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THE EFFECTS OF TARSAL TUNNEL RELEASE AND STABILIZATION PROCEDURES ON TIBIAL NERVE TENSION IN A SURGICALLY CREATED PES PLANUS FOOT

by

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A thesis submitted in conformity with the requirements
for the degree of Masters of Science
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The Effects of Tarsal Tunnel Release and Stabilization Procedures On Tibial Nerve Tension in a Surgically Created Pes Planus Foot.

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Abstract:

An *in vitro* study was performed on 11 cadaver legs to study the effects of tarsal tunnel release, distraction calcaneocuboid arthrodesis, and triple arthrodesis on tibial nerve tension in surgically created pes planus feet. Tibial nerve tension was measured during dorsiflexion, eversion, combined dorsiflexion-eversion, and cyclical load with 0°, 15°, and 25° of internal rotation, and repeated on the same leg following each surgical procedure.

A pes planus deformity significantly increased tibial nerve tension compared to baseline for all foot positions and cyclical load. A tarsal tunnel release significantly increased nerve tension during eversion, dorsiflexion-eversion, and cyclical load compared to the pes planus foot. A triple arthrodesis more effectively decreased nerve tension than a distraction calcaneocuboid arthrodesis.

Procedures producing skeletal instability (pes planus deformity and a tarsal tunnel release) increased tibial nerve tension. Procedures which corrected deformity and increased stability, decreased tibial nerve tension. In pes planus feet, if increased tibial nerve tension contributes to tarsal tunnel syndrome, then treatment with orthotics should be exhausted before considering surgery, which can further increase nerve tension.

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Introduction:

Tarsal tunnel syndrome is an uncommon disorder (43,105), which presents as a burning, or tingling pain located diffusely on the plantar aspect of the foot. Symptoms can be reproduced by compressing the tibial nerve, ankle dorsiflexion, and/or heel eversion. In 1960, Kopell and Thompson (58) described these symptoms and signs of tarsal tunnel syndrome. In 1962, the term tarsal tunnel syndrome was first used by Keck (55) and Lam (63) in independent publications. Initially, many authors (26,54,55,58,63,81,113,130) compared tarsal tunnel syndrome to carpal tunnel syndrome, and considered tarsal tunnel syndrome to be an entrapment neuropathy. However, further studies have shown that tarsal tunnel syndrome is more than just an entrapment neuropathy, and that these clinical entities differ with respect to anatomy, etiology, clinical presentation, and response to conservative and surgical treatment.

Since a significant number of the cases have an unknown etiology (15,105,113,135), and decompressing the tarsal tunnel has not been a satisfactory treatment for many patients (106), many authors (15,18,32,51,112) have studied increased tibial nerve tension in foot deformities to better understand the problem. This section of the thesis will focus on the anatomy, etiology, clinical presentation, diagnosis, treatment, and results of treatment in tarsal tunnel syndrome to understand the problem, and to understand the basis for the current investigation.

I. Anatomy:

Understanding the anatomy of the tarsal tunnel is important in understanding the clinical presentation, and the rationale of therapy. The tarsal tunnel consists of proximal and distal tarsal tunnels (44). The proximal tarsal tunnel contains the tibial nerve, and the distal tarsal tunnel contains the branches of the tibial nerve, which are the medial calcaneal nerve, medial plantar nerve, and lateral plantar nerve (44). The anatomy of the tarsal tunnel is illustrated in Figure 1a. Tarsal tunnel syndrome classically refers to the proximal tarsal tunnel, however, failure to recognize the existence of the distal tarsal tunnel has resulted in failure of therapy (32,44,81,126).

a. Tarsal Tunnel

The tarsal tunnel is a continuation of the deep posterior compartment of the leg leading to the sole of the foot (20). The proximal tarsal tunnel is a fibro-osseous space located behind the medial malleolus. It has an osseous floor formed by the medial surface of the talus, the sustentaculum tali, and the medial wall of the calcaneus, and is covered by the flexor retinaculum, which is also called the laciniate ligament. The flexor retinaculum is a continuation of the superficial and deep aponeurosis of the leg. The posterior border of the flexor retinaculum is continuous with the superficial and deep aponeurosis, and the anterior border is continuous with the dorsal aponeurosis of the foot. The base of the flexor retinaculum corresponds to the superior border of the abductor hallucis muscle, and separates into superficial and deep fascia for this muscle (124). Since the flexor retinaculum is continuous proximally and distally with the surrounding aponeurosis, it is difficult to accurately define the upper and lower borders (43). Dellon et al. (23) have defined the flexor retinaculum to be 2 cm on either side of the malleolar-calcaneal axis, which is a reference line drawn from the center of the medial malleolus to the center of the calcaneus. Thus, the flexor retinaculum roughly originates from the medial malleolus and extends posteriorly and inferiorly to the calcaneal tuberosity. Failure to recognize the fact that the flexor retinaculum is not a discrete structure and may extend up to 10 cm proximal to the medial malleolus may result in failure of surgical decompression (15).

The contents of the proximal tarsal tunnel include, from anterior to posterior: tibialis posterior tendon; flexor digitorum longus tendon; posterior tibial artery; tibial nerve; and flexor hallucis longus tendon. Each tendon has its own synovial sheath and is contained within a separate fibro-osseous compartment formed by fibrous projections from the undersurface of the flexor retinaculum to the periosteum of the calcaneus (25,26,50). The neurovascular bundle is often attached to these septa via a surrounding layer of dense areolar tissue (25). Since the tibial nerve is relatively fixed in its position, and is contained within a tight space formed by the fibrous septa, it is more susceptible to compression by a space-occupying lesion, or to tension.

b. Tibial nerve and its branches

The tibial nerve is one of two branches of the sciatic nerve within the popliteal fossa. It is a continuation of the sciatic nerve and enters the deep posterior compartment of the leg between the tibialis posterior and flexor digitorum longus. Then, it is found between the flexor digitorum longus and flexor hallucis longus, and emerges in the lower third of the leg medial to the medial border of the Achilles tendon to enter the proximal tarsal tunnel.

The branching of the tibial nerve is variable (23,43,48,78,124). In 93-96% cadaver feet studied, the tibial nerve bifurcated into the medial and lateral plantar nerves within the tarsal tunnel (23,43,48). In the remaining 4-7% of feet, the bifurcation was proximal to the tarsal tunnel. Dellon et al. (23) believed that a proximal bifurcation predisposed a patient to tarsal tunnel syndrome by "presenting a larger cross-sectional area at the narrowed entrance to the tarsal tunnel." Havel et al. (43) felt that the infrequent occurrence of a high bifurcation accounts for the low incidence of tarsal tunnel syndrome.

The medial calcaneal nerve arises most frequently from the tibial nerve (69-90%), and less frequently from the lateral plantar nerve (23,43,81). Havel et al. (43) reported that the medial calcaneal nerve had highly variable anatomy, with nine different branching patterns noted. A single nerve was identified in 79% of feet and multiple branches were noted in 21% of feet (43). However, the most common pattern was a single medial calcaneal nerve arising from the tibial nerve (43). The medial calcaneal nerve travels below the flexor retinaculum, or pierces it, and provides sensation for the medial and plantar aspects of the hindfoot.

The distal tarsal tunnel contains the medial and lateral plantar nerves (44). The medial and lateral plantar nerves pass through fibrous openings in the origin of the abductor hallucis. They are contained within separate fibrous tunnels separated by a partition originating from the medial side of the calcaneus and attached to the deep fascia of the abductor hallucis longus (44).

The medial plantar nerve, which is the largest division, passes superiorly to the abductor hallucis muscle and is bound by the plantar calcaneonavicular ligament. It is analogous to the median nerve of the hand. The medial plantar nerve provides sensation to the sole of foot and the medial three and half toes, and innervates the abductor hallucis, flexor digitorum brevis, flexor hallucis brevis, and first lumbrical muscle.

The lateral plantar nerve passes through a firm fibrous arch in the abductor hallucis muscle and passes beneath the muscle to run between the flexor digitorum below and the quadratus plantae above. It emerges between the flexor digitorum brevis and abductor digiti minimi muscles. It corresponds to the ulnar nerve of the hand. The lateral plantar nerve provides sensation to the lateral one and half toes and foot, and innervates the remaining intrinsic muscles of the foot.

II. Etiology:

The true incidence of tarsal tunnel syndrome is unknown (35). The specific cause of the tarsal tunnel syndrome can be identified in 60-80% of patients, but a significant number of patients have no identifiable cause (3,15,22,26,40,54,81,105,106,113,135). Many authors believe that tarsal tunnel syndrome is an entrapment neuropathy of the tibial nerve (10,15,25,26,55,58,63,64,78,81,96,113,129,130), but recently other etiological factors have been explored (18,32,51,58,81).

Cimino (15) reviewed the literature in tarsal tunnel syndrome in 1990. He reviewed 25 studies and case reports on tarsal tunnel syndrome. These studies included 164 patients with 186 tarsal tunnel syndromes. The ages ranged from 14 to 80 years old. There was a slight female predominance (56%) in the studies which reported sex. He noted that the commonest causes of tarsal tunnel syndrome were trauma (17%), varicosities (13%), heel varus (11%), fibrosis (9%), and heel valgus (8%). These factors can be broadly classified into: (1) trauma; (2) space-occupying lesions; and (3) foot deformity.

a. Trauma

The commonest cause of tarsal tunnel syndrome identified in Cimino's review was trauma (17%). Displaced fractures of the distal tibia, tarsal bones, and calcaneus, and ankle sprains involving the deltoid ligament can reduce the cross-sectional area of the tarsal tunnel, and compress the tibial nerve producing the syndrome (26,64,68,113,129). Since the tendons of the posterior tibialis, flexor digitorum longus, and flexor hallucis longus are in close proximity to the tibial nerve within the proximal tarsal tunnel, traumatic flexor tenosynovitis can also compress the tibial nerve within the closed space, producing the syndrome (15,32,51,58,64,125).

b. Space-Occupying Lesions

Space-occupying lesions can compress the tibial nerve either from inside or outside the tarsal tunnel. In Cimino's review, the commonest space-occupying lesion identified was varicosities (26,37,39). Other space-occupying lesions reported in the literature include, ganglia (56,78,83,113,134), perineural fibrosis (113), lipoma (104), neurilemoma (50,89,113,134), bony exostosis (15), medial talocalcaneal bar (81,135), hypertrophic flexor retinaculum (15), hypertrophic or accessory abductor hallucis muscle (25,26), flexor digitorum accessorial longus muscle (122), rapid weight gain (15,26), fluid retention (15), and chronic thrombophlebitis (15). Generalized inflammatory arthropathies such as, rheumatoid arthritis (3,13,29,40,69,96), and ankylosing spondylitis (28,61,101) produce a proliferative synovitis, which can have a mass effect on the tarsal tunnel, like flexor tenosynovitis.

c. Foot Deformities

When varus and valgus foot deformities are combined, foot deformity was the commonest identifiable cause of tarsal tunnel syndrome. In Cimino's review, 11% of patients had varus heels, and 8% of patients had valgus heels.

Radin (111) published the only article to discuss the varus heel deformity in tarsal tunnel syndrome. He stated that two-thirds of patients with tarsal tunnel syndrome had concurrent varus heel and pronated forefoot. He believed that the pronated forefoot deformity compensated for the varus heel allowing the midfoot and forefoot to contact the ground. The varus heel deformity would shorten the abductor hallucis muscle, making it appear hypertrophied, and decrease the cross sectional area of the tarsal tunnel. The pronated forefoot would stretch the plantar nerve. Thus, the combination of these deformities would compress and stretch the tibial nerve, producing the syndrome.

Many authors believe that the valgus heel and abducted forefoot in a pes planus foot causes tarsal tunnel syndrome by applying tension to the tibial nerve (18,22,32,51,55,58,63,69,78,81). Francis et al. (32) reported on 11 patients with pes planus deformity and tarsal tunnel syndrome. This is a rather unique study since no published reports have identified such a large group of patients with a pes planus deformity and tarsal tunnel syndrome, and of the 6 patients who underwent surgical release, no space-occupying

lesion could be identified. All patients had a pes planus deformity, and 8 patients had mobile deformities. Calcaneal hindfoot valgus was identified in 7 patients with mobile pes planus deformity, and 1 patient with fixed pes planus deformity. The majority of patients described paresthesias "like an electric shock with each step taken." Francis et al. believed that "pes planus, especially when mobile and aggravated by associated hindfoot valgus, puts the posterior tibial nerve and/or its medial and lateral plantar branches on stretch with each weight bearing step." This makes the nerve vulnerable to neuropraxia with any additional injuries. They felt that a trivial trauma could initiate the tarsal tunnel syndrome, and the pain would intensify as the patient continues with weight bearing.

Dynamic foot deformities producing tarsal tunnel syndrome have also been described in the literature (15,51,64,112). Rask (112) reported on "Jogger's foot," which is produced by valgus running that stretches the medial plantar nerve against the navicular tarsal bone above. Jackson et al. (51) believed that running with excessive pronation would stretch the tibial nerve, predisposing it to injury. In addition, occupations associated with repeated dorsiflexion (13,14,15), such as, repeated crouching, and squatting, have been reported to be associated with tarsal tunnel syndrome.

III. Pathophysiology of Neuropathy

Tarsal tunnel syndrome is produced by tibial nerve damage caused by either compression (25,58,64,115) or tension (18,32,51,58,81). Tibial nerve compression is clearly recognized as the commonest cause of tarsal tunnel syndrome, but tibial nerve tension has only recently become the focus of investigation.

a. Nerve Compression

In Kopell and Thompson's classic paper (58), an entrapment point was defined as a location "where the anatomic configuration may cause constriction of the nerve, or, if trauma has occurred to the area, the particular anatomic disposition will maintain the nerve in a state of inflammation." Entrapment occurs because the osseofibrous tunnel is made of strong and unyielding tissues compared to the nerve, and "there is little latitude for dimensional alteration in the cross-sectional area permitted for the passage of the nerve." This produces a pressure effect on the nerve and its blood supply, causing axonal demyelination. The nerve

is injured directly by neural compression, or indirectly by causing local vascular insufficiency (25).

In tarsal tunnel syndrome, the initial sensory dysfunction is thought to be due to nerve ischemia caused by local compression, while motor dysfunction, which occurs later, is due to direct neural compression and structural alteration (25,64). The tibial nerve is especially susceptible to entrapment because it is relatively fixed by fibrous septa from underneath the flexor retinaculum, is confined within a fibrous tunnel formed by these fibrous septa, and requires a good arterial blood supply (25,26,50,115).

Entrapment of the tibial nerve in the tarsal tunnel is most common at the anterior, inferior aspect of the canal (22,32,34,58), and the lateral plantar nerve is more commonly affected than the medial plantar nerve (32,35,50,54). The lateral plantar nerve passes through a fibrous opening in the abductor hallucis muscle separately and more proximal than the medial plantar nerve (26), and is probably more vulnerable (54). It has also been postulated that a high bifurcating tibial nerve is more susceptible to compression since it has a larger cross-sectional area at the narrowed entrance to the tarsal tunnel (23,43).

b. Nerve Tension

In a pes planus foot, the valgus heel and abducted forefoot stretches the tibial nerve (18,22,32,51,55,58,63,69,78,81). The tibial nerve can be damaged by an acute, and/or chronic stretch (5,19,125), and the severity of the injury depends on the magnitude of the force, and the rate of deformation (5).

Peripheral nerves are normally under tension *in situ* (5,117). Mechanisms exist which allow the nerve to shorten and lengthen to accommodate motion of the joint: (1) removal of slack from the nerve trunk; (2) the paraneurium above the epineurium allows the nerve to slide; (3) the endoneurial tissue accommodates compression, which occurs with reduction in cross-sectional area; and (4) the undulated arrangement of nerve fibers straighten with lengthening and coil up with shortening (5,90). However, these mechanisms have limitations, and sustained increases in stress, even < 10% of the ultimate stress of the nerve can have dramatic effects on nerve conduction (62).

Acute stretch injury to the tibial nerve can result from severe trauma due to a fracture-dislocation of the foot or ankle, or a severe ankle sprain. Chronic, recurrent microtrauma to

the tibial nerve, which is probably more common in patients with a pes planus deformity, is due to recurrent ankle sprains, or walking or running with a hyperpronated foot, which stretches the tibial nerve with each weightbearing step. The effects of acute stretch injuries on nerves have been extensively studied in animal models, but the effects of chronic, recurrent microtrauma have been less clearly defined.

In earlier studies, the amount of nerve elongation reported to produce nerve damage was inconsistent, ranging from 4-100% (6,24,42,45,46,47,71,117,132), due to inaccurate measurements of strain. In early *in vitro* measurements, a machine was used to stretch the nerve, but since the ends were difficult to fix, slippage occurred and the final distance between the clamps would overestimate the final length, and consequently overestimate the strain (5). Recently, strain has been more accurately measured *in vivo*, and *in vitro* by making two fixed marks on the nerve, and measuring the change in length between the two marks using more accurate techniques, such as digital calipers or video digital analyzers (5,75,100,117,138).

i. Acute Injury

As peripheral nerves are stretched, slack is removed from the nerve trunks and undulations in the nerve fiber straighten. With continued stretching, the fascicles are stretched, and ischemia results from two factors. Firstly, the normally coiled and tortuous vessels are straightened and with further stretching, extrinsic and intrinsic blood supply are strangulated (75). Secondly, tension reduces the cross sectional area within the epineurium, which increases the intrafascicular pressure, and compresses the blood vessels (75). Since peripheral nerves require an adequate and continuous supply of oxygen to function (73,75,100), ischemia impairs nerve conduction.

Venular flow obstructs at 8% elongation, and complete arterial occlusion occurs at 15% elongation, but perineural integrity remains intact (75,100). This agrees with compound nerve action potential studies which show reversible reduction in action potentials with 6% elongation, and irreversible reduction with 12% elongation (138).

Complete ischemia produces rapid deterioration in nerve function, which is reversible when blood flow is restored, but if the stretch is sufficiently prolonged, ischemia can cause irreversible damage (72,119). Obstruction to blood flow stops delivery of oxygen, and the

production of ATP by aerobic metabolism. Nerve function is impaired by interference with energy dependent mechanisms responsible for conduction: (1) axoplasmic transport of proteins are required for maintaining the structural integrity of the axon, supporting action potential propagation, and permitting neurotransmitter release at the presynaptic terminals, and (2) ATP-dependent sodium-potassium pumps are required for transfer of ions across axonal membrane and the propagation of nerve impulses (5,66,99,119). Restoration of blood flow restores nerve function (72).

If ischemia continues, endothelial vessel permeability increases leading to irreversible nerve damage despite restoring blood flow (72,119). The intrafascicular environment is highly specialized and is maintained by the selective permeability of the endoneurium and perineurium (60,73,102,118,119). With ischemia, endoneurial capillaries become more permeable, producing edema, which can impair nerve conduction directly, by altering ionic balance within the nerve fiber (72), or indirectly, by increasing endoneurial pressure and causing collapse of capillaries, further perpetuating the cycle (70,110). Later, fibroblasts producing intraneural scarring can damage neural structure (73). The perineurium, on the other hand, is highly resistant to ischemia (59,74,128).

As elongation approaches the elastic limit, which is the point where further elongation leads to permanent deformation, mechanical failure occurs sequentially (5,131) at the nerve fibers within the fascicles (second degree injury), endoneurial tubes within the fascicles (third degree injury), the perineurium (fourth degree injury), and the epineurium (fifth degree injury).

The nerve fiber is composed of axons, myelin sheath, and Schwann cells. Ultrastructural studies of axons in guinea pig optic nerves subjected to a sudden acute stretch show that mechanical failure of the axon can occur by one of two mechanisms depending on the amount of force used to stretch the nerve. With a severe stretching force, physical tearing of the axon occurs at the moment of injury resulting in primary axotomy. With lesser amounts of force, secondary axotomy results in axonal disconnection over a few hours due to axonal injury, which produces axonal swellings that undergo expansion, and rupture (85,88,109,136). These axonal changes produce significantly decreased mean amplitude and increased latency of visual evoked potentials in the acute post-traumatic phase (136). The key initiating event of secondary axotomy is thought to be the disruption of the axolemma

(87,88). This alters ionic homeostasis, and in particular, an influx of calcium ions (84,86,88) is believed to produce the disorganization and/or disintegration of the microtubules and neurofilaments seen (85,87,49,136), which interferes with axonal transport. Since axonal transport is disrupted, organelles accumulate at the site of injury, producing progressive axonal swelling and eventually rupture, which leads to axonal disconnection (88,109,136).

The strongest component of a peripheral nerve is the perineurium (5,24,62,117,132,133), which fails at 27% beyond *in situ* strain (117). After failure of the perineurium, epineural edema can alter the highly specialized environment within the fascicles (72), which impairs neural conduction acutely. Furthermore, leakage of proteins into the fascicles, may lead to fibrosis, which jeopardizes the return of normal structure (73,75). Thus, failure of the perineurium represents a major injury to the nerve (73,118,119).

ii. Chronic Injury

Unlike in acute stretch injuries, nerves can tolerate a greater stretch over a longer period of time (27,91,92). This is probably due to the tensile forces being more slowly and evenly distributed throughout the nerve compared to the acute situation (27,91,92), and/or greater stress relaxation of the nerve with a slow and gradual stretch (138). Studies in rat sciatic nerves elongated gradually with tissue expanders showed that elongation of the nerve by more than 50% significantly reduced conduction compared to 10-50% elongation, and that slow expansion over 15 days was better tolerated than over 3 days (91). With even more gradual and slow elongation of the sciatic nerve over 8 weeks, an average increase in length of 88% occurred without significantly altering the intraneural cytoskeleton (27). Though tissue expanders provide an ideal method for gradually elongating a nerve, nerve tension is not the only deforming force since the nerve is also subjected to compression by the expander, which will contribute to the overall pathology observed.

Over time, chronically injured nerves become stiffer because of fibrosis within the epineurium (4), and are unable to compensate for tension as easily as normal nerves, which makes them more susceptible to ischemia and injury. Though the effects of chronic and gradual elongation on peripheral nerves has been studied, the effects of chronic and repetitive elongation on peripheral nerves has not been quantified.

IV. Clinical Presentation:

Since the sensory modalities in the foot are not perceived as distinctly as those in the hand, the symptoms of tarsal tunnel syndrome can be diffuse and poorly localized (22,50). This makes the diagnosis of tarsal tunnel syndrome difficult, and unfortunately, it is often undiagnosed or misdiagnosed (15,22,25,34,35,43,50,55,64,78,81).

a. History

Patients with tarsal tunnel syndrome may have a previous history of significant or trivial trauma to the foot which precipitates the pain. It usually presents with an insidious onset of pain and paresthesia located diffusely on the plantar aspect of the foot. Symptoms are intermittent at first, and then become constant (40). The location of the pain will vary depending on which nerves are involved, and the pain is characterized as a burning, tingling or numb type of pain that can radiate distally along the involved nerve branches. In one third of patients with severe compression of the nerve, they will experience proximal migration of pain along the medial aspect of the leg, called the Valleix phenomenon (22,26,55,63,111).

