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Model-Based Estimation of Knee Stiffness

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Abstract

During natural locomotion, the stiffness of the human knee is modulated continuously and subconsciously according to the demands of activity and terrain. Given modern actuator technology, powered transfemoral prostheses could theoretically provide a similar degree of sophistication and function. However, experimentally quantifying knee stiffness modulation during natural gait is challenging. Alternatively, joint stiffness could be estimated in a less disruptive manner using electromyography (EMG) combined with kinetic and kinematic measurements to estimate muscle force, together with models that relate muscle force to stiffness. Here we present the first step in that process, where we develop such an approach and evaluate it in isometric conditions, where experimental measurements are more feasible. Our EMG-guided modeling approach allows us to consider conditions with antagonistic muscle activation, a phenomenon commonly observed in physiological gait. Our validation shows that model-based estimates of knee joint stiffness coincide well with experimental data obtained using conventional perturbation techniques. We conclude that knee stiffness can be accurately estimated in isometric conditions without applying perturbations, which presents an important step towards our ultimate goal of quantifying knee stiffness during gait.

I. Introduction

During unimpaired gait, humans vary not only the forces generated by their lower limbs, but also the limb stiffness. Depending on the terrain, one might either walk in a relaxed manner, or one might stiffen up joints by engaging antagonistic muscles to increase stability or to absorb impacts. This stiffening can happen subconsciously through activation-dependent changes in intrinsic muscle properties [1], [2] or changes in reflex behavior [3]. Lower-limb amputees, who rely on prostheses do not have this ability, and therefore are often challenged when locomoting across different terrain. With the advent of powered prostheses, modulation of the apparent stiffness of a prosthetic leg is now possible [4], [5]. Such modulation may allow the adaptability and complexity of unimpaired gait to be replicated in

prosthetic devices. First, however, more quantitative descriptions of the physiological variations in leg stiffness are required.

Few experimental estimates of knee stiffness have been reported. Two studies have quantified knee stiffness at varying levels of isometric torque, but neither considered co-contraction of agonist and antagonist muscles, as is present during many phases of locomotion [6], [7]. The effect of co-contraction on knee joint stiffness has only been addressed by one study [8], in which three subjects were asked to exert an unspecified amount of constant co-contraction. The resulting stiffness was higher than passive stiffness, but the muscle activation was not controlled, making it difficult to predict how specific patterns of muscle activation would influence knee stiffness.

We are unaware of any direct experimental estimates of knee stiffness during locomotion. This is likely due to the experimental challenges associated with making such measurements. These include the need to attach rigidly to the limb so that appropriate perturbations can be applied, while simultaneously not impeding natural behavior. Some have attempted to estimate knee stiffness by examining the slope of the torque angle curve [9], but this does not represent the instantaneous mechanical properties of the joint [10].

An alternative approach to direct experimental estimates is to obtain model-based estimates. It was recently demonstrated that the stiffness of the human arm can be accurately estimated during isometric tasks by considering only the geometry of the limb and the short-range stiffness properties of the active muscles [11]. While these results were encouraging, they only considered conditions in which minimal co-contraction was present. A critical step in the model-based approach is the estimation of muscle forces. During tasks in which there is little co-contraction, muscle forces can be estimated reliably by considering the forces generated by the limb, and using optimization to distribute those forces across the relevant muscles [12], [13]. However, such optimization strategies fail in the presence of co-contraction [14], [15]. In contrast, EMG-guided optimization can be used to estimate muscle forces during tasks that involve co-contraction [16], [17]. This hybrid experimental-modeling approach may also be useful for noninvasively estimating knee stiffness during co-contraction.

The long-term goal of this work is to develop a model-based estimate of knee stiffness that can be used to estimate stiffness during the dynamic conditions relevant to locomotion. This manuscript presents the first step in that process. Specifically, we have develop an EMG-guided, model-based approach for estimating knee stiffness during isometric contractions with substantial co-activation of antagonistic muscles. We validated this approach by comparing it to experimental estimates of knee stiffness. Our experimental measurements considered a range of perturbation amplitudes, so as to identify the range over which model-based predictions would be valid. A sensitivity analysis was used to evaluate the influence of model parameters on the accuracy of the estimated knee stiffness. Our results demonstrate that a model-based approach can accurately predict the activation-dependent changes in knee stiffness that occur during isometric contractions.

II. Model

To estimate active knee joint stiffness, we used an algorithm involving three successive steps (Fig. 1). First, we estimated individual muscle forces from torque or electromyogram (EMG) recordings. Second, we used these forces to estimate muscle stiffness. Finally, we mapped these muscle stiffnesses to joint stiffness, considering the individual muscle moment arms.

A. Musculoskeletal Model

We used the knee portion of the lower-limb model developed by Arnold et al. [18]. This knee model contained the muscles listed in Table I. Each muscle-tendon unit was described by the following parameters: maximal isometric force f_M^0, pennation angle α, optimal fiber, length l_M^0 and tendon slack length l_{TS}^0. The model also employs a generic Hill-type force-length relationship $f(l)$ [19]. All parameters represented a nominal subject, as described by Arnold et al., and were not modified for the purposes of our study. The outputs of the model were the vectors of moment arms $\boldsymbol{r}(\varphi_K)$ and muscle lengths $\boldsymbol{l}(\varphi_K)$.

B. Estimation of Muscle Forces

We used two methods to estimate muscle forces. The first involved static optimization, which is sufficient in situations without co-contraction. The second incorporated EMGs to estimate muscle forces when co-contraction was present.

1) Muscle Force Estimation from Net Joint Torque—The static optimization used a min-max objective function to distribute muscle forces equally after normalization by their maximum isometric muscle force at their current operating length [20]. Other cost functions have been proposed in the literature (see [13]), but tend to produce similar muscle force estimates [14]. Our preliminary studies also demonstrated that the estimated knee stiffness was not sensitive to the choice of cost function [21]. Hence, we have opted to use the one that is most computationally efficient.

2) EMG-Guided Muscle Force Estimation—To estimate muscle force when co-contraction was present, we employed a two-step process. First, we used EMGs to estimate the flexor (τ_{flex}) and extensor (τ_{ext}) contributions to the net joint torque τ. Then, we used static optimization to estimate the individual muscle contributions to τ_{flex} and τ_{ext}, as described above.

