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Recovery of Motoneuron Output Is Delayed in Old Men Following High-Intensity Fatigue

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Dalton BH, Harwood B, Davidson AW, Rice CL. Recovery of motoneuron output is delayed in old men following high-intensity fatigue. *J Neurophysiol* 103: 977–985, 2010. First published December 23, 2009; doi:10.1152/jn.00908.2009. Despite an age-related slowing in the contractile properties of the triceps surae, inherently low maximal motor unit firing rates (MUFRs) in the soleus are unchanged. Fatigue following high-intensity contractions is characterized by contractile slowing in conjunction with a reduction in MUFRs in young adults. Here we exploit the ageing model of the soleus to assess changes in neuromuscular function during fatigue and short-term recovery. We hypothesize that a high-intensity sustained contraction will cause minimal reductions in MUFRs in young and old subjects but that recovery of MUFRs will be delayed in aged subjects. We compared the effects of a high-intensity sustained task on the MUFRs of the soleus and triceps surae contractile properties in six young (~24 yr) and six old (~75 yr) men. Various measures of the contractile function of the triceps surae were tested during two to six sessions via maximal voluntary isometric contractions (MVCs) and tibial nerve stimulation. Populations of MUFR trains were recorded from the soleus during brief (~7 s) MVCs, a high-intensity (75% MVC) sustained fatiguing task, and brief MVCs following task failure at 1, 2, 5, and 10 min. Old men had greater time to task failure than the young (~138 and ~100 s, respectively). Voluntary activation was near maximal (>99%) for all subjects but at task failure, decreased to ~89% in both groups. Maximal MUFRs, for both groups, were reduced by ~44% and twitch contraction duration slowed by ~30% following task failure. Contraction duration recovered equally for both groups within 2 min, but maximal MUFRs did not recover until 5 min in the old compared with 1 min for the young. The surprising fatigue-induced reduction in MUFRs was similar for both groups, but despite a similar recovery of contractile properties for both, recovery of MUFRs was impaired in the old subjects.

INTRODUCTION

The ageing process is associated with diminished neuromuscular function in old adults compared with young (Klass et al. 2007). Despite these age-related changes, when neuromuscular fatigue is systematically tested, the results are equivocal. Older individuals experience more (Baudry et al. 2007; McNeil and Rice 2007), the same (Callahan et al. 2009; Laforest et al. 1990), or less (Callahan et al. 2009; Rubinstein and Kamen 2005) fatigue than the young. These discrepancies may be related to the task-dependent nature of fatigue or the muscle group tested (Enoka and Duchateau 2008).

Foundational studies of neuromuscular fatigue showed that average motor unit firing rates (MUFRs) declined during high-

intensity voluntary isometric contractions (high-intensity fatigue) as force declined and the muscle slowed (Bigland-Ritchie and Woods 1984; Bigland-Ritchie et al. 1983a,b), although there are exceptions reported (Macefield et al. 2000). The reduction in MUFRs may be attributed to alterations in descending drive (Hunter et al. 2008), central fatigue (Fuglevand and Keen 2003), reduced muscle spindle activity (Macefield et al. 1991), group III and IV afferent activity (Woods et al. 1987), or alterations in the intrinsic properties of the motoneuron (i.e., adaptation) (Carpentier et al. 2001).

In addition to contractile slowing with advanced age, several studies report a reduction in MUFRs at moderate to high-intensity nonfatiguing contractions in old compared with younger adults (Klass et al. 2007), but few studies (Christie and Kamen 2009; Rubinstein and Kamen 2005) have explored the effects of ageing on MUFRs in response to high-intensity fatigue, and none of these have concurrently assessed contractile speed. One study (Rubinstein and Kamen 2005) used a fixed time to fatigue (15 intermittent 30-s MVCs, separated by 10 s of rest), and the second (Christie and Kamen 2009) was a sustained 50% MVC until force could no longer be maintained at the target level. The former found the old group was less fatigable with a smaller reduction (~22%) in MUFRs compared with the young (~35%), and the latter found no age-related difference in fatigue with equivalent small reductions (~13%) in MUFRs. The differences between these two studies may be related to the task-dependent nature of fatigue (Enoka and Duchateau 2008), and thus further studies are needed to help elucidate the effects of ageing on the fatigue response of motoneuron output and its relationship to contractile properties. Furthermore, because of these natural alterations in neuromuscular properties with adult ageing (see review by Klass et al. 2007), this model may provide a greater understanding of the strength of association between motoneuron properties and muscle function.

The soleus offers an interesting model to explore these discrepancies. In addition to a high composition (>80%) of slow twitch muscle fibers (Johnson et al. 1973; Trappe et al. 2001), the soleus has low maximal MUFRs (Bellemare et al. 1983) compared with most other limb muscles studied (Enoka and Fuglevand 2001), and unlike most other muscles, maximal MUFRs are not reduced with ageing (Dalton et al. 2009). Overall the soleus does not seem to express the usual age-related alterations in structure and function attributed to the concept of age-related remodeling of motor units (MUs) (Dalton et al. 2008, 2009; Morse et al. 2005). Thus this model of ageing offers a unique opportunity to explore neural properties separate from contractile properties under conditions of stress

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and recovery. Although the inherently low MUFs of the soleus may be related to its slower contractile properties, it is of interest to know whether and how much these relatively low rates can be further reduced with fatigue in aged subjects and if contractile properties will slow to match these rates. Previous studies have not reported changes in contractile properties concomitantly with MUFs or followed these properties through a period of recovery after a high-intensity isometric fatigue protocol. There is some limited data to suggest that recovery of central fatigue is delayed in aged subjects compared with young adults (Hunter et al. 2008). Because of the known inherent limitations with voluntary activation assessed by peripheral nerve stimulation (Klass et al. 2007), a direct measure of motoneuron output during fatigue and recovery may provide additional and direct insight into this interesting observation.

Thus the purpose was to assess the effect of ageing on MUFs and contractile properties in the soleus during a sustained high-intensity fatigue protocol and throughout a period of recovery. We hypothesized that MUFs will decrease minimally during the fatigue protocol for both old and young men due to the inherently low maximal firing rates of the soleus (Bellemare et al. 1983; Dalton et al. 2009). However, the recovery of MUFs will be delayed in the old men despite similar slowing and recovery of contractile properties in both age groups.

