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# Decreased Nerve Conduction Velocity in Football Players

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***abstract***

***Background:*** Lower limbs nerves are exposed to mechanical injuries in the football players and the purpose of this study is to evaluate the influence of football on the lower leg nerves.

***Materials and Methods:*** Nerve conduction studies were done on 35 male college students (20 football players, 15 non active) during 2006 to 2007 in the Shiraz rehabilitation faculty. Standard nerve conduction techniques using to evaluate dominant and non dominant lower limb nerves.

***Results:*** The motor latency of deep peroneal and tibial nerves of dominant leg of football players and sensory latency of superficial peroneal, tibial and compound nerve action potential of tibial nerve of both leg in football players were significantly prolonged ($p$<0.05). Motor and sensory nerve conduction velocity of tibial and common peroneal in football players were significant delayed ($p$<0.05).

***Conclusion:*** It is concluded that football is sport with high contact and it causes sub-clinical neuropathies due to nerve entrapment.



## Introduction

Football is one of the most widely played sports in the world, that is playing professional and amateur. During this sport, body weight, the force of pressure and stress over 90 minutes playing time is loaded on the lower leg and the stress repetition in football causes physiological and pathological change, especially in the dominant leg. Lower limb nerve fibers are exposed to acute and chronic mechanical injuries in athletes because of the excessive physical demand as with other structures. Many existing studies evaluate the muscular change and activity required to follow the sport (training) but some studies have investigated nervous system changes in athletes [1-3]. This study was performed to evaluate the effect of playing football on the tibial and common peroneal nerves crossing lower leg.

## Materials and Methods

The football player group consisted of 20 male student (age mean±SD: 21.6±1.85 year) subjects who over 3 years for about 2 days a week continuously played football, the non-active (control) group consisted of 15 male student (age mean±SD: 21.98±2.42 yr) subjects who did not play football and any other sport that would have an effect on the lower limbs. Subjects who had previously peripheral nerve injuries, nervous and metabolic disease, vertebral column damage, lumbar disc herniation, vascular disease and played other sports that effect on lower limb, such as running, were excluded from the study.

The neurophysiological study consisted of motor and sensory nerve conduction studies of the tibial and common peroneal nerves. Superficial skin temperature of foot because of cold effect on nerve conduction checked and controlled between 31-32°C. Both the dominant (DL) and non-dominant (non DL) legs of all subjects were tested by an examiner using a Medelec Saphire Electromyography (Medelec, SaphireII, UK).

Nerve conduction studies (NCS) were performed using standard techniques of supra maximal percutaneous stimulation with a constant current stimulator and surface electrode recording on both limbs of each subjects. Sensory responses were obtained by antidromicaly stimulating at the lower leg and recording from the dorsum of foot (superficial peroneal nerve) and posterior to lateral maleollus (sural nerve) with disc electrodes. Compound nerve action potential (CNAP) of tibial nerve was obtained by stimulating at the palm of foot and recording with disc electrode posterior to medial maleollus.

The tibial motor nerve was examined by stimulating the tibial nerve at the ankle (posterior to medial maleollus), and the knee joint (posterior to knee joint). The tibial nerve was stimulated with bipolar surface electrodes and the recording was carried out over the abductor hallucis brevis muscle with surface electrodes. The deep peroneal motor nerve was examined by stimulating the nerve at the ankle (lateral to tibialis anterior tendon) and below the head of fibular bone (at the fibular neck) with bipolar surface electrodes. The deep peroneal motor response was

recorded from the extensor digitrom brevis with surface electrodes.

In the present study, the following tibial and common peroneal nerve measures were used: (I) distal peak latency of the sensory nerve action potential (DI-S); (II) conduction velocity of the sensory nerve (CV-S); (III) distal onset latency of the compound muscle action potential (DL-M); (IV) conduction velocity of the motor nerve fiber (CV-M); and peak latency of the tibial CNAP (CNAP).

The mean nerve conduction parameters of this population were compared with existing literature values. Simple biometric measurements were also carried out. The groups were matched according to weight, age, height, and superficial skin temperature of foots. All subjects were informed of the study procedure, purposes, and familiar risks, and all gave their informed consent. This study was conducted according to the guidelines of the Delisa et al. and approved by the ethics committee of our faculty. The results are presented as mean (SD). Differences between the groups were calculated using a non-parametric test for independent samples (Mann–Whitney *U* test). The SPSS-13 for personal computers was used to do statistical analysis. A *p*-value of $\leq$0.05 was considered significant.

## Results

Two groups of subjects were examined and compared with each other, the distal latency of motor deep peroneal and tibial nerves were given in table 1. Distal latency of motor deep peroneal ($p$=0.01) and tibial nerves ($p$=0.01) in non DL of football players were significantly prolonged compared to control group.

The distal latency of sensory superficial of both DL ($p$=0.01) and non DL ($p$=0.02) and sural nerves of both DL ($p$=0.01) and non DL ($p$=0.01) of football players compared to the control group were significantly prolonged, also tibial CNAP of both DL ($p$=0.01) and non DL ($p$=0.01) of football players were significantly prolonged as compared with the control group (Table 1).

Motor NCV of deep peroneal of both DL ($p$=0.01) and non DL ($p$=0.01) and tibial nerves of both DL ($p$=0.02) and non DL ($p$=0.01) of football players were significantly delayed compared to the control group (Table 2). Table 2 shows that sensory NCV of superficial peroneal of both DL ($p$=0.01) and non DL ($p$=0.01) and sural nerves of both DL ($p$=0.01) and non DL ($p$=0.01) of football players significantly were delayed as compared with the control group. Although reaching statistical significance between the groups, nerve conduction values within the football playing population fell within the normal range.

