Journal of Orthopaedic Research
15:422-426 The Journal of Bone and Joint Surgery, Inc.

Effects of Static Fingertip Loading on Carpal Tunnel Pressure

David Rempel, Peter J. Keir, W. Paul Smutz, and *Alan Hargens

*Ergonomics Laboratory, Department of Medicine, University of California-San Francisco, and *Life Science Division, NASA Ames Research Center, Moffet Field, California, U.S.A.*

Summary: The purpose of this study was to explore the relationship between carpal tunnel pressure and fingertip force during a simple pressing task. Carpal tunnel pressure was measured in 15 healthy volunteers by means of a saline-filled catheter inserted percutaneously into the carpal tunnel of the nondominant hand. The subjects pressed on a load cell with the tip of the index finger and with 0, 6, 9, and 12 N of force. The task was repeated in 10 wrist postures: neutral; 10 and 20° of ulnar deviation; 10° of radial deviation; and 15, 30, and 45° of both flexion and extension. Fingertip loading significantly increased carpal tunnel pressure for all wrist angles ($p = 0.0001$). *Post hoc* analyses identified significant increases ($p < 0.05$) in carpal tunnel pressure between unloaded (0 N) and all loaded conditions, as well as between the 6 and 12 N load conditions. This study demonstrates that the process whereby fingertip loading elevates carpal tunnel pressure is independent of wrist posture and that relatively small fingertip loads have a large effect on carpal tunnel pressure. It also reveals the response characteristics of carpal tunnel pressure to fingertip loading, which is one step in understanding the relationship between sustained grip and pinch activities and the aggravation or development of median neuropathy at the wrist.

High forces of grip and pinch have been identified as independent risk factors for carpal tunnel syndrome in epidemiological studies (2,16). However, the pathophysiology of carpal tunnel syndrome is still uncertain. Indirect evidence suggests that the pathophysiologic mechanism of this condition involves repeated and prolonged elevation of carpal tunnel pressure (6,13). Gelberman et al. (3) proposed that persistently elevated pressure in the carpal tunnel leads to impairment of blood flow, vascular leakage, edema, and eventually, epineural fibrosis.

Smith et al. (18), using a balloon transducer, found that pressure in the region of the median nerve in human cadaveric hands increased when the index and long finger flexor tendons were loaded. They also demonstrated that the nature of the pressure response to loading differed significantly between flexion and extension of the wrist. A number of previous *in vivo* studies demonstrated an increase in carpal tunnel pressure with deviations of the wrist from the neutral position (4,23). In five other studies, carpal tunnel pressure increased with grip or pinch activities, but none of those studies measured fingertip or muscular loading (8-10,15,22). No *in vivo* studies, to our knowledge, have systematically explored the relationship between fingertip loading and carpal tunnel pressure.

The purpose of this study was to determine the relationship between carpal tunnel pressure and fingertip loading. In healthy volunteers, this relationship was examined during a pressing task and repeated in various wrist postures of flexion-extension and radioulnar deviation.

METHODS

Fifteen volunteers (eight women and seven men; age range: 23-50 years) participated in this study after providing written, informed consent. Each participant was interviewed and examined by a hand surgeon for the signs and symptoms of carpal tunnel syndrome and evaluated for muscle strength (thumb opposition, interossei, and grip) and thenar atrophy. Sensation to touch was tested in the hand and fingers, as were Phalen's and Tinel's signs. A neurologist carried out electrodiagnostic studies of the median nerve by recording thenar muscle activity, measuring antidromic sensory conduction between the wrist and index finger, and recording the orthodromic short-segment between the palm and the wrist. The findings of the histories, physical examinations, and nerve conduction studies were normal for all subjects. This study was approved by the Committee on Human Research of the University of California at San Francisco.

