Pressure Dynamics of the Carpal Tunnel and Flexor Compartment of the Forearm

Tyson K. Cobb, MD, William P. Cooney, MD, Kai-Nan An, PhD, Rochester, MN

Although carpal tunnel pressures have been observed to increase as a result of repetitive flexion and extension of the wrist, and forearm compartment pressures have been shown to rise during and after muscle activity, the relationship between those two observations has not been studied. The flexor compartments of five cadavers were perfused with saline to determine whether elevated pressure in the flexor compartment of the forearm is transmitted to the carpal tunnel. The pressure in the carpal tunnel after the infusion was significantly different from the pressure in the flexor compartment of the forearm. Furthermore, pressures recorded in the carpal tunnel at the conclusion of the study were not statistically different from the preinfusion pressures. While the carpal tunnel may appear to be an open compartment anatomically, it functions as a relatively closed compartment with respect to transfer of pressure from the flexor compartment of the forearm under conditions that mimic elevated tissue pressure. (J Hand Surg 1995;20A:193–198.)

Elevated carpal canal pressures have been observed as a result of repetitive flexion and extension of the wrist.[1] Likewise, compartment pressures have been shown to rise during and following muscle activity.[2] However, the relationship between these observations has not been studied. From these two independent observations, the following hypothesis was established: Elevated pressure within the flexor compartment of the forearm resulting from sustained repetitive work tasks is transmitted to the carpal tunnel, with resulting median nerve dysfunction. This hypothesis assumes that the flexor compartment of the forearm is continuous with the carpal tunnel, allowing free flow of tissue pressure. The purpose of this study was to determine whether elevated pressure within the flexor compartment of the forearm is transmitted to the carpal tunnel.

From the Department of Orthopedics, Mayo Clinic and Mayo Foundation, Rochester, MN.

Received for publication Feb. 3, 1994; accepted in revised form Oct. 12, 1994.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

Reprint requests: William P. Cooney, MD, Biomechanics Laboratory Mayo Clinic, 200 First Street SW, Rochester, MN 55905.

Materials and Methods

Five fresh cadaveric upper extremities removed by mid-arm amputation were used for the study. A four-channel transducer system was constructed (Spectramed, Oxnard, CA) to allow simultaneous pressure monitoring at four sites. Slit catheters (Howmedica, Rutherford, NJ) were used to measure hydrostatic fluid pressure within the carpal canal and within the flexor compartment of the forearm. Before catheter insertion, the transducer/catheter systems were flushed with saline to remove all air bubbles and then calibrated.

Insertion of Catheters

A slit catheter (catheter 1) was placed into the carpal tunnel by the percutaneous technique described by Gelberman et al.,[3] modified on the basis of the findings of a previous anatomic study,[4] that allowed catheter placement at the level of the hook of the hamate. Catheter placement was performed by preinsertion of an angiocatheter 0.5 cm proximal to the proximal wrist flexion crease and just medial to the tendon of the flexor carpi radialis. It was directed dorsally and distally at an initial angle of 45°. Lines were drawn as previously described[4] to establish the position of the hook of the hamate. After penetration of the flexor retinaculum, the catheter was advanced within the carpal tunnel to the level of the hook of the hamate. The slit catheter was marked at the appropriate length (length from hook of hamate to site of percutaneous insertion) and inserted into the angiocatheter, and the latter was withdrawn. The correct position of the slit catheter was verified by the premeasured mark, and the position was confirmed by x-ray films.

A second slit catheter (catheter 2) was inserted into the flexor compartment of the forearm by the method described by McDougall and Johnston.[5] The catheter was placed into the region of the flexor digitorum profundus muscle at the junction of the proximal and distal halves of the forearm. With an ulnar approach, an angiocatheter was passed anterior to the ulna into the central portion of the compartment. The needle was withdrawn, the slit catheter was advanced into the angiocatheter, and the latter was withdrawn. A third slit catheter (catheter 3) was placed into the forearm at the junction of the proximal and middle thirds in the previously described manner. An additional angiocatheter (catheter 4) was placed into the central aspect of the proximal third of the forearm (proximal to catheter 3) in the previously described manner. Catheter 4 was used for saline infusion (Fig. 1).

