Fall 10-1999

Abnormal Electrical Potentials in Lower Limb Muscles After Ankle Sprain Grades I, II and III

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ABNORMAL ELECTRICAL POTENTIALS IN LOWER LIMB MUSCLES AFTER ANKLE SPRAIN GRADES I, II AND III

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B.Sc. June 1994, Kuwait University, Kuwait

A Thesis submitted to the
Faculty of The Graduate Program in Physical Therapy
In Partial Fulfillment of the Requirement for the Degree of Master of Science
Concentration in Physical Therapy

Old Dominion University
October, 1999

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Abstract

Fibrillation and positive sharp wave potentials have been found after ankle sprain in muscles around the injured joint. **The purpose** of this study was to better define and explain the reaction of the muscles around the ankle joint after grades I, II and III ankle sprains. **Methods:** Fifteen subjects (9 males and 6 females, mean age of 33.2 years) with ankle sprains voluntarily participated in this study. A stress x-ray was done to determine the grade of ankle sprain. Nerve conduction velocity and EMG studies were done three weeks after the injury. None of the patients in this study manifested any signs of sensory loss. **Results:** the subjects had normal latencys and conduction velocities for motor and sensory nerves of the involved ankle. Forty-seven percent of the subjects had fibrillation potentials in the abductor hallucis muscle and 40% had these potentials in the abductor digiti minimi muscle. Thirty-three percent of patients had positive sharp wave potentials in the abductor hallucis muscle, whereas 27% had the positive sharp waves in the abductor digiti minimi muscle. The results showed abnormal potentials in muscles supplied by both the peroneal and tibial nerves. There were no statistical differences among the three grades of ankle sprain groups. **Conclusions:** A greater than predicted percentage of subjects had fibrillation potentials and/or positive sharp waves in the small muscles of the foot following ankle sprain. Abnormalities in muscles innervated by two separate nerves indicates a cause other than direct nerve traction.

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CHAPTER I

Introduction.

Ankle sprains are one of the most frequently seen conditions in the emergency room. About one-fourth of all sports injuries involve the foot and ankle[1]. Physicians, physical therapists, and trainers usually treat these types of lower leg injuries. Injuries acquired in basketball and volleyball constitute the largest proportion of sprains of the ankle[1,2]. Of these injuries, inversion sprains (which primarily involves the lateral ligaments of the ankle) are the most common[1].

Injury of the deep peroneal nerve or posterior tibial nerve may or may not occur simultaneously with ankle sprains[3]. It is possible to have either direct or indirect nerve injury during an ankle sprain. Direct nerve injury occurs simultaneously with the sprain of the ankle. Indirect nerve injury may occur after the sprain as a result of swelling compression, hematoma, or nerve traction. Indirect nerve injury has been documented to occur in some ankle sprains at grades II and III[3].

Electromyography (EMG) is a method that can detect nerve damage by examining the intrinsic electrical activity output of muscles supplied by peripheral nerves. Following inversion ankle sprain, EMG can be used to examine the muscles supplied by the deep peroneal nerve and posterior tibial nerve. Symptoms such as numbness, decreased sensation, and weakness may indicate nerve damage. When a muscle is denervated, the EMG examination may reveal positive sharp waves and fibrillation potentials. The question raised here is whether the muscle fibers fibrillate because of lost innervation, or whether fibrillation is a reaction caused by other unknown influences as a result of the

injury. Denervated muscle can have fibrillation potentials as a result of a peripheral nerve lesion. However, numerous studies have reported EMG abnormalities similar to those seen with peripheral nerve lesions in patients with hemiplegia and spinal cord injury lesion[4,5,6,7,8]. In these cases, the peripheral nerves are intact, although the normal activity of the alpha motor neuron is disrupted.

Ankle anatomy

A sprain can be defined as a severe stretch or tear of soft tissue such as joint capsule, ligament, or tendon[9]. There are two types of ankle sprains: inversion sprains and eversion sprains. The most common ankle sprain is an inversion sprain. Simply stated, the mechanism of this injury can be explained by the movement of the tibia laterally, medially, backward, forward, or rotating while the foot is firmly fixed. This is a common injury in sports, especially where an athlete tries to quickly change direction. Stumbling off a curb, stepping in a hole, or losing balance are other potential causes for an ankle sprain.

An inversion sprain is a trauma or an injury to the lateral ligaments of the talocrural joint. The lateral ligaments of the talocrural joint consist of three main ligaments: the anterior talofibular ligament, the posterior talofibular ligament, and the calcaneofibular ligament. The anterior talofibular ligament can be sprained by a plantar flexion and inversion action of the foot. Excessive dorsiflexion and inversion can sprain the posterior talofibular ligament. In almost pure inversion, the calcaneofibular ligament is the ligament most likely to be sprained. Usually any talar tilt of more than 5 degrees inversion will result in ligament damage to the lateral ankle complex[9,10].

Forceful external tibial rotation, dorsiflexion or inversion can sprain the tibiofibular ligaments. This is due to the movement of the talus, which can push the tibia and the fibula apart, spraining the ligament[9].

Eversion sprains are not very common because of the support of the strong deltoid ligament medially and the bulwark created by the longer lateral malleolus[9]. Isolated injuries to the medial (deltoid) ligaments are rare and these injuries frequently involve malleolar fractures. The deltoid collateral ligament is a strong triangular band, attached to the apex and to the anterior and posterior borders of the medial malleolus[11,12,13]. This ligament can be divided into four main fibers; the superficial fibers (tibionavicular), the intermediate fibers (tibiocalcaneal), the posterior fibers (posterior tibiocalcaneal), and the deep fibers (anterior tibiotalar). Plantar flexion and eversion can sprain this strong ligament when an excessive force is applied to lateral side of the lower leg[11,12,13].

Classification of injuries

A physical examination, often including radiographs, is used to complete a diagnosis of an ankle sprain. Generally, ankle sprains are classified as first, second, or third degree. The classifications and symptoms are as follows:

Grade I sprain symptoms and injuries: mild injury, injuries stretch the ligament with microscopic, but not macroscopic tearing of fibers, pain, mild or no swelling, point tenderness, litle or no functional loss and no joint instability.

Grade II sprain symptoms and injuries: moderate injury, stretching of the ligament with macroscopic partial tearing, moderate to severe swelling, mild to moderate

hemorrhage, moderate functional loss and mild to moderate joint instability, pain, decreased motion and some loss of function.

Grade III sprain symptoms and injuries: severe injury, involves the complete rupture of the ligament with severe swelling and hemorrhage, an inability to bear weight and moderate to severe instability of the joint, pain, ligament separation, loss of function and the capsule is usually torn[3].

Nerve anatomy

The sciatic nerve is the broadest nerve in the body being about 2cm broad at its origin[13]. It branches from the sacral plexus. The nerve root, which form the sciatic nerve are; Lumbar 4 and 5, and Sacral 1, 2, and 3. This nerve runs along the posterior aspect of the thigh after leaving the pelvis through the greater sciatic foramen. It bifurcates into the tibial and common peroneal nerves just proximal to the knee[11,13].

The larger division of the sciatic nerve is the tibial nerve (figure 1). It arrives from the ventral branches of the 4^{th} and 5^{th} lumbar and 1^{st} to 3^{rd} sacral ventral rami. This nerve passes through the popliteal fossa lateral to the popliteal vessels. It is covered by the soleus and gastrocnemius proximally, and distally it passes between the medial malleolus and Achilles tendon where it is covered by skin and fasciae only. It terminates by passing under the flexor retinaculum where it divides into medial and lateral plantar nerves. The tibial nerve supplies the following muscles: gastrocnemius, plantaris, soleus, popliteus, tibialis posterior, flexor digitorum longus, and flexor hallucis longus.

The medial plantar nerve passes deep to the abductor hallucis then appears between it and the flexor digitorum brevis. It supplies the following muscles: abductor

hallucis, flexor hallucis brevis, and the first lumbricals. The lateral plantar nerve runs to the fifth metatarsal tubercle between the flexor digitorum brevis and accessorius. This nerve divides near the abductor digiti minimi into superficial and deep branches. The superficial branch supplies the flexor digitorum accessorius, abductor digiti minimi, flexor digiti minimi brevis, and the two interossei in the 4th intermetatarsal space. The deep branch supplies the following muscles: the second to 4th lumbricals, the adductor hallucis, and all interossei except the one which is supplied by the superficial branch[11,12,13].

The common peroneal nerve (figure 2), coming off the sciatic nerve, arises from the dorsal branches of the 4th and 5th lumbar and 1st and 2nd sacral ventral rami. It passes lateral to the fibular neck and deep to the peroneus longus and divides into superficial and deep peroneal nerves. The deep peroneal nerve (also called the anterior tibial nerve) descends to the ankle and divides there into lateral and medial terminal branches. This nerve provides muscular branches to the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius. The lateral terminal branch supplies the extensor digitorum brevis and the second dorsal interosseous muscle. The medial terminal branch supplies the first dorsal interosseous muscle[11,13].