After subcutaneous anesthesia was administered, an 18-gauge guide needle was inserted to a depth of 3.5 cm into the nondominant hand at a 45° angle, about 5 mm proximal to the distal volar wrist crease, immediately radial to the palmaris longus (10). Carpal tunnel pressure was measured by means of a saline-filled, blunt-tipped, multiperforated 20-gauge catheter (Burron Medical, Bethlehem, PA, U.S.A.) that was threaded through the needle. The needle was withdrawn over the catheter, and the catheter was connected to a pressure transducer that was maintained at the

Received July 22, 1996; accepted January 22, 1997.
Address correspondence and reprint requests to D. Rempel at University of California-San Francisco, Ergonomics Laboratory, 1301 South 46th Street, Building 112, Richmond, CA 94804, U.S.A. E-mail: rempel@itsa.ucsf.edu

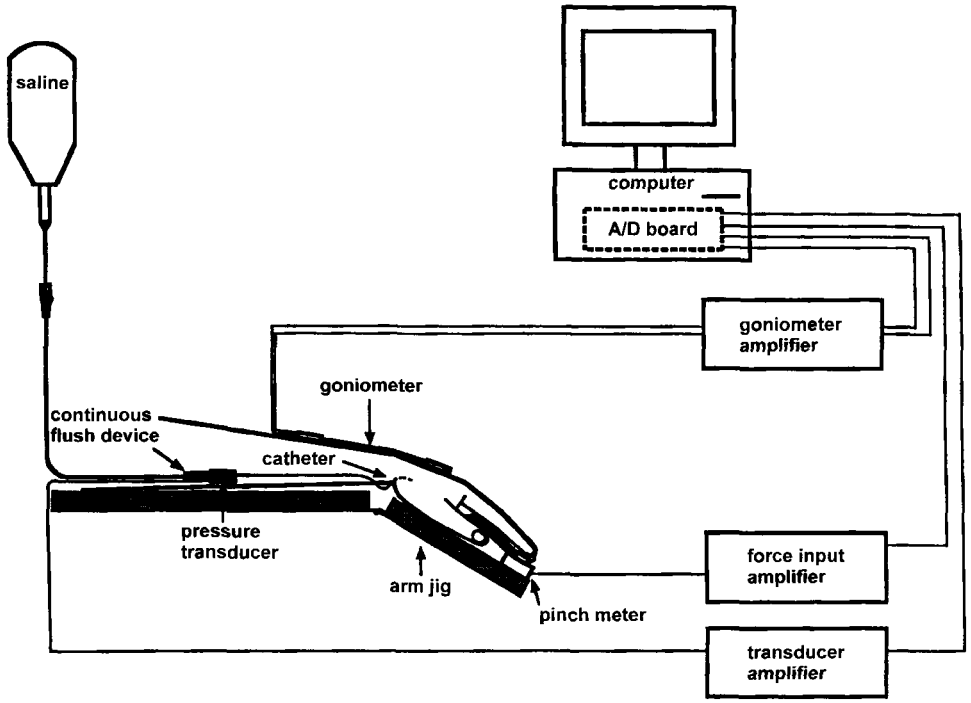

FIG. 1. Schematic drawing of the experimental setup, demonstrating the index finger pressing on an electronic pinch meter with the wrist in a flexed posture. The palm and the distal forearm are not in contact with the arm jig. Carpal tunnel pressure, goniometer values, and pinch force were sampled simultaneously at 40 Hz. A/D = analog to digital.

same elevation as the carpal tunnel. On the basis of the results of pilot studies on cadaveric hands and of the wrist radiographs of three subjects, the catheter tip was located near the center of the carpal tunnel at the level of the hook of the hamate. To minimize the possibility of occlusion, a slight positive flow of physiologic saline at a rate of 0.5 ml/hour was maintained using a low-flow continuous flush device (model 42002-02, Sorenson Intraflo II; Abbott Laboratories, North Chicago, IL, U.S.A.). Radioulnar deviation and flexion-extension angles of the wrist were monitored using a biaxial electrogoniometer (model M110; Penny and Giles, Santa Monica, CA, U.S.A.) that was secured to the dorsal surface of the hand and forearm. We used a previously defined reference system (14) for mounting and calibrating the goniometer; these methods have been described elsewhere (19).

An electronic load cell (Pinch meter; Greenleaf Medical Systems, Palo Alto, CA, U.S.A.) was used to measure fingertip force. The load cell was mounted on a test fixture that allowed for adjustment of the subject's wrist in flexion-extension and radioulnar deviation (Fig. 1). Outputs from the pressure transducer, the goniometer, and the load cell were digitally sampled at 40 Hz and stored on a computer. The calibration protocols for the pressure transducer and goniometer have been described previously (23).

