ORIGINAL ARTICLE

Nerve conduction studies of upper extremities in tennis players

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Br J Sports Med 2004;**38**:632–635. doi: 10.1136/bjsm.2003.008029

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Received 11 September 2003
Accepted for publication 14 September 2003

Objectives: The influence of regular and intense practice of an asymmetric sport such as tennis on nerves in the elbow region was examined.
Methods: The study included 21 male elite tennis players with a mean (SD) age of 27.5 (1.7) years and 21 male non-active controls aged 26.4 (1.9) years. Anthropometric measurements (height, weight, limb length, and perimeters of arm and forearm) were determined for each subject, and range of motion assessment and radiographic examination carried out. Standard nerve conduction techniques using constant measured distances were applied to evaluate the median, ulnar, and radial nerves in the dominant and non-dominant limb of each individual.
Results: The sensory and motor conduction velocities of the radial nerve and the sensory conduction velocity of the ulnar nerve were significantly delayed in the dominant arms of tennis players compared with their non-dominant arms and normal subjects. There were no statistical differences in the latencies, conduction velocities, or amplitudes of the median motor and sensory nerves between controls and tennis players in either the dominant or non-dominant arms. However, the range of motion of the upper extremity was significantly increased in tennis players when compared with control subjects. Tennis players were taller and heavier than control subjects and their dominant upper limb lengths were longer, and arm and forearm circumferences greater, than those of the control subjects.
Conclusions: Many of the asymptomatic tennis players with abnormal nerve conduction tests in the present study may have presymptomatic or asymptomatic neuropathy similar to subclinical entrapment nerve neuropathy.

Tennis is one of the most widely played sports in the world, is physically demanding, and imposes a high impact and weight bearing load upon the upper extremity. Repetitive stress on the dominant extremities of tennis players is responsible for physiological and pathological changes in the dominant elbow.[1–6] Peripheral nerves of the upper extremity are exposed to acute and chronic mechanical injuries in the athlete because of the excessive physiological demands. Various factors such as high repetition of motions, high muscular forces, and extreme elbow positions affect the peripheral nervous system with or without signs and symptoms. Identification of nerve injuries requires an understanding of their common sites in tennis.

This study was performed to evaluate the effect of playing tennis on the radial, ulnar, and median nerves crossing the elbow region.

METHODS

The group of tennis players consisted of 21 male (mean (SD) age 27.15 (1.7) years) subjects training for about 4 h/day since the age of 12–15. The reference group consisted of 21 non-active male (age 26.4 (1.9) years) subjects not participating in any kind of regular or organised sporting activity. Each individual was initially screened for any history, signs, or symptoms of either peripheral neuropathy or compression syndrome of the upper extremities.

The neurophysiological study consisted of motor and sensory nerve conduction studies of the median, ulnar, and radial nerves. All studies were performed with the subject in a supine position in a warm room with the temperature maintained at 26–28°C. The skin temperature of the upper limb was checked to eliminate the influence of temperature on the conduction parameters and, if necessary, the limb was warmed with the aid of an infra red heat lamp to maintain a temperature of 32°C or greater. Intercathodal distances were measured with an anthropometer. Both the dominant and non-dominant extremities of all subjects were tested by a neurologist using a Medelec Saphire Electromyograph (Medelec, Woking, UK). Nerve conduction studies were performed using standard techniques of supramaximal percutaneus stimulation with a constant current stimulator and surface electrode recording on both extremities of each subject. Sensory responses were obtained by antidromically stimulating at the wrist and recording from the index finger (median nerve) and little finger (ulnar nerve) with ring electrodes. Sensory responses of the radial nerve were obtained by antidromically stimulating at the middle of the forearm next to the cephalic vein and recording with a disc electrode between the first and second fingers.

The median motor nerve was examined by stimulating the median nerve at the wrist (between the tendons of the flexor carpi radialis and palmaris longus), the elbow (next to the brachial artery), and the upper arm (next to the brachial artery). The nerve was stimulated with bipolar surface electrodes and the recording was carried out over the abductor pollicis brevis muscle with surface electrodes.

