Acta orthop. scand. *54,* 312–316, 1983

PRESSURE AND NERVE LESION IN THE CARPAL TUNNEL

Carl-Olof Werner, Dan Elmqvist & Per Ohlin

Departments of Orthopaedic Surgery, Clinical Neurophysiology and Clinical Physiology, Hospital of Helsingborg, Sweden

In 16 patients, where the diagnosis carpal tunnel syndrome was electrophysiologically confirmed, the pressure between the median nerve and the carpal ligament was measured peroperatively.
At rest the pressure was 18–64 mmHg, mean 31 mmHg. Passive volar and dorsal wrist flexion increased the pressure about three times. Isometric or isotonic maximal contractions of wrist and finger muscles, elicited by tetanic nerve stimulation increased the pressure to three to six times the resting value. These high pressures may be one of the causes of the nerve lesion in the carpal tunnel syndrome.

Key words: carpal tunnel syndrome; median nerve; nerve compression; nerve lesion

Accepted 29.viii.82

The carpal tunnel syndrome is the most common peripheral nerve entrapment in man. The median nerve, passing beneath the firm carpal ligament, is easily compressed when the pressure in the carpal canal increases. Flexor synovitis, increased volume of fluid or any space occupying condition in the canal may increase the pressure and elicit symptoms of a carpal tunnel syndrome. Strenuous use of the hand may also aggravate the symptoms (Tanzer 1959, Phalen 1966, 1972, Birkbeck & Beer 1975).

The aim of this investigation was to record the magnitude of the pressure that the median nerve was subjected to beneath the carpal ligament during rest, passive volar and dorsal wrist flexion and during contraction of the wrist and finger flexor and extensor muscles in patients with a carpal tunnel syndrome.

The study was approved by the Human Ethics Committee, Medical Faculty, University of Lund, Sweden.

PATIENTS AND METHODS

Sixteen patients, 15 females and one male, aged 25–59 years, mean age 46 years, were included in this investigation. All patients, selected at random, showed characteristic symptoms of carpal tunnel syndrome.

Electrophysiological examinations were performed preoperatively in all patients. Both motor and orthodromic sensory nerve conduction in the median and ulnar nerves was studied using both surface and needle-electrodes. Only patients with abnormal findings restricted to the distal part of the median nerve were included. In 11 patients both sensory and motor conduction was abnormal. In four patients the distal motor latency was within normal limits but the sensory conduction velocities and the response amplitudes were reduced from both the thumb and middle finger. In the remaining patient the only abnormality was a reduced sensory conduction velocity and a reduced response amplitude when the middle finger was stimulated and the sensory potential was recorded at the wrist. The distal motor latency recorded with concentric needle electrodes in the abductor pollicis muscle after stimulation of the median nerve with needle electrodes just proximal to the wrist (Buchtal et al. 1974) was 5.3 ± 1.0 ms (normally below 4.7 ms). Sensory nerve con-

Support was received from the Swedish Medical Research Council (No B82-14X-00084-18A).

