Identification of Ankle Joint Stiffness During Passive Movements - A Subspace Linear Parameter Varying Approach

Ehsan Sobhani Tehrani, Kian Jalaleddini and Robert E. Kearney

Abstract— **This paper describes a novel method for the identification of time-varying ankle joint dynamic stiffness during large passive movements. The method estimates a linear parameter varying parallel-cascade (LPV-PC) model of joint stiffness consisting of two pathways: (a) an LPV impulse response function (IRF) for intrinsic mechanics and (b) an LPV Hammerstein cascade with time-varying static nonlinearity and a time-invariant linear dynamics for the reflex pathway. A subspace identification technique is used to estimate a state-space representation of the reflex stiffness dynamics. Then, an orthogonal projection decouples intrinsic from reflex response and subsequently identifies an LPV-IRF model of intrinsic stiffness. Finally, an LPV model of the reflex static nonlinearity is estimated using an iterative, separable least squares method. The LPV method was validated using experimental data from two healthy subjects where the ankle was moved passively by an actuator through its range of motion first *without* and then with perturbations. The identification results demonstrated that (a) the dynamic response of the intrisic pathway changes systematically with joint position; and (b) the static nonlinearity of the reflex pathway resembles a half-wave rectifier whose threshold decreases and gain increases as ankle is moved to dorsiflexed position.**

I. Introduction

Ankle joint mechanics can be described by the dynamic relationship between joint position and the torque acting about it, i.e. dynamic joint stiffness. Two distinct physiological mechanisms determine joint stiffness: (i) Limb inertia, viscoelasticity of muscle-tendon complex, and active properties of muscle contraction that together define *intrinsic* stiffness; and (ii) The stretch reflex feedback that changes muscle activation in response to muscle lengthening leading to *reflex* stiffness. Therefore, modeling joint stiffness and estimating its two components is critical for understanding human motor control and has far-reaching implications for design and control of active prostheses and orthotic devices.

However, measuring joint stiffness during movement is difficult because it is modulated strongly by joint position and neural activation, and is therefore time-varying (TV). Many studies have quantified these changes for quasi-stationary conditions, where the joint is perturbed around a position operating point (OP) while subjects remain relaxed or maintain a constant activation. Trials are repeated at different activation levels and position OPs and the changes in the time-invariant (TI) stiffness model parameters monitored. These approaches estimate a valid *local* model at each quasi-stationary condition but these local models cannot be simply interpolated to represent the *global* TV behvaior during movement. Nontheless, local models provide a model structure that can be utilized as the basis for TV identification. Specifically, the reflex stiffness is modeled by a Hammerstein system; i.e., a cascade of a static nonlinearity and a linear dynamic element [1], [2]. The intrinsic stiffness is modeled as either a non-parametric impulse response function (IRF) [1] or a second-order *mass-spring-damper* (IBK) model [3].

Quasi-stationary studies by Mirbagheri *et al.* [2], and more recently, Guarin *et al.* [3] showed that the intrisic elasticity and reflex gain were highly modulated with the position OP whereas damping and natural frequency of the reflex dynamics remained almost constant over the ROM. Furthermore, Jalaleddini and Kearney [4] showed that the reflex threshold changed with position. These results suggest that the TV reflex stiffness under passive condition (i.e., at rest) can be represented by a Hammerstein system with a TV static nonlinearity and TI linear dynamics.

Linear parameter varying (LPV) models have a structure resembling that of a linear system but parameters that change as a function of a scheduling variable (SV) [5], [6]. Recently, Sobhani *et al.* [7], [8] used LPV identification to model TV intrinsic stiffness and the reflex EMG response as having a functional dependence on ankle position. This makes it possible to predict the response to novel movement trajectories. This *cannot* be achieved using ensemble-based TV methods [9], [10].

This paper combines and extends the LPV methodologies developed for TV intrisic stiffness [7] and Hammerstein systems [8], and uses it to estimate total joint stiffness during passive movements. The new algorithm identifies an LPV Parallel-Cascade (LPV-PC) model of joint stiffness that comprises a LPV-IRF model for the intrinsic and an LPV Hammerstein model for the reflex pathway, using joint position as the SV. An LPV-IRF model structure is used since we have observed that the intrinsic stiffness is more complex than a second order LPV-IBK model.