Estimates of τ_{flex} and τ_{ext} were obtained using surface-EMGs from the 7 accessible muscles (see Table I), together with the following model:

$$\tau = \boldsymbol{c}^T \boldsymbol{a} - \boldsymbol{c}_{ext}{}^T \boldsymbol{a}_{ext} + \boldsymbol{c}_{flex}{}^T \boldsymbol{a}_{flex} = \tau_{ext} + \tau_{flex}. \quad (1)$$

In this model, $\boldsymbol{a}$ is the vector of average rectified EMGs for each isometric contraction and $\boldsymbol{c}$ is the parameter vector relating EMG to torque. The vectors $\boldsymbol{a}_{ext}$, $\boldsymbol{c}_{ext}$ contain only the values for the extensor muscles, and $\boldsymbol{a}_{flex}$, $\boldsymbol{c}_{flex}$ for the flexors. The parameter vector, $\boldsymbol{c}$, was estimated using linear regression, as described in Section III-C2.

C. Model for Musculotendinous-Stiffness

Musculotendinous stiffness was modeled as muscle stiffness k_M in series with tendon stiffness k_T: $k_{MT} = k_M k_T / (k_M + k_T)$ (e.g. Morgan et al. [22]).

Three different tendon models were evaluated. The first was that proposed by Zajac [23], including the nonlinear toe-region. The Zajac model scales the linear region of the tendon stiffness curve by the maximum isometric force of the muscle. Our second model replaced this scaling with that proposed by Cui et al. [24], based on the geometrical parameters of the tendon. This second model, which also contained a toe-region, was found to be most accurate and was used to generate all results except for the tendon model comparisons in Section IV-E. The last tendon model exactly matches that proposed by Cui. It contains the

same geometric scaling as the second model, but assumes that the tendon stiffness is linear without any toe-region.

Muscle stiffness was modeled as being proportional to muscle force f_M and inversely proportional to optimal fiber length l_M^0 [24], [25]: $k_M = \gamma f_M / l_M^0$.

D. Kinematic Mapping to Joint Stiffness

Musculotendinous forces $\boldsymbol{f}_{MT}$ and stiffnesses $\boldsymbol{k}_{MT}$ were mapped to joint stiffness K_a by

$$K_a = \frac{\partial \boldsymbol{r}^T}{\partial \varphi_K} \boldsymbol{f}_{MT} + \boldsymbol{r}^T \boldsymbol{K}_{MT} \boldsymbol{r}, \quad (2)$$

where $\boldsymbol{r}$ is the vector of moment arms and $\boldsymbol{K}_{MT}$ is a diagonal matrix containing the musculotendinous stiffnesses, $\boldsymbol{K}_{MT} = \mathrm{diag}(\boldsymbol{k}_{MT})$ (see [26]).

III. Evaluation Methods

A. Experimental Setup

Subjects were seated in a Biodex chair and secured with straps across the torso. Movement of the thigh was restricted by a strap and a cushioned wooden board placed above the edge of the chair (Fig. 2). A computer-controlled brushless servomotor (BSM90N-3150AX; Baldor Electric Company, Fort Smith, AR) was used to apply angular position perturbations to the knee joint. Angular feedback was provided by an encoder with an effective resolution of $6.3 \cdot 10^{-5}$ rad. Forces and moments were measured using a six degree-of-freedom load cell (630N80; JR3, Inc, Woodland, CA), and the motor was controlled in real-time using Matlab xPC. EMGs were acquired using an 8-channel amplifier (AMT-8; Bortec Biomedical, Calgary, Canada) with differential pre-amplifiers (APE-500; Bortec Biomedical). The electrodes were Ag-AgCl, pregelled, bipolar electrodes with a pole spacing of 2.5 cm (FT-007, MVAP Medical Supplies, Newbury Park, CA). All analog signals were passed through suitable antialiasing filters before data acquisition. Data were sampled at 5000 Hz for the first three subjects, and 2500 Hz for the other two.

Each subject's leg was attached to the motor using a custom-made thermoplastic cast extending from the toes to just below the knee. The knee center of rotation was aligned with the axis of the motor and locked into position at 60° knee flexion. The back rest was set at 85°. This configuration was selected to be consistent with previous studies on knee stiffness and to have a low contribution of passive joint stiffness [27].

B. Experimental Protocol

Five healthy male subjects (age: 27–31 years, height: 175–193 cm, weight: 71–89 kg) participated in the study. All procedures were approved by the Institutional Review Board at Northwestern University and all subjects gave informed consent. Only the right leg was measured.

Joint impedance describes the forces generated at a joint in response to external perturbations of posture. We estimated knee impedance using stochastic displacement perturbations with a standard deviation (SD) of 0.5°. The frequency spectrum of the perturbations was tailored for the purpose of stiffness estimation, which is the steady state component of impedance. Gaussian white noise was low-pass filtered with a transfer function having 2 poles at 5 Hz and 8 poles at 10 Hz. This reduced the torque due to inertia, which dominates the perturbation response at higher frequencies. These perturbations

allowed impedance to be estimated between 0–10 Hz, while keeping the experimental torques to values within measurement range of our load cell.

EMGs were collected from the 7 muscles acting about the knee that were easily accessible using surface electrodes (See Table I). Before the perturbation measurements, subjects performed at least two maximum voluntary contractions (MVCs) for knee extensors and knee flexors. The obtained torques were recorded and used to scale the target torque levels in the experiment. The experimental trials lasted for 35 seconds, during which subjects were instructed to produce constant knee torques, and to not react to the imposed perturbation.

There were two experiments, with a short break in between. The first experiment had a constant perturbation amplitude of 0.5° SD. The target torque levels were 10, 20, and 30 %MVC in the flexion and extension directions. Measurements also were made in a passive condition, during which subjects were instructed to relax (0 %MVC). Each of these 7 conditions without co-contraction was repeated twice, resulting in 14 trials. Similar trials were conducted with co-contraction. These had net torque levels of −10 %MVC, 0 %MVC and 10 %MVC. Two co-contraction levels were tested for each net torque level. These corresponded to a 5 %MVC (low level) and 10 %MVC (high level) contraction in the antagonist muscle. This resulted in 6 conditions with co-contraction with three repetitions each, yielding 18 trials.

Experimental estimates of stiffness are known to vary with perturbation amplitude. The second experiment was designed to quantify the magnitude of these variations relative to the activation-dependent changes predicted by the model. For this experiment, we used perturbations of 5 different SDs (0.5°, 1°, 1.5°, 2 ° and 2.5°). Only conditions without co-contraction, at −10 %MVC, 0 %MVC and 10 %MVC were considered. Two repetitions were obtained for each combination of perturbation amplitude and contraction level, resulting in 30 trials. For both experiments, the presentation order of the trials was randomized and there was a break of approximately 60 seconds between successive trials.

