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A Fluid-Immersed Multi-Body Contact Finite Element Formulation for Median Nerve Stress in the Carpal Tunnel

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Abstract

Carpal tunnel syndrome (CTS) is among the most important of the family of musculoskeletal disorders caused by chronic peripheral nerve compression. Despite the large body of research in many disciplinary areas aimed at reducing CTS incidence and/or severity, means for objective characterization of the biomechanical insult directly responsible for the disorder have received little attention.

In this research, anatomical image-based human carpal tunnel finite element (FE) models were constructed to enable study of median nerve mechanical insult. The formulation included large-deformation multi-body contact between the nerve, the nine digital flexor tendons, and the carpal tunnel boundary. These contact engagements were addressed simultaneously with nerve and tendon fluid-structural interaction (FSI) with the synovial fluid within the carpal tunnel. The effects of pertinent physical parameters on median nerve stress were explored. The results suggest that median nerve stresses due to direct structural contact are typically far higher than those from fluid pressure.

Keywords

Carpal tunnel syndrome; Finite element method; Mechanical insult; Median nerve; Flexor tendon; Solid stress; Fluid pressure

INTRODUCTION

Carpal tunnel syndrome (CTS) might fairly be described as the “flagship” of peripheral chronic nerve compression disorders. The condition is characterized by pain, numbness, and paresthesia in the distribution of the median nerve of the hand [1]. Untreated, there is often progressive loss of hand sensibility, strength, and dexterity. Mechanical insult to the median nerve is well recognized as the proximate cause, but to date the associated stresses have not been objectively quantified. Moreover, there is controversy as to the relative importance of fluid pressure versus nerve abutment contact with neighboring anatomic structures such as the flexor digitorum tendons and the carpal tunnel boundaries. Recent fundamental biologic studies [2] of the mechano-pathogenesis of CTS offer promise of substantial improvements over present treatment regimens. At present, however, the physiologic realism of the mechano-stimuli used in such work is limited by the lack of objective information as to the mechanical stress levels typically prevailing within the carpal tunnel.

In this study, a finite element (FE) formulation was developed to facilitate investigation of mechanical insult to the median nerve in the carpal tunnel. Exploratory parametric trials were undertaken to study model sensitivity to pertinent physical influences. In particular, stress levels in the median nerve and/or the flexor digitorum tendons were compared to levels of fluid pressure within the tunnel, to provide a basis for exploring whether direct structural contact(s) between solid elements, although presumably sporadic, might be a substantial factor precipitating or exacerbating carpal tunnel syndrome.

METHODS

An anatomical sectional image of the human carpal tunnel [3] was utilized to build carpal tunnel FE models (Figure 1). Source in-plane pixel dimension was 0.247 mm. Key anatomic attributes were the median nerve, the nine flexor digitorum tendons, and the carpal tunnel boundary. The margins of the median nerve, of the tendon, and of the carpal tunnel wall were manually segmented from the source images. The carpal tunnel FE model was built based on these detected anatomical boundaries, using ADINA/ADINA-F (Version 8.2, ADINA R&D, Inc.). There were two sub-models: one was a solid model for the nerve and tendons, and the other was a model for the fluid contained in the carpal tunnel, within which the nerve and tendons were immersed. As depicted in Figure 2, the two-dimensional carpal tunnel FE model (3,290 elements) was composed of 628 rectangular solid elements for the nerve and tendons, 100 individual straight line segments comprising the carpal tunnel boundary, and 2,562 triangular fluid elements for fluid. Also, a corresponding three-dimensional model was constructed by extruding the two-dimensional geometry along the longitudinal direction of the carpal tunnel. The three-dimensional model had 67,421 elements: 18,840 hexahedral solid elements for the nerve and tendons, 14,190 hexahedral elements for the tunnel wall, and 34,391 tetrahedral fluid elements for the fluid. The three-dimensional carpal tunnel had a 19.7 mm major diameter, a 9.7 mm minor diameter, and was 30 mm in length (Figure 3.)

Visco-elastic material properties from the literature [4] were applied as baseline conditions for the nerve and tendons. Shear modulus (G) was estimated by Equation (1). Poisson's ratio (ν) was taken as 0.45, and the bulk modulus (K) was estimated by Equation (2), using the relationship between K and G in Equation (3). The input baseline

$$G(t) = G_{\infty} + (G_0 - G_{\infty}) e^{-\beta t} \quad (1)$$

$$K(t) = K_{\infty} + (K_0 - K_{\infty}) e^{-\beta t} \quad (2)$$

$$K = \frac{2(1+\nu)}{3(1-2\nu)} G \quad (\nu=0.45) \quad (3)$$

conditions for nerve and tendon material properties are summarized in Table 1. Newtonian behavior (baseline viscosity $\mu = 20$ centipoise = 0.02 Ns/m^2) close to that of synovial fluid [5] was adopted for the fluid. Also, contact conditions were invoked between the nerve and tendons, between the individual tendons, between the nerve and the tunnel wall, and between the tendons and the tunnel wall, respectively, as depicted in Figure 4.

