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The Effect of Tendon Excursion Velocity on Longitudinal Median Nerve Displacement: Differences Between Carpal Tunnel Syndrome Patients and Controls

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Abstract

The subsynovial connective tissue (SSCT) is a viscoelastic structure connecting the median nerve (MN) and the flexor tendons in the carpal tunnel. Increased strain rates increases stiffness in viscoelastic tissues, and thereby its capacity to transfer shear load. Therefore, tendon excursion velocity may impact the MN displacement. In carpal tunnel syndrome (CTS) the SSCT is fibrotic and may be ruptured, and this may affect MN motion. In this study, ultrasonography was performed on 14 wrists of healthy controls and 25 wrists of CTS patients during controlled finger motions performed at three different velocities. Longitudinal MN and tendon excursion were assessed using a custom speckle tracking algorithm and compared across the three different velocities. CTS patients exhibited significantly less MN motion than controls ($p = 0.002$). While in general, MN displacement increased with increasing tendon excursion velocity ($p = 0.031$). These findings are consistent with current knowledge of SSCT mechanics in CTS, in which in some patients the fibrotic SSCT appears to have ruptured from the tendon surface.

Keywords

carpal tunnel; ultrasound; velocity; median nerve; flexor tendons

INTRODUCTION

Motion of the median nerve (MN) has found to be altered in patients with carpal tunnel syndrome (CTS).^{1,2} One plausible explanation for this is that the subsynovial connective tissue (SSCT) between the MN and tendons becomes severely damaged and ruptures.³ The SSCT is a multi-layered structure with interconnecting collagenous fibers that envelop the MN and tendons.^{4–6} Stretching of the interconnecting fibers between the layers and

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successive engagement of each consecutive layer allows the SSCT to serve as a friction-reducing unit between tendons and between tendons and the MN when tendons are in motion (Fig. 1). Ettema et al. showed through cadaveric study, subsequently confirmed clinically, that the SSCT of CTS patients falls into at least two distinctly different structure categories, either tightly adhering to the tendon or becoming completely disconnected from the tendon.³

The SSCT is a soft connective tissue and possesses viscoelastic properties.⁷ Abundant evidence exists demonstrating that increased strain rates increases stiffness and reduces deformation at failure in viscoelastic tissues.^{8–14} Filius et al. showed that peak forces and resistance energy of tendon excursion in the carpal tunnel were greater in specimens tested at higher velocities, most probably a direct result of viscoelastic stiffening of the SSCT.¹⁵ If SSCT stiffness, and thereby its capacity to transfer shear load, increases as a result of higher strain rates, MN displacement should increase at higher tendon excursion velocities, since the MN is a relatively loose structure within the carpal tunnel and the nerve cannot move actively.

In CTS patients, fibrotic changes in the SSCT have been observed.^{16–18} Fibrotic SSCT is stiffer and ruptures at smaller strains during tendon excursion.¹⁹ This combination of increased stiffness and failure at smaller tendon excursions could be a major contributor in a vicious cycle of initial SSCT injury, resulting fibrosis, decreased elasticity, further sensitivity to injury, and more fibrosis.^{3,20} Indeed, some authors have postulated that such a vicious cycle may be the root cause of CTS.^{3,21–23} This effect might be even more pronounced during higher velocity tendon excursions.¹⁵

Much of our knowledge of SSCT mechanical behavior has come through cadaveric experiments. While useful, there are a number of limitations in these studies that necessitate study in live subjects to identify similar response to confirm these models. In this study, we investigated the effect of increasing tendon excursion velocity on the resulting MN displacement in both CTS patients and healthy controls. Secondly, we investigated whether or not tendon excursions change in CTS patients.

METHODS

Subjects

Fourteen healthy controls and 18 CTS patients were selected for this study. Seven patients had bilateral CTS, so subsequently 25 CTS hands were analyzed in this study. CTS was confirmed based on clinical examination and nerve conduction studies.^{24,25} Subjects were excluded from this study if they had a medical history of upper extremity surgery or fractures of the wrist or hand or disorders to the musculoskeletal system. All procedures were reviewed and approved by our institutional review board.

