

Nerve conduction studies of the axillary, musculocutaneous and radial nerves in elite ice hockey players

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Aim. The peripheral nerves of the upper extremity are exposed to acute and chronic mechanical injuries in ice hockey players, because of the high repetition of motions, high muscular forces and extreme shoulder positions. This study was performed to evaluate the effect of ice hockey playing on the axillary, musculocutaneous and radial nerves crossing the upper arm region.

Methods. The ice hockey group consisted of 20 first-division male ice hockey players and the control group consisted of 20 non-active males. The neurophysiological study consisted of motor nerve conduction latency of the axillary, musculocutaneous and radial nerves.

Results. The values of distal motor latency (DML) of the axillary, radial and musculocutaneous nerves were significantly prolonged in the ice hockey players compared with the controls. Ice hockey can repetitively stress the upper extremity during shooting, because of forceful throwing to move the puck from the stick blade to the opponent's net. The mechanism of prolonged DML in the axillary nerve may both tract and compress as the axillary nerve stretches across the humerus during movement. From this study it emerged that using the biceps and coracobrachialis muscles can create notable muscle compartment pressure on the musculocutaneous nerve. The significant differences detected in the neurophysiologic study of the musculocutaneous nerve between the ice hockey players and controls may reflect the fact that the forces acting on the shoulder and the elbow during ice hockey matches can effectively influence DML.

Conclusion. The authors suggest two plausible causes for

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prolonged radial nerve DML: direct compression by the hypertrophied triceps muscle and stretching of the arm. The study suggests it is likely that a combination of skeletal muscle hypertrophy and excessive biomechanical demands on neurological structures during ice hockey matches may be a major etiologic factor in compression of the axillary, musculocutaneous and radial nerves.

Key words: Hockey - Nerve expansion - Motor activity.

Ice hockey is a fast collision sport with a high risk of injury. Muscle imbalance and structural asymmetry are common in ice hockey players due to the frequent rotational forces and collisions to which they are subjected.¹ Nerve injuries remain one of the most serious complications of contact sports.² There is an intrinsic risk for injury in ice hockey, with many injuries potentially affecting the nervous system.³ In ice hockey players, nerve injuries most commonly consist of concussions and spinal cord injuries, while peripheral nerve injuries are much less common.⁴ A few cases have been described of ice hockey players with peripheral neuropathies involving the axillary nerve,² peroneal nerve,⁵ medial and lateral plantar nerves⁶ and sural nerve.⁷

The glenohumeral joint is a potential area for nerve lesions, because it may be influenced by the dynamics of the shoulder girdle. Peripheral nerve injuries about the shoulder are being recognized with increasing frequency in athletes. The natural history of these injuries make it important to treat the athlete. Ice hockey players may be at risk of neurovascular injury to the shoulder as a result of strenuous activity of the upper extremity.²⁻¹⁰ The peripheral nerves of the upper extremity are exposed to acute and chronic mechanical injuries in ice hockey players, because of frequent repetition of motions, high muscular forces and extreme shoulder positions. Although these injuries present as distinct clinical syndromes, the signs and symptoms may remain subclinical. Identification of nerve injuries requires an understanding of the types and common sites unique to each sport. Therefore, this study was performed to evaluate the effect of playing ice hockey on the axillary, musculocutaneous and radial nerves crossing the upper arm region, as well as to evaluate whether or not the upper extremity movements of the ice hockey players have a compressive effect on the nerves.

Material and methods

The ice hockey group consisted of 20 first-division male ice hockey players (age 23.7 ± 7.32 years). They had been active in high-level ice hockey for 4.7 (2-8) years, and practiced 8 h/week. The control group consisted of 20 non-active males (age 24.6 ± 8.35 years) not participating in any kind of regular or organized sport activity. The controls were recruited from among students of the School of Medicine. Each subject was initially screened for any history, signs or symptoms of either peripheral neuropathy or compression syndrome of the upper extremities. None in either group had a history of previous severe trauma to the upper extremity. Before participation, informed consent was obtained from each subject. All were examined at the Kocaeli University Hospital Turkey.

