A STRAIN-ENERGY APPROACH TO SIMULATING SLOW FINGER MOVEMENTS AND CHANGES DUE TO LOSS OF MUSCULATURE

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INTRODUCTION

The neuromuscular interactions that produce slow and accurate finger movements are not understood. The geometry of finger anatomy is such that a slow finger movement (i.e., sequence of joint rotations) completely defines the necessary excursions of all tendons. Given that muscle tone prevents tendons from becoming slack, all tendons must undergo eccentric or concentric excursions during functional finger movements. In this paper, we describe a novel computational solver to model slow finger movements, and report how the mathematics of strain-energy of over-constrained systems can predict the impairment in finger motion that accompanies partial paralyses.

METHODS

A slow moving finger is best modeled as a first order dynamical system having low mass and performing quasi-static motion (which is not the case for fast finger movements [1]). Our 3-D model consists of a kinematic 3-link mechanism with 2 degrees of freedom (ad-abduction and flexion-extension) at the metacarpophalangeal joint (MCP) and 1 degree of freedom (flexion-extension) at the proximal interphalangeal (PIP) and the distal interphalangeal (DIP) joints. All seven muscles of the index finger were included: *flexor digitorum profundus* (FDP), *flexor digitorum superficialis* (FDS), *extensor indicis proprius* (EIP), *extensor digitorum communis* (EDC), *first lumbrical* (LUM), *first dorsal interosseous* (FDI), and *first palmar interosseous* (FPI). The routing of the tendons across finger joints is represented by the moment arm matrix obtained from [2]. Our focus here is the computational engine, thus for now we have neglected the extensor mechanism, but its effects will be considered in subsequent work. Torsional springs at the joints simulate the known passive stiffness arising from skin and soft tissue. The muscles actuating the index finger are modeled as tunable linear elastic springs that only exert force in tension. Changing the latent resting length of a spring, l_0, results in a 'muscle force' that is proportional to the difference between its current and resting lengths. Therefore the "activation signal" to a muscle is simulated as a change in resting length, Δl_0 (Fig. 1). Here, we are only interested in understanding how finger movement arises from Δl_0, and simply assume that Δl_0 can arise from a neural command. A set of commanded resting lengths to all muscles defines the posture attained by minimizing the strain-energy of the finger at equilibrium: where the net joint torques produced by the muscles, τ_{ext}, are exactly balanced by the torques produced by joint-stiffness, τ_{in}.

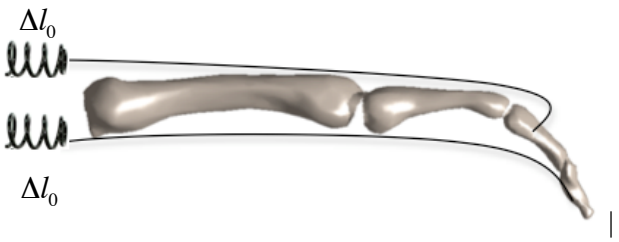

Figure 1: Model of the index finger being actuated by tunable springs.

$$\Delta s = R^T(\Delta\theta)$$
$$\tau_{ext} = RK_m(\Delta l_0 - \Delta s)$$
$$\tau_{in} = K_\theta(\Delta\theta)$$

where Δs is the change in tendon excursions, R is the moment arm matrix, K_m is the matrix of muscle spring constants, Δl_0 is the change in spring resting lengths, K_θ is the matrix of torsion spring constants at the joints and $\Delta\theta$ is the resulting change in joint angles.

Given that muscles behave like nonlinear springs that do not exert compressive force, there is an extra constraint defined by,

$$K_m \approx 0 \; if \; \Delta l_0 < R^T \Delta\theta$$

Also, we ensure that $\Delta l_0 > 0$ since muscle activation always leads to a net force greater than passive stretch force. We solve the forward kinematic problem iteratively using a Newton-Raphson iteration method.

As a first demonstration of our system, we simulated a simple quasi-static tapping motion by finding an appropriate trajectory of muscle resting length changes. The movement consisted of an initial retraction of the index finger followed by a downward flexion motion (Fig. 2). In addition, we simulated the resulting trajectory for acute radial, median and ulnar nerve palsies by removing the muscle commands to the i) two extensors, ii) the two flexors, and iii) the two interosseii and lumbrical, respectively and applying the same resting length change commands as before to the unaffected muscles. This simulates effects prior to neuromuscular adaptation (i.e., the robustness of finger trajectories to acute loss of some muscles). Note that the affected muscles continue to exert passive stretch forces even without the activation command.

RESULTS AND DISCUSSION

Not surprisingly, the fingertip deviates from the original trajectory when some muscle commands are removed. Some postures and directions of movement become impossible without the presence of specific muscles.

However, the value of these simulations is that they begin to explain the relative effect of different deficits and indicate which muscles are more or less critical to the execution of accurate and slow finger movements. In addition, finger movements are typically modeled using second order dynamics [3,4]. Here we are proposing that a simple quasi-static solver based on strain-energy equilibrium can model slow finger movements since mass and inertia properties of fingers are small. While muscle mechanics depends on neural activation and a muscle's force-length and force-velocity properties, the end product is a force command, which we generate by the adjustment of resting lengths of fictitious springs.

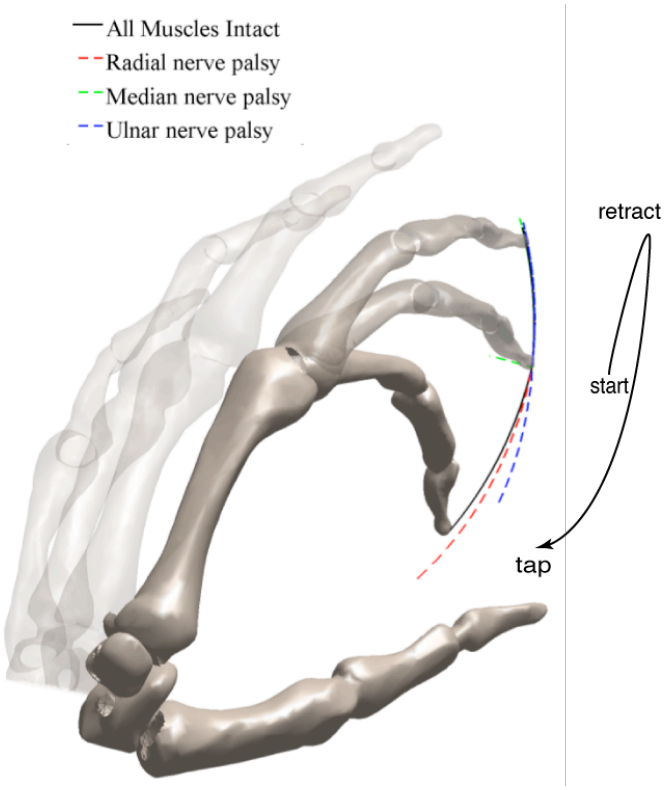

Figure 2. Fingertip trajectories for a simple tapping motion in unaffected and impaired cases.

Future work will incorporate tendon routing functions derived directly from cadaveric experiments to simulate more faithfully the extensor mechanism, as well as a more physiologically realistic muscle model.

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