Dissociation of recovery of muscle activation and force following a sustained maximal isometric contraction

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ABSTRACT

To investigate time-dependency of nervous system recovery following muscular contractions, subjects (n=10) performed two bouts (B1, B2) of sustained maximal isometric handgrip contractions separated by 10-min recovery. Force and surface EMG were collected continuously throughout contraction bouts but were analyzed at 15 sec intervals (1 sec segments). iEMG and mean power frequency (MPF) were calculated for the brachioradialis (BR), flexor carpi radialis (FCR) flexor carpi ulnaris (FCU), and flexor digitorum profundus (FDP) muscles. Muscle activation (iEMG-MPF: BR:68-20%; FCR:72-16%; FCU: 65-20%; FDP:48-50%, respectively) and isometric force (88%) decreased following B1(120-sec contraction). Following recovery, initial force of B2 was significantly less than B1 (23%); however muscle activation (iEMG) was similar to B1 for BR, FCR and FDP. Initial B2 FCU iEMG activity was lower but increased to B1 levels by 15 sec; remaining similar throughout. MPF was similar and decreased similarly over time in both bouts but tended to be higher at initiation of B2. The mechanical response was similar in both bouts despite differences in initial force generation (B1: $y = -0.17^2x+22.7$; R2=0.98; B2: $y = -0.182x+17.9$ R2=0.97). Incomplete recovery of force observed in B2 suggests interference in excitation-contraction coupling while fatigue within each bout appears specifically related to changes in muscle activation.

INTRODUCTION

Muscular fatigue is a natural consequence of repetitive muscle activity and is characterized by a change in muscle activation and/or maximal force generating capacity (Hunter *et al.*, 2004; Leonard, 1994; Garland *et al.*, 1987;). Additionally, muscle fatigue is associated with a decrease in mean power frequency (MPF) of the surface electromyogram (EMG) (Mills, 1981). Research has largely attributed this decrease in performance to neural changes (Leonard *et al.*, 1994, Garland *et al.*, 1987, Bigland-Ritchie *et al,* 1986) and multi-phase metabolic changes (Baker *et al.*, 1993) within the muscle. The phases of metabolic change suggest that muscle cells rotate through highly oxidative, intermediate, and highly glycolytic phases as identified through inflections in intracellular [H^+] (Kent-Braun, *et al.*, 1993).

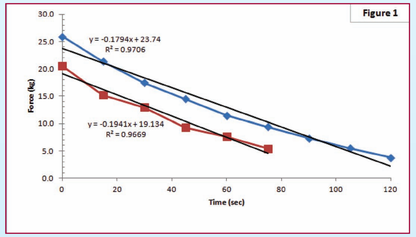
Consequently, recovery from muscle contractions has been shown to be phasic in that there is dissociation between mechanical and metabolic recovery (Brechue et al., 1992; Miller *et al.*, 1987). In these cases, metabolic recovery preceded restoration of mechanical function. Further, recovery of force generating capacity has been shown to be fiber-type dependent such that fast-twitch muscles fatigue faster and recover quicker than slow-twitch muscles (O'Drobinak and Brechue, 2000; O'Drobinak et al., 1999). These later experiments suggested that fast fatigue and recovery was primarily of neural origin. Longer duration contractions where mechanical and metabolic recovery was dissociated appeared to be related to changes in excitation-contraction coupling (Brechue *et al.*, 1992; Miller *et al.*, 1987). Studies have shown cases where [H^+] concentration did not change despite reduced force generation. This suggests a disparity between metabolic changes and contractile changes that might be occurring in the excitation-contraction coupling mechanism (Saugen, *et al.*, 1996) or during Ca^{2+} re-uptake. These contractile contributions to fatigue may be initiated by the metabolic or neural mechanisms discussed above (Leonard *et al.*, 1994).

METHODS

- Ten male volunteers (age = 21.1 + 2 years) volunteered to participate in the present study. All subjects received a description of the study and provided informed consent to participate.
- Muscle activation and force generation were assessed during two bouts of sustained maximal isometric handgrip contractions separated by a 10-minute recovery period. The duration of the contraction bouts were 2 minutes and 90 seconds, respectively.
- The isometric contraction bouts were conducted with a handgrip dynamometer (Biopac). Force output was continuously monitored online (Biopac ACKnowledge software). The dynamometer was calibrated with standard weights prior to each experiment.
- Subjects sat erect with forearms resting on the table surface, elbows at 90° and shoulder adducted 20o. The handgrip dynamometer was held vertically in the hand of the dominant arm.
- Muscle activation was assessed by electromyography (EMG) using surface electrodes (sEMG) affixed to standardized sites (Perotto, 1994) of the brachioradialis (BR), flexor carpi ulnaris (FCU), flexor carpi radialis (FCR), and flexor digitorum profundus (FDP). Prior to each experiment, a maximal voluntary isometric contraction (4 sec.) was recorded and used for each muscle to set the gain on the sEMG amplifier and to normalize EMG activity (nEMG).
- All data were collected at 1,000 Hz.

