Changes in muscle activation can prolong the endurance time of a submaximal isometric contraction in humans

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Hunter, Sandra K., and Roger M. Enoka. Changes in muscle activation can prolong the endurance time of a submaximal isometric contraction in humans. J Appl Physiol 94: 108–118, 2003. First published September 13, 2002; 10.1152/japplphysiol.00635.2002.—Fourteen young subjects (7 men and 7 women) performed a fatiguing isometric contraction with the elbow flexor muscles at 20% of maximal voluntary contraction (MVC) force on three occasions. Endurance time for session 3 (1,718 ± 1,189 [SD] s) was longer than for session 1 (1,225 ± 683 s) and session 2 (1,410 ± 977 s). Five men and four women increased endurance time between session 1 and 3 by 60 ± 28% (responders), whereas two men and three women did not (−3 ± 11%; nonresponders). The MVC force was similar for the responders and nonresponders, both before and after the fatiguing contraction. Fatiguing contractions were characterized by an increase in the electromyogram (EMG) amplitude and number of bursts during the fatiguing contractions. The responders achieved a similar level of EMG at exhaustion but a reduced rate of increase in the EMG across sessions. The rate of increase in EMG across sessions declined for the nonresponders, but it remained greater than that of the responders. The increase in burst rate during the contractions declined across sessions with a negative relation between burst rate and endurance time (r = −0.42). Normalized force fluctuations increased during the fatiguing contractions, and there was a positive relation (r = 0.60) between the force fluctuations and burst rate. Changes in mean arterial pressure and heart rate during the fatiguing contraction were similar for the responders and nonresponders across the three sessions. The results indicate that those subjects who increased the endurance time of a submaximal contraction across three sessions did so by altering the level and pattern of muscle activation during the fatiguing contraction (2, 13). The existence of a deficit in activation at exhaustion suggests that endurance time could be prolonged if it is possible to alter the size of the deficit.

Such adjustments in motor unit activity during fatiguing contractions can be modified by chronic interventions. Our laboratory found, for example, that several weeks of limb immobilization alters the pattern of muscle activation and influences the endurance time during a submaximal contraction performed with the elbow flexor muscles (35, 36). When the fatiguing contraction was performed before the arm was immobilized in a plaster cast for 4 wk, all subjects exhibited the progressive increase in EMG during an isometric contraction that was sustained at 15% of the maximal voluntary contraction (MVC) force. Immediately after the cast was removed, 7 of the 12 subjects (6 women and 1 man) used intermittent muscle activation and a lower rate of increase in the EMG, and this was associated with a 220% increase in endurance time. The other five subjects, who were all men, used the same EMG pattern as they did before the intervention, and they experienced no change in endurance time.

These observations raised two questions: Does muscle activation change with repeat performances of the task, and is the effect related to the sex of the individual? The purpose of the study was to compare the patterns of muscle activation and endurance time during a submaximal fatiguing contraction of the elbow flexor muscles performed on three occasions by men and women. Because young women appear to use the biceps brachii muscle more often than young men during activities of daily living (21, 36), we expected to find that any adaptations in muscle activation would be greater for the women. As with the immobilization study, we found that some subjects did exhibit an adaptation in muscle activation. However, these responders included both men and women. A preliminary account of these results has been published in abstract form (18), and related data have been published elsewhere (17).

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METHODS
Fourteen healthy adults [7 men and 7 women; age 27 ± 4 yr (SD), range 22–35 yr] volunteered to participate in the study. All the subjects were right handed with no known neurological disorders and were naive to the experimental protocol and procedures. The physical activity levels of the subjects were estimated with a questionnaire that evaluates a 7-day history of activity (33). Before participation in the study, all subjects gave informed consent, and the Human Subjects Committee at the University of Colorado approved the protocol.

Mechanical Recording
Subjects were seated upright in an adjustable chair with the left arm abducted slightly and the elbow resting on a padded support, and the elbow joint was flexed to 90°. The hand and forearm were placed in a modified wrist-hand-thumb orthosis (Orthomerica, Newport Beach, CA), which held the forearm in a position midway between pronation and supination. The force exerted by the elbow flexor muscles in a sagittal plane was measured with a force transducer (Force-Moment Sensor, JR-3, Woodland, CA) that was mounted on a custom-designed, adjustable support. The orthosis was rigidly attached to the force transducer. The vertical force detected by the transducer was recorded on digital tape (DAT Sony PC 116, Sony Data Recording, Montvale, NJ) and displayed on a 14-in. computer monitor that was located at eye level 1 m in front of the subject.