METHODS

Subjects

Six old [75.3 ± 4.1 (SD) yr] and six young men (23.5 ± 2.9 yr) participated in this study. The old and young men were similar in height, weight, and body mass index (177.0 ± 4.2 cm, 88.0 ± 12.2 kg, and 28.0 ± 3.5 kg/m² and 173.7 ± 8.5 cm, 82.3 ± 11.8 kg, and 27.1 ± 2.0 kg/m², respectively). The old and young men were considered recreationally active and healthy. Participants were included in the study if they were free of respiratory, cardiovascular, neuromuscular, and metabolic diseases and did not experience any cardiovascular or neuromuscular limitations to the experimental protocol. Oral and written consent were obtained prior to the testing of all subjects. The local university's ethical Review Board for Health Sciences Research Involving Human Subjects granted approval of the study.

Experimental arrangement

Testing sessions were performed on the right (dominant) leg in a custom-built isometric dynamometer used to record plantar flexion torque. Subjects were seated upright with the hip, knee, and ankle angle positioned at $\sim 90^\circ$. The soleus contributes $\sim 70\%$ to triceps surae torque with the knee extended (Fukunaga et al. 1992), and with the knee flexed to $\sim 90^\circ$, its contribution should be further maximized at the expense of the gastrocnemii (Cresswell et al. 1995; Kawakami et al. 1998). The ankle joint was aligned with the axis of rotation of the footplate. The leg was secured with a C-clamp pressing firmly against the distal aspect of the right thigh to minimize extraneous hip and knee movement during the plantar flexion contractions. Two Velcro straps, attached to the dynamometer footplate, were fastened across the dorsum and the toes. All plantar flexor torques were transmitted through a rigid footplate and strain gauge mounted at the joint axis of rotation. Torque output was sampled on-line at 500 Hz.

Bipolar surface electromyographic (EMG) signals were recorded from the soleus and medial gastrocnemius with self-adhering pediatric electrocardiogram cloth electrodes (H59P repositionable monitoring

electrodes; Kendall, Mansfield, MA). The interelectrode distance for both pairs was ~ 2 cm. One EMG electrode pair was positioned over the muscle belly of the medial gastrocnemius and a second pair ~ 2 cm below the gastrocnemius border, along the longitudinal axis over the soleus. A ground electrode was placed over the lateral malleolus. Surface EMG signals were preamplified ($\times 100$), amplified ($\times 2$), and sampled on-line at 2,000 Hz.

Single MUFs were recorded using the technique of Bigland-Ritchie et al. (1983a). Using custom-made insulated tungsten microelectrodes (125 μ m diam, 3–6 cm length, 5- μ m bared tip), two separate channels of intramuscular recordings were sampled: one from the medial and one from the lateral aspects of the soleus. A common reference surface electrode for both channels was placed over the anterior border of the tibia, and a ground was positioned over the medial malleolus. The skin was cleansed with 70% isopropyl alcohol prior to the placement of the surface electrodes and insertion of the two microelectrodes. Each microelectrode was manipulated by a separate operator. During the voluntary sustained contractions, each microelectrode was moved gradually (< 0.5 cm/contraction) to sample from as many muscle fibers, and presumably MUs, as possible. The recording of discrete single MU potential trains was facilitated by visual (computer monitor) and audio (audio speaker and headphones) feedback. To sample from different regions and different depths, the microelectrodes were often reinserted in slightly different locations after several contractions. Intramuscular EMG signals were preamplified ($\times 100$) and high-pass filtered (10 Hz) using a Neurolog NL824 (Hertfordshire, UK) preamplifier and filter. Both intramuscular and surface EMG channels were converted from A/D by a 12-bit A/D converter (model 1401 plus, Cambridge Electronic Design, Cambridge, UK). Intramuscular EMG signals were sampled on-line at 12 kHz.

Experimental procedures

To amass a large and representative sample of many different MUs from each soleus muscle, subjects visited the lab for multiple testing sessions (2–6 sessions) that consisted of identical procedures. Each visit was separated by ≥ 7 days. During each session, the maximum M-wave and corresponding twitch were elicited via supramaximal stimulation of the tibial nerve at the popliteal fossa using 100- μ s square-wave pulse set at a maximal voltage of 400 V (Digitimer stimulator, model DS7A; Digitimer, Welwyn Garden City, UK). The M-wave amplitude and twitch torque were monitored as the current was increased gradually until a plateau was achieved in both parameters. Once reached, the current (250–600 mA) was increased another 10–15% to ensure supramaximal stimulation.

Next the microelectrode EMG needles were inserted into the soleus, and once electrode placement was established, three maximal isometric voluntary contractions (MVCs) lasting ~ 7 s each were performed. All voluntary contractions were separated by ≥ 2 min of rest. A supramaximal twitch was delivered at rest ~ 1 s pre-MVC, during the peak plateau of the MVC (Ts) and ~ 1 s following (Tr), once the plantar flexors were fully relaxed. The supramaximal pulses were used to assess voluntary activation of the plantar flexors via the interpolated twitch technique { % activation = $[1 - (Ts/Tr)] \times 100$ } (Todd et al. 2004). All subjects were provided with visual feedback displayed on a computer monitor and strong verbal encouragement was consistently provided. A further attempt was given if the three MVCs varied in peak amplitude by $> 5\%$. Following the MVCs, intramuscular recordings were also collected during submaximal sustained (~ 10 s) isometric contractions at 25 and 50% MVC. These results were used for baseline measures reported in a previous study (Dalton et al. 2009). To aid in matching the desired target torque output, a line was placed on the computer screen for the subjects to follow. Each subject was instructed to ramp up to the torque target within 1–2 s and hold the contraction as steady as possible. Subjects alternated three attempts at each target level for a total of six contractions.