Subjects were provided with visual feedback to assist with the maintenance of the target net torque and co-contraction level. Net torque (target and actual) was displayed for all conditions. For conditions with co-contraction, the co-contraction level also was displayed. When the target torque was zero or in flexion direction, co-contraction was defined as a weighted sum of the three measured extensor muscles; the weight for each muscle was proportional to its modeled maximum isometric force. We preferred this scaling over the regression model described in Section II-B2, because the regression model would have been sensitive to variations in muscle force distribution among synergistic muscles with respect to the calibration trial. When the target torque was in extension, co-contraction was defined as the difference between the weighted sum of the extensor muscle EMGs and net torque measured by the load cell. All feedback signals were low-pass filtered (cutoff frequency at 0.5 Hz) to minimize the influence of the perturbation on the feedback.

C. Data Analysis

1) Stiffness Identification from Perturbation Measurements—Knee impedance was estimated by fitting a second-order system to the magnitude of the impedance frequency response function [28]:

$$\left|\frac{\tau_K(s)}{\varphi_K(s)}\right| = |Is^2 + Bs + K|. \quad (3)$$

The parameter I denotes inertia, B viscosity, and the static gain K corresponds to elasticity or stiffness, which we compared to model estimates. This model considers only the perturbation response of the joint, not the steady-state torques due to the constant level of muscle activation or the contributions of gravity; all steady-state torques were removed prior to system identification. For data fitting, the recorded signals were decimated to 100 Hz. The frequency response function was estimated using Fast Fourier Transforms with windows of 256 samples and an overlap of 128 samples. Position and torque data, and the corresponding coherence function [28] and frequency response function for a typical trial are depicted in Fig. 3. The first 5 seconds of each trial were discarded, and only the remaining 30 s were used, to eliminate possible transients at the start of each trial. To quantify how well the parametric fit described the frequency response function (Eq. 3), we also looked at the variance in the torque accounted for (VAF) by the parametric fit. Because the model only predicts active stiffness K_a, the passive stiffness K_p, estimated from the 0 %MVC trials, was subtracted from all measurements.

2) Model-Based Stiffness Estimation—For conditions without co-contraction, the model-based estimates of knee stiffness were described as a continuous function of torque, because the optimization criterion used to estimate muscle forces had a unique solution for any net torque.

For conditions with co-contraction, stiffness only could be estimated at the experimentally measured torques since the estimates of muscle activity depended on the measured EMGs within each experimental trial.

EMGs were band-pass filtered from 20–500 Hz using a 4th order, zero-phase digital Butterworth filter. The resulting signal was rectified and averaged to provide a single measure of muscle activity for each trial. The “activity” measured from a passive calibration trial without any joint perturbations was subtracted from that in the active trials in an effort to remove baseline noise.

Separate trials were used for estimating and evaluating the regression parameters describing the static EMG to torque relationship (Eq. 1). Of the 32 available trials, 20 were randomly selected for parameter estimation and 12 were used for evaluation. This procedure of random selection and cross-validation was repeated 500 times.

3) Perturbation-Evoked Muscle Activity—Our model considers only steady-state muscle activity, but some of this activity may arise from reflex pathways excited by the perturbation. To assess this possibility, we quantified the relationship between joint velocity and EMG for each of the measured muscles, by using the linear, non-parametric impulse response function and the variance it accounted for in the EMG. More complex models [29] did not improve the prediction accuracy.

4) Sensitivity Analyses—The influence of the selected tendon model was evaluated by comparing the knee stiffness estimated when using the three tendon models described in Section II-C.

Muscle moment arms vary substantially across individuals [30]. The influence of moment arm variability was assessed using a Monte Carlo analysis with 500 repetitions. For each repetition, the moment arm for each muscle was selected randomly from a Gaussian distribution with a mean and SD matched to that reported by Buford et al. [30]. Each moment arm was expressed as a percentage of the nominal moment arm used in our model. On average, this percentage was 22.3±5.1 % for extensors and 18.1 ± 4.8 % for flexors.

To investigate the effect of incomplete EMG recordings and to assess the importance of each muscle used in the process, we systematically removed muscles from the EMG-guided estimation procedure. The EMG-guided procedure (Section III-C2) was performed using all possible subsets of 5 and 6 out of the 7 measured muscles. We looked at the magnitude of the change in stiffness relative to the case where all 7 muscles were used for the estimation, to demonstrate the change that can be expected when certain EMGs are omitted. Mean and SD over all subjects and all trials were calculated.

IV. Results

A. Torque-Based Stiffness Estimates

Model-based estimates of knee stiffness matched the experimental estimates very well during conditions in which there was no intentional co-contraction (Fig. 4). Under these experimental conditions, muscle force was estimated from the measured joint torque using optimization (Section II-B1). Across all subjects, the model-based predictions of stiffness for each trial were 7.3 ± 14.1 % lower than the experimental estimates. The VAF in the torque data by the experimental estimates (Eq. 3) was 90 ± 2 % during the passive trials, and 87 ± 6 % in the active trials without co-contraction.

The experimental data showed a drop in the coherence at the resonance frequency. The resonance frequency shifted from around 1.5 Hz for the passive trials, up to around 4 Hz for the trials at 30 % MVC.

B. EMG-Guided Stiffness Estimates

When co-contraction was present, EMGs were used to estimate muscle activity in the flexors and extensors, as well as the corresponding torques produced by these muscles. The sum of the EMG-estimated flexion and extension torques provided an estimate of the net torque about the knee. These predictions of net torque based on EMG measurements closely matched the net torque measured by the load cell in all conditions. In conditions without target co-contraction, the mean difference between measured and predicted torque was −0.4±2.5 Nm over all trials and all subjects (Fig. 5a). Conditions with target co-contraction resulted in an mean difference of 0.3 ± 1.8 Nm (Fig. 5b).

The EMG-guided predictions of knee stiffness agreed well with the experimentally estimated values in conditions both without and with co-contraction. Experimental values were slightly lower than model predictions. Over all subjects and all trials without target co-contraction (passive trials excluded), the experimental estimates were 0.01 ± 24.4 % lower than the EMG-guided model-based estimates (Fig. 6a). For the trials with co-contraction, the experimental estimates were 21.0±22.3 % lower than the model-based estimates (Fig. 6b). The VAF in the torque data by the experimental estimates (Eq. 3) was 80 ± 7 % for the trials with high co-contraction, and 84 ± 5 % for the trials with low co-contraction.