Infusion Technique

Normal saline was infused through the angiocatheter (catheter 4) into the proximal forearm by gravitational flow at approximately 50 mm Hg. Normal saline was added intermittently to maintain the hydrostatic pressure at catheter 4 while pressures were continuously monitored (0.5 Hz) at catheters 1, 2, and 3. Time was recorded from the onset of infusion to a maximum of 2 hours. The rate of infusion varied from specimen to specimen, as did the total amount of saline infused. Approximately 500–1500 mL was infused into each specimen. The pressures at the proximal (P3) and central (P2) aspects of the flexor compartment were compared with each other and with the pressure measured at the carpal canal (P1), and a paired t-test was used to determine whether a significant difference existed. Values were considered significant, $p < .05$.

Contrast Injection Studies

Injection studies with a contrast material, Hypaque, (Winthrop Pharmaceutical, New York, NY) were performed with fluoroscopy to demonstrate the degree of infiltration within the flexor compartment and the extent of fluid movement into the carpal tunnel. These studies were performed in three settings: (1) infusion into the proximal third of the flexor compartment, (2) infusion into the carpal canal, and (3) infusion into the synovial lining of the flexor tendons of the little finger. The degree of filling and transmission from one compartment to the next was evaluated in each of these injections. A latex injection was performed in one cadaver specimen through the synovial sheath of the flexor tendon

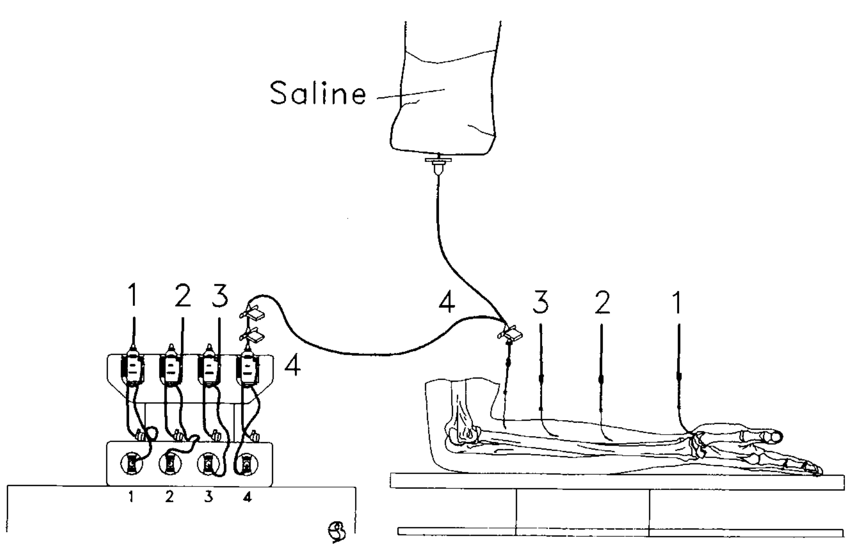

Figure 1. Line drawing demonstrating position of catheters. Catheter 1 is in carpal tunnel at the level of the hamate hook. Catheters 2 to 4 are in muscle tissues of the flexor compartment of the forearm. (By permission of Mayo Foundation.)

of the little finger. The synovial sheath of the flexor tendon of the small finger was dissected free and a catheter was placed into the sheath and advanced to the level of the metacarpophalangeal joint. An elastic band was placed around the sheath distal to the catheter opening, and this forced the liquid latex in a proximal direction. Following injection and after allowing time for the latex to set up, a dissection was performed to determine the amount and location of transfer of the latex solution through the carpal tunnel and into the flexor compartment of the forearm.

