ISB 2013 BRAZIL
XXIV CONGRESS OF THE INTERNATIONAL SOCIETY OF BIOMECHANICS
XV BRAZILIAN CONGRESS OF BIOMECHANICS

SHORT- AND MEDIUM-LATENCY STRETCH REFLEXES STUDIED BY MEANS OF A BIOLOGICALLY BASED NEUROMUSCULAR MODEL

[1]Leonardo A. Elias and [1]André F. Kohn
[1]Biomedical Engineering Laboratory, Escola Politécnica, University of São Paulo, São Paulo 05508-900, Brazil
Email: leoabdala@leb.usp.br, Web: http://remoto.leb.usp.br/

SUMMARY
A biologically based neuromuscular simulator was used to produce short- (SLR) and medium-latency reflexes (MLR) observed after a stretch of the SO muscle. As expected, SLR was exclusively mediated by the activity of Ia afferents passing through a monosynaptic pathway. The MLR was generated by a summation of effects from Ia and type-II afferents, though it could be generated without the activity of type-II afferents. When the reflex was mediated exclusively by type-II afferents, only MLR was observed as found in patients with a loss of Ia afferents.

INTRODUCTION
At least two electromyographic (EMG) responses are frequently observed after a sudden stretch of the Triceps Surae evoked by a passive ankle rotation (dorsiflexion) [1-4]. The first, referred to as the SLR, has been mainly attributed to the activity of Ia muscle spindle afferents through a reflexive monosynaptic spinal pathway. However, the mechanisms underlying the MLR have been the focus of several studies in the literature [3-5]. Some authors stated that this response is due to the activity of type-II muscle spindle afferents, which have a slower axonal conduction velocity and pass through an excitatory di-synaptic pathway [3]. Conversely, others argued that the prolonged burst from Ia afferents caused by the stretch maintains the excitation of the motor neuron (MN) pool, which tends to discharge synchronously as a rebound effect [4, 5].

The aim of this computer simulation study was to evaluate the mechanisms behind the genesis of SLR and MLR by means of a biologically based neuromuscular model, encompassing MN and interneuron (IN) pools, Hill-type muscles, muscle spindles, and afferent fibers. The simulations will be used to test the following hypotheses: *i)* if SLR and MLR may be generated solely by the activity of Ia afferents; *ii)* if these reflexes may arise from the interplay between monosynaptic Ia and di-synaptic type-II activities; and *iii)* if the MLR might be generated exclusively by a di-synaptic type-II reflex pathway, mimicking the loss of Ia afferents during a neuromuscular pathology [3].

METHODS
Part of the neuromuscular model follows the structure previously described elsewhere [6, 7]. Briefly, each motor pool encompasses conductance-based MN (without persistent currents) and excitatory group-II IN models. MN soma and INs included three ionic channels (Na^+, fast K^+ and slow K^+), with simplified dynamics (see [7]). Three independent motor nuclei were used to represent the Soleus (SO), Medial Gastrocnemius, and Lateral Gastrocnemius. Independent descending commands drive spinal MNs to evoke a sustained basal activity. The activity of the descending axons was modeled as homogeneous Poisson point processes with a given mean rate. Figure 1 depicts the schematic diagram of a given motor pool, its innervated muscle, and the afferent fibers from spindles. The number of elements of the whole neuromuscular model follows the estimates provided by the experimental literature.

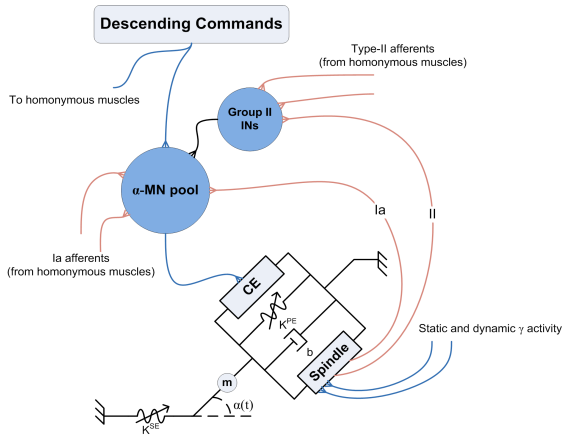

Figure 1: Schematic of the neuromuscular simulator for a given muscle of the Triceps Surae. In this study, we explored the EMG responses of the SO muscle.

Hill-type muscle models [8] were included in the system's structure. The model encompasses: a nonlinear series element, representing the muscle tendon and distal aponeurosis; parallel elements representing the viscoelasticity of the muscle fiber; and contractile elements (CE) (see Figure 1). The force generated by each muscle was multiplied by the muscle moment arm, providing the muscle torque. The resultant plantarflexion torque at the ankle joint was given by the sum of the torques generated by the SO, MG and LG muscles. Motor unit action potentials (MUAPs) were modeled as Hermite-Rodriguez functions, and the muscle EMG was the sum of the MUAPs of all active MUs. The EMG was assumed to be recorded from the SO muscle as is usually done experimentally.

