Effects of Forearm Pronation/Supination on Carpal Tunnel Pressure

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The effects of forearm rotation and metacarpophalangeal (MP) flexion on carpal tunnel pressure were investigated in 17 healthy adults who had no evidence of carpal tunnel syndrome (CTS). Pressure was continuously recorded with a saline-filled catheter inserted into the carpal tunnel and connected to a pressure transducer while test subjects slowly rotated the forearm from full pronation to full supination. Forearm rotation was repeated with MP flexion of 0°, 45°, and 90°. Both forearm rotation and MP flexion, and their interaction term, significantly affected carpal tunnel pressure and accounted for most of the variability in the data. Highest mean pressures (55 mmHg) were recorded in full supination and 90° MP flexion and lowest pressures (12 mmHg) were recorded at 45° pronation and 45° MP flexion. These data may be useful in the design of tasks and hand tools in the management and prevention of CTS. (J Hand Surg 1998;23A:38–42. Copyright © 1998 by the American Society for Surgery of the Hand.)

Carpal tunnel syndrome (CTS) has been attributed to repetitive, hand-intensive work, specifically work involving static, awkward wrist postures.[1] It is unlikely that epidemiologic studies will ever clarify the dose–response relationship of posture to the risk of aggravating or causing CTS. It is possible, however, to determine the dose–response relationship between changes in posture and changes in carpal tunnel pressure. If sustained, elevated pressure retards long-term improvement of CTS or precedes the onset of CTS, then knowledge of how postures influence pressure can be used for managing or preventing CTS.

Although carpal tunnel pressure is higher in patients with CTS than in healthy people,[2–6] the exact role of daily changes in carpal tunnel pressure in the pathophysiology of CTS is uncertain. Gelberman et al. have proposed that prolonged elevated pressures within the carpal tunnel limit microvascular flow and lead to protein leakage, epineural edema, and, eventually, epineural fibrosis.[2] In human and animal studies, it has been found that fluid pressures of 40–50 mmHg sustained for 60 minutes can cause transient changes in nerve function.[7,8] Elevated carpal tunnel pressure can also cause more prolonged effects on tissues. For example, pressures of 30 mmHg applied to the sciatic nerves of rats for 4 hours will cause edema and a persistent elevation of endoneurial pressure that lasts for up to 24 hours.[9] In addition, histologic examinations of the flexor tendon sheaths of patients with CTS demonstrate edema and vascular changes consistent with an ischemic process,[10,11] and it has been proposed that synovial hypertrophy leads to further increases in pressure.[12] Although a critical pressure–

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time threshold associated with CTS has yet to be determined, the pressure is likely to be below the critical threshold apparent for acute median neuropathy, which is approximately 30 mmHg.[2,13] Overall, these studies suggest that if we understand the factors that change carpal tunnel pressure, we will be better equipped to manage and prevent CTS.

Wrist flexion/extension,[3,14] ulnar/radial deviation,[14] and fingertip loading[15] have been shown to modify carpal tunnel pressure, but the role of pronation/supination has not been examined. In addition, metacarpophalangeal (MP) joint flexion and the resulting incursion of the lumbricals into the carpal tunnel have been shown to increase carpal tunnel pressure in cadaver hands,[16] but no *in vivo* studies have been conducted to confirm these findings. This laboratory experiment was designed to evaluate the effects of forearm pronation/supination and MP joint angle on the carpal tunnel pressure of healthy test subjects.

Materials and Methods

Seventeen test subjects (mean age, 31 ± 6 years) without symptoms, signs, or electrodiagnostic evidence of CTS participated in the study. The nondominant hand was used in the experiment. Test subjects had no paresthesias in the median nerve distribution and had no disorders associated with peripheral neuropathies (e.g., diabetes, thyroid disease). One neurologist (Y. S.) conducted tests for muscle strength (interossei and grip), thenar atrophy, sensibility in the fingers, and Phalen and Tinel signs and administered the electrodiagnostic tests. The electrodiagnostic tests of the median nerve were performed by recording from the thenar muscle, measuring antidromic sensory conduction between the wrist and index finger, and recording the orthodromic short segment between the palm and wrist. The palmar skin temperature was maintained at or above 31°C.

Carpal tunnel pressure measurement was accomplished with a blunt-tipped 20-gauge (0.8-mm–diameter) saline-filled catheter inserted percutaneously into the carpal tunnel using an 18-gauge epidural needle as previously described.[17] The needle was inserted at a 45° angle approximately 5 mm proximal to the distal volar wrist crease, immediately radial to the tendon of palmaris longus. Needle insertion was performed under local anesthesia. After the needle was withdrawn over the catheter, the catheter was attached at its proximal end to an in-line pressure transducer (CDXPress, Cobe Cardiovascular, Arvada, CO) and the transducer was affixed to the forearm midway between the wrist and the elbow with tape. Each test subject sat upright with the upper arm hanging passively at the side and the elbow held in 90° flexion so that the pressure transducer remained level with the carpal tunnel.

