Median Nerve Displacement Through the Carpal Canal

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We determined the direct relationships between wrist position and displacement of the median nerve during active contraction of the flexor tendons at the wrist with an intact, transected transverse carpal ligament (TCL). Nine fresh cadavers were mounted in an apparatus to allow variable wrist position. Excursions of the tendons and displacement of the median nerve were measured by tracking markers with a video camera. Each limb was tested at 0°, 30°, and 60° of wrist extension before and after release of the TCL. Excursion of the flexor tendons required for full finger flexion ranged from 2.3 to 3.1 cm (mean, 3 cm). Median nerve displacement ranged from 0.9 to 1.4 cm (mean, 1 cm). The relationship between median nerve and flexor tendon excursion was consistently linear. Finger motion alone allows for median nerve displacement after surgery in the carpal tunnel. (J Hand Surg 1994; 19A:901-906.

Because the path of the median nerve lies anterior to the centers of rotation of the joints of the hand, extension of the wrist and fingers will shorten the bed of the nerve, allowing it to slide proximally. Similarly, extension of the wrist and fingers lengthens the course of the median nerve in the hand, causing it to slide distally. Distal-proximal sliding of the median nerve at the wrist in response to hand motion has been observed in live subjects and in cadavers.

To the extent that the normal sliding of the median nerve is restricted, for example, as a result of adhesions or compressive entrapments, lengthening the nerve bed in response to joint motion must be accommodated by local elongation of the nerve. Nerve stretching or traction of this type can result in temporary or permanent disruption of action potential propagation, causing impairment of sensory and motor function. We hypothesize that altered kinematics may be an etiologic element in nerve entrapment syndromes, such as carpal tunnel syndrome.

The purpose of this study is to describe the normal motion of the median nerve at the wrist in relation to stimulated active finger flexion, with the wrist immobilized in a neutral position or in extension.

Methods

Nine fresh cadaver forearms disarticulated at the elbow were used. Each was rigidly mounted in a jig by means of three Steinmann pins, one each in the radius and ulna proximally and one distal, traversing both radius and ulna (Fig. 1). Two 3-mm external fixation half-pins in the index metacarpal were attached to the jig by a pivoting clamp that allowed immobilization of the wrist in any desired position of flexion or extension. Each finger was independently loaded with a 75-gram counterweight attached to the finger tip with a heavy suture to extend the finger. The forearm was superficially dissected to expose the median nerve, transverse carpal ligament, and flexor digitorum superficialis (FDS) tendons. The flexor digitorum profundus (FDP) tendons were exposed by deeper dissection in the mid-forearm. Care was taken not to disrupt the natural tendon-nerve positional relationship, and all fascia was left intact. Stainless steel sutures were attached to the FDS and FDP tendons at the muscle-tendon junction, routed...
proximally along the muscle bellies, and attached to a load cell in series with a computer-controlled stepper motor. The stepper motor simulated slow muscle contraction by producing 12.5 mm/min of flexor displacement and was positioned to maintain the normal line of action of the flexor muscles. Each kinematic study was initiated with the fingers fully extended, and the stepper motor was used to flex the fingers until they reached the palm.

Excursion of the FDS tendons and displacement of the median nerve relative to the transverse carpal ligament was measured by tracking the positions of markers attached to each structure (Fig. 2). Steel sphere markers 0.5 mm in diameter were placed in the substance of the tendon of the flexor digitorum superficialis of the ring finger and within the substance of the median nerve. With this placement, markers moved with the nerve or tendon, and not with the overlying fascia. Reference markers were placed in the transverse carpal ligament. As the fingers were actively flexed, positions of the markers were recorded with a Pulnix CDC video camera (Pulnix America, Sunnyvale, CA) placed directly above the wrist. Images were recorded with the fingers extended and at each 5-mm increment of flexor tendon motion proximally, as determined by displacement at the stepper motor. Marker positions were measured in each captured frame using IMAGE software (Wayne Rasband, Research Services Branch, National Institute of Mental Health), and displacements of the flexor tendons and median nerve at the wrist were calculated.