II. Theory [1]

A. Problem Formulation

The objective is to identify the LPV-PC model of ankle dynamic stiffness depicted in Fig. 1 using N samples of input position, $pos(k)$, output torque, $tq(k)$, and SV, $\rho(k)$.

E. Sobhani Tehrani, K. Jalaleddini, and R. E. Kearney are with the Department of Biomedical Engineering, McGill University, 3775 University, Montréal, Québec H3A 2B4, Canada. {ehsan.sobhani,seyed.jalaleddini}@mail.mcgill.ca. and robert.kearney@mcgill.ca

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[1] In this paper, matrices, vectors, and scalars are denoted by bold-face uppercase, uppercase and lowercase letters, respectively.

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Internal signals that cannot be measured include: the reflex nonlinearity output,$z(k)$, intrisic torque,$tq_i(k)$, and reflex torque, $tq_r(k)$. It is assumed that there is no voluntary contraction and $\nu(k)$ is an arbitrary colored noise signal that is uncorrelated with the $pos(k)$:

$$\tilde{tq}(k) = tq_i(k) + tq_r(k) + \nu(k) \tag{1}$$

Define vectors comprising all sampled data:

$$\begin{aligned} \boldsymbol{T\tilde{Q}} &= \left[\begin{array}{cccc} \tilde{tq}(0) & \cdots & \tilde{tq}(N-1) \end{array}\right]^T \\ \boldsymbol{TQ_i} &= \left[\begin{array}{cccc} tq_i(0) & \cdots & tq_i(N-1) \end{array}\right]^T \\ \boldsymbol{TQ_r} &= \left[\begin{array}{cccc} tq_r(0) & \cdots & tq_r(N-1) \end{array}\right]^T \\ \boldsymbol{V} &= \left[\begin{array}{cccc} v(0) & \cdots & v(N-1) \end{array}\right]^T \end{aligned} \tag{2}$$

Represent intrinsic stiffness by an LPV-IRF model:

$$tq_i(k) = \sum_{l=-L}^{+L} h_l\left(\rho(k)\right) pos(k-l) \tag{3}$$

with the IRF weights given by a basis expansion of $\rho(k)$:

$$h_l \triangleq \sum_{j=1}^{n_i} h_{lj} g_j\left(\rho(k)\right) \tag{4}$$

where h_{ij} is the $(i,j)^{\text{th}}$ coefficient for the i^{th} lag of IRF, h_i; $g_j\left(\rho(k)\right)$ is the j^{th} basis expansion of the SV; and n_i is the expansion order.

Rewrite (3) using matrices to obtain a data equation for the intrinsic pathway. Define a tall vector containing the LPV-IRF weights for lag l:

$$\boldsymbol{H_l} = \left[\begin{array}{cccc} h_{l1} & \cdots & h_{ln_i} \end{array}\right]^T \tag{5}$$

and $\boldsymbol{\theta_i}$ that stacks H_l for all lags:

$$\boldsymbol{\theta_i} = \left[\begin{array}{ccccc} \boldsymbol{H_{-L}} & \cdots & \boldsymbol{H_0} & \cdots & \boldsymbol{H_{+L}} \end{array}\right]^T \tag{6}$$

Define vectors for the basis expansion of SV:

$$\boldsymbol{G_i(k)} = \left[\begin{array}{cccc} g_1\left(\rho(k)\right) & \cdots & g_{n_i}\left(\rho(k)\right) \end{array}\right]^T \tag{7}$$

and lagged samples of position:

$$P(k) = \left[\begin{array}{ccccccc} pos(k+l) & \cdots & pos(k) & \cdots & pos(k-l) \end{array}\right] \tag{8}$$