Subjects then performed the fatigue task, which consisted of a sustained isometric contraction starting at 75% MVC until the torque dropped <50% MVC for a period of 3 s. To assess voluntary activation failure during the fatigue protocol, a supramaximal twitch was delivered to the tibial nerve just prior to termination of the task and two more each separated by ~1 s delivered as soon as the muscle was relaxed. Recovery measures were then recorded at 1, 2, 5, and 10 min following task failure. Each recovery point included a resting twitch, a ~7-s MVC with a superimposed twitch, and a post-MVC twitch.

Data analysis, reduction, and statistics

As previously described (Connelly et al. 1999), off-line analysis of the unprocessed intramuscular EMG signal consisted of manual comparison of individual action potentials from an identified MU train using software consisting of key features including a window discriminator, shape recognition, and overlay of sequential action potentials (Spike 2, Cambridge Electronic Design). Figure 1 displays an example of three MU action potential trains during the fatigue task. To qualify as an acceptable MU action potential train for firing rate

analysis, a minimum of four contiguous interspike intervals (i.e., 5 action potentials) were needed (Bigland-Ritchie et al. 1983a). Finally, to be accepted as a MU action potential train, the MUFV variability assessed as the coefficient of variation (CV) had to be <30% { $CV (\%) = [SD (\text{Hz})/\text{mean firing rate} (\text{Hz})] \times 100$ }. Only MU trains that fit all these criteria were accepted for further analysis.

Because time to task failure varied among the subjects, MUFV data collected during the voluntary sustained isometric fatigue task were grouped into four 25% bins based on time to task failure. To analyze surface EMG data of the soleus and medial gastrocnemius, a root mean square (RMS) value was calculated over a 1-s interval about the peak torque for all baseline and recovery MVCs and normalized to the RMS of the M-wave. To compare RMS values for the fatigue protocol, the surface EMG signal was related to the duration of the fatigue task and expressed as a percentage of time and divided into four 25% bins as described for the MUFVs in the preceding text. However, the surface EMG signal was interpreted with caution because cancellation of positive and negative peaks in the signal can underestimate muscle activity (Keenan et al. 2005) and may lead to erroneous conclusions based on this signal especially during cross-sectional experiments involving fatigue protocols.

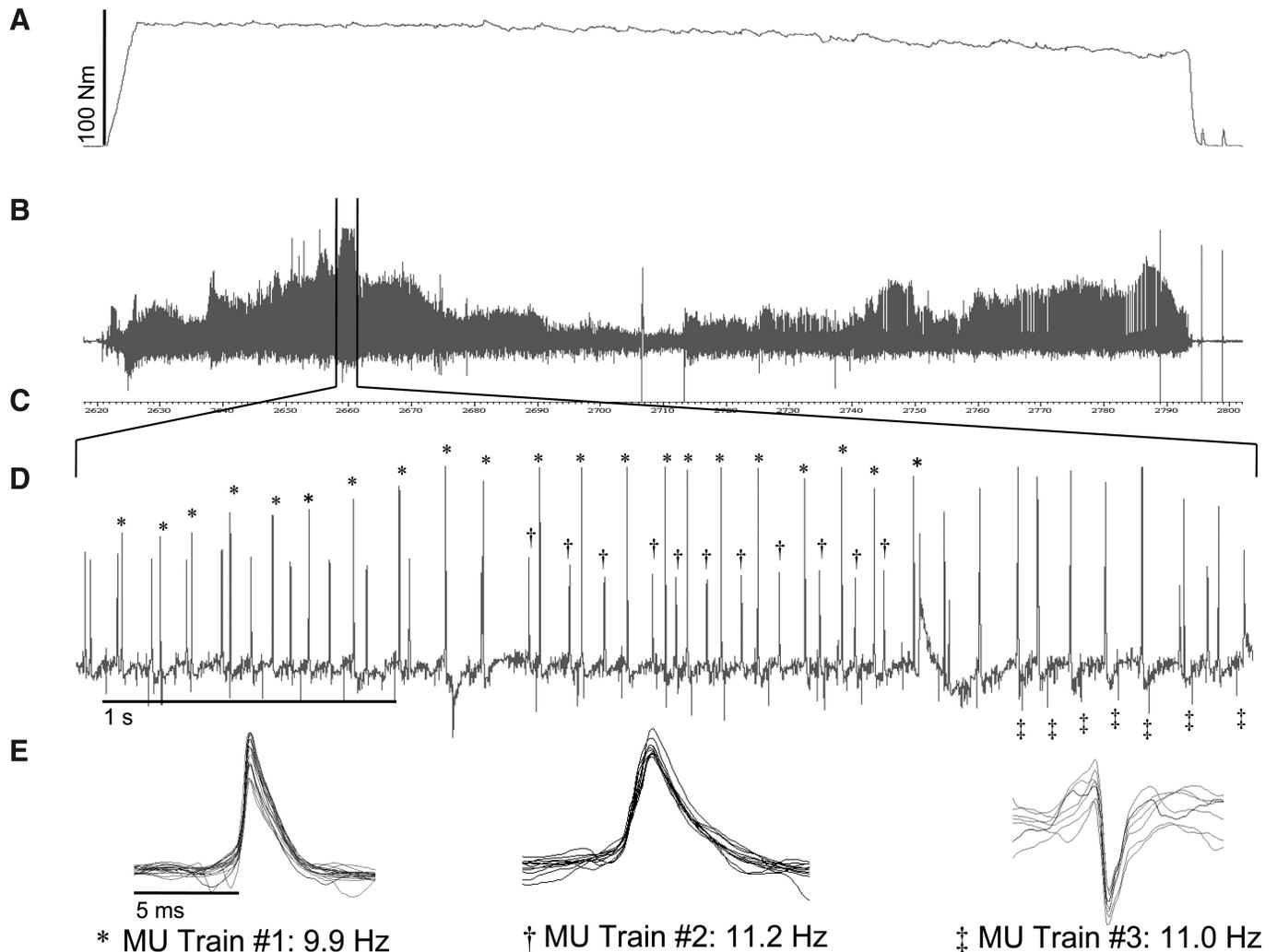


FIG. 1. Example of a typical microelectrode recording during a high-intensity isometric fatigue task. *A*: a torque tracing. *B*: electromyographic (EMG) recording from 1 of 2 microelectrodes in the soleus. As the intramuscular microelectrode was advanced through the muscle during the protocol, amplitudes of the action potential recordings varied in height in relation to the distance from the recording surface. *C*: the time scale of the protocol in seconds. *D*: a 4-s expanded view of the microelectrode EMG recording, highlighting distinct motor unit (MU) action potential trains. *E*: the shapes of distinct action potentials (overlaid) used to extract separate MU trains from the unprocessed microelectrode EMG signal with firing rates of each discrete MU action potential train. *, MU action potentials of MU train 1; †, MU action potentials of MU train 2; ‡, MU action potentials of MU train 3.

