Effects of Finger Posture on Carpal Tunnel Pressure During Wrist Motion

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Persistent elevations in carpal tunnel pressure may aggravate carpal tunnel syndrome. This study examined the effects of finger posture on carpal tunnel pressure during wrist motion. Carpal tunnel hydrostatic pressure was measured using a saline-filled catheter inserted into the nondominant wrists of 14 healthy individuals. Range of motion tasks of wrist flexion-extension and radioulnar deviation were repeated with metacarpophalangeal (MCP) joint angles of 0°, 45°, and 90° flexion. Pressures were significantly greater with the fingers straight (MCP = 0°) than when the MCP joints were flexed to 45° for all radioulnar deviation angles and from 10° of wrist flexion to all angles of wrist extension tested. Pressures were also significantly higher with MCP joints at 0° than at 90° for wrist extension angles from 10° to 40°. Pressures increased to over 30 mm Hg (4.0 kPa) in some wrist extension and ulnar and radially deviated postures. Finger and wrist postures should be considered when designing splints or evaluating tasks for patients with carpal tunnel syndrome. (J Hand Surg 1998;23A:1004–1009. Copyright © 1998 by the American Society for Surgery of the Hand.)

The conservative management of carpal tunnel syndrome (CTS) remains somewhat controversial because the mechanisms for its development and aggravation are not fully understood. As knowledge of the specific hand postures associated with CTS increases, rehabilitation can be improved by altering the design of splints, tools, and common tasks.[1] Although epidemiologic studies have associated pinch force, awkward wrist postures, and repetitive motion with CTS,[2,3] an understanding of underlying mechanisms is necessary to advance conservative management and prevention.

Carpal tunnel pressure (CTP) is an important factor in the pathophysiology of CTS, although the evidence linking persistent elevated pressure to the development of CTS is indirect. Carpal tunnel syndrome patients have elevated CTPs compared with healthy control subjects.[1,4–10] Furthermore, extreme wrist postures will increase CTP, and these postures are associated with the development of CTS in epidemiologic studies.[5,6,9–12] Luchetti et al[8] identified a correlation between CTP and nerve conduction. The reduction in sensory conduction velocity action potential amplitude was greatest in the distal portion of the carpal tunnel where the pressure was highest. Experimentally increased CTPs can produce short-term sensory and motor nerve conduction deficits and elicit the symptoms of median neuropathy.[13]

Of the many studies of CTP, only 5 have specifically reported finger postures in the design of the study,[5,7,12,14,15] 2 of which formed a grip or fist,[5,7] which adds an element of muscular force (known to increase CTP[11,16,17]) and may also include palm con-

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tact by the fingers. Some studies report a "rest posture" of the fingers but do not define this posture. Werner et al[12] measured pressure in CTS patients undergoing surgery during passive, maximal wrist flexion-extension motions with straight and flexed fingers. These investigators found a relatively small difference between the pressures at either extreme of motion. In another study, Werner et al[14] found pinch and relaxed finger postures to result in slightly lower pressures than a closed hand or straight fingers with the wrist in a neutral posture.

Recent studies have implicated lumbrical incursion into the carpal tunnel as a possible mechanism for CTS. In patients undergoing carpal tunnel release[18] and in cadavers,[19] it has been demonstrated that the lumbricals enter the distal end of the carpal tunnel during finger flexion. Using a cadaver model, Cobb et al[15] found that the effects of finger flexion on CTP were negated by removal of the lumbricals, strengthening their hypothesis that the lumbricals enter the carpal tunnel and act to increase the pressure. The results of these studies present a feasible mechanism for the pressure increase associated with finger and wrist flexion, but the mechanism cannot explain the higher pressures observed with wrist extension.[1]

These studies demonstrate the need to accurately determine the effects of finger posture on CTP over a range of functional wrist postures. Although other studies have evaluated the effects of extreme wrist postures (eg, full flexion and full extension) on CTP, these postures are seldom maintained during normal daily activities. The aim of this study was to determine the effect of metacarpophalangeal (MCP) joint posture on CTP during functional wrist motions of flexion-extension and radioulnar deviation. These data may add to our understanding of causal or aggravating mechanisms and may improve strategies for the management and prevention of CTS.

