Knee stiffness estimation in physiological gait

Serge Pfeifer[1], Robert Riener[1] and Heike Vallery[1,2]

Abstract— **During physiological gait, humans continuously modulate their knee stiffness, depending on the demands of the activity and the terrain. A similar functionality could be provided by modern actuators in transfemoral prosthesis. However, quantitative data on how knee stiffness is modulated during physiological gait is still missing. This is likely due to the experimental difficulties associated with identifying knee stiffness by applying perturbations during gait. It is our goal to quantify such stiffness modulation during gait without the need to apply perturbations. Therefore, we have recently presented an approach to quantify knee stiffness from kinematic, kinetic and electromyographic (EMG) measurements, and have validated it in isometric conditions. The goal of this paper is to extend this approach to non-isometric conditions by combining inverse dynamics and EMG measurements, and to quantify physiological stiffness modulation in the example of level-ground walking. We show that stiffness varies substantially throughout a gait cycle, with a stiffness of around 100 Nm/rad during swing phase, and a peak of 450 Nm/rad in stance phase. These quantitative results may be beneficial for design and control of transfemoral prostheses and orthoses that aim to restore physiological function.**

I. Introduction

In physiological gait, varying muscle activations change not only the forces generated by the lower limbs, but also the limb stiffness. Humans can walk in a relaxed manner, or, if stability needs to be increased or impacts absorbed, joints can be stiffened by employing antagonistic muscles. Stiffening mainly happens subconsciously due to the activation-dependent changes in intrinsic muscle properties [1], [2] and due to changes in reflexes [3]. Amputees using lower-limb prostheses do not have such abilities, and the lack of stiffness modulation may be part of the reason why they have difficulties to walk across different terrain, and have a lower balance confidence in general [4]. Current powered prostheses allow a modulation of the apparent stiffness [5], [6], and may allow the versatility of unimpaired gait to be at least partly restored by prosthetic devices.

Following the assumption that knowledge of physiological impedance in the lower limbs could facilitate control design for prostheses, several research groups have recently started working on quantifying physiological joint stiffness during gait using special apparatus that apply perturbations [7], [8]. These efforts focused on the ankle joint so far, and quantitative descriptions of how knee joint stiffness is modulated are still missing. Therefore, it is unknown in how far stiffness modulation in current prostheses, which is usually found by manual tuning or heuristic strategies [9], [10], follow physiological knee stiffness profiles. It should be noted that physiological stiffness is defined as differential changes in joint moment in response to differential changes in joint position. It must not be confused with the *quasi-stiffness*, which describes the apparent covariation of joint moment and joint angle [11].

Recently, we presented a model-based method to estimate physiological knee stiffness only from conventional kinematic, kinetic, and electromyographic (EMG) recordings, and have validated it in isometric contractions [12]. This method lays the foundation to estimate stiffness profiles during diverse activities, for example level-ground gait, stair ascent, or stair descent, without applying perturbations. In this paper, we show how this method can be extended and applied to such activities in the example of level-ground walking.

II. Experimental Setup and Protocol

To estimate knee joint stiffness in physiological gait, a gait analysis with an able-bodied subject (age 29, height 1.84 m, weight 72 kg) was performed. The subject walked on even ground equipped with three force plates (9260AA6, Kistler Holding AG, Winterthur, Switzerland) at a speed of 3.5 km/h. For kinematic measurements an 8-camera Vicon optical tracking system (Vicon Motion Systems Ltd., Oxford, United Kingdom), and processing described by Wolf et al. [13] was used. The marker setup closely follow the setup described in [13], except for the feet, which used less markers because the subject was wearing sneakers and we did not evaluate angles of the metatarsophalangeal joint. Joint moments were obtained using inverse dynamics as described by Vaughn et al. [14].