For M-wave characteristics, peak-to-peak amplitude, duration, and area were analyzed. Twitch characteristics used for analysis were peak twitch torque (Nm), time to peak twitch torque (TPT; ms), half relaxation time (HRT; ms), contraction duration (TPT + HRT; ms), maximal rate of torque development (s^{-1}), and maximal relaxation rate (s^{-1}). All evoked characteristics, except for voluntary activation, were taken from the pre-MVC twitch and M-wave for baseline, fatigue, and recovery values.

Data were analyzed using the statistical software, SPSS version 15 (SPSS, Chicago, IL). A two-way ANOVA (age \times time course) with repeated measures was used to analyze all data except time to task failure in which an unpaired *t*-test was used. The level of significance was set at $P < 0.05$. If a significant main effect or interaction was present, paired *t*-test were performed with a Bonferroni correction factor to determine where differences existed. Pearson correlation coefficients (*r*) were calculated for subjects' twitch HRT and MUFs to analyze baseline and task failure values. Descriptive statistics for group data are given as means \pm SD for text and figures.

RESULTS

Torque properties, voluntary activation and time to task failure

All baseline values and subject characteristics were previously reported (Dalton et al. 2009). The old men were 35% weaker ($P < 0.05$) than the young men and sustained the fatigue task 39% longer ($P < 0.05$; Table 1). The old men maintained the 75% MVC target torque for 47.5 ± 18.1 s compared with 28.8 ± 18.7 s for the young men ($P < 0.05$). There was a main effect for time for the MVC ($P < 0.01$) and voluntary activation ($P < 0.05$). At task failure, the MVC was reduced equally to 50% of baseline in each group and did not recover (Fig. 2). Voluntary activation decreased similarly in the old and young men from >99 to $89.9 \pm 9.1\%$ at task failure, and recovered within 1 min for each age group (Fig. 2).

The old men exhibited a 25% slower TPT ($P < 0.05$) and 20% longer contraction duration ($P < 0.05$) of the baseline twitch than the young men, respectively, with a trend ($P = 0.08$) toward a 30% lower peak twitch torque (Table 1). Immediately following task failure and throughout recovery, there was a main effect for time ($P < 0.01$), but no main effect for age ($P = 0.10$ to $P = 0.67$) or interaction ($P = 0.11$ to $P = 0.76$) for twitch contractile properties, and thus these data were

TABLE 1. *Baseline values and time to task failure*

Neuromuscular Property	Young	Old
MVC, Nm	146.9 \pm 47.2	96.2 \pm 16.8*
Voluntary activation, %	99.2 \pm 0.7	99.0 \pm 0.7
Peak Twitch, Nm	12.6 \pm 3.8	8.6 \pm 3.3†
Time to Peak Twitch, ms	122.6 \pm 13.1	153.2 \pm 10.8*
Half Relaxation Time, ms	94.8 \pm 12.9	108.2 \pm 13.3
Contraction Duration, ms	217.3 \pm 14.2	261.5 \pm 18.8*
Soleus M-wave Peak to Peak amplitude, mV	10.5 \pm 2.5	5.3 \pm 1.8*
MU Firing Rates 100% MVC, Hz	16.4 \pm 5.3	16.6 \pm 4.9
MU Firing Rates 75% MVC, Hz	14.9 \pm 4.2	14.2 \pm 4.1
Time to Task Failure, s	100.0 \pm 25.5	138.5 \pm 29.2*

Values are means \pm SD. All baseline values have been previously reported (Dalton et al. 2009). The maximal voluntary contraction (MVC) was weaker, time to peak twitch and contraction duration were slower, and time to task failure was longer for the old men compared to the young (* $P < 0.05$). The old men tended to have lower peak twitch torque compared to the young († $P = 0.08$). MU, motor unit.

collapsed between groups and compared over time. Peak twitch torque was unchanged immediately following task failure but increased to $129.7 \pm 18.7\%$ of the baseline values at 1 min following the fatigue task and persisted until 2 min ($125.9 \pm 17.2\%$). Peak twitch torque was recovered within 5 min. Time to peak twitch torque was shortened to $83.1 \pm 14.8\%$ of the baseline value for both age groups immediately following task failure and returned to baseline values by 1 min of recovery. Half-relaxation time was lengthened to $192.2 \pm 34.9\%$ of baseline values immediately following task failure and remained $113.2 \pm 15.8\%$ slower until 5 min of recovery. Contraction duration of the twitch was lengthened to $129.4 \pm 15.4\%$ of baseline values and remained elongated ≤ 2 min of recovery (Fig. 3). Maximal rate of torque development was $136.7 \pm 11.1\%$ faster than baseline values immediately following task failure and remained faster for ≤ 10 min of recovery ($110.7 \pm 7.3\%$). Maximal relaxation rate of the twitch slowed to $55.8 \pm 11.4\%$ of baseline values and did not recover until 5 min following task failure, but at 10 min of recovery, it was faster ($115.9 \pm 10.8\%$).