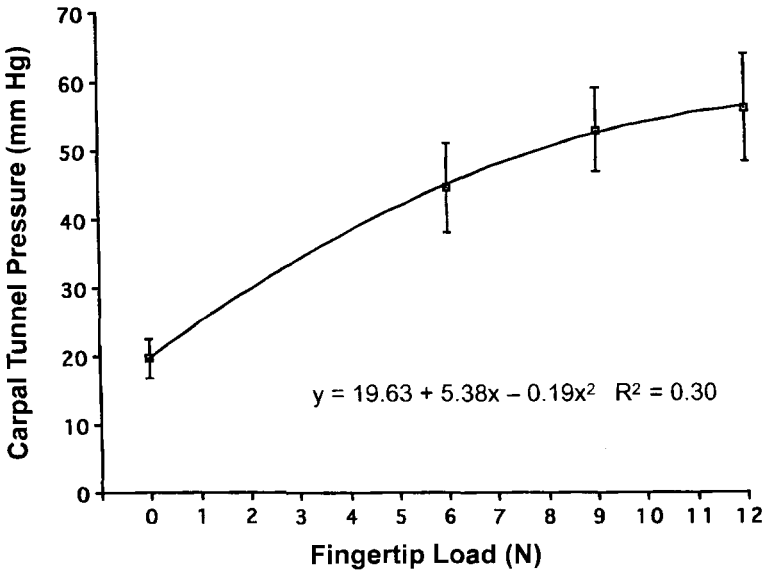

FIG. 2. Mean carpal tunnel pressure relative to fingertip load for 15 subjects with the wrist in the neutral position. Error bars represent SEM. The line represents a second-order polynomial fit of the data.

TABLE 1. *Mean carpal tunnel pressures and SEMs in mm Hg (Pa) of 15 healthy subjects for each of the experimental conditions.*

Wrist angle	Fingertip force (N)			
	0	6	9	12
45° of extension	36.0 ± 4.3 (4,799 ± 573)	63.9 ± 8.0 (8,518 ± 1,066)	70.8 ± 10.4 (9,438 ± 1,386)	73.7 ± 10.8 (9,824 ± 1,440)
30° of extension	27.7 ± 4.9 (3,692 ± 653)	53.5 ± 5.6 (7,132 ± 746)	61.6 ± 7.1 (8,211 ± 946)	62.3 ± 6.8 (8,305 ± 906)
15° of extension	18.5 ± 3.9 (2,466 ± 520)	41.1 ± 5.6 (5,479 ± 746)	50.8 ± 6.4 (6,772 ± 853)	52.7 ± 6.2 (7,025 ± 826)
Neutral	19.7 ± 3.0 (2,626 ± 400)	44.6 ± 6.4 (5,945 ± 853)	53.1 ± 6.1 (7,078 ± 813)	56.5 ± 7.9 (7,531 ± 1,053)
15° of flexion	16.9 ± 2.8 (2,253 ± 373)	42.4 ± 9.5 (5,652 ± 1,266)	51.4 ± 9.7 (6,852 ± 1,293)	54.5 ± 10.0 (7,265 ± 1,333)
30° of flexion	18.8 ± 4.2 (2,506 ± 560)	32.9 ± 5.5 (4,386 ± 733)	41.4 ± 5.9 (5,519 ± 786)	43.7 ± 7.1 (5,825 ± 946)
45° of flexion	26.6 ± 3.6 (3,546 ± 480)	41.5 ± 5.4 (5,532 ± 720)	44.0 ± 6.8 (5,865 ± 906)	46.3 ± 8.4 (6,172 ± 1,120)
10° of radial	27.8 ± 4.5 (3,706 ± 600)	48.3 ± 6.9 (6,438 ± 920)	56.6 ± 8.6 (7,545 ± 1,146)	57.9 ± 9.8 (7,718 ± 1,306)
10° of ulnar	15.4 ± 2.9 (2,053 ± 387)	36.1 ± 9.0 (4,812 ± 1,200)	44.9 ± 10.7 (5,985 ± 1,426)	45.9 ± 10.5 (6,118 ± 1,400)
20° of ulnar	21.5 ± 5.0 (2,866 ± 667)	40.9 ± 6.6 (5,452 ± 880)	46.6 ± 7.0 (6,212 ± 933)	47.9 ± 7.7 (6,385 ± 1,026)

The mean baseline pressure, identified before the subject assumed the neutral test posture, was 7.2 ± 5.4 mm Hg (960 ± 720 Pa).