Electrical Recordings
EMG signals were recorded with bipolar surface electrodes (Ag-AgCl, 8-mm diameter; 20-mm distance between electrodes) that were placed over the long head of biceps brachii, the short head of biceps brachii, brachioradialis, and the medial head of triceps brachii. Reference electrodes were placed on a bony prominence at the elbow or shoulder. The EMG of the brachialis muscle was measured with an intramuscular bipolar electrode inserted into the muscle ~3 cm proximal to the antecubital fold. The electrode comprised two stainless steel wires (100-μm diameter) that were completely insulated with Formvar (California Fine Wire, Grover Beach, CA) except for the cut ends. A surface electrode (8-mm diameter) placed on a bony prominence served as the reference electrode. The EMG signal was amplified (~500–2,000) and band-pass filtered (20–800 Hz for the surface EMG and 20–1,000 Hz for the intramuscular EMG; Coulbourn Instruments, Allentown, PA) before being recorded on digital tape and displayed on an oscilloscope.

Cardiovascular Measurements
Heart rate and blood pressure were monitored throughout the fatiguing contraction with an automated, beat-by-beat blood pressure monitor (Finapres 2300, Ohmeda, Madison, WI). The blood pressure cuff was placed around the middle finger of the right hand, and the arm was placed in a sling so that it was relaxed with the hand at heart level. The blood pressure signal was recorded on digital tape.

Experimental Protocol
Subjects were required to perform the protocol on three occasions, with 2 wk separating each session. Before the first experimental session, each subject visited the laboratory for an introductory session to become familiar with the equipment and the procedures, and each performed several trials of the MVC task. All subjects were naive to the experimental protocol and procedures before the familiarization session. The experiments for each subject were performed at the same time of the morning on each occasion. The protocol comprised 1) MVCs with the elbow flexor and elbow extensor muscles, 2) an isometric contraction with the elbow flexor muscles that was sustained at 20% MVC force until exhaustion, and 3) another MVC with the elbow flexor muscles immediately after the fatiguing contraction.

MVC force. Subjects performed three MVC trials with the elbow flexor muscles and three MVC trials with the elbow extensor muscles. The MVC task consisted of a gradual increase in force from zero to maximum over 3 s, with the maximal force held for 2–3 s. Subjects were able to observe the exerted force on a monitor and were verbally encouraged to achieve maximal force. Subjects rested for 60–90 s between trials. If the peak forces from two of the three trials were not within 5% of each other, additional trials were performed until this was accomplished. The greatest force achieved by the subject was taken as the MVC force and used as the reference to determine the target force for the fatiguing contraction. Subsequently, the MVC force for the elbow extensor muscles was determined by using the same procedures. Within 20 s of completion of the fatiguing contraction, an MVC was performed with the elbow flexor muscles.

Fatiguing contraction. The fatiguing contraction was performed at a target force of 20% MVC as determined from the MVC performed on that day. The absolute change in target force relative to session 1 was –0.7 ± 5.1 N and did not differ between the subjects who increased endurance time (responders) and those who did not (nonresponders) (Table 1). The subject was required to match the target force displayed on the monitor and was verbally encouraged to sustain the force for as long as possible. The contraction was terminated when the target force was <16% MVC force for ~3 s, despite strong verbal encouragement; this time was recorded as the endurance time. Subjects were not informed of the endurance times until completion of the third session.

The rating of perceived exertion (RPE) was recorded by using the modified Borg 10-point scale (3). The subjects were instructed to focus the assessment of effort on the arm muscles performing the task. The scale was anchored so that 0 represented the resting state and 10 corresponded to the strongest contraction that the arm muscles could possibly perform. The RPE was measured at regular time intervals.

Table 1. Target forces for the fatiguing contraction across the three sessions and the two groups of subjects

<table>
<thead>
<tr>
<th></th>
<th>Absolute Target Force, N</th>
<th></th>
<th>Relative Target Force, N</th>
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<tbody>
<tr>
<td></td>
<td>Session 1</td>
<td>Session 2</td>
<td>Session 3</td>
</tr>
<tr>
<td>Responders</td>
<td>9</td>
<td>58.4 ± 25.2</td>
<td>58.6 ± 23.7</td>
</tr>
<tr>
<td>Nonresponders</td>
<td>5</td>
<td>57.1 ± 31.8</td>
<td>58.4 ± 30.0</td>
</tr>
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Values are means ± SD; n, no. of subjects. Relative target forces indicate the difference between the 2 sessions averaged across the subjects in each group.
(1–5 min between recordings) during the fatiguing contraction. Once the subject had attained a score of 8 on the Borg scale, the RPE was recorded every minute.

Data Analysis

All data collected during the experiments were recorded on digital tape and subsequently digitized (analog-to-digital converter, 12-bit resolution) and analyzed off-line by using the Spike2 data-analysis system (Cambridge Electronic Design, Cambridge, UK). The force and blood pressure were digitized at 200 samples/s, whereas the EMG signals were digitized at 2,000 samples/s.

The MVC force was quantified as the average value over a 0.5-s interval that was centered about the peak force. Similarly, the maximal EMG for each muscle was determined as the average value over a 0.5-s interval that was centered about the peak rectified EMG.