C. Dependence of Experimentally Measured Stiffness on Perturbation Amplitude

Experimental estimates of knee stiffness varied with perturbation amplitude. During 10 %MVC contractions in the flexion and extension directions, net stiffness decreased as perturbation amplitude increased. Over the range of tested amplitudes, stiffness decreased by 27.0±17.2 Nm/rad in flexion, and by 53.9±13.2 Nm/rad in extension (Fig. 7a). For the same change in perturbation amplitude, passive stiffness decreased by 27.9 ± 5.9 Nm/rad (Fig. 7b). These results were not related to a change in the system dynamics. The 2nd-order model fits had a VAF of 89±4 % for the smallest perturbations and 93 ± 2 % for the largest perturbations. These results suggest that more than half of the amplitude-dependent change in net knee stiffness during extension contractions comes from the passive properties of the

joint, and that nearly all of the observed changes during flexion come from these same passive properties (Fig. 7c).

D. Perturbation-Evoked Muscle Activity

There was no substantial relationship between joint velocity and muscle EMG during the passive trials (VAF < 3 % for all amplitudes). In contrast, a larger percentage of the EMG variance could be predicted during the active trials. Across all muscles, the VAF ranged from 18 ± 14 % for the 0.5° perturbation amplitude, up to 32 ± 20 % for the 2.5° perturbation amplitude. These results suggest that some of the muscle activity recorded during the active trials could be attributed to reflex mechanisms excited by the continuous, stochastic perturbations.

E. Model Sensitivity

Knee stiffness predicted by the model varied substantially with the choice of tendon model. Predictions using Zajac's tendon model were generally higher than experimentally identified stiffness (Fig. 8). In extension, predictions were 14.4 ± 15.8 % higher, in flexion they were 59.8 ± 25.0 % higher than experimental values. Cui's tendon model yielded a good agreement between model predictions and experimental data at high torque levels. Overall, predictions were still higher than experimentally identified values; in extension, they were 3.8 ± 17.1 % higher, in flexion they were 22.4 ± 27.4 % higher. The adjusted Cui model, incorporating a toe region, produced the most accurate results across all tested conditions. In extension, predictions were 5.9 ± 13.3 % lower than experimentally identified stiffness, in flexion they were 8.8 ± 15.0 % lower.

Model predictions of joint stiffness were sensitive to changes in moment arms, as expected due to the quadratic dependence on that parameter (Eq. 2). The SD in stiffness obtained from the 500 Monte-Carlo simulations was 22.3±1.1 % in flexion, and 27.4±1.6 % in extension, with respect to the mean over the considered torque range from −40 Nm to 60 Nm. Compared to variation of the moment arms (18.1 % in flexion and 22.3 % in extension), this resulted in a relative sensitivity of approximately 1.2 in both flexion and extension.

The EMG-guided stiffness estimates were sensitive to which EMGs were used. This was assessed by systematically removing individual EMGs and pairs of EMGs. The effect of removing any single muscle on the predicted stiffness was modest, ranging from approximately 2.2–11.9 % across all measured muscles. As would be expected, omitting two EMG signals in the estimation process generally had a higher influence on joint stiffness prediction than omitting one. The largest effect was seen when two highly used synergistic muscles were removed, e.g. VM and VL (change of 41.9 %), or ST and BFL (change of 29.1 %), presumably making it difficult to estimate torque in direction of the synergistic pair. When omitting other combinations of two EMG signals, the average change in predicted stiffness was less than 16 %.

V. Discussion

Conventional approaches for estimating joint stiffness require perturbing the joint in a controlled manner, usually placing the limb inside a cast. Our goal was to develop a less disruptive method that may eventually be applied during functional tasks such as natural gait. Specifically, we developed a model-based method to estimate activation-dependent knee joint stiffness based on measurements of EMG, joint torque and joint angle, and evaluated this approach against experimental measurements during isometric tasks. We evaluated performance with substantial co-contraction, which is common in many functional tasks including locomotion. The EMG-guided stiffness predictions were most accurate when

little co-contraction was present, having an error of only 0.01 % on average. These errors were comparable to those based only on measures of joint torque (average error of 7 % across the same trials). EMG-guided errors increased to approximately 21 % when substantial co-contraction was present. These results provide the basis for model-based estimates of knee stiffness that can be used during functional tasks in which experimental measurements are not feasible. They also place bounds on the accuracy that can be expected from such model based approaches.

A. Experimental Characterization of Knee Stiffness

Our experimental estimates of knee stiffness determined are very similar to values reported by Zhang et al. [27], who considered only conditions without co-contraction. There are no reports on knee joint stiffness during co-contraction that could be directly compared to our results. Tai and Robinson [8] measured stiffness during co-contraction, but did not control for the amount of co-contraction. Nevertheless, their findings of increased stiffness during co-contraction, relative to passive conditions, are consistent with our findings. Our data further suggests that active stiffness due to the flexors and extensors adds linearly during co-contraction, as would be expected from muscles with minor mechanical interactions [31]. We also observed a decrease in VAF for more demanding trials such as trials with co-contraction, indicating that the subjects could not perfectly maintain a constant effort.

If reflex contributions to muscle activation were present in our experiments, they do not appear to have altered the model's ability to predict knee stiffness. This finding either suggests that reflex contributions were minimal, or that they simply contributed to the steady-state activation of the muscles. In an effort to distinguish between these possibilities, we quantified the relationship between the applied perturbations and the measured EMGs. While there was no substantial relationship during passive trials (VAF below 3 %), it was possible to predict 15–30 % of the EMG variance during the active trials. This suggests that reflex responses elicited by the small stochastic perturbations used in this study may have contributed to the measured knee stiffness. Given that our model only considers steady-state muscle activations, it appears that any reflex contributions to knee stiffness arose through contributions to the steady state muscle activation. More transient contributions to muscle activation and stiffness, as would arise from non-stochastic perturbations [3], could not be predicted by our model.

B. Model-based Estimates of Knee Stiffness

To our knowledge, this is the first model-based approach to predict active knee joint stiffness. A similar approach was recently used to model the force-dependent endpoint stiffness variations of the human arm [11], but conditions with co-contraction were not considered. Hence, EMG was not used in that previously published estimation process. Some models for the upper limb have incorporated EMG, but they either fit parameters to best match the experimentally determined stiffness [32], or used a musculoskeletal model that was determined for the specific subject and task [33]. In contrast, our approach was not fit to the experimental data or customized for these specific tasks. While further validation is necessary, especially for dynamic conditions, our approach may be useful for any subject and any situation where kinematic measurements and EMG can be obtained.

Our EMG-guided approach tended to overestimate knee stiffness when there was significant co-contraction. This is not surprising given the low levels of co-contraction considered and the fact that our torque-based estimates (Fig. 4), which ignore possible antagonist activity, were most accurate at low torque levels. Regardless of the source, these errors point to the limits on the accuracy that can be expected from this model-based approach to stiffness estimation.

