Clinical Biomechanics 41 (2017) 48–53

Contents lists available at ScienceDirect

Clinical Biomechanics

journal homepage: www.elsevier.com/locate/clinbiomech

Delineation of the mechanisms of tendon gliding resistance within the carpal tunnel

Anika Filius [a,b,c], Andrew R. Thoreson [a], Yasuhiro Ozasa [a], Kai-Nan An [a], Chunfeng Zhao [a], Peter C. Amadio [a,*]

[a] Biomechanics Laboratory, Division of Orthopedic Research, Department of Orthopedic Surgery, Mayo Clinic, 200 1st Street SW, Rochester, MN, USA
[b] Department of Plastic and Reconstructive Surgery and Hand Surgery, Erasmus MC, University Medical Centre Rotterdam, The Netherlands
[c] Department of Rehabilitation Medicine, Erasmus MC, The Netherlands

ARTICLE INFO

Article history:
Received 2 February 2016
Accepted 1 December 2016

Keywords:
Biomechanics
Carpal tunnel syndrome
Gliding resistance
SSCT
Tendon

ABSTRACT

Background: Forceful, high-velocity, and repetitive manual hand tasks contribute to the onset of carpal tunnel syndrome. This study aimed to isolate and identify mechanisms that contribute to tendon gliding resistance in the carpal tunnel.
Methods: Eight human cadaver hands (four pairs) were used. Tendon gliding resistance (force, energy, and stiffness) was measured under different conditions: with intact and with divided subsynovial connective tissue, at 2 mm/s and 60 mm/s tendon excursion velocity, and with and without relaxation time before tendon excursion.
Results: Subsynovial connective tissue stretching substantially contributed to increased gliding resistance force and energy during higher tendon excursion velocities, and subsynovial connective tissue stiffening was observed. Poroelastic properties of the tendon (and possibly the subsynovial connective tissue) also appear to be involved because relaxation time significantly increased gliding resistance force and energy ($P < 0.01$), and the difference in energy and force between high- and low-velocity tendon excursions increased with relaxation time ($P = 0.01$ and $P < 0.01$). Lastly, without relaxation time, no difference in force and energy was observed ($P = 0.06$ and $P = 0.60$), suggesting contact friction.
Interpretation: These findings are consistent with the hypothesis that the mechanics of tendon motion within the carpal tunnel are affected by the integrity of the subsynovial connective tissue. While not tested here, in carpal tunnel syndrome this tissue is known to be the fibrotic, thickened, and less-fluid-permeable. An extrapolation of our findings suggests that these changes in the subsynovial connective tissue of carpal tunnel syndrome patients could increase contact friction and carpal tunnel pressure.

1. Introduction

Forceful, high-velocity, and repetitive manual hand tasks can contribute to the onset of carpal tunnel syndrome (CTS) (Atroshi et al., 1999; Barcenilla et al., 2012; Moore et al., 1991; Palmer, 2011; Viera, 2003), a commonly diagnosed neuropathy of the median nerve. Wrist flexion and finger flexion, especially isolated flexion of a single finger (as may occur in typing or other repetitive work), are associated with high tendon gliding resistance (GR) and increased carpal tunnel pressure, which also increases GR (Ettema et al., 2004; Filius et al., 2014; Kociolek et al., 2015; Seradge et al., 1995; Zhao et al., 2011), but little work has been done to elucidate the mechanisms that contribute to tendon GR in the carpal tunnel.

Filius et al. (2014) and Vanhees et al. (2012) reported that the GR response of the flexor digitorum superficialis (FDS) tendon within the carpal tunnel had an initial "toe" region that was followed by a plateau, in turn followed by an ascending force response. The ascending response has been attributed to stretching of the subsynovial connective tissue (SSCT) (Ettema et al., 2006), a multi-layered structure that surrounds the flexor tendons and median nerve within the carpal tunnel. The SSCT is thought to provide an interface that reduces friction between these structures; however it also restricts tendon motion, especially differential motion between adjacent tendons (Ettema et al., 2006). This assumption is based on previous anatomic research that showed that the FDS tendons within the carpal tunnel, except for the FDS of the little finger, are connected to the SSCT but had no directly interconnecting fibers to other tendons (Leijnse et al., 1997).