The ulnar motor nerve was examined by stimulating the ulnar nerve at the wrist, below the elbow (about 4–5 cm below the medial epicondyle), and above the elbow (1–6 cm above the tip of the medial epicondyle) with bipolar surface electrodes. The motor response was recorded from the abductor digiti minimi muscle with surface electrodes.

The radial motor nerve was examined by stimulating the radial nerve in the region of the elbow (about 5 cm proximal to the lateral epicondyle of the humerus), in the dorsum of the forearm (about 8 cm proximal to the styloid process of ulna), and in the arm (about 4 cm posterior to the insertion of the deltoid muscle). Bipolar surface electrodes were used

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Table 1 Comparison of the biometric data of professional tennis players and sedentary non-athletic subjects

Characteristics		Normal	Tennis	p
Age (years)		26.46 (0.5)	27.56 (0.7)	0.123
Height (cm)		174.9 (0.3)	176.1 (0.6)	0.095
Weight (kg)		72.26 (1.2)	74.65 (1.4)	0.057
Perimeter forearm (cm)	Dominant	25.5 (1.4)	28.7 (1.3)	**0.03**
	Non-dominant	24.9 (1.3)	25.1 (1.4)	0.255
	p	0.06	**0.03**	
Perimeter arm (cm)	Dominant	29.4 (2.0)	31.9 (1.2)	**0.02**
	Non-dominant	28.4 (1.9)	28.2 (1.0)	0.349
	p	0.06	**0.01**	
Length upper extremity (cm)	Dominant	72.63 (0.8)	73.03 (0.9)	0.491
	Non-dominant	72.50 (1.8)	71.92 (0.7)	0.552
	p	0.627	**0.03**	

Values are given as mean (SD). Significant p values are shown in bold.

to stimulate the radial nerve. Recording was carried out over the brachioradialis muscle using surface disc electrodes.

In the present study, the following median and radial nerve measures were used: (i) baseline to peak amplitude of the sensory nerve action potential (Amp-S); (ii) distal peak latency of the sensory nerve action potential (DL-S); (iii) conduction velocity of the sensory nerve fibres (CV-S); (iv) baseline to peak amplitude of the compound muscle action potential (Amp-M); (v) distal onset latency of the compound muscle action potential (DL-M); and (vi) conduction velocity of the motor nerve fibres (CV-M).

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, height, and limb lengths. We measured the length of the upper limbs as the distance between the acromial angle and the tip of the third digit of the hand, when the shoulder was flexed to 90° with the elbow extended. Perimeters of arm and forearm were measured to allow comparison between the dominant side and the non-dominant side. Using a tape measure, we obtained the perimeter of the forearm 10 cm proximal to the styloid process of the ulna and the perimeter of the arm 10 cm proximal to the medial epicondyle of humerus.

The range of motion of the upper extremities was evaluated by using a Biodex System 3 (Biodex Medical Systems, Shirley, NY, USA). Comparative plain films of both elbows were obtained from players in an anteroposterior projection to determine the radiographic manifestations of musculoskeletal stress at the elbow associated with tennis.

All subjects were informed of the study procedure, purposes, and known risks, and all gave their informed consent. This study was conducted according to the guidelines of the Declaration of Helsinki and approved by the ethics committee of our faculty.

When performing nerve conduction studies, and during the assessment of movement and radiological findings, the observers were not blinded to the subjects' tennis playing habits.

The results are presented as means (SD). Differences between the groups were calculated using a non-parametric test for independent samples (Mann-Whitney U test). The SPSS package (SPSS for Windows v 11.0, SPSS, Chicago, IL, USA) for personal computers was used for the statistical analyses. A p value of <0.05 was considered significant.

RESULTS

Two groups of subjects were examined. Their biometric characteristics are given in table 1. The tennis players were slightly larger and heavier, but as the difference was small this is not significant. The perimeters of the arm and forearm were chiefly influenced by muscle mass. A very marked hypertrophy was observed in all the tennis players in the dominant arm and forearm.

We observed significant differences in upper extremity length between the two arms in the tennis players. Anteroposterior roentgenograms of the elbows of tennis players showed a highly significant hypertropy of bone in response to exercise. Visual inspection of the radiographs of tennis players revealed an obvious difference in bone structure between the stroke arm and the non-dominant arm; bone development was clearly greatly increased in the dominant extremity. There were no bony changes such as callus formation, arthritic spurs, fracture fragments, or changes in carrying angle.