The amplitude of the force fluctuations was quantified as the coefficient of variation (CV = SD/mean × 100) of the first, middle, and last 60 s of the fatiguing contraction. To compare the force fluctuations at the same absolute time across the three sessions, the CV was calculated for five 30-s epochs at the same absolute time relative to session 1 for each subject. The five 30-s epochs were the first 30 s; 15 s on both sides of the time at 25, 50, and 75% of endurance time; and the last 30 s of the endurance time for session 1.

The average EMG during the fatiguing contraction was quantified in two ways: 1) for statistical analyses, as averages of the rectified EMG (AEMG) for the first, middle, and last 60 s of the fatiguing contraction; and 2) for graphic presentation, as AEMG for every 1% of the endurance time. The EMG for each muscle was normalized to the maximum EMG (percentage of MVC) obtained during its respective MVC. The normalized EMGs for the short and long heads of biceps brachii did not differ during the fatiguing contraction on the basis of a statistical comparison; hence the two signals were averaged to yield a single value for biceps brachii.

Because low-force fatiguing contractions of long duration are characterized by bursts of EMG activity, we quantified the number and duration of these bursts throughout the fatiguing contraction for each muscle (Fig. 1). The rectified EMG signal was 1) smoothed with a low-pass filter at 2 Hz for surface EMG signals and at 3.8 Hz for the intramuscular EMG (brachialis), 2) differentiated to identify rapid changes in the EMG signal, and 3) divided by the AEMG so that muscles with different EMG amplitudes could be compared. A burst was identified when the smoothed, differentiated EMG signal increased by >0.4 s⁻¹ for the surface EMG and 0.36 s⁻¹ for the intramuscular EMG. These values represented 3 SDs above the mean of the smoothed, differentiated EMG signal that had been calculated from 25 samples when the EMG signal displayed minimal bursting during the contraction. The end of a burst was identified as the time when the smoothed EMG signal decreased to the same amplitude as at the start of the burst. When the EMG signal did not decline to the same EMG amplitude at the start of the burst, however, the end of the burst was then identified as the time that the smoothed, differentiated EMG signal was most negative before the start of the next burst. This criterion represented the time at which the signal decreased most rapidly before the beginning of the next burst. Based on pilot observations, the start of two bursts was constrained to be >2 s apart and the minimum burst duration was 0.5 s. Burst rate and duration were compared at relative and absolute times during the fatiguing contractions.

Heart rate and mean arterial pressure during the fatiguing contraction were analyzed by comparing 10- to 15-s averages at 10% intervals throughout the endurance time. For each interval, the blood pressure signal was analyzed for the mean peaks (systolic blood pressure [SBP]), mean troughs (diastolic blood pressure [DBP]), and the number of pulses per second (multiplied by 60 to determine heart rate). Mean arterial pressure (MAP) was calculated for each epoch with the following equation: \( \text{MAP} = \text{DBP} + \frac{1}{3} \times \text{SBP} - \text{DBP} \).

Statistical Analysis

Data are reported as means ± SD within the text and displayed as means ± SE in the figures. On the basis of the distribution of the change in endurance time, subjects were categorized as responders and nonresponders. The responders (5 men and 4 women) increased endurance time over the three sessions by >15% from session 1. The assignment of subjects to the responder and nonresponder groups was based on cluster analysis (SPSS, Chicago, IL) and the pattern of change in endurance time across the three sessions. The responders group initially included those subjects who changed endurance time by ≥15% after cluster analysis. However, we included the subject who improved her endurance time by 15% into the nonresponders group after inspection of the pattern of change in endurance time across the three sessions. A two-factor (response × session) ANOVA (StatView, SAS Institute) was used to compare the dependent variables for endurance time, and changes in heart rate and MAP for the fatiguing contraction. A three-factor ANOVA (response × session × time) was used to compare the
dependent variables for heart rate and MAP, MVC force, AEMG, RPE, and force fluctuations during the fatiguing contraction and the force fluctuations at the same absolute time in the three sessions.

A four-factor (muscle × response × session × time) ANOVA was used to compare the AEMG of the elbow flexor muscles and burst rate and burst rate at the same absolute time of session 1. Because bursts were absent during some time periods for most subjects, averages of the burst duration are reported and the results of independent t-tests reported where these analyses were possible. Post hoc analyses (Tukey-Kramer) were used to test for differences when appropriate. A significance level of \( P < 0.05 \) was used to identify statistical significance.

RESULTS

The purpose of the study was to compare changes in muscle activation and endurance time across repeat performances of a fatiguing contraction. Because the changes in endurance time comprised a bimodal distribution, we categorized the subjects into those who increased endurance time across sessions (responders) and those who did not (nonresponders) (Fig. 2A). Accordingly, we first describe the changes in endurance time for these two groups of subjects before examining the associated changes in muscle activation.