The superficial peroneal nerve runs deep to the peroneus longus then pierces the deep fascia in the distal third of the leg to divide into medial and lateral branches. The medial branch passes anterior to the ankle and the lateral branch travels to the dorsum of the foot. The sensory portion of the superficial peroneal nerve supplies part of the skin on

the dorsum of the foot. The motor portion of the superficial peroneal nerve supplies the peroneus longus and brevis muscles.

Electromyography

Electromyography (EMG) is a tool used to aid in the diagnosis of various neuromuscular disorders. It is the study of the intrinsic electrical activity of muscles [14]. EMG is the most common procedure for screening patients with neuropathies and myopathies and remains the most important technique for assessing the course of the disease over time. During diagnostic EMG, the electrical activity of a muscle is detected using needle electrodes, the resulting waveform is displayed on a computer screen or oscilloscope. Various diseases, degeneration and/or regeneration of nerves result in characteristic waveform that indicates the status of the perepheral nerves. Abnormal findings such as fibrillation potentials, positive sharp waves, myotonic or complex repetitive discharges, as well as polyphasic potentials are non-specific and may occur in both myopathic and neurogenic lesions. After an acute peripheral nerve injury, fibrillations and positive sharp waves can be recorded in the denervated muscle.

Fibrillation potentials are spontaneous contractions of individual muscle fibers[15,16,17]. Fasciculation potentials are a spontaneous twitching of a group of muscle fibers or a motor unit[15,16,17]. Positive sharp waves are biphasic, positive-negative action potentials initiated by needle movement and recurring in a uniform manner, which may be found in association with fibrillation potentials[15,17].

Nitz et al found that 17% of the subjects with grade II ankle sprains had mild to moderately severe denervation in the peroneal nerve distribution and 10% in the tibial

nerve. They also found that 86% of subjects with grade III sprains sustained a peroneal nerve injury and 83% sustained tibial nerve injury resulting in moderate to severe denervation. They stated that possible causes of this nerve injury include compartment syndrome, epineural hematoma, or nerve traction[3].

Statement of purpose

The purpose of this study is to better define and explain the electrophysiological changes in the muscles around the ankle joint after grade I, II, and III ankle sprains. Theories such as nerve stretching or epineural hematoma explain how nerves get injured after ankle sprains due to the stretching of the nerve. This nerve stretching can cause damage only to the pulled nerve, but when two nerves are involved, the traction theory might not be enough to explain the abnormal potentials in muscles supplied by two nerves. Adding to that, nerves move between 5 and 8 mm during ankle inversion[32], which probably is not enough to cause nerve injury.

Literature review

An ankle sprain is a commonly experienced injury. Sprains are a tearing of the ligaments that connect bones and typically involve the outer ligaments that support your ankle. Most sprains occur when the foot plantar flexes and inverts. In sports, a sudden weight change or shift can cause a sprain. Factors that can make you more prone to an ankle sprain include: collisions or falls during contact sports, loss of balance on uneven surfaces, calf and leg muscle imbalances, tight Achilles tendons, improper footgear, and fatigue[1,2].

Luciano and Davis examined the mechanism of lateral ankle sprain in 39 amputation specimens[18]. All specimens had a normal range of motion of the ankle joint.

The skin, all soft tissue, and muscles were removed. They left the ankle joint and the tibial fibular syndesmosis with all ligaments and capsule intact. The foot was attached to a special apparatus, which was made to move the foot in different directions. The foot was tested in three different positions: supination with inversion, supination with internal rotation, and supination with plantar flexion. They found that as the degree of inversion increased the following injuries to the ligaments could be detected. First, partial tears of the fibulocalcaneal ligament, second, rupture to the anterior fibulotalar ligament, then, complete tear of the fibulocalcaneal ligament and posterior fibulotalar ligament, and finally, a partial tear of the deltoid ligament.

Many studies have reported peroneal nerve palsy following inversion ankle injury[3,19,20,21,22,23]. Stoff and Greene reported two cases[23]. They were both males, a twenty-eight and a forty-two year old, who had each sustained an inversion injury to the ankle joint. They were found to have swelling, pain, and loss of motor power in the muscles of the foot and ankle, and a diminished sensation to pin pricks. Electromyographic examination with monopolar needles electrode revealed fibrillation potentials and positive sharp waves in the tibialis anterior, extensor digitorum longus, and extensor digitorum brevis muscles. Those three muscles are supplied by the deep peroneal nerve. Complete recovery of function occurred three months after the injury in these two cases.

Connolly, Fitzgibbons, and Weber reported a case of a 20 year old female who sustained an inversion injury to the left ankle[20]. She complained of numbness on the anterolateral aspect of the distal portion of the leg extending onto the dorsal medial aspect

of the foot. EMG and nerve conduction studies were performed and abnormal potentials of fibrillation and positive sharp waves were found. All evidence in this case showed a common peroneal nerve injury after the ankle sprain. After four months the patient showed complete return of peroneal nerve function with full range of motion in the ankle and no residual numbness.

In another case a thirty-two year old female sustained an inversion injury to her left ankle while stepping off a curb. She complained of pain over the lateral ankle and the dorsolateral aspect of the foot immediately after the injury. Swelling was noted in the region of the anterior talofibular ligament, but there was no ligamentous instability of the ankle. X-ray showed no evidence of fracture. Motor and sensory function was intact. Her treatment was an elastic bandage and the use of crutches non-weight bearing for 2 weeks. One month after her injury, the patient noted to have a positive percussion sign over the intermediate dorsal cutaneous branch of the superficial peroneal nerve. The patient continued to have this neuritic type of pain at the dorsolateral aspect of the foot for over 2 years. This pain was aggravated even by light pressure such as a sock. The authors of this case report, Acus and Flanagan, noted that this pain was caused by a 2 cm long region of severe perineural fibrosis of the intermediate dorsal cutaneous branch of the superficial peroneal nerve, which was identified just anterior to the lateral malleolus at the level of the ankle joint[19]. A neurolysis was performed with transposition of the nerve into subcutaneous fat. Complete relief of the neuritic pain was noted postoperatively. The authors suggested that traction injuries to the superficial peroneal nerve and perineural

fibrosis should be considered in the differential diagnosis of patients with persistent pain after ankle sprains.

Kleinrensink et al conducted a study to determine the motor conduction velocity of the peroneal nerve after ankle sprains[22]. This study focused on ankle sprain injuries, which had no clinical signs of peroneal nerve palsy. The authors examined the motor nerve conduction velocity of the superficial and deep peroneal nerves. Twenty-two patients, eighteen men and four women, between seventeen to forty-five years of age participated in the study. Patients were selected after inversion injury from the Department of Traumatology of the University Hospital Rotterdam, Dijkzigt. This group sample included seven patients with grade I ankle sprains, three grade II sprains, and twelve-grade III sprains. The authors used the anterior drawer test to grade the ankle sprains. All patients with fractures, neuropathy, and diseases of the central nervous system were excluded from the study. Normally we compare the injured ankle with the contralateral ankle (non-injured ankle) to look for any abnormalities. The authors stated this might be misleading because a unilateral injury to the ankle joint could lead to functional alterations at both the ipsilateral and the contralateral joints. For this reason they compared the injured ankle with the contralateral joint and a healthy volunteers who never complained of ankle injury.

The healthy group consisted of twenty-eight subjects, ten men and eighteen women, between nineteen to thirty-seven years of age. Three recordings were taken from all patients at three different times: 4-8 days, 18-22 days, and 32-36 days post-trauma to record the conduction velocity of the peroneal nerve. The peroneal nerve was stimulated

at the knee, the fibular head, and the ankle. A square wave pulse of 0.3 ms duration and a voltage up to 300 mV was used to stimulate the nerve. After testing the superficial peroneal nerve, the authors found a reduction in the motor nerve conduction velocity when compared to the contralateral leg and the control group in all three grades of ankle sprains. When the deep peroneal nerve was tested, it showed no differences in motor nerve conduction velocity when compared to the contralateral leg, but when it was compared to the healthy group, all values of the injured leg were slower than values of the control group. The authors suggested that this damage to the peroneal nerve was due to traction or stretching of the nerve beyond a certain percentage, which lead to acute insufficiency in intraneural circulation and thus to alteration in the conduction velocity.

Theories

Several authors have offered explanations of peroneal nerve injury after ankle sprains (figure 3). In 1911 Oppenheim suggested that nerve injury symptoms were due to traction or stretching of the peroneal nerve at the fibular head or neck, secondary to inversion of the foot[24]. This traction would apply pressure on the nerve between the tendon of the biceps muscle and the head of the fibula. The author stated that this forceful sudden inversion would affect the superficial peroneal nerve more than the deep peroneal nerve.

In 1941 Hyslop noted that the deep peroneal nerve passes laterally and distally around the neck of the fibula and pierces the peroneus longus as it runs through this muscle[21]. Then the nerve penetrates the extensor digitorum longus and extensor hallucis longus muscles. The superficial peroneal nerve passes underneath the peroneus longus

muscle. She concluded that when a sudden tightness of the peroneus longus at the fibular head occurs, the peroneal nerve become compressed and causes paralysis. This theory became known as Hyslop's compression theory.