C. Sensitivity of Model Estimates

Muscles are thought to operate in the region of short-range stiffness for fiber length changes of 1–3 % [1]. This is often used to explain the decrease in measured stiffness for increasing perturbation amplitude [8], [34]. Our results demonstrated that the passive properties of the joint can also contribute to a substantial portion of the observed amplitude dependence. While many studies have focused on the short-range stiffness of actively contracting muscles, this phenomenon also exists in passive muscles (see Proske and Morgan [35] for a review). Furthermore, the short-range stiffness of passive muscles exhibits an amplitude dependence, as reported in our work [36], [37]. In the active stiffness, almost no amplitude dependent change was observed for flexion. In extension, the change in stiffness from the lowest to the highest tested amplitude corresponded to a change in active stiffness that could be expected from a voluntary contraction change of approximately 5 Nm. Hence, at least for the knee, this amplitude dependence is relatively small compared to the large activation dependent changes in knee stiffness.

In contrast to perturbation amplitude, the tendon model had a large effect on the accuracy of the estimated knee stiffness. The tendon model which scaled stiffness properties according to the geometry of the muscle tendon [24] produced more accurate joint stiffness estimates than the model by Zajac, which scaled stiffness properties according to muscle force. By incorporating a nonlinear toe-region at low forces, estimation accuracy at low torque levels could be further increased. These results appear to differ from previous work in the upper limb, demonstrating that a linear tendon model with Zajac's scaling parameters for tendon stiffness was sufficient. This apparent difference arises from the fact that the difference between the tendon stiffnesses predicted by the Zajac and Cui methods is smaller for muscles of the upper limb than for those of the lower limb (unpublished results). While we achieved our goal of selecting the most appropriate tendon model for the knee, it is important to reevaluate the applicability of this model for other joints, before employing our same model-based approach for stiffness estimation.

Model-based estimates of knee stiffness also depended on the accuracy of muscle moment arms. These results are similar to previous findings for the arm [11], and can be attributed to the square relationship between muscle moment arms and joint stiffness. The relative sensitivity of stiffness estimates to variations in moment arms was approximately 1.2. Even with this sensitivity, there was a remarkable consistency in the knee stiffness measured across subjects (Fig. 4). For applications that require more accuracy, the uncertainty due to moment arm variations may be further reduced by considering population specific, or even subject specific parameters [38].

The number of EMG recordings affected the ability to generate accurate, model-based estimates of knee stiffness. This likely due to the corresponding effect on the estimation of muscle activity during co-contraction. The detrimental effect of reducing the number of EMGs was most dramatic when multiple muscles from a synergistic group were removed from the analysis. For the extensor muscle group, omitting VM and VL in the estimation process had the biggest influence; for the flexor group, omitting ST and BFL yielded the biggest change. For the extensors, these were precisely the muscles that are able produce the biggest force at the investigated joint angle. For the flexors, the ST is not able to produce a very large force, however, its EMG is presumably highly correlated with the neighboring SM [39], the strongest knee flexor at the tested posture. Eliminating any one muscle had a much less dramatic effect, as did the elimination of smaller muscles or muscles not likely used extensively in the tested posture (e.g. the GM and GL). These results suggest that a reduced muscle set could be sufficient for estimating knee stiffness, especially if it provided significant experimental advantages.

VI. Conclusions

This paper presents a model-based approach for unobtrusively estimating activation-dependent knee stiffness from measurements of EMG, joint angle and torque. It does not require the application of perturbations to the joint, as done conventionally to assess joint stiffness. Our indirect method was validated against the more direct perturbation-based approach and found to provide reasonably similar results. Due to the use of EMG, it was possible to quantitatively estimate activation of antagonistic muscles and, therefore, predict joint stiffness during co-contraction, a common feature of many functional tasks. An important feature of our method is that it was not customized to the subjects or fit to any experimental data. Rather, it uses scalable muscle models that have also been shown to work well for estimating the stiffness of the human arm. As a result, it represents a general approach that should be applicable to joints throughout the body, provided that the musculoskeletal architecture is available.

It should be noted that elastic stiffness is not the only component that defines the mechanical properties of a joint. Viscous and inertial components contribute substantially to joint impedance. Our model focuses only on joint stiffness, which is supposed to play a major role in the control of posture and movement [40]. We have used a model for short-range muscle stiffness to estimate the stiffness of the knee joint. Given our intended application to a variable-impedance prosthetic knee joint, a similar model for the viscous behavior could be interesting for future work.

The major limitation of our current approach is that it has only been validated for isometric conditions. While intrinsic muscle stiffness has been shown to follow muscle force during dynamic contractions [41], dynamic conditions may also invoke changes in neural commands, such as reflex activations, that are not contained in our current model. While it will be necessary to investigate such possibilities, the current work presents an important step towards quantification of stiffness-modulation during functional tasks, including those with dynamic contractions such as locomotion. Such quantification is important for the development of transfemoral prostheses that aim to replicate those behaviors.

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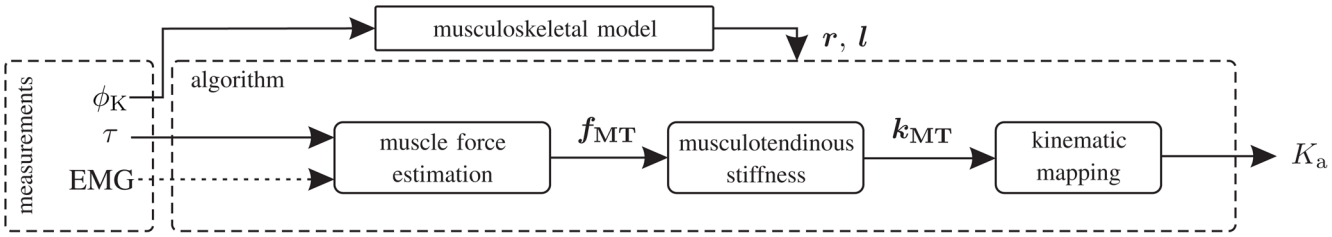

Fig. 1.

Overview of model to estimate active knee joint stiffness k_a. Two different methods are used for muscle force estimation, depending on whether co-contraction was present or not: one is based on net torque (Section II-B1) and one is based on EMG (Section II-B2). Inputs to the algorithm are knee flexion angle φ_K and knee torque τ measured by a load cell, or EMG signals. Intermediate variables are the vectors of musculotendinous forces $\boldsymbol{f}_{MT}$ and musculotendinous stiffnesses $\boldsymbol{k}_{MT}$, the vector of moment arms $\boldsymbol{r}$ and the vector of muscle lengths $\boldsymbol{l}$.