Some patients may complain of pain with each step (32,78). Generally, the amount of burning and aching pain is proportional to the amount of standing and walking during the day (22,26,40,50,52,81,101). The pain is relieved by rest, elevation and loose shoes. Some patients have pain at night, severe enough to wake the patient, which is relieved by walking (25,26,40,58,63,64,78,89). This may be due to venostasis and engorgement of the tibial veins at night (25).

b. Physical Examination

The foot is examined for any significant hindfoot varus or valgus deformity (15,22,32,51,55,58,63,69,78,81,111). The course of the nerve and its branches is palpated for any evidence of thickening or swelling, which may indicate evidence of a space-occupying lesion (10,50,63,64,81,125). The range or motion of the ankle, subtalar, and transverse tarsal joints is measured.

Although patients may complain of dysesthesias and numbness, it is difficult to detect areas of numbness on the bottom of the foot (22,64,81). However, the earliest sign of this condition will be decreased two point discrimination on the plantar aspect of the foot (64),

and as the condition progresses, hypoesthesia to pinprick occurs in the distribution of the involved nerve. Weakness of the intrinsic muscles is assessed by testing plantarflexion of the toes, especially the lateral toes. However, motor weakness is an uncommon finding, and is difficult to assess (22,50,78). In the advanced stages, atrophy of the abductor hallucis or abductor digiti quinti may be observed. On the other hand, the medial arch may appear full due to hypertrophy of the abductor hallucis or an accessory abductor hallucis, which will compress the nerve (15).

More objective physical findings of tarsal tunnel syndrome are found by exacerbating tibial nerve compression or tension, which reproduces symptoms. Palpation and percussion of the tibial nerve, and its branches can cause tenderness, and reproduce symptoms in the irritated regions of the nerve. Tinel's sign, which is the reproduction of pain by compression of the tibial nerve behind the medial malleolus, is a very specific finding (10,22,55,63,64,68,78,129). This may also produce proximal migration of the pain into the leg. The Cuff test involves inflating a pneumatic pressure cuff around the leg, which acts as a venous tourniquet that causes venous engorgement, increases local ischemia, and reproduces symptoms (15,25,26,40,64,68,78,111).

Forcing the ankle into maximal dorsiflexion (13,14), or forcing the heel into eversion (15,22,26,40,55,101,106) may aggravate symptoms, by increasing the tension on the tibial nerve. Some authors believe that forcing the heel into inversion relieves symptoms by reducing tibial nerve tension (26,40,55,106), while others believe that it reproduces symptoms by reducing the cross-sectional area of the tunnel, and compressing the tibial nerve (15,25,50,64,111). These conflicting results can be resolved when the pathology of the tarsal tunnel syndrome is considered. Since tibial neuropathy results from nerve compression or tension, the physical maneuver which exacerbates the causative factor will reproduce symptoms. Thus, in a tibial nerve which is stretched, heel eversion will increase nerve tension and reproduce symptoms, while heel inversion will reduce nerve tension and relieve symptoms. Though heel inversion reduces the cross-sectional area of the tunnel, the nerve can accommodate the reduction in volume without any deleterious effects. On the other hand, tarsal tunnel syndrome resulting from a compression cannot accommodate any further reduction in tarsal tunnel cross-sectional area, and so inversion of the heel would further compress the nerve, and reproduce symptoms.

c. Conclusion

Misdiagnosis of tarsal tunnel syndrome is common because the symptoms are poorly localized, and physical findings are variable. To improve diagnostic accuracy, the clinician must have a high degree of suspicion for the diagnosis, and must search for common etiological factors such as, trauma, space-occupying lesions, and foot deformity, on history and physical examination. Maneuvers which reproduce symptoms, such as the Tinel's sign, Cuff test, ankle dorsiflexion, or heel inversion or eversion, are the most objective physical findings.

V. Diagnosis:

Since the history and physical examination for tarsal tunnel syndrome may not rule out other diagnoses, electrodiagnostic studies and radiological imaging are useful tests for clarifying the diagnosis of tarsal tunnel syndrome.

a. Electrodiagnostic Studies

Electrodiagnostic studies measure nerve function, and can be classified into motor, sensory, or mixed compound nerve tests. The rate an action potential travels along a nerve can be expressed as: (1) velocity, which is the ratio of distance and time; and (2) latency, which is the time interval between the stimulation and response. Latency is more useful in measuring conduction in short nerve segments (37). Though electrodiagnostic studies are useful objective diagnostic tests, controversy surrounds which test is the most sensitive and specific for diagnosis (22,34,35,37,41,52,54,105). The development of electrodiagnostic studies in tarsal tunnel syndrome parallels the evolution of the technology.

i. Motor Studies

The motor conduction velocity, and distal motor latency of the medial and lateral plantar nerves were the first studies used to diagnose tarsal tunnel syndrome. Goodgold et al. (37) believed that abnormal distal motor latencies in either the medial or lateral plantar nerves or both, were objective evidence for tarsal tunnel syndrome. Johnson et al. (52) reported that abnormal distal motor latencies were more sensitive than conduction velocity in detecting tarsal tunnel syndrome. They considered distal motor "latencies in excess of one

standard deviation as indicating the presence of tarsal tunnel syndrome." Fu et al. (34) standardized the measurement of distal motor latencies to control for limb temperature, and distance of segment stimulated, both of which influenced measurements, and defined abnormal latencies as those in excess of two standard deviation. Since Johnson et al. defined abnormal latencies as those in excess of one standard deviation, up to 16% of normal patients would be considered abnormal. In the presence of normal distal motor latencies, Fu et al. (34) advocated electromyographic studies of the intrinsic muscles supplied by the medial and lateral plantar nerves, which often showed positive sharp waves, and fibrillation potentials. Kaplan et al. (54) showed that reduced amplitude and increased duration of motor evoked potentials were more sensitive indications of neuropathy than motor distal latencies, especially for the medial plantar nerve.

ii. Sensory Studies

The next development in electrodiagnostic studies was the measurement of sensory action potentials. Since sensory action potentials are much smaller than motor action potentials, measurement of these action potentials was more difficult due to limitations in equipment (12,41,105,108). Guiloff et al. developed a technique for measuring sensory action potential (41), which was further refined by Ponsford (108), Buchthal et al. (12), and Oh et al. (105). Oh et al. (105) reported that 90.5% of patients with tarsal tunnel syndrome had abnormal sensory conduction compared to only 54.4% of patients with abnormal terminal motor latency. Abnormal sensory conduction was defined as absence of nerve potential, or slow sensory nerve conduction velocity in excess of two standard deviations. They concluded that normal distal motor latencies alone were inadequate to rule out tarsal tunnel syndrome, and that sensory nerve conduction velocity was superior to distal motor latency for making the diagnosis. Since sensory function is impaired before motor function, and patients usually present with sensory complaints before motor findings (25,64), it makes sense that sensory conduction velocity is affected before distal motor latency.

iii. Mixed Motor and Sensory Studies

The most recent development in electrodiagnostic studies are the mixed motor and sensory action potential. Compared to sensory action potentials, mixed motor and sensory

action potentials are much larger, and predominantly, represent sensory fibers (22). Saeed et al. (120) measured mixed motor and sensory action potentials across the tarsal tunnel, and showed that they were more sensitive for detecting early change. DeLisa et al. (22) recommended that "medial and lateral plantar motor distal latencies, sensory or mixed compound nerve studies, and the needle examination of selected intrinsic foot muscles" be performed for suspected tarsal tunnel syndrome. Galardi et al. (35) studied the sensitivity and specificity of distal motor latencies, sensory conduction velocities, and mixed motor and sensory conduction velocities in 13 patients with tarsal tunnel syndrome. The distal motor latencies were relatively insensitive (21.5%), but the sensory conduction velocities were too sensitive (92.8-100%). Sensory conduction velocity was abnormal in 8% of normal controls tested, which reflects its sensitivity for detecting early peripheral neuropathy (35). There was no abnormal mixed motor and sensory conduction velocities in asymptomatic limbs or normal controls, but 85.7% of patients with tarsal tunnel syndrome had abnormal studies. They concluded that only the coexistence of abnormal sensory, and mixed motor and sensory conduction velocities, especially if asymmetric, were highly sensitive and specific for tarsal tunnel syndrome.

Mann (81) recommends that tarsal tunnel syndrome be diagnosed only when the following triad exists: pain and paresthesia in the foot; positive Tinel's sign; and positive electrodiagnostic studies. If all three findings are present, then tarsal tunnel syndrome is diagnosed. If only two of the three findings exist, then the patient should be carefully followed, and if the findings are reproducible, then the diagnosis should be considered. If only one of these findings are present, then other diagnosis should be considered.

However, Skalley et al. (126) reported that the clinical history and physical examination were more helpful in defining the extent and location of the tibial nerve irritation than electrophysiologic studies. Likewise, Pfeiffer et al. (106) reported no correlation between results of electrodiagnostic studies and clinical results. They recommended that electrodiagnostic studies should only be used to confirm the clinical diagnosis. Cimino calculated the false negative rate of electrodiagnostic studies to be at least 9.5%, and he believed that these studies were relatively insensitive, and that a negative test did not preclude the diagnosis (15).

Electrodiagnostic studies definitely have a role in the diagnosis of tarsal tunnel syndrome. They should be performed on the medial and lateral plantar nerves. Clearly, one electrodiagnostic study alone is not adequate to diagnose or to rule out tarsal tunnel syndrome. Sensory conduction velocity, mixed motor and sensory conduction velocity, and electromyographic studies have proven to be the most useful combination of tests. However, since electrodiagnostic studies have an accuracy of only 90% under the best circumstances (35,81), in cases with a clear history and physical examination, negative studies do not rule out the diagnosis of tarsal tunnel syndrome (15).

b. Radiological Investigations

Radiological investigations also play a key role in diagnosis by identifying the anatomy within the tarsal tunnel. Plain x-ray examination of the foot identifies displaced fractures, accessory ossicles, or bony exostoses within the vicinity of the tarsal tunnel. Weight bearing x-rays of the foot and ankle are also useful for determining the presence of a foot deformity. Venography may be indicated in patients with a positive cuff test (139).

In the absence of a bony abnormality on plain x-rays, magnetic resonance imaging is the most effective method for assessing the contents of the tarsal tunnel, the flexor retinaculum, and the branches of the tibial nerve (29,33,142). It can image the foot and ankle in all three orthogonal planes, and different computer algorithms can be used to study different tissues: (1) T1 weighted images demonstrate bony anatomy; and (2) T2 weighted images delineate soft-tissue masses, fluid collections, and inflammatory conditions (142). For surgical planning, it is extremely useful for localizing a lesion, determining the extent of a lesion, and determining the location of the lesion relative to the tibial nerve (29,33,142). This provides invaluable information for planning surgical exploration, especially considering the anatomical variation within the region, and the possibility of an inadequate decompression.

In Frey et al.'s study (33) involving 29 feet with tarsal tunnel syndrome proven by electrodiagnostic studies, 88% of patients had a lesion identified by magnetic resonance imaging. These lesions included: tenosynovitis, varicosities, masses, and scar tissue. They recommended the use of magnetic resonance imaging in children, who tended to have

neoplastic mass lesions, and in surgical candidates, to help plan surgery. Zeiss et al. (142) also recommended its use for failed decompressions of the tarsal tunnel.

VI. Treatment and Results:

The treatment of tarsal tunnel syndrome is classified into non-operative, and operative methods. In all cases, an adequate trial of non-operative treatment should be instituted before considering surgical therapy. If symptoms do not improve after 6-8 weeks, or if the symptoms worsen, then surgical decompression is indicated. However, many authors believe that non-operative treatments provide transient relief, and that surgery is the only effective treatment (26,68,78,135).

a. Non-Operative Treatment

The non-operative treatments recommended in tarsal tunnel syndrome are rest, immobilization, oral non-steroidal anti-inflammatory drugs, local corticosteroid injections, physiotherapy, orthotics, and stockings (25,26,32,51,54,61,69,81,89,96,101,104,111,112,125, 130,135).

The immobilization methods include, ankle-foot orthosis (54), and occasionally, a walking cast (81,89,139). Oral non-steroidal anti-inflammatory drugs, and local corticosteroid injections into the region have only a transient effect in many cases (26,32,54,106), but in inflammatory arthritis, such as rheumatoid arthritis or ankylosing spondylitis, where tenosynovitis is a factor, they have proven to be effective treatments (13,61,69,96,101,106,130). Physiotherapy to strengthen the intrinsic muscles of the feet, the flexor digitorum longus, flexor hallucis longus, and tibialis posterior (32,61,112,125), have been recommended to restore the medial longitudinal arch, and stabilize the foot in a neutral position. In runners with "Jogger's foot" where a hyperpronated foot stretches the tibial nerve, patients are instructed to toe in slightly to strike the lateral aspect of the foot, decreasing the deformity (112).

Orthotics, which hold the foot in a neutral position, are effective if the foot deformity is flexible (15,25,32,111). In flexible valgus heels, medial longitudinal arch supports, and medial heel wedges have been successfully used (25,32). For more severe deformities, UCBL (University of California Biomechanics Laboratory) type inserts, which hold the

calcaneus in neutral alignment and force the abducted forefoot into neutral, can be used (82). This can be supplemented with a polypropylene ankle foot orthosis for more control of the deformity. Lateral heel wedges effectively treat flexible varus heels, while medial arch supports aggravate symptoms and should be avoided (111). In patients who have symptoms reproduced by dorsiflexion, Chater reported that an one inch heel lift helps by decreasing tension on the tibial nerve (13,14).

Stockings are used to decrease the amount of swelling in the leg, and the amount of venous stasis present, which may be a factor when varices are present within the tarsal tunnel (25,81,89,125). In obese patients, some authors recommend weight loss (26,112).

In Cimino's review, only 16% of patients were treated non-operatively. Orthotic management was successful in 34% of patients, but all patients had flexible deformities. Spontaneous resolution or corticosteroid injection was successful in 24%.

Though non-operative therapy is the first line of treatment, patients should be warned that it is effective in only certain situations. The most effective non-operative treatments are directed at specific etiological factors such as, (1) orthotics for flexible foot deformities; and (2) oral non-steroidal anti-inflammatory drugs, and/or local corticosteroid injections for tenosynovitis, and inflammatory arthritis. On the other hand, non-operative treatments will have no significant effects on space-occupying lesions within the tarsal tunnel, with the exception of tenosynovitis and other inflammatory conditions (15,106,135).

b. Operative Treatment

When tarsal tunnel syndrome results from a space-occupying lesion, surgical treatment provides predictably good results (10,25,26,50,81,89,106,135). Surgical treatment was described by Lam (64), and consists of release of the flexor retinaculum, neurolysis of the medial and lateral plantar nerves as far distally as possible, and surgically treating any etiological factors, such as repairing fractures to restore normal spatial arrangement, excising a mass lesion, or resecting synovium from tenosynovitis.

The tibial nerve is exposed proximal to the flexor retinaculum through a curved medial incision starting 10 cm proximal to the medial malleolus and 2 cm posterior to the margin of the tibia, and extending towards the talonavicular joint. The flexor retinaculum is completely divided, and the tibial nerve is mobilized along its course as far distally as

possible. The medial and lateral plantar nerves must be adequately decompressed at least 2 cm distally into the sole of the foot. The medial plantar nerve is followed distally as it passes in its fibrous tunnel inferior to the talonavicular joint. The lateral plantar nerve travels behind the abductor hallucis muscle, and part of the origin of the abductor hallucis may have to be released to adequately expose the nerve. Post-operatively, the patient remains non-weight bearing for 3 weeks, and then weight bearing is permitted as tolerated, and active and passive range of motion is encouraged. Failure to recognize the proximal extent of the flexor retinaculum, and to decompress the distal tarsal tunnel can result in failure of therapy (126,142).

In Cimino's review (15), 65% of patients required surgical treatment, and 91% had good or improved results. This agrees with surgical results from the literature, which have a range of 79-95% good results (26,54,64,68,78,105,111). Surgical results were better when patients were younger (135), when surgery was performed early within the disease process before motor involvement (64,135), and when a localized lesion was identified (10,25,26,50,81,89,106,135). Takakura et al. (135) reported that coalition and tumours gave the best surgical results, while idiopathic causes, and trauma were the worst. They felt that trauma caused hemorrhage within the tarsal tunnel producing subsequent adhesions.

With poor results from non-operative treatment methods and excellent results from tarsal tunnel release, many authors have advocated surgical therapy for lasting results in tarsal tunnel syndrome (25,26,78,81). However, a recent study by Pfeiffer and Cracchiolo (106) has cast doubt on the long term effectiveness of surgery. In 30 patients, they reported that only 44% of patients benefited markedly from surgical treatment (106). Compared to previous studies, these results differed because they used more precise and stringent subjective questionnaires, and they had a minimum follow-up of 24 months with an average of 31 months, which is considerably longer than many previous studies (26,54,64,78,105,111). They noted that early results of surgery were encouraging, but later, results deteriorated. They concluded that "unless there is an associated lesion near or within the tarsal tunnel preoperatively, decompression of the posterior tibial nerve should be considered with caution," and patients with previous surgery for pain in the foot, plantar fasciitis, or symptoms of systemic inflammatory disease should be managed conservatively.

Rationale:

Tarsal tunnel syndrome is commonly idiopathic (3,15,22,26,40,54,81,105,106,113, 135). In a recent review (106), long term follow-up of tarsal tunnel syndrome treated surgically showed poor results. In idiopathic cases, many authors have discouraged tarsal tunnel release because of poor clinical results (81,106,135). To improve clinical results, the etiological factors causing tarsal tunnel syndrome must be recognized, and understood better so that appropriate therapy can be performed to correct the cause. Tarsal tunnel syndrome can be produced by compression or tension of the tibial nerve. Compression is a clearly recognized factor (10,15,25,26,55,58,63,64,78,81,96,113,129,130), but very few authors (26,32,51,55,58,63,81) have discussed nerve tension in a pes planus deformity as a cause.

The normal foot is a unique biomechanical structure. It becomes supple during the early part of stance phase to adapt to uneven terrain, and then becomes rigid during the latter part of stance phase for push off (80). However, pes planus feet are excessively everted and supple, and cannot convert to a rigid structure for push off (7,8,9). Thus, the pes planus foot is relatively unstable and hypermobile compared to a normal foot. The pes planus deformity consists of excessive valgus of hindfoot, and abduction of the forefoot compared to a normal foot (see Figure 1b). As a result of the valgus heel and abducted forefoot, the lateral plantar nerve is stretched as it wraps around the calcaneus (18), which may produce nerve damage and impair nerve conduction (see Figure 1a). Forced eversion of the heel (15,22,26,40,55,101,106) and dorsiflexion (13,14) of the ankle reproduce symptoms by increasing nerve tension.

The only treatment method in the literature designed to correct excessive tibial nerve tension in tarsal tunnel syndrome is orthotics designed to: (1) neutralize the foot deformity, such as the medial heel wedge with posting along the medial aspect of the first metatarsal, and UCBL-type insert +/- polypropylene ankle foot orthosis, or (2) restrict dorsiflexion, such as an one inch heel lift. However, orthotics are only useful in shoes, and have a limited amount of correction in flexible deformities. Surgical options to correct a severe and rigid pes planus deformity consist of bony realignment procedures, such as calcaneal osteotomies, distraction calcaneocuboid arthrodesis, and double or triple arthrodesis.

The goal of this investigation was to study the role of nerve tension in tarsal tunnel syndrome and pes planus feet. This investigation was divided into two parts: (1) Part I: The

effect of foot position and load on tibial nerve tension; and (2) Part II: The effects of tarsal tunnel release and stabilization procedures on tibial nerve tension in a surgically created pes planus foot. In the first part of the study, a new method for measuring nerve tension had to be developed so that accurate and reproducible measurement was possible during foot position and cyclical load, and the validity of measuring nerve tension after surgically creating a pes planus deformity had to be determined. After successfully completing the first part of the study, the second part of the study examined the effects of treatments, such as tarsal tunnel release and stabilization procedures, on tibial nerve tension so that treatments directed specifically at nerve tension are used appropriately.

Hypotheses:

1. Tension on the tibial nerve is influenced by foot position and load with internal rotation.

2. The amount of tension transferred to the nerve is dependent on skeletal stability of the foot.

3. A tarsal tunnel release has no effect on tibial nerve tension.

4. A stabilization procedure, such as a distraction calcaneocuboid arthrodesis or triple arthrodesis, decreases tibial nerve tension in a surgically created pes planus foot.

Objectives:

1. To measure tibial nerve tension in dorsiflexion, eversion, and combined dorsiflexion-eversion, and cyclically loaded with increasing internal rotation of 0°, 15°, and 25°.

2. To measure tibial nerve tension in dorsiflexion, eversion, and combined dorsiflexion-eversion, and cyclically loaded with increasing internal rotation of 0°, 15°, and 25° in a surgically created pes planus foot.

3. To measure tibial nerve tension in dorsiflexion, eversion, and combined dorsiflexion-eversion, and cyclically loaded with increasing internal rotation of 0°, 15°, and 25° in a surgically created pes planus after a tarsal tunnel release.

4. To measure tibial nerve tension in dorsiflexion, eversion, and combined dorsiflexion-eversion, and cyclically loaded with increasing internal rotation of 0°, 15°, and 25° in a surgically created pes planus foot with a tarsal tunnel release after a stabilization procedure, such as a distraction calcaneocuboid arthrodesis or triple arthrodesis.

Part I: The effects of foot position and load on tibial nerve tension (18)

Introduction:

Tarsal tunnel syndrome is a debilitating problem for patients, and the results of surgical decompression are less than satisfactory. Patients with tarsal tunnel syndrome of unknown etiology do poorly following surgical decompression (81,106,135). Surgical results in this patient population are so poor that some authors recommend that it not be considered a treatment option (106). Several studies that have closely assessed ligamentous laxity and the shape of the foot have found a high incidence of a pes planus deformity in this subgroup (3,32,40).

A pes planus deformity results in increased abduction of the forefoot and valgus of the hindfoot. The increased abduction of the forefoot may increase the stretch on the tibial nerve as it wraps around the plantar surface of the calcaneus; in addition, the valgus position of the hindfoot may place increased compressive forces on the tarsal tunnel contents. Releasing the tarsal tunnel contents would not alter stretch forces on the tibial nerve, nor would it relieve the compressive forces on the tibial nerve due to stretch. These abnormal forces could only be addressed by stabilizing the foot in a corrected position.

If instability of the hindfoot and midfoot could be demonstrated to be an etiologic factor in tarsal tunnel syndrome, it would greatly alter our understanding of this problem. The primary goal of this study was to determine the effects of tarsal instability on the tibial nerve tension within the tarsal tunnel. Baseline measurements of tibial nerve tension were recorded with the foot in dorsiflexion, eversion, and combined dorsiflexion-eversion, and under cyclical load with internal rotation of the tibia at 5° intervals from 0° to 20° to simulate the early stance phase of gait. Then, a pes planus deformity was surgically created and its effect on tibial nerve tension under the previous conditions was reassessed.

