

COMPARISON OF NERVE CONDUCTION VELOCITIES OF LOWER EXTREMITIES BETWEEN FOOTBALL PLAYERS AND CONTROLS

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ABSTRACT

This study aimed to compare neuronal function in the lower limbs of elite footballers and an age-matched control group. Conduction velocity, response latency and amplitude following electrical stimulation were investigated. Thirty male football players and 30 untrained males comprised the subject and control groups, respectively. We showed that professional football players have significantly slower sural nerve conduction velocity than untrained controls ($p < 0.05$). Although we found no signs of neuropathy, mild changes in nerve conduction velocity were evident in the sural nerve of the players. Motor conduction velocity of tibial nerve was also significantly prolonged in this group when comparing dominant and non-dominant extremities ($p < 0.05$). The tibial nerve muscle action potential amplitude was lower in the players' dominant limbs compared with the controls ($p < 0.05$). We found no statistical difference in motor conduction velocities, distal latencies and amplitudes of the common peroneal nerve when comparing players and controls in both dominant and non-dominant limbs ($p > 0.05$). Our results showed abnormalities of function in the sural and tibial nerves in football players compared with untrained controls, which may indicate early signs of neuropathy.

Keywords: Amplitude; Nerve conduction; Nerve injuries; Neurophysiological tests.

INTRODUCTION

Football is the most exercised sport worldwide and is associated with a risk of injuries in players (Pérez-Gómez *et al.*, 2020). Lower extremity is the most commonly injured region in football players due to high physical activity exposure (Longo *et al.*, 2012). Toth (2009) suggested that injuries to the lower extremity in footballers were due to a number of mechanisms. These include repetitive motion, rapid forceful foot position changes, large muscular forces and excessive load upon the foot, which all result in the lower limbs being exposed to a wide range of physical forces. These excessive biomechanical and physiological demands may cause neurophysiological changes, especially in peripheral nerves.

Zuckerman *et al.* (2019) studied high school athletes over a 2-year period and reported sport-related nerve injuries. They reported that the peripheral nerve injury rate (71.3%) was higher

in football than all other sports. Hainline (2014) suggested that peripheral nerve injuries in players may also occur because of either repetitive compression, stretching, or both. Chronic exposure of the nerve to physical disruption eventually exceeds the compensation ability of the tissue. As a result of this, peripheral nervous system injuries may occur in the lower extremities of the players. These injuries may also remain subclinical, without signs and symptoms (Bamac *et al.*, 2014).

Nerve conduction studies are essential tools in the assessment of the peripheral nerves (Tavee, 2019). These tests evaluate distal latency, conduction velocity and response amplitudes as neurological responses following electrical stimulation of a peripheral nerve (O'Bryan & Kincaid, 2021). These nerve conduction parameters can be affected by the demographic features and anthropometric measurements of the subjects. These parameters are used to investigate the effects of training and physical activity in athletes (Shah *et al.*, 2022). We hypothesised that excessive lower extremity mechanics have an impact on the nerves that traverse the popliteal and ankle regions in football players. Therefore, this study aimed to compare the effects of football playing on the tibial, common peroneal and sural nerves found in the distal lower limb and ankle, in football players and untrained controls.

METHODOLOGY

Ethical clearance

The study complied with the Declaration of Helsinki. Ethical approval for the study was granted by the local Research Ethics Committee of Kocaeli University with ethical clearance number 2014/167.

Subjects

Thirty male professional football players (age: 19.30±0.83 years) and 30 untrained, age-matched males (age: 19.33±0.84 years) volunteered to participate in this study. The control group consisted of participants who did not engage in any regular or organised sports activities. Demographic features of the football players and controls are presented in Table 1.

Table 1. DEMOGRAPHIC FEATURES OF THE FOOTBALL PLAYERS AND CONTROLS (N= 30 FOR EACH GROUP)

	Football players (mean±SD)	Controls (mean±SD)	<i>p</i>
Age (years)	19.30±0.83	19.33±0.84	0.853
Height (m)	1.77±0.06	1.78±0.05	0.369
Weight (kg)	70.27±5.63	70.37±7.71	0.947
Body mass index (kg/m ²)	22.34±1.14	22.02±1.96	0.647

SD=standard deviation.