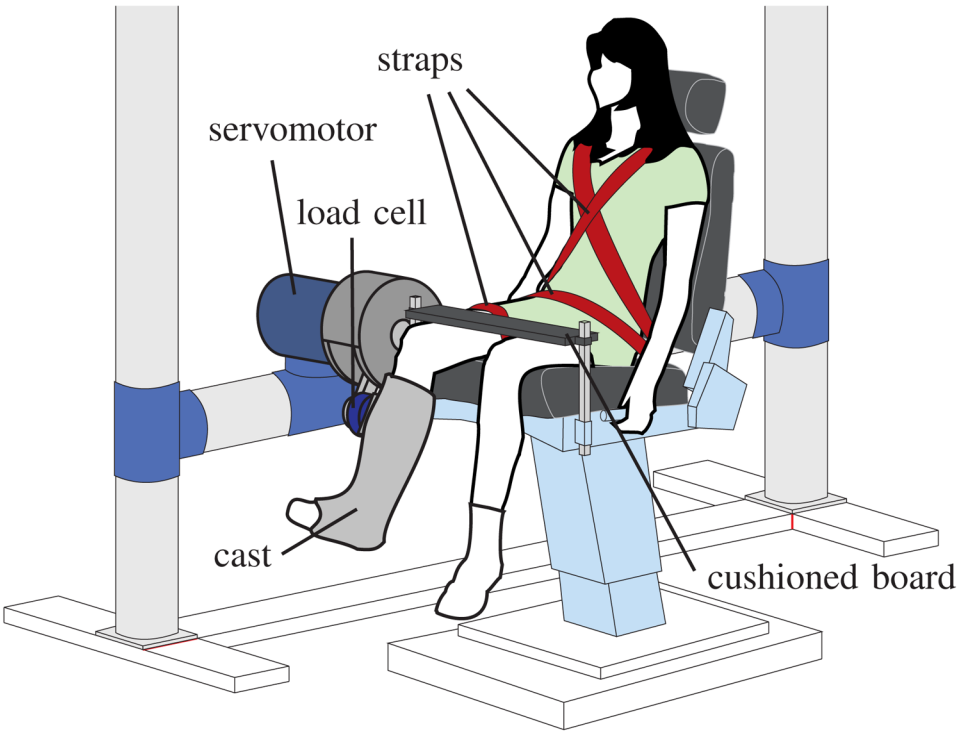

Fig. 2.
Experimental Setup. The subject's torso and thigh were fixed by straps, the thigh was also secured by a cushioned wooden board placed above the edge of the chair. The servomotor applied stochastic angular position perturbations. A load cell between the crank arm and the cast was used to measure knee torque.

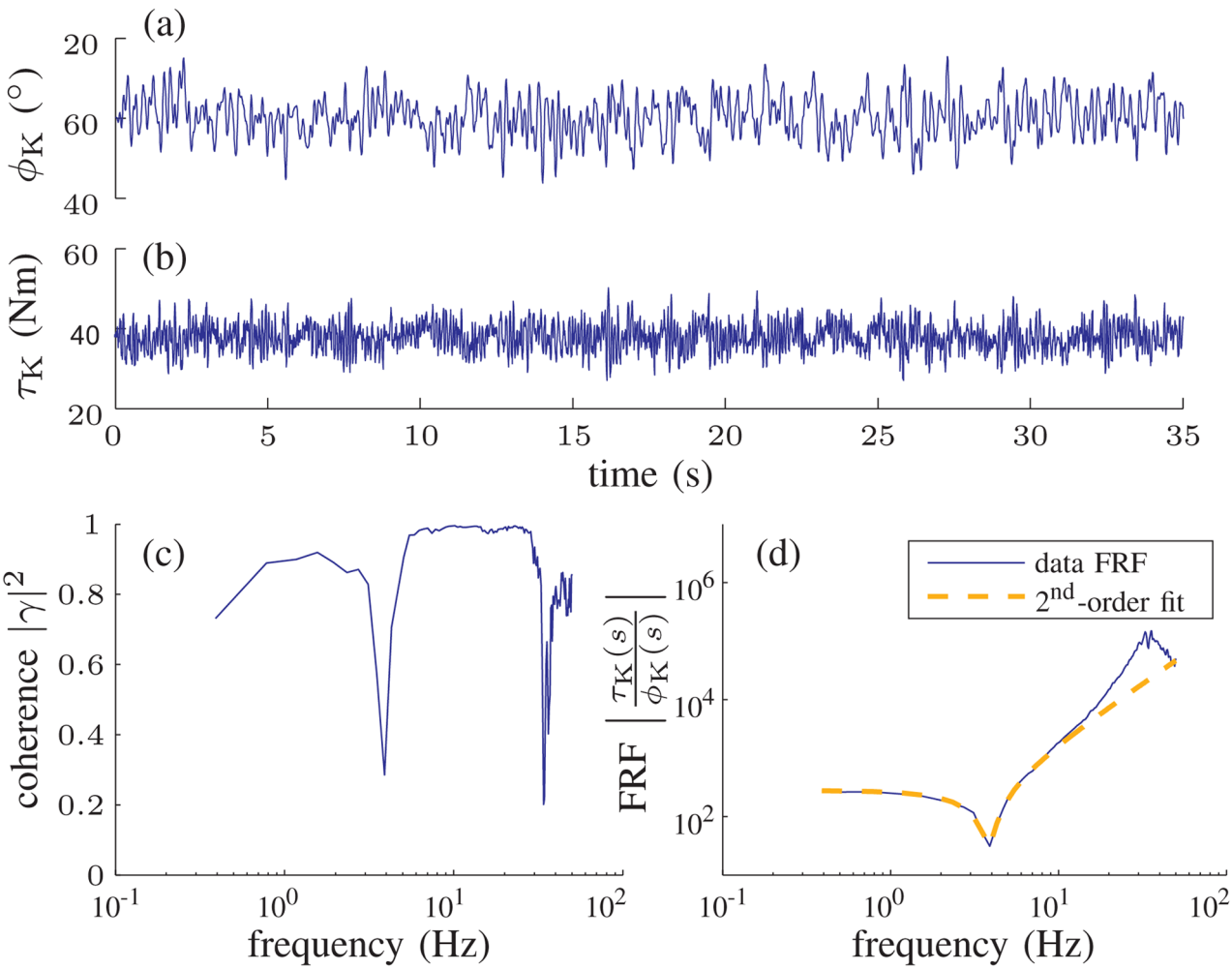

Fig. 3.

Data from a typical trial: position φ_K (a), torque τ_K (b), corresponding coherence (c) and frequency response function (FRF) and its 2nd-order fit (d). For this trial, the VAF was 86 %.