The responders (5 men and 4 women) increased endurance time from session 1 to session 3 by >15% [60 ± 28% (SD), range 25–115%], whereas the nonresponders (2 men and 3 women) did not experience a change in endurance time (−3 ± 11%, −12–15%). The endurance times for session 1 were similar for the responders and nonresponders (1,265 ± 753 vs. 1,153 ± 607 s; \( P > 0.05 \)). An interaction between session and response (\( P < 0.05 \)) indicated that the responders significantly increased endurance time from session 1 to session 3 (Fig. 2B). The estimated physical activity levels were not different for the responders (49.7 ± 11.0 kcal·day\(^{-1}\)·kg body mass\(^{-1}\)) and the nonresponders (44.5 ± 8.6 kcal·day\(^{-1}\)·kg body mass\(^{-1}\)).

The MVC force was similar for the responders (289 ± 117 N) and nonresponders (290 ± 140 N) before the fatiguing contraction and across sessions (\( P > 0.05 \)). The MVC force after the fatiguing contraction was reduced to similar levels for both the responders (35.9 ± 18.8%) and the nonresponders (31.3 ± 16.8%; \( P > 0.05 \)), and this decrease was consistent across sessions (Fig. 3, A and B).

RPE

RPE increased during the fatiguing contraction for both groups of subjects (\( P < 0.05 \)). The responders had a lower RPE at the start and midpoint of the fatiguing contraction for session 2 (1.3 ± 0.7 and 6.9 ± 1.6) and session 3 (1.3 ± 0.9, 7.7 ± 1.4) compared with session 1 (1.7 ± 1.0 and 8.2 ± 1.2; \( P < 0.05 \)). Conversely, the nonresponders had a similar RPE at the start of the contraction for the three sessions (1.8 ± 0.8, 1.9 ± 1.0, and 1.8 ± 0.8, respectively), but at the midpoint of the contraction it increased from session 1 (7.3 ± 1.2) to sessions 2 (8.1 ± 1.2) and 3 (7.7 ± 1.2). At exhaustion, all subjects had achieved a rating of 10.

Fig. 2. A: percent change in endurance time between sessions 1 and 3 for individual subjects assigned as either responders (\( n = 9 \)) or nonresponders (\( n = 5 \)). B: mean (± SE) endurance time for the fatiguing contractions across 3 sessions for the responders (\( n = 5 \) men, 4 women) and nonresponders (\( n = 2 \) men, 3 women). Endurance times for the responders and nonresponders were similar for session 1, but the responders increased endurance times in sessions 2 and 3 (\( P < 0.05 \)).
Adjustments in AEMG

The amplitude of the normalized AEMG for all elbow flexor muscles increased progressively during the fatiguing contractions for the responders and nonresponders in all sessions (P < 0.05; Figs. 4 and 5). The increase in the normalized AEMG at exhaustion for the elbow flexor muscles during the fatiguing contraction, however, was less in session 3 compared with sessions 1 and 2 (interaction of time and session; P < 0.05). An interaction between time, session, and response indicated that the reduced increase in AEMG was specific to the nonresponders (P < 0.05), due to an attenuation of the marked increase in the terminal AEMG for biceps brachii and brachioradialis (P < 0.05; Fig. 5, D–F). For the responders, however, the AEMG achieved similar values at exhaustion across all three sessions (P < 0.05; Fig. 5, A–C).

Because endurance time increased across sessions for the responders but reached comparable terminal AEMG values, the rate of increase in AEMG declined across sessions (P < 0.05). For the responders, the average rate of increase in AEMG for the elbow flexor muscles was 0.87 ± 0.54%/min for session 1, 0.78 ± 0.64%/min for session 2, and 0.68 ± 0.62%/min for session 3. Furthermore, the average rate of increase in AEMG for nonresponders was greater than that for responders across sessions (P < 0.05). For the nonresponders, the average rate of increase in AEMG for the elbow flexor muscles was 2.00 ± 2.04%/min for session 1, 1.76 ± 1.57%/min for session 2, and 1.31 ± 1.81%/min for session 3. These rates for the nonresponders were influenced by the marked increase in terminal AEMG exhibited by some muscles (Fig. 5, D–F). For example, the average rate of increase in AEMG for the elbow flexor muscles of the nonresponders over the first 600 s was 1.23 ± 0.95%/min for session 1, 1.01 ± 0.95%/min for session 2, and 0.52 ± 0.61%/min for session 3.