Then, the input to the intrinsic pathway will be the Kronecker product of (8) and (7):

$$U_i(k) = P(k) \otimes G_i(k) \tag{9}$$

so that the intrinsic torque defined by (2) and (3) is:

$$\boldsymbol{TQ_i} = \Psi_i \boldsymbol{\theta_i} \tag{10}$$

$$\text{where :} \qquad \Psi_{\text{i}} = \left[\begin{array}{cccc} U_i(0) & \cdots & U_i(N-1) \end{array}\right]^{\text{T}} \tag{11}$$

Reflex stiffness is modeled as an LPV static nonlinearity cascaded with an LTI linear system. The output of the reflex nonlinearity is:

$$z(k) = f\left(dvel(k), \rho(k)\right) \simeq \sum_{i=1}^{n} \omega_i\left(\rho(k)\right) g_i\left(dvel(k)\right) \tag{12}$$

where $dvel(k)$ is the ankle velocity delayed by reflex delay and ω_i is a basis expansion on SV:

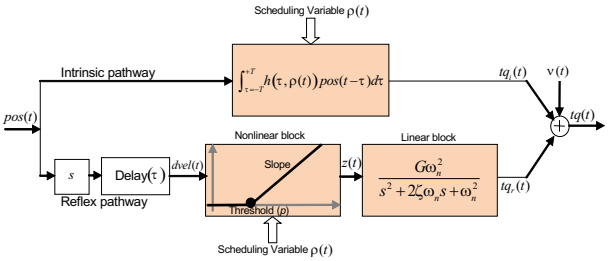

Fig. 1. The schematic of the LPV-PC model of ankle stiffness.

$$\omega_i = \sum_{j=1}^{n_r} \omega_{ij} g_j\left(\rho(k)\right) \tag{13}$$

where ω_{ij} is the coefficient of the product of the i^{th} basis expansion of the delayed velocity, $g_i\left(dvel(k)\right)$, with the j^{th} basis expansion of the SV, $g_j\left(\rho(k)\right)$. Define $G_r(k)$ as the basis expansion of SV with order n_r and $DV(k)$ as the basis expansion of delayed velocity with order n:

$$\begin{aligned} \boldsymbol{G_r(k)} &= \left[\begin{array}{cccc} g_1\left(\rho(k)\right) & \cdots & g_{n_r}\left(\rho(k)\right) \end{array}\right]^T \\ DV(k) &= \left[\begin{array}{cccc} g_1\left(dvel(k)\right) & \cdots & g_n\left(dvel(k)\right) \end{array}\right] \end{aligned} \tag{14}$$

Represent the LTI component of the reflex pathway by the state-space model:

$$\begin{cases} \boldsymbol{X}(k+1) & = A\boldsymbol{X}(k) + \boldsymbol{B}z(k) \\ tq_r(k) & = C\boldsymbol{X}(k) + Dz(k) \end{cases} \tag{15}$$

where, $\boldsymbol{X}(k)$ is a $m \times 1$ state vector; and $A_{m\times m}$, $\boldsymbol{B}_{m\times 1}$, $C_{1\times m}$ and $D_{1\times 1}$ are the state-space matrices:

$$\boldsymbol{B} = [b_1, \cdots, b_m]^T; \quad \text{D} = [\text{d}] \tag{16}$$

Define:

$$\begin{aligned} \boldsymbol{\Omega_i} &= [\omega_{i1}, \cdots, \omega_{in_r}]^T \\ \boldsymbol{\Omega} &= [\boldsymbol{\Omega_1} \cdots \boldsymbol{\Omega_n}]^T \\ \boldsymbol{U_r}(k) &= DV(k) \otimes G_r(k) \end{aligned} \tag{17}$$

Substitute (17) in (15) to yield:

$$\begin{cases} \boldsymbol{X}(k+1) & = A_r\boldsymbol{X}(k) + B_\Omega \boldsymbol{U_r}(k) \\ tq_r(k) & = C_r\boldsymbol{X}(k) + D_\Omega \boldsymbol{U_r}(k) \end{cases} \tag{18}$$

where:

$$B_\Omega = \boldsymbol{B} \otimes \boldsymbol{\Omega} = \left[\begin{array}{ccc} b_1\Omega_1^T & \cdots & b_1\Omega_n^T \\ \vdots & \ddots & \vdots \\ b_m\Omega_1^T & \cdots & b_m\Omega_n^T \end{array}\right] \tag{19}$$

$$D_\Omega = D \otimes \boldsymbol{\Omega} = \left[\begin{array}{ccc} d\Omega_1^T & \cdots & d\Omega_n^T \end{array}\right] \tag{20}$$