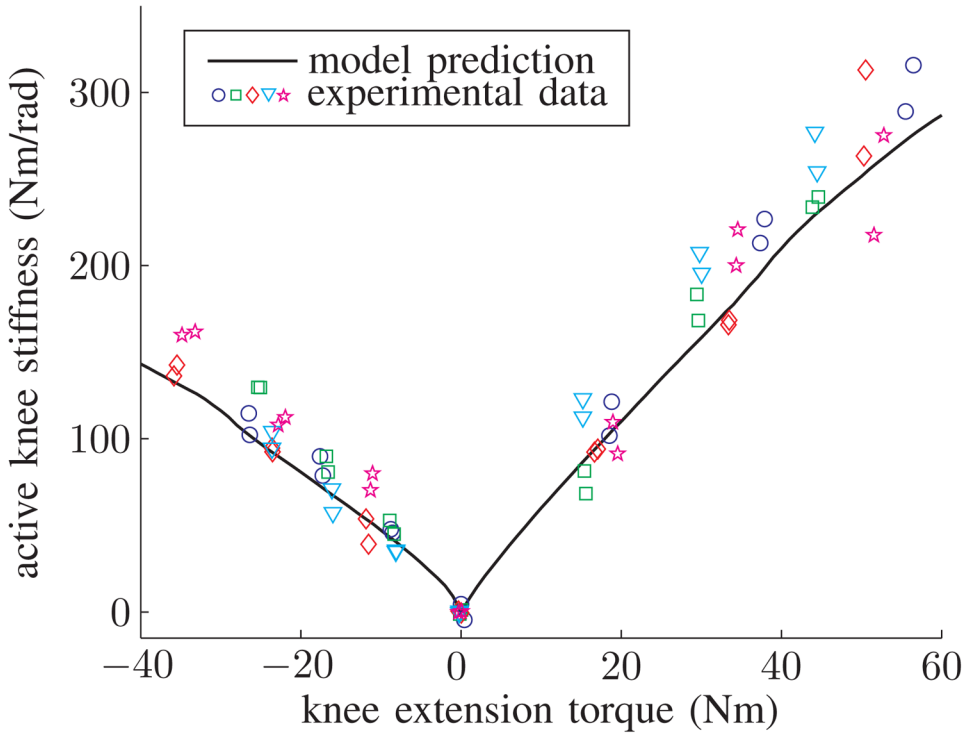

Fig. 4.
Torque-based model predictions and active stiffness estimated during perturbation experiments for 5 subjects, without co-contraction. The different symbols represent the different subjects.

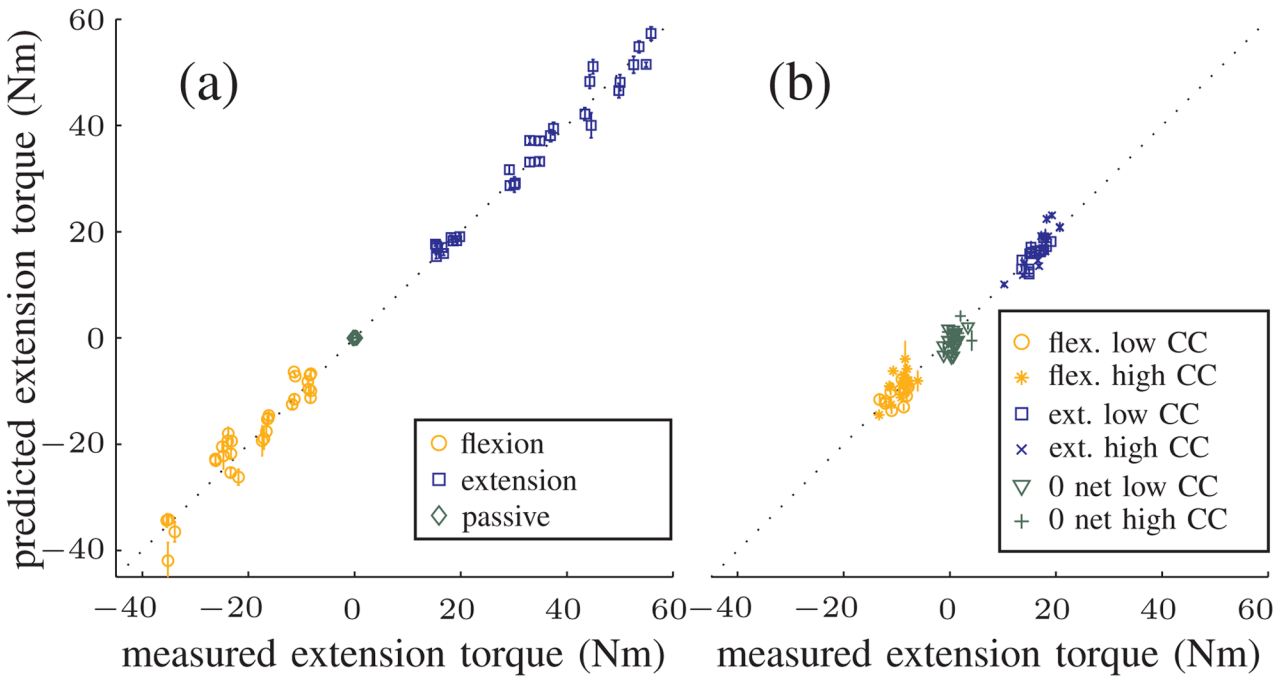

Fig. 5.

Torque prediction accuracy based on EMG for conditions without target co-contraction (a) and with co-contraction (b). The dashed line in both figures represents the unity line. In (a), different symbols are used for flexion, extension and passive trials. In (b), different symbols are used for each experimental condition that involved co-contraction (CC).