Materials and Methods

Fourteen volunteers (7 women and 7 men with a mean age of 31.5 ± 8.2 years) participated in this study after providing written informed consent. The subjects had no evidence of CTS based on their histories, physical examinations, and nerve conduction studies. Specifically, each participant had no symptoms of paresthesia, pain, or weakness in the hands. Muscle strength (thumb opposition, interossei, grip), thenar atrophy, and sensation to touch in the hand and fingers were evaluated, as were Phalen's and Tinel's signs. A neurologist conducted electrodiagnostic examination of the median nerve, which included thenar muscle recording, antidromic sensory conduction between the wrist and index finger, and orthodromic short-segment between the palm and the wrist. This study was approved by the Committee of Human Research of the University of California at San Francisco.

Carpal tunnel pressure was measured using a saline-filled, blunt-tipped, multiperforated, 20-gauge catheter (Burron Medical, Inc, Bethlehem, PA). Under local anesthesia (injected just subcutaneously at the site of insertion, not into the carpal tunnel), the catheter was inserted percutaneously into the carpal tunnel of the nondominant hand and connected to a pressure transducer, as described previously.[20] Based on pilot studies in cadaver hands and on wrist x-rays of 3 subjects, the catheter tip was located near the center of the carpal tunnel at the level of the hook of hamate. During finger flexion and extension, the catheter did not move at the insertion site. It did, however, move slightly (approximately 5 mm) during wrist flexion and extension (this small movement should not affect pressure measurement in healthy wrists[8]). A suture was used to maintain the position of the catheter while allowing this slight movement. The pressure transducer was maintained at the same elevation as the carpal tunnel. A slight positive flow of physiologic saline at a rate of 0.5 mL/hr was maintained using a low-flow continuous-flush device (model 42002-02; Sorenson Intraflow II, Abbott Laboratories, N. Chicago, IL) to minimize the possibility of occlusion. Radioulnar deviation and flexion-extension angles of the wrist were monitored using a biaxial electrogoniometer (model M110; Penny and Giles, Inc, Santa Monica, CA) secured to the dorsum of the hand and forearm. Methods of goniometer mounting and calibration have been described previously,[21] as has the calibration of the pressure transducer.[1] Pressure and goniometer data were sampled at 40 Hz, then reduced (by averaging) and stored at 4 samples/s for all tasks in this study.

After insertion of the catheter, the subject was seated with the upper arm vertical and adjacent to the body and the elbow flexed to 90°. The subject was then instructed to move the forearm and wrist through a full range of motion, which included flexion and extension, radioulnar deviation, pronation, and supination. During this task, the posture of lowest pressure was recorded. Each subsequent task was initiated from this posture. The subject then progressed through a series of flexion-extension and