Results

Because of variation of specimen size, we were unable to standardize the distance between each pressure measurement. Likewise, the amount of fluid infused varied among specimens. Each infusion study was terminated when the extremity was grossly edematous and enlarged. For each of the five specimens, the curves were similar, in that the catheter closest to the infusion site (proximal flexor compartment, catheter 3) responded first and that in the next closest site (central flexor compartment, catheter 2) responded in a similar fashion except for a delay in response. However, the catheter in the carpal tunnel (catheter 1) had a minimal, if any, response. Figure 2 demonstrates the distribution of

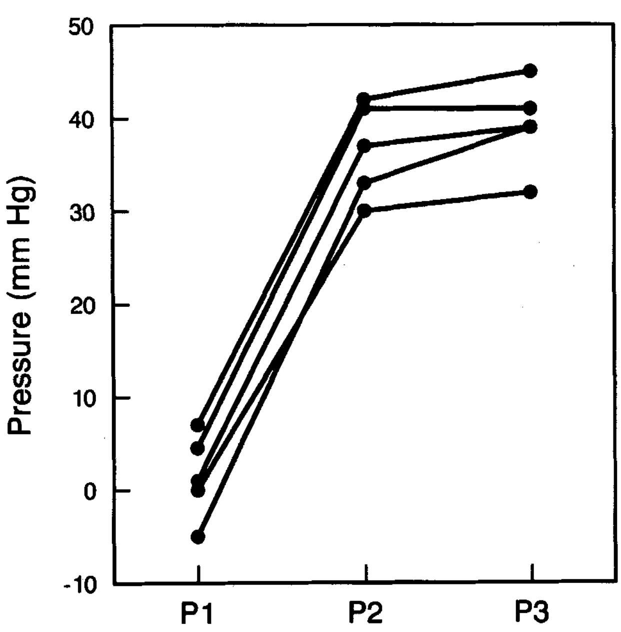

Figure 2. Line graph of three pressure points for five specimens. P1, pressure at carpal tunnel at conclusion of infusion; P2, pressure at central flexor compartment at conclusion of infusion; P3, pressure at proximal flexor compartment at conclusion of infusion.

the three pressures measured for each specimen. Note that the pressures within the carpal canal (P1) are significantly different from the pressures in the central (P2) or proximal (P3) aspect of the flexor compartment. In one specimen the infusion was continued for 12 hours. Even with this lengthy infusion, the pressures within the carpal tunnel did not show any significant change in comparison with preinfusion pressures.

When the pressures at the conclusion of the study for each site were analyzed relative to the others, the pressures within the flexor compartment (P3 and P2) were not significantly different ($p > .05$), whereas the pressures within the carpal tunnel (P1) were significantly different from the pressures in the central or proximal flexor compartment (P2 or P3) ($p < .0001$). The mean pressure at the carpal tunnel at the conclusion of the infusion for the five specimens was 1.46 mm Hg (SD $\pm$ 4.55). This is in contrast to the mean pressures in the proximal and central aspects of the flexor compartments, which were 39.2 mm Hg (SD $\pm$ 4.71) and 36.6 mm Hg (SD $\pm$ 5.13), respectively. No significant ($p > .2$) changes in the carpal tunnel pressures were found when preinfusion and postinfusion pressures were compared.

Hypaque injection studies performed with fluoroscopy demonstrated easy infiltration of the contrast solution within the flexor compartment. However, there was resistance to flow from the flexor compartment into the carpal tunnel, and a great deal of pressure was required to expedite this transfer. This feature is demonstrated by the amount of infiltration of Hypaque into the flexor compartment before transfer of the solution into the carpal tunnel (Fig. 3). When contrast solution was injected into the carpal tunnel under minimal pressure, the distribution of the solution was limited by the bursa into which it was injected. Two specimens were injected with Hypaque solution through the synovial lining of the flexor tendons of the little finger. The ulnar bursa and the quadrilateral space (Parona's space) were the first to fill under minimal pressure. This phenomenon is demonstrated by direct injection (Fig. 4). With increasing degrees of pressure and volumes of Hypaque, the solution was transferred into the proximal aspect of the flexor compartment of the forearm and into the hand, including the lumbrical muscles (Fig. 4).