Materials and Methods:

This study was performed in two parts. The first section concentrated on the tension placed through the tibial nerve for a baseline foot. The second section measured nerve tension following the creation of a pes planus deformity.

An *in vitro* study was performed on 9 fresh frozen cadaver limbs. The cadaver limbs came from 5 males and 2 females, with an mean age of 75.5 years (1 unknown). There were

5 right, and 4 left specimens, with 2 bilateral specimens. All the cadavers were received in the anatomy department within 24 hours of death, and preserved by freezing without embalming. There was no major structural abnormality present in the cadaver limbs. There was no gross subtalar, talonavicular, or calcaneocuboid arthritis present.

The fresh frozen cadaver limbs were amputated through the knee joint. All specimens were thawed for 24 hours at room temperature. Each leg was prepared by removing the skin and subcutaneous tissues on the distal aspect of the foot and ankle, which helped in the exposure of the tibial nerve and vessels. The dissection was carefully performed to preserve supporting ligaments and joint capsules. The tibial nerve was identified and isolated just proximal to the fascia of the abductor hallucis muscle, and the transducer was attached to the lateral plantar nerve within the tarsal tunnel.

The proximal tibia was rigidly positioned in an aluminum pot with set screws. The leg specimen was then placed on the MTS base-plate, and loaded proximally through a hinged articulation connecting the servohydraulic actuator.

A transducer, MLP-10 Low-Range Load Cell (Transducer Techniques, California), was used to measure the tensile forces placed through the nerve. It was attached to an adjustable machined rig that was secured to the distal tibia by a plate with screws. The transducer was attached to a specially designed supporting arm of a rig through a universal joint, which allowed it to swivel, and orient itself in the same direction as the forces being placed through the nerve. The supporting arm was constructed so that the transducer could be adjusted in both a vertical and horizontal direction, which allowed it to be positioned so that there was no slack within the system prior to loading the foot. A 2-0 ethibond suture was placed through the lateral plantar nerve using a Bunnell type suture technique. The suture was secured to the center of the transducer, and aligned to follow the direction and position of the tibial nerve. The proximal and distal branches of the tibial nerve were undisturbed. The experimental set up is shown in Figure 2. The attachment of the lateral plantar nerve to the transducer with 2-0 ethibond suture is shown in Figure 3.

The method used to measure nerve tension in this study interfered with the normal excursion of the nerve because it fixed the tibial nerve proximally via a suture attached to the transducer. This simulated the *in vivo* situation (90) since the tibial nerve was relatively fixed proximally by its many branches to the posterior compartment muscles. Furthermore, because

the nerve was fixed distally by nerve branching in the foot, which remained undisturbed during the experiment, the transducer was optimally attached to the tibial nerve between the relatively fixed proximal and distal portion of the nerve to measure nerve tension.

The transducer recorded nerve tension in real time. With the leg loaded to 700 N (newtons), which simulated one body weight of 70 kg, the nerve was pre-tensioned to 1 N, and zeroed. This was considered the reference point from which nerve tension was measured. This reference point of 1 N was not meant to simulate the *in vivo* tibial nerve tension, which is unknown. The reference point of 1 N was chosen to remove slack from the nerve so that when the foot was manipulated into different positions or cyclically loaded with internal rotation of the leg, the nerve tension recorded did not include removal of the slack from the nerve trunk, and represented only the tension transferred to nerve by the maneuver. Also, since normal and abnormal tibial nerve tensions are unknown, the relative change of tibial nerve tension in the pes planus foot compared to the baseline was more important than the absolute value of the nerve tension. If we were interested in the absolute value of the nerve tension recorded, the *in vivo* tibial nerve tension would be extremely important since overestimation or underestimation of the *in vivo* nerve tension would result in underestimation or overestimation of the true value, respectively.

a. Baseline Measurement

i. Foot Position

The foot was loaded to 700 N (newtons), the nerve was pre-tensioned to 1 N, and then zeroed. The specimen was unloaded, and allowed to hang free, making the foot and ankle freely mobile. The foot was then manually manipulated into maximal dorsiflexion, eversion, and combined dorsiflexion-eversion. The same experimenter manipulated the foot with the goal of achieving the maximal position with the greatest possible nerve tension. Since the transducer recorded nerve tension in real time, nerve tension was recorded until a maximum plateau was reached, and no further increase in nerve tension was possible. The maximum nerve tension was measured and sustained for 3 seconds. Any further force applied to the foot did not result in an increase in nerve tension.

The manipulation of the foot into dorsiflexion or eversion represents a combination of movements at the ankle, subtalar, transverse tarsal, and tarsal-metatarsal joints (36,79,80). Dorsiflexion of the foot results from dorsiflexion through the ankle, transverse tarsal, and tarsal-metatarsal joints. Eversion of the foot results from valgus tilt of the ankle, hindfoot eversion, and forefoot abduction. For example, isolated fusion of the ankle only decreases dorsiflexion of the foot by 50.3%, and eversion by 27.8%, while the remainder of motion occurs through the unfused joints (36). To accurately measure isolated foot and ankle joint motion, sophisticated equipment is required to track the movement of the bones in three-dimensional space (2,21,36).

ii. Cyclical Load

Before testing in a loaded position, the transducer was pre-tensioned to 1 N and zeroed while loaded at 700 N again. The specimen was placed on the MTS base-plate, and loaded to simulate the early part of stance phase of gait. The specimen was loaded cyclically at a frequency of 1 Hz from 0 N to 700 N for 3-4 cycles before nerve tension was recorded to accommodate for stress relaxation. The nerve tension was then recorded under a cyclical load from 0 N to 700 N at a frequency of 1 Hz with simultaneous internal rotation of the tibia relative to the foot of 0°, 5°, 10°, 15°, and 20°.

The walking cycle consists of the stance phase (62%), and swing phase (38%). The events which produce calcaneal eversion during the early part of stance phase form the basis for this experimental model (79,80). While the foot is fixed to the ground, the joints of the foot, ankle, and leg are interconnected, forming a closed kinetic chain, with motion at one end of the chain altering motion at the other end. At heel strike, the foot passively collapses into hindfoot eversion and forefoot abduction due to the configuration of the joints and ligaments. Since the axis of the subtalar joint is obliquely oriented, it acts as a mitered hinge, transmitting the forefoot abduction and hindfoot eversion into internal rotation of the tibia (79,80). Because of the specific linkage of the leg to the foot through the subtalar joint, hindfoot eversion and forefoot abduction must occur simultaneously with internal rotation of the leg. This is mostly a passive process with only the anterior tibial compartment muscles contracting to prevent foot slap.

With the foot flat, external rotation of the tibia begins, which is an active process originating proximally. The swinging of the contralateral leg produces external rotation of the pelvis on the stance leg, which is transmitted into the foot through the leg and subtalar joint, producing hindfoot inversion and forefoot adduction. The metatarsal break, and the contraction of the intrinsic and extrinsic muscles of the foot also contribute to the hindfoot inversion and forefoot adduction, and through the subtalar joint, external rotation of the tibia. Thus, hindfoot inversion and forefoot adduction must occur simultaneously with external rotation of the tibia due to the linkage of these joints.

The cadaver limb was mounted and cyclically loaded from 0 N to 700 N to simulate the loading of one body weight. The tibia was simultaneously internally rotated relative to the foot to 0°, 5°, 10°, 15°, and 20° to simulate the normal transverse rotations of the lower extremity. Since the foot was in contact with the MTS base-plate and formed a closed kinetic chain, the internal rotation of the tibia relative to the foot produced hindfoot eversion and forefoot abduction through the subtalar joint. With increasing internal rotation of the tibia, the hindfoot eversion and forefoot abduction would also increase. Though the average amount of internal rotation produced during the early part of stance phase is 11° (67), more internal rotation is present with a more flexible pes planus foot. Since there is no muscle activity which restricts motion of these joints at initial ground contact, this is a valid biomechanical model for the early part of stance phase.

The first measurements of tibial nerve tension under different foot positions, and cyclical load were defined as the baseline measurements before intervention. Then, lateral plantar nerve tension was measured sequentially on the same specimen following the surgical creation of a pes planus deformity.

b. Pes Planus foot

Deland et al. (20) demonstrated that a pes planus deformity could be recreated by releasing the posterior tibial tendon, talonavicular joint, spring ligament, plantar fascia, calcaneocuboid joint, subtalar joint, and interosseous ligament. This created a pes planus foot which resembled the clinical situation radiographically. After the release of the structures described by Deland et al., the nerve tension was measured for foot position and load according to the baseline protocols described above.

c. Statistical Analysis

For foot position and cyclical load, nerve tension was defined as the difference of the maximum and minimum nerve tension recorded. Under foot position and cyclical load, two interventions were studied: baseline, and pes planus foot. After surgically creating a pes planus foot, the lateral plantar nerve tension of the pes planus foot were compared to the baseline measurement. Since each specimen served as its own control, this decreased the variability in the data by using a repeated measures design (116). Thus, even though a pes planus deformity in a mobile foot would have a greater nerve tension than a relatively stiffer foot, comparison of the pes planus nerve tension to the baseline nerve tension would account for this variability since the mobile foot would have a greater baseline nerve tension than the stiffer foot. All statistical analysis was performed using the SAS statistical software with a significance of $p < 0.05$.

The overall statistical analysis for foot position and cyclical load were performed differently, and are described separately below. However, the paired t-test (116) was used for comparison of the baseline and pes planus groups for foot position and cyclical load after determining a statistically significant overall effect. This was a valid test since the two sample means were continuous variables derived from a normally distributed population.

i. Foot Position

The lateral plantar nerve tensions for dorsiflexion, eversion, and combined dorsiflexion-eversion were analyzed separately. A t-test of significance (116) was used to demonstrate whether a significant increase in lateral plantar nerve tension existed, and a paired t-test (116) was used to determine whether the lateral plantar nerve tension was significantly greater in an unstable pes planus foot compared to a baseline foot while in dorsiflexion, eversion, and combined dorsiflexion-eversion.

ii. Cyclical Load

A t-test (116) was used to determine whether a significant increase in nerve tension existed. Nerve tension was affected by two factors under cyclical load: (1) internal rotation, and (2) intervention. A two factor repeated measures analysis of variance (116) was used to

determine whether the increased tibial nerve tension in the unstable pes planus foot compared to the stable baseline foot interacted with increasing internal rotation. A repeated measures analysis of variance comparing each increase in internal rotation to 0° was used to determine if the increased nerve tension with increasing internal rotation was significant. Within the baseline and pes planus groups, a single factor repeated measures analysis of variance (116) was performed to determine if the increased nerve tension with each increment of internal rotation compared to 0° was significant.

Results:

a. Foot Position

The data from the nine specimens are summarized in Tables 1, and 2. Table 1 displays the data for the effect of foot position on the peak nerve tension measured in newtons. The cadaver foot was placed in a position of maximal eversion, dorsiflexion, or combined eversion-dorsiflexion, and the peak nerve tension was measured.

In a stable baseline foot, the mean peak nerve tensions and standard deviations with dorsiflexion was 8.44 +/- 4.61 N, with eversion was 7.79 +/- 3.35 N, and with combined dorsiflexion-eversion was 13.23 +/- 4.59 N. In an unstable pes planus foot, the mean peak nerve tensions and standard deviations with dorsiflexion was 10.61 +/- 5.89 N, with eversion was 10.90 +/- 3.98 N, and with combined dorsiflexion-eversion was 16.42 +/- 5.17 N. These results are presented in Figure 4.

Lateral plantar nerve tension was significantly increased in baseline and pes planus feet with eversion ($p=0.0001$), dorsiflexion ($p=0.0006$), and combined dorsiflexion-eversion ($p=0.0001$). The increased lateral plantar nerve tension in the unstable pes planus foot compared to the stable baseline foot was statistically significant for eversion ($p<0.002$), dorsiflexion ($p<0.01$), and combined dorsiflexion-eversion ($p=0.0004$).

b. Cyclical Load

The tracing of the lateral plantar nerve tension measurements under a cyclical load appeared like a sinusoidal wave. The peak of nerve tension corresponded to the maximum load, which resulted from the maximum hindfoot eversion and forefoot abduction during

loading, and the minimum nerve tension corresponded to the minimum load. With increasing internal rotation of the leg, the peak nerve tension increased due to increasing hindfoot eversion and forefoot abduction transferred to the foot through the subtalar joint. During the measurement of nerve tension under these conditions, the peaks and troughs of the sinusoidal wave were consistent.

Before an analysis of variance was performed, the data were scrutinized for distributional properties. With increasing internal rotation, the raw data demonstrated more skewness and kurtosis. A transformation of the raw data with the natural logarithm improved the normality of the data and the analysis of variance was performed on the transformed data. The mean tibial nerve tensions reported in Table 2 represents the anti-logarithm of the mean transformed nerve tension.

The mean nerve tension in a stable baseline foot which was axially loaded and internally rotated 0° was 0.28 +/- 1.60 N, 5° was 0.31+/- 1.77 N, 10° was 0.42 +/- 1.88 N, 15° was 0.50 +/- 2.22 N, and 20° was 0.64 +/- 2.23 N. The mean nerve tension in an unstable pes planus foot which was axially loaded and internally rotated 0° was 0.50 +/- 1.46 N, 5° was 0.67 +/- 1.46 N, 10° was 0.84 +/- 1.71 N, 15° was 1.05 +/- 2.18N, and 20° was 1.12 +/- 2.29 N. These results are shown in Figure 5.

In the stable baseline foot, lateral plantar nerve tension was significantly increased during axial loading and internal rotation of 0° ($p=0.0001$), 5° ($p=0.001$), 10° ($p<0.003$), 15° ($p<0.007$), and 20° ($p<0.005$). In the unstable pes planus foot, lateral plantar nerve tension was significantly increased during axial loading and internal rotation of 0° ($p=0.0001$), 5° ($p=0.0001$), 10° ($p<0.001$), 15° ($p<0.03$), and 20° ($p<0.03$).

Throughout the range of increasing internal rotation, lateral plantar nerve tension was significantly increased in an unstable pes planus foot compared to a stable baseline foot ($p=0.0002$). In the unstable pes planus and stable baseline feet, the lateral plantar nerve tension was significantly increased with axial loading and increasing internal rotation ($p=0.0001$). Though decreased stability of the foot and increasing internal rotation increased lateral plantar nerve tension independently, these two factors acting together did not significantly compound the increase in lateral plantar nerve tension ($p=0.28$). The increased lateral plantar nerve tension for the unstable pes planus and stable baseline feet with

increasing internal rotation was significantly greater when 10° ($p<0.01$), 15° ($p<0.02$), and 20° ($p<0.006$) was compared to 0° of internal rotation.

In the stable baseline foot, the lateral plantar nerve tension was significantly increased with increasing internal rotation compared to the nerve tension at 0° of rotation ($p=0.0001$). The increased lateral plantar nerve tension in the stable baseline foot was significant with increasing internal rotation when 10° ($p<0.02$), 15° ($p<0.02$), and 20° ($p<0.003$) was compared to 0° internal rotation. In the unstable pes planus foot, the lateral plantar nerve tension was significantly increased with increasing internal rotation compared to the nerve tension at 0° of internal rotation ($p=0.0001$). The increased lateral plantar nerve tension in the unstable pes planus foot was significantly greater when 5° ($p<0.02$), 10° ($p<0.02$), 15° ($p<0.02$), and 20° ($p<0.02$) was compared to 0° internal rotation.

Part II: The effects of tarsal tunnel release and stabilization procedures on tibial nerve tension in a surgically created pes planus foot

Introduction:

In Part I of the thesis, a new method for measuring nerve tension was developed. We showed that lateral plantar nerve tension was increased in a pes planus foot compared to a normal foot for foot position and cyclical load, which confirms the beliefs of many authors (26,32,51,55,58,63,81). Tensile forces placed through the lateral plantar nerve were measured: (1) when the foot was placed into maximal dorsiflexion, eversion, combined dorsiflexion-eversion; and (2) under cyclical load with increasing internal rotation of the leg at 5° increments from 0° to 20° to simulate the stance phase of gait. Then, a pes planus deformity was surgically created, and the nerve tension measurements were repeated. Analysis of the data showed that lateral plantar nerve tension was significantly increased in a pes planus foot compared to a normal foot during dorsiflexion, eversion, combined dorsiflexion-eversion, and under cyclical load with increasing internal rotation.

After failure of non-operative therapy, surgical therapy with a tarsal tunnel release is recommended. However, the effect of tarsal tunnel release on tibial nerve tension is unknown. Furthermore, if tibial nerve tension is dependent on skeletal stability, then stabilization of the pes planus foot deformity should decrease tibial nerve tension. The goal of Part II of this investigation was to study the effects of tarsal tunnel release and stabilization procedures, which correct the pes planus deformity, on tibial nerve tension in a surgically created pes planus foot.

The surgical procedures used in this investigation to correct the pes planus deformity were the distraction calcaneocuboid arthrodesis, and the triple arthrodesis. The distraction calcaneocuboid arthrodesis is based on the concept that the lateral column is relatively shorter than the medial column in a pes planus foot, and lengthening of the lateral column is required to correct the deformity (30). Evans (30) lengthened the lateral column by lengthening the calcaneus through a calcaneal osteotomy 1.5 cm proximal to the calcaneocuboid joint. Long term follow-up in young patients showed overall good results, but arthritic changes were noted in the calcaneocuboid joint (107). Thus, to lengthen the lateral column and eliminate late degeneration of the calcaneocuboid joint, Deland et al. (21)

proposed lateral column lengthening by distraction calcaneocuboid arthrodesis. The triple arthrodesis involves fusion of the subtalar, talonavicular, and calcaneocuboid joints in a neutral position. This is useful for correcting very severe deformities, and especially in cases where significant arthritis is present within the joints.

Part I of this study (18) was the first to confirm that lateral plantar nerve tension was greater in a pes planus foot than a normal foot. Furthermore, it provides a feasible and reproducible method for measuring tibial nerve tension, and surgically creating a pes planus deformity. Though Part I of this investigation laid down the ground work for Part II, modifications were made to the "Materials and Methods" for Part II to study the effects of tarsal tunnel release, and corrective procedures on tibial nerve tension in a pes planus foot. Firstly, in Part II, the flexor retinaculum was carefully preserved, and the transducer was attached to the tibial nerve proximal to the flexor retinaculum. Secondly, since internal rotation of the leg from 0° to 20° at 5° increments showed increasing nerve tension in Part I, tibial nerve tension was only measured at 0°, 15°, and 25° of internal rotation in Part II, because smaller amounts of internal rotation would not provide any additional information. Finally, in Part II, the tibial nerve was pre-tensioned to 1 N while the leg was loaded to 100 N, unlike in Part I, where the nerve was pre-tensioned to 1 N while the leg was loaded to 700 N. During pre-tensioning of the nerve, the higher loads would produce greater foot deformity, which would increase nerve tension. Since our reference point for pre-tensioning of the nerve was this initial load, the higher nerve tension during the higher load would be our baseline of 0 N, and the greater nerve tension would not be accounted for during testing. Thus, a lower load for pre-tensioning of the tibial nerve was chosen for Part II to ensure that our reference point did not include the load during our testing conditions. The goal of this investigation was to better understand tibial nerve tension in a pes planus foot so that treatments which specifically address nerve tension are used appropriately.

Tarsal tunnel syndrome results from tibial nerve neuropathy caused by nerve compression and/or tension. Treatment options that successfully relieve tibial nerve compression include, anti-inflammatory agents for inflammatory conditions causing tenosynovitis; orthotics use for varus heels; repair of fractures to restore spatial orientation; and tarsal tunnel release and excision of space-occupying lesions. However, the only treatment directed towards nerve tension is orthotics for valgus heels, and for patients with

symptoms reproduced by dorsiflexion. The effects of tarsal tunnel release and stabilization procedures on tibial nerve tension are unknown. Part I of this study establishes a feasible, and reproducible model for studying the effects of tarsal tunnel release and stabilization procedures on tibial nerve tension in a surgically created pes planus foot.

Material and Methods:

An *in vitro* study was performed on 11 fresh frozen cadaver limbs. The cadaver limbs came from 4 males and 3 females, with an mean age of 64.0 years. There were 5 right, and 6 left specimens, with 3 bilateral specimens. All the cadavers were received in the anatomy department within 24 hours of death, and preserved by freezing without embalming. There was no major structural abnormality present in the cadaver limbs, except one specimen had a fifth toe amputation. There was no gross subtalar, talonavicular, or calcaneocuboid arthritis present.

The fresh frozen cadaver limbs were amputated through the knee joint. All specimens were thawed for 24 hours at room temperature. Each leg was prepared by removing the skin and subcutaneous tissues on the distal aspect of the foot and ankle, which helped in the exposure of the tibial nerve and vessels proximal to the flexor retinaculum. The tibial nerve was identified and isolated just proximal to the flexor retinaculum. The dissection was carefully performed to preserve the supporting ligaments, joint capsules, and the flexor retinaculum.

The proximal tibia was rigidly positioned in an aluminum pot with set screws. The leg specimen was then placed on the MTS base-plate, and loaded proximally through a hinged articulation connecting the servohydraulic actuator.

A transducer, MLP-10 Low-Range Load Cell (Transducer Techniques, California), was used to measure the tensile forces placed through the nerve. The transducer recorded nerve tension in real time. It was attached to an adjustable machined rig that was secured to the distal tibia by a plate with screws. The transducer was attached to a specially designed supporting arm of a rig through a universal joint, which allowed it to swivel, and orient itself in the same direction as the forces being placed through the nerve. The supporting arm was constructed so that the transducer could be adjusted in both a vertical and horizontal direction, which allowed it to be positioned so that there was no slack within the system prior to loading the foot. A 2-0

ethibond suture was placed through the tibial nerve proximal to the flexor retinaculum using a Bunnell type suture technique. The suture was secured to the center of the transducer, and aligned to follow the direction and position of the tibial nerve. The proximal and distal branches of the tibial nerve were undisturbed. The experimental set up is shown in Figure 2. The attachment of the lateral plantar nerve to the transducer with 2-0 ethibond suture is shown in Figure 3.

a. Baseline Measurement

i. Foot Position

The foot was loaded to 100 N (newtons), and the nerve was pre-tensioned to 1 N, then zeroed. This was considered the reference point. First, the specimen was unloaded, and allowed to hang free, making the foot and ankle freely mobile. The foot was then manually manipulated into maximal dorsiflexion, eversion, and combined dorsiflexion-eversion. This was consistent between different specimens because the same experimenter manipulated the foot with the goal of achieving the maximal position with the greatest possible nerve tension. Since the transducer recorded nerve tension in real time, nerve tension was recorded until a maximum plateau was reached, and no further increase in nerve tension was possible. The maximum nerve tension was measured and sustained for 3 seconds. Any further force applied to the foot did not result in an increase in nerve tension.

ii. Cyclical Load

Before testing in a loaded position, the transducer was pre-tensioned to 1 N and zeroed while loaded at 100 N again. The specimen was placed on the MTS base-plate, and loaded to simulate the early part of stance phase of gait. The specimen was loaded cyclically at a frequency of 1 Hz from 0 N to 700 N for 3-4 cycles before nerve tension was recorded to accommodate for stress relaxation. The nerve tension was then recorded under a cyclical load from 0 N to 700 N at a frequency of 1 Hz with simultaneous internal rotation of the tibia relative to the foot of 0°, 15° and 25°.