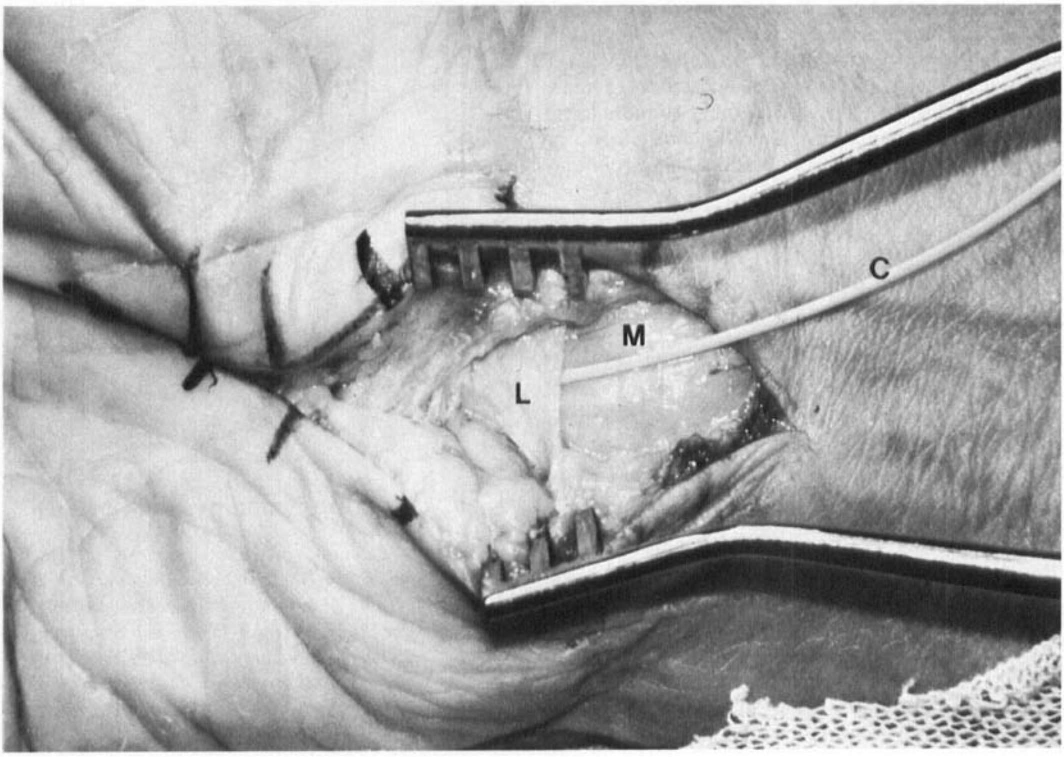

Figure 1. The pressure recording catheter (C) in place on the volar surface of the median nerve (M) beneath the carpal ligament (L).

duction velocity, obtained with needle electrodes placed close to the median nerve just proximal to the wrist and stimulating the thumb and the middle finger with ring electrodes, was 28.3 ± 9.1 m/s and 32.8 ± 5.0 m/s, respectively (normally above 40 m/s).

Surgery was performed under general anaesthesia. Muscle relaxants were not used. The intersection between the median nerve and the proximal edge of the carpal ligament was dissected free in a bloodless field. The torniquet was removed and to prevent postischaemic oedema the forearm was elevated for about 10 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 sideholes placed 2 and 5 mm from the end was inserted beneath the ligament and placed on the volar surface of the nerve (Figure 1). 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). The pressure recording system is a routine set-up for pressure determination in experimental situations such as the present. It should be noted that the position of the tip of the catheter is critical and it was therefore anchored with a suture.

At rest the pressure between the nerve and the ligament was recorded with the wrist in neutral position and the fingers relaxed. The pressure was then recorded during maximal passive volar wrist flexion with the fingers both straight and flexed. Similar recordings were performed during maximal passive dorsal wrist flexion. The pressure occurring during stimulus elicited muscle contraction was recorded both at "isometric" and "isotonic" conditions. During isometric contractions of the wrist and finger flexor muscles the wrist was held in a neutral position and the fingers were kept straight. During isotonic contraction the hand and fingers were allowed to move freely during the stimulation. Similar recordings were performed during contraction of the wrist and finger extensor muscles. Muscle contraction was elicited by tetanic stimulation of the median nerve for the flexor muscles and the radial nerve for the extensor muscles. Needle electrodes (DISA 13L62), placed close to the nerves above the elbow, were used for stimulation. We considered the needle electrodes being close to the nerve when stimulus currents below 1 mA caused a visible muscle twitch. The stimulus parameters were: pulse duration 0.3 ms, frequency 40 Hz, tetanus duration 5 s. To observe the relation between the muscle contraction force and the pressure between the nerve and the carpal ligament the stimulus current was increased stepwise until the stimulus intensity was well supramaximal. Thus, there was a successive increase from minimal to maximal muscle contractions.

RESULTS

The pressure between the median nerve and the carpal ligament at rest was 31 ± 12 (mean ± S.D.) mmHg.

During passive volar wrist flexion with the fingers straight the pressure increased to 75 ± 59 mmHg, and with the fingers flexed to 60 ± 29 mmHg. During passive dorsal wrist flexion with the fingers straight the pressure increased to 105 ± 56 mmHg. When this procedure was performed with the fingers flexed a similar pressure increase, to 113 ± 53 mmHg, was observed.

When maximal muscle contraction was elicited with the nerve stimulation technique described above the pressure increased three to six times the resting value. Thus, during isometric contraction of the wrist and finger flexors the pressure increased to 180 ± 107 mmHg. Isotonic contraction of wrist and finger flexors increased the pressure to 150 ± 110 mmHg. During isometric contraction of wrist and finger extensors the pressure increased to 128 ± 83 mmHg. Isotonic contraction of wrist and finger extensors increased the pressure to 105 ± 67 mmHg. When the muscle contraction became stronger as the stimulus intensity was increased the pressure changes increased concomitantly (Figure 2).