The FE model was driven by volarly directed nerve and tendon displacements, characteristic of a wrist flexion event. These displacements were prescribed in terms of motion of two central points (coincident with element nodes) located near the geometric centroid of each of these

individual solids. The magnitude and direction for nerve and tendon displacements were prescribed by considering an actual pattern of tendon excursions apparent on MRI for a power grip maneuver. The total physical time simulated was 0.5 seconds. To account for the interaction between the solid and fluid models, a fluid structure interaction (FSI) boundary condition was applied to every solid anatomic surface, and to corresponding loci in the fluid (Figure 4). Adaptive remeshing was utilized to avoid excessive fluid mesh distortion during solid motion. The adaptive remeshing function, which was implemented for triangular elements in the case of the two-dimensional model and for tetrahedral elements in the case of the three-dimensional model, was used to automatically modify the fluid mesh during the finite nerve and tendon excursions relative to the initial fluid mesh. Contact offset was invoked to avoid fluid mesh overclosure in the narrow gap between solid elements, i.e., to ensure fluid mesh preservation between contacting solids.

ADINA/ADINA-F (Version 8.2, ADINA R&D, Inc.) was used as the solver for the multi-body fluid/structure interaction solutions. Typical processing times required for two-dimensional simulations of the 0.5-second physical gripping maneuver were about 35 seconds, on a four-processor 1.5 GHz ITANIUM workstation (Hewlett-Packard Integrity rx4640) with 12 GB of RAM. The corresponding three-dimensional solid contact simulations required typically about 55 seconds of computation time per 0.05-second physical increment of the gripping maneuver.

RESULTS

Solid stress for the nerve and tendons, fluid pressure, and fluid velocity were first computed with the baseline material properties (Table 1) for the two-dimensional model. For simplicity, the carpal tunnel wall was defined as rigid. Beginning from the defined baseline analysis conditions, the simulation results at intermediate ($t = 0.35$ s) and final ($t = 0.5$ s) time points are depicted in Figure 5, 6, and 7, respectively. Overall solid and fluid mesh configuration (Figure 5), solid stresses and fluid pressure distribution (Figure 6), and fluid velocity distribution (Figure 7) are shown under multi-body immersed contact conditions. As expected, the original fluid mesh structure evolved into a substantially new configuration as the tendons traversed through the fluid (Figure 5). The data showed that fluid pressure was orders of magnitude below the median nerve stresses that developed due to tendon contact, even under the idealized assumption of full fluid containment in the carpal tunnel. Maximum values of median nerve stress, tendon stress, fluid pressure, and fluid velocity are summarized in Table 2. The predominance of solid-solid contact stress, relative to fluid pressure, was manifest regardless of the viscosity value assumed for the fluid. The median nerve underwent finite deformation of its cross-section due to solid-solid contact.

Although nerve stresses monotonically increased during volar migration of the tunnel contents, nerve stresses showed their greatest increase only after the nerve was fully captured between entrapping tendons and the tunnel wall (Figure 6). Also, maximum fluid velocity (which in this simulation occurred at $t = 0.45$ s) developed in a region of narrow gap between two tendons, rather than in the vicinity of the nerve (Figure 7). Since the particular kinematic pattern of tendon migrations input was somewhat arbitrary, it seems reasonable to infer that the maxima and distributions of fluid velocity would vary appreciably with motions of the structures inside the carpal tunnel. During activities of daily living and/or in the workplace, such motions are likely quite idiosyncratic, depending on individual anatomy [6] and specific functional activity [7].

Next, the effects of pertinent constitutive parameters on median nerve stress were investigated using the two-dimensional model. Fluid viscosity and tendon material properties were viewed as the key parameters likely affecting median nerve stress. Varying the input viscosity of the carpal tunnel fluid across a range from 0.05, 0.1, 10, and 25 times the baseline value of 0.02

Ns/m², it was found that the level of solid stress in the contacting regions of the nerve and tendons was always far greater than the fluid pressure (Table 3). Despite these three-order-of-magnitude fluid viscosity variations, peak values of nerve/tendon stress and of fluid velocity changed almost imperceptibly. By contrast, the computed fluid pressure showed an approximately linear relationship with fluid viscosity, although for very high viscosity (for example, Cases A4 and A5), a non-linear relationship between fluid viscosity and fluid pressure began to emerge.