The events which produce calcaneal eversion during the early part of stance phase form the basis for this experimental model. The cadaver limb was mounted and cyclically loaded

from 0 N to 700 N to simulate the loading of one body weight. The tibia was simultaneously internally rotated relative to the foot to 0°, 15°, and 25° to simulate the normal transverse rotations of the lower extremity. Since the foot was in contact with the MTS base-plate and formed a closed kinetic chain, the internal rotation of the tibia relative to the foot produced hindfoot eversion and forefoot abduction through the subtalar joint. With increasing internal rotation of the tibia, the hindfoot eversion and forefoot abduction would also increase. Though the average amount of internal rotation produced during the early part of stance phase is 11° (67) more internal rotation is present with a more flexible pes planus foot. Since there is no muscle activity which restricts motion of these joints at initial ground contact, this is a valid biomechanical model for the early part of stance phase.

The first measurement of tibial nerve tension under different foot positions, and cyclical load were defined as the baseline measurements before intervention. Then, tibial nerve tension was measured sequentially on the same specimen following four interventions: surgically created pes planus, tarsal tunnel release, distraction calcaneocuboid arthrodesis, and triple arthrodesis. Since each specimen served as its own control, this decreased variability in the data by using a repeated measures design.

b. Pes Planus foot

Deland et al. (20) demonstrated that a pes planus deformity could be recreated by releasing the posterior tibial tendon, talonavicular joint, spring ligament, plantar fascia, calcaneocuboid joint, subtalar joint, and interosseous ligament. This created a pes planus foot which resembled the clinical situation radiographically. After the release of the structures described by Deland et al., the nerve tension was measured for foot position and load according to the baseline protocols described above.

c. Tarsal Tunnel Release

The proximal and distal tarsal tunnels were released to simulate surgical treatment. The proximal tarsal tunnel was decompressed by incising the flexor retinaculum and exposing the tibial nerve within the proximal tarsal tunnel. The distal tarsal tunnel was released by identifying and following the medial and lateral plantar nerves, and releasing part of the origin of the abductor hallucis brevis. The experimental set up is shown is Figure 6.

Measurement of tibial nerve tension for foot position and load were then repeated according to the baseline protocols.

d. Distraction Calcaneocuboid Arthrodesis

The calcaneocuboid joint was exposed through an incision directly over the joint, and the cartilage removed using an oscillating saw. The calcaneocuboid joint was distracted using a 7.5-10 mm block of lexan to lengthen the lateral column of the foot, which corrected the hindfoot valgus and forefoot abduction, while maintaining subtalar motion. The calcaneocuboid joint was then stabilized using a staple. The experimental set up is shown in Figure 7. The tibial nerve tension was remeasured for foot position and load according to the baseline protocols.

e. Triple Arthrodesis

The talonavicular, calcaneocuboid, and subtalar joints were fused in a neutral position to correct the pes planus deformity, and eliminate any motion within these joints. The talonavicular joint was fused by reducing the navicular in a neutral position relative to the talus, and drilling two heavy Steinman pins anteriorly from the navicular into the talus. The subtalar joint was fused by holding the calcaneus in a neutral position and drilling a heavy Steinman pin from the posterior-inferior non-weightbearing surface of the calcaneus to the talus in a superior-medial direction.

The joint fusions were verified directly by using the scalpel to probe the joints to verify that the pins crossed the joint, and indirectly by manipulating the joint to determine whether subtalar or talonavicular motion existed. In all cases, the talonavicular and subtalar joints were solidly fused using this technique. The experimental set up is shown in Figure 8. Measurement of tibial nerve tension for foot position and load were then repeated according to the baseline protocols.

f. Statistical Analysis

For foot position and cyclical load, nerve tension was defined as the difference of the maximum and minimum nerve tension recorded. Under foot position and cyclical load, five interventions were studied: baseline, pes planus foot, tarsal tunnel release, distraction

calcaneocuboid arthrodesis, and triple arthrodesis. All statistical analysis was performed using the SAS statistical software program with a significance of $p<0.05$.

The overall statistical analysis for foot position and cyclical load were performed differently, and are described separately below. However, the paired t-test (116) was used for comparison of two interventions for foot position and cyclical load after determining a statistically significant overall effect. This was a valid test since the two sample means were continuous variables derived from a normally distributed population.

i. Foot Position

The effects of the five interventions on dorsiflexion, eversion, and combined dorsiflexion-eversion were analyzed separately. For each position, an one factor repeated measures analysis of variance (116) was performed on the data to determine whether nerve tension was significantly different within the group.

Since a difference was detected, multiple comparisons using paired t-tests (116) were performed based on our hypothesis to determine where the differences existed: (1) pes planus and baseline; (2) tarsal tunnel release and pes planus; (3) distraction calcaneocuboid arthrodesis and tarsal tunnel release; and (4) triple arthrodesis and tarsal tunnel release. However, since the surgery was performed sequentially on each specimen, the tibial nerve tension after a tarsal tunnel release could not be compared to the baseline foot because a pes planus deformity was already created, and such a comparison would ignore the contribution of the pes planus deformity to the nerve tension before the tarsal tunnel release. Furthermore, the tibial nerve tension after each stabilization procedure could not be compared to the pes planus foot since each foot had a tarsal tunnel release prior to stabilization, and if the tarsal tunnel release affected tibial nerve tension, then the effect of the tarsal tunnel release would be inappropriately ignored in the comparison.

ii. Cyclical Load

Nerve tension was affected by two factors under cyclical load: (1) internal rotation, and (2) intervention. Nerve tension was recorded with 0°, 15°, and 25° of internal rotation of the leg. The interventions studied were baseline, pes planus foot, tarsal tunnel release, distraction calcaneocuboid arthrodesis, and triple arthrodesis.

A two factor repeated measures analysis of variance (116) was performed to determine whether: (1) internal rotation affected nerve tension alone; (2) intervention affected nerve tension alone; and (3) internal rotation and intervention combined to compound the effect on nerve tension. Since internal rotation and intervention did not significantly interact, internal rotation and intervention were analyzed separately to determine their individual effect on nerve tension. For intervention under cyclical load, multiple comparisons using paired t-tests (116) were performed based on our hypothesis to determine where the differences existed: (1) pes planus and baseline; (2) tarsal tunnel release and pes planus; (3) distraction calcaneocuboid arthrodesis and tarsal tunnel release; and (4) triple arthrodesis and tarsal tunnel release.

Results:

a. Foot position

The mean tibial nerve tension for all the data on foot position was 24.80 N. The effect of intervention on tibial nerve tension was analyzed separately for each position using a one factor repeated measures analysis of variance. The interventions had a significant effect on tibial nerve tension for dorsiflexion ($p=0.0001$), eversion ($p=0.0002$), and combined dorsiflexion-eversion ($p=0.0001$). For each position, multiple comparisons were performed using a paired t-test (116) to determine which interventions significantly affected tibial nerve tension.

During dorsiflexion, the mean nerve tension and standard deviation measured was 20.64 +/- 7.55 N for baseline, 31.90 +/- 12.59 N for pes planus, 31.47 +/- 11.29 N for tarsal tunnel release, 25.55 +/- 8.84 N for calcaneocuboid arthrodesis, and 23.62 +/- 7.88 N for triple arthrodesis. These data are shown in Table 3a and Figure 9.

During eversion, the mean nerve tension and standard deviation measured was 10.54 +/- 5.50 N for baseline, 16.35 +/- 9.30 N for pes planus, 19.92 +/- 10.42 N for tarsal tunnel release, 19.83 +/- 8.65 N for calcaneocuboid arthrodesis, and 14.52 +/- 9.07 N for triple arthrodesis. These data are shown in Table 3b and Figure 10.

During combined dorsiflexion-eversion, the mean nerve tension and standard deviation measured was 25.69 +/- 10.29 N for baseline, 32.41 +/- 12.93 N for pes planus,

35.69 +/- 13.04 N for tarsal tunnel release, 34.68 +/- 12.03 N for calcaneocuboid arthrodesis, and 29.21 +/- 10.11 N for triple arthrodesis. These data are shown in Table 3c and Figure 11.

When the pes planus foot was compared to baseline, tibial nerve tension was significantly increased during dorsiflexion ($p=0.0001$), eversion ($p<0.03$), and combined dorsiflexion-eversion ($p=0.0004$). Tarsal tunnel release significantly increased tibial nerve tension during eversion ($p<0.04$), and combined dorsiflexion-eversion ($p<0.03$) compared to the pes planus foot, while it had no significant effect on tibial nerve tension during dorsiflexion ($p=0.72$). These data are summarized in Figure 12.

When distraction calcaneocuboid arthrodesis was compared to the tarsal tunnel released pes planus foot, tibial nerve tension was significantly decreased during dorsiflexion ($p<0.004$). It had no significant effect on tibial nerve tension during eversion ($p=0.93$), and combined dorsiflexion-eversion ($p=0.45$).

When triple arthrodesis was compared to the tarsal tunnel released pes planus foot, tibial nerve tension was significantly decreased during dorsiflexion ($p=0.0005$), and combined dorsiflexion-eversion ($p<0.002$). It decreased tibial nerve tension during eversion, which approached significance ($p=0.065$).

b. Cyclical Load

The tracing of the tibial nerve tension measurements under a cyclical load appeared like a sinusoidal wave. The peak of nerve tension corresponded to the maximum load, which resulted from the maximum hindfoot eversion and forefoot abduction during loading, and the minimum nerve tension corresponded to the minimum load. With increasing internal rotation of the leg, the peak nerve tension increased due to increasing hindfoot eversion and forefoot abduction transferred to the foot through the subtalar joint. During the measurement of nerve tension under these conditions, the peaks and troughs of the sinusoidal wave were consistent.

The mean tibial nerve tension for all the data on cyclical load with internal rotation was 1.93 N. Before an analysis of variance was performed, the data were scrutinized for distributional properties. With increasing internal rotation, the raw data demonstrated more skewness and kurtosis. A transformation of the raw data with the natural logarithm improved the normality of the data and the analysis of variance was performed on the transformed data.

The mean tibial nerve tension reported represents the anti-logarithm of the mean transformed nerve tension.

A two factor repeated measures analysis of variance (116) on the transformed data demonstrated that internal rotation ($p=0.0001$) and intervention ($p=0.0001$) significantly affected tibial nerve tension, but intervention and internal rotation combined did not significantly compound the change in nerve tension ($p=0.67$). These data are shown in Tables 4a-4b, and Figures 13 and 14.

The mean tibial nerve tension and standard deviation of a specimen cyclically loaded and internally rotated 0° was 0.36 +/- 1.83 N, 15° was 1.24 +/- 2.64 N, and 25° was 2.41 +/- 2.57 N. The tibial nerve tension at 15° ($p=0.0005$) and 25° ($p=0.0001$) of internal rotation were significantly higher compared to 0° of internal rotation. These data are shown in Table 4a and Figure 13.

The mean tibial nerve tension and standard deviation was 0.70 +/- 2.96 N for baseline, 1.15 +/- 3.14 N for pes planus, 1.44 +/- 3.18 N for tarsal tunnel release, 0.95 +/- 3.46 N for calcaneocuboid arthrodesis, and 1.05 +/- 3.02 N for triple arthrodesis. These data are shown in Table 4b and Figure 14.

During cyclical load and increasing internal rotation, tibial nerve tension was significantly increased when the pes planus foot was compared to baseline during 0°, 15°, and 25° of internal rotation ($p=0.001$, see Figure 15). Tarsal tunnel release significantly increased tibial nerve tension compared to the pes planus foot during 0°, 15°, and 25° of internal rotation ($p<0.03$, see Figure 16). Distraction calcaneocuboid arthrodesis ($p<0.03$, see Figure 17) and triple arthrodesis ($p<0.03$, see Figure 18) significantly decreased tibial nerve tension compared to the tarsal tunnel released pes planus foot during 0°, 15°, and 25° of internal rotation.

Further analysis was performed for baseline and pes planus feet. Baseline measurements of tibial nerve tension and standard deviation for cyclical load and internal rotation of 0° was 0.28 +/- 1.51 N, 15° was 0.84 +/- 2.71 N, and 25° was 1.49 +/- 2.70 N. In the pes planus foot, tibial nerve tension and standard deviation with cyclical load and internal rotation of 0° was 0.44 +/- 1.69 N, 15° was 1.19 +/- 2.91 N, and 25° was 2.87 +/- 2.54 N. Tibial nerve tension was significantly increased in the pes planus foot compared to baseline during 0°, 15°, and 25° of internal rotation ($p=0.001$). Tibial nerve tension was significantly

increased with increasing internal rotation ($p=0.0001$). Though the trend in the data suggests that increasing internal rotation and a pes planus deformity compounds the increase in tibial nerve tension, the interaction between the intervention and internal rotation was not significant ($p=0.19$). To demonstrate a significant interaction, sample size would have to be increased. Nevertheless, the pes planus foot compared to the baseline, and internal rotation from 15° to 25° significantly interacted to compound the increase in tibial nerve tension ($p<0.02$).

Discussion:

Tarsal tunnel syndrome has been thought of as an entrapment neuropathy (26,55,58,63,81,113,130). However, simply decompressing the tarsal tunnel has not produced results as successful as decompressing the carpal tunnel (81,106,135). Thus, other unrecognized factors may be contributing to the tarsal tunnel syndrome. In the initial descriptions by Keck (55) and Lam (63), both authors postulated that "poor foot structure" could be a cause of this syndrome. Many authors (26,51,58,81) believe that a severely pronated and valgus foot deformity stretches the tibial nerve, producing a tarsal tunnel syndrome. In a series of 11 patients with pes planus deformity and tarsal tunnel syndrome treated surgically, 6 patients had no evidence of a space-occupying lesion identified at surgery (32). It was stated that "pes planus, especially when mobile and aggravated by associated hindfoot valgus, puts the posterior tibial nerve and/or its medial and lateral plantar branches on stretch with each weight bearing step." This makes the nerve vulnerable to neuropraxia with any additional injuries (32). Rask (112) and Jackson (51) believed that while running, eversion of the foot brings the medial plantar nerve into stretch against the tunnel's superior surface. Also, positions requiring extreme dorsiflexion of the ankle (15) are associated with tarsal tunnel syndrome (eg. repeated crouching, and squatting).

Currently, there are no studies in the literature that quantify lateral plantar nerve or tibial nerve tension. This current study quantifies lateral plantar nerve tension in a baseline, and surgically created pes planus foot; and tibial nerve tension in a baseline, pes planus foot, tarsal tunnel released foot, distraction calcaneocuboid arthrodesis, and triple arthrodesis. Because there are currently no methods for measuring nerve tension *in vivo*, the absolute

value of the nerve tension measurement was not as important as the magnitude of the change of the measurements with each intervention. The method used to measure nerve tension in this study interfered with the normal excursion of the nerve because it fixed the tibial nerve proximally via a suture attached to the transducer. This simulated the *in vivo* situation (90) since the tibial nerve was relatively fixed proximally by its many branches to the posterior compartment muscles. Furthermore, because the nerve was fixed distally by nerve branching in the foot, which remained undisturbed during the experiment, the transducer was optimally attached to the tibial nerve between the relatively fixed proximal and distal portion of the nerve to measure nerve tension.

Since the tibial nerve and its branches cross the ankle and subtalar joints, they must accommodate for foot and ankle motion (90) by lengthening during ankle dorsiflexion and hindfoot eversion, and shortening during ankle plantarflexion and hindfoot inversion. As they are stretched, slack is removed from the nerve trunks, the paraneurium above the epineurium allows the nerve to slide, and undulations in the nerve fiber straighten (5,90). With increasing tension and stretch, these mechanisms fail and nerve conduction is impaired (62,75,100,138) either acutely, such as in severe trauma due to a fracture-dislocation of the foot or ankle, or a severe ankle sprain (26,64,68,113,129); or chronically, such as in pes planus feet (18,22,32,51,55,58,63,69,78,81), recurrent ankle sprains, or walking or running with a hyperpronated foot (51,112), which stretches the nerve with each weight bearing step.

Tension stretches a nerve, and the acute pathological and physiological consequences have been extensively studied in animals, while the effects of chronic, recurrent microtrauma are less clearly defined. With continued elongation of the nerve, the fascicles are stretched, and ischemia results from two factors. Firstly, the normally coiled and tortuous vessels are straightened and with further stretching, extrinsic and intrinsic blood supply are strangulated (75). Secondly, tension reduces the cross sectional area within the epineurium, which increases the intrafascicular pressure, and compresses the blood vessels (75). Elongation of rabbit tibial nerves by 8% produces a reduction of flow in venules, while elongation by 15% produces complete arterial vascular occlusion, but perineural integrity remains intact (75,100). Since peripheral nerves require an adequate and continuous supply of oxygen to function (73,75,100), ischemia impairs nerve conduction, and compound nerve action potential studies show reversible reduction in action potentials with 6% elongation, and

irreversible reduction with 12% elongation (138). Complete ischemia produces rapid deterioration in nerve function, which is reversible when blood flow is restored, but if the stretch is sufficiently prolonged, ischemia can cause irreversible damage (72,119).

With increasing elongation acutely, the components of the peripheral nerve fail sequentially (5,131). First, the nerve fibers within the fascicles (second degree injury) are disrupted either by a severe stretch at the time of injury, or by delayed axonal disconnection resulting from axolemma injury by a lesser force (85,87,88,109). Then, mechanical failure occurs at the endoneurial tubes within the fascicles (third degree injury), followed by the perineurium (fourth degree injury). The perineurium is the strongest component of the nerve (5,24,62,117,132,133), and fails at 27% beyond *in situ* strain (117). Failure of the perineurium represents a major injury to the nerve, and disrupts its ability to act as a diffusion barrier and maintain the highly specialized intrafascicular environment (73,117,118,119). A fifth degree injury occurs when the epineurium is disrupted and complete disruption of the nerve trunk results. In chronic injuries, greater amounts of stretch applied slowly and gradually are better tolerated without interfering with conduction (27,91,92).

The foundation of the tarsal tunnel consists of mobile joints. The amount of tibial and lateral plantar nerve tension depends on the amount of foot and ankle motion. Factors that destabilized the bony foundation, such as surgically creating a pes planus deformity and tarsal tunnel release, increased foot and ankle motion, which produced a greater deformity and increased nerve tension. On the other hand, procedures that corrected deformity and stabilized the bony foundation, such as distraction calcaneocuboid and triple arthrodesis, stabilized an unstable foundation, and decreased nerve tension.

With increasing foot and ankle motion, tibial and lateral plantar nerve excursion must increase to accommodate this motion. For all the data in both Parts I and II of this thesis, manipulation of the foot into maximal dorsiflexion, eversion, and combined dorsiflexion-eversion produced the greatest tibial nerve tension measured compared to cyclical load with increasing internal rotation of the leg, which simulated the early part of the stance phase of gait. This was due to the greater passive range of motion, which is 10° of hindfoot eversion and 20° of ankle dorsiflexion in normal patients (8), compared to the range of motion during walking, which is only 3° of hindfoot eversion (141) and 10° of ankle dorsiflexion (94,95).

At the extremes of motion, the greatest possible nerve tension was generated, and the maximum nerve excursion was required to accommodate this motion. Thus, tibial and lateral plantar nerve tension increased with increasing foot and ankle motion. This explains the reproduction of clinical symptoms due to increased tibial nerve tension with forcible dorsiflexion of the ankle (13,14) and eversion of the heel (15,22,26,40,55,101,106).

During axial loading and increasing internal rotation of the leg in Part I and Part II of the thesis, an unstable pes planus foot had a significantly higher nerve tension than a stable baseline foot, and increasing internal rotation of the leg significantly increased nerve tension independently. In Part I of the thesis, the increased lateral plantar nerve tension with increasing internal rotation of the leg was significantly higher when 10°, 15°, and 20° of internal rotation was compared to 0° in unstable pes planus or stable baseline feet. Though Figure 5 demonstrates a greater increase in lateral plantar nerve tension as internal rotation exceeds 10° in the unstable pes planus foot, skeletal stability and internal rotation acting together did not significantly compound the increase in nerve tension, however, increasing sample size may demonstrate a significant finding. In Part II of the thesis, the increased tibial nerve tension was significantly higher when 15°, and 25° of internal rotation was compared to 0° of internal rotation for all the data combined. Furthermore, Figure 15 shows a significantly greater increase in tibial nerve tension with increasing internal rotation from 15° to 25° in the unstable pes planus foot compared to baseline. With increasing internal rotation of the leg, calcaneal eversion, produced through the subtalar joint, increased and produced greater valgus of the hindfoot, which stretched the tibial and lateral plantar nerves, and increased nerve tension.

Furthermore, in both Parts I and II, nerve tension was significantly increased in an unstable pes planus foot compared to a stable baseline foot for all three positions, and cyclical loading with internal rotation of the leg. Surgically creating a pes planus deformity resulted in destabilization of the foot, and a generalized increase in motion, which increased valgus of the hindfoot and abduction of the forefoot. These positions combined to stretch the tibial and lateral plantar nerves, and increase nerve tension under all the testing conditions. This supports the observation that the functional range of motion of the subtalar joint is 6° in normal feet, and 12-16° in pes planus feet (17,141). Thus, in a pes planus foot, increased tibial nerve tension can be a cause of tarsal tunnel syndrome, which supports Francis et al.

(32), who reported cases of tarsal tunnel syndrome with pes planus feet and no evidence of a space-occupying lesion at surgery.

Part II of this thesis is the first study to show that tarsal tunnel release significantly increased tibial nerve tension compared to the pes planus foot during eversion, combined dorsiflexion-eversion, and calcaneal eversion produced by cyclical load with internal rotation of the leg. The flexor retinaculum is a medially based structure that originates from the medial malleolus and inserts on the calcaneal tuberosity, and can contribute to the stability of the medial side of the foot and ankle. Releasing the flexor retinaculum destabilized the medial side of the foot and ankle. On the other hand, surgically creating a pes planus foot required the release of many ligaments and joints, which increased the overall foot and ankle motion. This resulted in greater valgus hindfoot and forefoot abduction in all foot positions, which increased nerve tension, and accounted for the increased nerve tension in all foot positions. Since tarsal tunnel release only destabilized the medial side of the foot and ankle, the valgus of the hindfoot and abduction of the forefoot increased nerve tension only during calcaneal eversion.

Tarsal tunnel release in pes planus feet may further increase tibial nerve tension, which can exacerbate the underlying cause, and may produce poor clinical results. Since many authors do not comment on foot deformity in their patients (54,78,106,135), failure to recognize the presence of a pes planus deformity before tarsal tunnel release may partly explain the significant number of idiopathic cases, and following tarsal tunnel release in unrecognized pes planus feet, further increase in tibial nerve tension may explain the poor results in idiopathic cases reported in the literature (106,135).