In order to see if the pressures recorded had any relation to the severity of the nerve lesion in the carpal tunnel, the electrophysiological parameters and the pressures were compared. For the electrophysiological parameters a rank

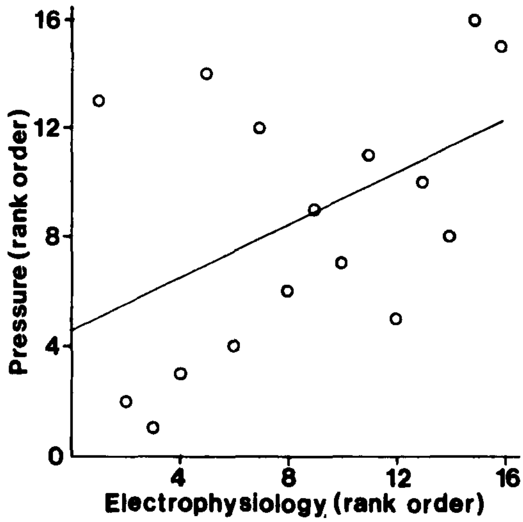

Figure 3. Relation between preoperative electrophysiological abnormalities in the median nerve over the carpal tunnel and peroperatively recorded pressures between the median nerve and the carpal ligament. The patients have been ranked according to the severity of the electrophysiological abnormalities and the magnitude of the pressures recorded. $r_s = 0.46$ (Spearman rank correlation coefficient).

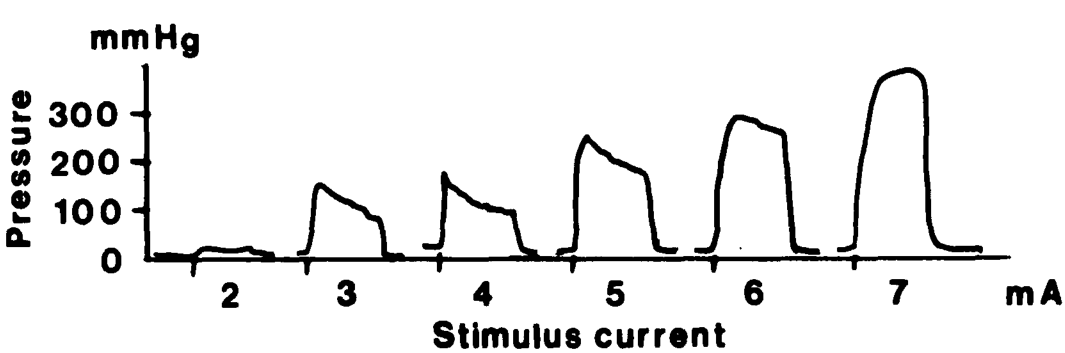

Figure 2. Pressure between the median nerve and the carpal ligament when muscle contractions were elicited by median nerve stimulation (40 Hz, 5 s) at the elbow with increasing stimulation intensity.

order of the patients was established. The patient with the most abnormal findings was given the highest rank number. A similar rank order was established for the pressures. The patient with the highest pressures was given the highest rank number. Figure 3 shows the patients plotted according to their rank numbers for the electrophysiological parameters and for the pressures. The pressure between the median nerve and the carpal ligament was significantly ($P < 0.05$) higher in patients with more severe electrophysiological signs of a nerve lesion.

DISCUSSION

In the carpal tunnel syndrome an indentation of the median nerve, sometimes combined with a proximal swelling of the nerve, located under the proximal part of the carpal ligament is a common finding at surgery. Neary et al. (1975) reported from autopsy findings alteration in the connective tissue of the nerve with this location. By placing the holes in the catheter at this point we made the pressure recordings from the site where the main lesion appears to occur.

Gelberman et al. (1981) measured the pressure in the carpal tunnel in normal subjects using a wick catheter percutaneously inserted into the tunnel. They observed a pressure at rest of about 3 mmHg. Volar and dorsal wrist flexion increased the pressure to about 30 mmHg. The pressure in the carpal tunnel in patients with a carpal tunnel syndrome has been measured by Tanzer (1959) and Gelberman et al. (1981). In six patients Tanzer, using a distensible bag filled with mercury, peroperatively recorded pressures of about 50 mmHg under the proximal part of the carpal ligament on both volar and dorsal flexion of the wrist. Gelberman et al., using their wick catheter technique, observed a pressure of about 30 mmHg at rest in 15 patients. The pressure increased to about 100 mmHg on volar and dorsal wrist flexion. The present observations of the pressure in the carpal tunnel at rest and on volar and dorsal wrist flexion are thus in good agreement with those of Tanzer and Gelberman et al.