In this study the range of motion of the upper extremities of the tennis players was clearly increased except for elbow flexion. Measurements showed significant differences in hyperextension, adduction, internal and external rotation degree of the shoulder, and pronation and supination of the forearm, and in flexion, extension, and ulnar and radial deviation of the wrist between tennis players and control subjects in the dominant extremity. Ranges of motion values of the upper extremity are shown in table 2.

There were no statistical differences in conduction velocities, latencies, or amplitudes of the median motor and sensory nerves between the control group and tennis players in both dominant and non-dominant arms (table 3).

The sensory and motor conduction velocities of the radial nerve were significantly delayed in the dominant arms of tennis players compared with their non-dominant arms and with normal subjects (table 4).

Table 2 Range of motion of upper extremity (dominant arm)

	Normal	Tennis	p
Shoulder flexion	180.0 (0.0)	180.0 (0.0)	1.000
Shoulder hyperextension	54.1 (4.1)	57.5 (6.1)	**0.000**
Shoulder abduction	179.8 (2.4)	180.0 (0.0)	0.317
Shoulder adduction	38.9 (7.7)	43.3 (3.8)	**0.01**
Shoulder internal rotation	64.4 (7.6)	70.6 (2.9)	**0.000**
Shoulder external rotation	84.2 (6.6)	89.9 (3.1)	**0.000**
Elbow flexion	144.1 (4.5)	133.6 (5.3)	**0.000**
Forearm supination	78.3 (9.7)	84.3 (3.0)	**0.000**
Forearm pronation	78.8 (9.3)	84.3 (0.2)	**0.000**
Wrist flexion	85.8 (4.4)	89.7 (1.7)	**0.000**
Wrist extension	64.6 (5.3)	68.9 (4.8)	**0.01**
Wrist ulnar deviation	27.6 (3.2)	29.2 (6.9)	**0.009**
Wrist radial deviation	18.8 (1.3)	20.0 (0.0)	**0.006**

Values are given as mean (SD).
Significant p values are shown in bold.

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Table 3 Nerve conduction results of the median nerve

	Normal		Tennis	
	Dominant	Non-dominant	Dominant	Non-dominant
Amp-S (μv)	47.6 (6.9)	46.9 (7.3)	45.8 (5.9)	45.1 (6.5)
DL-S (ms)	2.8 (0.15)	3.01 (0.18)	2.9 (0.14)	3.03 (0.22)
CV-S (m/s)	54.4 (3.12)	53.8 (0.18)	55.1 (3.06)	54.3 (5.8)
Amp-M (mV)	8.8 (2.5)	9.1 (2.3)	8.7 (2.4)	9.2 (3.2)
DL-M (ms)	3.28 (0.19)	3.31 (0.22)	2.99 (0.2)	3.2 (0.21)
CV-M (m/s)	57.5 (0.17)	58.3 (0.23)	59.84 (3.64)	58.33 (0.2)

Values are mean (SD).

The distal latencies (motor and sensory) of the radial nerve in the tennis players were significantly prolonged compared with control subjects ($p<0.05$) (table 4).

Latencies of the ulnar sensory nerve potential were considerably greater and conduction velocities were smaller in the tennis players' dominant arms compared with the controls ($p<0.05$) (table 5).

Although reaching statistical significance between the groups, nerve conduction values within the tennis playing population fell within the normal range.

DISCUSSION

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. Several authors have discussed the effect of regular and intense practice of an asymmetric sport, such as tennis, on player morphology. Exercise and loading promotes bone mineral acquisition and changes bone geometry (bone width, and cortical wall thickness and strength) in the dominant arm as compared with the contralateral arm in tennis players.[1–7]

In this study roentgenographic evaluation of the upper extremities of tennis players showed obvious hypertrophy of the humerus, radius, and ulna in the dominant arm. This was accepted as a natural response to exercise. Differences in the length of the dominant extremity of tennis players as observed in this study have been described before and can be regarded as a result of the specific strain induced by tennis practice and as a biopositive adaptive reaction; mechanical strain and hyperaemia seem to be able to stimulate the germinative cell layer of the epiphyseal plate in some way.[3 8]

Physical exercise causes hypertrophy in the muscles involved. The perimeters of the arm and forearm were principally influenced by muscle hypertrophy in this study. A very marked hypertrophy was measured in all the tennis players in the dominant forearm and arm. We were surprised to find a significant range of motion decrease in elbow flexion in tennis players and suggest that hypertrophy of the elbow flexors was the possible reason. In the tennis players, the range of motion of other upper extremity joints was clearly increased in the dominant limb.