The latex injection study performed through the synovial sheath of the flexor tendon of the little finger in a cadaveric specimen demonstrated transfer of latex through the carpal tunnel and into the flexor compartment of the forearm. The transfer occurred deep within the region of the profundus muscle. This phenomenon of transfer through the deep substance

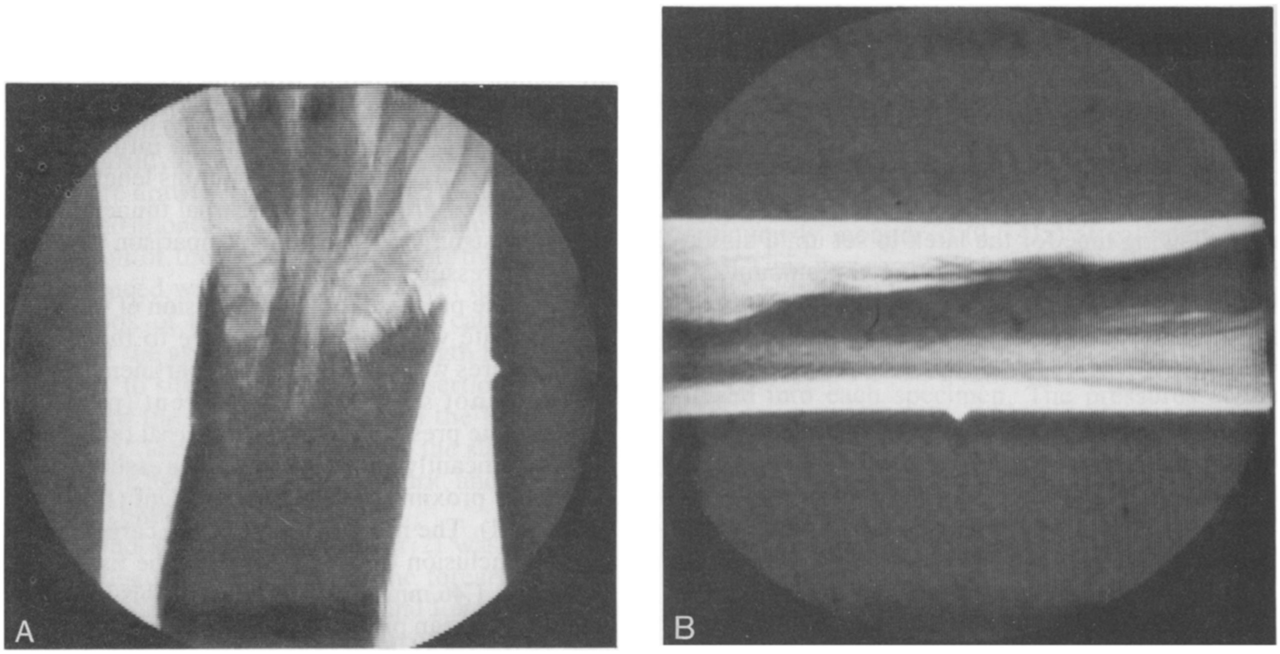

Figure 3. (A) Anteroposterior x-ray film of Hypaque infusion study in fresh frozen cadaveric specimen. Hypaque solution was infused into the proximal aspect of the flexor compartment of the forearm. Note the degree of infiltration into flexor compartment before transfer across the carpal canal. Also note how the Hypaque solution is extracted from the central aspect of the canal. Top, distal. (B) Lateral x-ray film of another specimen, demonstrating that the initial distribution of Hypaque solution into the forearm from the carpal tunnel is in the deep aspect of the flexor compartment. Right, proximal.