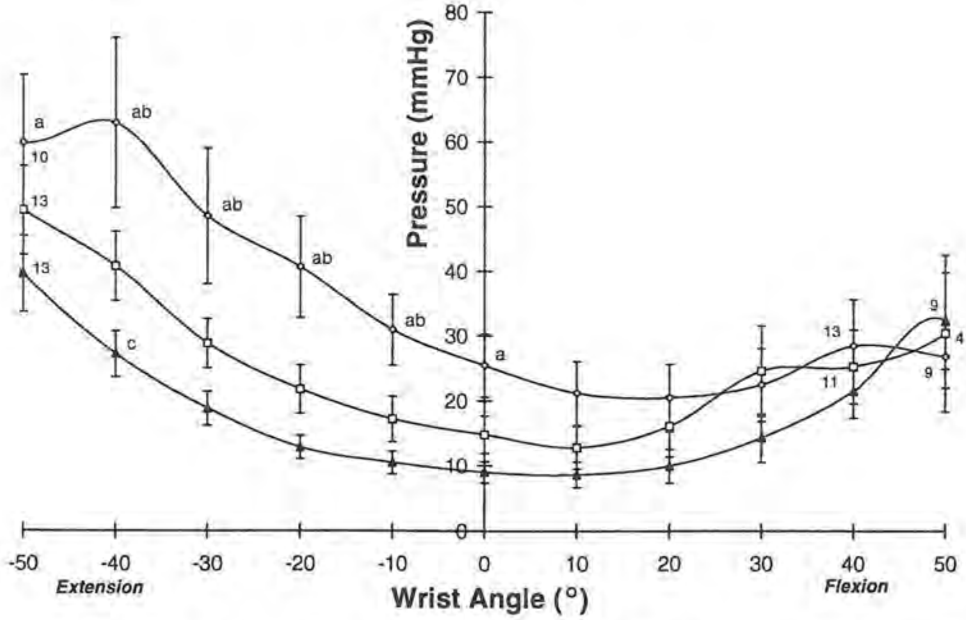

Figure 1. Mean CTP (±SE) of 14 healthy volunteers during wrist flexion and extension with 3 finger postures. Letters beside the data points indicate the significant critical difference of 13.02 mm Hg ($\alpha = 0.01$) from Tukey's studentized range test at each wrist angle: a, MCP 0° versus 45°; b, MCP 0° versus 90°; and c, MCP 45° versus 90°. Numbers beside the data points indicate the number of subjects who reached the angle (if <14). Open diamond, MCP 0°; solid triangle, MCP 45°; open box, MCP 90°.

radioulnar deviation tasks. A flexion-extension task consisted of the subject actively extending the wrist to a comfortable end range of motion, then flexing the wrist to a comfortable end range. This was repeated such that 3 complete motions from end range to end range were performed, with each angle being crossed 4 times. The same procedure was completed in radioulnar deviation. These 2 range of motion tasks were completed with 3 different finger postures: the MCP joint at 0° (MCP and interphalangeal [IP] joints straight), the MCP joint at 45° (IP joints in a relaxed position), and the MCP joint at 90° (IP joints in a relaxed position). The relaxed proximal IP joint angle was typically less than 20° and the relaxed distal IP angle was typically 5° or less. Before collection of the data, subjects were trained using a manual goniometer to ensure MCP angles (0°, 45°, and 90°). This training also ensured that the relaxed IP angles were maintained throughout the range of motion and between tasks.

Mean pressures at each 10° increment in wrist angle were calculated using the data from the 3 complete motions. The effect of finger flexion on CTP was analyzed using a repeated-measures ANOVA with follow-up tests at each wrist angle using Tukey's studentized range test.

Results

The results of the repeated-measures ANOVA for the flexion-extension tasks found the main effects of MCP angle and wrist angle to be significant ($p < .0001$). The interaction of these effects was also significant with $p < .0001$. The significant interaction effect is made evident by the convergence of the curves of the 3 MCP postures with wrist flexion (Fig. 1). Post hoc comparisons were accomplished by performing a separate Tukey's studentized range test at each wrist angle using $\alpha = 0.01$, resulting in an overall procedure-wise error rate of 0.11 (11 angles each tested with $\alpha = 0.01$). The critical difference was 13.02 mm Hg, which is identified by letters on Figure 1. An MCP joint angle of 0° induced significantly greater pressures than an MCP flexion of 45° for all wrist extension angles and up to 10° of wrist flexion. Similarly, the pressures with 0° of MCP flexion were significantly greater than with 90° of MCP flexion for wrist angles between 10° and 40° of extension (inclusive). In the midrange wrist extension (20° to 40°), these differences were approximately 30 mm Hg and 20 mm Hg for MCP joint angles of 45° and 90°, respectively (0.134 mm Hg = 1 kPa).

The results of the repeated-measures ANOVA for the radioulnar deviation tasks determined the main effects of MCP angle and wrist radioulnar angle to be statistically significant ($p < .0001$). The interaction of these terms was not significant ($p = .7677$). Using Tukey's test, significantly higher CTPs were obtained with the MCP joints at 0° flexion than with the MCP joints flexed to 45° ($\alpha = 0.05$). Pressures were higher with 0° MCP flexion than with the fingers flexed to 90°, but the difference was not significant. Similarly, the difference between the 2 flexed finger postures was not significant.