Each limb was tested in three positions: neutral, 30° wrist extension, 60° wrist extension. The wrist positions selected reflect a sequential increase in flexion that parallel increasing carpal tunnel pressures noted with increasing wrist extension in people with and without carpal tunnel syndrome. Following these tests, the transverse carpal ligament was surgically sectioned to simulate a release used to treat carpal tunnel syndrome, and the kinematic experiments were repeated.

For each specimen, the FDS tendon excursion was plotted against displacement of the corresponding median nerve, and this relationship was analyzed by linear regression. A three-factorial analysis of variance was then conducted with the regression line slopes to determine the significance of specimen variation, wrist position, and surgical release of the transverse carpal ligament. This was followed by a two-factorial blocked design analysis of variance (ANOVA) with specimen as blocking factor and wrist position and surgical release as the experimental factors.

**Results**

Excursion of the FDS tendons required for full finger flexion ranged from 2.3 to 3.1 cm (mean, 3 cm). Corresponding displacement of the median nerve ranged from 4.3 to 5.1 mm (mean, 4.5 mm), regardless of wrist position. Excursions of the median nerve, regardless of wrist position, correlated significantly with excursion of the FDS tendon. Median nerve displacement continued to increase after release of the transverse carpal ligament.
Example of video frames used to measure median nerve (MN) and flexor digitorum superficialis (FDS-ring finger) displacement. Measurements are references to a line drawn parallel to the edge of the transverse carpal ligament (TCL) and through a marker placed within the substance of the ligament. At the beginning of loading (A), the MN and FDS-ring markers are approximately equidistant from the TCL. At the end of the test (B), both have moved proximally in a nerve/tendon ratio of approximately 0.4. Palmaris longus (PL) is labelled for reference.

The relationship between excursion of the FDS tendons and median nerve displacement was consistently linear, regardless of wrist position or whether the transverse carpal ligament was intact (Fig. 3). Values of $r^2$ for nerve displacement versus tendon excursion varied from 0.893 to 0.998 (mean, 0.96). Because of the consistently linear relationship between nerve and tendon displacement, slope of the linear regression was used to characterize each test. These nerve/tendon displacement ratios were used for statistical comparison of the experimental groups (Fig. 4).

The ANOVA revealed significant differences between specimens ($p = .002$), but differences owing to surgical release of the transverse carpal ligament ($p = .142$) and wrist position ($p = .349$) were not significant. The effect of ligament release and wrist position remained insignificant and had similar $p$-values when eliminating the effect of specimen variability with the two-way blocked ANOVA.

Load versus motor displacement data were ap-

![Image of nerve and tendon displacement plots]
proximately linear; \( r^2 = 0.95 \) by regression analysis on compiled data from all experiments. Loads never exceeded 45 N. No slipping at the tendon-suture was noted. Passive flexion of the fingers at each wrist position resulted in bunching up of the nerve and tendon distal to the transverse carpal ligament, with little evidence of sliding.

**Discussion**

Carpal tunnel syndrome (CTS) has classically been considered a nerve compression injury; however, it is clearly multifactorial in origin.\(^5\) CTS is known to be aggravated by activities requiring prolonged wrist flexion or repeated wrist or finger flexion and power grip.\(^6\) Using wick catheter pressure measurements, we determined that hydrostatic pressure increases in the carpal canal when the wrist is held in either flexion or extension.\(^7\) Furthermore, repetitive wrist flexion and extension cause a sustained increase in intracarpal pressure.\(^8\) Recent magnetic resonance imaging studies have demonstrated that in flexion the finger flexor tendons are shifted anteriorly against the transverse carpal ligament, while in extension they are aligned more dor-sally.\(^9\) In its interposed position between the anterior row of tendons and the transverse carpal ligament, the median nerve is subject to shearing and compression as the fingers are flexed. The extent to which these shear forces result in tensile strain in the median nerve has not been investigated.

A related problem concerns “spot-welding” of the median nerve caused by postoperative adhesions. Postoperative splinting regimens and physical therapy programs have been recommended to prevent this problem without any knowledge of normal nerve mechanics. For instance, a longstanding debate concerns whether splinting the wrist after carpal tunnel surgery to prevent bowstringing of the flexor tendons is harmful because of limiting median nerve displacement by adhesions.