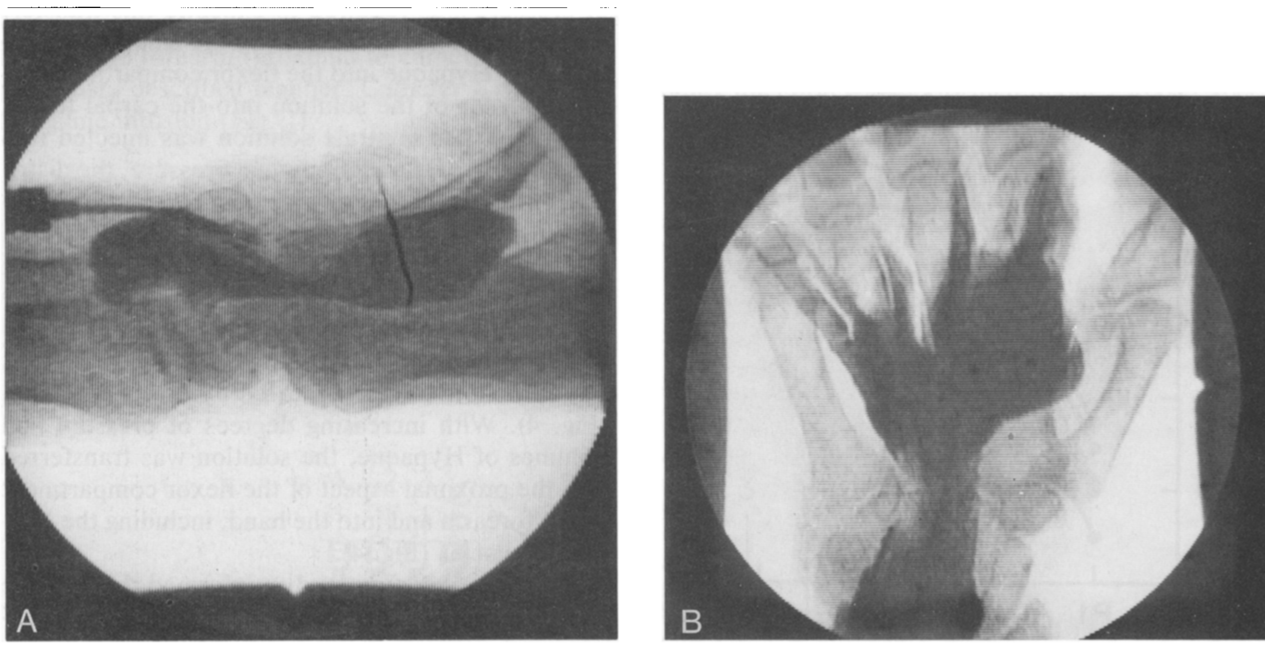

Figure 4. (A) Lateral x-ray film of cadaveric specimen after infusion of Hypaque solution directly into the carpal canal. This was accomplished under minimal pressure. Note proximal limitation of contrast medium at the distal forearm-carpal tunnel junction. Right, distal; top, palmar. (B) Anteroposterior x-ray film of same specimen. With increasing pressure and volume of Hypaque solution, the proximal forearm and central compartment of the hand also fill. Top, distal.