The neurophysiological study consisted of motor nerve conduction of the axillary, musculocutaneous and radial nerves. The nature of the procedure was explained to the subjects. They had to lay supine on a padded table with the upper limb supported. All studies were performed in a warm room maintained at 26-28 °C. The skin temperature of the upper limb was checked to eliminate its influence on conduction para-

meters. If necessary, the limb was warmed with the aid of an infra-red heat lamp to maintain a temperature of 32 °C or more. In an effort to reduce diurnal variation, all measurements were made at approximately the same time of the day for each subject. Intercathodal distances were measured with an anthropometer. Both the dominant and non-dominant extremities of the subjects were tested by a neurologist using a Neuropack M1, MEB-9204K (Nihon Kohden, Japan). The electrophysiologic study was conducted according to the American Association of Electrodiagnostic Medicine practice guidelines.

The nerve conduction studies were performed using standard techniques of supramaximal percutaneous stimulation with a constant current stimulator and surface electrode recording on both extremities of each subject. Distal motor latency was selected to assess the effects on axillary, radial and musculocutaneous nerves motor conduction properties. Generally, measurement of DML is important in diagnosing peripheral neuropathy. Longer DML reflects worse nerve dysfunction.

The axillary motor nerve was examined by stimulating Erb's point (supraclavicular fossa, just posterior to the sternocleidomastoid muscle). The nerve was stimulated with bipolar surface electrodes, and the recording was carried out over the deltoid muscle with surface electrodes. A surface reference electrode was placed distally over the deltoid tendon.

Motor conduction of the musculocutaneous nerve was performed by stimulating Erb's point with bipolar surface electrodes. Motor response was recorded from the biceps muscle. A surface reference electrode was placed distally over the biceps tendon.

The radial motor nerve was examined by stimulating Erb's point. The nerve was stimulated with bipolar surface electrodes, and the recording was carried out over the triceps muscle with surface electrodes. A surface reference electrode was placed distally over the triceps tendon.

DML was calculated from the start of the electrical artefact to the first positive peak of the nerve potential. Radial motor nerve conduction latency was measured between the Erb point and the triceps muscle. The DML values were calculated both below (dominant arms $N=21$, non-dominant arms $N=21$) and above (dominant arms $N=19$, non-dominant arms $N=19$) the midpoint of the mean distance (33 cm) separately. The DML values of the axillary nerve were measured

between the Erb point and the deltoid muscle. The DML values were calculated both below (dominant arms N.=20, non-dominant arms N.=23) and above (dominant arms N.=20, non-dominant arms N.=17) the midpoint of the mean distance (18 cm) separately. Musculocutaneous motor nerve conduction latency was measured between the Erb point and the biceps muscle. The DML values both below (dominant arms N.=20, non-dominant arms N.=23) and above (dominant arms N.=20, non-dominant arms N.=17) the midpoint of the mean distance (26 cm) were calculated separately.

Simple biometric measurements were also carried out. The groups were matched according to weight, height, and limb length. Upper limb length was measured 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 and the non-dominant side. Using a tape measure, 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 were obtained.

Data were analyzed using SPSS for Windows Version 15.00. A P value <0.05 was considered statistically significant. After tests for normality, statistical significance was calculated using an independent sample t test for normally distributed data and the Mann-Whitney U test for data not normally distributed.

The study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Ethics Committee of Kocaeli University.

Results

Comparison of the biometric data of the ice hockey players and the non-athletes (controls) is given in Table I. There were no statistical differences in age or height between the controls and the ice hockey players. The ice hockey players were heavier than the controls (P=0.005). The perimeters of the arm and forearm were chiefly influenced by muscle mass. A very marked hypertrophy was observed in all the ice hockey players in both the dominant and the non-dominant arm and forearm compared with the controls. There were no significant differences between the controls and the ice hockey players with respect to upper extremity length of both the dominant and non-dominant side.