Combine the intrinsic (3) and reflex (18) models to form a multi-input-single-output (MISO) state-space representation:

$$\begin{cases} \boldsymbol{X}(k+1) & = A_r\boldsymbol{X}(k) + B_{tot}\boldsymbol{U_{tot}}(k) \\ \tilde{tq}(k) & = C_r\boldsymbol{X}(k) + D_{tot}\boldsymbol{U_{tot}}(k) + v(k) \end{cases} \tag{21}$$

where:

$$U_{tot}(k) = \left[\begin{array}{cc} U_r(k) & U_i(k) \end{array}\right] \tag{22}$$

$$\begin{aligned} B_{tot} &= \left[\begin{array}{cc} B_\Omega & \underbrace{0 \cdots 0}_{(2L+1)n_i \text{ columns}} \end{array}\right] \\ D_{tot} &= \left[\begin{array}{cc} D_\Omega & \theta_i \end{array}\right] \end{aligned} \tag{23}$$

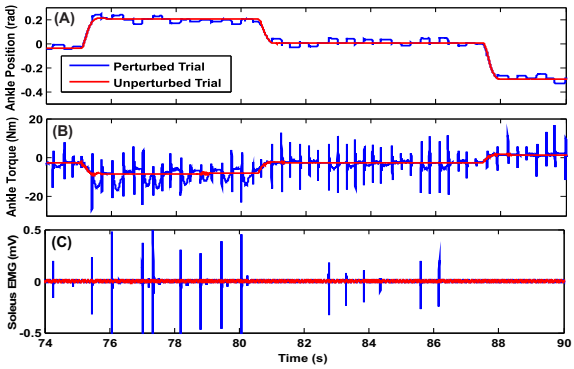

Fig. 2. Experimental signals from subject S1 during a PT and a UT passive movement trial: (A) ankle position, (B) torque, and (C) Soleus EMG.

B. Identification Algorithm

The objective is to identify (i) the parameters of the intrinsic LPV-IRF, $\boldsymbol{\theta_i}$; (ii) the vector $\boldsymbol{\Omega}$ of the reflex nonlinearity; and (iii) the state-space matrices of the reflex LTI dynamics.

Use *multivariable output error state-space* (MOESP) subspace identification algorithms to estimate the state-space matrices $\hat{A}_r$ and $\hat{C}_r$ [11]. Then, form the data equation:

$$\tilde{\boldsymbol{TQ}} = \boldsymbol{TQ_i} + \boldsymbol{TQ_r} + \boldsymbol{V} = \Psi_i \boldsymbol{\theta_i} + \Psi_r \boldsymbol{\theta_r} + \boldsymbol{V} \tag{24}$$

where the reflex regressor, Ψ_r, and parameters, $\boldsymbol{\theta_r}$ are [8]:

$$\Psi_r = \begin{bmatrix} 0 & \boldsymbol{U_r}^T(0) \\ \vdots & \vdots \\ \sum_{\tau=0}^{N-2} \boldsymbol{U_r}^T(\tau) \otimes \hat{C}_r \hat{A}_r^{N-2-\tau} & \boldsymbol{U_r}^T(N-1) \end{bmatrix} \tag{25}$$

$$\boldsymbol{\theta_r} = \left[\boldsymbol{B}^T d\right]^T \otimes \boldsymbol{\Omega} \tag{26}$$

Use the *orthogonal projection* from [12] to decompose the intrinsic and reflex torques and estimate intrinsic stiffness parameters:

$$\hat{\boldsymbol{\theta}}_i = C^{\dagger} \Psi_i^{\dagger} \left(I - \Psi_r \Psi_r^{\dagger}\right) Y \tag{27}$$

where:

$$C = I - \Psi_i^{\dagger} \Psi_r \Psi_r^{\dagger} \Psi_i \tag{28}$$

Use $\hat{\boldsymbol{\theta}}_i$ to predict intrinsic torque using (10) and then estimate the reflex torque:

$$\hat{TQ}_r = \tilde{TQ} - \Psi_i \hat{\theta}_i = \Psi_r \theta_r \tag{29}$$

Use this with the subspace LPV Hammerstein identification method [8] to estimate $B = \{b_1, \cdots, b_m\}$, d and Ω.