During physiological gait, the amount of co-contraction is substantial [15], [16] and accurate estimation of the muscle forces from moment measurements is not practical. Therefore, an EMG-guided approach was used to estimate muscle forces. Transcutaneous EMG of seven easily accessible muscles (rectus femoris (rf), vastus lateralis (vl), vastus medialis (vm), semitendinosus (st), biceps femoris long head (blh), gastrocnemius medialis (gm), gastrocnemius lateralis (gl)) was recorded. EMGs were collected using a Noraxon TeleMyo 2400R system (Noraxon USA Inc., Scottsdale, AZ, USA), sampled at 3000 Hz after analog low-pass filters (8th

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[1]S. Pfeifer and R. Riener are with the Sensory-Motor Systems Lab, ETH Zurich and Medical Faculty, University of Zurich, Switzerland pfeifers at ethz.ch, riener at hest.ethz.ch

[2]P. H. Vallery is with the Department of BioMechanical Engineering, Delft University of Technology, 2628 CD Delft, The Netherlands and the Sensory-Motor Systems Lab, ETH Zurich h.vallery at tudelft.nl

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order Bessel) with a cut-off frequency of 1000 Hz. The recorded EMG was rectified, and the envelope was extracted using smoothing with a 4th order Butterworth filter with a cut-off frequency of 6 Hz. The signals were normalized to values obtained during maximum voluntary contractions (MVC), yielding the EMG-based estimate of muscle activity a_{EMG}. The analysis was done for 30 gait cycles.

The gait analysis was preceded by calibration movements necessary to determine functional joint axes and centers [13], and by MVC trials for all muscle groups for which EMG-measurements were involved.

III. Stiffness Estimation During Gait

A. Estimation of Muscle Forces

We used the musculoskeletal lower-limb model by Arnold et al. [17] analog to our approach described in [12]. Because muscle activations and moment arms vary heavily during gait, we used an approach that takes these variables into account. Of the 12 muscles present in the model, seven were measured using EMG as described above. Examplary profiles are depicted in Fig. 1. The remaining five muscles in the model spanning the knee joint, which are less easy to access, were estimated in analogy to Barrett et al. [18]. These muscles were: vastus intermedius (vi), the biceps femoris short head (bsh), the semimembranosus (sm), the gracilis (gr) and the sartorius (sr). The respective activations a were:

$$a_{\mathrm{vi}} = 0.5 \cdot (a_{\mathrm{vm}} + a_{\mathrm{vl}}), \quad (1)$$

$$a_{\mathrm{bsh}} = a_{\mathrm{blh}}, \quad (2)$$

$$a_{\mathrm{sm}} = a_{\mathrm{st}}, \quad (3)$$

$$a_{\mathrm{gr}} = 0.5 \cdot (a_{\mathrm{blh}} + a_{\mathrm{st}}), \quad (4)$$

$$a_{\mathrm{sr}} = 0.5 \cdot (a_{\mathrm{blh}} + a_{\mathrm{st}}). \quad (5)$$

A delay of 40 ms accounted for the delay between EMG signal and muscle force [19]. These estimates for each muscle i were multiplied by $f^0_{\mathrm{M},i} \cdot \cos\alpha_i$ to obtain the muscle force; $f^0_{\mathrm{M},i}$ is the muscle's maximum isometric force and α_i is its pennation angle (values from the literature [20]).

The estimated muscle forces contain inaccuracies, stemming from the noisy and highly variable nature of EMG signals, and from the mapping to the five muscles that were not measured. These inaccuracies were already apparent in a previous study, where a discrepancy between EMG-guided moment estimation and moment determined using inverse dynamics was observed, leading to a discrepancy in stiffness estimates [22]. To correct for these inaccuracies, we now correct the resulting flexion and extension moments by a method similar to Cholewicki and McGill [21]. It adjusts the individual muscle forces as much as needed such that they produce the measured moment (as determined with inverse dynamics). Because we observed in an earlier study that distribution among synergistic muscles does not have a substantial influence on resulting stiffness estimates [22], we summarized the individual muscle contributions to extensor moment contribution τ_{ext} and flexor moment contribution τ_{flex}. These were then modified by minimizing