Hyslop reported three cases to support her theory. The first case was of a 26 year old female who slipped on a polished floor and turned her left ankle with the foot inverted. After a couple of weeks the patient could not lift her toes from the floor. The patient showed about 50% loss of power in the tibialis anterior muscle and a slight weakness in the peroneus longus and brevis muscles. There also was a decreased sensibility to light touch and pain on the dorsum of the foot and the lower anterior and lateral aspect of the leg. The second case was of a 55 year old female who lost her balance while walking in the street and sprained her left ankle. This patient complained of numbness on the dorsum of her foot, and she was unable to extend her toes. Two weeks post injury the patient showed weakness and wasting of the tibialis anterior and toes extensors. The third case was of a 49 year old male who caught his toe on a door-sill and lost his balance. His foot was inverted with a forced planter flexion that sprained the left ankle. The patient complained of numbness on the dorsum of his left foot. Upon examination, the patient showed a weakness of the tibialis anterior and a slight weakness in the toes extensors. The author suggested that a sudden forced movement to the ankle would lead to tensing the muscle mass lateral and posterior to the neck of the fibula. This tensing would pull and compress the deep and superficial portion of the peroneal nerve, which in turn traumatized the nerve trunk[21].

In 1966, Noble explained a peroneal nerve injury as an excursion of the common peroneal nerve which could be caused by a distal traction of the superficial peroneal nerve[25]. After ankle sprain the distal part of the nerve (superficial peroneal nerve) will become stretched and cause further pulling to the proximal part of the common peroneal nerve. As Noble explained, this stretching will cause an injury to that stretched nerve. He noted that sometimes severe traction on the nerve would cause rupture to the nutrient vessels, which supply it. This rupture would lead to a gradually expanding hematoma within the nerve sheath, which explains the delayed onset of the peroneal nerve paralysis. Most cases showed neurological symptoms several weeks after the injury. Those delayed symptoms were stated to be due to the time that the bleeding accumulation in the nerve sheath took to cause sufficient compression.

Nobel examined a case with a spiral tibial fracture in the lower third of the leg and another case with a spiral fibular fracture in the proximal third of the leg. The injury was due to a forced twisting of the ankle while skiing. During the first week the pain started increasing in the ankle and lower leg. Later the patient complained of numbness of the third and fourth toes and the dorsolateral aspect of the ankle. By the tenth day the patient complained of a severe pain that couldn't be tolerated any more. Under anesthesia, a nine-inch incision was made along the posteromedial edge of the biceps femoris tendon, over the fibular neck, then extended distally along the anterolateral aspect of the leg. When the peroneal nerve sheath was exposed, there was a bluish swollen area from the sciatic bifurcation to below the knee. A six-inch long hematoma was found in the nerve sheath encircling the perineurium and compressing the nerve. This was due to rupture of the

blood vessels (vas nervorum). Upon opening the sheath, the vessel was still oozing. The vessel was repaired and the hematoma was evacuated. After the operation, the neuritic pain was completely gone. On the seventh postoperative day, the patient recovered completely with normal sensation and no pain. The author said that the vas nervorum is a very tiny and fragile vessel, about 2 millimeters in diameter, that enters the epineurium to supply a much tougher structure than the vessel itself. This structure is the peroneal nerve, which is about 8 millimeters in diameter. During the nerve movement, back and forth, this mechanical pulling puts the vas nervorum vessel under extreme stress. The vessel enters the epineurium, which is a relatively fixed tissue, and then penetrates the perineurium, which is a more freely gliding tissue. At this point, the rupture of the vessel occurs.

Nobel tested twelve cadavers to study the mechanism of nerve injury in situ. Two small limited incisions were done: one at the sciatic bifurcation and the other at the terminal cutaneous branch of the superficial peroneal nerve near the ankle joint. Using a clamp, the superficial peroneal nerve was pulled distally. This pulling caused between ten to twenty-five millimeters of excursion of the common peroneal nerve. Further traction of the nerve caused it to rip at its exit from the fascia near the ankle before any motion of the nerve could be seen at the bifurcation. The author concluded that an inversion ankle sprain will not apply enough load and stress to pull the nerve to a degree that tears the blood vessel. The author further noted that in order to have this great a pull on the nerve, you would need a skier whose average speed is thirty miles per hour and a stopping force on the legs as great as 6600 foot pounds. This amount of torsion force might be enough to

exert traction on the terminal branches of the deep and superficial peroneal nerve, which can be transmitted along the entire length of the nerve to tear the nutrient vessel of the common peroneal nerve at the sciatic bifurcation[25].

In 1967, Meals added to the traction theory that compression would also injure the peroneal nerve because it passes underneath a narrow arcade of fibrous tissue at the fibular head where it can easily be damaged[26]. He stated that as the nerve is pulled distally and the arcade is tightened, traction or compression, or both could cause injury to the nerve after an ankle sprain. The author reported two cases, a 24 year old male and a 23 year old male, who both sustained an inversion injury in their right ankle while running and walking on irregular ground. Both cases showed muscles weakness in the lateral and anterior compartments of the leg (case 1, one week after the injury / case 2, four weeks after the injury). After examination, the peroneal nerve conduction velocities and latency times were normal. The electromyogram showed irritability and fibrillation potentials in the right tibialis anterior muscle. The author noted that the cause for nerve injuries vary from one case to another. The mechanism of injury depended on the location, size, and the rate of formation of the hematoma. Some patients showed symptoms right after injury and others had delayed symptoms. The author concluded that both theories could be true. The traction theory can explain the injury to the nerve and gives symptoms right after the injury whereas the compression theory needs some time for the hematoma to accumulate and cause compression to the nerve.

Tibial nerve injury associated with sprained ankle is less frequent than peroneal nerve injury. Nitz, Dobner, and Kersey studied 66 patients with grade II (30) and grade III

(36) sprained ankles[3]. They excluded 45 patients with grade I sprain. The reason they did not study patients with grade I is that EMG studies on the first 12 patients revealed no evidence of denervation potentials in the sprained leg muscles. All patients were tested on the 14th day post injury, and the examination included a nerve conduction study of the peroneal and the tibial nerves. The following muscles were tested: the gluteus maximus, medius, tensor fascia latae, iliopsoas, medial and lateral hamstrings, adductor magnus and longus, vastus medialis, tibialis anterior and posterior, medial head of the gastrocnemius, extensor hallucis longus, and the peroneus longus.

Nitz et al found that all nerve conduction studies were normal for both the peroneal and the tibial nerves[3]. Seventeen percent (5 out of 30 patients) showed denervation potentials (positive sharp waves and fibrillation) in the muscles supplied by peroneal nerve in grade II sprains, and ten percent (3 patients) showed denervation potential in the muscles supplied by the tibial nerve. In grade III sprains, eighty-six percent (31 patients) showed denervation potentials in the muscles supplied by the peroneal nerve, where eighty-three percent (30 out of 36 patients) showed denervation potential in the muscles supplied by the tibial nerve. The authors hypothesized that the reason for the abnormal potentials in both nerves were due to a combination of nerve traction and epineural hematoma at the level of the bifurcation of the sciatic nerve into the common peroneal nerve and the tibial nerve. The authors stated that this would explain why some patients with ankle sprains complain of tenderness in the popliteal fossa. The authors noted that the nerve moves between 5 to 8 mm during ankle inversion

which is enough to cause a small degree of traction on the nerve in both the peroneal and the posterior tibial nerves.

Nitz et al did not study the intrinsic muscles of the feet. The authors claimed that normally the intrinsic muscles of the feet have a high percentage of EMG abnormalities. Gatens and Saeed, however, published a study on EMG findings in the intrinsic muscles of normal feet[27]. They studied seventy (51 men and 19 women) normal individual's feet with ages ranging from 19 to 67 years. One foot was studied in each subject. The muscles examined were the abductor hallucis, the first dorsal interosseous, the extensor digitorum brevis, and the abductor digiti minimi. Fibrillation potentials were rarely seen in examined muscles. The only muscle showing significant numbers of fibrillation potentials (5.7%) was the abductor digiti minimi. In twenty-seven out of the seventy individuals, one of the four muscles examined showed abnormal potentials. The only muscle that was high in positive sharp wave potentials (15.7%) was the extensor digitorum brevis (11 out of 70 subjects). The authors suggested that possibly denervation and reinnervation are regular phenomena in the extensor digitorum brevis of many people as a result of episodic trauma.

In another study, Wiechers noted that minor trauma can affect the feet particularly in active individuals or in women who wear tight fitting or high heeled shoes[28]. Wiechers stated that this type of trauma from activity or poor shoes might explain these abnormal potentials in extensor digitorum brevis.

The sensory feedback system

The sensory system can be affected by ankle sprain too. The sensory system provides information from the muscles, ligaments and joints to the central nervous system. Mechanoreceptors located in the joint capsule and the ligaments relay information via afferent fibers to the spinal cord. These receptors relay information that allows for reflex stabilization of the joint, adjustment of joint position and movement, muscle activation, and control of the gamma muscle spindle system to influence the stability of the joint. Mechanoreceptors are located within joint structures and can be damaged during ankle sprain. Any damage to them will affect the information going to the spinal cord, which will alter its ability to control movement and position.