Participants provided written informed consent prior to taking part. The footballers reported their kicking leg preference, which was assumed to be the dominant limb. The elite athletes in the study trained at least 8 hours per week and took part in one competitive match per week. Participants were initially assessed for any history, signs or symptoms of either peripheral

neuropathy or compression syndrome in the lower extremities. Participants were excluded if they had a recent history of lower limb injury.

Research procedures

Anthropometric measurements and neurophysiological tests were performed on each participant. Each player was informed of the testing procedures, benefits and risks. After the anthropometric measurements of the participants were taken, neurophysiological measurements were made by an experienced medical neurologist using a Neuropack M1, MEB-9204K (Nihon Kohden, Japan). The neurologist was blinded as to whether the subject was a footballer or a control. Both legs were tested. Neurophysiological investigation included motor nerve conduction studies of tibial and common peroneal nerves and sensory nerve conduction of sural nerve. Skin temperature was recorded in the participants at a point behind the medial malleolus. Mean skin temperature was 32.1°C and ranged from 30–34°C. Mean room temperature was 26.5°C. The subjects were examined in the morning and asked to avoid tiring physical activity on the testing day. An anthropometer was used to measure the distance between proximal and distal stimulation points. Nerve conduction studies were carried out using standardised techniques to obtain and record the action potentials. The supramaximal percutaneous stimuli were delivered in order to get adequate responses from both extremities of each subject with a constant current stimulator and surface recording electrodes of electromyograph (Garg *et al.*, 2013). The mean nerve conduction results of the subjects and controls were compared with reference data obtained from the literature.

Neurophysiological tests

Skin purifier was applied to clean the stimulation and recording sites. The tibial nerve was stimulated from the popliteal fossa and behind the medial malleolus. Responses of the abductor hallucis muscle were recorded by surface disc electrodes. The active electrode was positioned 1-cm below and behind the navicular tuberosity, while the reference electrode was placed on the first metatarsophalangeal joint. Similarly, for stimulation and measurement of conduction in the common peroneal nerve, the stimulation was applied from below the head of fibula and on the dorsum of the ankle. The active electrode was placed on the extensor digitorum brevis muscle, while the reference electrode was positioned on the fifth metatarsophalangeal joint. For the calculation of nerve conduction velocity, the measured distance between stimulating electrodes at distal and proximal sites was entered into the machine (Colak *et al.*, 2005; O'Bryan & Kincaid, 2021).

Sural nerve sensory action potential was analysed using an antidromic method. The active electrode was placed just behind the lateral malleolus and the reference electrode was positioned between 2- and 3-cm distally and below and behind the lateral malleolus. The sural nerve was stimulated at the posterior midline of the calf, while sensory action potential was recorded 15-cm distal to the points of stimulation. Distal latency was computed by the difference in time between the beginning of the electrical artefact to the first positive peak of nerve potential. Sensory conduction velocity was calculated by dividing the distance between the stimulating and recording electrodes by the calculated distal latency (Colak *et al.*, 2005; O'Bryan & Kincaid, 2021).

Anthropometric measurements

Simple anthropometric data were collected from each participant using a flexible, non-elastic measuring tape. The length of both lower extremities was measured as the distance between the umbilicus and medial malleolus while participants were standing, feet together and arms at rest by their sides. The circumference of the thigh and calf was measured with the participant again standing, with legs slightly apart to accommodate the measuring tape and weight-bearing equally on both feet. The circumference of the thigh was measured 15-cm proximal to the patella; the circumference of the calf was measured 15-cm proximal to the medial malleolus.

Statistical analysis

The Statistical Package for Social Sciences (SPSS) version 18.0 was used for statistical analysis (SPSS, IBM Inc., Armonk, NY, USA). The results are presented as mean \pm standard deviation (SD). Kolmogorov–Smirnov test was performed to analyse normality in the distribution of data. Differences between the footballer and control groups were compared by using a non-parametric test, Mann-Whitney U, as data were not normally distributed. A p value <0.05 was considered statistically significant.

RESULTS

No significant differences existed between the subjects and controls for any of the demographic characteristics investigated. These included age, height, weight and body mass index (BMI) (Table 1). Anthropometric leg measurements including total length and thigh and calf circumferences were compared between the footballers and the controls (Table 2). There were no statistical differences between football players and controls in height, weight, age, lower extremity length, or the measured circumferences of either thigh or calf.