Biophysically based spindle models were included parallel to each muscle model (see Figure 1). The mean firing activity of Ia and type-II afferents provided by the spindle model were converted to spike trains following inhomogeneous Poisson point processes. In addition, we adopted a model for the recruitment of afferents similar to [6]. Fusimotor activity was 100 Hz in all simulations.

Simulation Protocols. All simulations were performed with a basal muscle contraction so that the resultant plantarflexion torque was at approximately 10% MVC. Ankle joint angle was maintained at 0° (neutral position) for 1 s followed by a ramp-and-hold perturbation with a 150°/s velocity and a steady state amplitude equal to 9°. Three different systems were tested: *i)* the full model as described above; *ii)* a model in which type-II afferents (and group-II INs) were absent; and *iii)* a model in which Ia afferents were absent. Three simulations were performed in each condition, and their average is reported here.

RESULTS AND DISCUSSION

Figure 2a shows the SO EMG for the three simulated conditions. The first EMG response (i.e. SLR) is observed at a latency of ~50 ms for the full model (black trace) and when only Ia afferents are present in the system (red trace). The reflex amplitude is similar in both conditions, suggesting that this response is mainly generated by the Ia activity. MLR is observed in all three conditions, with a latency of ~70 ms. Figure 2b (note the longer time interval in the abscissa) shows clearly the contribution of the reflexes to plantarflexion torque and SO muscle length (black response) as compared to the case of a passive muscle (blue response) subjected to the same stretch.

When the full model was simulated, the MLR amplitude was similar to the condition with only Ia afferents, suggesting that this response is also influenced by the sustained activity of the primary afferents. Nonetheless, when the Ia afferents were absent, only a delayed (+10 ms) version of the MLR was observed. To represent the larger amplitude of MLR observed experimentally [3] a gain adjustment was required, perhaps corresponding to chronic changes in the spinal cord following the lack of Ia inputs. The MLR/SLR ratio was ~0.50 for conditions (i) and (ii), similar to those observed experimentally [2].

CONCLUSIONS

Stretch reflexes are important neurophysiological mechanisms in the control of movements. For instance, they have a key role in joint stiffness regulation. In this study, we are presenting a large-scale biologically based neuromuscular model that can be used to simulate stretch reflexes of ankle extensor muscles. The main findings suggest that both Ia and type-II spindle afferents are responsible for the genesis of the MLR, whereas the SLR is mainly due to the Ia afferent activity. This model can be used to test hypotheses and to raise new questions regarding the mechanisms behind spinal reflexes in both health and neuromuscular diseases.

ACKNOWLEDGEMENTS

This study has been supported by FAPESP (São Paulo Research Foundation) and CNPq (Brazilian NSF). LAE holds a scholarship from the FAPESP (process #2009/15802-0).

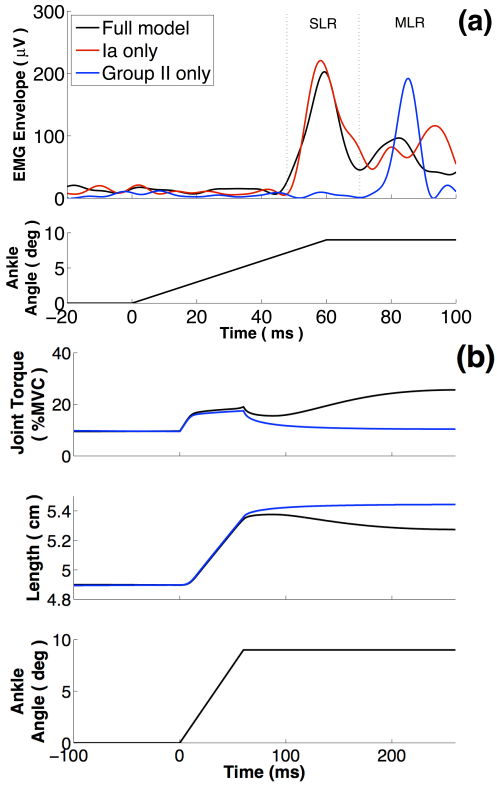

Figure 2: Simulation input and output signals. **(a)** EMG envelope of the SO muscle and ankle angle (input). Black, red and blue curves represent three different simulated conditions (see Methods). **(b)** Ankle joint torque, SO muscle length, and ankle angle (input). Black curves have a reflex contribution, whereas the blue curves represent the passive (no reflex) behavior. The horizontal scale is different in (b).

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