Nerve entrapment syndromes can occur in athletes. Many neurological injuries remain subclinical and are not recognised before neurological damage is permanent. Our results demonstrated that the sensory and motor conduction velocities of the radial nerve were significantly delayed in the dominant arms of tennis players compared with their non-dominant arms and with control subjects. We suggest that repetitive motion and overload of wrist extensors and the supinator muscle are major etiological factors in the delayed radial nerve motor and sensory conduction velocity. Forceful repetitive movements may cause stretching or impingement of the radial nerve within unyielding musculotendinous compartments followed by fascial thickening.

Table 4 Nerve conduction results of the radial nerve

	Normal		Tennis	
	Dominant	Non-dominant	Dominant	Non-dominant
Amp-S (μv)	11.4 (13.45)	12.9 (12.5)	12.8 (11.7)	11.7 (37)
DL-S (ms)	2.91 (11.5)	3.40 (0.15)	3.98 (8.8)*	3.1 (10.5)
CV-S (m/s)	64.4 (9.71)	63.7 (8.9)	60.4 (6.7)*	62.82 (5.0)
Amp-M (mV)	11.2 (2.4)	12.6 (7.8)	11.81 (0.2)	12.1 (3.8)
DL-M (ms)	2.60 (3.13)	2.95 (2.5)	3.22 (2.6)*	2.81 (6.2)
CV-M (m/s)	68.61 (0.19)	69.3 (0.22)	66.80 (3.68)*	68.73 (5.2)

Values are mean (SD).
*$p<0.05$.

From an anatomical point of view, the inseparable origin of the wrist extensors and supinator muscle seems to link tennis elbow with radial tunnel syndrome. Compression of the deep branch of the radial nerve during its passage through Frohse's arcade in the supinator muscle is one of the classical explanations advanced for epicondylalgia.[9 10]

Rath *et al* defined the anatomy of the deep branch of the radial nerve and indicated that it is most affected during its passage through the supinator muscle because of the marked fibrous thickening of the perineurium and the interstitial connective tissue. They founded that the nerve is also closely related to the radial head around which it winds in supination and extension. Safran indicated that radial nerve entrapment is aggravated by active supination and passive pronation as well as by resisted extension of the long finger while the elbow is extended depending on the site of entrapment.[10–12]

Studies show that wrist extensors are highly involved in all strokes in tennis.[13 14] In our opinion if the extensor group and supinator muscle are at near maximum contraction, stiffness and overload of the muscles will cause repeated microtrauma to the radial nerve during its course. Repetitive motion and overuse of the upper extremity make the tennis player vulnerable to radial nerve disorders.

Spontaneous entrapment of the superficial radial nerve in the forearm in the region where the nerve passes to a subcutaneous position has occasionally been reported. In 15 of the tennis players, the radial sensory and motor conduction velocities were slower than in the control subject in this study. These results show that competitive tennis players have a tendency to radial nerve damage although they were asymptomatic. We think that dynamic pressure on the radial

Table 5 Nerve conduction results of the ulnar nerve

	Normal		Tennis	
	Dominant	Non-dominant	Dominant	Non-dominant
Amp-S (μv)	27.8 (11.3)	26.9 (9.6)	25.8 (12.8)	25.2 (5.3)
DL-S (ms)	2.17 (0.25)	2.62 (0.29)	3.31 (7.2)*	2.69 (0.36)
CV-S (m/s)	52.7 (4.8)	54.1 (5.1)	50.7 (5.8)*	54.7 (5.2)
Amp-M (mV)	5.3 (2.2)	5.8 (2.0)	5.4 (1.8)	5.6 (2.3)
DL-M (ms)	3.3 (0.37)	3.01 (0.36)	2.9 (0.38)	2.98 (0.27)
CV-M (m/s) (below elbow to wrist)	56.8 (5.3)	57.2 (5.4)	57.6 (5.2)	57.9 (5.5)
CV-M (m/s) (above elbow to below elbow)	58.6 (5.4)	58.7 (5.6)	57.4 (5.1)	58.0 (5.4)

Values are mean (SD).
*$p<0.05$.