The average rates of increase in AEMG varied among the individual muscles. For both groups of subjects, there was a close association between the AEMG values for biceps brachii and brachioradialis, except for the nonresponders near the end of the task in sessions 1 and 2 (Fig. 5, D and E). Similarly, the brachialis AEMG consistently began at a greater value (18 ± 6%) compared with the biceps brachii and brachioradialis (11 ± 3%), ended at a similar value (31 ± 10%), and increased at a lower average rate (P < 0.05). Nonethe-
less, there were differences among the muscles in the increase in AEMG of the individual muscles for the two groups of subjects. For the responders (Fig. 5, A–C), the average rate of increase in AEMG across sessions was similar for brachialis (0.60 ± 0.70%/min), whereas the average rate for biceps brachii and brachioradialis declined from session 1 (1.09 ± 0.50 and 0.96 ± 0.51%/min, respectively) to session 3 (0.66 ± 0.55 and 0.71 ± 0.62%/min, respectively). Although the average rate of increase in AEMG also declined for the nonresponders across sessions (Fig. 5, D–F), the rates did not achieve values similar to the responders, except for the brachialis muscle in session 3 (0.52 ± 0.69%/min). For example, the average rate of increase in AEMG of the nonresponders in session 3 was 2.19 ± 2.97%/min for biceps brachii and 1.22 ± 0.95%/min for brachioradialis. Qualitatively, the AEMG for the nonresponders appeared to change in all three sessions from the progressive linear increase at ~600 s when the AEMG reached ~20% MVC.

Compared with the elbow flexor muscles, the change in AEMG for the triceps brachii during the fatiguing contractions was much less (P < 0.05) and was similar for the responders and nonresponders (P > 0.05). The average values of the triceps brachii AEMG across all three sessions was 1.67 ± 0.93% for the first 60 s and increased to 3.78 ± 2.10% for the last 60 s (P < 0.05). This increase in triceps brachii AEMG (Fig. 5) was similar across sessions for both the responders (0.10 ± 0.10%/min) and nonresponders (0.17 ± 0.14%/min; P > 0.05). There was, however, a significant interaction between session and time, which indicated that the rate of increase in triceps brachii AEMG declined across sessions for both the responders and nonresponders. The average rate of increase in triceps brachii AEMG was 0.17 ± 0.13%/min for session 1 compared with 0.07 ± 0.05%/min for session 3.

Bursting of EMG Activity

Fatiguing contractions were characterized by an increase in the number of EMG bursts with time during the fatiguing contraction. For example, the number of bursts identified in the records of one subject in session 1 (Fig. 4A) were 39 for brachialis, 18 for brachioradialis, 0 for the long head of biceps brachii, and 41 for the short head of biceps brachii. These counts corresponded to burst rates of 2.96, 1.37, 0, and 3.11 bursts/min, respectively. When this subject performed the task in session 3 (Fig. 4B), the burst count was 31 for brachialis, 34 for brachioradialis, 19 for the long head of biceps brachii, and 70 for the short head of biceps brachii, which corresponded to rates of 1.10, 1.20, 0.67, and 2.48 bursts/min, respectively.

The burst rate for all elbow flexor muscles and the triceps brachii increased progressively during the fatiguing contractions in all sessions for both the responders and nonresponders (P < 0.05; Fig. 6A). Burst rate was similar across all muscles (P > 0.05) and similar for responders and nonresponders (P > 0.05). There was, however, an interaction between time and session because the burst rate of the elbow flexor muscles in the last third of the endurance time of sessions 2 and 3 was less than that for session 1 (P < 0.05; Fig. 6A).
reduction in burst rate across sessions such that the burst rate at the same absolute time was less in sessions 2 and 3 compared with session 1 (Fig. 6B; \( P < 0.05 \)). There was an interaction between session and time (\( P < 0.05 \)) because burst rate was less for sessions 2 and 3 during the middle and last thirds of the endurance time (on the basis of the endurance time of session 1) (Fig. 6B; \( P < 0.05 \)). Burst rate was similar for the elbow flexor muscles during sessions 2 and 3 (\( P > 0.05 \)) when compared at the same absolute time of session 1. In contrast, the responders showed a greater reduction in average burst rate across sessions compared with the nonresponders at the same absolute time of session 1 (Fig. 6C; \( P < 0.05 \)). There was a significant negative relation (\( r = -0.42; \ P < 0.05; \ n = 42 \)) between endurance time and burst rate, which meant that longer endurance times were associated with a reduction in the rate of bursts in the AEMG.

These differences in burst rate were attributable to the two groups of subjects producing similar numbers of EMG bursts. Across all three sessions, the average number of bursts was 19 ± 29 for the responders and 10 ± 26 for the nonresponders (\( P > 0.05 \)). Furthermore, the burst number did not change across sessions for the responders (21 ± 39, 17 ± 23, and 20 ± 24; \( P > 0.05 \)) and the nonresponders (13 ± 15, 7 ± 8, 10 ± 11; \( P > 0.05 \)). Because the responders increased endurance time, they were therefore able to delay the bursts of EMG activity.