Wilgis and Murphy\(^2\) observed longitudinal sliding of the median nerve at the wrist in response to passive motion of the hand in cadaver limbs. Because we are concerned that passive motion of a cadaver arm may not accurately reflect nerve kinematics in an active person,\(^10\) we simulated active finger flexion in cadaver preparations by pulling on the finger flexor tendons with a motor. We investigated median nerve displacement with power grip in which the FDP and FDS act in unison. The similarity of...
The architecture of the flexor digitorum superficialis and flexor digitorum profundus muscles appears to justify tendodesis of their tendons and use a single motor for finger flexion. The displacement of the median nerve may be different in activity such as typing that require independent FDS activity.

The range of longitudinal sliding of the median nerve we observed (9-14 mm) in response to passive finger flexion is similar to that observed by Kwan et al. and Murphy in response to passive wrist flexion/extension in cadavers. A similar range of sliding was noted in live subjects by McLelland and Swash, who estimated nerve sliding by observing deflections of microelectrodes inserted in the nerve. In addition to longitudinal sliding, displacement of the median nerve in the palmar-dorsal direction and radial-ulnar direction have been observed in dynamic ultrasound and magnetic resonance imaging studies.

One of the forces causing the nerve to slide longitudinally in response to hand motion is the nerve's internal elastic tension. Commonly if the median nerve is cleanly transected, the ends will retract 1-2 cm, indicating that the nerve was under tension before being cut. Kwan et al., in a rabbit study, measured on 11% shortening in segments of peripheral nerve after explanation. If we take the extensor length as the gauge length, then the nerve stumps are strained 11%, and therefore is continually under tension. Any imbalance in tensile forces caused by motion of joints would result in sliding of the nerve until equilibrium is reestablished.

In addition to internal tension, shear forces between the nerve and adjacent tissues may affect nerve motion. Our finding that the flexor digitorum tendons consistently translocate at two to three times the rate of the median nerve indicates that the adjacent tendons exert proximally directed, frictional shear forces on the nerve as the fingers are flexed. We know of no published measurements of these forces; our observation that passive finger flexion produced little motion of the median nerve suggests that they may be important. In addition, membranous connections between the median nerve and flexor tendons may contribute to the synchrony of their movement. Goldstein et al. measured strain in the digital flexor tendons proximal and distal to the transverse carpal ligament in response to stepwise increasing tendon loads and concluded that significant shear forces exist between the flexor tendons and the transverse carpal ligament. Wrist flexion and active finger flexion increased these traction effects. Since the median nerve passes adjacent to the transverse carpal ligament, we may expect that similar shear forces will affect the median nerve; these would be aligned opposite to the shear forces resulting from friction with the proximally moving tendons. We speculate that increased pressure in the carpal tunnel might amplify the relative role of shear forces in median nerve motion. Such a pressure increase could result from abnormal anatomy or simply from forceful contraction of the flexor digitorum profundus muscle. Similarly, any adhesions between nerve and tendon or between nerve and transverse carpal ligament or entrapment would be expected to alter the normal nerve kinematics.

The failure of surgical release of the transverse carpal ligament to affect nerve and tendon excursion in this study is not surprising. Disruption of normal hydrostatic relations in a cadaver hand would be expected to alter carpal tunnel pressure dynamics; in addition, there was no evidence that any of these cadavers suffered from carpal tunnel syndrome when alive. Increasing wrist extension caused a trend toward diminished sliding of the median nerve. Wrist flexion would be more likely to elicit kinematic abnormalities; however, technical problems with the wrist-flexed preparation prevented us from collecting data in this position.

The observation that both flexor tendons and median nerve exhibit substantial translocation at the wrist in response to finger motion when the wrist is rigidly immobilized is significant with respect to care following surgical repair of nerve or tendon injuries. It appears that active finger motion alone would provide sufficient motion of the median nerve and flexor tendons in the vicinity of the wrist to prevent adhesion formation even if the wrist is immobilized. Conversely, if one desires complete immobilization of the median nerve or finger flexor tendons after repair, digital motion must be prevented.

References

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