In order to quantitatively isolate the effect of tendon stiffness, linear elastic material property conditions were input for the tendons in a subsequent parametric series. A baseline Young's modulus ($E = 65$ MPa) was assumed [8], and was scaled by 0.01, 0.2, 5, and 10 times, respectively (Table 4). The results again showed that the level of solid member stress due to contact between the nerve and tendons remained far greater than the fluid pressure, with tendon stress being greater than nerve stress. When tendon Young's modulus was changed, the peak stress in tendon showed the expected linear relationship with tendon modulus. However, fluid pressure and fluid velocity remained almost unchanged. There was a strongly non-linear relationship between tendon stiffness and nerve stress. From this, it is apparent that tendon and nerve stresses in the carpal tunnel would strongly depend on the effective transverse stiffness of the tendon, in turn presumably a function of activity-dependent longitudinal tension in the tendons.

Finally, the tractability of extending a multi-body solid contact simulation to three-dimensions was investigated (Figure 8). Because of the far larger problem size in the three-dimensional solid contact model (33,030 elements), the computation time required to simulate the 0.5-second physical event of the baseline solution increased up to approximately 550 seconds. Young's modulus of the deformable tunnel wall was assumed as 65 MPa, and several nodes on the outer surface of the tunnel wall were completely fixed. The simulation results showed that the nerve was again gradually squeezed between the nearby tendons and inner surface of the wall boundary during the tendons' excursions (Figure 9a). Also, contact stresses developed on the surface of the carpal tunnel wall due to nerve abutment (Figure 9b). From this, it could be inferred that direct compressive solid contact(s) due to tendon motions could potentially cause appreciable mechanical insult to the nerve, above and beyond any effects from fluid pressure. Although the three-dimensional solution showed obvious end-effect influences near the proximal and distal ends of the (extended 2-D) study region, solution output near the midsection was reasonably prismatic, and the absolute stress values that emerged reasonably approximated those for the two-dimensional counterpart.

DISCUSSION

The purpose of this exploratory study was to develop and preliminarily exercise a numerical formulation, by means of which it would be possible to estimate mechanical stress on the median nerve of the carpal tunnel. Such stress would be due to effects of direct solid contact with nearby anatomical structures (flexor digitorum tendons, carpal tunnel boundary), and simultaneously due to pressurization of the synovial-like fluid within which the carpal tunnel contents are immersed. To our knowledge, this represents the first instance of an immersed multi-body contact finite element formulation in the field of orthopaedic biomechanics.

Both the two- and three-dimensional human carpal tunnel FSI finite element models were numerically well-behaved, and the parametric results were intuitively credible. Although important near-term steps are obviously needed regarding material property determination, kinematic input delineation, and physical validation, this new numerical formulation opens the way for systematic quantitative study of median nerve mechanical insult for immersed multi-body finite-deformation contact in the carpal tunnel.

Typical resting fluid pressures measured experimentally in the carpal tunnel are on the order of 1 to 2 kPa for normal subjects [9] and 1 to 5 kPa for CTS patients [10], with additional activity-induced fluctuations being on the order of 2 to 4 kPa [9]. Given the vagaries of biological variability and measurement artifact present experimentally, along with the various assumptions and simplifications in the numerical model, these ranges of measured fluid pressure (1 to 10 kPa) are very reasonably consistent with the ranges of fluid pressure computed in the finite element model (1 to 12–24 kPa). In view of that nominal parity, and even allowing for the obvious approximations and uncertainties in these exploratory numerical series, median nerve stresses resulting from this general range fluid pressure values and fluctuations thus appears to be far below the levels plausibly resulting from direct structural contact (~ 200 kPa). Moreover, the particular nerve entrapment configuration presently simulated was relatively mild, whereas it is possible for the nerve to be extruded even fully across the tunnel in vigorous functional activity (Figure 10). Admittedly, unlike chronic elevation of resting fluid pressure, these dramatic levels of contact-associated median nerve stress elevation would be expected to arise only during transient provocative activities. In principle, however, such episodic contact stress elevations would be no more infrequent than the (much smaller) fluid pressure elevations conventionally viewed as CTS-provocative, arising from those same transient activities. Hence, while very preliminary, the present computational results suggest that direct structural contact may be a substantial contributor to the pathogenesis, and possibly even a dominant influence.