Gelberman et al. (1981) were not able to correlate the magnitude of the pressure with the electrophysiological findings. In contrast, our observations showed a relation between electrophysiological abnormalities and pressure which supports the view that a raised pressure between the nerve and the ligament is an important factor for the nerve lesion in the carpal tunnel syndrome. In two of our patients, however, the pressures were high in spite of minimal electrophysiological evidence of a nerve lesion (Figure 3). The duration of symptoms in these two patients was rather short, about 6 months, which might explain why no severe nerve lesion had developed.

Apparently the pressure in the carpal tunnel increases considerably during contraction of the wrist and finger muscles. Even if supramaximal tetanic nerve stimulation may cause muscle contractions that are stronger than those occurring during physiological muscle work there is a relation between increasing muscle contraction and increasing pressure on the nerve. The pressure between the median nerve and the carpal ligament during maximal muscle contraction increased to about 150 mmHg and thus compares well with the pressure in the radial tunnel during stimulus elicited contraction of the supinator muscle in patients with a radial tunnel syndrome (Werner et al. 1980).

There are no experimental animal studies on the effect of intermittent compression of peripheral nerves. Studies in animals, where static local compression for 2 h or more was used, showed a reduction of the venous blood flow in the nerve at pressures of 20–30 mmHg and ischaemia occurred at 60–80 mmHg (Rydevik et al. 1981). Blockade of the nerve impulse conduction occurs at pressures of 130–150 mmHg (Bentley & Schlapp 1943, Sharpless 1975). It seems possible that mechanical damage of the nerve may result from iterative pressure increases of the magnitude observed in our study. Possible mechanisms are that intermittent pressure increases cause telescoping of the internodes with swelling and folding of the myelin sheets (Ochoa & Marrotte 1973) or that they impair the blood flow and cause a protein leakage through the walls of the epineural venulus resulting in oedema and epineural fibrosis (Rydevik & Lundborg 1977).

In conclusion, the present investigation showed that the median nerve sustains rather high local pressures during contraction of wrist and finger muscles and that there is a correlation between nerve compression and electrophysiological abnormalities in patients with a carpal tunnel syndrome.

REFERENCES

Bentley, F. H. & Schlapp, W. (1943) The effects of pressure on the conduction in peripheral nerve. *J. Physiol.* **102**, 72–82.

Birkbeck, M. Q. & Beer, T. C. (1975) Occupation in relation to the carpal tunnel syndrome. *Rheumatol. Rehabil.* **14**, 218–221.

Buchtal, F., Rosenfalck, A. & Trojaborg, W. (1974) Electrophysiological findings in entrapment of the median nerve at wrist and elbow. *J. Neurol. Neurosurg. Psychiat.* **37**, 340–360.

Gelberman, R. H., Hergenroeder, P. T., Hargens, A. R., Lundborg, G. N. & Akeson, W. H. (1981) The carpal tunnel syndrome. A study of carpal canal pressures. *J. Bone Joint Surg.* **63**-A, 380–383.

Neary, D., Ochoa, J. & Gilliat, R. W. (1975) Sub-clinical entrapment neuropathy in man. *J. Neurol. Sci.* **24**, 283–298.

Ochoa, J. & Marotte, L. (1973) The nature of the nerve lesion caused by chronic entrapment in the guinea-pig. *J. Neurol. Sci.* **19**, 491–495.

Phalen, G. S. (1966) The carpal-tunnel syndrome. Seventeen years' experience in diagnosis and treatment of six hundred fifty-four hands. *J. Bone Joint Surg.* **48**-A, 211–228.

Phalen, G. S. (1972) The carpal-tunnel syndrome. Clinical evaluation of 598 hands. *Clin. Orthop.* **83**, 29–40.

Rydevik, B. & Lundborg, G. (1977) Permeability of intraneural microvessels and perineurium following acute, graded experimental nerve compression. *Scand. J. Plast. Reconstr. Surg.* **II**, 179–187.

Sharpless, S. (1975) Susceptibility of spinal roots to compression block. In: *The research status of spinal manipulative therapy* (Ed. Goldstein, M.), NINCDS Monograph no 15, 155–162. US Department of Health, Education and Welfare. Bethesda, Maryland.

Tanzer, R. C. (1959) The carpal-tunnel syndrome. A clinical and anatomical study. *J. Bone Joint Surg.* **41**-A, 626–634.

Werner, C. O., Haeffner, F. & Rosén, I. (1980) Direct recording of local pressure in the radial tunnel during passive stretch and active contraction of the supinator muscle. *Arch. Orthop. Traumat. Surg.* **96**, 299–301.

Correspondence to: C. O. Werner, M.D., Department of Orthopaedic Surgery, Hospital of Helsingborg, S-251 87 Helsingborg, Sweden.