NATA RESEARCH & EDUCATION FOUNDATION

Grant Information Summary:

The Relationship Between Muscle Stiffness and Muscle Spindle Sensitivity in the Triceps Surae

Practical Implications:

Soleus spinal stretch reflex (SSR) latency and amplitude were not affected by varying levels of triceps surae (TS) stiffness. Low levels of muscle stiffness require greater neural drive to the muscle spindle to reach these set points, while high levels of muscle stiffness require less neural drive to the muscle spindle. These results may suggest that rehabilitation programs aimed at improving dynamic joint stability should incorporate increases in muscle stiffness.

Background

Muscle stiffness provided by the series elastic element potentially contributes to joint stability from both mechanical and neuromuscular perspectives. The spinal stretch reflex (SSR) is a component of neuromuscular control, which contributes to joint stability. Sensitivity of the spinal stretch reflex operates on a set-point basis, whereby the latency and amplitude responses of a muscle are functions of its stiffness and neuromotor drive. Increased muscle stiffness may heighten SSR sensitivity via enhancement of the mechanical coupling of the muscle spindle and the stretch reflex stimulus. A reflexive response of greater magnitude in a timelier manner following the onset of joint perturbation may allow for enhanced dynamic joint stabilization.

Objective

To determine if higher series elastic muscle stiffness would shorten soleus spinal stretch reflex latency, while increasing its amplitude during a mechanical perturbation.

Design and Setting

Triceps surae (TS) muscle group stiffness, the soleus Hoffmann reflex (H-reflex), and the soleus SSR responses were compared between males and female. All testing was performed in the Sports Medicine Research Laboratory at the University of North Carolina at Chapel Hill.

Subjects

Twenty males (ht = 1.81±0.06 m, mass = 81.83±12.21 kg, age = 22±3 yrs) and 20 females (ht = 1.67±0.07 m, mass = 63.55±9.84 kg, age = 22±3 yrs) participated.

Measurements

TS stiffness, the soleus H-reflex, and the soleus SSR were compared across sex. TS stiffness was estimated from the damped frequency of oscillation of the shank about the ankle following perturbation. The soleus H-reflex was elicited by electrically stimulating the tibial nerve, providing an indication of total motorneuron pool excitability. The soleus SSR was evoked via mechanical ankle dorsiflexion perturbation. For each assessment, subjects were seated with the soleus active to ~15%MVC.

Results

TS stiffness was significantly greater in males than in females ($t_{38} = 6.160$, $P < 0.001$), thus verifying high (males) and low (females) stiffness groups. Soleus H-reflex latency ($F_{1,37} = 0.030$, $P = 0.862$; ANCOVA, covariate = leg length) and amplitude ($t_{38} = 0.412$, $p = 0.683$), and SSR latency ($F_{1,37} = 0.026$, $P = 0.872$; ANCOVA, covariate = leg length) and amplitude ($t_{38} = 0.755$, $P = 0.455$) were not significantly different between stiffness groups. Similarly, the respective relationships between TS stiffness and soleus SSR latency ($r = 0.072$, $P = 0.659$) and amplitude ($r = 0.047$, $P = 0.772$) were not significant.

Conclusions

Similarity of SSR latency and amplitude across varying levels of muscle stiffness was due to the combined effects of series elastic stiffness and neural drive to the muscle spindle to achieve set-point values for SSR latency and amplitude for standardized testing conditions. Given the limited sample variability in SSR latency and amplitude and the relationship between muscle stiffness and neural drive to the muscle spindle suggested by previous literature, these results indicate that low levels of muscle stiffness require more neural drive to the muscle spindle to attain SSR latency and amplitude set points compared to high levels of stiffness.

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