Motor unit properties

There was no main effect for age ($P = 0.15$ to $P = 0.27$) or time ($P = 0.31$ to $P = 0.45$) or interaction ($P = 0.20$ to $P = 0.34$) for M-wave properties (peak-to-peak amplitude, duration, and area) as a result of the fatigue task, despite a $\sim 50\%$ lower peak-to-peak amplitude ($P < 0.05$) of the baseline M-wave in the old compared with young men (Table 1). For example, M-wave amplitude ranged from 96.7 ± 6.1 to $101.1 \pm 11.0\%$ and 94.7 ± 10.9 to $101.9 \pm 14.2\%$ at task failure and throughout recovery for the old and young men, respectively. During the fatigue task, there was a main effect for age ($P < 0.05$) and time ($P < 0.01$) for normalized RMS of the soleus surface EMG. The normalized RMS was not changed by task failure in the old men, but it was reduced to $77.7 \pm 13.1\%$ by task failure compared with the beginning of the fatigue task in the young men. For the normalized RMS values of the soleus during the recovery MVCs, there was a main effect for age ($P < 0.05$) and time ($P < 0.01$). In the old men, RMS values during the recovery MVCs remained depressed for ≤ 5 min of recovery compared with the baseline MVC, but RMS values remained depressed for ≤ 10 min recovery for the young men. The old had higher RMS values at 5 min of recovery compared with the young (Fig. 4A). For the normalized RMS of the medial gastrocnemius during the fatigue task, there was only a main effect for time ($P < 0.01$). The medial gastrocnemius normalized RMS values were significantly reduced to $84.0 \pm 10.7\%$ by task failure in both age groups ($P < 0.05$). For the normalized medial gastrocnemius RMS values of the recovery MVCs, there was a main effect for age ($P < 0.05$) and time ($P < 0.01$). The normalized RMS values remained depressed for ≤ 5 min of recovery for the old and ≤ 10 min for the young. At 1 min of recovery, the old had higher maximal RMS values than the young (Fig. 4B). However, when the RMS data were normalized to task failure (data not shown), the RMS values recovered similarly in the old and young men.

A total of 424 MU trains in the old and 570 MU trains in the young were collected during the fatigue task, and 184 and 149 MVC MU trains, respectively, were collected during the four

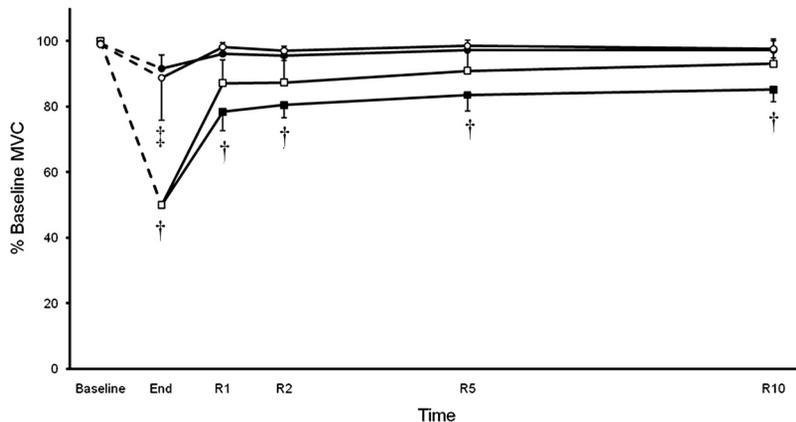


FIG. 2. Fatigue and recovery of maximal voluntary isometric contraction (MVC) and voluntary activation. ---, the fatigue task. Recovery at 1 (R1), 2 (R2), 5 (R5), and 10 (R10) min. The MVC for both the old (\square) and young (\blacksquare) men was reduced to 50% by task failure (End) and did not recover ($\dagger P < 0.05$). Voluntary activation for both the old (\circ) and young (\bullet) men was reduced similarly by $\sim 9\%$ at task failure and recovered within 1 min ($\ddagger P < 0.05$).

recovery time points. Each subject contributed ~ 83 MU trains during the fatigue task and ~ 28 MU trains during recovery. For MUFs during the fatigue task, there was only a main effect for time ($P < 0.01$). During the fatigue task, average MUFs starting at 75% MVC were progressively reduced in both the old and young men by 35 and 36%, respectively, with no age effect (Fig. 5). There were negative correlations between the linear decline in maximal MUFs and HRTs for both the old ($r = -0.75$) and young ($r = -0.63$) men for baseline and task failure values ($P < 0.05$; Fig. 6). The MUF values at task failure and during the recovery MVCs were also compared with values reported for a previous study of baseline MVC data from the same sessions (Dalton et al. 2009), and there was a main effect for time ($P < 0.01$) and age ($P < 0.01$) with a trend ($P = 0.07$) for an interaction. These MUFs were reduced by 45 and 43% at task failure in the old and young men, respectively. However, for the young men, MVC MUFs recovered within 1 min, but not until 5 min after task failure did these rates recover in the old group (Fig. 7). Furthermore, the old men had lower maximal MUFs than the young at 1 min of recovery.

DISCUSSION

The response of the aged neuromuscular system to fatigue has focused mainly on factors in the muscle related broadly to metabolism (Allman and Rice 2002; Kent-Braun 2009). Surprisingly few studies have explored possible differences in maximal motoneuron output (Christie and Kamen 2009; Rubinstein and Kamen 2005), and none previously have con-

currently assessed motoneuron output in relation to muscle contractile properties or these factors during a period of short-term recovery. The aged soleus in response to high-intensity fatigue and recovery provides a useful model and a unique experimental paradigm to assess the influence of neural output in relation to neuromuscular function. Our results show that despite similar decreases in MVC torque, MUFs, voluntary activation, and slowing of evoked contractile speed, the weaker old men demonstrated a longer time to task failure than the young. The recovery of maximal strength and contractile properties was similar for both groups but in neither was MVC recovered by 10 min. However, the recovery of an equal fatigue-induced reduction in MUFs was delayed in the old men compared with the young. This profile was not reflected in the surface EMG of the old for the soleus as evident by a smaller reduction in RMS amplitude than the young and therefore recovery of surface EMG was faster. These results indicate that during high-intensity sustained fatigue, the neuromuscular system responds similarly in old and young subjects, but despite similar recovery in contractile characteristics, motoneuron output remains depressed in the old men. This may suggest that recovery of soleus spinal motoneuron output is delayed in aged subjects due to central drive insufficiencies following a high-intensity isometric sustained task.