III. Experimental Validation

The utility of the LPV-PC model and identification algorithm was examined for two healthy, male subjects.

A. Methods and Input Signals

Both subjects gave informed consent to the experimental procedures which had been approved by McGill University's *institutional review board*. Subjects lay supine with their the left foot firmly attached to the pedal of a hydro-electric actuator operating as a stiff position servo. Details of the experimental setup are described in [2]. Two types of trials were performed: (i) *Unperturbed* trials (UT) where the ankle was moved by the actuator through a multi-level trajectory that spanned ankle positions from -0.4rad (*plantarflexed*, PF) to +0.2rad (*dorsilflexed*, DF); and (ii) *Perturbed* trials (PT)

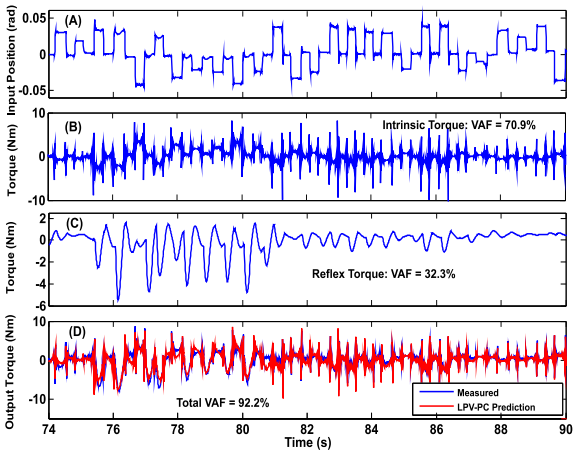

Fig. 3. Identification data and results for subject S1: (A) input: position perturbations computed as the difference between measured positions of a perturbed and an unperturbed trial, (B) the identified intrinsic torque, (C) the identified reflex torque, and (D) output: torque in response to perturbations - computed from measurements and predicted.

where small signal, piecewise constant PRALDS (pseudo random arbitrary level distributed signal) perturbations were added to the multi-level trajectory for identification. The multi-level trajectory was the same for both UT and PT trials. It was low-pass filtered to 2.5Hz with a second-order Butterworth filter to avoid sharp transitions from one level to another. The levels were selected randomly from a set having 10 levels. The ankle was maintained at each level for a duration selected from a uniform distribution with the range $[4, 7]$s. The peak to peak amplitude of PRALDS in PTs was ± 0.04rad with pulse durations selected from a uniform random process with the range $[250, 350]$ms. This range was selected to have a wide-band, persistently exciting, perturbation signal for identification and yet avoid reflex response suppression at high mean absolute velocities. Each trial lasted 2 minutes. Data was sampled at 1kHz and decimated to 100Hz for identification.

B. Results

Fig. 2 shows a typical segment of a UT trial (red) with the corresponding PT superimposed (blue). Soleus EMG is shown in Fig. 2C confirms that (a) there was no voluntary activation and (b) reflex responses were present only during PT and were modulated by joint position; decreasing as ankle was moved into plantarflexion.

The first step for the LPV-PC identification was to estimate the responses evoked by the small amplitude perturbations; to do this, position and torque records from the UT trial were subtracted from those of the corresponding PT. Fig. 3A shows the resulting position input; Fig. 3D shows the torque response (blue).