The mean burst duration during the fatiguing contractions and across the three sessions was 5.8 ± 7.2 s. There was an increase in burst duration across the sessions for the elbow flexor muscles: session 1 (4.4 ± 2.2 s) was less than session 2 (7.8 ± 10.0 s; \( P = 0.02 \)), and there was a trend toward significance in session 3 (6.6 ± 7.8 s; \( P = 0.08 \)). The burst duration across sessions was similar for the responders (6.5 ± 9.1 s) and nonresponders (5.7 ± 4.7 s; \( P > 0.05 \)), and the two groups showed a similar burst duration across the three sessions. Furthermore, the burst duration of the elbow flexor muscles was similar over the three sessions (\( P > 0.05 \)) short head of biceps brachii (6.6 ± 8.1 s), long head of biceps brachii (5.9 ± 7.4 s), brachioradialis (7.0 ± 7.2 s), and brachialis (6.9 ± 11.5 s). The burst duration for triceps brachii was similar across sessions (3.4 ± 1.5, 4.1 ± 1.6, and 4.1 ± 2.6 s; \( P > 0.05 \)) and similar between responders (4.1 ± 2.3 s) and nonresponders (3.7 ± 1.5 s; \( P > 0.05 \)). The burst duration for triceps brachii (3.8 ± 1.9 s) tended to be briefer (\( P = 0.08 \)) than that for the elbow flexor muscles (6.2 ± 7.8 s).

**Force Fluctuations**

The amplitude of the normalized force fluctuations (CV for force) increased progressively during the fatiguing contractions for the responders and nonresponders (\( P < 0.05 \); Fig. 7, A and B). There was an interaction between session and time (\( P < 0.05 \)) due to less of an increase in the CV during the fatiguing contraction for session 3 (8.8 ± 3.9% at exhaustion)

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Fig. 6. Burst rate of the rectified EMG of the elbow flexor muscles for the fatiguing contractions. Values are means ± SE. There was no difference in the burst rate between the elbow flexor muscles. A: average burst rates for the first third, middle third, and last third of the endurance time for sessions 1–3. There was an increase in the burst rate during the fatiguing contraction (\( P < 0.05 \)) for all 3 sessions. However, an interaction between session and time indicated that the burst rate in the last third of the endurance time was less in sessions 2 and 3 compared with session 1. B: average burst rates for sessions 1–3 at the same absolute endurance time of session 1. An interaction between session and time indicated that the burst rates in the middle third and last third of the endurance time in sessions 2 and 3 (on the basis of session 1) were less than that in session 1. C: average burst rates for the responders and nonresponders for sessions 1–3 over the same duration of endurance time of session 1. An interaction between session and response indicated a greater reduction in the burst rate across sessions for the responders compared with the nonresponders.
compared with session 1 (11.3 ± 6.5% at exhaustion). However, the nonresponders had larger increases in the CV compared with the responders at the end of the fatiguing contraction for session 1 (15.2 ± 8.5 vs. 9.1 ± 4.1% at exhaustion) but similar increases to the responders for sessions 2 and 3 (9.2 ± 3.8 vs. 9.7 ± 2.7% at exhaustion). Conversely, the responders had similar increases in the CV across the three sessions (8.7 ± 4.2% at exhaustion), and these values were similar to those of the nonresponders for sessions 2 and 3. There was a significant positive relation \((r = 0.60; P < 0.05; n = 42)\) between the CV and burst rate, which indicated that the increase in the CV was related to an increase in the rate of bursts in the AEMG.

The CV for force was compared between the responders and nonresponders at the same absolute time across sessions on the basis of the endurance time of session 1 for each subject. At the start of the fatiguing contraction, the CV for force in sessions 1–3 was similar for the responders (2.7 ± 1.0%) and nonresponders (2.7 ± 0.5%; \(P > 0.05\)). At the endurance time of session 1, however, the CV for sessions 2 and 3 was less than for session 1 (\(P < 0.05\)) for both the responders and nonresponders. The CV for the responders at the endurance time of session 1 was 9.3 ± 4.7% for session 1, 6.8 ± 3.0% for session 2, and 4.7 ± 1.9% for session 3. The CV for the nonresponders at the endurance time of session 1 was 17.2 ± 10.2% for session 1, 8.6 ± 3.6% for session 2, and 9.9 ± 3.0% for session 3. The attenuation of the increase in the CV for force across sessions was greater for the responders compared with the nonresponders (\(P < 0.05\)).

**MAP and Heart Rate**

MAP increased similarly for responders and nonresponders during the fatiguing contraction (\(P < 0.05\)) across all three sessions, although less during session 3 (\(P < 0.05\)) for both the responders and nonresponders. The MAP was similar for the responders and nonresponders at rest (83.5 ± 4.3 vs. 82.0 ± 6.0 mmHg), at the start of fatiguing contraction (103.9 ± 8.2 vs. 105.8 ± 8.6 mmHg), and at exhaustion (130.9 ± 19.0 vs. 140.8 ± 18.3 mmHg).