Ultrasound Assessment and Analysis

Subjects were seated with the arm positioned on an adjacent table. The arm was positioned such that the forearm was supinated, the elbow flexed to 60 degrees, and the wrist was in the neutral position. Subjects were asked to flex their fingers from full extension until the tip of

their fingers touched the palm of the hand. Subjects were asked to perform this motion over time periods of 8, 4, and 2 s (defined, respectively as low, medium, and high velocity tendon excursion). High frequency ultrasound scans of the MN, the flexor digitorum superficialis tendon of the middle finger (FDS3) and the flexor digitorum profundus tendon of the middle finger (FDP3) were made using a Philips iU33 ultrasound machine (Philips Electronics, Best, The Netherlands) with a 7–15 MHz linear array transducer. The full range of frequencies was used, depending on the depth of the structure. The transducer was placed at the distal wrist crease at the level of the inlet of the carpal tunnel defined as the proximal border of the pisiform bone (Fig. 2). After the structure of interest (i.e., the MN, FDS3, or FDP3) was clearly visualized, there was zoomed in on the structure using the “zoom” function. The ultrasound imaging settings were set at maximum frame rate; lossless imaging formats were used.

Data processing to calculate longitudinal displacement of the aforementioned structures has been described previously by Korstanje et al.^{1,26} In brief, ultrasound images were imported into the program OsiriX (version 4.1.2 64-bits, <http://osirix-viewer.com>), rotated if needed and exported as uncompressed audio-video interleave (AVI) files. The uncompressed AVI's were imported into Matlab (Version 2011a, The MathWork Inc., Natick, MA) and analyzed with an inhouse developed and validated speckle tracking algorithm (Fig. 3).²⁷

Absolute displacements of the MN, FDS3, and FDP3 were calculated. The FDS3 and FDP3 displacements were calculated for solely the low velocity. Relative MN displacements were characterized by normalizing absolute MN displacement with FDS3 displacement. Defining MN motion relative to FDS3 displacement in this way was done because the FDS3 is the tendon closest to the nerve and assumed to have the greatest influence on MN motion.²⁸ Relative tendon excursion was defined as the ratio of the FDP3 displacement to FDS3 displacement.

Statistical Analysis

Differences between the controls and CTS group characteristics, absolute displacement of the MN and tendons as well as the MN/FDS3 and FDS3/FDP3 ratios between the CTS patients and control group were analyzed using the unpaired *t*-test or the chi-square test for binominal data. Repeated measures ANOVA was used to evaluate differences in relative MN displacement at different tendon excursion velocity levels within the two groups. *p* values of 0.05 or less were considered significant. The statistical analysis was done with Statistical Package for Social Science software (SPSS version 17.0, Chicago, IL).

RESULTS

Twenty-five hands of 18 CTS patients were imaged, as were 14 hands of 14 healthy controls. No significant differences were found for age, gender or body mass index between the two groups (Table 1).

Patients had significantly less absolute and relative median nerve motion regardless of the tendon excursion velocity, with $p = 0.002$ and $p < 0.001$, respectively (Table 2). The largest difference in relative MN motion was observed at high velocity tendon excursion. No

significant differences between patients and controls were found for either absolute FDS3 or FDP3 displacement. However, the FDP3/FDS3 ratio for CTS patients was significantly smaller than that for controls ($p = 0.001$).

In the control group, there was a significant difference in relative MN displacement between the slow tendon excursion velocity and medium and high tendon excursion velocity ($p = 0.005$ and $p = 0.001$ respectively). In the CTS patient group a difference in MN displacement was seen between low and high tendon excursion velocity ($p = 0.012$) (Fig. 4).

DISCUSSION

In this study, we investigated the effect of tendon excursion velocity on MN displacement in CTS patients and healthy controls. We found that in the healthy control group there was more MN displacement compared to the CTS group, and a trend of increased MN displacement at higher tendon excursion velocities. This trend, however less obvious, was also observed in the CTS patient group.