The finding of a toe region and plateau is consistent with the response observed in isolated flexor tendon gliding in the zone II-A2

Abbreviations: CTS, carpal tunnel syndrome; F, force; FDS3, flexor digitorum superficialis tendon of the middle finger; GR, gliding resistance; SSCT, subsynovial connective tissue.

* Corresponding author at: Department of Orthopedic Surgery, Mayo Clinic, 200 First Street SW, Rochester, MN 55905, USA.
E-mail address: pamadio@mayo.edu (P.C. Amadio).

http://dx.doi.org/10.1016/j.clinbiomech.2016.12.001
0268-0033/

pulley area, suggesting that this behavior is caused by contact friction between tissues (Uchiyama et al., 1995; Uchiyama et al., 1997). It may then be speculated that tendon GR in the carpal tunnel may have a component attributed to contact friction. If the GR could be evaluated by isolating the effect of contact friction and removing other effects (i.e., the SSCT), this speculation could be verified.

Like all living tissues, tissues in the carpal tunnel are infused with an ionic interstitial fluid; interaction of this fluid with the solid phase of these tissues during deformation is a phenomenon known as poroelasticity (Simon, 1992), a behavior which has been demonstrated in other soft tissues such as cartilage (DiSilvestro and Suh, 2001). Poroelasticity of the tendon may become evident when the tendon is pulled through the narrow carpal tunnel and fluid is dispersed out of the tendon as it is deformed, affecting its cross-sectional shape. The poroelastic properties of the tendon, and possibly the SSCT, may also affect GR (Cao and Tang, 2005; Elliott et al., 2003; Sud et al., 2002).

The purpose of this study was to identify factors contributing to tendon GR within the carpal tunnel by sequentially isolating different resistance components related to structural integrity, tendon excursion velocity, and relaxation time allowed between excursions. We hypothesized that the total GR measured during tendon excursion in the carpal tunnel would be the result of a combination of SSCT deformation, (poroelastic) tendon deformation, and contact friction between the flexor tendon and the surrounding tissues (Eq. (1)) (Fig. 1) (Chimich et al., 1992; Clemmer et al., 2010; Lynch et al., 2003; Pioletti et al., 1998; Sud et al., 2002; Zhang, 2005).

$$F_{\text{total gliding resistance}} = F_{\text{SSCT deformation}} + F_{\text{tendon deformation}} + F_{\text{contact friction}} \quad (1)$$

To test our hypothesis regarding the role of SSCT, we aimed to compare the total GR to the response after disrupting the SSCT connections to the tendon. We hypothesized that the ascending part of the curve would be eliminated when severing all SSCT connections to the tendon, and that the GR response over a given tendon excursion would then be similar to that observed with tendon excursion in the digits, i.e., a constant GR throughout the excursion, confirming the role of contact friction in the carpal tunnel when the SSCT is not functioning. Lastly, the opportunity to test the isolated tendon GR through the carpal tunnel permits studying the effect of fluid on gliding resistance. We hypothesized that a poroelastic response, if present, would cause a higher GR force and energy for excursions with relaxation time allowed between pulls compared with excursions without relaxation (Cao and Tang, 2005; Chimich et al., 1992; Yin and Elliott, 2004). Allowing no relaxation time between pulls would be expected to nullify any poroelastic effect, and in so doing, the contribution of the contact friction could be confirmed and evaluated. If contact friction were present, it would result in a measurable, nonzero GR which would not be sensitive to the excursion velocity.