In the ice hockey players the axillary DMLs were significantly prolonged compared with the controls in both the dominant and non-dominant arms in subjects with an Erb-deltoid distance longer than 18 cm. There were no statistical differences in motor action potential distal latency of the axillary nerve between the controls and the ice hockey players in either the dominant or the non-dominant arms below 18 cm (Table II).

The musculocutaneous nerve DMLs of the dominant arms of the ice hockey players were significantly prolonged in comparison to those of the non-dominant arms of the controls in subjects with an Erb-biceps distance shorter than 26 cm. The DMLs of the musculocutaneous nerve were not significantly different in the dominant arms of the ice hockey players compared with the dominant arms of the controls above 26 cm.

TABLE I.— Comparison of the biometric data of professional ice hockey players and sedentary non-athletic subjects.

Characteristics	Control	Ice hockey	P
Age (years)	22.50 (1.3)	21.90 (1.8)	0.383
Height (cm)	177.2 (3.6)	181.2 (7.7)	0.091
Weight (kg)	68.75 (6.4)	77.97 (11.1)	0.005
Perimeter forearm (cm)	Dominant	22.3 (1.8)	25.4 (2.7)
	Non-dominant	22.1 (1.8)	25.1 (2.5)
	P	0.718	0.659
Perimeter arm (cm)	Dominant	26.3 (1.8)	31.3 (2.1)
	Non-dominant	27.9 (2.1)	31.1 (2.1)
	P	0.620	0.718
Length upper extremity (cm)	Dominant	78.27 (3.8)	78.7 (1.5)
	Non-dominant	78.25 (1.5)	78.8 (0.7)
	P	0.527	0.767

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

TABLE II.—Mean distal latency(ms) results of the axillary nerve.

Distance (erb-deltoid)	Dominant		P	Non-dominant		P
	Controls (N=20)	Ice hockey players (N=20)		Controls (N=20)	Ice hockey players (N=20)	
<18 cm	3.84±0.31 (N=10)	3.66±0.48 (N=10)	0.472	3.69±0.22 (N=12)	3.72±0.39 (N=11)	0.375
≥18 cm	3.97±0.34 (N=10)	4.33±0.30 (N=10)	0.034*	4.07±0.29 (N=8)	4.57±0.36 (N=9)	0.024*

Values are given as mean±SD. *Significantly different than the dominant and non-dominant arms of the controls.

TABLE III.—Mean distal latency(ms) results of the musculocutaneous nerve.

Distance (erb-deltoid)	Dominant		P	Non-dominant		P
	Controls (N=20)	Ice hockey players (N=20)		Controls (N=20)	Ice hockey players (N=20)	
<26 cm	4.12±0.26 (N=12)	4.39±0.26 (N=8)	0.045*	4.20±0.34 (N=14)	4.48±0.36 (N=9)	0.073
≥26 cm	4.53±0.46 (N=8)	4.66±0.52 (N=12)	0.316	4.68±0.26 (N=6)	4.67±0.50 (N=11)	0.58

Values are given as mean±SD. *Significantly different than the dominant and non-dominant arms of the controls.

TABLE IV.—Mean distal latency (ms) results of the radial nerve.

Distance (erb-deltoid)	Dominant		P	Non-dominant		P
	Controls (N=20)	Ice hockey players (N=20)		Controls (N=20)	Ice hockey players (N=20)	
<33 cm	4.18±0.24 (N=10)	4.73±0.31 (N=11)	0.002*	4.22±0.44 (N=11)	4.87±0.37 (N=10)	0.007*
≥33 cm	4.75±0.30 (N=10)	5.15±0.39 (N=9)	0.066	4.84±0.42 (N=9)	5.33±0.37 (N=10)	0.014*

Values are given as mean±SD. *Significantly different than the dominant and non-dominant arms of the controls; *significantly different than the non-dominant arm of the controls.

There were no statistical differences between the controls and the ice hockey players in the non-dominant arms in subjects with an Erb-biceps distance below and above 26 cm (Table III).

The DMLs of the radial nerve were significantly prolonged in the ice hockey players in both the dominant and non-dominant sides compared with the controls in subjects with an Erb-triceps distance below and above 33 cm (Table IV).