Table 2. ANTHROPOMETRIC FEATURES OF FOOTBALL PLAYERS AND CONTROLS

		Football players (mean \pm SD)	Controls (mean \pm SD)	p
Length of the lower extremity (cm)	Right	96.83 \pm 4.46	97.88 \pm 4.07	0.260
	Left	96.65 \pm 4.87	98.03 \pm 3.98	0.207
Circumference of thigh (cm)	Right	52.43 \pm 3.21	53.28 \pm 3.81	0.583
	Left	52.96 \pm 2.92	52.65 \pm 4.02	0.641
Circumference of calf (cm)	Right	36.40 \pm 1.56	36.55 \pm 3.09	0.597
	Left	36.26 \pm 1.46	36.40 \pm 3.17	0.628

We found no statistical differences in motor conduction velocities, distal latencies and amplitudes of the common peroneal nerve between players and controls in both dominant and non-dominant limbs (Tables 3 & 4). We also found no statistical differences for these parameters when comparing the dominant and non-dominant extremities of the players themselves ($p>0.05$). Muscle action potential amplitudes of the tibial nerve were lower in the players' dominant limbs compared with the controls. There were no statistical differences in conduction velocities and latencies of the tibial nerve between the players and controls in either the dominant or non-dominant legs (Tables 3 & 4). However, motor conduction velocity of the

tibia I nerve in the players was significantly prolonged when comparing dominant and non-dominant extremities ($p=0.036$).

Table 3. NERVE CONDUCTION CHARACTERISTICS OF THE DOMINANT LEG

		Latency (msec) (mean±SD)	p	Amplitude (mV) (mean±SD)	p	Conduction Velocity (m/sec) (mean±SD)	p
Tibial nerve	Players	4.27±0.81	0.089	14.32±4.71	0.026*	45.84±3.61	0.395
	Controls	3.93±0.86		17.54±5.48		46.56±3.24	
Common peroneal nerve	Players	3.80±1.50	0.264	8.41±3.32	0.117	49.28±3.76	0.084
	Controls	3.93±1.15		7.14±2.69		51.46±4.68	
Sural nerve	Players	2.69±0.41	0.407	15.96±4.71	0.038*	46.33±4.00	0.027*
	Controls	2.58±0.39		20.51±8.54		49.14±5.17	

*Significant difference ($p<0.05$).

Table 4. NERVE CONDUCTION CHARACTERISTICS OF THE NON-DOMINANT LEG

		Latency (msec) (mean±SD)	p	Amplitude (mV) (mean±SD)	p	Conduction velocity (m/sec) (mean±SD)	p
Tibial nerve	Players	4.31±1.07	0.784	14.92±4.36	0.056	47.75±3.42	0.842
	Controls	4.11±0.72		17.64±5.32		47.81±3.86	
Common peroneal nerve	Players	3.49±0.53	0.756	8.43±3.47	0.149	49.94±3.66	0.228
	Controls	3.48±0.74		7.12±2.18		48.60±3.90	
Sural nerve	Players	3.01±0.39	0.120	13.87±3.87	0.002*	47.57±4.14	0.012*
	Controls	2.84±0.37		18.01±5.90		50.19±4.06	

*Significant difference ($p<0.05$).

The sural nerve conduction velocity was significantly delayed and amplitude was significantly smaller in the players compared with controls in both the dominant and non-dominant legs. We found no statistical significance between the groups for distal latency (Tables 3 & 4). However, in footballers the distal latency in the dominant leg was significantly delayed when compared with the non-dominant leg ($p=0.002$).

DISCUSSION

Sport-related peripheral nerve injuries may result following chronic and repetitive stress in athletes, which may lead to pathological changes in the lower leg. This study evaluated the differences between professional football players and non-active individuals in conduction velocities of tibial, common peroneal and sural nerves in the lower leg and ankle. The most significant result was the slower conduction velocity and lower amplitude of the sural nerve in

both dominant and non-dominant limbs of players compared with the corresponding legs of control subjects. Moreover, sural nerve distal latency in the dominant extremity was significantly delayed in players compared with their non-dominant extremity.