Zimny studied the morphology, distribution, and function of the mechanoreceptors in joint capsule, ligaments, knee joint menisci, and articular disc[29]. The author investigated three types of receptors: Ruffini-like receptors, Golgi tendon organ receptors, and encapsulated Pacinian-like corpuscle receptors. Tissues from the knee joint were examined to locate the free nerve endings and mechanoreceptors in the joint structures. The nerve fibers enter from the synovial connective tissue and terminate in receptors located in the joint structures. The author found that most of the receptors were located in the distal portion of the ligaments. In the meniscus, nerves penetrate the outer and middle one-third of the body and the horns from the perimeniscal tissue. The greater concentrations of the receptors are in the horns of the meniscus. The concentration of those receptors appears greater in areas related to the extremes of movement in order to act as the first line of defense in sensing these extremes. Usually the joint

mechanoreceptors are present within the joint intra-articular structure. In the temporomandibular articular disk, the mechanoreceptors are concentrated at the periphery and decreased in the center. Those receptors fire afferent discharges to alert the central nervous system of impending injury that can be avoided by reflex mechanisms. Those are two examples of two different joints in the body that have receptors in the joint structure. Zimny noted that the ankle joint structure is similar to other joints in the body in that it includes these types of receptors[29].

In 1995, Bullock-Saxton studied the effect of ankle sprains on the sensory perception system[30]. Two groups participated in this study. Group one was the injured group (20 men aged between 18 and 35 years of age) who sustained a unilateral ankle sprain (grade II and III). Group one was assessed no sooner than 2 months and no later than 18 months post injury to allow pain and swelling to heal. Group two was the control group. Eleven men participated in group two, and they were matched for age, height, and weight to group one. Group two had no history of musculoskeletal injury to the lower limbs, and no neurological disorder. The subjects were tested for vibration perception, two-point discrimination and balance in one-legged standing. The vibration perception was measured by a mechanical oscillator, which was connected to a power oscillator to increase the voltage and amplitude of the head progressively. The authors used a range of frequencies between 50 to 450 Hz intervals to assess vibration sense. This was applied to the inferior fibular head of both lower limbs. Two-point discrimination was measured using a set of dial calipers accurate to 0.1 mm. The skin was marked at a point 60 mm from the lateral malleolus on a line running along the fibula. The subject had to tell the

assessor if one or two points could be felt with eyes closed. Balance was tested by asking the subjects to cross arms and to raise one leg with 90 degrees flexion of the hip. The time in seconds was recorded for each subject while standing balanced on one leg. All results were compared with subjects in group two. Results for vibration perception and two point discrimination showed that group one had a significant deficit in sensory perception on the injured side. Impaired balance could be seen by asking the subjects to stand on the injured side. The authors stated this could be explained by loss of the integrity of the sensory motor system. The author confirmed the assumption that ligamentous and capsular injury are associated with significant deficits in local sensory receptor function, which could be associated with further muscle function changes.

Justification

Most studies of abnormal potentials in the foot and ankle post ankle sprain have suggested that a denervation injury is the cause of the abnormalities[3,19,20,21,23,24,25]. Researchers have hypothesized that nerve injury occurs as a result of a mechanical pulling or compression directly caused by the sprain. However, when more than one nerve is involved in the injury, this will lead us to think about another explanation because it is unlikely that nerves in two different locations are simultaneously pulled by the same mechanism of injury. Inversion sprains should cause pulling on the peroneal nerve but not on the tibial nerve, which passes medially to the talocrural joint.

Nerves are made of viscoelastic tissues, which allow the nerves to stretch to a certain limit before reaching the failure point where damage occurs. When the nerve is stretched beyond this elastic limit, a mechanical failure occurs. Beyond this point the

nerve elongates quickly until complete separation of the nerve occurs. The epineurium, the perineurium, and loose connective tissues surround and fill the spaces within the nerve fascicles. These structures enable the nerves to elongate and shorten, adapting to different positions. The perineurium is the tube which envelops all structures in the endoneurial space. This tube adapts to lengthening and shortening by changing its diameter. The endoneurial components have a wavy shape structure that can increase the number of undulations shortening or straighten this wavy pattern while elongating. The perineurial tube diameter increases while shortening and decreases while elongating[31].

An important question in studying nerve injury is how far the nerves can be elongated before reaching their elastic limit. Liu et al studied 22 human nerves, 3 sciatic, 4 ulnar, 7 tibial, and 8 peroneal nerves to determine the tensile strength of human nerves[32]. All nerves were taken six to twelve hours after death from patients who died from diseases other than neurological conditions. The authors used a tensiometer to study the stress strain limit for each nerve. The results showed that a 10% elongation of the nerve could cause a complete tear of all nerve fibers, where as a 6% elongation may injure one nerve fascicle with preservation of the other bundles. The range of over-stretching limits in all tested nerves was between 3.2% to 5.2%. Damage to the peroneal nerve occurred with stretching over 4.1% of its total length.

Inversion injury of the ankle joint applies inversion stress on the peroneal nerve. This stress can stretch the nerve and elongate it for about 5 to 8 mm during an ankle sprain[3]. If the peroneal nerve of an average sized adult was measured from the sciatic bifurcation to the dorsal part of the foot, its length would roughly be about 50 cm. When

4.1 percent of its total length is calculated, it equals about 2 cm. This means that the peroneal nerve has to be stretched about 2 cm (20 mm) in order to damage the nerve. The total length of the sciatic nerve and the peroneal nerve if measured from the lumbosacral region to the dorsum of the foot, is about 112.5 cm. Four point one percent of this total nerve length is about 4.6 cm (46 mm). Nerves have an elastic mechanical property to allow them to elongate and shorten, adapting with the joint angles and body movements. Based upon evidence from the above research, five to eight millimeters of peroneal nerve stretching is not enough to cause damage to this nerve. The peroneal nerve needs to be stretched more than 20 mm in order for damage to occur.

When we look at studies conducted during the late Sixties and the early Seventies, we find another theory which might explain the abnormal potentials after ankle sprains. Spielholz et al examined thirty-two patients with spinal cord injuries[4]. Electromyography was performed on the anterior tibialis, soleus, peroneus longus, quadriceps, and gastrocnemius muscles. Two patients showed no abnormalities. Fifteen patients showed abnormal potentials in all the muscles tested. Those 15 patients showed abnormal nerve conduction readings, which indicated essentially complete degeneration of peripheral nerves, which might be due to cauda equina damage. The last 15 patients demonstrated fibrillation potential and positive sharp waves in all tested lower extremity muscles. The authors suggested the following theory to explain their findings:

> Anterior horn cells exert more effects on their associated muscle fibers than simply signaling them when to contract. More and more, one reads of the "trophic effects" of nerve on muscle. These effects are presumed to be mediated by factors, possibly protein in nature, synthesized in anterior horn cells and transported down the axon to the associated muscle fibers. Perhaps one or more of

> these trophic factors exert an influence, which prevents fibrillation in normally innervated muscle fibers. We are not claiming that prevention of fibrillation is the physiological role of this factor-we are merely stating that in its absence, muscle fibrillates. For this factor, or combination of factors, we propose the tentative name of antifibrillation factor (AFF)[4].

It is possible that upper motor neurons exert trophic effects on anterior horn cells as anterior horn cells exert trophic effects on muscles.

Although the identity of the trophic factors are not known, this theory might explain the function of these trophic factors. Eliminating the target muscle eliminates the factors, and an increase in the size of the target increases the size of the supply. Anything that decreases neuromuscular transmission also decreases the supply of trophic factors to the spinal motor neuron. This leads to a greater degree of motor neuron death[16]. In this way, the target muscle has a critical role in the survival of the spinal motor neurons. The reason that some neurons die is because nerve terminals compete with each other for a limited amount of an essential nutrient or trophic factors provided by the target muscle[16]. A healthy target muscle may be necessary for spinal motor neuron survival.

After ankle injury the muscles around the joint will be affected. This could lead to a decrease in the trophic factors coming from the target muscles, which could in turn lead to death of the motor neuron in the spinal cord. The decrease in the trophic factors and neuron death will affect the function and the control of the spinal motor neuron. The spinal motor neuron will not be able to control the electrical potentials, which will lead to the firing of abnormal potentials. The whole mechanism works as a loop. The muscles and articular structures send feedback to the spinal cord which keeps the trophic factors active to supply the neurons in the spinal cord in order to control the activities in the

muscles. Higher levels, such as the brain, have some control on the trophic factors too. This is how Spielholz's findings may explain the abnormal potentials in skeletal muscles in patients with spinal cord injuries (figure 4).