Although nerve conduction values reached statistical significance between the two groups, the values fell within the normal range in the ice hockey players.

Discussion

Overuse injuries occur over time rather than after a single, identifiable event. The cumulative effect of submaximal forces leads to injury. The injury mech-

anism for overuse conditions is more likely related to the history of activity over a period of time.¹¹ These injuries are thought to be due to repetitive application of relatively small loads over many repetitive cycles.^{12, 13}

The peripheral nerves are susceptible to injury in athletes, because of the excessive physiological demands made on both the neurological structures and the soft tissues that protect them. In sports medicine, most peripheral nerve injuries of the shoulder are first degree injuries, or neuropraxias, consisting of a conduction block in the presence of intact neural elements, including the axons and their connective tissue sheaths.¹⁴ While most acute and catastrophic neurological injuries are usually obvious, many remain subclinical and are not recognised before neurological damage is permanent.⁸ Definition of nerve injuries requires a knowledge of the peripheral nervous system and an understanding of the types and common sites

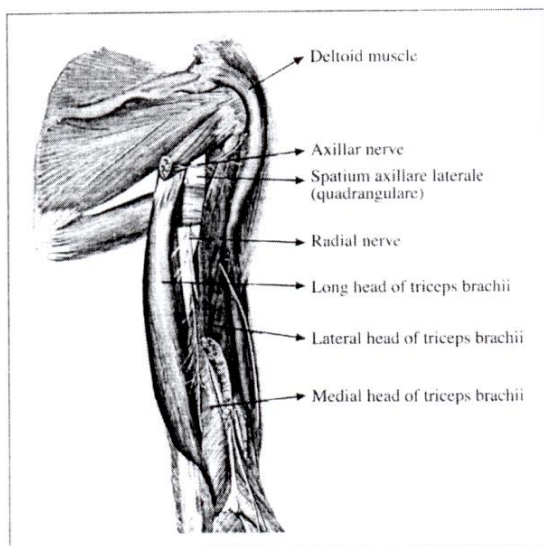


Figure 1.—Drawing of posterior view of upper arm region. Lateral and long head of the triceps brachii has been divided to show the radial nerve.

of nerve injuries unique to each sport. The purpose of this study was, therefore, to examine how ice hockey playing affects the nerves in the upper arm region.

Axillary nerve injuries in ice hockey have been defined before. The most common cause of axillary nerve injury is direct blow or shoulder dislocation. Blunt trauma to the anterior shoulder without dislocation is implicated in axillary nerve trauma.^{10, 14, 15} Axillary neuropathy due to direct contact without shoulder dislocation has been reported in two hockey players.² The axillary nerve is susceptible to injury at several sites, including the origin of the nerve from the posterior cord, the anteroinferior aspect of the subscapularis muscle and shoulder capsule, the quadrilateral space, and within the subfascia of the deltoid muscle.¹⁶ The nerve leaves the axilla through the quadrilateral space, which is formed by the humerus and the teres minor, teres major and long head of the triceps muscles¹⁵ (Figure 1). Axillary nerve compression within the quadrilateral space had been postulated to be secondary to abnormal fibrous bands and muscle hypertrophy of the muscular boundaries of the space, which cause static and/or dynamic compression of the axillary nerve.^{17, 18} The present results

demonstrated that the DML of the axillary nerve was significantly prolonged in the dominant and non-dominant arms of the ice hockey players compared with the controls (subjects with an Erb-deltoid distance longer than 18 cm [N.=10]). We can speculate on the reasons for these differences. The demands placed on the upper extremities in ice hockey generally occur bilaterally. In ice hockey, the shoulders have to abduct and adduct with the hips to maintain balance, momentum and increased velocity, whether the players have one or two hands on the stick for high-performance skating. Practicing forceful shoulder/arm movement by keeping the skates on the ice and forcefully abducting and adducting the shoulders to produce forward movement while maintaining a deep bend are recommended for enhancing performance.¹⁹ We suggest that the exercise intensity was such that it would have an effect on the axillary nerve conduction velocity in both the dominant and non-dominant arms of the ice hockey players.