Fatigue

The old men were less fatigable than the young (~ 139 and ~ 100 s, respectively), which is a similar finding to previous studies with isometric tasks (Callahan et al. 2009; Hunter et al.

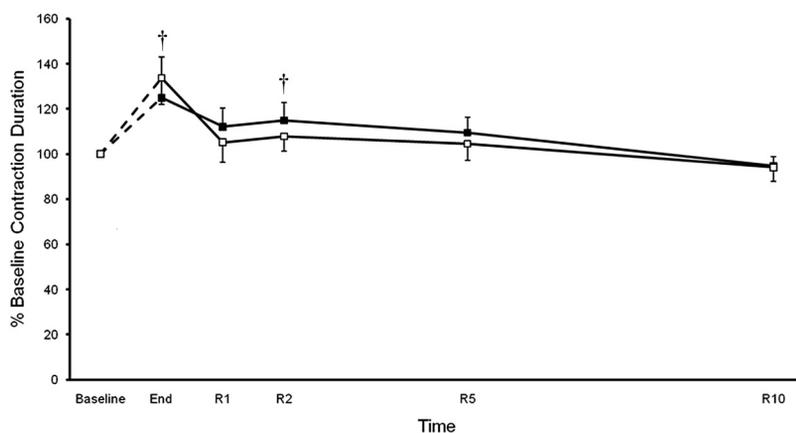


FIG. 3. Fatigue and recovery of twitch contraction duration. ---, the fatigue task. Recovery at 1 (R1), 2 (R2), 5 (R5), and 10 (R10) min. Contraction duration of the twitch was lengthened similarly in both the old (\square) and young (\blacksquare) men ($\dagger P < 0.05$).

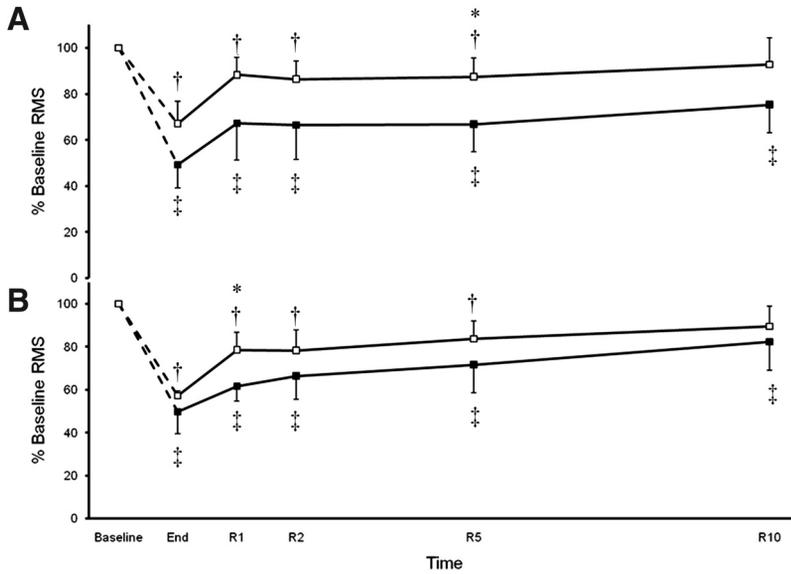


FIG. 4. Fatigue and recovery of root mean square (RMS) amplitude of the soleus and medial gastrocnemius. - - -, the fatigue task. Recovery at 1 (R1), 2 (R2), 5 (R5), and 10 (R10) min. The old men (□) had lower RMS values for the soleus (A) and medial gastrocnemius (B) throughout 5 min of recovery compared with baseline values († $P < 0.05$). The young men (■) had lower RMS values for the soleus and medial gastrocnemius throughout 10 min of recovery compared with baseline values († $P < 0.05$). The old men had greater RMS values at 5 min of recovery for the soleus and 1 min of recovery for the medial gastrocnemius compared with the young (* $P < 0.05$).

2004; Mademli and Arampatzis 2008a). These reports suggest that with isometric contractions, the age-related differences in fatigue are due mainly to peripheral factors, although specific mechanisms may depend on the task and measures used (Enoka and Duchateau 2008). In the present study, electrophysiologic indices indicate that peripheral mechanisms were fatigued to the same extent despite longer time to task failure for the old men compared with the young. For example, for both groups, maximal relaxation rate and HRT of the twitch slowed by ~45 and ~92%, respectively, from baseline, and each group exhibited a ~17% faster time to peak twitch and a ~27% greater peak twitch torque immediately following task failure. The faster and larger twitch responses after fatigue indicate muscle potentiation, which has been reported previously, but only for young subjects in this muscle group (Behm and St-Pierre 1997; Kuchinad et al. 2004). Thus our results demonstrate that the relationship between fatigue and potentiation in the plantar flexors is maintained in the old men.

Furthermore, the old men had smaller M-wave amplitudes than the young. This age-related alteration may be attributed to desynchronization of soleus motoneurons or impairment in muscle membrane excitability (Scaglioni et al. 2003). However, the disparity in fatigability between both age groups cannot be explained by differences in muscle membrane excitability because there were no changes in the M-wave prop-

erties for either group immediately following the task and throughout recovery. Although not documented previously for the plantar flexors, this finding is similar to previous reports from other muscle groups (Bilodeau et al. 2001; Lanza et al. 2004).