The load cell was calibrated prior to the study, and data from it were found to be nonlinear in the force range of this study. Therefore, a nonlinear correction function was applied to all force data.

The subject was seated with the upper arm at the side, the elbow at 90°, and the forearm parallel to the floor. Initially, the subject freely moved the wrist, forearm, and fingers while the carpal tunnel pressure was observed in real time to identify the lowest, or baseline, carpal tunnel pressure (23). The hand was then placed palm down (forearm pronated) in the test fixture so that only the proximal and mid-forearm was in contact with the fixture; no external pressure was applied to the wrist or palm. Contact in the mid-forearm region does not increase carpal tunnel pressure, according to results from pilot studies. The subject pressed down on the load cell with the tip of the index finger to force levels of 0, 6, 9, and 12 N without changing the position of the fingers or hand. Visual feedback from the pinch meter of the force level was provided to the subject, and the subject was instructed and observed to maintain each force at a constant level for at least 2 seconds. A pilot study of two subjects determined that the pressure remained constant whether the loading duration was 2 or 20 seconds. No other fingers were loaded. The task was repeated with the wrist in 10 different postures: neutral; 10 and 20° of ulnar deviation; 10° of radial deviation; and 15, 30, and 45° of flexion and extension.

The data were analyzed by a repeated measures analysis of variance (ANOVA) with specific *post hoc* analyses using the Tukey-Kramer honestly significant difference (HSD) test ($\alpha = 0.05$).

RESULTS

After the forearm was set in the jig, the mean baseline pressure in the carpal tunnel increased from 7.2 ± 5.4 mm Hg (960 ± 720 Pa) to between 16.9 and 36.0 mm Hg (2,253 and 4,799 Pa), depending on the posture of the wrist. The increase in "resting" pressure can be attributed to the fully pronated forearm posture (11). In the neutral wrist position (0° of flexion-extension and 0° of radioulnar deviation), the mean pressure in the carpal tunnel increased from 19.7 mm Hg (2,626 Pa) in the unloaded state to 44.6, 53.1, and 56.5 mm Hg (5,945, 7,078, and 7,531 Pa) with fingertip loads of 6, 9, and 12 N, respectively. The response of carpal tunnel pressure to fingertip force in the neutral posture (Fig. 2) was fitted with the second-order polynomial, $y = 19.63 + 5.38x - 0.19x^2$ ($R^2 = 0.30$).

Carpal tunnel pressure increased with increasing fingertip loads for all postures of the wrist (Table 1). Repeated measures ANOVA demonstrated that carpal tunnel pressure was significantly affected by fingertip force ($p = 0.0001$) and wrist angle ($p = 0.0013$), but the interaction effect of force and angle was not significant ($p = 0.9551$). Application of the Tukey-Kramer HSD test demonstrated that carpal tunnel pressure in any loaded state (6, 9, or 12 N) was significantly higher than carpal tunnel pressure in the unloaded state (0 N) for all wrist postures tested ($p < 0.05$). The pressure associated with a fingertip force of 12 N was also significantly higher than the pressure with 6 N of fingertip force for all wrist postures ($p < 0.05$). For the target forces of 0, 6, 9, and 12 N, the actual mean forces applied to the load cell were 0.3 ± 0.4, 6.3 ± 0.4, 9.3 ± 0.5, and 12.1 ± 0.5 N, respectively.

DISCUSSION

This is the first study to measure carpal tunnel pressure during controlled fingertip loading *in vivo*, to our knowledge. It demonstrated that carpal tunnel pressure increases with active, static loading of the fingertip and that this increase is independent of the angle of the wrist. Under the conditions of this experiment, finger force had a greater effect than wrist angle on carpal tunnel pressure. In the neutral posture, the relationship between carpal tunnel pressure and fingertip load loosely fit the form of a second-order polynomial.

The pressures recorded with a fingertip load of 0 N in this study (Table 1, column 1) demonstrate a trend of increasing pressure with increasing wrist deviation from neutral, a finding observed by others (20-23). With the arm in a jig, the minimum carpal tunnel

pressure was associated with postures near neutral, with slight flexion and ulnar deviation, as previously reported by Weiss et al. (23). However, the pressure associated with a neutral wrist posture is higher than the absolute lowest pressure measured in the same subjects prior to positioning in the jig and is higher than that reported by other studies of normal subjects (3,7,12,23). This elevated "resting" pressure can be attributed to the fully pronated forearm posture used in the protocol — a posture that independently increases carpal tunnel pressure (11). Therefore, the increased pressures noted during fingertip loading should be considered relative to the "resting" pressure that is determined by posture.

