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Pressures recorded in ulnar neuropathy

The pressure between the ulnar nerve and the arcade bridging the two heads of the flexor carpi ulnaris muscle was recorded peroperatively in ten patients with electrophysiologically confirmed ulnar neuropathy at the elbow. At rest, with the elbow extended, pressures ranged from 0 to 19 mm Hg but increased in flexion and during isometric contraction of the flexor carpi ulnaris muscle to maximal values above 200 mm Hg.

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The aim of this investigation was to determine the pressure that the ulnar nerve sustained in the cubital tunnel in patients with an electrophysiologically confirmed ulnar neuropathy at the elbow.

Patients and methods

Ten patients, nine males and one female, aged 40–65 years, had characteristic symptoms of ulnar neuropathy, electrophysiologically confirmed to be located at the elbow level. In six patients the dominant side was involved, in four the nondominant. The only patient with bilateral symptoms had 6 months previously successfully undergone surgery for ulnar neuropathy at the other elbow.

Preoperative electrophysiological examinations were performed in all patients. Motor and orthodromic sensory nerve conduction in the ulnar nerves in both arms and in the median nerve on the affected side was studied using both surface and needle electrodes. In the ulnar nerve the motor nerve conduction velocity was measured over the segments: axilla-above elbow, above-below elbow and below elbow-wrist. The amplitude of the muscle response was measured for all simulation sites. The following electrophysiological parameters were used for this study: sensory nerve action potential amplitude at the wrist recorded both with surface and needle electrodes, motor nerve conduction velocity in the elbow segment, amplitude of maximal muscle response of the hypothenar muscle when the ulnar nerve was stimulated at the wrist, and the percentage fall of maximal muscle response when the ulnar nerve was stimulated above the elbow in relation to the amplitude when the nerve was stimulated at the wrist.

The pressure in the cubital tunnel was determined under general anaesthesia. Muscle relaxants were not used. The intersection between the ulnar nerve and the proximal edge of the arcade which bridges the two heads of the flexor carpi ulnaris muscle was dissected free in a bloodless field. The tourniquet was then removed. To prevent postischaemic oedema the forearm was elevated for about 15 min. At least 20 min with intact circulation was allowed before the pressure recording started. A teflon catheter (outer diameter 1.5 mm), with an open end and two side holes placed 2 and 5 mm from the end, was inserted beneath the flexor carpi ulnaris arcade and placed on the surface of the nerve. The catheter was filled with heparinized saline and connected to a pressure transducer and an ink recorder (Siemens-Elema, Sweden). To keep the recording system patent the catheter was continuously perfused with heparinized saline (50 µl/min). This pressure recording system is a routine set-up for pressure determinations in experimental situations and has previously been used when investigating the pressure in the carpal tunnel (Werner et al. 1983).

The pressure was first recorded with the elbow extended and then flexed 120 degrees. Thereafter the pressure during isometric contraction of the flexor carpi ulnaris muscle was recorded with the elbow in both extended and flexed position. Muscle contraction was elicited by tetanic stimulation of the ulnar nerve with a needle electrode (DISA 13 L 62) placed close to the nerve above the elbow. The needle electrode was considered to be close to the nerve when stimulus currents below 1 mA caused a visible muscle twitch in distal muscles innervated by the ulnar nerve. The stimulus parameters were: pulse duration 0.3 ms, frequency 40 Hz, and tetanus duration 5 s. To observe the relation between the muscle contraction force and the pressure occurring in the cub-

Table 1. Pressure on the ulnar nerve at the elbow and the electrophysiological abnormalities in ten patients with ulnar neuropathy. Group I consists of the five patients with the less marked neuropathy and Group II of those five patients with the most marked neuropathy. Figures are given as mean ± SD.

	Elbow extended		Elbow flexed	
	Rest	Stimulation	Rest	Stimulation
Group I	7±8	54±32	41±33	163±136
Group II	10±7	131±92	86±66	247±107

ital tunnel, the stimulus current was increased stepwise until the stimulus intensity was clearly supramaximal. Thus, there was a successive increase from minimal to maximal muscle contractions.

Results

At rest, with the elbow extended, the pressure was 9 (0–19) mm Hg. When the elbow was flexed the pressure increased to 63 (2–187) mm Hg. When maximal contraction of the flexor carpi ulnaris muscle was elicited, the pressure increased to 92 (28–238) mm Hg when the elbow was extended and to 209 (52–413) mm Hg when the elbow was flexed.

When the strength of the muscle contraction was increased due to augmented stimulus intensity, there was a concomittant increase in pressure. To investigate whether the pressures recorded were related to the severity of the nerve lesion in the cubital tunnel, the electrophysiological parameters and the pressures were compared (Table 1). The mean pressures in the five patients with a more severe nerve lesion were higher than in the five patients with less severe nerve lesion, but the difference was not significant ($p>0.05$).

Discussion

The ulnar nerve has limited space when it passes the cubital tunnel beneath the fibrous arcade which bridges the two heads of the flexor carpi ulnaris muscle. By dividing the fibrous arcade in patients with ulnar neuropathy at the elbow, nerve decompression is achieved and good results have been reported (Thomsen 1977, Lugnegård et al. 1982).

Osborne (1957, 1970), Feindel & Stratford (1958) and Apfelberg & Larson (1973) have reported that the space available to the ulnar nerve decreases when the elbow is flexed. In cadaver experiments, Pechan & Julis (1975) observed that the intraneural pressure in the ulnar nerve increased during elbow flexion, and our investigation confirmed this. In animals, Rydevik et al. (1981) observed that static local nerve compression for 2 h caused a reduction of the venous blood flow at 20–30 mm Hg and ischaemia at 60–80 mm Hg. An increase of the pressure in the cubital tunnel as observed here seems likely to compromise the capillary circulation of the ulnar nerve.

In the carpal tunnel syndrome the median nerve is subjected to increased pressure during volar flexion of the wrist (Phalen test). In normal subjects Gelberman et al. (1981) observed a pressure of 30 mm Hg in this position. In patients with the carpal tunnel syndrome, a pressure of about 90 mm Hg was observed by both Gelberman et al. (1981) and Werner et al. (1983). Contractions of the wrist and finger flexor muscles increase the pressure in the carpal tunnel to about 150 mm Hg in patients with a carpal tunnel syndrome (Werner et al. 1983). A similar increase of pressure occurs in the radial tunnel in patients with a radial tunnel syndrome when the supinator muscle is electrically stimulated to maximal contraction (Werner et al. 1980). In our investigation the ulnar nerve was found to sustain similarly high pressures on maximal contraction of the flexor carpi ulnaris muscle and especially high pressures when the elbow was in a flexed position. Mechanical damage to the nerve seems likely to occur as a result of iterative pressure increases of this magnitude. Telescoping of the internodes, with swelling and folding of the myelin sheets (Ochoa & Marotte 1973), might occur. The blood flow might be impaired and thus cause a protein leakage through the epineural venules, resulting in oedema and epineural fibrosis (Rydevik & Lundborg 1977, Rydevik et al. 1981).

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