Heart rate increased during the fatiguing contraction (\(P < 0.05\)), with a greater increase in session 1 compared with sessions 2 and 3, for both responders and nonresponders (\(P < 0.05\)). Heart rate was less for responders compared with nonresponders throughout the fatiguing contraction (\(P < 0.05\)), and this was consistent across all three sessions. At rest, heart rate was similar for responders and nonresponders (61 ± 9 vs. 67 ± 8 beats/min; \(P = 0.22\)). At the start of the contraction, the heart rate for responders was less than that of nonresponders (70 ± 9 vs. 82 ± 7 beats/min; \(P < 0.05\)) and also at exhaustion (95 ± 16 vs. 110 ± 12 beats/min; \(P < 0.05\)). The change in heart rate from the beginning to the end of the fatiguing contraction was similar for responders and nonresponders (35 ± 22 vs. 37 ± 10%; \(P > 0.05\)).

**DISCUSSION**

The purpose of this study was to compare the endurance time and muscle activation patterns of men and women during a fatiguing contraction performed on three occasions with the elbow flexor muscles. Endurance time increased across the three sessions for some men and women (responders) but not others (nonresponders). The responders exhibited a reduced rate of increase in muscle activation across sessions but similar levels of activation at exhaustion. Although the nonresponders displayed a significant reduction in the rate of increase in muscle activation across sessions, the rate of increase in AEMG was greater than that for the responders. The interference EMG comprised bursts of activity, which increased in number during the fatiguing contraction for both the responders and nonresponders. However, the increase in burst rate during the contraction declined across sessions, and there was a significant negative relation between burst rate and endurance time. In contrast to these differences in muscle activation, the pressor response was similar for the responders and nonresponders across sessions. Consequently, alterations in the level and pattern of muscle activation and not cardiovascular adjustments were associated with changes in endurance time for fatiguing contractions performed with the elbow flexor muscles. Furthermore, alterations in
muscle activation and endurance time were not specific to either the sex or the strength (MVC force) of the individual.

**Comparable Fatigue for the Responders and Nonresponders**

The endurance time increased across the three sessions for 9 of the 14 subjects, and this was independent of the sex of the individual. Those subjects who exhibited this increase were denoted as responders (5 men and 4 women), and those who did not were identified as nonresponders (2 men and 3 women). Despite the progressive increase in endurance time across sessions for the responders, the two groups of subjects experienced comparable RPE scores at exhaustion and similar reductions in MVC force after the fatiguing contractions. These results suggest that the responders and nonresponders experienced similar amounts of fatigue across the three sessions.

Previous analyses of these data (17) have indicated that women had a longer endurance time than men and that this difference was associated with both a lower absolute target force during the sustained contraction and a reduced pressor response. The sex difference in endurance time, target force, and pressor response was consistent across sessions. In the present analysis, however, both men and women demonstrated the adaptations in endurance time across sessions while maintaining a constant target force across the three sessions. Thus, despite the acute adjustments over the three sessions for some men and women (responders) when performing the contraction at the same target force, the sex difference in endurance time persisted. These results indicate that the relatively acute adaptation of endurance time across sessions was independent of the sex of the subject and also the target force (Table 1). Consequently, the mechanisms underlying the longer endurance time of women compared with men must be different from those responsible for the increases in endurance time exhibited by the responders.

**Adaptations of Endurance Time Were Not Associated With the Pressor Response**

To understand the differential changes in endurance time across sessions, we examined the relative contributions of muscle activation and the pressor response. The pressor response is a reflex-mediated adjustment in MAP that attempts to rectify the mismatch between perfusion and muscle metabolism during an isometric contraction (28, 31, 32). Both the central command and peripheral reflexes are involved in mediating the pressor response (1, 12, 28, 31, 32), although the reflex is largely driven by the metaboreflex and is indicative of the metabolic state of the active muscle (19, 20, 34).

In this present study, both the responders and nonresponders had a reduced increase in the MAP and heart rate in session 3 compared with sessions 1 and 2. This reduction in the rate of increase in the pressor response suggests there was an adaptation in the central command or the metaboreflex in session 3 for both groups of subjects. Because the increase in MAP and heart rate was similar for the responders and nonresponders, however, the increase in the endurance time for the responders across sessions was independent of the adaptations in the pressor response. Consequently, the differential changes in endurance time for the responders and nonresponders could not be explained by circulatory adjustments or the metabolic state of the muscle. Furthermore, the mechanism for the sex difference in endurance time (17) that was related to the pressor response and the target force exerted was consistent across the three sessions and must be different from that for the increase in the endurance time of the responders.

**Muscle Activation Differed for Responders and Nonresponders**

Previous reports have indicated that when a submaximal contraction is sustained for as long as possible, the AEMG at exhaustion is often less than the value recorded during a MVC (2, 13, 27). Furthermore, the magnitude of the deficit is greater for low-force contractions (8, 13, 25, 39). The present results show that details of the EMG activity during a submaximal contraction can vary with practice and that these adjustments are associated with changes in endurance time. The adjustments included changes in the rate of increase in AEMG, the rate of bursts in EMG activity, and the distribution of EMG activity among the synergist muscles. The magnitude of the AEMG at exhaustion, however, did not vary across repeat performances of the contraction for the responders despite prolongation of the task.