$$J = (1 - c_{\mathrm{ext}}[k])^2 + (1 - c_{\mathrm{flex}}[k])^2 \quad (6)$$

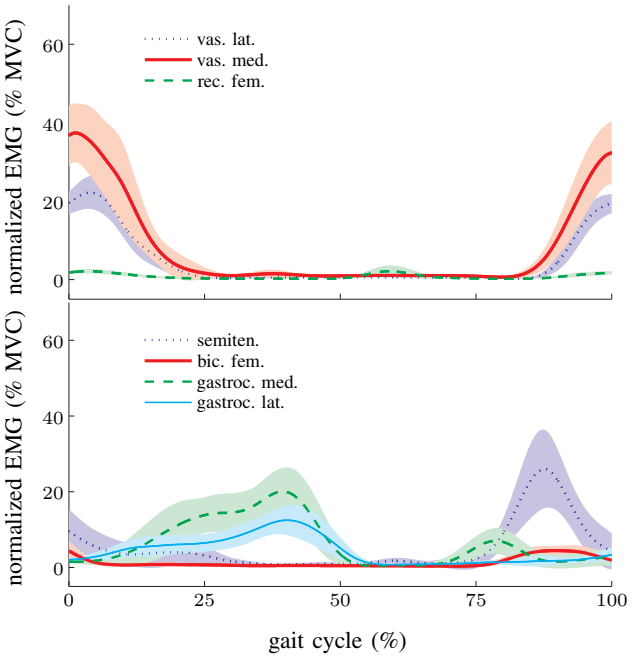

Fig. 1. EMG profiles in level-ground walking for extensor muscles (top) and flexor muscles (bottom). Mean and standard deviation over 30 gait cycles are shown.

with

$$c_i[k] = \begin{cases} g_i[k], & g_i[k] \geq 1 \\ 1/g_i[k], & g_i[k] < 1 \end{cases} \quad i = \mathrm{ext}, \mathrm{flex} \quad (7)$$

subject to the constraint

$$\tau_{\mathrm{meas}}[k] = g_{\mathrm{ext}}[k]\tau_{\mathrm{ext}}[k] + g_{\mathrm{flex}}[k]\tau_{\mathrm{flex}}[k], \quad (8)$$

where $\tau_{\mathrm{meas}}[k]$ represents the moment measured using inverse dynamics, and $g_{\mathrm{ext}}[k]$ and $g_{\mathrm{flex}}[k]$ represent the correction factors at each sample time k. This increases $\tau_{\mathrm{ext}}[k]$ and decreases $\tau_{\mathrm{flex}}[k]$ (or vice versa) as little as possible but as much as needed such that the moment as determined using inverse dynamics is exactly reproduced. Exemplary net moment measured using inverse dynamics during level-ground walking and estimated extension and flexion moments before and after adjustment with $g_{\mathrm{ext}}[k], g_{\mathrm{flex}}[k]$ are shown in Fig. 2.

B. Stiffness Estimation

In a next step, the flexion moment was distributed among the flexor muscles, and the extension moment among the extensor muscles. Using these estimated muscle forces, active joint stiffness was calculated (analog to [12]).

This active stiffness only takes into account stiffness due to muscle activation. To account for passive stiffness due to soft tissue and ligaments, we extracted average values reported by Zhang et al. [23] at different knee flexion angles (0 °, 30 °, 60 ° and 90 °) and interpolated for values in between. Resulting passive stiffness was relatively constant around 35-45 Nm/rad (Fig. 3). The sum of active and passive stiffness represents the total joint stiffness.

The estimated active knee stiffness reached the maximum (around 450 Nm/rad) shortly after heel-strike, and

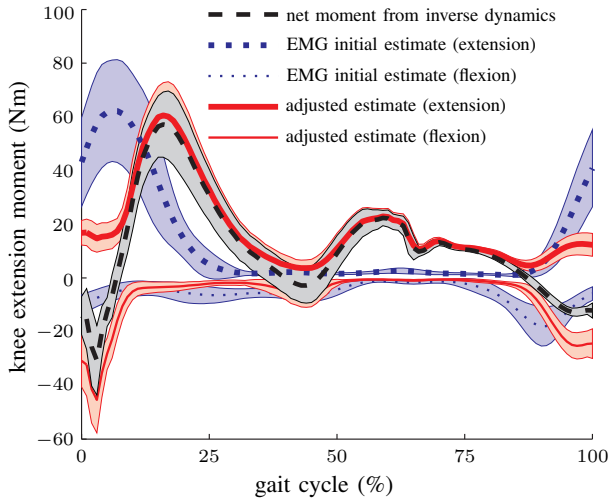

Fig. 2. Inverse dynamics moment and estimated extension and flexion moments estimated from EMG. Dashed blue lines are the initial estimates before adjustment with (6) and solid red lines are after adjustment. Mean and SD over 30 gait cycles are depicted.

