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

RESPONSE OF NEURAL OSCILLATORS TO VELOCITY-DEPENDENT SENSORY AFFERENTS AT THE ANKLE

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INTRODUCTION

Neural oscillators, also known as spinal pattern generators (SPG) may play an important role in generating rhythmic movements [1]. In this study, SPG refer to neural networks that do not require a central input from the brain so as to create a motor output. It has been demonstrated that coordinated electromyographic (EMG) patterns were induced in patients with complete or incomplete paraplegia while walking on a treadmill with reduced loading [2]. Such findings support the view that humans may use SPG in generating locomotion. The main afferents essential in generating locomotor-like activity in humans were found to be load receptors, consisting of proprioceptive afferents in muscles and exteroceptive afferents from mechanoreceptors in the feet, and afferents related to hip position were identified [3,4].

Walking at a slower or faster pace creates different motor demands on the neural system. A number of gait components such as stance and swing phase intervals, in addition to muscle activations, change at different speeds. However, in healthy humans, it is not known whether these changes are a result of sensory cues to the neural network in the spinal cord, since higher commands from the brain can intervene.

The aim of our study was to determine whether neural networks in the spinal cord can adapt to changing sensory afferents, and directly influence muscular activity to meet the motor demands of locomotion. So, we studied the response of the SPG model in situations where gait components in a gait cycle will be different to normal walking: change in walking speed and performing "silly walks".

METHODS

Seven healthy male subjects volunteered to participate in this study. Each subject was requested to walk at his normal self-selected speed (4.8±0.5 km/h), at 3.5 km/h, 4.0 km/h and 4.5 km/h on a treadmill. In addition, they were asked to perform movements unlike normal walking i.e. "silly walks". Force data, hip angles, and EMG measurements of the Soleus (SOL) and Tibialis Anterior (TA) from six consecutive strides were captured simultaneously.

A Matsuoka oscillator [5] consisting of two mutually inhibiting neurons was used (Figure 1); one neuron will activate the SOL and the other will activate the TA. The outputs from the oscillator represented the corresponding muscle activation of each muscle.

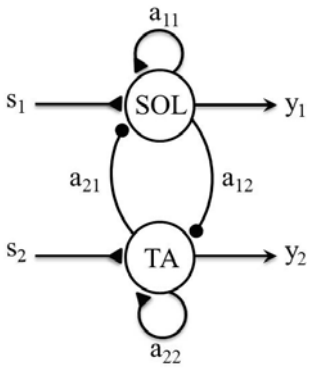

Figure 1: SPG model consisting of two neurons. Dark triangles represent excitatory connections, dark spheres represent inhibitory connections.

The oscillator is adapted from Matsuoka, 1985 [5]:

$$\dot{x}_i + x_i = \sum_i^j a_{ij} y_j + s_i - b_i f \quad (1)$$

$$T_i \dot{f}_i + f_i = y_i \quad (2)$$

$$y_i = \max(0, x_i) \quad (3)$$

where f is the adaptation in the neuron, T and b are the parameters that determine the time course of the adaptation. x is the inner state of the neuron, y is the generated output of the neuron, s is the input signal, and a is the strength of the connection between both neurons; a_{ij} <0 for $i \neq j$ (mutual inhibition) and >0 for $i = j$ (self-excitation). Vertical force and hip flexion/extension angles of the ipsilateral limb were used to determine the inputs s_i.

A nonlinear least squares fitting algorithm was used to determine a set of parameters that would fit the model outputs to experimental EMG data. Gait components (maximum normalised force, maximum range of hip flexion-extension angles, stance and swing phases) were calculated for each stride. In analysing the rectified EMG signals for different speeds, cumulative numerical integration for each EMG signal was calculated [6]. To determine significant differences in the model parameters, multivariate analysis of variance along with Tukey's post-hoc test were performed.

RESULTS AND DISCUSSION

The results showed that neural networks in the spinal cord can activate muscles at the ankle to generate stepping motion during steady-state walking. In humans, it is difficult to determine whether the elevated EMG patterns during walking are from supraspinal control, a result of activations

from sensory inputs, or an interaction from both supraspinal and spinal control. However, the generated outputs by our model consisting of only spinal neurons, suggest that muscle activations can be generated by sensory inputs from loading and hip angles at the spinal level [7].