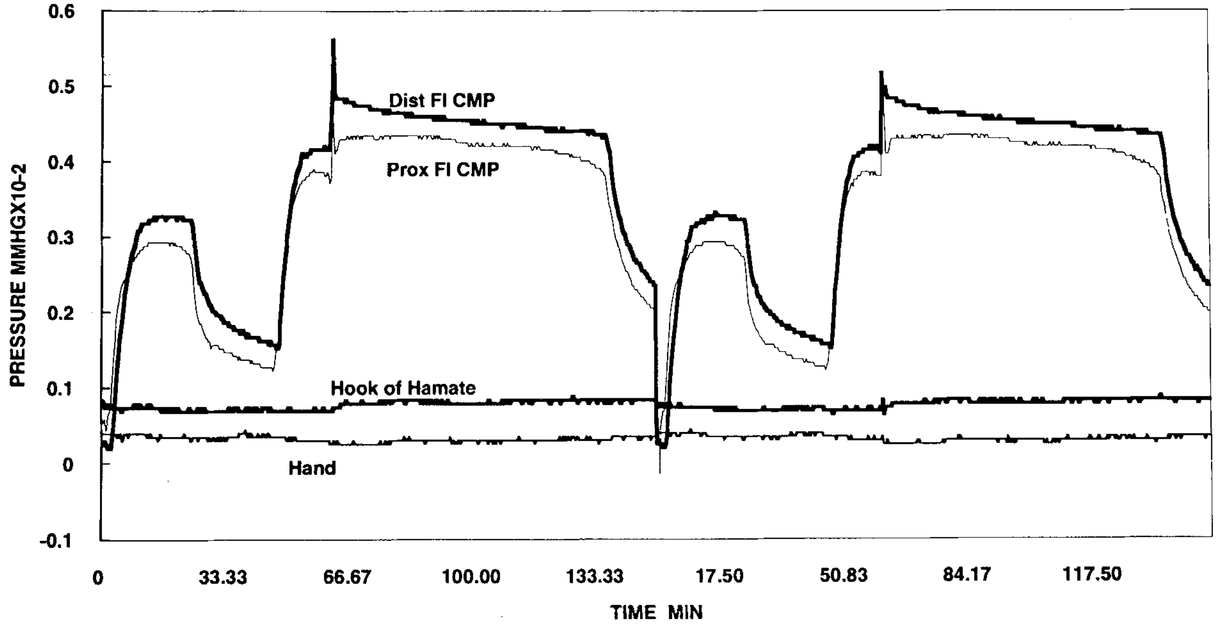

Figure 5. Infusion study with four sites of pressure measurement. Infusion was discontinued on multiple occasions to allow dissipation of pressure within the flexor compartment. Note, however, that the pressure at the carpal tunnel and palmar compartment of the hand remained essentially unchanged throughout the study. The pressures measured within the flexor compartment correlated highly with one another, as did pressures taken at the carpal canal and hand. However, the pressures measured within the flexor compartment appeared not to correlate with those taken at the hand or carpal canal. Dist F1 CMP, distal portion of flexor compartment; Prox F1 CMP, proximal portion of flexor compartment.

of the profundus muscle is also demonstrated in the Hypaque injections (Fig. 3B). Both retrograde and antegrade transfers through the carpal tunnel were observed with Hypaque injections; however, the resistance to transfer from the flexor compartment of the forearm into the carpal tunnel is quite high, whereas the converse is less true.

A number of preliminary infusion studies have been performed in our laboratory. One included infusion into the proximal portion of the flexor compartment and measurements of pressure in the distal and proximal portions of the flexor compartment of the forearm and also within the carpal tunnel and palms of the hand (Fig. 5). The results of this infusion complement the findings of the present study. The pressures within the flexor compartment of the forearm dissipated each time the infusion was terminated and then quickly increased each time the infusion was reinitiated. However, the pressures taken at the carpal canal and in the palm remained essentially unchanged and showed no correlation with the pressures within the forearm.

Discussion

Carpal tunnel syndrome has been linked to repetitive hand and wrist motion in the workplace[1,6–9] (Goebel ME, Goldner JC, Ferlic TP, read at the 59th annual meeting of the American Academy of Orthopaedic Surgeons, Washington, DC, February 20–25, 1992, unpublished data). Although the cause of carpal tunnel syndrome in this population has not been elucidated, these same authors have suggested that carpal tunnel syndrome in this specialized population differs from idiopathic carpal tunnel syndrome. Szabo and Chidgey[1] demonstrated an increase of canal pressure as a result of repetitive flexion/extension of the wrist. Furthermore, elevated pressures were maintained for a longer period in patients with carpal tunnel syndrome compared with normal controls. These authors pointed out previous studies that had demonstrated this phenomenon in patients with chronic compartment syndrome. Thruston and Krause[10] used the slit catheter to demonstrate a continuous increase in canal pressures when wrist extension was maintained. They proposed vascular congestion as a possible explanation for this phenomenon and thus as a possible cause of carpal tunnel syndrome.