In young adults, Macefield et al. (2000) reported that MUFRRs did not significantly decrease in the extensor hallucis longus with a high-intensity sustained fatigue task (100% MVC), and they suggested that this was related to this muscle's high composition of slow twitch muscle fibers. In contrast, Kuchinad et al. (2004) reported a decrease in soleus (>80% slow twitch) MUFRRs in young adults as a result of moderate to high-intensity fatigue (sustained >40% MVC). It is unclear why the long toe extensor muscle did not show the usual fatigued-induced reduction in MUFRRs (Macefield et al. 2000), but with our high-intensity fatigue task in the soleus, we found that average MUFRRs were reduced and in both age groups. Thus despite an already slow muscle (Bellemare et al. 1983), which has a limited range in rate coding (Dalton et al. 2009; Oya et al. 2009), average high-intensity MUFRRs were reduced by 34%. Additionally, voluntary activation in both age groups was reduced by ~10%, which is a similar finding to previous studies focused on ageing in this (Mademli and Arampatzis 2008a) and other muscle groups (Bilodeau et al. 2001). However, it seems that the small loss of voluntary activation cannot

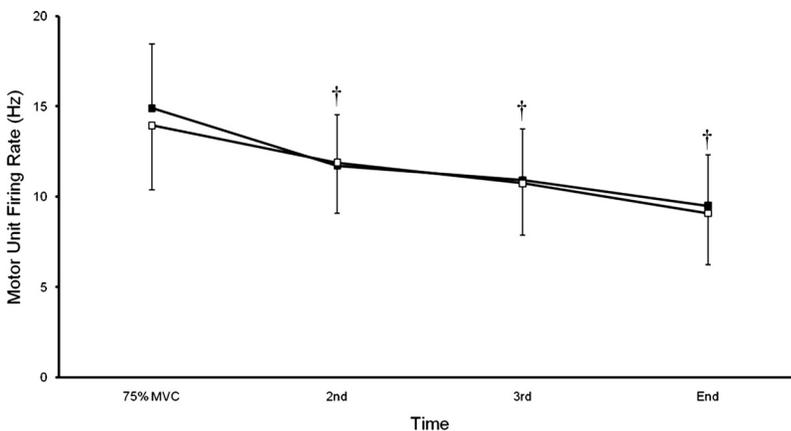


FIG. 5. Motor unit firing rates during the fatigue task. Motor unit firing rates were similarly lower throughout the fatigue task compared with the beginning (75% MVC) for the old (□) and young (■) men († $P < 0.05$).

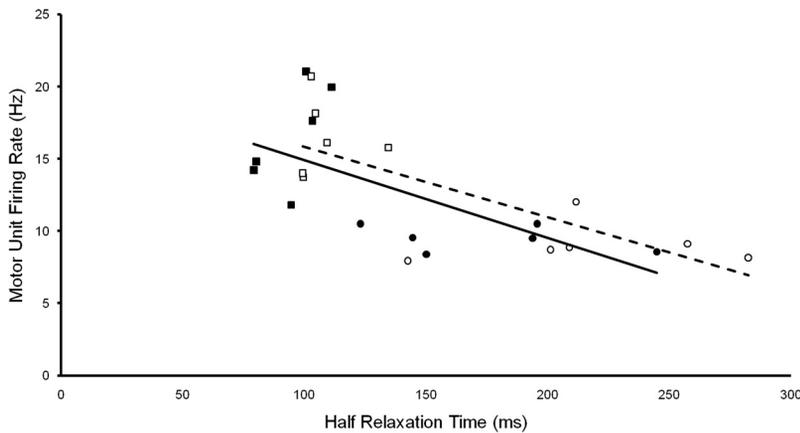


FIG. 6. The relationship for individual mean half relaxation times and individual mean motor unit firing rates (MUFRRs) collected at 100% MVC (baseline; \square , \square) and at the end of the fatigue task (\bullet , \circ) for old (\square , \circ) and young (\blacksquare , \bullet) men. Pearson correlation coefficients (r) were $r = -0.75$ and -0.63 for the old and young men, respectively ($P < 0.05$). The R^2 values were 0.56 and 0.40 for the old (---) and young (—) men, respectively ($P < 0.05$).

fully explain the large $\sim 34\%$ reduction in MUFRRs of the old and young men. These rates may have been reduced by mechanisms that are insensitive to the measure of voluntary activation, including reductions in central drive (Hunter et al. 2008), muscle spindle disfacilitation (Macefield et al. 1991), increase in group III and IV afferent feedback activity (Woods et al. 1987), or alterations in the intrinsic properties of the motoneuron (Carpentier et al. 2001).

In addition to a decline in MUFRRs, this type of fatigue usually is noted by a concomitant slowing in muscle contractile speed (Bigland-Ritchie and Woods 1984; Bigland-Ritchie et al. 1983b; Kuchinad et al. 2004). Despite a few studies (Christie and Kamen 2009; Rubinstein and Kamen 2005) that have documented decreases in MUFRRs in old subjects following high-intensity fatigue, with equivocal results, there are no reports investigating the relationship between contractile properties and MUFRRs during and following such a task. In this study, we found that at task failure, HRT was lengthened by $\sim 90\%$ of prefatigue values in both age groups with a concomitant reduction in MUFRRs. Although the concept of muscle wisdom has been questioned (Fuglevand and Keen 2003), the moderate to strong correlations ($r = -0.75$ and -0.63 , old and young, respectively) suggest that a compensatory mechanism in which MUFRRs decrease during a sustained task to match the slowing in contractile properties of the muscle to retain efficient torque production (Bigland-Ritchie et al. 1983b; Kuchinad et al. 2004), is maintained in the aged muscle (Fig. 6).

Despite a better maintenance of muscle mass and functional MUs in the soleus of old men well into their eighth decade (Dalton et al. 2008; Morse et al. 2005) compared with other

limb muscles tested (McNeil et al. 2005; Morse et al. 2005), the greater fatigue resistance for the old men compared with the young likely has a metabolic basis. Because the old men had weaker absolute plantar flexion strength than the young, it is reasonable to suggest they may have lower intramuscular pressures and less blood occlusion to the working muscles leading to less fatigue (Hunter et al. 2004, 2008) during this task. Our old adults were able to maintain the target torque of 75% MVC for a 65% longer duration than the young; this probably indicates that the young men were working at a maximal effort sooner than the old, leading to a shorter time to task failure. It has previously been proposed that due to shorter fascicle lengths in older adults, there is a lower ratio between active muscle volume and active cross-sectional area at least in the medial gastrocnemius, providing an energetic advantage for the old adults during isometric contractions at the same ankle angle compared with young adults (Mademli and Arampatzis 2008b). Therefore the improved fatigue resistance could be related to reduced metabolic byproducts harmful to muscle contraction in the old men. Support for this is found in the RMS data in which the old men maintained higher relative RMS amplitudes for the soleus than the young by the end of the fatigue task, suggesting that MUs in the soleus of the old men were more fatigue resistant. Thus the older individual may have greater fatigue resistance due to greater reliance on oxidative phosphorylation and a lower glycolytic flux, leading to less acidosis, and lower accumulation of P_i and $H_2PO_4^-$ (Kent-Braun 2009).