Since tibial nerve tension depends on foot and ankle motion and deformity, stable correction of the deformity would decrease tibial nerve tension. In Part II of this thesis, the distraction calcaneocuboid arthrodesis significantly reduced tibial nerve tension during dorsiflexion and cyclical load, while the triple arthrodesis significantly reduced tibial nerve tension during dorsiflexion, combined dorsiflexion-eversion, and cyclical load, and eversion approached significance. Since tibial nerve tension increases with increasing foot and ankle motion, the effectiveness of corrective surgical procedures to decrease nerve tension depends on the stability of the arthrodesis. Following distraction calcaneocuboid arthrodesis, 48-67% of talonavicular and 70-92% of subtalar motion remained (2,21). On the other hand, a triple

arthrodesis decreased dorsiflexion of the ankle by 12.5%, hindfoot eversion by 60.5%, and supination and pronation of the foot by 50% (36). The triple arthrodesis was more effective at decreasing tibial nerve tension because it maintained correction of the deformity better than the distraction calcaneocuboid arthrodesis by fusing the unstable bony foundation of the tarsal tunnel in a corrected, neutral position. Thus, tibial nerve tension dependent on the amount of foot and ankle motion remaining after surgical stabilization.

These conclusions are supported by Astion et al. (2) who reported that 73% of posterior tibial tendon excursion remained after calcaneocuboid arthrodesis, while only 25% remained after triple arthrodesis. They concluded that "the amount of excursion remaining after a particular simulated arthrodesis was associated with the amount of motion remaining in the triple joint complex." Since the posterior tibial tendon is a structure within the tarsal tunnel, the forces transmitted to the posterior tibial tendon would be similarly transferred to the tibial nerve.

Limitations of this Study:

To definitively answer the question of whether increased tibial nerve tension causes tarsal tunnel syndrome in patients with pes planus feet, an *in vivo* study would have to be performed, but clearly this is impossible. An *in vitro* model using cadaver legs was developed to measure tibial nerve tension during passive range of motion, and the simulated early part of stance phase with different interventions performed. Since this is an *in vitro* model, there are many limitations which must be recognized before drawing conclusions from the data.

The cadaver legs used for this study were fresh frozen without embalming, and thawed for testing. The effects of freezing and thawing on the biomechanical properties of human peripheral nerves are unknown. Menisci (1), ligament (98,140), and cartilage (57) maintain their mechanical properties after freezing and thawing because they are mainly composed of collagen, which is highly resistant to autolysis (38). However, freezing and thawing significantly reduces tensile strength of muscle tissue compared to fresh specimens (38,65). This may be due to the high water content of muscle, which causes volume changes at low temperature, potential rupture of cell membranes, and damage to the intracellular contractile mechanism. Unlike muscle, nerves do not have a high water content, and consist

mainly of collagen, elastin, and lipids. Thus, the mechanical properties of peripheral nerves would be expected to be minimally affected by freezing and thawing, like other similar tissues. However, further investigation is required to clarify this issue.

To simulate the early part of stance phase, the cadaver leg was amputated through the knee joint, and the tibial plateau mounted in a pot. Any soft tissue attachments proximal to the knee would not be accounted for in this model. However, the proximal and distal branches of the tibial nerve were intact, and attachments of the nerve to the surrounding soft tissues were undisturbed. Since the transducer was situated between relatively fixed regions of the nerve proximally and distally, it was ideally situated to measure nerve tension (18,90).

During the analysis of the data, the relative changes in nerve tension were more important than the actual value of nerve tension recorded. Firstly, the tibial nerve tension measured did not represent the *in vivo* nerve tension because the tibial nerve was fixed proximally, which limited the excursion of the tibial nerve, and does not reflect the situation *in vivo*. Secondly, though the amount of strain and tension causing damage in rabbit peripheral nerves has been thoroughly studied (75,100,117), these data cannot be extrapolated to humans because the biomechanical properties of animal and human peripheral nerves are different. Even the biomechanical properties of different human peripheral nerves differ because the number of fascicles and volume of epineurium varies for different nerves (131). Since the amount of nerve tension producing damage in human tibial nerves is unknown, the nerve tension measured is meaningless without normal and abnormal reference values, making the relative change in tibial nerve tension for different interventions more important. However, further study is required to determine whether the statistically significant differences in this study are clinically significant.

Though the surgically created *in vitro* pes planus model simulates a pes planus foot radiographically, it represents the most severe form of pes planus deformity. Clinically, it is unlikely that a patient will have complete incompetence of all the ligaments and tendons released in this model. Furthermore, since it was acutely created without allowing for the soft tissues to accommodate the deformity, the soft tissues are under the greatest amount of tension. Since foot position and cyclical load with internal rotation of the leg were sustained for only a few seconds to measure nerve tension, stress relaxation, which results from a longer sustained force of at least 10 minutes (137), may not be a significant factor. In the *in*

vivo situation where a nerve is gradually stretched by a slowly progressive deformity, stress relaxation may be significant factor, which allows for elongation of the nerve without alteration in function (27,91,92,138).

Motion of the foot and ankle joints were not measured because the accurate measurement of foot and ankle joint motion requires sophisticated equipment, and techniques using three dimensional tracking of the joints (2,21,36), which is beyond the scope of this study. However, since there was no significant arthritis in the joints of the foot, the normal passive and functional range of motion in the literature would apply to our specimens. To reduce variability, interventions were performed sequentially on the same specimen, and data analysis accounted for this repeated measures design. Furthermore, for foot position, the same examiner manipulated the foot with the goal of achieving the greatest possible position.

Significance:

Tarsal tunnel syndrome is due to compression and/or tension of the tibial nerve or its branches. In the presence of a space-occupying lesion, tarsal tunnel release is indicated to relieve nerve compression (10,25,26,50,81,89,106,135). However, in idiopathic cases, tarsal tunnel release has had poor results and has been discouraged by some authors (81,106,135). The pes planus foot consists of a valgus hindfoot and abducted forefoot, which together stretches the tibial nerve and compresses it against bone. Though the contribution of nerve compression and tension to tarsal tunnel syndrome in pes planus feet is unknown, tibial and lateral plantar nerve tension have been quantified in this study. This study has shown that tibial and lateral plantar nerve tension is dependent on foot position, cyclical load with internal rotation, and skeletal stability. Surgical procedures, such as surgically created pes planus deformity and tarsal tunnel release, destabilize the foot, increase motion, and increase deformity, which results in increased nerve tension. Surgical procedures, such as distraction calcaneocuboid and triple arthrodesis, stabilize the foot, decrease motion, and correct deformity, which results in decreased nerve tension.

In pes planus feet without extrinsic nerve compression, increased tibial and lateral plantar nerve tension may be a cause of tarsal tunnel syndrome which is unrecognized. Tarsal tunnel release increases tibial nerve tension, and can further exacerbate the underlying condition. However, even in the absence of a space-occupying lesion and pes planus

deformity, repetitive ankle dorsiflexion can increase tibial and lateral plantar nerve tension and be a cause of tarsal tunnel syndrome. Physicians must ask specifically about repetitive ankle dorsiflexion, and carefully examine the foot structure in all patients with tarsal tunnel syndrome to ensure a pes planus deformity is not present.

Though simulated arthrodesis corrects foot deformity, restricts motion, and decreases nerve tension, the use of these procedures is highly experimental, and is not indicated for treating this syndrome currently. However, orthotics have identical treatment goals, which explains the reported improvement in symptoms with their use in these patients (13,14,25,28,32,111), and should be extensively used before considering surgery. Since orthotics are externally applied, unlike simulated arthrodesis which uses internal fixation, the correction of the foot deformity and the maintenance of the correction is much more variable, and only useful for flexible deformities. After failure of non-operative treatment with orthotics, tarsal tunnel release in patients with a pes planus foot should be carefully considered, and if performed, post-operative stabilization of the foot and ankle externally with orthotics is recommended to decrease nerve tension.

Summary:

Many authors have postulated that tarsal tunnel syndrome can result from increased tibial nerve tension in pes planus feet. This is the first study to measure tibial nerve tension in an *in vitro* surgically created pes planus foot model during dorsiflexion, eversion, combined dorsiflexion-eversion, and cyclical load with internal rotation of the leg.

This study has shown that tibial nerve tension is significantly increased in a pes planus foot compared to the baseline foot for all conditions, which supports the hypothesis of many authors. After tarsal tunnel release, tibial nerve tension was further increased during eversion, combined dorsiflexion-eversion, and cyclical load compared to the pes planus foot. The triple arthrodesis was more effective at decreasing tibial nerve tension than the distraction calcaneocuboid arthrodesis.

Skeletally instability resulting due to a surgically created pes planus deformity or a tarsal tunnel release increased tibial nerve tension. Procedures which corrected deformity and increased stability, decreased tibial nerve tension. However, since this is a biomechanical study, further investigation is required to determine whether these statistically significant increases in nerve tension are clinically significant. If increased tibial nerve tension in pes planus feet contributes to tarsal tunnel syndrome, then stabilization of the foot may be an interesting new direction to consider in patients with severe pes planus feet and no space-occupying lesion.

Future Directions:

In a surgically created pes planus foot, we have measured tibial nerve tension after tarsal tunnel release, and after stabilization procedures. However, as the lateral plantar nerve wraps around the calcaneus, the tibial nerve is stretched and also compressed against the bony floor of the tarsal tunnel. As a result, the final tibial neuropathy in tarsal tunnel syndrome and pes planus feet may be due to variable amounts of tibial nerve compression and tension. This current study quantifies the effect of pes planus deformity, tarsal tunnel release, and stabilization procedures on tibial nerve tension, but, the same model could be used to study the effect of these procedures on the compressive forces of the tibial nerve as it is stretched. The compression of the tibial nerve against the bony floor of the tarsal tunnel could be measured with Fuji film or a transducer.

In surgically created pes planus feet, this current investigation demonstrated that tibial nerve tension was significantly decreased by fusion of the pes planus deformity in a neutral position. However, could correction of the pes planus deformity without fusion of joints alter tibial nerve tension? A medial displacement calcaneal osteotomy corrects the hindfoot valgus deformity without fusion of joints. Using the same experimental design as this current investigation, we could measure the effect of a medial displacement calcaneal osteotomy on tibial nerve tension in a surgically created pes planus foot.

Tables

Table 1: Effect of foot position on nerve tension (newtons).

	Dorsiflexion		Eversion		Both	
Specimen	**Baseline**	**Pes Planus**	**Baseline**	**Pes Planus**	**Baseline**	**Pes Planus**
1	3.70	3.00	2.68	6.06	6.37	9.10
2	2.53	2.61	3.98	5.46	7.20	9.33
3	13.57	16.43	11.06	11.85	18.62	20.52
4	12.34	14.41	11.80	15.58	16.11	20.09
5	5.67	5.79	6.68	6.79	13.65	14.98
6	6.51	9.83	11.76	16.40	17.33	19.45
7	7.65	11.06	9.23	12.03	10.59	14.23
8	16.10	19.45	6.27	11.64	17.77	24.37
9	7.85	12.87	6.70	12.33	11.42	15.73
Mean	8.44	10.61	7.79	10.90	13.23	16.42
Standard Deviation	4.61	5.89	3.35	3.98	4.59	5.17
Mean Square*	42.35		86.94		91.78	
F value*	11.42		22.08		34.53	
Pr > F*	0.0097		0.0015		0.0004	

Legend:

* = Comparison of nerve tension for baseline and pes planus feet using a paired t-test.

Table 2a: 2 factor repeated measures ANOVA for stability (pes planus vs. baseline) and internal rotation on nerve tension (newtons).

Factor	Mean Square	F value	Pr > F
Stability	10.02	42.89	0.0002
Internal Rotation	1.99	12.01	0.0001
Interaction	0.04	1.34	0.2769

Table 2b: Contrasts of nerve tension (newtons) between the nth degree of internal rotation and 0 degrees of internal rotation for all data using a paired t-test.

Nth Degree of Internal Rotation	Mean Square	F value	Pr > F
5 degrees	1.40	5.01	0.0556
10 degrees	7.47	11.69	0.0091
15 degrees	15.61	9.50	0.0151
20 degrees	24.01	13.89	0.0058

Table 2c: Effect of cyclical axial load and increasing internal rotation on nerve tension (newtons) in stable baseline feet.

	BASELINE INTERNAL ROTATION (degrees)				
Specimen	**0**	**5**	**10**	**15**	**20**
1	0.25	0.18	0.32	0.18	0.36
2	0.31	0.23	0.23	0.39	0.58
3	0.17	0.22	0.39	0.60	0.94
4	0.21	0.18	0.21	0.19	0.20
5	0.46	0.52	0.60	0.72	0.89
6	0.50	0.79	1.27	1.89	2.25
7	0.13	0.19	0.20	0.25	0.23
8	0.33	0.45	0.63	0.84	1.00
9	0.42	0.52	0.67	0.88	1.06
Mean*	0.28	0.31	0.42	0.50	0.64
Standard Deviation*	1.60	1.77	1.88	2.22	2.23
Mean Square+	--	0.11	1.42	3.03	6.15
F value+	--	1.22	9.37	9.08	19.77
Pr > F+	--	0.3008	0.0156	0.0167	0.0021

Legend:

* = Anti-logarithm of the mean and standard deviation of the transformed data.

+ = Contrast of nerve tension between nth degree of internal rotation and 0 degrees using a paired t-test.

Table 2d: Effect of cyclical axial load and increasing internal rotation on nerve tension (Newtons) in unstable pes planus feet.

	PES PLANUS INTERNAL ROTATION (degrees)				
Specimen	**0**	**5**	**10**	**15**	**20**
1	0.91	0.85	0.76	0.69	0.71
2	0.37	0.47	0.58	0.58	0.69
3	0.34	0.56	0.85	1.28	1.57
4	0.41	0.37	0.33	0.36	0.35
5	0.51	0.75	1.13	1.60	1.91
6	0.68	1.28	2.21	5.40	5.81
7	0.46	0.58	0.63	0.69	0.57
8	0.33	0.61	0.83	0.97	1.08
9	0.80	0.94	1.27	1.43	1.55
Mean*	0.50	0.67	0.84	1.05	1.12
Standard Deviation*	1.46	1.46	1.71	2.18	2.29
Mean Square^{+}	–	0.73	2.38	4.88	5.86
F value^{+}	–	10.19	10.10	8.91	9.08
Pr > F^{+}	–	0.0128	0.0130	0.0175	0.0167

Legend:

* = Anti-logarithm of the mean and standard deviation of the transformed data.

+ = Contrast of nerve tension between nth degree of internal rotation and 0 degrees using a paired t-test.

Table 3a: The effect of interventions on tibial nerve tension (newtons) during dorsiflexion.

Specimen	Baseline	Pes Planus	Tarsal Tunnel Release	CC Arthrodesis	Triple Arthrodesis
1	24.79	33.46	31.17	19.40	17.20
2	19.38	30.02	36.54	30.47	28.57
3	13.24	20.25	22.66	19.28	18.01
4	34.06	57.19	51.52	36.95	34.73
5	7.25	7.42	8.15	9.61	7.03
6	20.59	34.37	34.06	33.52	26.56
7	17.85	31.91	35.62	28.63	30.55
8	20.45	30.84	27.03	19.88	18.36
9	23.34	35.00	37.38	25.27	27.85
10	30.47	44.34	39.53	38.12	28.32
11	15.58	26.13	22.50	19.88	22.66
Mean	20.64	31.90	31.47	25.55	23.62
Standard Deviation	7.55	12.59	11.29	8.84	7.88
Mean Square	1396.65*	2.06+	385.98#	677.64$	
F value	44.87*	0.13+	14.23#	26.07$	
Pr > F	0.0001*	0.7224+	0.0036#	0.0005$	

Legend:

* = Comparison of nerve tension between pes planus and baseline using a paired t-test.

+ = Comparison of nerve tension between tarsal tunnel release and pes planus using a paired t-test.

= Comparison of nerve tension between CC arthrodesis and tarsal tunnel release using a paired t-test.

$ = Comparison of nerve tension between triple arthrodesis and tarsal tunnel release using a paired t-test.

Table 3b: The effect of interventions on tibial nerve tension (newtons) during eversion.

Specimen	Baseline	Pes Planus	Tarsal Tunnel Release	CC Arthrodesis	Triple Arthrodesis
1	11.21	17.31	21.15	20.35	11.29
2	5.49	4.61	13.28	13.85	8.65
3	4.12	5.08	14.36	16.02	7.87
4	7.89	31.17	40.39	34.10	17.93
5	3.97	9.08	4.38	9.51	1.93
6	17.54	29.40	33.52	35.08	29.08
7	17.89	19.57	17.66	22.15	31.21
8	9.18	15.61	20.94	17.97	17.31
9	6.43	7.81	12.97	13.75	11.60
10	15.39	24.84	27.62	24.73	15.70
11	16.87	15.35	12.81	10.58	7.15
Mean	10.54	16.35	19.92	19.83	14.52
Standard Deviation	5.50	9.30	10.42	8.65	9.07
Mean Square	370.69*	139.93+	0.09#	319.97$	
F value	7.20*	6.00+	0.01#	4.30$	
Pr > F	0.0229*	0.0342+	0.9329#	0.0648$	

Legend:

* = Comparison of nerve tension between pes planus and baseline using a paired t-test.

+ = Comparison of nerve tension between tarsal tunnel release and pes planus using a paired t-test.

= Comparison of nerve tension between CC arthrodesis and tarsal tunnel release using a paired t-test.

$ = Comparison of nerve tension between triple arthrodesis and tarsal tunnel release using a paired t-test.

Table 3c: The effect of interventions on tibial nerve tension (newtons) during combined dorsiflexion-eversion.

Specimen	Baseline	Pes Planus	Tarsal Tunnel Release	CC Arthrodesis	Triple Arthrodesis
1	30.04	35.72	38.28	33.59	27.07
2	24.16	29.65	40.04	40.39	30.61
3	13.20	13.83	21.04	26.97	22.75
4	46.68	55.12	57.97	55.74	44.06
5	10.29	12.29	13.67	13.07	9.59
6	25.35	41.00	49.38	46.37	36.68
7	30.66	41.46	45.90	37.74	43.60
8	24.81	30.66	31.60	28.98	24.96
9	18.61	27.30	30.74	27.50	24.18
10	36.45	44.53	39.72	45.86	34.50
11	22.30	24.96	24.26	25.27	23.32
Mean	25.69	32.41	35.69	34.68	29.21
Standard Deviation	10.29	12.93	13.04	12.03	10.11
Mean Square	497.23*		118.34+	11.22#	461.88$
F value	26.98*		6.42+	0.61#	18.65$
Pr > F	0.0004*		0.0297+	0.4519#	0.0015$

Legend:

* = Comparison of nerve tension between pes planus and baseline using a paired t-test.

+ = Comparison of nerve tension between tarsal tunnel release and pes planus using a paired t-test.

= Comparison of nerve tension between CC arthrodesis and tarsal tunnel release using a paired t-test.

$ = Comparison of nerve tension between triple arthrodesis and tarsal tunnel release using a paired t-test.

Table 4a: The mean tibial nerve tension (newtons) for cyclical load with increasing internal rotation.

Internal Rotation (degrees)	0	15	25
Mean Tibial Nerve Tension (N)*	0.36	1.24	2.41
Standard Deviation*	1.83	2.64	2.57
Mean Square+	--	415.51	981.84
F value+	--	25.89	63.90
Pr > F+	--	0.0005	0.0001

Legend:

* = Anti-logarithm of the mean and standard deviation of the transformed data.

+ = Contrast of nerve tension between nth degree of internal rotation and 0 degrees using a paired t-test.

Table 4b: The mean tibial nerve tension (newtons) for each intervention.

Intervention	Baseline	Pes Planus	Tarsal Tunnel Release	CC Arthrodesis	Triple Arthrodesis
Mean Nerve Tension (N)!	0.70	1.15	1.44	0.95	1.05
Standard Deviation!	2.96	3.14	3.18	3.46	3.02
Mean Square	23.75*	4.97+	16.95#	9.82$	
F value	20.33*	6.44+	6.89#	6.73$	
Pr > F	0.0011*	0.0295+	0.0253#	0.0267$	

Legend:

! = Anti-logarithm of the mean and standard deviation of the transformed data.

* = Comparison of nerve tension between pes planus and baseline using a paired t-test.

+ = Comparison of nerve tension between tarsal tunnel release and pes planus using a paired t-test.

= Comparison of nerve tension between CC arthrodesis and tarsal tunnel release using a paired t-test.

$ = Comparison of nerve tension between triple arthrodesis and tarsal tunnel release using a paired t-test.

Table 5a: The effect of cyclical load with internal rotation on tibial nerve tension (newtons) for the baseline foot.

	Baseline Internal Rotation (degrees)		
Specimen	**0**	**15**	**25**
1	0.14	1.40	3.02
2	0.45	0.85	0.92
3	0.48	0.35	0.64
4	0.35	2.02	2.78
5	0.17	0.43	0.75
6	0.31	3.27	6.63
7	0.30	0.94	2.71
8	0.32	2.26	3.41
9	0.19	0.18	0.26
10	0.36	1.64	2.48
11	0.21	0.21	0.54
Mean*	0.28	0.84	1.49
Standard Deviation*	1.51	2.71	2.70
Mean Square+	0.38	1.07	--
F value+	1.11	7.76	--
Pr > F+	0.3177	0.0192	--

Legend:

* = Anti-logarithm of the mean and standard deviation of the transformed data.

+ = Comparison of the effects of the interaction between pes planus and baseline, and nth degree of internal rotation and 25 degrees of internal rotation on nerve tension using a paired t-test.

Table 5b: The effect of surgically created pes planus release and cyclical load with internal rotation on tibial nerve tension (newtons).

	Pes Planus Internal Rotation (degrees)		
Specimen	**0**	**15**	**25**
1	0.44	1.48	3.88
2	0.37	0.40	1.03
3	0.73	0.51	1.80
4	1.03	4.75	9.21
5	0.21	0.47	1.31
6	0.51	3.47	7.87
7	0.38	1.33	3.18
8	0.73	3.87	5.84
9	0.31	0.21	0.60
10	0.61	3.07	8.12
11	0.20	0.87	1.75
Mean*	0.44	1.19	2.87
Standard Deviation*	1.69	2.91	2.54

Legend:

* = Anti-logarithm of the mean and standard deviation of the transformed data.

Table 5c: The effect of tarsal tunnel release and cyclical load with internal rotation on tibial nerve tension (newtons).

	Tarsal Tunnel Release Internal Rotation (degrees)		
Specimen	**0**	**15**	**25**
1	0.39	1.55	2.74
2	0.38	0.53	0.64
3	0.46	1.19	2.96
4	0.99	6.02	12.72
5	0.18	0.88	1.65
6	0.75	4.91	9.60
7	0.51	2.33	3.88
8	1.41	4.80	7.22
9	0.20	1.21	2.56
10	0.69	3.80	7.80
11	0.26	0.77	1.11
Mean*	0.47	1.87	3.38
Standard Deviation*	1.92	2.33	2.55

Legend:

* = Anti-logarithm of the mean and standard deviation of the transformed data.