Similarly, investigators have demonstrated elevated pressures within the muscular compartments during and after exercise.[2,11] Anatomically, the palmar boundary of the flexor compartment of the forearm has been shown to be continuous with the palmar boundary of the carpal tunnel.[12] Hypaque injection studies have also demonstrated continuity between the flexor compartment of the forearm and the carpal tunnel.[13] Previous studies have suggested that increased pressures within the flexor compartment of the forearm resulting from repetitive activity in a workplace could be transmitted into the carpal tunnel. From the results of the present study, this does not appear to be the case. In some specimens the pressure within the carpal tunnel did increase, but in other specimens the pressure actually decreased by several millimeters of mercury during the infusion. In no specimen did the pressure curves indicate that the carpal tunnel functions as a continuous canal with the flexor compartment; rather, there appears to be a barrier preventing free transmission of pressure from the flexor compartment into the carpal tunnel. However, other studies in our laboratory have demonstrated that when saline is infused into the carpal tunnel, there is less restriction to flow into the flexor compartment of the forearm. This finding is consistent with the results of the Hypaque injections performed in the present study. Thus, there appears to be somewhat of a one-way valve between the flexor compartment of the forearm and the carpal tunnel with respect to the flow of fluid and transmission of pressure. This phenomenon could be explained by the fascial limits (epimysium) of the individual muscles of the flexor compartment, which prevent flow into the carpal tunnel. However, when infusion takes place into the carpal tunnel, saline is able to flow between the musculotendinous units and therefore affect the pressure within the flexor compartment of the forearm.

While the carpal tunnel may appear to be an open compartment anatomically, it functions as a relatively closed compartment with respect to transfer of pressure from the flexor compartment of the forearm. The flexor compartment is closed with respect to transfer of pressures to the carpal tunnel under conditions that mimic elevated tissue pressure within a physiologic range.

The authors thank Dean R. Fisher, Department of Anatomy, for help with cadaveric specimens.

References

1. Szabo RM, Chidgey LK. Stress carpal tunnel pressures in patients with carpal tunnel syndrome and normal patients. J Hand Surg 1989;14A:624–7.
2. Rydholm U, Werner CO, Ohlin P. Intracompartmental forearm pressure during rest and exercise. Clin Orthop 1983;175:213–5.
3. Gelberman RH, Hergenroeder PT, Hargens AR, Lundborg GN, Akeson WH. The carpal tunnel syndrome: a study of carpal canal pressures. J Bone Joint Surg 1981;63A:380–3.
4. Cobb TK, Cooney WP, An K-N. Relationship of deep structures of the hand and wrist to topographical landmarks. Clin Anat 1993;6:300–7.
5. McDougall CG, Johnston GHF. A new technique of catheter placement for measurement of forearm compartment pressures. J Trauma 1991;31:1404–7.
6. Phalen GS. The carpal-tunnel syndrome: seventeen years' experience in the diagnosis and treatment of six hundred fifty-four hands. J Bone Joint Surg 1966; 48A:211–28.
7. Phalen GS. The carpal-tunnel syndrome: clinical evaluation of 598 hands. Clin Orthop 1972;83:29–40.
8. Smith EM, Sonstegrad DA, Anderson WH Jr. Carpal tunnel syndrome: contribution of flexor tendons. Arch Phys Med Rehabil 1977;58:379–85.
9. Armstrong TJ, Chaffin DB. Some biomechanical aspects of the carpal tunnel. J Occup Med 1979;12: 567–70.
10. Thruston AJ, Krause BL. The possible role of vascular congestion in carpal tunnel syndrome. J Hand Surg 1988;13B:397–9.
11. Reneman RS. The anterior and the lateral compartmental syndrome of the leg due to intensive use of muscles. Clin Orthop 1975;113:69–80.
12. Cobb TK, Dalley BK, Posteraro RH, Lewis RC. Anatomy of the flexor retinaculum. J Hand Surg 1993; 18A:91–9.
13. Cobb TK, Dalley BK, Posteraro RH, Lewis RC. The carpal tunnel as a compartment: an anatomic perspective. Orthop Rev 1992;21:451–3.