Increased tendon excursion velocities resulted in increased MN displacement. The SSCT is a multilayered structure that interconnects structures within the carpal tunnel. The MN is a structures that moves only passively, by traction either of its attachments or of adjacent structure, presumably driven by loads of moving tendons that are transmitted through the SSCT.^{6,29} High velocity tendon excursions require more force to overcome sliding resistance compared to low velocity tendon excursions in healthy subjects.¹⁵ A plausible explanation is that this is a result of the viscoelasticity of the SSCT, with the SSCT becoming stiffer at higher velocities. A stiffer SSCT will resist stretching more and will move more closely associated with the driving tendon and taking along the MN. This was indeed observed in our study.

The relative MN displacement was not as strongly affected by different velocities of tendon excursion in the CTS patient group compared to the controls. Moreover, the average relative MN displacement in the CTS patient group was well below that observed in the control group, as noted by others as well.^{1,2} This may suggest that in this patient population the SSCT between the MN and FDS3 was at least partly disrupted. We made the assumption that any observed MN motion would be a result of SSCT action, and that if MN velocity and excursion matched that of the adjacent tendon, the most probable explanation was a fibrotic SSCT binding the nerve and tendon together, similar to what has been shown with regard to the SSCT and FDS by Ettema et al.³ Their study showed a non-normal distribution of motion, representing varying degrees of adherence of SSCT and FDS3, ranging from tight adherence and linked motion in some CTS patients, to a complete failure of the SSCT/FDS3 connection in other CTS patients, with no correlation between the movement of one structure and another. This latter phenomenon, we saw especially in our low excursion nerves, but to some extent in all the CTS nerves. This finding suggests that, unlike the SSCT around the FDS, which is often adherent in CTS,³ with regard to the MN, the SSCT between it and the underlying tendons tend to fail, so that the nerve and tendons are less closely associated. This may result in more fixation of the nerve to the overlying flexor retinaculum, resulting in less nerve dorsal-palmar mobility, a phenomenon observed by others.^{30,31}

In this study there was a trend of increased FDS excursion. Korstanje et al. found significantly more FDS excursion and less FDP excursion in CTS patients, as compared to controls.¹ Their rationale for this finding was that fibrotic SSCT tissue causes adhesions between the FDS and FDP tendon, causing the FDS tendon to be more adherent to the FDP tendon, thereby increasing FDS excursion and restricting FDP excursion.³¹ More hands were included into that study and the wrists within the same subject were compared, which will affect the statistical power. Comparable to the current study, Hough et al. and Doesburg et al. also observed a trend of increased FDS excursion, although not significant.^{2,32} This finding may be the result of increased adhesions of the SSCT in a subset of subjects, as noted above. To our knowledge finger motion in CTS patients has not been investigated and therefore it is unknown if this motion path is altered. We find the possibility of changes in FDP/FDS synergy in CTS patients intriguing and would find it an interesting topic for further investigation.

Our study has several limitations. First, we measured MN motion and not the SSCT. Measuring the SSCT would be relevant, because it is an important component of the carpal tunnel and implicated in the etiology of CTS. Studies have shown that duration and type of motion effect SSCT/tendon ratios,²³ and these ratios are affected as well by CTS.^{1,3} However, obtaining a consistent and reliable SSCT displacement measurements is difficult, because the SSCT is a very thin structure (<1 mm), at the limit of resolution of our ultrasound method, especially at high tendon velocities. Few studies have been conducted evaluating accuracy of the speckle tracking results of the tendon or SSCT in a laboratory setting or in vivo. Doesburg et al. showed that the mean absolute error of the SSCT speckle tracking results was approximately 3 mm. The current speckle tracking method was found to have a mean absolute error of 0.08 mm for different tendon excursions; no measurements for speckle tracking of the SSCT were performed. Currently, new very-high frequency ultrasound machines (e.g., VisualSonics Vevo 2100, VisualSonics, FUJIFILM VisualSonics, Inc., Toronto, CANADA) are being developed and used in studies with human subjects. These machines may increase the accuracy of speckle tracking measurements of the SSCT for such movements. In addition, more experience and further development taking into account the layered properties of SSCT may also lead to more accurate measurements. For now, our rationale to focus on the MN is that CTS is ultimately a disorder of the MN. Moreover, in this study we made the assumption that any observed MN motion would be a result of SSCT action, as described above. Therefore MN motion was considered to be a good alternative to measuring SSCT motion directly, although this could be a matter of debate based on findings of this study. A second limitation is that we have not investigated factors that could also play a role in affecting the MN motion, such as the size of the MN, which is altered in patients with CTS.^{33,34} Third, tendon excursion was only measured at one speed and was assumed constant. Yoshii et al. showed that tendon excursion remained constant regardless of different tendon velocities for the same motion.³⁵ Therefore, we believe that this is a reasonable assumption, but if excursions were different at different velocities, this would have affected the results. Lastly, we did not take into consideration the difference between dominant versus non-dominant hand or right versus left hand. Filius et al. did not find difference in transversal plane tendon and nerve motion between the two hands of one individual.²⁸ However, the effect on hand dominance and tendon and nerve motion in the