Table 5d: The effect of calcaneocuboid arthrodesis and cyclical load with internal rotation on tibial nerve tension (newtons).

	CC Arthrodesis Internal Rotation (degrees)		
Specimen	**0**	**15**	**25**
1	0.97	1.99	2.93
2	0.66	0.59	0.88
3	0.94	0.72	1.77
4	0.11	2.89	4.68
5	0.11	0.41	0.96
6	0.53	4.13	9.02
7	0.24	1.33	2.57
8	0.19	2.78	4.84
9	0.25	0.31	0.70
10	0.17	4.31	8.82
11	0.39	0.28	0.81
Mean*	0.31	1.17	2.34
Standard Deviation*	2.23	2.82	2.59

Legend:

* = Anti-logarithm of the mean and standard deviation of the transformed data.

Table 5e: The effect of triple arthrodesis and cyclical load with internal rotation on tibial nerve tension (newtons).

	Triple Arthrodesis Internal Rotation (degrees)		
Specimen	**0**	**15**	**25**
1	0.73	0.53	0.80
2	0.43	1.13	1.40
3	0.52	0.67	1.06
4	0.26	5.65	7.67
5	0.22	0.46	1.18
6	0.15	2.79	7.15
7	0.23	2.89	6.00
8	0.43	2.71	4.39
9	0.32	0.73	1.36
10	0.69	3.11	4.10
11	0.38	0.53	1.29
Mean*	0.36	1.35	2.39
Standard Deviation*	1.65	2.47	2.37

Legend:

* = Anti-logarithm of the mean and standard deviation of the transformed data.

Figures

Figure 1a: The anatomy of the tibial nerve and its branches.

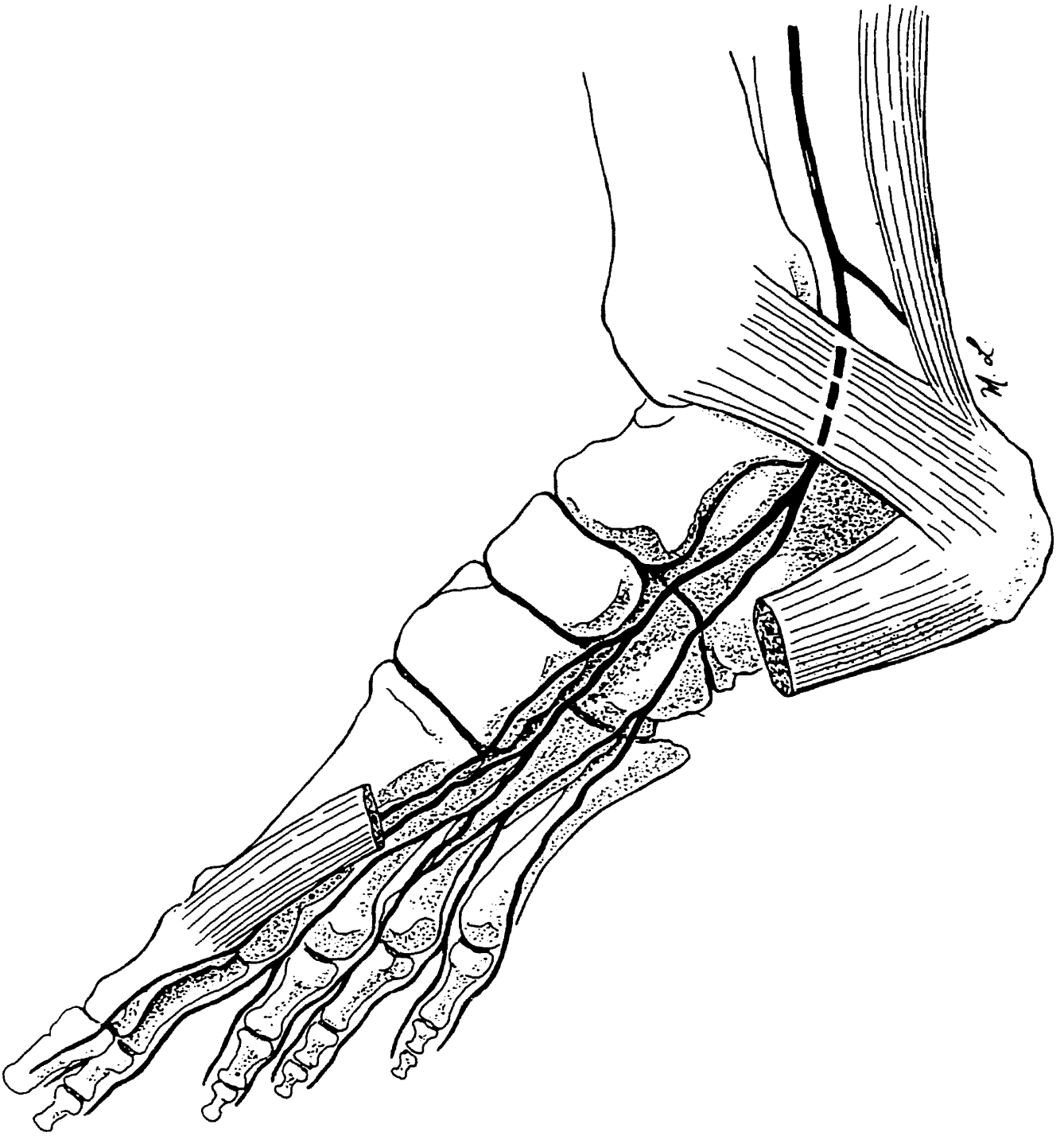
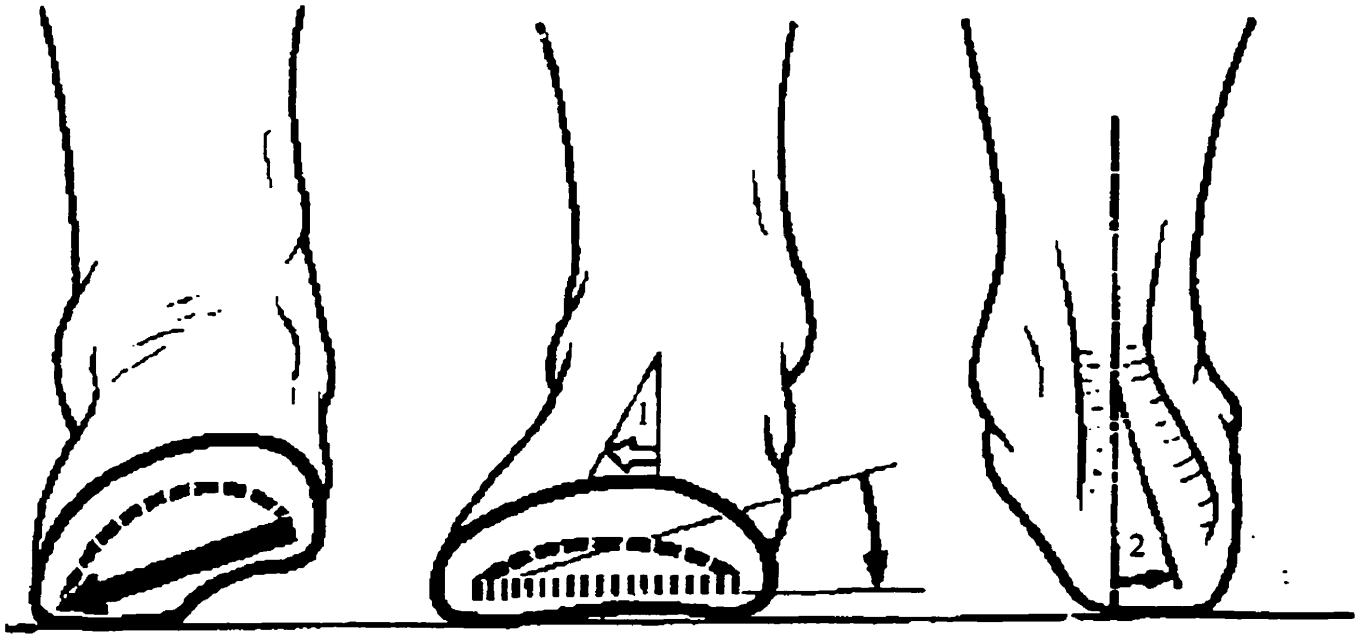


Figure 1b: Pes planus foot deformity consisting of: (1) abducted forefoot, and (2) valgus heel.



Reproduced with permission from Kapandji I.A.: The Plantar Vault, in Kapandji, I.A. (ed.): *Physiology of the Joints: Volume 2*, New York, NY, Churchill Livingstone, 1987, p. 239. © Churchill Livingstone, 1987.

Figure 2: The proximal tibia was rigidly positioned in an aluminum pot with set screws. The leg specimen was placed on the MTS base-plate, and loaded proximally through a hinged articulation. The tibial nerve was sutured to the transducer in a physiological direction, and attached to the supporting arm of an adjustable machined rig secured to the distal tibia by a plate with screws.

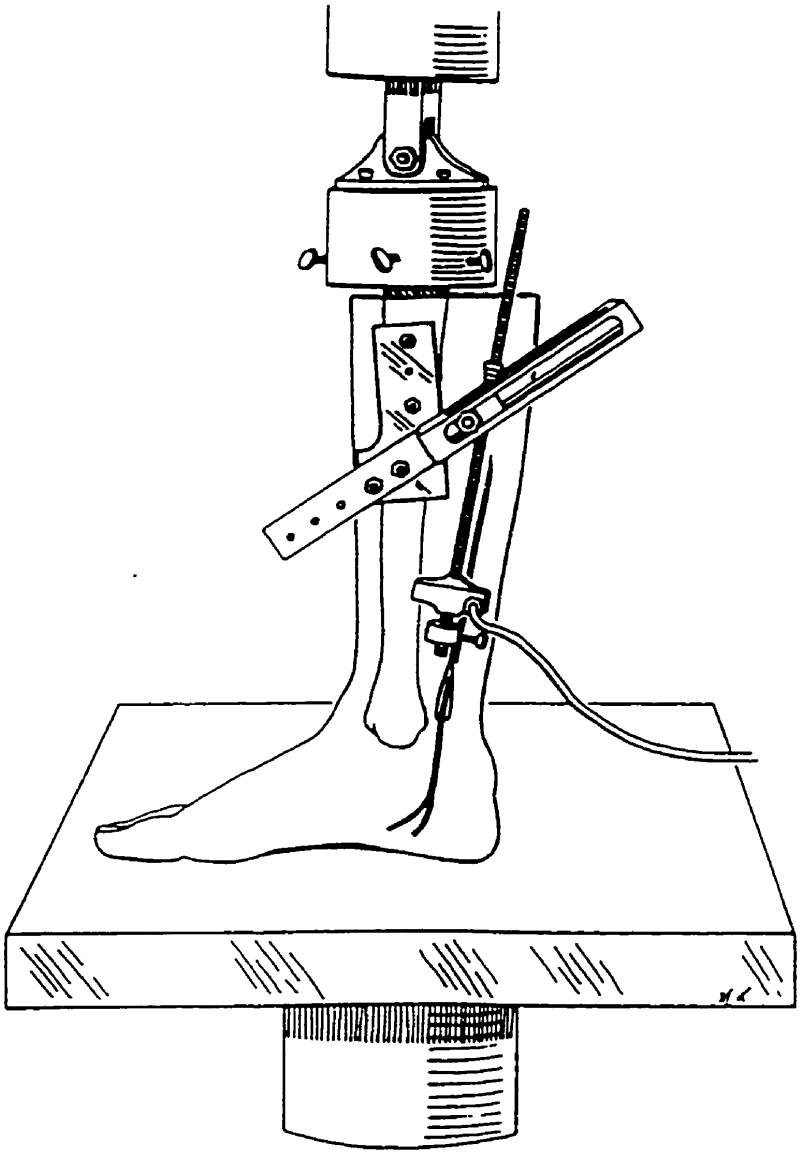
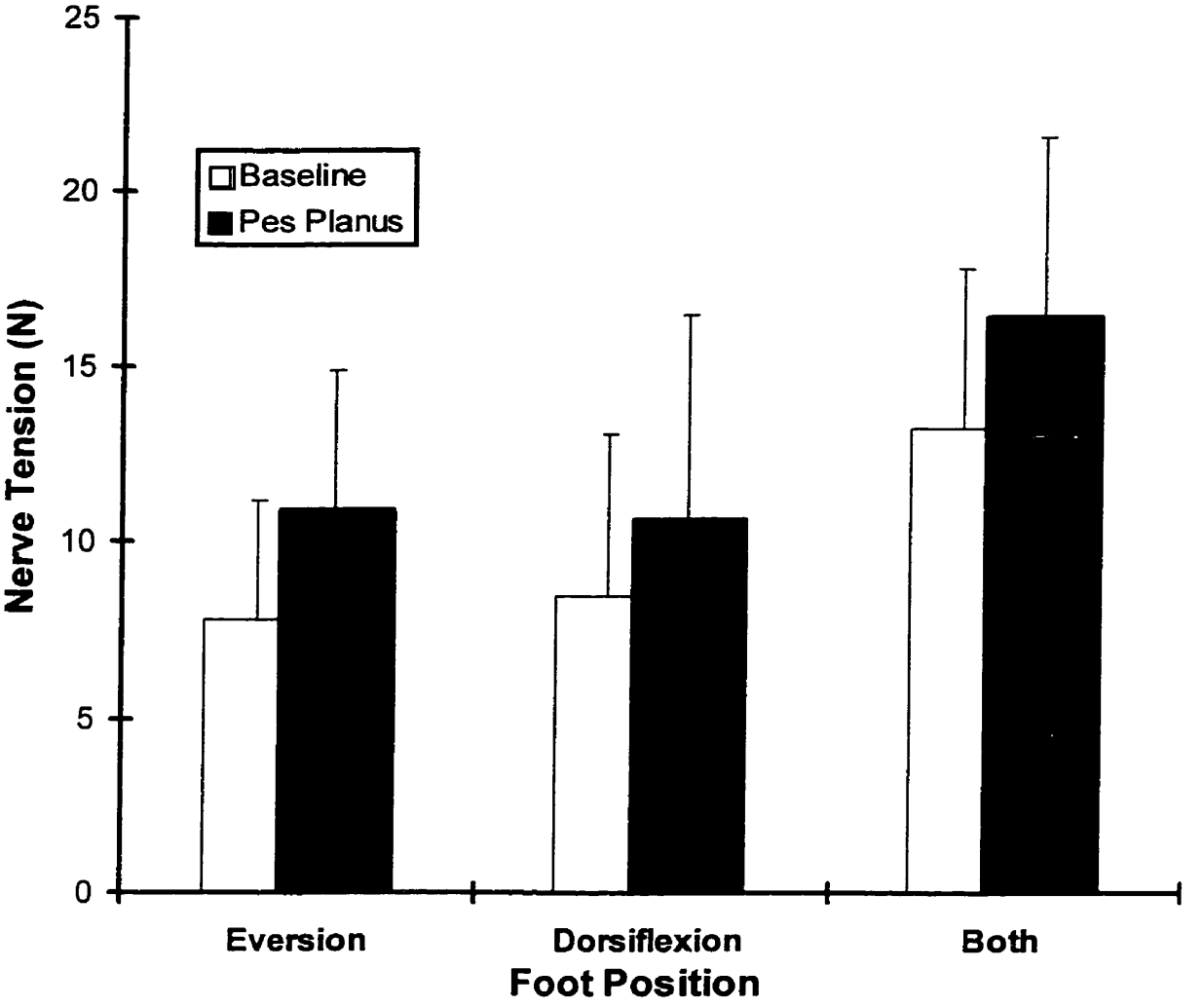


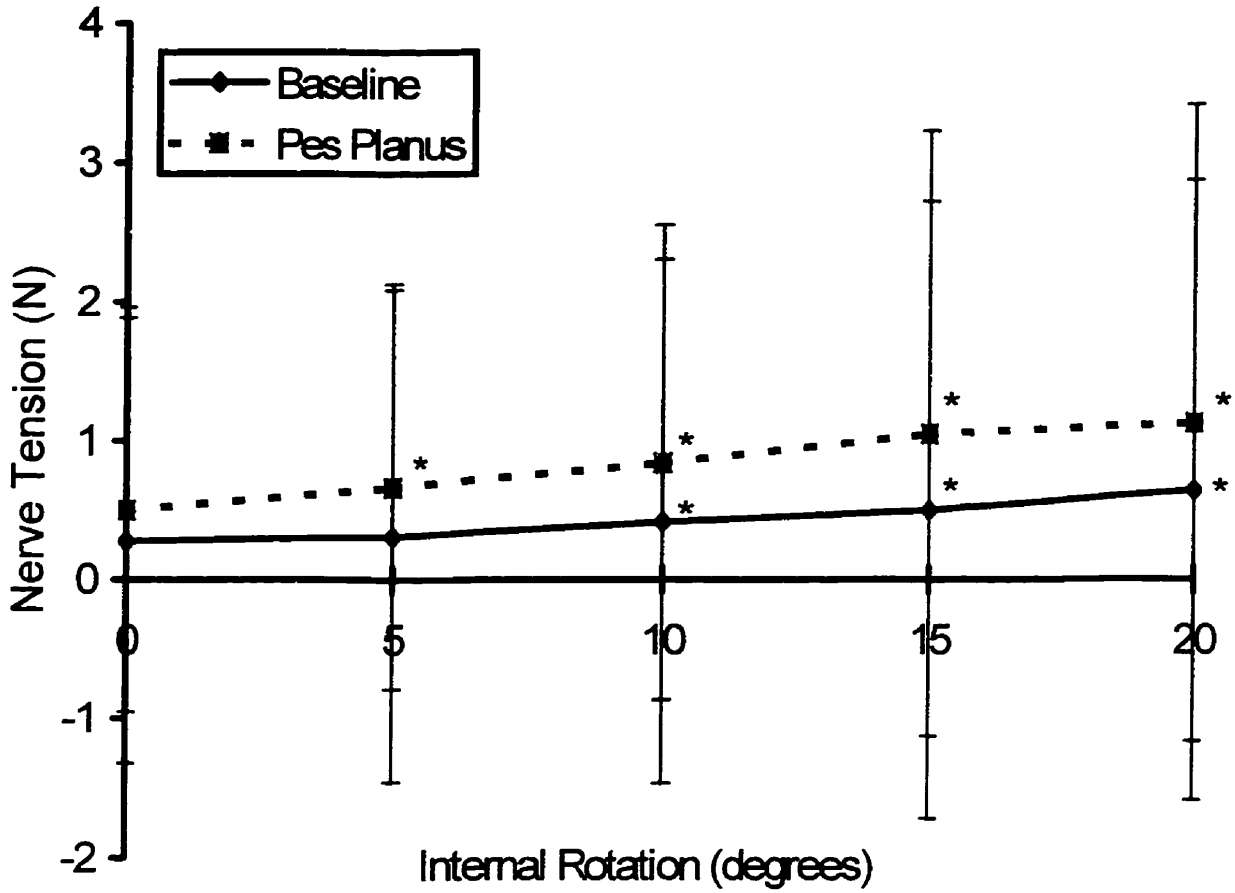
Figure 3: A photograph of the lateral plantar nerve attached to the transducer by a 2-0 Ethibond suture.

Figure 4: The Effect of Foot Position On Nerve Tension (Mean +/- SD)



Pes Planus foot significantly increased lateral plantar nerve tension compared to baseline for all foot positions ($p<0.05$).

Figure 5: The Effect of Axial Load and Internal Rotation on Nerve Tension (Mean +/- SD)



* = Significantly greater nerve tension than 0° of internal rotation.

Using a 2 factor repeated measures ANOVA,

- Pes Planus significantly increased lateral plantar nerve tension compared to baseline during 0°, 5°, 10°, 15°, and 20° of internal rotation ($p=0.0002$).
- Increasing internal rotation significantly increased lateral plantar nerve tension ($p=0.0001$)
- No significant interaction between intervention and internal rotation ($p=0.2769$).

Figure 6: A photograph of the cadaver leg set up on the MTS base-plate following tarsal tunnel release.

Figure 7: A photograph of the cadaver leg set up on the MTS base-plate following distraction calcaneocuboid arthrodesis.

Figure 8a: A photograph of the cadaver leg set up on the MTS base-plate following triple arthrodesis. For testing, the Steinman pins were cut to the level of the skin.

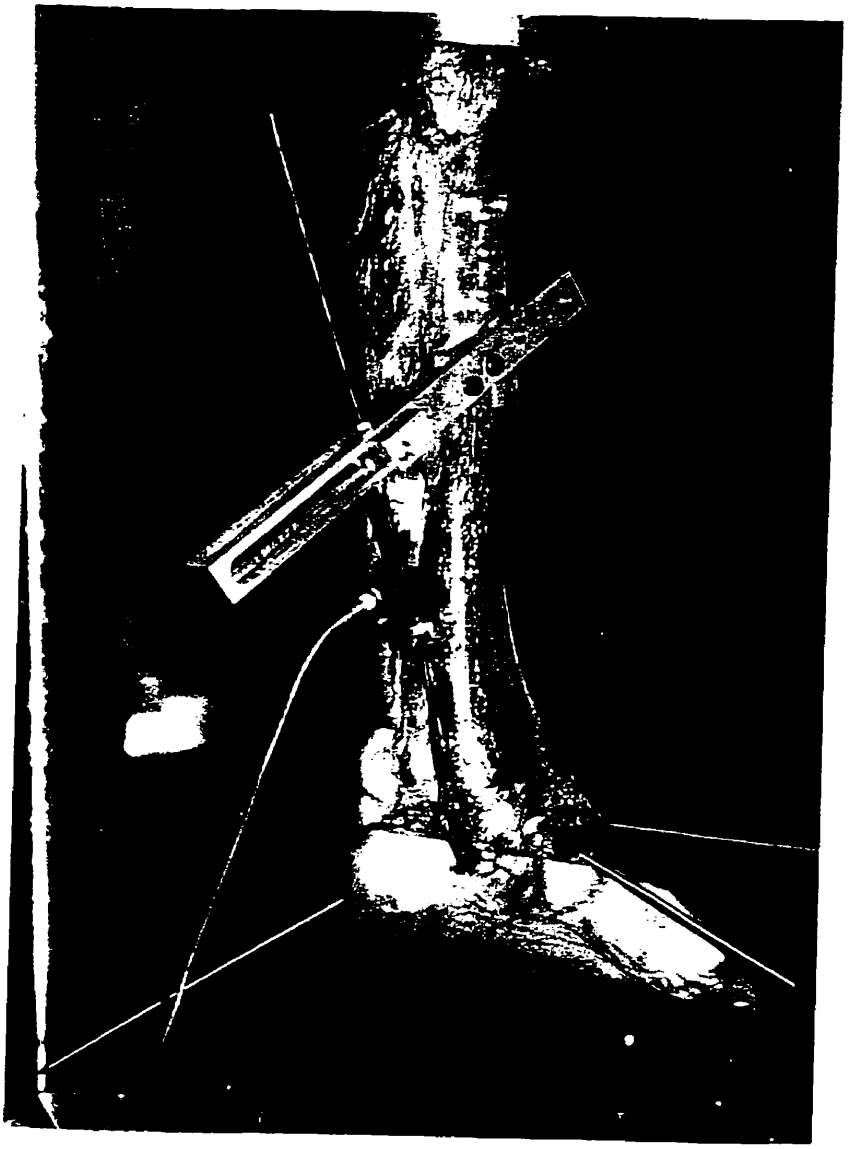


Figure 8b: A photograph of the cadaver leg set up on the MTS base-plate following triple arthrodesis. For testing, the Steinman pins were cut to the level of the skin.