In athletes it is unusual for the sural nerve to become trapped. If the sural nerve does become trapped it is most often due to poorly fitting shoes, direct trauma, compression, or over stretching following an ankle sprain (Schon, 1994; Meadows & Finnoff, 2014). Our findings may be explained by various factors. We hypothesise that the sural nerve, which passes in close proximity to the gastrocnemius, may become adversely affected by muscular hypertrophy of the gastrocnemius in football players, leading to nerve compression (Figure 1A). Thus, increased muscle compartment pressure due to contraction may cause chronic and repetitive microtrauma of the nerve. There may also be fascia thickening after repetitive movements. Alternatively, previous inversion trauma may have an effect on conduction properties in players. In football, the powerful kicking motion is performed with a plantar-flexed ankle. The anatomy of the ankle makes it likely that a player will sprain the outside of the ankle (an inversion sprain) far more often than the inside of the ankle (an eversion sprain) (Kirkendall, 2011). Repetitive microtrauma may increase the vulnerability of the sural nerve, and chronic stretching may also affect sural nerve electrophysiological properties.

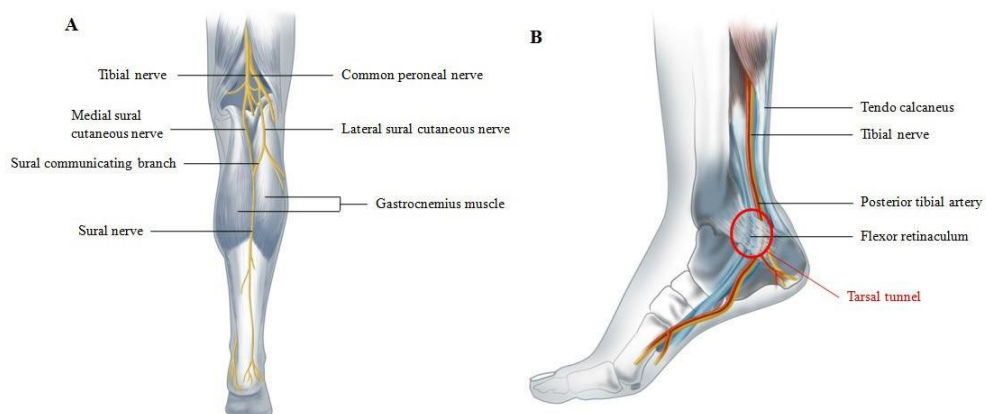


Figure 1 (A) THE COURSE OF THE SURAL NERVE IN THE POSTERIOR COMPARTMENT OF THE LEG. (B) THE COURSE OF THE TIBIAL NERVE IN THE MEDIAL ASPECT OF THE ANKLE.

Proximal tibial neuropathy has been reported to occur in the popliteal fossa. This may happen because the tibial nerve passes both over the popliteus muscle and under an arch formed from the tendons of the soleus muscle, and it is therefore vulnerable in this location (Beltran *et al.*, 2010). However, in athletes, trapping of the tibial nerve most often occurs in the distal limb at the foot or ankle (McKean, 2009). It has been reported that in elite athletes it is common to find tibial nerve damage owing to trapping, as the distal branches pass through the tarsal tunnel (Nakano, 1978; Donnell & Barrett, 1991; McCrory *et al.*, 2002) (Figure 1B). Nerve compression may occur due to tibial nerve tension with inward rotation of the talocalcaneal joint. Injury to

the tarsal tunnel or tissues therein may result from repeated plantar flexion and dorsiflexion of the ankle when running (Daniels *et al.*, 1998; McKean, 2009). Thus, in sports where the athletes run for extended periods, tarsal tunnel damage may occur. If the mechanical characteristics of an individual athlete are suboptimal when running, this will increase the likelihood of neuronal damage in the tarsal tunnel region (Colak *et al.*, 2005).

Didehdar *et al.* (2014) reported delayed motor and sensory conduction velocity of the common peroneal and tibial nerves in healthy football players compared with controls. Ka men *et al.* (1984), studying marathon runners, reported delayed posterior tibia I nerve conduction velocity compared with non-athletes. In this study, prolonged conduction velocity of the tibia I nerve in the players in their dominant leg compared with their non-dominant leg was evident, whereas there was no difference between the players and control groups for either leg. From this data, we hypothesise that excessive biomechanical demands on the tibial nerve during running could be an aetiologic factor for decreased conduction velocity in the players' dominant extremity. In football, players tend to make short sprints (10 to 30 yards, equivalent to 9 to 27 m) regularly during the game so that this type of sprint occurs every 45 to 90 seconds. Kirkendall (2011) has reported that there is an increase in physiological load of 15% when running at any speed and dribbling a ball. Thus foot position and increased tension on the tibial nerve during running, which is exacerbated by running with the ball, maybe aetiological factors in the delayed nerve conduction velocity of the dominant leg in players.