Perhaps researchers should think of factors other than mechanical stretching affecting the peripheral nerves following ankle sprains. All aspects and all the possible theories should be focuses of investigation of this problem. The mechanoreceptors located in the joints send information to the spinal cord and to the central nervous system. These sensory signals might be altered or diminished after an ankle sprain. The motor spinal neurons will not receive those feedback signals, which could inhibit the antifibrillation factors (AFF). As a result, the AFF will no longer exert any control on the muscle fibers. This is one theory that explains the abnormal fibrillation potentials in muscles around an injured joint. This study may open a new line of investigation with the potential to answer unanswered questions.

Hypothesis

Alternative Hypothesis (H_1):

Subjects will have a greater than normal incidence of abnormal EMG potentials (positive sharp waves and fibrillation potentials) in the muscles supporting the ankle (tibialis anterior, peroneus longus, abductor hallucis, abductor digiti minimi, and gastrocnemius) three weeks following grades I, II, and III ankle sprain.

Null hypothesis (H_0):

Subjects will have no incident of abnormal EMG potentials (positive sharp waves and fibrillation potentials) in the muscles supporting the ankle (tibialis anterior, peroneus longus, abductor hallucis, abductor digiti minimi, and gastrocnemius) three weeks following grades I, II, and III ankle sprain.

CHAPTER II

Methods.

Subjects

Fifteen patients (9 males and 6 females) with ankle sprains, grade I, II, or III, voluntarily participated in this study. These patients were selected from Al-Razi Hospital, which is the longest orthopedic center in Kuwait. Their ages ranged from 19 to 53 years old. All patients were free of diabetes, neurological and cardiovascular diseases. None of the patients had a history of lumbar spine, hip, thigh, or knee injury, nor did any have a fracture during the past two years. There were five patients in each ankle sprain grade group. All patients agreed to participate in the study by signing the consent form (Appendix A). All information regarding the patients was treated with strict confidentiality.

Instrumentation and Procedure

The patients were selected from the emergency room in Al-Razi Hospital as acute cases. All patients were sent for x-rays to make sure that there were no fractures. Because of pain and swelling, patients with grade II and III sprains were casted with placed in Plaster Of Paris (POP) for a duration of two weeks. Patients were advised to rest, while elevating their feet above the level of their hearts. Analgesic drugs were prescribed as needed to decrease pain in patients who had severe pain.

Subjects were referred to an orthopedic surgeon two weeks after the injury. The POP was removed, and anterior drawer stress x-ray and varus tilt stress x-rays were taken for both left and right ankles for comparison. The orthopedic surgeon performed a manual instability test to determine the ankle sprain grade. He combined the results of the manual

test with the x-rays to confirm the grading. After determining the ankle sprain grade, the orthopedist advised the patients to rest for another week and to begin gentle ankle exercises to maintain joint mobility. All injured ankles were bandaged to minimize joint mobility and help in stabilization, in order to protect them from further damage.

A consultant clinical neurophysiologist performed the tests on the subjects one week after removing the POP. All patients were referred to the Department of Clinical Neurophysiology in Ibn Sina Hospital to perform Nerve Conduction Velocity and EMG tests. A Sapphire II[§] 2M (2 channel) was used to perform the nerve conduction velocity and EMG tests.

Nerve Conduction Velocity Test

All tests were performed in a room with temperature of 24°C (75.2°F). Subjects were given 30 minutes to rest and adapt to the room's temperature and environment.

The peroneal nerve was stimulated at three different locations: the posterior knee, the fibular head, and the anterior ankle. The reference electrode (positive) was placed on the fifth toe's extensor tendon. The ground electrode was placed on the lateral malleolus, and the recording electrode (negative) was placed on the belly of extensor digitorum brevis muscle[14].

The tibial nerve was stimulated at two locations: the mid popliteal fossa of the posterior knee and posterior to the medial malleolus of ankle. The reference electrode (positive) was placed at the first metatarsal head. The ground electrode was placed on the

[§] TECA Sapphire II, Oxford Instruments Medical System Division. Pleasantville, New York 10570, U.S.A. 1995.

lateral malleolus, and the recording electrode (negative) was placed on the belly of the abductor hallucis[14].

The sensory test was performed to the sural nerve. The stimulating electrode was placed on the posterior surface of the lower leg. The reference electrode was placed in line with the course of the sural nerve. The ground electrode was placed on the medial malleolus. Finally, the recording electrode was placed behind and below the lateral malleolus exactly on the nerve[14].

For motor nerve conduction velocity, a stimulus of a duration of 0.1 msec and a rate of 1 pulse/sec was applied. For sensory nerve conduction velocity, a stimulus was applied with a duration of 0.05 to 0.1 msec with a rate of 1 pulse/sec. The signals were captured on an oscilloscope and displayed on the computer screen. The consultant neurologist applied about five shocks and chose the best response.

Electromyogram

The five muscles chosen test all three nerve supplies to the leg and foot muscles. The abductor digiti minimi muscle is supplied by the lateral plantar branch of the tibial nerve. The abductor hallucis muscle is supplied by the medial plantar branch of the tibial nerve. The gastrocnemius muscle is supplied by the tibial nerve. The tibialis anterior muscle is supplied by the deep peroneal nerve. Finally, the peroneus longus is supplied by the superficial peroneal nerve.

A coaxial (concentric) needle electrode was used to accomplish the EMG test. The consultant neurologist tested the following muscles: both heads of gastrocnemius, peroneus longus, tibialis anterior, abductor hallucis, and abductor digiti minimi. The

ground electrode was placed on the medial tibial shaft. After locating the muscle by palpation, the area was wiped with an alcohol swab. The patients were asked to relax the muscles in order to insert the needle. A standard clinical scale of 0 to 4 was used to grade the fibrillation potential activity at rest: (0) represented absence of abnormalities, (1+) represented spontaneous activity caused by the needle insertion, (2+) represented moderate output of spontaneous activity, (3+) represented spontaneous activity between (2+) and (4+), and (4+) represented continuous spontaneous activity that filled the oscilloscope screen[28].

Data Analysis

Descriptive and frequencies statistics were used to calculate the mean age, number of male and female subjects, grades of fibrillation potentials in each tested muscle, and the mean latencies and velocities for motor and sensory nerves.

Non-parametric statistics were used in the process of data analysis. A Binomial test was used for both fibrillation and positive sharp wave potentials. This test was used to determine which muscles had statistically significant levels of abnormal potential values at the level of 0.05 probability, to determine whether the amount of abnormal potential occurrence was significantly greater than what is expected in the normal muscle.

The Kruskal-Wallis test was used to determine whether there was a statistically significant difference in abnormal potentials (at level of 0.05 probability) between the three ankle sprain grades for each muscle. This test was performed for both fibrillation and positive sharp wave potentials. The chi-square values were used separately to calculate and compare between ankle sprain grade groups in each tested muscle.

CHAPTER III

Results

Fifteen patients between the ages of 19 and 53 years old, with a mean age of 33.2 (standard deviation 10.64), participated in this study. The tested sample included 6 females (40%) and 9 males (60%). Subjects were divided into three groups for mild, moderate, and severe ankle sprain. Each ankle sprain grade group consisted of 5 patients, for a percentage of 33.33% in each group.

The means and standard deviations of the latency and motor conduction velocity of the peroneal and tibial nerves were calculated. The peroneal nerve mean latency was 3.70 ms (standard deviation 0.42). The tibial nerve mean latency was 3.70 ms (standard deviation 0.53). The peroneal nerve motor conduction velocity mean was 49.06 M/Sec with (standard deviation 1.86). The tibial nerve motor conduction velocity mean was 48.30 M/Sec with (standard deviation 1.61) (Table 1).

The sural nerve conduction velocities and amplitudes are shown in Table (1). The sural nerve sensory conduction velocity mean was 49.15 M/Sec (standard deviation 3.22). The mean amplitude of the sural nerve was 22.85 μV (standard deviation 4.70).

A binomial test was used to compare expected incidence of fibrillation potentials with actual fibrillation potentials observed in each muscle. Since 6% of normal individuals have been reported to have fibrillation potentials in abductor digiti minimi and adductor hallucis muscles, this was used as the threshold for significant fibrillation in these muscles[27]. In other words, more than 6% of the subjects had to show fibrillation potentials in these muscles for the findings to be considered significant (Table 2).

As with the data for fibrillation potentials, a binomial test was used to compare expected incidences of positive sharp wave potentials with actual positive sharp wave potentials observed in each muscle. Since 12% of normal individuals have been reported to have positive sharp wave potentials in abductor digiti minimi and adductor hallucis muscles, this was used as the threshold for significant positive sharp waves in these muscles[27]. In other words, more than 12% of the subjects had to show positive sharp waves in these muscles for the findings to be considered significant (Table 3).

For gastrocnemius, peroneus longus and tibialis anterior, no fibrillation or positive sharp wave potentials were expected to be seen in normal people[27]. Based on these criteria, all tested muscles that revealed significant fibrillation and/or positive sharp wave potentials were considered abnormal.

One patient out of 15 had fibrillation and positive sharp waves in the tibialis anterior muscle. Two patients showed fibrillation and positive sharp waves in the peroneus longus muscle. Two patients showed fibrillation potentials in the gastrocnemius muscle, whereas one patient showed positive sharp wave in the same muscle. Seven patients showed the fibrillation potentials in the adductor hallucis muscle where as 5 patients showed positive sharp waves in the same muscle. Six patients showed fibrillation potentials in the abductor digiti minimi muscle, and 4 patients showed positive sharp wave potentials in the same muscle.