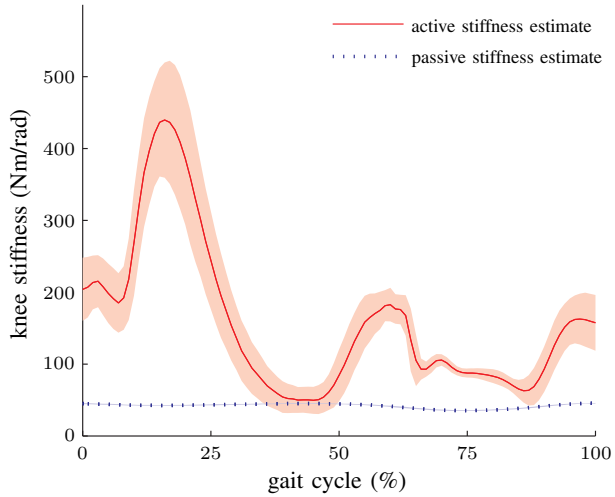

Fig. 3. Stiffness estimated assuming no co-contraction and using the EMG-/guided approach. Mean and SD over 30 gait cycles are depicted.

comparably low stiffness could be observed during swing phase (Fig. 3). Compared to the active stiffness, the passive stiffness estimated using a regression model based on Zhang et al. [23] was negligible in large portions of the gait cycle and almost constant over a gait cycle (Fig. 3).

IV. Discussion

In this paper, we have presented a method to estimate activation-dependent knee stiffness from standard gait lab data (kinematics, kinetics, EMG). EMG-measurements and inverse dynamics are combined to obtain estimates of co-contraction, which is necessary to accurately estimate stiffness. We applied the method in the example of level-ground walking. The results may be useful for future hardware and control design of prostheses and orthoses.

Stiffness was estimated during gait for only one subject, but we believe that estimated stiffness during gait is still representative, because the underlying measurements agreed well with data from the literature: EMG signals were similar to other data [16], [24] and joint moments determined using inverse dynamics as well [25], [26].

Our approach to estimate physiological stiffness has so far been validated by comparison to isometric perturbation experiments [12]. Model-based stiffness estimates could need adjustments, if isometric findings cannot be directly transferred in this form to movements. It has not yet been validated during gait, because it is difficult to apply perturbations to the joint without impeding natural gait. It is well known that reflexes play an important role in physiological gait [33], [34] and for a comprehensive description of knee behavior in response to perturbations, it may be necessary to consider such mechanisms separately. It should also be noted that the method to estimate muscle forces presented here contains potential sources of inaccuracies that have not yet been quantified (i.e. the mapping to unmeasured EMG muscles [18] and the correction of flexion and extension moments similar to [21]). Literature suggests that stiffness during movement could be lower than what would be expected from observations in the static case, for example, in the elbow joint [27]. It has also been observed that joint stiffness decreases during movement onset [28], [29]. Future perturbation experiments during gait (for example with the device by Tucker et al. [30]) will show how well our approach estimates stiffness in locomotor activities. Nevertheless, our method presents the first attempt to quantify activation-dependent knee joint stiffness during gait.

These estimates can be used for the design and control of transfemoral prostheses, for example by designing impedance controllers replicating such stiffness profiles. However, other factors may have to be considered when doing so. Previous studies (our own and research by other groups) have shown that experimentally measured knee joint stiffness decreases with perturbation amplitude [31], [32], [12]. It is possible that stiffness in response to larger excursions better reflects joint behavior during gait, and other contributors to joint behavior may have to be considered in more detail. Human motor control is commonly thought of consisting of several layers that are tightly interlinked: cerebral control, spinal afferent feedback loops (also called reflexes), and intrinsic muscle stiffness [35]. Cerebral control may possibly be replaced by the intention estimation of a prosthesis. However, additional *reflexive* components to stiffness may have to be considered when physiological stiffness estimates are used to mimic physiological behavior in a prosthesis.

It is also possible that physiological stiffness, even if it could be determined perfectly, should not directly be copied in a prosthetic device. Controlling a prosthesis is very different from controlling a physiological knee that is tightly integrated into the human sensory-motor loop. First, the mechanical attachment differs greatly, and movement of the shaft with respect to the residual limb may influence stiffness required at the joint. There is also only limited sensory feedback from the prosthetic leg and actuator, in contrast

to a physiological joint where constant afferent feedback is available; this might influence the stiffness necessary in a prosthetic joint.

V. Conclusion

We have presented a method to estimate knee joint stiffness during gait by combining inverse dynamics and EMG measurements, and using our recently developed model-based estimation approach [12]. While there is still work to be done, it provides first quantitative estimates of knee stiffness during gait. These estimates are important to understand the contribution of such stiffness mechanics to physiological gait, and may help design and control of prostheses and orthoses in the future.

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