

RESIDUAL FORCE DEPRESSION IS NOT ABOLISHED FOLLOWING A QUICK SHORTENING STEP

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INTRODUCTION

When a muscle is actively shortened and then held at a constant length until all transient force recovery has disappeared, the force following shortening is smaller than the corresponding steady-state force obtained for a purely isometric contraction at the corresponding length. This phenomenon has first been described systematically by Abbott and Aubert (1952) and is referred to as (residual) force depression.

Recently, the idea that force depression is caused by an inhibition of cross-bridge attachment in the actin-myosin zone that is newly formed during shortening has received strong support (Herzog, 2004). The “cross-bridge inhibition theory” predicts that when stress on actin is released, force depression should be abolished. This observation has been made when force-depressed muscles were deactivated just long enough for force to drop to zero.

Stress in force-depressed muscles can also be released by shortening the muscle at great speed. The purpose of this study was to shorten a force-depressed muscle quickly so that force dropped to zero. We hypothesized, in accordance with the cross-bridge inhibition theory, that force depression would be abolished following the quick shortening step.

METHODS

All testing was performed with cat soleus muscles ($n=8$) as described in detail previously (Herzog and Leonard, 1997).

Each set of tests consisted of ten contractions. The first two and last two contractions were isometric reference contractions performed at optimal length (hereafter referred to as 0mm length) and 9mm shorter than optimal length (-9mm). The reference contractions were complemented by six test contractions. The first consisted of a 1s isometric contraction at a length 9mm greater than optimal, followed by shortening at 4.5mm/s, followed by an isometric contraction at 0mm. The second test contraction was identical except that shortening occurred from +9mm to -9mm. The third was identical to the second, except that the speed of shortening was 200mm/s. The remaining three test contractions consisted of slow shortening (4.5mm/s), followed by quick shortening (200mm/s) with a delay between the slow and quick shortening of 0, 1 and 2s.

Mann-Whitney statistics were used to identify differences between the forces obtained for the reference and shortening contractions. Kruskal-Wallis statistics were used to identify differences in force depression between the slow, fast and the slow-followed-by-fast shortening tests. The level of significance was 0.05 for all tests.

RESULTS AND DISCUSSION

Slow shortening of soleus produced statistically significant force depression for both magnitudes of shortening (Figure 1) with mean values of 5.3% ($\pm 1.9\%$) for the 9mm shortening and 5.8% ($\pm 1.3\%$) for the 18mm shortening distance.

Active shortening at the quick speed (200mm/s) resulted in a drop of the active force to zero (Figure 2) and there was no force depression. When force depression was induced by shortening at the slow speed and then followed by the quick shortening step, active forces dropped to zero. However, there remained a significant force depression of 3.2% ($\pm 0.5\%$), 3.7% ($\pm 0.5\%$); and 4.2% ($\pm 1.5\%$) when the quick and slow shortening were delayed by 0, 1, and 2s, respectively.

If the cross-bridge inhibition theory was correct, force depression should have been abolished after the quick shortening step. However, that was not observed in this study. Rather, quick shortening steps following slow shortening known to induce force depression were associated with a small but consistent and statistically significant force depression (Figure 2), while the quick shortening steps alone were not. This result indicates that loss of force depression during deactivation is not caused by stress release on actin, but by changes associated with deactivating the muscle.

SUMMARY/CONCLUSIONS

We conclude from these results that a small amount of force depression persists following stress release caused by quick shortening, and therefore that the cross-bridge inhibition theory cannot be the sole cause of force depression. We suggest that force depression might be associated with an

inhibition of cross-bridge attachment that is controlled in the cross-bridge itself.

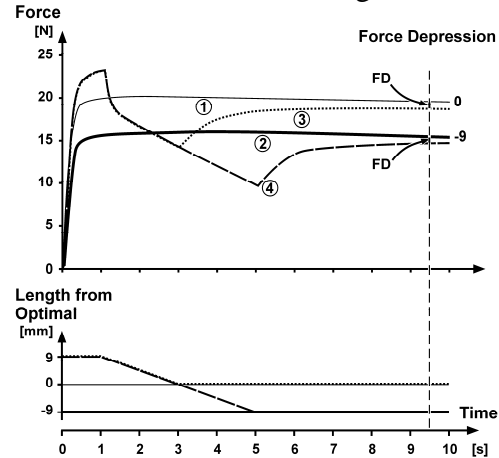


Figure 1: Force depression following slow shortening of soleus for 9 and 18mm.

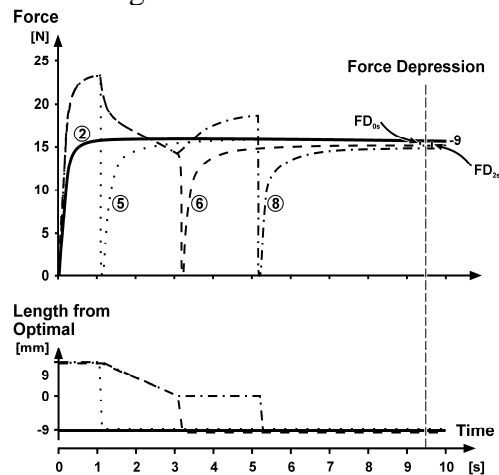


Figure 2: Force depression persists when slow shortening is followed by a quick shortening step

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