Discussion

The angle of the MCP joint had a significant effect on CTP during active wrist flexion-extension and radioulnar maneuvers. Motions performed with 0° MCP flexion were associated with the highest pressures, followed by an MCP angle of 90°, the lowest pressures occurred with 45° of MCP flexion. The difference between finger postures was greatest with the wrist extended (Fig. 1). At 40° of wrist extension (the greatest wrist extension angle attained by all subjects), the mean pressures were 63 mm Hg, 27 mm Hg, and 41 mm Hg for MCP angles of 0°, 45°, and 90°, respectively. The 0° MCP flexion finger posture increased CTP to a much greater extent than the other 2 postures, surpassing 30 mm Hg by a wrist extension angle of 10°. This pressure level has been shown to damage isolated nerves[22] and exacerbate the symptoms of CTS.[23]

Few previous studies examining CTP *in vivo* have explicitly stated finger posture and only 2 investigated the effect of finger posture on CTP. Given the present results, interpretation of CTP without knowledge of the finger postures may be cause for concern, especially considering the pressure differences between finger postures with wrist extension. Werner et al[12] found smaller differences than the present study for maximal passive wrist flexion and extension with the fingers straight (wrist flexion, 75 ± 59 mm Hg; extension, 105 ± 56 mm Hg) and flexed (wrist flexion, 60 ± 29 mm Hg; extension, 113 ± 53 mm Hg) in CTS patients. It is interesting that these investigators found that straight fingers resulted in higher pressures in the flexed wrist and lower pressures in the extended wrist. Werner et al[14] found very small differences between the finger postures with wrist flexion and extension, but their wrist extension angle data were stratified into 4 categories, which may have masked the differences seen in this study. In the present study, the difference in pressure due to finger posture was greatest with the wrist extended; the difference between an MCP angle of 0° and an MCP angle of 45° was as much as 35 mm Hg. At 50° of wrist extension, the mean pressures were 60 mm Hg and 40 mm Hg for MCP angles of 0° and 45°, respectively. Similarly, at 50° of wrist flexion, the mean pressures were 27 mm Hg and 32 mm Hg for the same angles. For wrist flexion angles of 20° and greater, finger posture does not seem to have much effect on CTP (Fig. 1). Thus, the results of this study indicate that studies measuring CTP can reduce measurement variances by maintaining constant finger joint postures between trials and subjects.

In this study, subjects extended IP joints with 0° MCP flexion while using relaxed IP joints with 45° or 90° MCP flexion. A recent study indicated that IP joint flexion from fully extended to relaxed (as used in this study) had little effect on CTP (Bach and Rempel, presented at the 43rd Annual Meeting of the Orthopaedic Research Society, 1997). The postures used in this study were selected to minimize the intrinsic finger muscle forces required to maintain a flexed MCP joint while the IP joints are extended. Activation of the intrinsic finger musculature may have induced undesirable pressure changes that would have masked those induced by changes in MCP joint angle.

Plausible biomechanical mechanisms for these findings emerge from an examination of the anatomy of the carpal tunnel. Although changes in the cross-sectional area of the carpal tunnel with wrist motion have been documented as a possible factor for increasing CTP,[24] this mechanism can be excluded from the present discussion as finger posture should have no effect in altering the shape of the carpal tunnel. Potential mechanisms that may increase pressure with finger flexion include folding of skin at the distal palm and lumbrical movement into the carpal tunnel[15,18,19] (both mechanisms would increase pressure with increasing flexion). A potential mechanism for a pressure increase with finger extension is flexor muscle belly movement into the carpal tunnel. The latter 2 mechanisms both relate directly to the movement of the flexor digitorum profundus from which the lumbrical muscles originate.

Using the lumbrical incursion data reported by Cobb et al[19] and the regression equations for tendon excursion developed by Armstrong and Chaffin,[25] we can evaluate these possible mechanisms. A fifth percentile female wrist was used for all calculations as the excursions of such a wrist should represent the

