Electrodiagnostic confirmation of long thoracic nerve palsy

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SUMMARY Long thoracic nerve latencies were measured in 25 normal subjects. The nerve was stimulated at Erb's point. Monopolar electrodes were used to record the motor evoked response from the serratus anterior muscle. The mean long thoracic nerve latency was 3.9 ± 0.6 ms. Four athletes with unilateral, isolated long thoracic nerve palsies were compared with the control group and with the uninvolved extremities. Long thoracic nerve latency examinations may help confirm the presence of long thoracic nerve palsy and test proximal nerve conduction.

In the past 20 years, latencies of muscle responses from excitation of nerves to muscles of the shoulder girdle have been studied repeatedly.1-4 The axillary, musculocutaneous, and suprascapular nerves have been stimulated proximally at Erb's point or at the axilla, and the latencies recorded have involved travel of the electrical impulse across parts of the brachial plexus. Indeed, at least one study has referred to them as "brachial plexus latencies".3

A long thoracic nerve latency would represent a less complicated example of proximal nerve conduction. However, none of the above studies have determined latencies from long thoracic nerve stimulation. Recently, four athletes presented with unilateral, isolated long thoracic nerve palsies and, therefore, presented a need for the electrodiagnostic study of this nerve.

The long thoracic nerve is formed directly from the fifth, sixth, and seventh cervical spinal nerve roots.6 As a result, it is not a part of the brachial plexus but passes posterior to it and upon the anterior surface of the scalenus medius (fig 1). The nerve is 15–20 mm behind the clavicle and angulates over the first or second ribs following the thoracic wall. It is attached at its end to the serratus anterior which it innervates. During its course down the neck and thoracic wall, traction will be exerted upon the nerve with shoulder depression or with contralateral bending movements of the cervical spine.

Method

Twenty-five healthy male and female unpaid volunteers (control group) were studied. None of the subjects had any evidence of peripheral neuropathy, diabetes, renal disease, shoulder girdle atrophy, or any other abnormalities. The age range was 19–55 years, and the median was 33 years.

Four male athletes (neuropathy group) were
studied. Two were football players, and two basketball players. None had any past history of peripheral neuropathy, diabetes, renal disease, or shoulder girdle atrophy before the onset of the injury. All were injured during the course of a game. The age range was 18–22 years, and the median was 19 years. All were evaluated five to seven weeks after the injury.

The temperature of the room in which the examinations were performed was 21–23° C. A TECA TE-4 electromyograph was used for all studies with a storage oscilloscope. The latency values were measured to the first deflection from the isoelectric line of the evoked potential. Infrared direct rewriting equipment as well as Polaroid photographs were used to record the evoked potentials.

The long thoracic nerve was stimulated at Erb’s point (above the upper margin of the clavicle lateral to the clavicular head of the sternocleidomastoid muscle). The cathode of the surface stimulation electrode was placed at Erb’s point, and the anode was located above and medially. The earth (ground) electrode was placed upon the thoracic wall at the anterior axillary line at the T2 rib level. A monopolar electrode was placed into the digitation of the serratus anterior at the T5 rib level at the midaxillary line. The position of the electrode was verified by having the subject attempt protraction of the shoulder. The inactive surface electrode was placed 20 mm caudally to the active electrode. Distances were measured with obstetric calipers. This method has given more reliable measurements than did tape measurements. All four subjects in the neuropathy group had electromyographic examination of the involved extremity.

Results

All the athletes showed fibrillations and sharp waves in the involved serratus anterior. On attempted voluntary contraction, a decreased number of motor unit action potentials were recruited. The motor unit potentials that were recruited fired at a more rapid rate than expected for the strength of the contraction. In all the athletes, the deltoid, biceps, triceps, infraspinatus, latissimus dorsi, abductor pollicis brevis, and erector spinae muscles were normal.

![Figure 2](http://jnnp.bmj.com/)

**Fig 2.** Motor evoked potential after stimulation of a normal long thoracic nerve. The height of the time line is 1 mV. Each dot is 0.1 ms.

![Figure 3](http://jnnp.bmj.com/)

**Fig 3.** Motor evoked potential after stimulation of the involved long thoracic nerve from the neuropathy group. The height of the time line is 1 mV. Each dot is 0.1 ms.

All four athletes recovered from their injuries within a year of examination. The mean response latency of the neuropathy group after recovery was 4.0 ± 0.5 ms.
Discussion

It has been shown that either surface or deeply placed concentric recording electrodes are probably preferable to monopolar recording electrodes. This situation is especially true with regard to the amplitude of the evoked potential and to the sharpness of the deviation from the isoelectric baseline. However, the stimulation of the long thoracic nerve at Erb’s point was not limited to that nerve. Therefore, a major disadvantage of stimulation at Erb’s point lies in the inadvertent recording of a volume conducted potential from the latissimus dorsi. This situation is particularly true for patients with long thoracic nerve palsy. Recording percutaneously from the serratus anterior is a way of making that objection. On the other hand, the comfort, safety, and convenience of monopolar electrodes were important advantages when compared to the use of concentric electrodes. Use of a hollow electrode might result in production of tension pneumothorax.

Long thoracic nerve palsy has been produced by downward strains on the shoulders caused by well-loaded knapsacks. It has also been noted after strenuous exertion and in patients with brachial plexus neuritis. Indeed, downward shoulder traction and lateral flexion of the neck may also lead to neuropathy of the upper trunk of the brachial plexus as well as palsy of the long thoracic nerve.

However, even though the mechanisms of injury are similar, the brachial plexus and long thoracic nerve injuries may be separate and distinct. Prescott and Zollinger reported that abduction of the shoulder beyond 90° might lead to an isolated palsy of the long thoracic nerve and that excellent recovery from this lesion could be observed. This last situation is probably related to the long thoracic nerve palsies noted in the four athletes. Both basketball and football may, on occasion, require sudden and forceful abduction of the shoulder beyond 90°. All four of the injured athletes recovered completely. Goodman et al have reported traumatic long thoracic nerve palsy with only partial or no recovery. Our patients were more like those reported by Prescott and Zollinger.

Whether long thoracic nerve palsy is isolated or is accompanied by neuropathy of the upper trunk of the brachial plexus, determination of the long thoracic nerve latency will help confirm a palsy of this nerve. The long thoracic nerve latency may also be a method for evaluating proximal nerve conduction in systemic diseases.

References