We assessed triceps brachii activity during the sustained contractions because an increase in antagonist muscle activity can exacerbate the appearance of fatigue in an agonist muscle (29). However, the increase in the level of coactivation was minor and similar for both the responders and nonresponders across sessions. Consequently, coactivation of the antagonist muscle did not account for the different adaptations in endurance time for the responders and nonresponders.

The increase in EMG activity of an agonist muscle during a fatiguing contraction at a submaximal intensity is attributable to an increase in motor unit activity (7, 9, 26), perhaps including a contribution from a reduction in signal cancellation (6, 41). Because the modulation of discharge rate during such contractions is rather modest, the increase in AEMG is largely due to the recruitment of motor units (4, 5, 11, 14, 37, 38). The gradual increase in AEMG that we observed, therefore, likely involved the progressive recruitment of more fatigable motor units. Accordingly, the prolongation of endurance time across sessions should have been associated with a decline in the rate of increase in AEMG and a reduction in the rate of EMG bursts.

The results that were most consistent with this rationale were the more pronounced decline in burst rate across sessions for the responders and the negative
relation between burst rate and endurance time. This effect is evident in the records of the individual shown in Fig. 4, which indicates that the number of bursts in session 1 (39, 18, 0, and 41 for brachialis, brachioradialis, biceps long, and biceps short, respectively) declined to almost zero (0, 0, 0, and 5, respectively) over the same absolute time in session 3. Because a burst of EMG activity corresponded to the transient recruitment of additional motor units, the decline in burst rate indicated a delay in the recruitment of more fatigable motor units and hence enabled the prolongation of endurance time.

Changes in the rate of increase in AEMG across sessions were also consistent with the rationale that the increase in endurance time was attributable to the activation of fewer fatigable motor units. Although the average rate of increase in AEMG decreased across sessions for both groups of subjects, the rate remained greater for the nonresponders. Furthermore, the AEMG for the nonresponders exhibited a qualitative change at ~20% MVC when it went from a linear increase to a much more variable signal, including marked increases in the terminal values for some muscles. The responders did not use this pattern of activity. The significance of this observation may be related to reports (16, 24, 30) that find target forces of 15–20% MVC force to distinguish between short and long endurance times for sustained contractions. On the basis of the cumulative sum of the tetanic forces of motor units, a force of 20% MVC would require the activation of about two-thirds of the motoneuron pool (10, 22), which can correspond to the region in which the fatigability of motor units changes (23). Once the descending drive is sufficient to recruit more fatigable motor units during a fatiguing contraction, they appear to be activated transiently in bursts of activity with burst rate increasing up to exhaustion.

This interpretation that the improvement in endurance time across sessions experienced by some subjects was due to adjustments made in motor unit activity raises a significant but unanswered question. What adjustment enabled the target force to be sustained with a lesser rate of increase in motor unit activity? A likely explanation, which awaits investigation, is that the input received by the motoneuron pool can change with practice, including changes in either the descending drive or the feedback from peripheral receptors. Our results indicate that the perceived effort of the responders was less during the contractions performed during sessions 2 and 3, although similar at exhaustion. These results further suggest that modulation of the excitatory descending drive partly contributed to an enhanced endurance time of the responders. In contrast, there was no difference in the pressor response between the two groups of subjects, which suggests that the feedback from group III and IV afferents did not contribute to the adaptation across sessions.

Fatiguing isometric contractions performed at low to moderate forces are characterized by a progressive increase in the amplitude of the force fluctuations (27, 36). This phenomenon appears to be adaptable because we observed that the increase in the fluctuations of the force exerted at the wrist during the fatiguing contractions declined in sessions 2 and 3 for the responders and nonresponders when compared with session 1. The adaptations in the force fluctuations paralleled the changes in AEMG across sessions for the two groups. Furthermore, there was a significant association between EMG burst rate and force fluctuations. Consequently, the mechanisms responsible for the adaptations in the force fluctuations and EMG activity may have been similar and underscore the modulation of the force fluctuations by central sources.

In summary, practice of a sustained isometric contraction at a submaximal intensity with the elbow flexor muscles enabled some men and women, but not others, to prolong the endurance time to exhaustion. This improvement in performance was independent of the sex of the individual, target force, and the pressor response. However, the changes in endurance time were associated with a reduced rate of increase in motor unit activity, as indicated by a decrease in EMG burst rate, a decline in the rate of increase in the AEMG, and an alteration in the distribution of AEMG activity among the synergist muscles. These results indicate that the maximum duration of a low-force contraction can be limited by the neural activation of muscle.

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