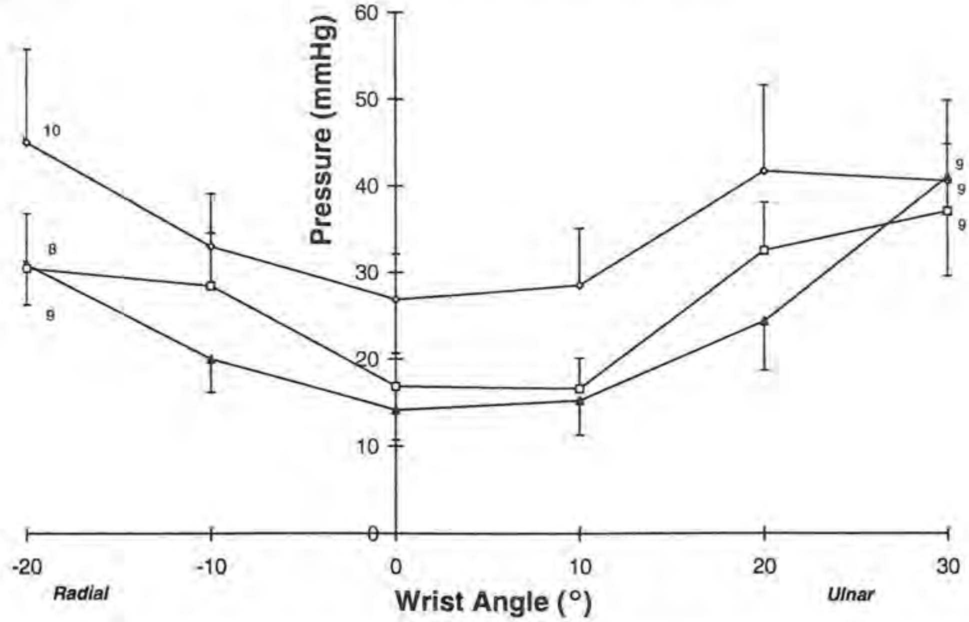

Figure 2. Mean CTP (±SE) of 14 healthy volunteers during radioulnar deviation of the wrist using 3 finger postures. A follow-up Tukey's test ($\alpha = 0.05$) indicated that MCP angles of 0° induced significantly greater pressures than the MCP angles of 45°. Numbers beside the data points indicate the number of subjects who reached the angle (if <14). Open diamond, MCP 0°; solid triangle, 45°; open box, 90°.

smallest magnitudes one is likely to see and thus represent conservative estimates of tendon excursion. Using the calculated excursion values, we expect the lumbricals to enter the distal end of the carpal tunnel in all postures of radioulnar deviation and all flexion-extension angles tested with the MCP joints flexed to 90° and most flexion-extension angles with the MCP flexed to 45°. The lumbricals are always within the carpal tunnel with the MCP joints flexed to 90°, even at 50° wrist extension. This may explain the difference between the 45° and 90° MCP joint conditions in the wrist extension portion of the curve (Fig. 1). With only 45° MCP flexion, we expect that in some patients the lumbricals will not enter the distal end of the carpal tunnel until a more neutral wrist posture is approached (eg, 20° extension).

Neither movement of the lumbrical muscles nor palmar skin folding can explain the higher pressures found with the fingers and wrist extended, where the lumbricals in some individuals can be assumed to remain distal to the carpal tunnel (until near 20° of wrist flexion). It also appears that there is relatively little effect of finger posture when the wrist is flexed past 30°, where the 3 curves are seen to converge (Fig. 1). Based on unpublished data from our laboratory, the distal reaches of the extrinsic flexor muscle bellies are located such that in some individuals the muscle bellies will move past the pisiform (often used to denote the proximal border of the carpal tunnel) with wrist extension. On average, flexor muscle bellies will enter the carpal tunnel by 30° of wrist extension. This would provide a mechanism that acts in the opposite manner to lumbrical movement, ie, with wrist and finger extension, the flexor muscle bellies may enter the proximal end of the tunnel.

The interaction effects between wrist and finger posture (wrist flexion-extension data) indicate that there is most likely a wrist flexion angle beyond which there is no further effect of lumbrical incursion. With radioulnar deviation, the location of the lumbrical and flexor muscle bellies will remain relatively constant in relation to the carpal tunnel. Thus, one would expect the constant differences in pressure as observed in the radioulnar deviation data (Fig. 2; lack of a wrist angle-finger posture interaction effect in radioulnar deviation, ANOVA). This strengthens support for the pressure-elevating mechanisms of both lumbrical and flexor muscle belly incursions since both of these mechanisms are dependent on flexion-extension motions only.