Decreased muscle action potential amplitudes of the tibia I nerve was found in the dominant limbs of players when compared with corresponding limbs of controls in this study ($p=0.026$). This has been reported previously in the upper limbs of uninjured athletes (Pawlak & Kaczmarek, 2010). These findings of lower amplitude in athletes may be attributed to training-induced changes in peripheral nerves. However, in contrast to these findings, Sharma *et al.* (2017) found increased tibial muscle action potential amplitudes in football players. Pawlak and Kaczmarek (2010) suggested that these differences in findings may be due to the different sports, and therefore different training regimes and limb usage during play. We believe that additional neurophysiological studies are needed to evaluate the effects of endurance-type training on peripheral nerves of the lower limb, which may need to take the sport practised by the individual athlete into account.

The motor conduction velocity, distal latency and amplitude of the common peroneal nerve was the same in both the players and the control subjects. A slight difference in nerve conduction velocity between the dominant limb of players and the corresponding limbs of controls was observed, although this was not significant. The usual location for injury to the common peroneal nerve is adjacent to the fibular neck and the injury type is usually either compression or traction. Repetitive ankle inversion and pronation, generalised ligamentous laxity, and genu varum may all lead to traction-type injury (Peck *et al.*, 2010; Meadows & Finnoff, 2014). Additionally, this nerve is susceptible to traumatic injury due to its superficial and exposed location in the leg. Thus in contact sports, including football, traumatic injuries have been reported to occur (Lorei & Hershman, 1993). Several studies have reported asymptomatic electrophysiological abnormalities of the common fibular nerve following ankle sprains (Nitz *et al.*, 1985; Benchortane *et al.*, 2011). Jazayeri-Shooshtari *et al.* (2007) reported that in football players following ankle sprain, nerve conduction velocity of the deep peroneal and tibial nerves showed a significant decrease in comparison with healthy football players.

Although a decrease in nerve conduction velocity in football players might be expected, it did not differ between players and controls in our study. This is probably due to an anatomic feature of the nerve. As the peroneal nerve has only one muscular branch to the short head of the biceps femoris muscle this may affect the accuracy of motor conduction velocity measurements of the common peroneal nerve.

Earlier studies have investigated the effects of strength and endurance training on peripheral nerves. These have suggested that there is an adaptive effect that enables motor neurones to continue to function as demand increases. It has been observed that intense, repetitive physiologic exercise can result in morphological adaptive changes in peripheral nerves, such as increased neuronal size, which affected conduction velocity (Roy *et al.*, 1983; Sleivert *et al.*, 1995). Elam and Barth (1986) investigated football players in college. They reported no relationship between muscle strength and conduction velocity. However, there was an inverse association between conduction velocity and vertical jumping ability. They hypothesised that this was due to an adaptive increase in the number of terminal neurones which would innervate a greater proportion of the muscle but, at the same time, reduce conduction velocity by greater dispersion of the signal. This hypothetical mechanism may account for the non-significant difference in conduction velocity found in our cohort of footballers when compared with untrained controls. There is a need for neurophysiological parameters evaluated both at the beginning and after a long and intensive training period to confirm the evolving adaptive changes specific to football training in elite football players.

CONCLUSIONS

In this cohort of elite-level footballers, both sural and tibial nerves exhibited abnormalities of function when compared with a control group. The most significant finding was slower conduction velocity and lower amplitude of the sural nerve in both dominant and non-dominant extremities of football players when compared with untrained subjects. These may indicate either presymptomatic or asymptomatic neuropathies or an adaptation to chronic intense training required to achieve elite status in this sport. Informing the trainers about these subclinical conditions may be beneficial to plan and organise specific training and rehabilitation programmes for the players in order to prevent injuries.

Conflict of interest

No conflict of interest declared

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