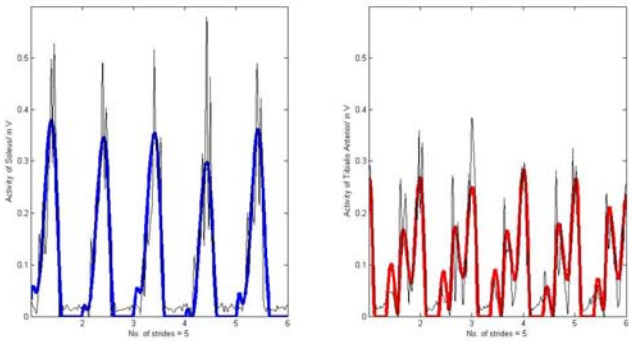

Figure 2: R_{ave}=0.88. Muscle activation of the SOL (R_{SOL}=0.90) and the TA (R_{TA}=0.87) of subject #1 walking at 4.5km/h (bold lines represent the output from the SPG model, thin lines represent the experimental EMG data).

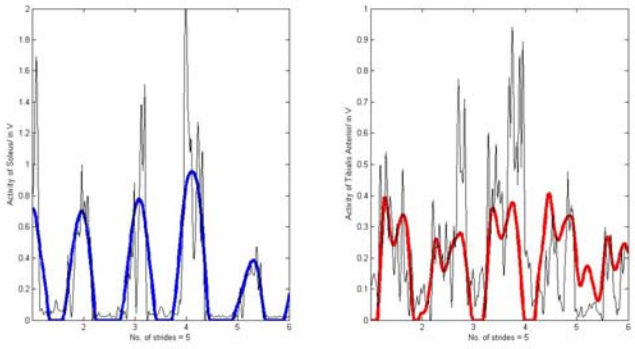

Figure 3: R_{ave}=0.63. Muscle activation of the SOL (R_{SOL}=0.79) and the TA (R_{TA}=0.46) of subject #1 performing silly walks (bold lines represent the output from the SPG model, thin lines represent the experimental EMG data).

As expected, an increase in walking speed is demonstrated by a significant decrease in the relative stance phase duration, an increase in the relative swing phase duration, an increased range of hip flexion-extension angles, increase in maximum normalised force, and increase in the peak activation values of the SOL and TA [6]. This meant that inputs to the SPG model, and the resulting model outputs were significant differently for all walking types.

However, no significant differences were found in all model parameters. No significant differences were also found in correlation *R* between normal walking at self-selected speeds and other speeds (Figure 2). For normal walking at different speeds, this might imply that an insignificant change in a parameter is sufficient in causing a significant change in the output. Since the control of these parameters, which determine the neuronal properties of the SPG, could possibly come from interneurons, presynaptic inhibition [5,8] or descending pathways from supraspinal structures, the insignificant changes might imply that regulation from the brain or inter-spinal circuitries are not required to modulate the activation patterns during walking. Thus, higher control is not needed to alter the motor output of moving limbs, but the corrections may instead, be predominantly performed by spinal structures using sensory information that is available [9]. In addition, since the data was captured during steady-state walking without a threat to equilibrium, the same neural network will be utilised [10,11]. Perhaps, changes to gait components are secondary, and might be a result of changes to stride length, rather than the result of a different motor control mechanism.

We postulated that the muscle activations during silly walks were due to a command from the brain. So, we were expecting to observe differences in the silly walks since the subjects were intentionally performing 'something silly'. However, we found significant changes only in T_2, the time constant responsible for the time lag of the adaptation effect in the TA (equation 2). *R* calculated for silly walks (R_{mean}=0.70±0.08) were significantly lower than the other walking types (Figure 3).

It was speculated that persistent sodium and calcium inward currents levels, which play an essential role in the firing activities of motoneurons, are expressed more in the extensors, than the flexors [12,13]. Since extensors are mostly activated during walking, it would be more functional to modulate the flexors which do not require long lasting bursts. Nevertheless, it remains uncertain here if the TA requires more intervention from the brainstem or additional neural circuitries than the SOL. The significant difference in T_2 could also be due to the SPG model, which requires a strong adaptation effect in generating stable oscillations [5] ($T_{2,\ silly\ walks}$=0.12±0.17 compared to $T_{2,\ self\text{-}selected}$=0.03±0.04). So, the significantly higher value of T_2 could just be a way for the model to continue generating stable oscillations.

CONCLUSIONS

We proposed that SPG in the spinal cord can interpret and respond accordingly to velocity-dependent afferent information. Changes in walking speed do not require a different motor control mechanism so long as equilibrium is not affected.

ACKNOWLEDGEMENTS

This study was supported by the German Federal Ministry of Education and Research (BMBF), project no. 01EC1003A.

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