The Kruskal-Wallis test was used to test for differences between ankle sprain grades I, II, III, for fibrillation potentials occurrences showed no significant difference between each ankle sprain grade groups in all tested muscles. The Chi-Square test

confirmed these results with a value of 5.991. Which showed that there was no difference between each grade group (Table 4).

The Kruskal-Wallis test, for positive sharp wave potentials, also showed no significant difference between ankle sprain groups in all tested muscles. The Chi-Square test confirmed this result with a value of 5.991, which showed that there was no significant difference between each ankle sprain grade group (Table 5).

CHAPTER IV

Discussion

This study showed normal latencies and conduction velocities for motor and sensory nerves tested (tibial, peroneal and sural). The values obtained from subjects in the study compare favorably with normal published values[17] (Table 1).

Severe ankle sprain may cause some sensory changes as noted by Bullock-Saxton[30]. These sensory changes are due to damage to the local sensory receptors in the ligaments and the capsule of the ankle joint. The results of this study revealed sensory and motor nerve conduction after grade I, II, III ankle sprains. These results provide evidence that sensory abnormalities following ankle sprains are caused by factors other than nerve damage.

With the exception of 3 of the patients who complained of numbness immediately after the injury, none of the patients in this study manifested any signs of sensory loss. Those 3 subjects who experienced numbness immediately after injury noted this numbness to be on the dorsum of the foot. This may have been due to the swelling that occurred post injury, causing compression of the sensory nerve. The peripheral nerve trunks are very strong structures which derive their strength from the resilient connective tissue sheaths. This strong nerve structure can protect the nerve trunk from traction injury. However, small cutaneous nerves and unmyelinated nerves are not protected well against compression forces. Compression may have caused decrease to the blood flow of these small nerves and compromised their function. As soon as the swelling decreased, the sensation returned to normal[23].

In order for a traction injury to occur, a nerve must be stretched beyond its normal limit. During an ankle sprain this limit is not exceeded[25]. As Liu et al stated, a nerve may be damaged if it's stretched more than 4.1% of its total length[32]. This kind of stretching can occur in cases like fractures. Ankle sprains can move the nerve for about five to eight mm only, which according to Liu is not enough to damage the nerve. This may explain why we have normal values for latencies and nerve conduction velocities of the tested nerves (Table 1). None of the subjects appears to have sustained direct nerve damage.

As shown in Table (2), 47% of the total patients had fibrillation potentials in the abductor hallucis muscle and 40% had them in the abductor digiti minimi muscle. These percentages were compared to an expected proportion of 6% occurrence of fibrillation potentials[27]. Fibrillation potentials were found in the gastrocnemius muscle and peroneus the longus muscle in 13% of the subjects. Fibrillation was found in the tibialis anterior muscle in only one patient (6%).

Table (3) shows muscles which had positive sharp waves. According to Gatens and Saeed, 12% of the normal population has positive sharp wave potentials in the abductor hallucis muscle and abductor digiti minimi muscle[27]. This expected proportion was compared with the proportion found in this study. Thirty-three percent of the patients had positive sharp waves in the abductor hallucis muscle, whereas 27% had them in the abductor digiti minimi muscle. Thirteen percent of the patients had positive sharp waves in the peroneus longus muscle. Six percent of the patients had positive sharp waves in both the tibialis anterior and the gastrocnemius muscles. All statistical tests of these results for both fibrillation and positive sharp wave potentials showed a statistical

significance difference between our study subjects and the reported incidence in the literature at the .05 level.

Muscles supplied by the tibial nerve showed more abnormal potentials than muscles supplied by the peroneal nerve after an ankle sprain. Most studies we discussed in the literature review noted peroneal nerve injury after an ankle sprain[19,20,21,22,23,25]. Some of the studies did not mention any injury to the tibial nerve. Nitz et al found 10% of the patients with grade II ankle injury had denervation potentials in muscles supplied by the tibial nerve[3]. Nitz et al found the results of motor nerve conduction studies within normal limits for all the patients. They found 83% of the patients with grade III ankle sprains had denervation potentials in muscles supplied by the tibial nerve. They also found that 17% of the patients with grade II ankle sprain had denervation potentials in muscles supplied by the peroneal nerve and 86% of the patients with grade III ankle sprains had denervation potentials in muscles supplied with the same nerve. The authors suggest that both tibial and peroneal nerves can be injured due to the stretching of the nerves at the bifurcation of the sciatic nerve.

If we determine that nerve injury after an ankle sprain is caused by the mechanical stretching of the nerve, then the peroneal nerve is the only nerve that should be injured after an inversion sprain. Our results showed abnormal potentials in muscles supplied by both the peroneal and tibial nerves. Nitz et al suggested a mechanical stretching at the bifurcation of the sciatic nerve, but this would require a large amount of force to exert traction on the terminal branches of the peroneal nerve that must be transmitted along the

entire length of the nerve to damage it near the bifurcation. As noted previously Nobel noted that the forces needed would be enormous.[25]

In Table (4) and (5), we found no significant difference between ankle sprain grade groups at a level of ($p \leq 0.05$). Abnormal potentials in the same amounts were present in all grade groups. Fibrillation and positive sharp wave potentials were present in all ankle sprain grades equally. Nitz said that abnormal potentials differ from one grade to another. He found no abnormal potentials in patients with grade I sprains, and he noticed that patients with grade III ankle sprains have more abnormal potentials than patients with grade II sprains. Nitz suggested that the greater the grade the more unstable the joint, and the more unstable the joint, the greater the traction on the nerve. This was his explanation of why there were more abnormal potentials in higher ankle sprain grades.

In this study there was no statistical difference between the three ankle sprain grade groups. This suggests that grades of ankle sprains have no relationship with the incidence of abnormal potentials. Furthermore, if the ankle sprain grade level is not statistically related to the occurrence abnormal potentials then all traction theories fail to explain the cause of these potentials.

Abnormal EMG potentials such as fibrillation potentials and positive sharp waves are associated with motor nerve damage. However, it does not appear that direct trauma to the tibial nerve from an ankle sprain is sufficient to cause nerve damage. Spielholz's hypothesized that the abnormal potentials seen post injury were the result of the sensory receptor damage.

If we follow Spielholz's theory, step by step, and hypothesize that after ankle sprains the mechanical receptors in the ligaments and the joint capsule will be damaged. This would decrease the sensory feedback, traveling towards the spinal cord. In the spinal cord Spielholtz hypothesized the presence of factors called antifibrillation factors which were present in the cell body of the alpha motor neurons. These factors control the occurance or lack of occurrence of fibrillation potentials and positive sharp wave potential activities in the muscles by controlling and regulating the outgoing impulses from alpha motor neurons the muscles fibers. In order for these factors to function properly, they need to receive feedback from the joints and other structures. After an ankle sprain, the sensory feedback will be disturbed or diminished, which will lead to a decrease in the stimulation of the alpha motor neuron which will lead to a decrease in AFF. The influence of these factors on the muscles will be decreased, and the abnormal potentials will become present in the muscles around the injured joint (figure 4).

In summary we found abnormal potentials in muscles supplied by both tibial and peroneal nerves. We found abnormal potentials in all ankle sprain grades. Our subjects had normal nerve conduction studies for both motor and sensory nerves. Therefore it seems the findings in this study can not be explained by the traction theory. Although swelling could have caused some compression damage, all subjects were placed in Plaster Of Paris casts, which helped to control it. Therefore the antifibrillation factors theory might be considered as a way to explain the occurence of the abnormal potentials in the muscles surrounding the injured ankle. The findings of this study do provide additional

evidence for the presence of the antifibrillation factors, and it suggests that factors other than traction and compression be taken into consideration.

One of the limitations of this study was the small sample size. In the future it would be beneficial to have a larger sample. Another limitation of this study is that there was no control group. A group of non-ankle sprain subjects with matched characteristics would have strengthened the study or subjects could have been their own controls by studying the non-injured leg. The further limitation was the large age range within a small sample size. The older are likely to have physiological changes and abnormal potentials as a result.

The study needs to be repeated with a larger sample size a wider variety of muscles tested. Further studies are needed to investigate the antifibrillation factors theory. Studies are also recommended to examine injury to other joints to see if abnormal potentials occur in the muscles around other joints.

Studying the contralateral joint (the uninjured ankle joint) could help in understanding the antifibrillation factor theory. If antifibrillation factors located in the alpha motor neurons can control abnormal potentials in the muscles, there might be some influence from the joint structures in both the injured and the uninjured joint limbs.

Acknowledgments

I am greatly indebted to Professor John Echternach for his suggesting the topic for this thesis. His continual support and encouragement were invaluable. I would especially like to thank Professor Martha Walker and Professor Michael Tamburello for effort, time and guidance. I would like to acknowledge the help of the clinicians Dr. Adnan Khuraibet and Dr. Fathi Khalaaf in Kuwait who helped me in collecting the data needed to carry out the study.