Active gripping is known to increase carpal tunnel pressure (9,10,22,24), but the relationship between level of force and pressure has not been previously quantified, to our knowledge. The shape of the response in our data indicates that, after an initial rise in carpal tunnel pressure with application of low-level forces, there is a plateau or ceiling effect on the relationship between carpal tunnel pressure and fingertip load. Chronic exposure to relatively low-level forces may therefore be sufficient to elevate carpal tunnel pressures to levels associated with ischemia (13) and to initiate events leading to median nerve injury, as suggested by Gelberman et al. (3). In nerve studies on animals, brief exposures to pressures as low as 30 mm Hg (3,999 Pa) caused physiologic changes that persisted for much longer (8).

The protocol for this study involved systematic loading of the extrinsic flexor muscles of the index finger, an experiment previously done only in cadavers (5,18). In general, cadaveric carpal tunnel pressures measured with a technique similar to the one used in this study have been shown to be representative of those *in vivo* (5). However, the effects of fingertip loading on carpal tunnel pressure observed in this study differed somewhat from the results of the cadaver study of Smith et al. (18), in which the median nerve was replaced with a balloon transducer after transection distal to the carpal tunnel. Although Smith et al. observed a large effect of tendon loading with flexion of the wrist, little or no effect was observed with extension of the wrist. We observed a significant effect of loading regardless of wrist posture. The levels of tendon loading in our study were comparable with those of Smith et al. (18) based on tendon force models. Thus, we must attribute the differing results to experimental technique. Cadaveric response may differ from the *in vivo* response due to tissue damage secondary to freezing or to disruption of fluid flow due to alterations in tissues or tissue planes caused by the invasive procedures employed with the cadaver. More importantly, the present study used a multiperforated catheter, while Smith et al. used a bulb apparatus that was inserted into the carpal tunnel where the median nerve had been excised. This bulb technique is more representative of contact pressure than the true hydrostatic pressure that is measured by the multiperforated catheter, and thus the results of Smith et al. may be indicative of a different mechanism (5).

There are no known explanations for the observed increase in carpal tunnel pressure with fingertip loading. It is possible that elevated pressures are due to localized phenomena in the carpal tunnel. Previous studies have shown that gradients of carpal tunnel pressure exist in patients with carpal tunnel syndrome (7). Flexor tendons migrate toward the transverse carpal ligament with wrist flexion (5,17) and may also migrate when the flexor tendons are loaded. This type of tendon movement may create local pressure gradients or regions of elevated pressure within the carpal tunnel.

This study has demonstrated that active fingertip loading, of the type used in a pinch grip, consistently increases carpal tunnel pressure across a wide range of functional postures of the wrist. Although deviations of the wrist from neutral also increase pressure, the effect of fingertip load on pressure is independent of wrist posture. Furthermore, the shape of the load-to-pressure curve is nonlinear. Although it is unlikely that repeated brief episodes of fingertip loading will initiate an ischemic process, relatively low fingertip loads are capable of elevating the carpal tunnel pressure to levels that, if sustained (especially in nonneutral wrist postures), may initiate an ischemic process and trigger symptoms in patients with carpal tunnel syndrome or may initiate events leading to the development of carpal tunnel syndrome. If this theory of the mechanism of disease aggravation is correct, then designing tools that minimize the duration and level of pinch force and of extreme wrist postures may benefit patients and contribute to the prevention of carpal tunnel syndrome.

Acknowledgment: This study was partially supported by Grant K01OH00121-01 from the National Institute of Occupational Safety and Health of the Centers for Disease Control.