The continuous CTP recording technique combined with the range of finger postures used in this study provide insight into factors affecting CTP. Finger flexion is an important factor in determining CTP. A reduction in CTP can be obtained by avoiding full finger extension, especially when combined with wrist extension. This information may be relevant to the design of splints and tasks in the management of patients with CTS. This study is limited to a population of healthy individuals; therefore, extrapolation to the CTS individual wrist may not be appropriate, although past studies have shown that CTS wrists demonstrate trends in flexion and extension similar to those of healthy wrists with higher CTP magnitudes.[1] If the trends are similar in patients with CTS, the information will be relevant to the design of splints, tasks, and training of patients for appropriate medical management. These findings, however, should be confirmed in patients with CTS.

References

1. 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 1995;77A:1695–1699.
2. Marras WAS, Schoenmarklin RW. Wrist motions in industry. Ergonomics 1993;36:341–351.
3. Silverstein BA, Fine LJ, Armstrong TJ. Occupational factors and carpal tunnel syndrome. Am J Ind Med 1987;11:343–358.
4. Hamanaka I, Okutsu I, Shimizu K, Takatori Y, Ninomiya S. Evaluation of carpal canal pressure in carpal tunnel syndrome. J Hand Surg 1995;20A:848–854.
5. Seradge H, Jia YC, Owens W. In vivo measurement of carpal tunnel pressure in the functioning hand. J Hand Surg 1995;20A:855–859.
6. 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 1990;72B:516–518.
7. 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 1989;71A:679–683.
8. Luchetti R, Schoenhuber R, Alfarano M, Deluca S, DeCicca G, Landi A. Carpal tunnel syndrome: correlations between pressure measurement and intraoperative electrophysiological nerve study. Muscle Nerve 1990;13:1164–1168.
9. Szabo RM, Chidgey LK. Stress carpal tunnel pressures in patients with carpal tunnel syndrome and normal patients. J Hand Surg 1989;14A:624–627.
10. 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:380–383.
11. Thurston AJ, Krause BL. The possible role of vascular congestion in carpal tunnel syndrome. J Hand Surg 1988;13B:397–399.
12. Werner C, Elmqvist D, Ohlin P. Pressure and nerve lesion in the carpal tunnel. Acta Orthop Scand 1983;54:312–316.
13. Gelberman RH, Szabo RM, Williamson RV, Hargens AR, Yaru NC, Minteer-Convery MA. Tissue pressure threshold for peripheral nerve viability. Clin Orthop 1983;178:285–291.
14. Werner R, Armstrong TJ, Bir C, Aylard MK. Intracarpal canal pressures: the role of finger, hand, wrist and forearm position. Clin Biomech 1997;12:44–51.
15. Cobb TK, An K, Cooney WP. Effect of lumbrical muscle incursion within the carpal tunnel on carpal tunnel pressure: a cadaveric study. J Hand Surg 1995;20A:186–192.
16. Smith EM, Sonstegard DA, Anderson WH. Carpal tunnel syndrome: contribution of flexor tendons. Arch Phys Med Rehabil 1977;58:379–385.
17. Keir PJ, Wells RP, Ranney DA, Lavery W. The effects of tendon load and posture on carpal tunnel pressure. J Hand Surg 1997;22A:628–634.
18. Yii NW, Elliot D. A study of the dynamic relationship of the lumbrical muscles and the carpal tunnel. J Hand Surg 1994;19B:439–443.
19. Cobb TK, An K, Cooney WP, Berger RA. Lumbrical muscle incursion into the carpal tunnel during finger flexion. J Hand Surg 1994;19B:434–438.
20. 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 1994;19A:106–110.
21. Smutz P, Serina E, Rempel D. A system for evaluating the effect of keyboard design on force, posture, comfort, and productivity. Ergonomics 1994;37:1649–1660.
22. Hargens AR, Romine JS, Sipe JC, Evans KL, Mubarak SJ, Akeson WH. Peripheral nerve-conduction block by high compartment pressure. J Bone Joint Surg 1979;61A:192–200.
23. Lundborg GN, Gelberman RH, Minteer-Convery M, Lee YF, Hargens AR. Median nerve compression in the carpal tunnel—functional response to experimentally induced controlled pressure. J Hand Surg 1982;7:252–259.
24. Winn FJ, Habes DJ. Carpal tunnel area as a risk factor for carpal tunnel syndrome. Muscle Nerve 1991;13:254–258.
25. Armstrong TJ, Chaffin DB. An investigation of the relationship between displacements of the finger and wrist joints and the extrinsic finger flexor tendons. J Biomech 1978;11:119–128.