As a personal note, I would like to convey deepest appreciation to my parents for giving me infinite love, support and motivation for my entire academic career. I wish to express my deepest gratitude to Sarah Al-Shoura for her never ending support, inspiration, and love. Finally I would like to thank my friends, especially Ahmad Shah, for their support. Without all these people this endeavor would have been impossible.

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Appendix A

INFORMED CONSENT
Old Dominion University
College of Health Sciences
Physical Therapy Department

TITLE OF RESEARCH: ABNORMAL ELECTRICAL POTENTIALS IN LOWER LIMB MUSCLES AFTER ANKLE SPRAIN GRADES I, II AND III.
INVESTIGATORS: TALAL A. AL-SHATTI, B.Sc. June 1994, Kuwait University, Kuwait.

DESCRIPTION OF RESEARCH:

Several studies have been conducted testing fibrillation potentials and positive sharp waves in the muscles of the leg and foot after ankle sprains. The purpose of this study is to better define and explain the reaction of the muscles around the ankle joint after grades I, II and III ankle sprains.

You will be participating in a study involving needle insertion in the leg and foot muscles along with electrical shocks.

EXCLUSIONARY CRITERIA:

You have completed casting, x-ray, and manual instability test by the orthopedic surgeon. To the best of your knowledge, you should not have diabetes, neurological, and cardiovascular diseases. You should not have history of lumbar spine, hip, thigh, or knee injury, and fractures for the past two years that would prohibit your participation in this study.

RISK AND BENEFITS:

The testing procedures that you undergo may result in pain, faintness, or discomfort. There also exists the possibility that you may be subject to risks that have not yet been defined. These risks are minimal and all precautions will be taken to ensure your safety. The main benefit to accrue from this study in the attainment of information relative to electrical potentials in muscles around the injured joint after ankle sprain. Pertinent information relative to your responses to this study will be discussed with you by the investigator of this study.

COSTS AND PAYMENTS:

Your efforts in this study are voluntary, and you will not receive some remuneration to help defray incidental expenses with participation.

NEW INFORMATION:

Any new information obtained during the course of this research that is directly related to your willingness to continue to participate in this study will provided to you.

CONFIDENTIALITY:

Any information obtained about you from this research, including medical history and laboratory findings will be kept strictly confidential. Data derived from this study could be used in reports, presentations, and publications, but you will not be individually identified.

WITHDRAWAL PRIVILEGE:

You are free to refuse to participate in this study or to withdraw at any time and your decision to withdraw will not adversely affect your care at this institution or cause a loss of benefits to which you might otherwise be entitled. If you do decide to withdraw, you agree to undergo all trial evaluations necessary for your safety and well-being as determined by the investigator. The investigator reserves the right to withdraw your participation at any time throughout this investigation if they observe any contraindication to your continued participation.

COMPENSATION FOR ILLNESS AND INJURY:

In the unlikely event of injury or illness resulting from the research protocol, no monetary compensation will be made, but any immediate emergency medical treatment which may be necessary will be available to you without charge by the investigator. If any injury result from your participation in this research project, OLD Dominion University does not provide insurance coverage, free medical care or any other compensation for such injury. In the event that you suffer injury as a result of participation in this study, you may contact Talal AL-Shatti at 5316507.

VOLUNTARY CONSENT:

I certify that I have read the preceding sections of this document, or it has been read to me; that I understand the contents; and that any questions I have pertaining to the research have been, or will be answered by Talal Al-Shatti (5316507). A copy of this informed consent form has been given to me. My signature below indicated that I have freely agreed to participate in this study.

_____ _____

Subject's Signature Date

_____ _____

Witness's Signature Date

INVESTIGATOR'S STATEMENT:

I certify that I have explained to the subject whose signature appears above the nature and purpose of the potential benefits and possible risks associated with participation in this study. I have answered any questions that have been raised by the subject and have encouraged him/her to ask additional questions at any time during the course of this study. I have witnessed the above signature on the date stated on this consent form.

_____ _____

Investigator's Signature Date

Table 1. Descriptive statistics to calculate the mean and standard deviation of the motor and sensory nerves velocities, latencies, and amplitudes.

	Mean	Normal values[17]
Peroneal nerve Latency ms	3.71 ± 0.42	3.77 ± 0.86
Peroneal nerve MCV m/s	49.10 ± 1.87	48.30 ± 3.90
Tibial nerve Latency ms	3.70 ± 0.53	3.96 ± 1.00
Tibial nerve MCV m/s	48.31 ± 1.61	48.50 ± 3.60
Sural nerve Sensory m/s	49.15 ± 3.23	52.50 ± 5.60
Sural nerve sensory Amplitudes μV	22.85 ± 4.71	20.90 ± 8.00

A comparison between the mean of the patients latencies and conduction velocities values, and normal values[17].

Table 2. Fibrillation potentials in each muscle.

	Category	N	Observed Proportion	Test Proportion	Asymptotic Significant
Abductor digiti minimi	No	9	.6	.06	.000
	Yes	6	.4		
Abductor Hallucis	No	8	.53	.06	.000
	Yes	7	.47		
Gastrocnemius	No	13	.87	0	.000
	Yes	2	.13		
Peroneus Longus	No	13	.87	0	.000
	Yes	2	.13		
Tibialis Anterior	No	14	.93	0	.000
	Yes	1	.06		

No = no fibrillation potentials in the tested muscle.
Yes = presence of fibrillation potential in the tested muscle.
A comparison between the expected incidence of fibrillation potentials and the actual fibrillations observed in each muscle using a binomial test.

Table 3. Positive sharp wave potentials in each muscle.

	Category	N	Observed Proportion	Test Proportion	Asymptotic Significant
Abductor digiti minimi	No	11	.73	.12	.000
	Yes	4	.27		
Abductor Hallucis	No	10	.67	.12	.000
	Yes	5	.33		
Gastrocnemius	No	14	.93	0	.000
	Yes	1	.06		
Peroneus Longus	No	14	.93	0	.000
	Yes	1	.06		
Tibialis Anterior	No	13	.87	0	.000
	Yes	2	.13		

No = no positive sharp wave potentials in the tested muscle.
Yes = presence of positive sharp wave potential in the tested muscle.
A comparison between the expected incidence of positive sharp wave potentials and the actual positive sharp wave observed in each muscle using a binomial test.

Table 4. Kruskal-Wallis test and Chi-Square test for fibrillation potentials between each sprain grade.

	Abductor digiti minimi	Abductor Hallucis	Gastrocnemius	Peroneus Longus	Tibialis Anterior
Chi-Square	.000	3.500	1.077	1.077	2.000
df	2	2	2	2	2
Asymptotic Significant	1.000	.174	.584	.584	.368

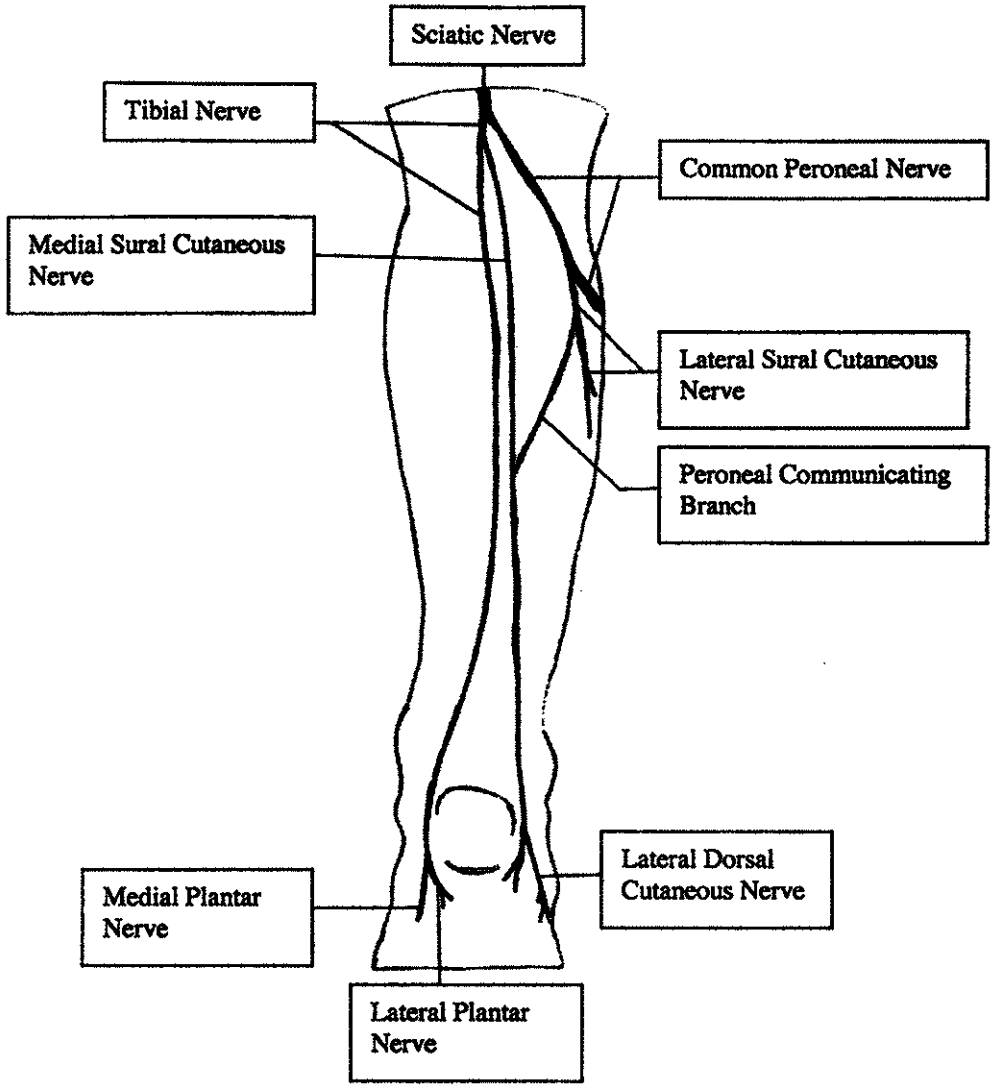
No difference between each ankle sprain grade group in all tested muscles at a significant level of $p \leq 0.05$ (chi-square value 5.991).

