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ALTERED LENGTH-FORCE CHARACTERISTICS AND EPIMUSCULAR MYOFASCIAL FORCE TRANSMISSION IN SPASTIC RAT CALF MUSCLES

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SUMMARY

The aims of the present study were to investigate the effects of spasticity (1) on muscle tendon unit (MTU) length-force characteristics of rat m. gastrocnemius (GAS), m. soleus (SOL) and m. plantaris (PL), and (2) on epimuscular myofascial force transmission between synergistic GAS, SOL and PL, as well as between the calf muscles and antagonistic m. tibialis anterior (TA). The effects of spasticity on rat calf muscles were found to be muscle specific. Length-active force characteristics of spastic GAS and PL muscles were narrower with a reduced maximal active force, whereas maximal active force of spastic SOL was nearly twice as high as from the control SOL. The main difference in length-passive force characteristics were related to the shorter MTU in the spastic calf muscles. The distribution of myofascial force transmission between spastic GAS and SOL seems to be different, but not greater, than in the controls. No sign of antagonistic epimuscular myofascial force transmission between the calf muscles and TA was found in neither spastic nor control rats.

INTRODUCTION

Studies investigating the mechanical characteristics of skeletal muscles in spastic limbs have been limited to biopsies [1], non-invasive methods like ultrasound imaging [2], or single muscles tested during surgery in humans [3]. These studies have found indications that human spastic muscles have a shorter MTU and a shorter muscle belly [2] than typically developed muscles. The mechanical properties of several spastic muscles from the same compartment have not yet been investigated. There are indications that the extracellular matrix in spastic muscles is stronger and more abundant [4]. Evidence of force transmission between spastic muscles via epimuscular myofascial linkages has been reported [3], but not compared with controls.

The aims of the present study were to investigate the effects of spasticity (1) on MTU length-force characteristics of rat GAS, SOL and PL, and (2) on epimuscular myofascial force transmission between synergistic GAS, SOL and PL, as well as between the calf muscles and antagonistic TA.

METHODS

Spastic rats from a mutant Han Wistar strain (SP; n=4, 146±15g, 46-55 days) were compared with typically

developed Wistar rats (weight matched controls WMC; n=4, 140 ± 19 g, 36-52). In deeply anesthetized rats, skin and m. biceps femoris was removed from the left hindlimb. The distal tendons of GAS, SOL, PL and TA were dissected and each connected to separate force transducers. Connective tissues between muscle bellies were left intact. The common peroneal nerve was cut just distally of its branching from the sciatic nerve. Electrode cuffs were placed on the cut common peroneal nerve, innervating TA, and the sciatic nerve at the middle of the thigh innervating GAS, SOL and PL. In the setup, the femur was fixed vertically with a metal clamp and the foot was vertically fixed on a plastic plate making the knee and ankle angle 90° and the lower leg horizontal.



Figure 1: Schematic illustration of experimental protocols.

Total isometric forces exerted at the distal tendons of all four muscles on supramaximal stimulation of common peroneal and/or sciatic nerves (100 Hz, 500ms) were measured during tetanus and in passive state for 2 protocols (Fig. 1): (*i*) Lengthening of all calf muscles simultaneously from active slack length (L_{slack}) to 2 *mm* above optimum length (L_{opt}) by 1 *mm* increments. TA was kept at a length corresponding to 90° ankle and knee angles (L_{ref}). Common peroneal and sciatic nerves were stimulated simultaneously. (*ii*) Lengthening of GAS from L_{slack} to 2 *mm* above L_{opt} by 1 *mm* increments. SOL, PL and TA were kept at L_{ref} . Only the sciatic nerve was stimulated.

Active forces were calculated by subtracting passive force from total force at equal MTU length. Active and passive length-force data were fitted to calculate means and SDs, as well as to assess maximal active force ($F_{a,max}$), L_{opt} and L_{slack} . Electromechanical delay (EMD) was calculated as the latency from nerve stimulation to force development with muscles at L_{ref} and L_{opt} . Following the mechanical measurements, GAS and SOL muscles were excised to determine muscle belly mass. . Length-force curves were compared with two-way ANOVAs; groups (SP vs. WMC) crossed with length as repeated measurement.

RESULTS AND DISCUSSION

Length-active force characteristics of spastic GAS (Fig. 2) and PL (not shown) were significantly narrower than those of controls. Optimum MTU length was significantly lower in spastic GAS and PL ($\Delta L_{ref} = 1.49 \pm 0.84 mm$ and $0.29\pm0.53mm$ resp.) compared to controls (ΔL_{ref} = 3.46±0.88mm and 2.02±1.17mm resp.). No difference in L_{slack} was found. $F_{a,\text{max}}$ of PL was significantly lower in spastic rats (1.56±0.27N) compared to control rats (2.31±0.29N). Similar results were found for GAS, but differences were not statistically significant. This is consistent with previous findings on length-force characteristics in spastic human muscles [4]. In contrast, spastic SOL differed from controls by significantly higher $F_{a,max}$ (0.947±0.136N vs. 0.644±0.083N, Fig. 2) and not in the measured lengths. This indicates that SOL was affected differently by spasticity than GAS and PL. These results suggest that spastic SOL has adapted to compensate for the weakened spastic PL.



Figure 2: Active and passive length-force characteristics of GAS (top) and SOL (bottom), obtained with protocol *i*. MTU length is expressed as deviation from L_{ref} . Means \pm SD are plotted. + denotes interaction between SP and WMC (p < 0.05) and (*) denotes a tendency of higher force in SP than WMC (p = 0.06).

The narrower length-active force curve in spastic GAS and PL may be explained by fewer sarcomeres in series within muscle fibers, a decrease in the physiological cross-sectional area or a shorter MTU.

When comparing the length-passive force curves for GAS interaction effects were found between SP and WMC. No differences were found in EMD. Similar results were found for SOL (not sig.) and PL (sig. interaction). These findings suggest that the main difference in length-passive force characteristics were related to the shorter MTU in the spastic calf muscles, and not stiffness.

Active force of restrained SOL and PL in protocol *ii* decreased significantly with lengthening GAS distally (data not shown). This can be explained by effects of epimuscular myofascial force transmission. The length-force curve for SOL showed significant interaction effects between SP and WMC, which indicates that the force transmitted myofascially between GAS and SOL differed between SP and WMC rats. However, the magnitude in force decline in spastic SOL and PL ($49.50\pm12.40\%$ and $14.70\pm9.61\%$ resp.) did not differ significantly from the decline in controls ($32.32\pm25.75\%$ and $25.19\pm17.17\%$ resp.). Thus, further investigation is needed to identify the specific difference. No effects of epimuscular myofascial force transmission between the calf muscles and antagonistic TA was found in neither spastic nor control rats.

CONCLUSIONS

Length-force characteristics of calf muscles in spastic rats were different from those in controls. Effects of spasticity were muscle specific. Previous studies [1,2,3] have investigated one muscle of synergistic group only. Our results indicate that this may not be representative for the whole group of synergistic muscles.

Epimuscular myofascial force transmission was found between calf muscles in both groups, and interaction effects between groups were found. No sign of antagonistic epimuscular myofascial force transmission between the calf muscles and TA was found in neither spastic nor control rats. Epimuscular interaction between spastic muscles has not previously been compared with controls. Thus, this is the first indication of altered epimuscular interaction between spastic calf muscles.

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