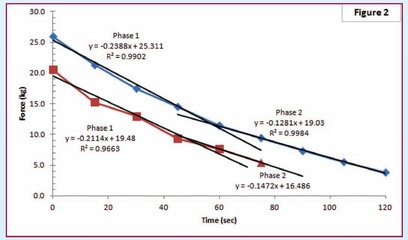
DATA ANALYSIS

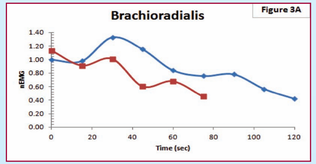
- sEMG signals were rectified and integrated (iEMG) in 1 second intervals. The iEMG was normalized to the initial EMG at Bout 1 for each muscle group.
- The frequency of the EMG signal (mean power frequency; MPF) was analyzed with a FFT transform and filtered using a Hamming window technique. The signal was then normalized to 1 volt and MPF was determined as the frequency at 0.5 volts.
- Data were analyzed with repeated measures ANOVA. A priori alpha level was set at 0.05.

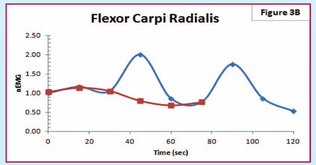


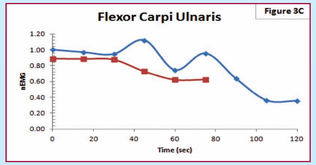
Bout 1

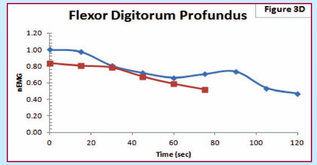
Bout 2







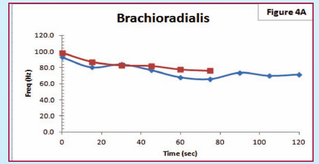


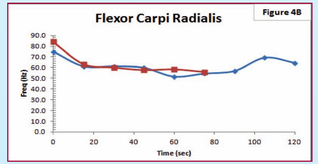


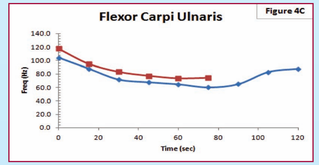
RESULTS

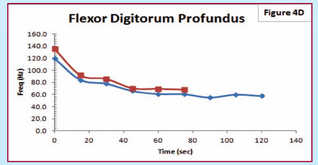
Data are presented as means. Standard deviations are not shown for clarity but are less than 10% of the mean.

- Force generation decreased ~85% during Bout 1 and decreased 75% during Bout 2. Initial force of Bout 2 was ~80% of Bout 1 (see Figure 1).
- The rate of fatigue was the same in both bouts (Bout 1_m = -0.17994; Bout 2_m = -0.1941) despite differences in initial force (see Figure 1).
- Analysis revealed an apparent two-phase fatigue; inflection point 60 sec.
- Phase 1 slopes were greater than Phase 2; slope of Phase 1 was similar between bouts as were the slopes of Phase 2 (see Figure 2).
- Muscle activity (nEMG) was the same in Bout 2 as it was in Bout 1 in all muscles except for the BR and initial FDP (see Figure 3A, B, C, D).
- Initial MPF was greater in Bout 2 than in Bout 1 and similar thereafter (see Figure 4A, B, C, D).









CONCLUSIONS

- Muscle fatigue within bouts is associated with a decrease in central motor drive as indicated by the decline in muscle activity (nEMG and MPF).
- There appear to be two phases of fatigue evidenced by the change in slope of the force-time curve (at or around 60 sec) and lack of recovery of peak force between bouts. This suggests a shift in the mechanism of fatigue.
- Decrease in muscle activity within each bout suggests Phase 1 fatigue is the result of neural impairment. Subsequently, Phase 2 fatigue suggests changes in excitation-contraction coupling which appears later in the bout and recovers slower between bouts.

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