This new FE formulation provides a basis for systematically studying median nerve mechanical insult from CTS-provocative activities, at a level of quantitative rigor not previously possible. From further work with this model, useful information presumably can be obtained to help choose reasonable stress levels for cell culture or molecular mechano-biological studies of CTS pathogenesis, including the effect pathways of potential therapeutic agents [2]. Also, FE stress results may provide a basis for better identifying risk factors of individual subjects' anatomy, and for studying biomechanical aspects of surgical interventions such as transverse carpal ligament release.

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FIGURE 1.
Anatomical Image of the Human Carpal Tunnel

FIGURE 2.
Carpal Tunnel 2-D FE Model

FIGURE 3.
Carpal Tunnel 3-D FE Model

FIGURE 4.
Basic Configuration of Carpal Tunnel FE Model

FIGURE 5.

Comparison of Solid and Fluid Mesh Structure for Baseline Analysis Conditions in the 2-D Model. Note the more complete entrapment of the nerve against the tunnel wall as the tendons migrate upward (volarly) from $t=0.35 \text{ sec}$ to $t=0.5 \text{ sec}$.

FIGURE 6.
Comparison of Solid Stresses and Fluid Pressure for Baseline Analysis Conditions in the 2-D Model

FIGURE 7. Comparison of Fluid Velocity at two Instants for Baseline Analysis Conditions in the 2-D Model

FIGURE 8.

Three-Dimensional Solid Contact Carpal Tunnel FE Model. Nodes on the external surface of the tunnel wall are modeled as fixed.

a) Solid Mesh Structure ($t = 0.5$ s)

b) Solid Stress Distribution ($t = 0.5$ s)

FIGURE 9.
Simulation Results of 3-D Carpal Tunnel FE Model under Solid Contact Conditions

FIGURE 10.

Magnetic Resonance Image (3 Tesla, DESS Sequence, TR/TE=14/4.4 msec) of Tendon and Median Nerve Migrations during an Isometric Wrist Flexion Resistance Activity. Beginning from its volar position in the resting state (left panel), the nerve is extruded dorsally (right panel) due to the volar-ward migrations of the tensed flexor tendons.

Table 1

A Baseline Material Properties of Shear and Bulk Modulus for Nerve and Tendons

	Long-term (G_{∞}, K_{∞})	Short-term (G_0, K_0)	Decay factor (β)
Shear (G)	150 kPa	500 kPa	35 s^{-1}
Bulk (K)	1,450 kPa	4,830 kPa	35 s^{-1}

Table 2

Computed Physical Values in 2-D Carpal Tunnel FE Model with Baseline Conditions

Max. Median Nerve Stress	201.0 (kPa) at $t = 0.5$ s
Max. Tendon Stress	258.9 (kPa) at $t = 0.5$ s
Max. Fluid Pressure	1.482 (kPa) at $t = 0.5$ s
Max. Fluid Velocity	135.6 (mm/s) at $t = 0.45$ s

Table 3
Effects of Fluid Viscosity (Baseline Parameter Case A3)

	Fluid Viscosity		Max. Tendon Stress		Max. Nerve Stress		Max. Fluid Pressure		Max. Fluid Velocity	
	[Ns/m ²]	Ratio	[kPa]	Ratio	[kPa]	Ratio	[kPa]	Ratio	[mm/s]	Ratio
Case A1	0.001	0.05	258.9	1.000	201.2	1.001	0.080	0.054	131.7	0.971
Case A2	0.002	0.10	258.9	1.000	201.2	1.001	0.154	0.104	137.5	1.014
Case A3	0.02	1.00	258.9	1.000	201.0	1.000	1.482	1.000	135.6	1.000
Case A4	0.2	10.0	259.6	1.003	199.8	0.994	12.78	8.623	127.5	0.940
Case A5	0.5	25.0	260.3	1.005	198.6	0.988	24.06	16.23	117.9	0.869

Table 4
Effects of Tendon Young's Modulus (Baseline Parameter Case B3)

	Tendon Young's Modulus		Max. Tendon Stress		Max. Nerve Stress		Max. Fluid Pressure		Max. Fluid Velocity	
	[MPa]	Ratio	[MPa]	Ratio	[kPa]	Ratio	[kPa]	Ratio	[mm/s]	Ratio
Case B1	0.65	0.01	0.118	0.011	93.7	0.514	0.489	1.043	62.6	1.011
Case B2	13.0	0.20	2.212	0.200	167.5	0.918	0.455	0.970	62.0	1.002
Case B3	65.0	1.00	11.06	1.000	182.4	1.000	0.469	1.000	61.9	1.000
Case B4	325.0	5.00	55.30	5.000	185.7	1.018	0.472	1.006	61.9	1.000
Case B5	650.0	10.0	110.6	10.00	186.1	1.020	0.472	1.006	61.9	1.000