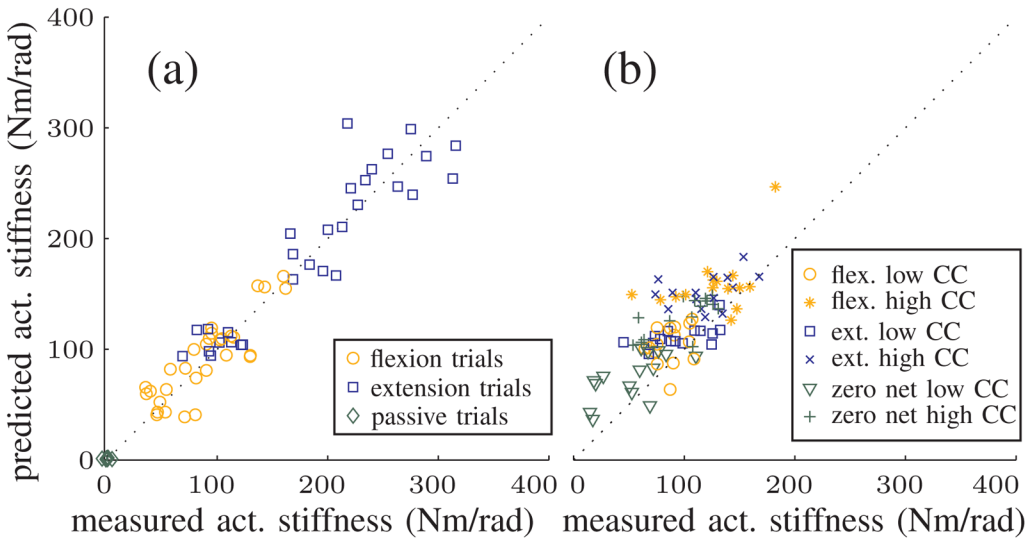

Fig. 6.

Active stiffness prediction accuracy based on EMG for conditions without (a) and with (b) target co-contraction (CC). The dashed line in both figures represents the unity line. Each data point represents a trial.

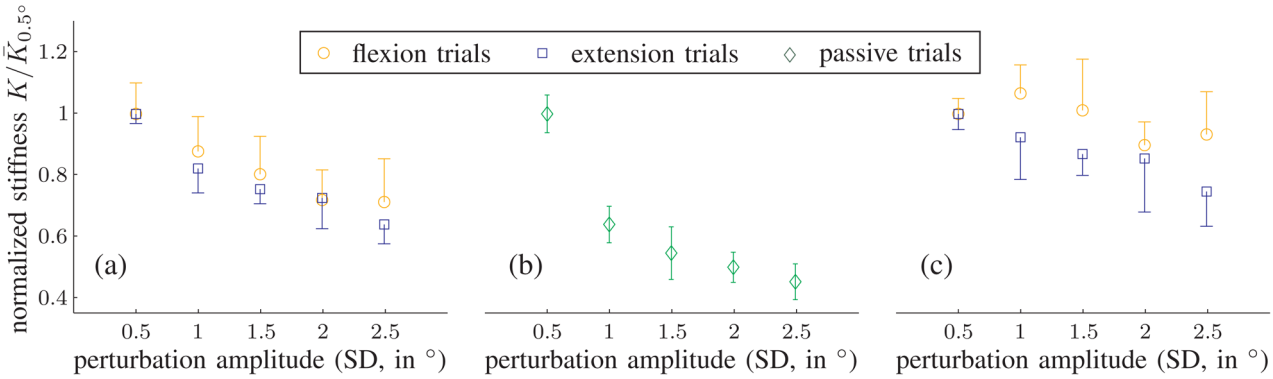

Fig. 7.

Dependence of identified stiffness on perturbation amplitude. For each trial, the total stiffness was normalized by the average stiffness of the respective condition (passive, flexion torque, extension torque) identified at the lowest amplitude ($\bar{K}_{0.5°}$), to illustrate the changes relative to the lowest amplitude. We show the mean values and SD over all subjects and over the following trials: all active trials (a); all passive trials (b); and the difference between the stiffness in the active trials and the passive trials at the same amplitude, normalized by this difference at the lowest amplitude (c), which illustrates the change in active stiffness K_a only.

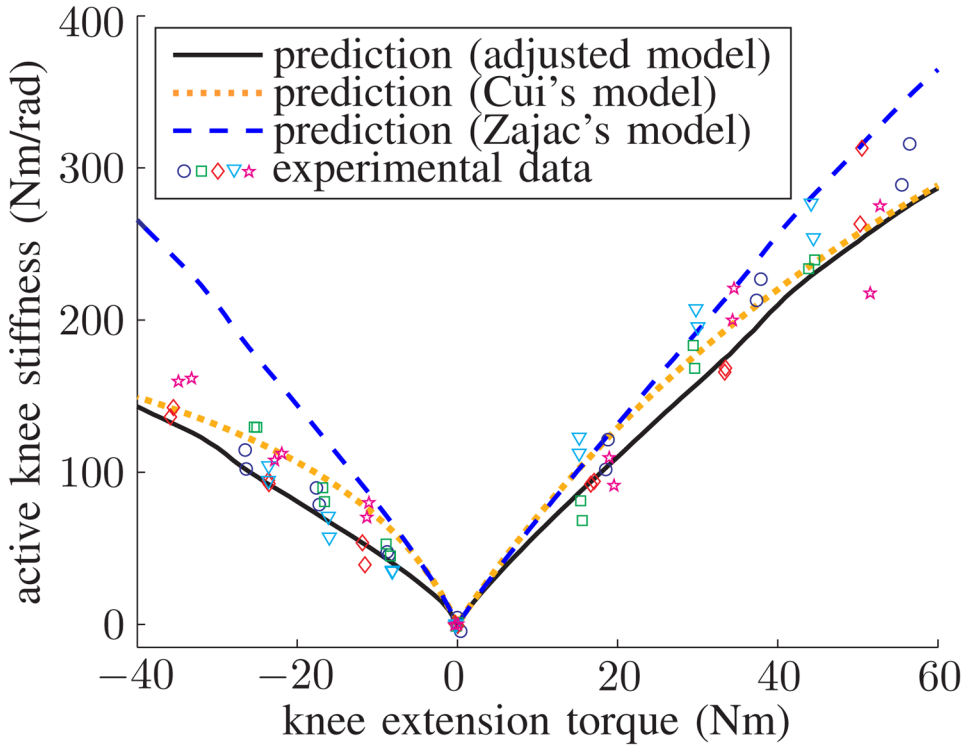

Fig. 8.

Torque-based predictions of active knee stiffness using different tendon models described in the text. The adjusted tendon model (solid black line) is a combination of Zajac's and Cui's model, incorporating a toe region for low forces, as used by Zajac, and a stiffness in the linear region that corresponds to Cui's model. The experimental data are the same as in Fig. 4.

TABLE I

Muscles used in the knee stiffness estimation process.

muscles	abbreviation	function	EMG recorded
Rectus femoris	RF	extension	yes
Vastus medialis	VM		yes
Vastus lateralis	VL		yes
Vastus intermedius	VI		no
Biceps femoris long head	BFL	flexion	yes
Biceps femoris short head	BFS		no
Semitendinosus	ST		yes
Semimembranosus	SM		no
Gracilis	GR		no
Sartorius	SR		no
Gastrocnemius medialis	GM		yes
Gastrocnemius lateralis	GL		yes