## Discussion

Our study demonstrated that the sensory and motor conduction velocity of tibial and common peroneal nerves of both DL and non DL of football players as compared with the control group significantly were delayed. Retrospective studies indicate that muscles, tendons, bones, and nerves tend to adapt in response to high training loads. However, these particular adaptations are not beneficial to performance and may be associated with increased injury risk [1-6]. Also, some study evaluated effect of sport on NCV and changes following them and they recorded that NCV is decreased [7-9].

**Table1.** Distal onset latency of motor tibial and peroneal nerves, distal peak latency of the sensory superficial and sural nerves, and tibial CNAP

| Characteristics | Football player (msec) (Mean±SD) | Control (msec) (Mean±SD) | *p*-Value (Mean±SD) |
|---|---|---|---|
| Deep peroneal motor DL | 3.79 ±0.99 | 3.49 ±0.342 | 0.14 |
| Deep peroneal motor non DL | 3.77 ±0.64 | 3.03 ±0.8 | 0.01 |
| Tibial motor DL | 3.5 ±1.22 | 3.22 ±0.22 | 0.06 |
| Tibial motor non DL | 3.72 ±1.3 | 3.12 ±0.57 | 0.01 |
| Superficial peroneal sensory DL | 3.54±0.52 | 3.11 ±0.38 | 0.01 |
| Superficial peroneal sensory non DL | 3.57 ±0.52 | 3.02±0.3 | 0.02 |
| Sural DL | 3.64 ±0.5 | 3.22 ±0.28 | 0.01 |
| Sural non DL | 3.79 ±0.42 | 3.24 ±0.3 | 0.01 |
| Tibial CNAP DL | 3.68 ±0.18 | 3.09 ±0.23 | 0.01 |
| Tibial CNAP non DL | 3.5 ±0.19 | 2.97 ±0.2 | 0.01 |

DL: Dominant Leg

**Table 2.** Motor NCV of tibial and deep peroneal nerves, sensory NCV of sural and superficial peroneal nerves

| Characteristics | Football player (m/sec) (Mean±SD) | Control (m/sec) (Mean±SD) | *p*-Value (Mean±SD) |
|---|---|---|---|
| Deep peroneal motor DL | 47.68 ±2.02 | 51.2 ±2.75 | 0.01 |
| Deep peroneal motor non DL | 49.5 ±1.09 | 51.5 ±2.02 | 0.01 |
| Tibial motor DL | 44.8 ±2.94 | 47.97 ±3.98 | 0.02 |
| Tibial motor non DL | 48.38 ±1.69 | 50.52 ±2.77 | 0.01 |
| Superficial peroneal sensory DL | 38.68 ±2.52 | 43.78 ±2.34 | 0.01 |
| Superficial peroneal sensory non DL | 38.41 ±2.03 | 45.61 ±1.65 | 0.01 |
| Sural DL | 23.94 ±0.86 | 28 ±1.35 | 0.01 |
| Sural non DL | 24.8 ±1 | 28.86 ±1.26 | 0.01 |

DL: Dominant Leg

Nerve entrapment syndrome in athletes can happen and peripheral nerves in athletes could be exposed to effect of difference damage; trauma can be microscopic or macroscopic, that is associated with connective tissue changes closed to nerve, or direct trauma to nerve because of repeated stress, pressure, stretch, tissue alignment changes and friction.

Nerves may be exposed to the effect of short time severe pressure, or long time low pressure; nerve and vessels of nerve may be exposed to pressure in tight fibrous tissue or fibro-osseus tissue, by bone anomaly, muscle hypertrophy, soft tissue inflammation, scar tissue, tumor and orthopedic abnormality posture; therefore, axon, myelin cover, connective tissue cover of nerves or the mixture of could be injured [10-12].

Pressure neuropathies can occur in athletes who are due to repeated motion or repeated blunt trauma [11-14]. These nerve damages maybe remains sub-clinical without significant symptom and couldn't be recognized before neurological damage is permanent. Colack et al. defined that the sensory latency of medial plantar and sural nerves in middle distance runners significantly were prolonged as compared with non-runners, also sensory NCV of these nerves in middle distance runners as compared with non runners significantly was decreased [7]. Kamen et al. defined the motor NCV of posterior tibial nerve in marathon runner as compared with non-athletes significantly was delayed [9]. In a study by Colack et al. on median, ulnar and radial nerves of tennis players it was found that motor and sensory NCV of radial and sensory NCV of ulnar nerve in dominant arm of tennis players as compared with non-active group significantly were delayed. They defined that the delayed NCV was caused because of damage, repeated movements, trauma through sport and following sub clinical neuropathy [8]. Several authors have performed NCS on football players with ankle sprain [12, 14]; we could find no studies in the literature suggesting electro diagnostic abnormalities in asymptomatic football players. This study is the first reported observation of delayed motor and sensory conduction velocity of the common peroneal and tibial nerves in healthy football players as compared with the control group. The athletes' limbs involved in sport were exposed to different external and internal damage and trauma; on other hand, football is a sport with high contact which has caused sub-clinical neuropathies because of chronic or acute nerve entrapment and in conclusion caused no significant clinical decrease of NCV. We proposed that repeated movements, damage and trauma through sport to lower limbs are probably major etiological factors in the delayed NCVs. If the biomechanical and physiologic stresses inherent in the game of football have been correctly analyzed and understood, the clinician can rehabilitate the patient, plan a preventive conditioning program, and modify biomechanics scientifically.

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### Authors' Contributions

All authors had equal role in design, work, statistical analysis and manuscript writing.

### Conflict of Interest

The authors declare no conflict of interest.

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