Output from the transducer was amplified (ProPac 104, Protocol Systems, Beaverton, OR) and stored on a computer. To minimize the possibility of occlusion, a slight positive flow (0.5 mL/h) of physiologic saline was maintained using a low-flow continuous-flush device built into the pressure transducer. The transducer was calibrated at room temperature with a 70-cm H_2O fluid column. A baseline pressure was collected with the hand in the position associated with the lowest carpal tunnel pressure.[14] The mean baseline pressure was 8 ± 6 mmHg.

Pronation/supination and MP joint angles were measured using manual goniometers.[18] The accuracy of these devices was considered to be ± 5°.[19] Beginning with the wrist in 0° flexion/extension and 0° ulnar/radial deviation, the test subject slowly moved his or her fingers until an MP angle of 0°, 45°, or 90° was reached. The test subject then very slowly began to supinate the forearm. At full supination, the test subject reversed direction and slowly rotated the arm to full pronation and finally back to the resting position. The movements were practiced prior to data collection while the joints were observed and measured to ensure that the wrist position of neutral flexion/extension and radial/ulnar deviation remained constant throughout forearm rotation. The motion from full supination to full pronation was completed in approximately 1 minute. The experimenter continuously measured the pronation/supination angle manually and time-marked the data at 45° rotational intervals throughout the cycle. Cycles were then performed at each of the 2 remaining MP angles.

The data were analyzed with repeated-measures analysis of variance using the JMP statistical analysis package (SAS Institute, Cary, NC). A 2-factors repeated-measures analysis was performed using 2 within-subjects factors—pronation/supination angle and MP angle—with carpal tunnel pressure as the outcome measure. The pronation/supination angle factor had 5 levels and the MP factor had 3. A Tukey's studentized range test was performed using the JMP results to determine significant differences between the various levels of the 2 factors.

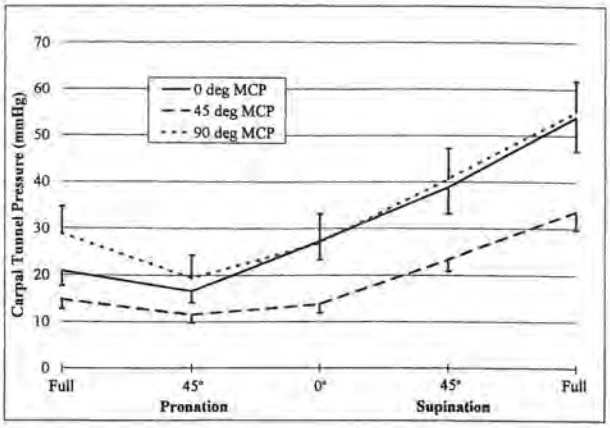

Figure 1. Graph of the mean carpal tunnel pressure versus forearm pronation/supination angle for 17 healthy test subjects. The data are shown for 3 different metacarpophalangeal (MCP) angles. The error bars represent standard errors. deg, degrees.

Results

The carpal tunnel pressure values from all subjects were combined to determine means and standard errors for each pronation/supination–MP angle combination. These are plotted in Figure 1 as carpal tunnel pressure versus pronation/supination angle for each of the 3 MP angles. The same data are represented in Figure 2 as carpal tunnel pressure versus MP flexion at each of the 5 pronation/supination angles to demonstrate the contribution of MP flexion. The minimum carpal tunnel pressure occurred at 45° pronation for all MP angles (17 mmHg at 0° MP angle, 12 mmHg at 45°, and 19 mmHg at 90°). The mean carpal tunnel pressures at full supination were 54, 34, and 55 mmHg at the respective MP joint angles of 0°, 45°, and 90°. Standard errors ranged from 1.8 to 7.3 mmHg.