longitudinal plane is unknown. In summary, ultrasonography in living subjects confirms that MN displacement increases with increasing tendon excursion velocity. Less MN displacement is found in CTS patients overall and there is a diminished effect of velocity tendon excursion on the MN displacement. This could be caused by a disruption of the SSCT between the MN and nearby tendons, an observation consistent with other studies of CTS patients. These findings may have clinical utility. Whether individual variations in the sonographic findings of CTS patients might have therapeutic or prognostic implications is unknown, but could be studied.

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Abbreviations

cm	centimeter
SD	standard deviation
MN	median nerve
FDP3	flexor digitorum profundus of the middle finger
FDS3	flexor digitorum superficialis of the middle finger

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Figure 1.

A–B Shown is a macroscopic (A) and electron microscopic (B) image of the SSCT between tendons. Normally, the SSCT is a flimsy structure that envelops the structures within the carpal tunnel. The SSCT consists of layers with interconnecting fibers. These interconnecting fibers are also connected to the tendon, stretching the interconnecting fibers between consecutive layers when the tendon moves. Copyright permissions will be asked for upon acceptance of the manuscript (Plast Reconstr Surg. 2006 Nov;118 6):1413–22).

Figure 2.
Placement of the probe on the wrist when making ultrasound scans of the median nerve and tendons at the carpal tunnel.

Figure 3.
Representative example of a longitudinal plane ultrasound scan of the median nerve with placement of the region of interest (ROI).

Figure 4.

Relative MN displacements at different tendon excursion velocity levels for CTS patients and controls. Significant differences exist for relative MN motion between the different tendon excursion velocity levels for CTS patients and controls.

Table 1

Study Group Characteristics

	Patients (<i>N</i> = 18)	Controls (<i>N</i> = 14)	<i>p</i>-value
Age	50 (SD 12)	42 (SD 12)	0.066
Gender (male)	44%	36%	0.618
BMI	32 (SD 7)	30 (SD 13)	0.503

SD, standard deviation; BMI, body mass index.

Table 2

Excursions of the Tendons and of the MN at Different Tendon Excursion Velocities

	CTS hands (N = 25)	Control hands (N = 14)	p-value
Absolute MN displacement (cm) at			
Low velocity tendon excursion	0.18 (SD 0.10)	0.21 (SD 0.07)	<0.001
Medium velocity tendon excursion	0.21 (SD 0.12)	0.33 (SD 0.10)	0.002
High velocity tendon excursion	0.23 (SD 0.12)	0.40 (SD 0.11)	<0.001
Absolute tendon displacement (cm)			
FDS3 tendon excursion	1.88 (SD 0.48)	1.56 (SD 0.52)	0.064
FDP3 tendon excursion	2.73 (SD 0.49)	2.82 (SD 0.51)	0.608
Relative MN displacement at			
Low velocity tendon excursion	0.10 (SD 0.06)	0.22 (SD 0.07)	<0.001
Medium velocity tendon excursion	0.12 (SD 0.07)	0.23 (SD 0.07)	<0.001
High velocity tendon excursion	0.13 (SD 0.08)	0.28 (SD 0.11)	<0.001
FDP3/FDS3 excursion ratio	1.53 (SD 0.40)	1.95 (SD 0.44)	0.005

cm, centimeter; SD, standard deviation; MN, median nerve; FDP3, flexor digitorum profundus of the middle finger; FDS3, flexor digitorum superficialis of the middle finger.