2. Materials and methods

This study was approved by the Mayo Clinic Institutional Review Board. Specimens were obtained from the Mayo Clinic Institutional Anatomical Bequest Program. Eight human cadaver hands (four pairs) were used, with 2 pairs of hands from male subjects. The mean age of the cadaver specimens was 74 years (range 57–83 years). We verified that cadaver specimens did not have a medical history of CTS, upper extremity surgery or fractures of the wrist or hand or disorders to the musculoskeletal system. Specimens were thawed at 5 °C for approximately 10 h before testing.

2.1. Carpal tunnel total gliding resistance (tendon plus SSCT) testing

Hands were prepared as previously described (Filius et al., 2014; Vanhees et al., 2012). Briefly, all finger flexor tendons were exposed proximal to the carpal tunnel and the flexor digitorum superficialis (FDS) 2, FDS4 and the flexor digitorum profundus (FDP) 3 tendon were each connected to a 50 g weight. Next, the wrist was mounted onto a jig with the wrist fixed in the neutral position and all fingers fixed with a Kirschner wire in the extended position, except for the middle finger. After determining the physiologic excursion of the FDS3 tendon, the middle finger also was fixed in a fully extended position. The FDS3 and FDP3 tendons were exposed at the mid palm, but distal to the carpal tunnel and the FDS3 was sharply transected at the level of the metacarpophalangeal joint. The distal end of the FDS3 tendon was connected to a 50-g weight and the proximal end was then connected to a 25 N load cell (MDB-5; Transducer Techniques) (Fig. 2).

A stepper motor-driven test actuator controlled the velocity and displacement for each cycle. Four hands were subjected to a displacement velocity of 2 mm/s and four contralateral hands from the same donors were subjected to a velocity of 60 mm/s. Displacement of the stage was monitored with a potentiometer sensor (TR 75; Novotechnik US). Force and displacement data were recorded at a sample rate of 100 Hz for low-velocity tendon excursion (2 mm/s) and 1000 Hz for high-velocity tendon excursion (60 mm/s). The FDS3 tendon was preconditioned by repeating 3 tendon excursions to 5 mm at 2 mm/s. Next, after 15 min of relaxation time, the jig was moved 55 mm, resembling FDS3 tendon flexion, which was sufficient to include the 95% CI of the physiologic excursion of the FDS3 tendon, and an additional 5 mm were included for deceleration, so the that the total displacement was 60 mm (Filius et al., 2014; Vanhees et al., 2012). Specimens were tested at room temperature (20 °C) and were moistened throughout testing

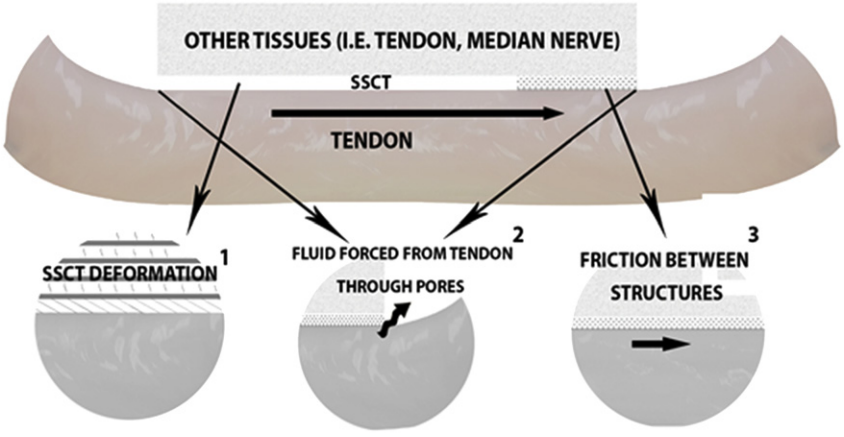

Fig. 1. Carpal tunnel components thought to have a role in gliding resistance. Resistance may be affected by 1) subsynovial connective tissue (SSCT) deformation, 2) tendon deformation (poroelasticity), and 3) residual contact friction.