To minimize the contribution of the gastrocnemius, the knee angle was flexed to 90° . Based on magnetic resonance imaging in healthy subjects, the soleus was estimated to contribute

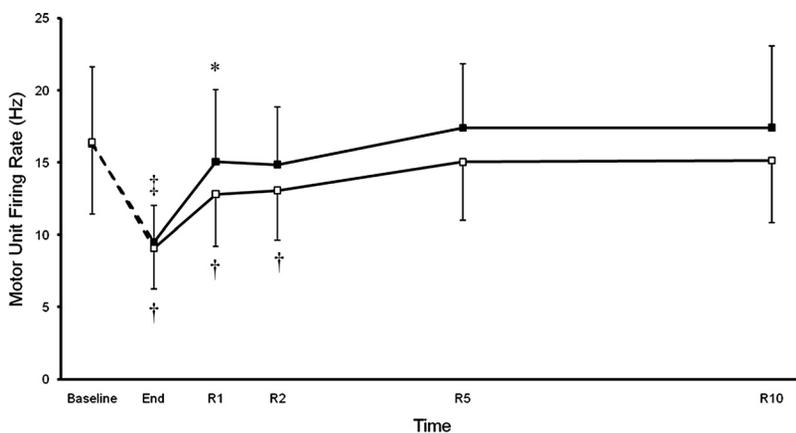


FIG. 7. Fatigue and recovery of maximal MUFRRs. ---, the fatigue task. Recovery at 1 (R1), 2 (R2), 5 (R5), and 10 (R10) min. Mean maximal MUFRRs were lower by task failure (End) and did not recover in the old men (\square) until 5 min of recovery ($\ddagger P < 0.05$) but was recovered in the young (\blacksquare) men by 1 min ($\ddagger P < 0.05$). The old had lower MUFRRs at 1 min of recovery compared with the young men ($*P < 0.05$).

~70% of plantar flexion torque of the triceps surae in the extended knee position (Fukunaga et al. 1992). In the flexed knee position (90°), the contribution of the gastrocnemii to plantar flexion torque was further minimized as the two-joint gastrocnemii are more sensitive to length change than the soleus (Cresswell et al. 1995; Kawakami et al. 1998), thus allowing the soleus to be the predominant plantar flexor. Despite this, there was EMG activity in the gastrocnemii that decreased equally by ~16% for both age groups by the end of the fatigue task. However, it is unknown the degree to which gastrocnemii activity contributed to plantar flexor torque and indeed whether or how the interplay among all plantar flexors is affected by ageing.

Recovery

Our results show similar declines in MUFs between the two groups during fatigue but a delay in recovery of these MUFs in the old subjects compared with the young. Because the muscle contractile properties were equally recovered in both age groups, MUF recovery may be more related to alterations at the spinal or supraspinal levels rather than within the muscle. A recent study (Mademli and Arampatzis 2008a) reported similar recovery of voluntary activation between age groups in the plantar flexors, using the interpolated twitch technique, despite greater fatigue resistance in the old. However, in that study, the old had a lower voluntary activation prior to the fatigue task. In contrast, our results suggest there was no difference in voluntary activation at baseline and despite a small reduction (~10%) at task failure, voluntary activation was equally and fully recovered within 1 min. Perhaps due to the limitations of the interpolated twitch technique (Klass et al. 2007), particularly at high activation levels (>90%), this technique is not sensitive to differences in rate coding especially in a muscle that maintains recruitment of MUs even at high contraction levels (Oya et al. 2009). Because presumably all MUs are recruited at MVC, maximal MUFs are a more direct estimate of neural drive during fatigue and recovery. Support for our findings can be found in a recent report by Hunter et al. (2008), who studied central drive using transcranial magnetic stimulation (TMS) during an intermittent high-intensity isometric fatiguing task of the elbow flexors in old and young adults. In agreement with our study, they reported that the old were less fatigable than the young with no age-related difference in the small decrease in central activation at the end of each sustained MVC. The superimposed twitch evoked by TMS increased similarly by ~7.5% in both age groups. However, during recovery, central drive remained depressed longer for the old subjects compared with that of the young. Our results of a delay in recovery of MUFs in the old men corroborate this finding.

Slower MUF recovery in the old men compared with the young may be related to a decrease in excitability of intracortical pathways (Peinemann et al. 2001) that may not be expressed effectively until the system is altered or stressed (i.e., fatigued) and therefore unable to recover at the same rate as the young. The factors associated with the age-related delay in recovery of the CNS as expressed by greater motor-evoked potential size during sustained contractions (Hunter et al. 2008) and now reductions in maximal MUFs are not known but may be related to reductions in cortex size (Raz et al. 2007),

structural degradation of cortical neurons (Dickstein et al. 2007), reduced motor cortical excitability (Oliviero et al. 2006), a decline in effective connectivity between distant motor-related cortical areas (Rowe et al. 2006) or age-related alterations at the spinal level (Kido et al. 2004; Sale and Semmler 2005; Scaglioni et al. 2003; Van Asseldonk et al. 2003).

Summary

When reduced to identical relative plantar flexion strength from a high-intensity sustained fatigue task, both age groups exhibited similar alterations in twitch contractile properties, muscle membrane excitability, and MUFs. This indicates that similar processes occurred during fatigue to reduce plantar flexion torque to 50% MVC. However, greater fatigue resistance and time to task failure in the old men may reflect a greater reliance on oxidative pathways and less metabolic byproducts compared with the young despite similar reductions in MUFs. Finally, the recovery of motoneuron output, as indicated by maximal MUFs, was delayed in the old men even though there was a similar recovery in all other measures. Thus our data suggest that overall, the old men are less fatigable than the young, but age-related alterations in spinal and supraspinal processes may be responsible for the delayed recovery of MUFs following a high-intensity fatiguing task in the soleus.

GRANTS

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