Ice hockey can repetitively stress the upper extremity during shooting, because of forceful throwing to move the puck from the stick blade to the opponent's net. The mechanism of delayed motor conduction velocity in the axillary nerve may both tract and compress as the axillary nerve stretches across the humerus during movement. The anatomical features are such that the axillary nerve is only free for a short distance in the axilla; for the remainder of its length, it is attached to the deltoid muscle by its numerous branches. This appears to render it susceptible to stretch injury.^{14, 18} There may also be traction injury at the point where the nerve exits the quadrangular space in forceful movement of the shoulder or muscle hypertrophy of the muscular boundaries of the space may cause compression of the axillary nerve. On the other hand, the mechanism of injury in the majority of athletes studied was a direct blow to the anterior deltoid muscle.^{2, 18} The mechanism of axillary nerve injury is typically a direct blow to the anterolateral deltoid muscle, such as that which occurs during hockey collisions and when attempting to tackle an opposing player. A compressive force is applied to the axillary nerve as it travels on the deep subfascial surface and within the deltoid muscle.¹⁶ We suggest that the axillary nerve might be injured from a compressive force as it travels on the deep deltoid muscle in repetitive direct blows during practices and games.

The musculocutaneous nerve arises directly from

the lateral cord of the brachial plexus. In the upper arm, it pierces the coracobrachialis muscle to run in the fascia between the biceps and brachialis muscles. It innervates the biceps, brachialis and coracobrachialis.¹⁵ Statistical differences in DML of the musculocutaneous nerve between the ice hockey players and the controls in the dominant arms are evident (subjects with an Erb-biceps distance shorter than 26 cm [N.=8]). To our knowledge, cases of musculocutaneous nerve injury resulting from an athlete's participation in ice hockey have not been mentioned in the literature before.

The musculocutaneous nerve may be vulnerable to injury in certain situations. Musculocutaneous nerve lesions have been reported in association with vigorous upper extremity activity.²⁰⁻²³ Mastaglia,²⁰ reported three cases of isolated musculocutaneous neuropathy related to heavy physical activity. Hypertrophy or strong contraction of the coracobrachialis (as in weight-lifters) is assumed to determine both direct mechanical and ischemic nerve injury.^{23,24} Injuries to the musculocutaneous nerve most commonly involve its sensory portion.²⁵

In ice hockey, upper extremity training exercises include biceps curls, triceps pushdowns, incline dumbbell bench press, and dumbbell flat press. It seems that using the biceps and the coracobrachialis muscle can create notable muscle compartment pressure on the musculocutaneous nerve. The significant differences in neurophysiologic study of the musculocutaneous nerve between the ice hockey players and the controls may reflect that the forces acting on the shoulder and elbow during ice hockey matches can effectively influence motor conduction velocity.

The radial nerve is a direct continuation of the posterior cord of the brachial plexus. On leaving the axilla, the radial nerve winds around the back of the arm, first between the long and medial heads of the triceps, then in the spiral groove on the back of the humerus, between the lateral and medial heads of the triceps²⁷ (Figure 1). The most common radial neuropathy occurs at the spiral groove. Here, the nerve lies juxtaposed to the humerus and is quite susceptible to compression.¹⁵

The radial nerve is not infrequently injured during athletic or other strenuous activities.²⁸ It can be compressed or undergo traction injury at multiple sites along its course. Radial nerve injuries may result from muscular effort with the arm. Woltman *et al.*,²⁹