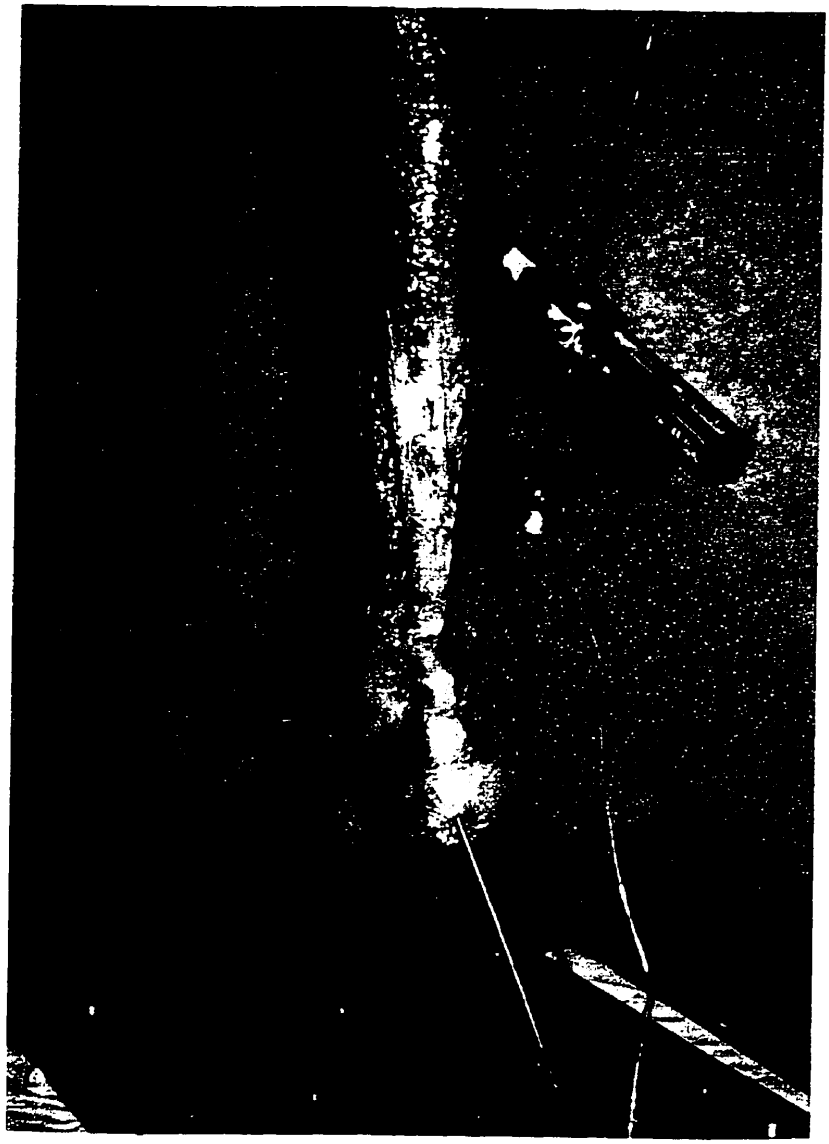


Figure 8c: A photograph of the cadaver leg set up on the MTS base-plate following triple arthrodesis. For testing, the Steinman pins were cut to the level of the skin.

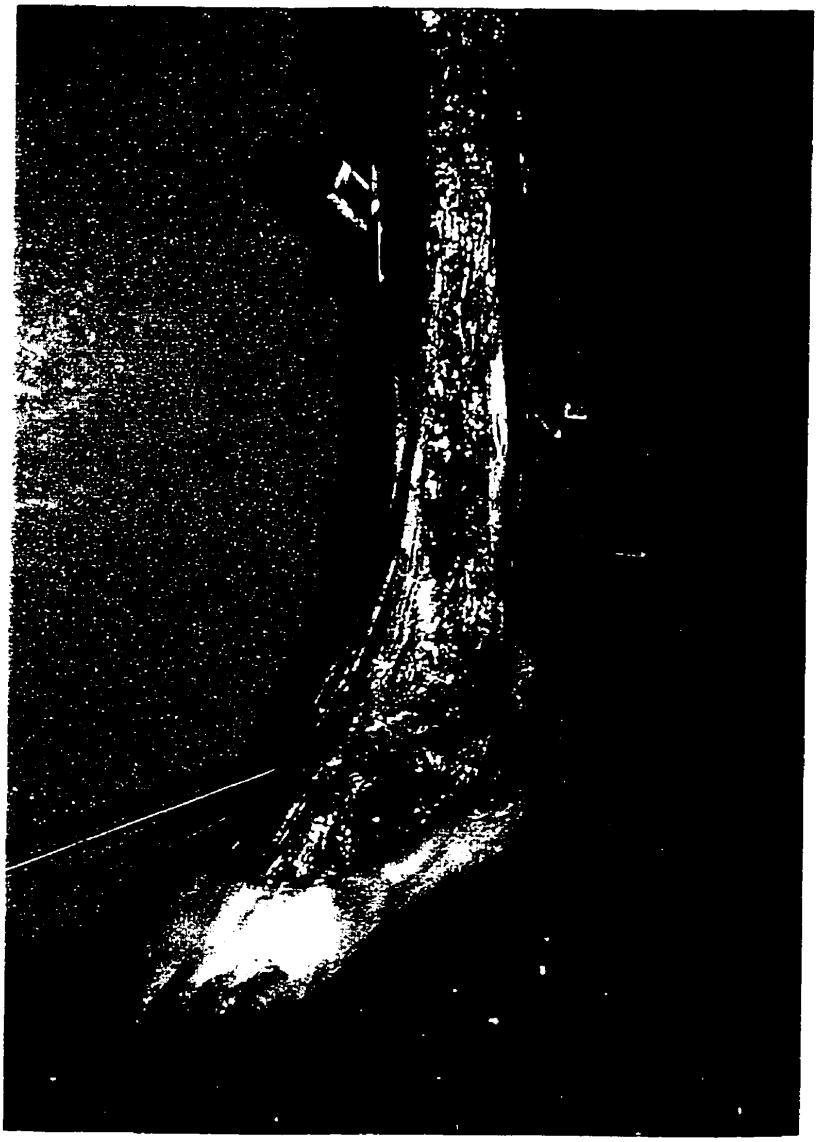


Figure 9: The Effect of Interventions on Tibial Nerve Tension During Dorsiflexion (Mean +/- SD)

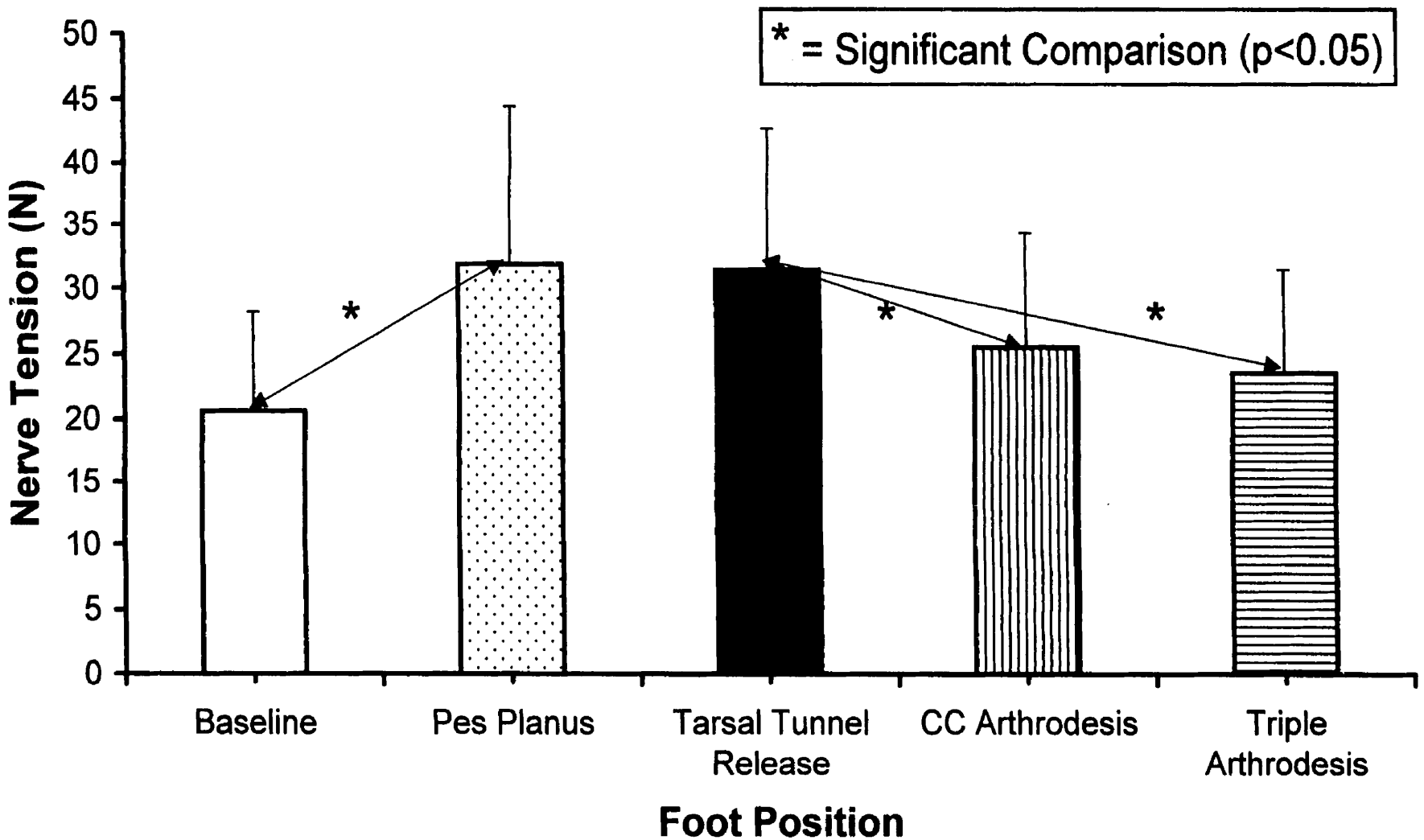


Figure 10: The Effect of Interventions on Tibial Nerve Tension During Eversion (Mean +/- SD)

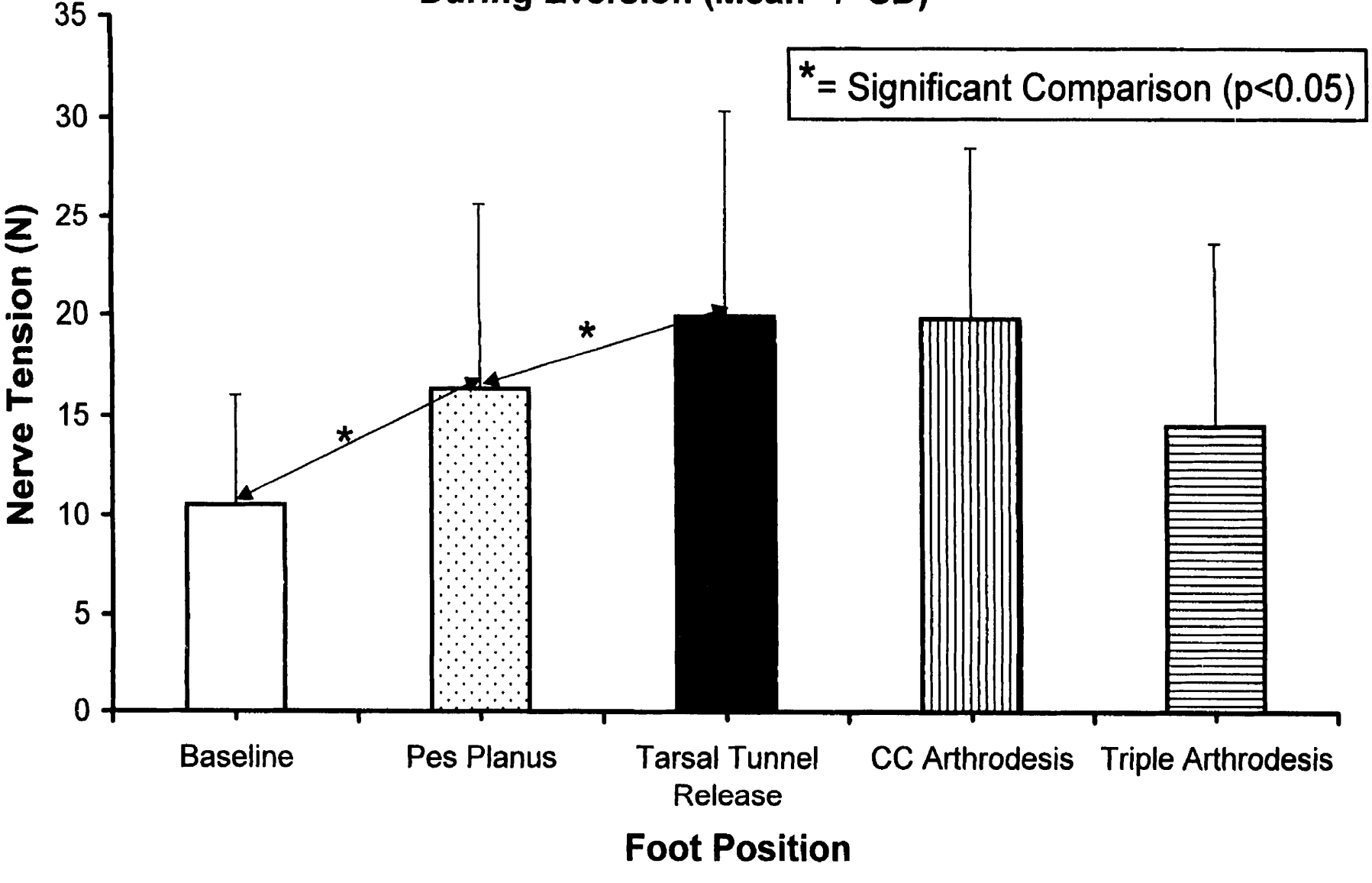
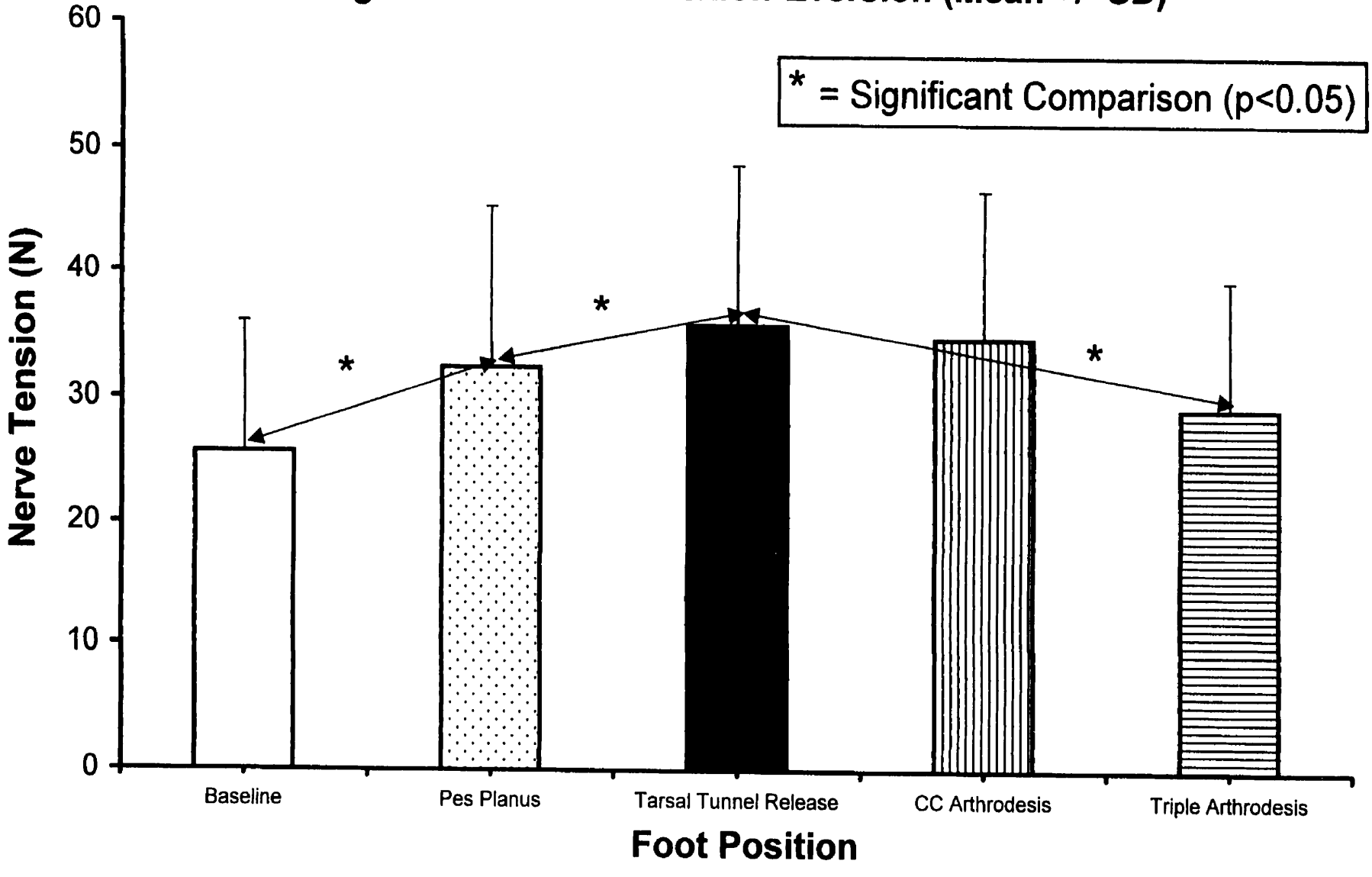


Figure 11: The Effect of Interventions on Tibial Nerve Tension During Combined Dorsiflexion-Eversion (Mean +/- SD)



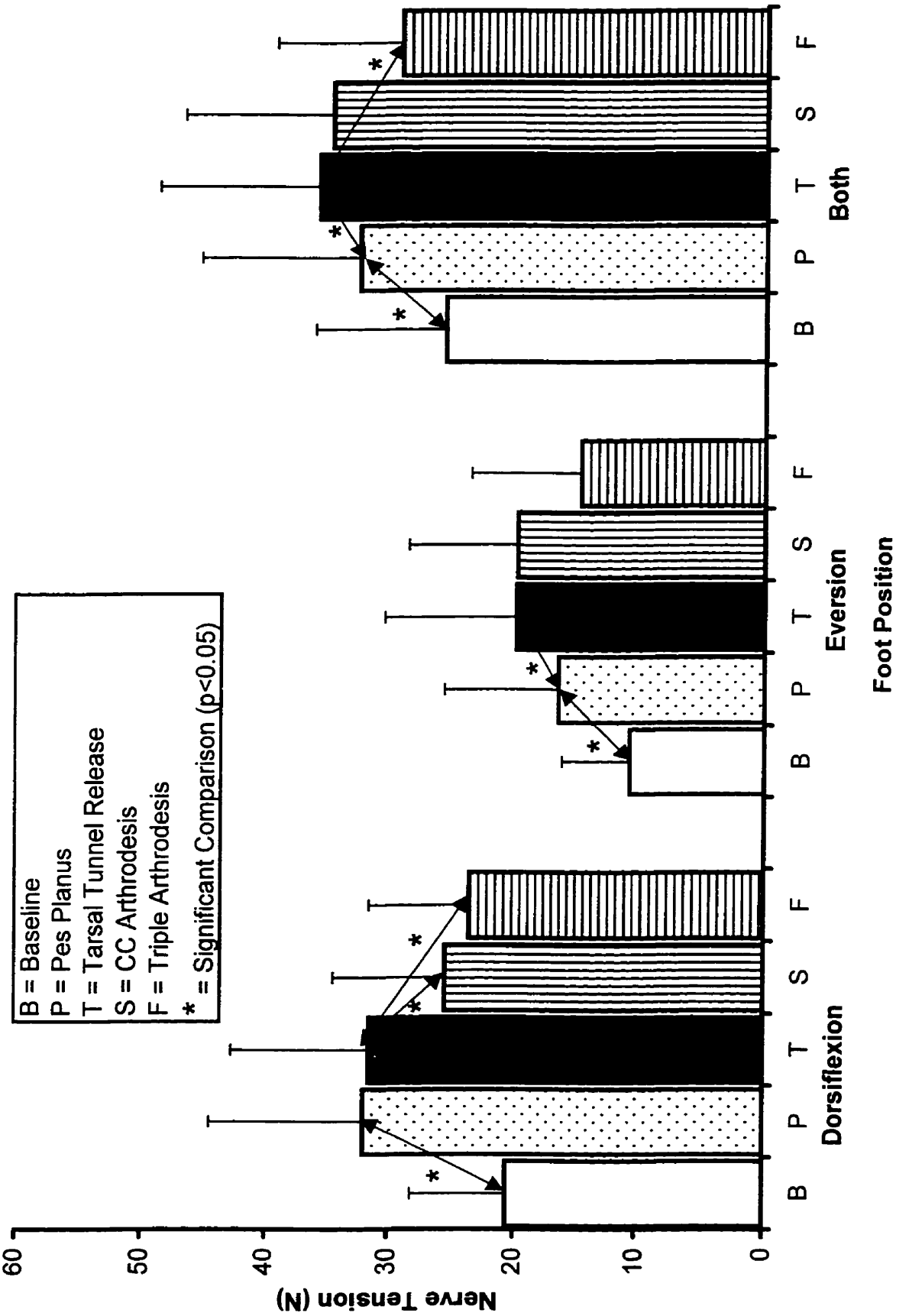


Figure 12: The Effect of Foot Position on Nerve Tension (Mean +/- SD)

Figure 13: The Effect of Cyclical Load with Internal Rotation on Tibial Nerve Tension (Mean +/- SD)