The LPV-PC model estimated from these data accurately predicted the torque; Fig. 3D shows that the predicted (red) and observed torques (blue) were very similar. Indeed, the *variance-accounted-for* (VAF) was greater than %92 for both subjects. The intrinsic (Fig. 3B) and reflex (Fig. 3C) responses predicted by the LPV model clearly vary with joint position. Moreover, changes in the reflex torque were consistent with those of the EMG response in Fig. 2C. We

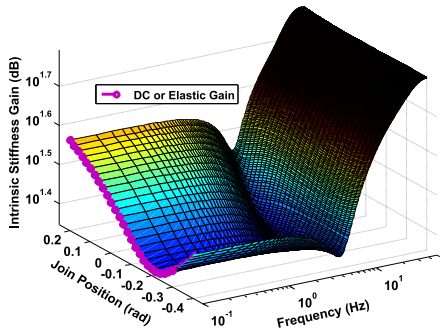

Fig. 4. The gain of the intrinsic stiffness frequency response computed from its estimated IRF as a function of joint position for subject S1. The low-frequency or *elastic* gain of intrinsic stiffness is also shown.

have planned to further validate the LPV model by simulating its response to other movement trajectories and compare the results with the corresponding experimental data.

Fig. 4 shows LPV-IRF model of intrinsic stiffness in terms of the gain of intrinsic stiffness frequency response computed from the IRF as a function of joint poisiton. Significant modulations with joint position are evident; the low-frequency gain increased towards DF and was minimum near the mid-point between Neutral and PF positions. Similar changes are evident in the location of the resonant frequency.

Fig. 5 shows the estimated LPV Hammerstein model of reflex pathway. Fig. 5A shows that the static nonlinearity has a strong uni-directional sensitivity to velocity and a slope (corresponding to the reflex gain) that increases from PF to DF ankle position. Fig. 5B shows that the estimated TI reflex IRF resembles a second-order low-pass system.

IV. Discussion

We presented a novel method for LPV identification of ankle joint stiffness during large passive movements. The method identifies an LPV-PC model of ankle stiffness consisting of an LPV-IRF model for intrinsic pathway, and a reflex pathway comprising of a TV nonlinearity and LTI dynamics. The LPV-PC method can characterize changes in intrinsic stiffness and reflex stiffness static parameters such as gain and threshold as a function of joint position. The application of the method was demonstrated by using it to follow the changes in stiffness dynamics accompanying large passive movements of the ankle in two healthy subjects.

The position dependent trends estimated in intrinsic and reflex parameters were similar to those observed in quasi-stationary identification studies at multiple position OPs [2]. The LPV approach provides this information much more efficiently than the OP approach. It uses *only* two trials (UT and PT) while the OP-based identification requires a large number of trials to estimate trends with a similar resolution.

This LPV-PC methodology has other important advantages: (i) It identifies the position-dependent nonlinear dynamics that cause TV behavior during movement. As such, it provides a continous-time, *global* model of TV joint mechanics instead of *local* models at each position OP, which *cannot* be interpolated to reproduce the global behavior. Therefore, the LPV-PC model can predict joint response to *novel* movement trajectories. (ii) LPV models are well-suited

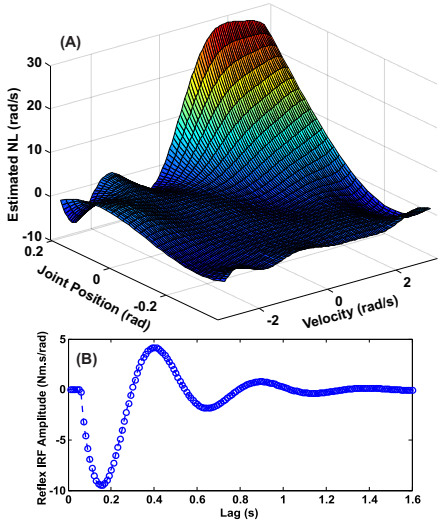

Fig. 5. The identified LPV Hammerstein model of the reflex pathway of subject S1: (A) estimated static nonlinearity as a function of joint velocity and position, and (B) identified IRF.

for designing *robust* controllers of prostheses and orthotic devices since the LPV control theory is well developed.

The new LPV approach can be applied to characterize biomechanics of movement during other functional tasks such as upright stance, and reaching and pointing. To identify stiffness during *active* movements where both position and activation level change, will require extending the identification methodology to LPV systems where (a) the SVs of both pathways are muscle activation in addition to position and (b) the reflex linear dynamics are also time-varying.

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