described a radial nerve injury resulting from throwing a discus. Sinson *et al.*,²⁸ presented two cases of severe radial nerve injury precipitated by the "windmill" arm motion of softball pitchers. Proximal (high) radial nerve palsy after strenuous muscle activity has been reported.³⁰⁻³³ Both unilateral and bilateral compression syndrome of the nerve at the lateral head of the triceps muscle has been reported and attributed to repetitive movement and muscle hypertrophy.^{25,31,34,35-37} Many cases of radial nerve entrapment following muscular overexertion have been reported to be caused by the fibrous arch at the lateral head of the triceps.^{30,31,34,38} To explore this mechanism further, Lotem *et al.*³⁰ performed a number of cadaver dissections of the radial nerve. They found a fibrous arch of tendon giving rise to muscle fibres of the lateral head of the triceps in "almost every case". Furthermore, they observed that it appeared generally more conspicuous and stronger in muscular subjects. The passage of the radial nerve under the arch was usually loose; however, in some cases it was tighter than in others. They suggested that a tight arch may cause compression of the radial nerve. Fibrous arches can also occur at the long head of the triceps muscle. Prochaska *et al.*³³ reported high radial nerve palsy to have occurred in a tennis player after practicing his tennis serve. Their surgical exploration showed a fibrous arch coming from the long head of the triceps and causing entrapment of the radial nerve.

In this study, it was found that the DML of the radial nerve was significantly prolonged in both the dominant and non-dominant arms of the ice hockey players. The level of the compression may be below the origin of the branches to the triceps and proximal to the posterior cutaneous branch. Two plausible causes for prolonged radial nerve DML have been postulated: direct compression by the hypertrophied triceps muscle and stretch of the arm. Physical exercise causes hypertrophy in the muscles involved. In this study the perimeters of the arm and forearm were principally influenced by muscle hypertrophy. A very marked hypertrophy was measured in both the dominant and non-dominant arm and forearm of the ice hockey players. According to the authors, the muscular hypertrophy of the triceps produced an extrinsic mass effect on the underlying radial nerve. It is clear that ice hockey helps to develop more muscle mass. One of the most critical muscles used in ice hockey is the triceps brachii. A proper training program will balance exercising of

the biceps and the triceps. These areas should be worked equally. Hockey players do not realize that when they are on the ice, they use their triceps much more than the biceps. Although one of the most used muscles is the biceps brachii, ice hockey players use their triceps much more than the biceps. Any time a player straightens his arm the triceps are working. When a forward powers around a defenseman with one hand on his stick and the other arm to separate him from the opposing player, the triceps are being exhausted. The triceps also work during take-outs in front of the net and along the wall, holding the opponent off with one arm while carrying the puck.²⁶ As a full collision sport, ice hockey requires muscular overexertion for efficient movement. In the authors' opinion, repetitive motion and overload of the triceps muscle are the major etiological factors in prolonged radial nerve DML. Because of forceful repetitive movements and muscular overexertion, the radial nerve is compressed by the overlying lateral head of triceps. Prolongation may also be caused by compression by a fibrous arch related to the triceps. It is possible that prolonged DML may result from repetitive shoulder activity, leading to nerve ischemia. On the other hand, a traction force may be placed on the axillary nerve when stretching the arm. The possible mechanism for these injuries appears to be stretching of the arm during extreme muscular effort. The sudden and forceful extension of the elbow and shoulder movements may provide traction sufficient to compromise the radial nerve as it traverses in the triceps muscle or spiral groove.

It is helpful for the clinician to be familiar with the demands of a given sport to better understand the connection between various training practises and overload to specific musculoskeletal structures.¹¹ According to the results of the present study, it is likely that a combination of skeletal muscle hypertrophy and excessive biomechanical demands on the neurological structures during ice hockey may be the main etiologic factor in the compression of axillary and radial nerves.

Conclusions

To our knowledge, these data represent the first evaluation of motor nerve conduction latency of the axillary, radial and musculocutaneous nerves in ice hockey players. In the present study, many of the asymptomatic ice hockey players with slowing nerve con-

duction tests may represent a presymptomatic or asymptomatic neuropathy similar to subclinical entrapment neuropathy. We can speculate about the reasons for prolonged DMLs. Hockey players are subjected to high-velocity impacts with players, pucks, sticks and the unforgiving ice surface and boards.¹ On the other hand, the hockey equipment is probably not sufficiently effective for the physical demands of the game. If the physiological stresses inherent in the game of ice hockey have been correctly understood, the authors can modify equipments scientifically.

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