Table 5. Kruskal-Wallis test and Chi-Square test for positive sharp wave potentials between each sprain grade.

	Abductor digiti minimi	Abductor Hallucis	Gastrocnemius	Peroneus Longus	Tibialis Anterior
Chi-Square	.636	2.240	2.000	1.077	2.000
df	2	2	2	2	2
Asymptotic Significant	.727	.326	.368	.584	.368

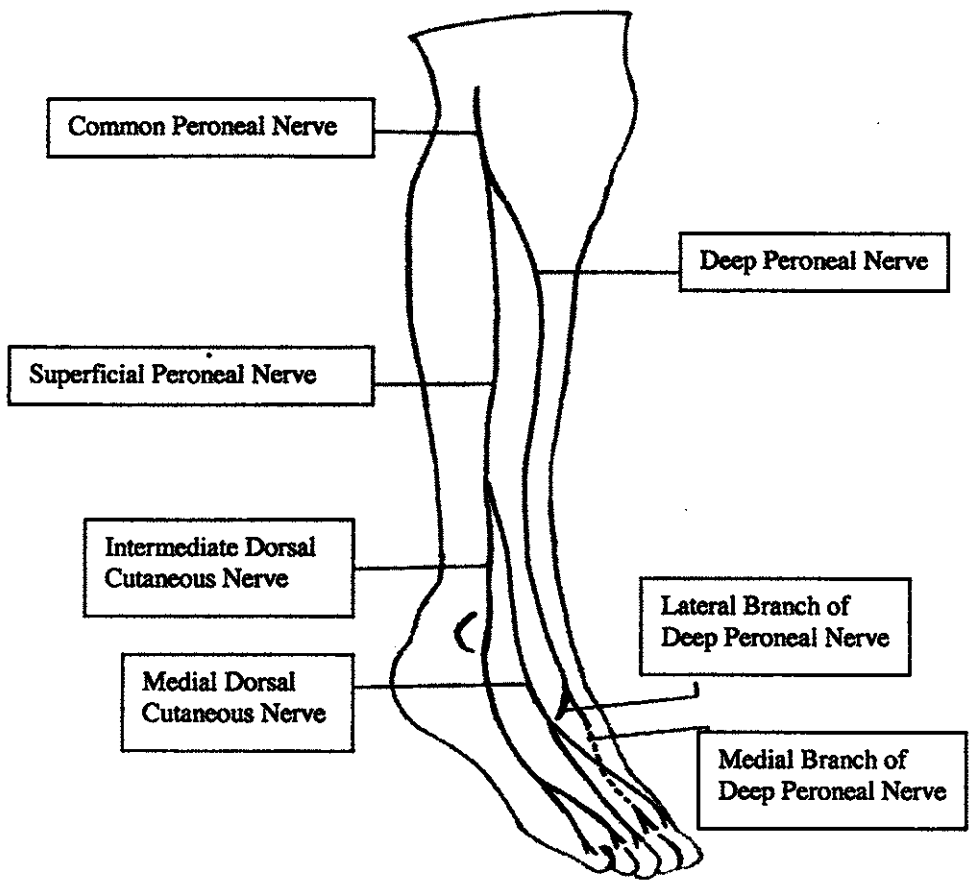
No difference between each ankle sprain grade group in all tested muscles at a significant level of $p \leq 0.05$ (chi-square value 5.991).

Figure 1. Tibial nerve and its branches.



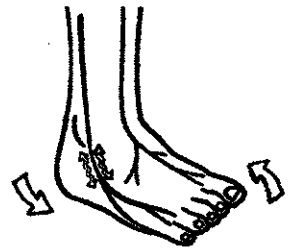
Posterior view of the right lower limb illustrating the anatomy of the tibial nerve and its branches.

Figure 2. Common Peroneal nerve and its branches.

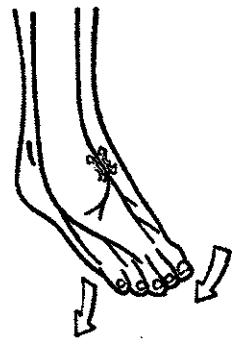


Lateral view of the right lower limb illustrating the anatomy of the Common Peroneal nerve and its branches.

Figure 3. Nerve traction during ankle inversion.



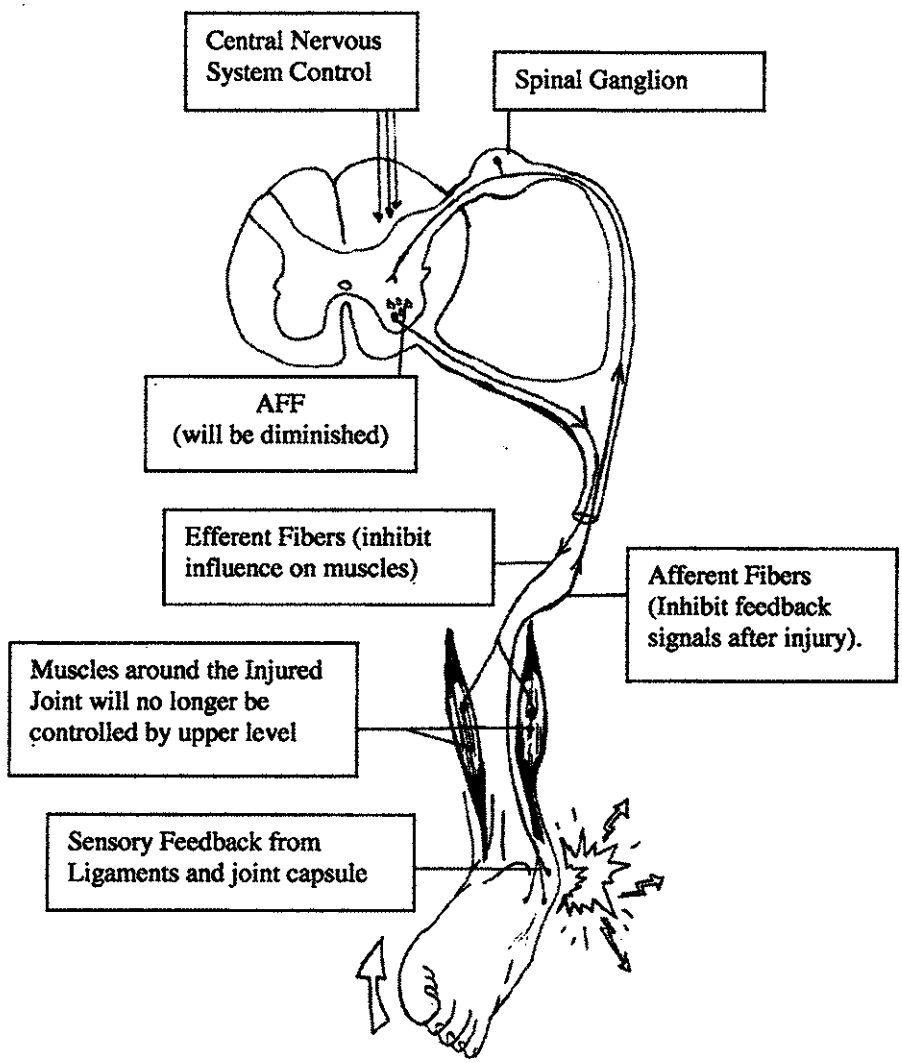
a



b

A. Traction on the intermediate dorsal cutaneal branch of the superficial peroneal nerve.
B. Traction on the deep peroneal nerve during inversion.

Figure 4. The antifibrillation factors theory (AFF).



After ankle sprain, feedback from ligament and joint capsule will be diminished. The spinal cord will not receive information from the joint. This will lead the spinal motor neuron to decrease the release of AFF. Therefore abnormal potentials may be present in the muscles around the injured joint.