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nerve is the result of delayed motor and sensory nerve conduction velocity.

The elbow is also the most common site for ulnar nerve compression. Potential compressive sites include the arcade or ligament of Struthers, the medial intermuscular septum, the cubital tunnel, and the medial head of the triceps. Normally, the ulnar nerve is subjected to stretch and compression forces that are moderated by its ability to glide in its anatomic path around the elbow. When normal excursion is restricted, irritation ensues. This results in a cycle of perineural scarring, further loss of excursion, and progressive nerve damage. The peripheral location of the motor and the sensory fibres may explain the usual clinical presentation in cases of early compression.[15–18]

In this study, we found that the sensory conduction velocity of the ulnar nerve was significantly delayed in the tennis players' dominant arms. In the tennis players the elbow is subjected to valgus stresses during the throwing motion which cause increased force to the medial elbow. We suggest that the valgus extension overload leads to possible subclinical pathologies in the ulnar nerve.

Another factor leading to dynamic compression of the ulnar nerve is elbow flexion. In clinical studies, increased cubital tunnel pressures with elbow flexion have been reported. The decrease in canal volume with elbow flexion is consistent with findings of increased intraneural and extraneural pressures within the canal. On flexing the elbow, ulnar nerve traction and excursion occurs because the course of the nerve is behind the axis of rotation of the elbow (nerve compression in this region explains why so many patients with ulnar nerve lesions at the elbow have no abnormality of the joint itself and no history of injury). Excursion of the nerve at the elbow is further increased when the shoulder is placed in abduction and the wrist in extension. These traction forces on the ulnar nerve are increased in situations where there are valgus forces, as in throwing.[15 19–21] We propose that because of the superficial location of the ulnar nerve, excursion of the nerve at the elbow with repetitive motion, abnormal elbow mechanics, and overuse of the triceps muscle in playing tennis may initiate the injury. Sensory fibres may be affected earlier than motor fibres so sensory conduction studies using ulnar neuropathy might be more useful than motor studies in early diagnosis. The high prevalence of decreased ulnar nerve conduction without corresponding symptoms may suggest subclinical entrapment neuropathy.

In this study we did not find statistical differences in conduction velocities of the median motor and sensory nerves between tennis players and control subjects in either dominant or non-dominant arms, because the median nerve is not in a vulnerable position as it lies through the cubital fossa and is not easily damaged in the elbow region. Entrapment of the median nerve in the proximal forearm is considered a rather rare condition. Median nerve entrapment may occur at the distal humerus when the rarely present ligament of Struthers connects an anomalous bony spur of the humeral shaft to its medial epicondyle.[22] Our study showed that the median nerve has no predisposition to nerve entrapment in the elbow region during a game of tennis.

Several authors have performed nerve conduction studies on patients with tennis elbow. We could find no studies in the literature suggesting electrodiagnostic abnormalities in asymptomatic tennis players. This study is the first reported observation of delayed motor and sensory conduction velocity of the radial nerve and sensory conduction velocity of the ulnar nerve in competitive tennis players' dominant arms compared with control subjects. The structural and functional changes may have a crucial effect on the radial and ulnar nerves in this study. Large numbers of neurological injuries remain subclinical and are not identified before damage is irreversible.

If the biomechanical and physiological stresses inherent in the game of tennis have been correctly analysed and understood, the clinician can rehabilitate the patient, plan a preventive conditioning program, and modify biomechanics scientifically.

Take home message

Many neurological injuries remain subclinical and are not identified before damage is irreversible. However, the clinician can rehabilitate the patient, plan a preventive conditioning program, and modify biomechanics scientifically.

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Conflict of interest: none declared.

This study was presented at the ''2nd European Congress of Sport Traumatology'' which was held in Monaco, May 1–3, 2003.

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