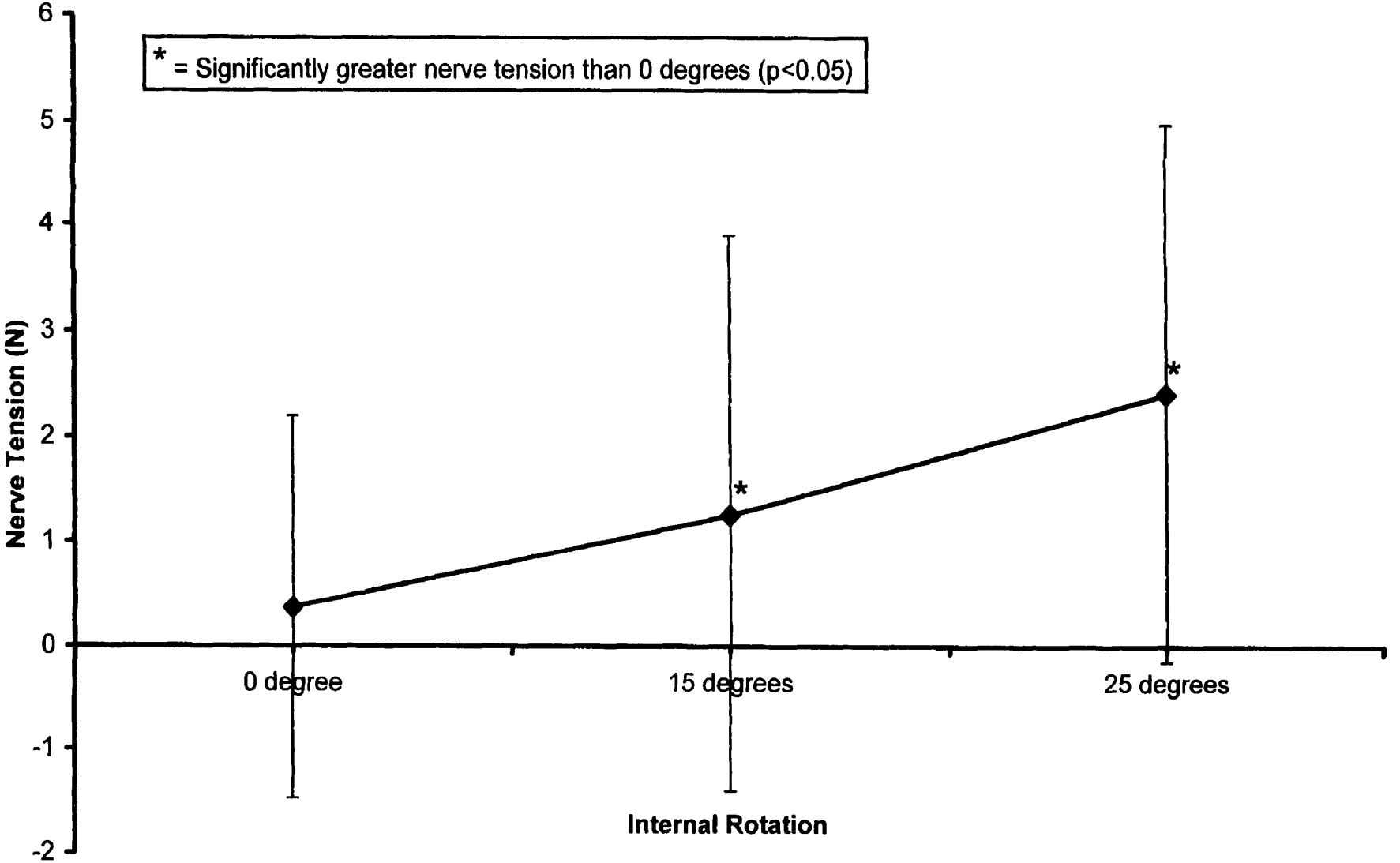


Figure 14: The Effect of Interventions on Tibial Nerve Tension under Cyclical Load (Mean +/- SD)

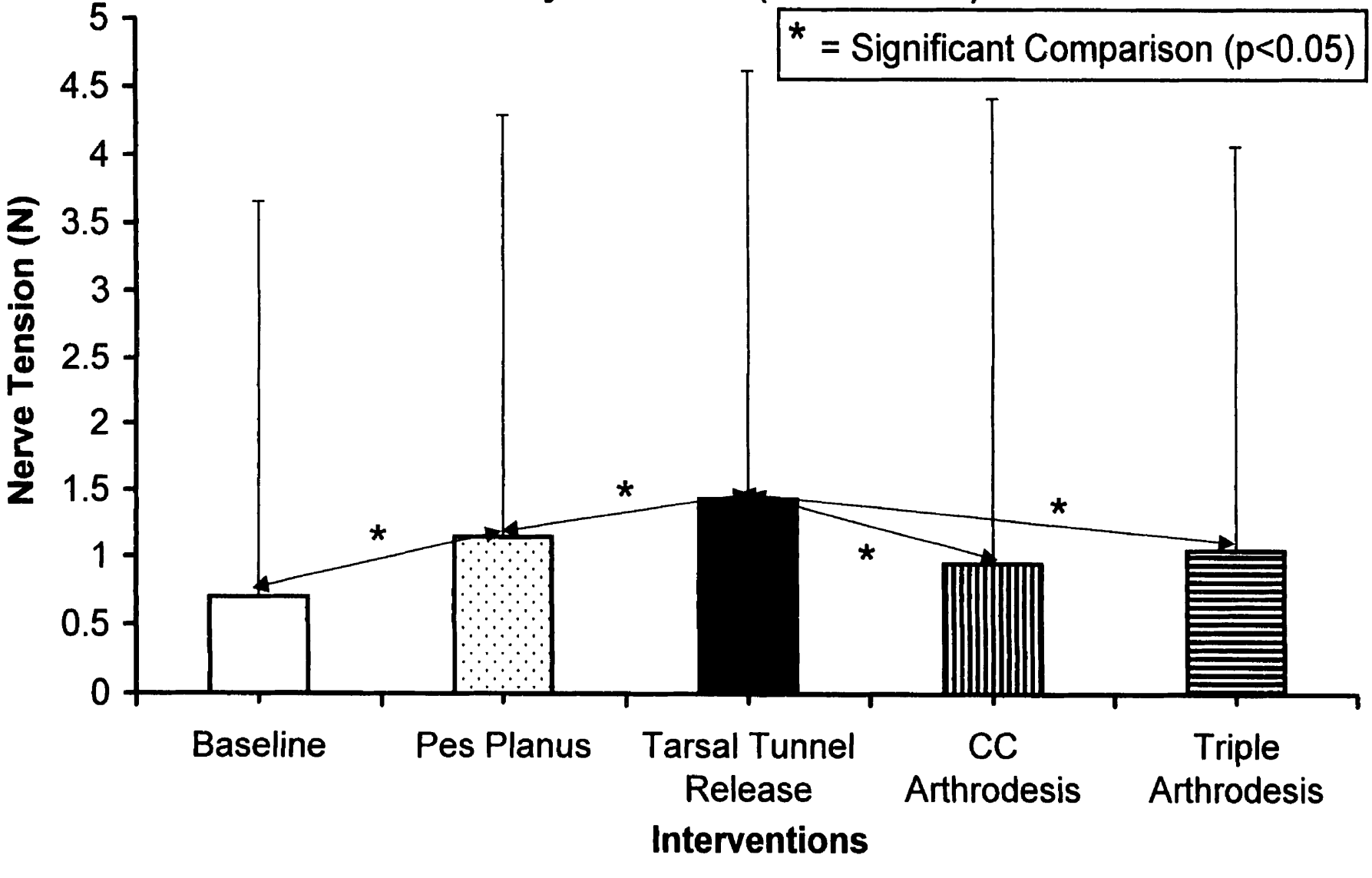
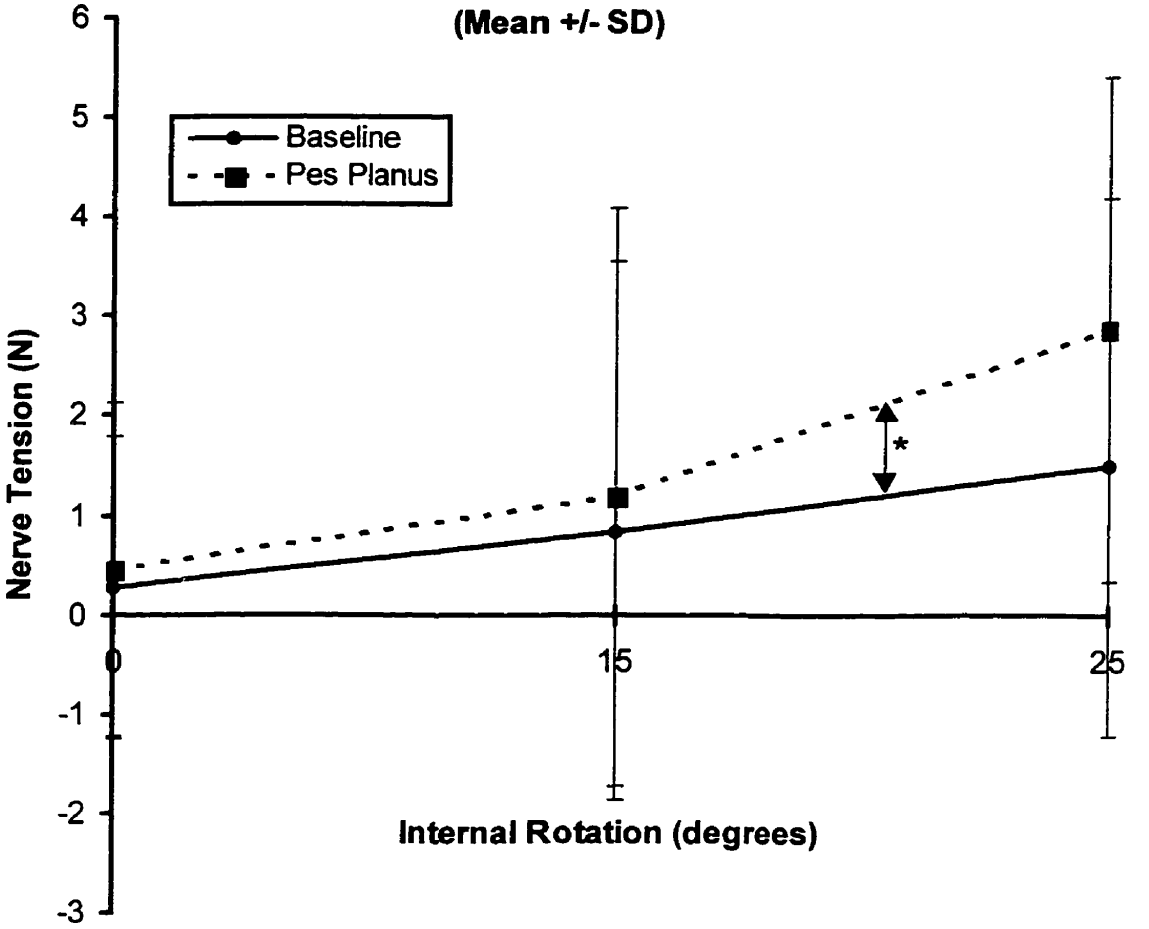


Figure 15: The Effect of Pes Planus Compared to Baseline on Tibial Nerve Tension during Cyclical Load (Mean +/- SD)

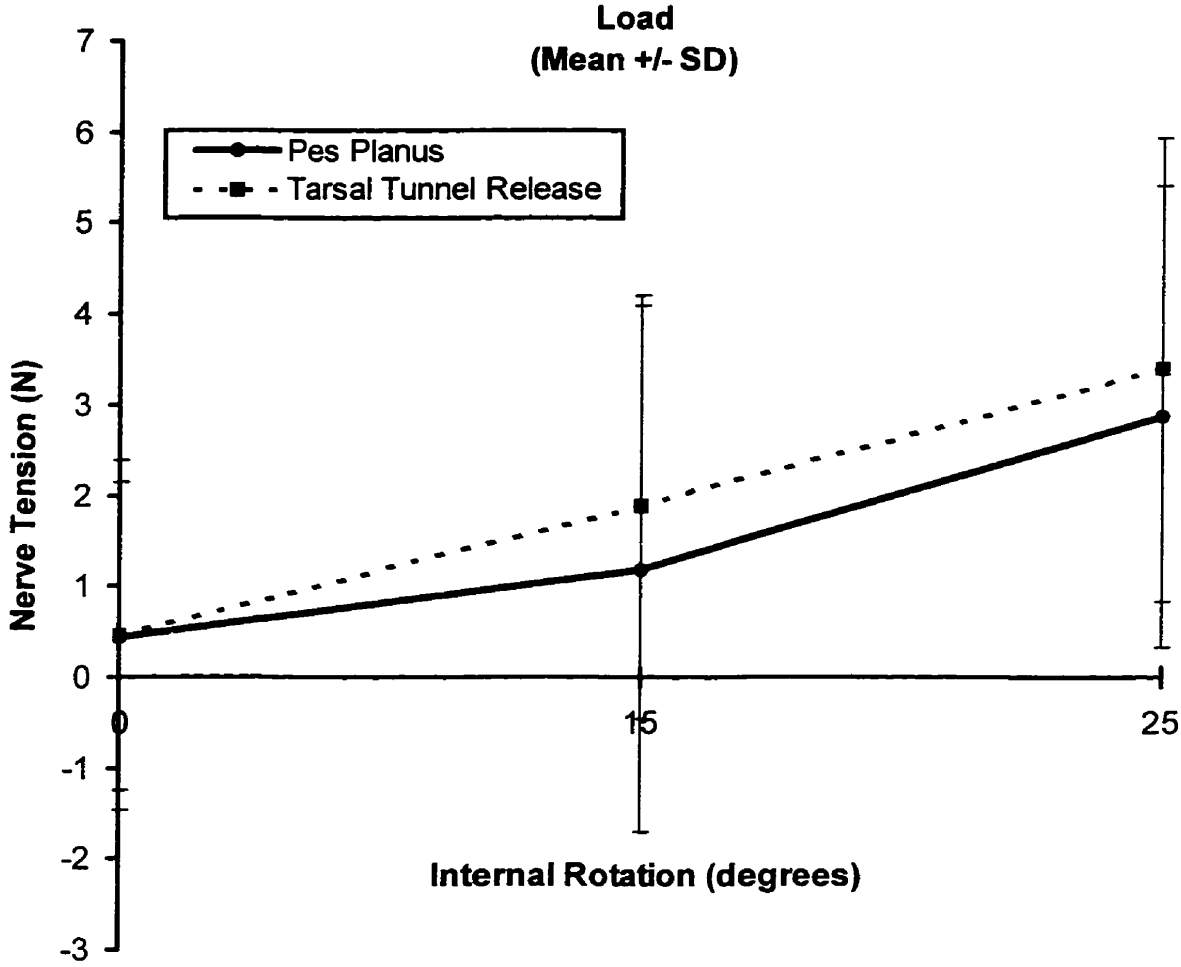


* = Pes planus foot compared to the baseline, and internal rotation from 15° to 25° significantly interacted to compound the increase in tibial nerve tension (p=0.0192).

Using a 2 factor repeated measures ANOVA,

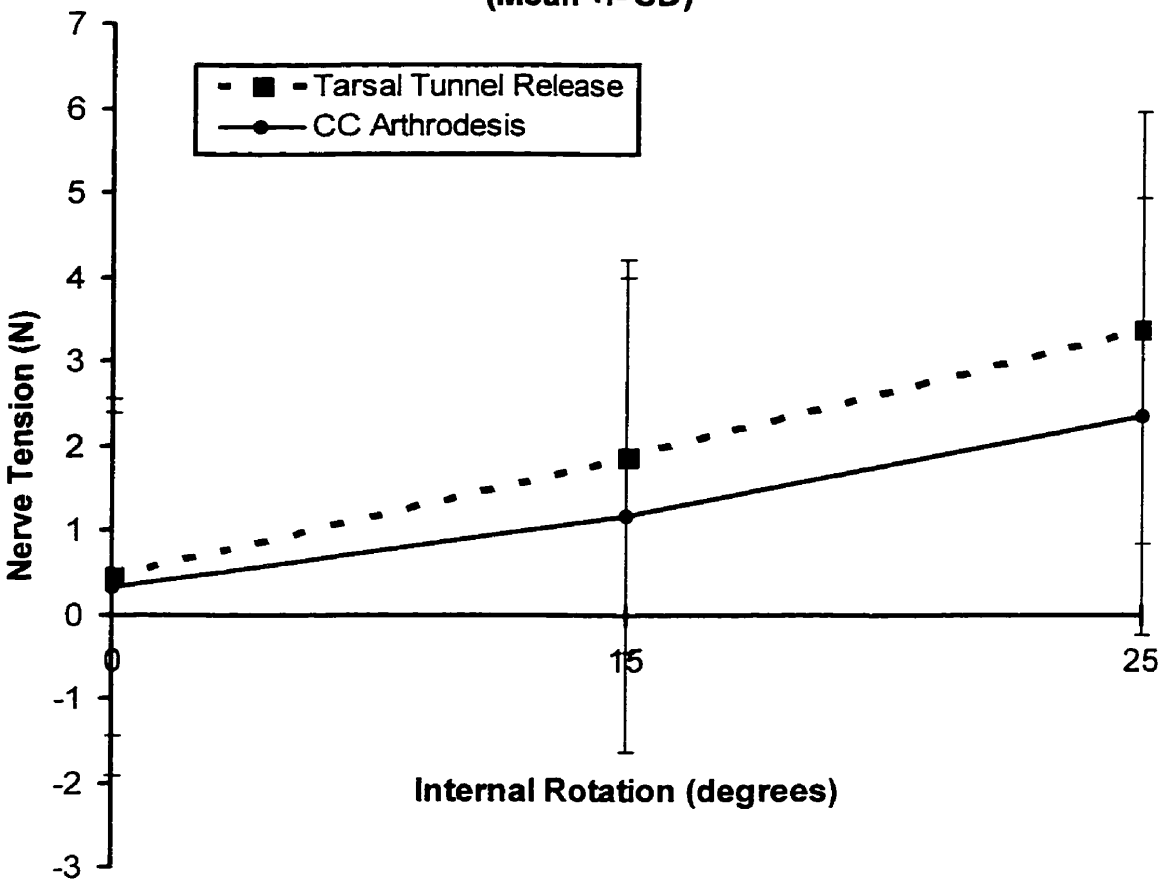
- Tibial nerve tension was significantly increased in the pes planus foot compared to baseline during increasing internal rotation (p=0.0011).
- Tibial nerve tension was significantly increased with increasing internal rotation (p=0.0001).
- No significant interaction between intervention and internal rotation (p=0.1915).

Figure 16: The Effect of Tarsal Tunnel Release Compared to Pes Planus Feet on Tibial Nerve Tension during Cyclical Load (Mean +/- SD)



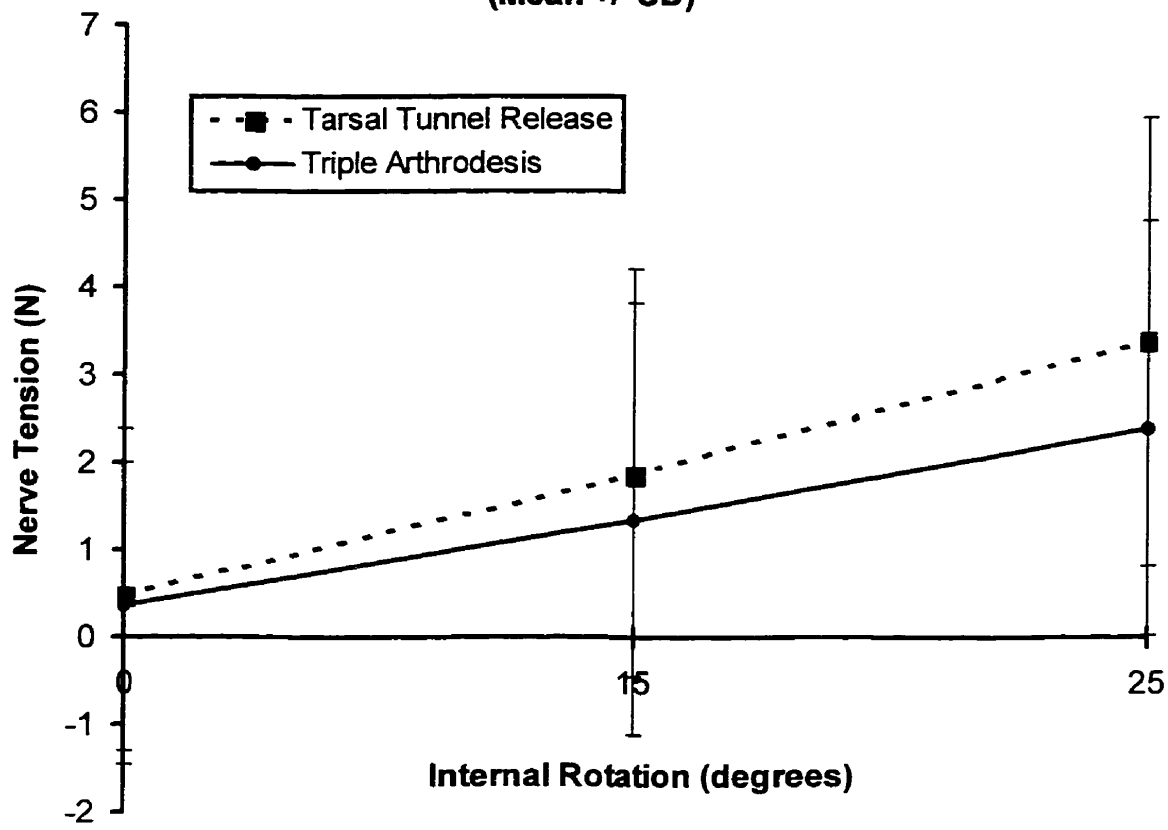
Using a paired t-test, tarsal tunnel release significantly increased tibial nerve tension compared to the pes planus foot during increasing internal rotation ($p=0.0295$).

Figure 17: The Effect of Calcaneocuboid Arthrodesis Compared to Tarsal Tunnel Released Feet on Tibial Nerve Tension during Cyclical Load (Mean +/- SD)



Using a paired t-test, distraction calcaneocuboid arthrodesis decreased tibial nerve tension compared to the tarsal tunnel released pes planus foot during increasing internal rotation ($p=0.0253$).

Figure 18: The Effect of Triple Arthrodesis Compared to Tarsal Tunnel Released Feet on Tibial Nerve Tension during Cyclical Load (Mean +/- SD)



Using a paired t-test, triple arthrodesis significantly decreased tibial nerve tension compared to the tarsal tunnel released pes planus foot during increasing internal rotation ($p=0.0267$).

Appendix

Appendix:

Three articles will be published from this thesis. One article (1) has been published, one article (2) has been accepted for publication, and one article (3) is submitted for publication.

In the first article published (1), which represents Part I of the thesis, Dr. Lau is the second author. He analyzed the data, and prepared the manuscript.

In the remaining two articles (2,3), Dr. Lau is the first author. In Part II (3), Dr. Lau participated in the design of the study, and performed all experimental work, data collection, data analysis, and preparation of the manuscript.

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