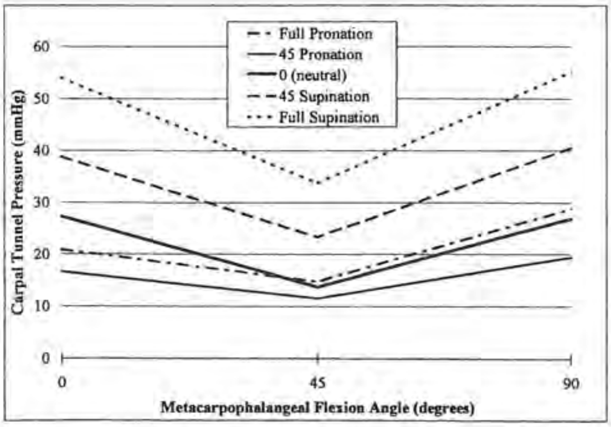

Figure 2. Graph of the mean carpal tunnel pressure versus metacarpophalangeal angle for 17 healthy test subjects at 5 forearm pronation/supination angles. These are the same data as in Figure 1.

Table 1. Mean Carpal Tunnel Pressures (mmHg) at Different Forearm Rotation and Metacarpophalangeal Angles (n = 17)

	Metacarpophalangeal Angle		
Forearm Rotation Angle	*0°*	*45°*	*90°*
Full supination	54*	34*	55*
45° supination	39†	23†	41†
0°	27‡	14‡	27‡§
45° pronation	17§	12‡	19§
Full pronation	21‡§	15‡	29‡

Significant effects ($p < .05$) of joint angles on pressure are indicated by the superscripts (Tukey's studentized range test); within a column, values with the same superscript are not significantly different.

The repeated-measures analysis of variance revealed that the effects on carpal tunnel pressure of pronation/supination angle ($F = 44.4$, $p = .0001$), MP angle ($F = 5.9$, $p = .007$), and their interaction term (pronation/supination multiplied by metacarpophalangeal) ($F = 4.0$, $p = .0003$) were significant. Follow-up tests using Tukey's method of multiple comparison are presented in Table 1. In the table, means that differ by more than 8.84 mmHg are significantly different at the $\alpha = .05$ level and are identified by different superscripts. For example, the pressure increases significantly when rotating the forearm from 0° to 45° supination and from 45° supination to full supination across all MP angles. When the forearm is in pronation, the trend is that the pressure is lowest at 45° pronation, although this difference is only statistically significant at 90° of MP flexion.

Discussion

This *in vivo* study evaluated the effect of forearm pronation/supination and MP flexion on carpal tunnel pressure. In general, the tissues within the carpal tunnel are subjected to increasing fluid pressure as the forearm is rotated from 45° of pronation to full supination. In addition, the pressure is the lowest at an MP flexion angle of 45° and increases with full MP flexion or extension. Because there is a nonadditive interaction between pronation/supination and

MP angles, the pressure is further elevated when these pronation/supination and MP flexion postures occur in combination.

The mechanism whereby pronation/supination increases carpal tunnel pressure is unclear. We suggest 2 possibilities: (1) pronation/supination changes the orientation of the tendons passing through the tunnel, altering the volume and thereby the pressure; (2) the proximal boundary of the "effective" carpal tunnel space (as defined by the flexor muscle bellies) translates and/or changes shape with pronation/supination. We could identify no studies that address these possible mechanisms.

A study of cadaver hands[16] has shown that lumbrical incursion into the distal end of the carpal tunnel leads to an elevated pressure. This incursion, or the effect of skin folding in the palm, may explain the increase in pressure as the fingers are flexed beyond 45° but does not explain the increase in pressure as the MP joints are extended. Our hypothesis regarding this mechanism is that as the MP joints are extended relative to 45°, the muscle bellies of the finger flexors, particularly flexor digitorum superficialis, move distally into the "effective" carpal tunnel space. This invasion of the carpal tunnel space by the distal end of these muscles would act as a space-occupying lesion, resulting in an elevated carpal tunnel pressure.

The results of our study provide additional information into the mechanism that may underlie the relationship between finger and forearm position and CTS. It has previously been shown that carpal tunnel pressure varies with wrist flexion/extension and ulnar/radial deviation,[2,3,5,14,20–24] with static fingertip loading,[15,24] and with palm loading.[25] These findings, in conjunction with our results, indicate that carpal tunnel pressure is a complex function of finger, wrist, and forearm postures, finger loads, and hand loads.

The extension/flexion and ulnar/radial deviation postures associated with lowest carpal tunnel pressure[14] can now be expanded to include a forearm rotation angle near 45° pronation and an MP joint angle of 45°. This set of postures should be considered during the design of hand-intensive tasks and hand tools in order to minimize carpal tunnel pressure during repetitive activity. These postures can also assist in planning rehabilitation for patients with CTS. Splint designs and usual daily hand postures that prevent prolonged, elevated pressure will provide maximal blood flow and nutrient supply to the tissues in the carpal tunnel. If carpal tunnel pressure plays a role in the cause of activity-related CTS, then redesigning tools and tasks to minimize carpal tunnel pressure might decrease the risk of developing CTS.

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