REFERENCES

1. An KN, Chao EY, Cooney WP, Linscheid RL: Forces in the normal and abnormal hand. *J Orthop Res* 3:202-211, 1985
2. Armstrong TJ, Chaffin DB: Carpal tunnel syndrome and selected personal attributes. *J Occup Med* 21:481-486, 1979
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 [Am]* 63:380-383, 1981
4. Gelberman RH, Szabo RM, Mortensen WW: Carpal tunnel pressures and wrist position in patients with Colles' fractures. *J Trauma* 24:747-749, 1984
5. Keir PJ: Functional implications of the musculoskeletal anatomy and passive tissue properties of the forearm [Ph.D. dissertation]. Waterloo, Ontario, Canada, University of Waterloo, 1995
6. Lluch AL: Thickening of the synovium of the digital flexor

tendons: cause or consequence of the carpal tunnel syndrome? *J Hand Surg [Br]* 17:209-212, 1992
7. Luchetti R, Schoenhuber R, Alfarano M, Deluca S, De Cicco G, Landi A: Carpal tunnel syndrome: correlations between pressure measurement and intraoperative electrophysiological nerve study. *Muscle Nerve* 13:1164-1168, 1990
8. Lundborg G, Myers R, Powell H: Nerve compression injury and increased endoneurial fluid pressure: a "miniature compartment syndrome." *J Neurol Neurosurg Psychiatry* 46:1119-1124, 1983
9. Okutsu I, Ninomiya S, Hamanaka I, Kuroshima N, Inanami H: Measurement of pressure in the carpal canal before and after endoscopic management of carpal tunnel syndrome. *J Bone Joint Surg [Am]* 71:679-683, 1989
10. Rempel D, Manojlovic R, Levinsohn DG, Bloom T, Gordon L: The effect of wearing a flexible wrist splint on carpal tunnel pressure during repetitive hand activity. *J Hand Surg [Am]* 19:106-110, 1994
11. Rempel DM, Bach J, Gordon L, Tal R: Effects of forearm pronation/supination and metacarpophalangeal flexion on carpal tunnel pressure. Unpublished data
12. Rojviroj S, Sirirchativapee W, Kowsuwon W, Wongwiwattananon J, Tamnanthong N, Jeeravipoolvarn P: Pressures in the carpal tunnel: a comparison between patients with carpal tunnel syndrome and normal subjects. *J Bone Joint Surg [Br]* 72:516-518, 1990
13. Rydevik B, Lundborg G: Permeability of intraneural microvessels and perineurium following acute, graded experimental nerve compression. *Scand J Plast Reconstr Surg* 11:179-187, 1977
14. Ryu J, Palmer AK, Cooney WP: Wrist joint motion. In: *Biomechanics of the Wrist Joint*, pp 37-60. Ed by KN An, RA Berger, and WP Cooney. New York, Springer-Verlag, 1991
15. Seradge H, Jia YC, Owens W: In vivo measurement of carpal tunnel pressure in the functioning hand. *J Hand Surg [Am]* 20:855-859, 1995
16. Silverstein BA, Fine LJ, Armstrong TJ: Occupational factors and carpal tunnel syndrome. *Am J Ind Med* 11:343-358, 1987
17. Skie M, Zeiss J, Ebraheim NA, Jackson WT: Carpal tunnel changes and median nerve compression during wrist flexion and extension seen by magnetic resonance imaging. *J Hand Surg [Am]* 15:934-939, 1990
18. Smith EM, Sonstegard DA, Anderson WH Jr: Carpal tunnel syndrome: contribution of flexor tendons. *Arch Phys Med Rehabil* 58:379-385, 1977
19. Smutz P, Serina E, Rempel D: A system for evaluating the effect of keyboard design on force, posture, comfort, and productivity. *Ergonomics* 37:1649-1660, 1994
20. Szabo RM, Chidgey LK: Stress carpal tunnel pressures in patients with carpal tunnel syndrome and normal patients. *J Hand Surg [Am]* 14:624-627, 1989
21. Tanzer RC: The carpal-tunnel syndrome: a clinical and anatomical study. *J Bone Joint Surg [Am]* 41:626-634, 1959
22. Thurston AJ, Krause BL: The possible role of vascular congestion in carpal tunnel syndrome. *J Hand Surg [Br]* 13:397-399, 1988
23. Weiss ND, Gordon L, Bloom T, So Y, Rempel DM: Position of the wrist associated with the lowest carpal-tunnel pressure: implications for splint design. *J Bone Joint Surg [Am]* 77:1695-1699, 1995
24. Werner C-O, Elmqvist D, Ohlin P: Pressure and nerve lesion in the carpal tunnel. *Acta Orthop Scand* 54:312-316, 1983