

ABSTRACT: The purpose of the study was to identify factors that could predict differences among individuals in the time to failure of a submaximal contraction. Twenty subjects (10 men, 25 ± 6 years) supported an inertial load equivalent to 20% of the maximal voluntary contraction (MVC) force with the elbow flexor muscles for as long as possible. The time to failure was predicted by the frequency of electromyographic bursts in the long head of biceps brachii during the first 20% of the contraction, the amplitude of bursts in the brachioradialis during the first 20% of the contraction, and the target torque. Subjects who could sustain the task longer exhibited greater initial (first 20% of contraction) electromyographic burst frequency in the long head of biceps brachii, lower initial burst amplitudes in the brachioradialis muscle, and lower target torque. Knowing the main predictors of a submaximal fatiguing contraction with the elbow flexor muscles may assist clinicians in personalizing therapeutic interventions.

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TIME TO FAILURE OF A SUSTAINED CONTRACTION IS PREDICTED BY TARGET TORQUE AND INITIAL ELECTROMYOGRAPHIC BURSTS IN ELBOW FLEXOR MUSCLES

THORSTEN RUDROFF, PhD,¹ EVANGELOS A. CHRISTOU, PhD,¹ BRACH POSTON, MS,¹ JENS BOJSEN-MØLLER, PhD,² and ROGER M. ENOKA, PhD¹

¹ Department of Integrative Physiology, University of Colorado, Carlson 202G, 354 UCB, Boulder, Colorado 80309-0354, USA

² Institute of Sports Medicine, Copenhagen/Team Danmark Test Center, Copenhagen, Denmark

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The mechanisms that contribute to the fatigue experienced during a sustained submaximal contraction include impairments that range from the input received by the motor cortex to the force-generating capacity of the contractile proteins.^{18,49} The prevailing mechanism that limits force production during a fatiguing muscle contraction varies with the task parameters^{5,11,18} and depends on such factors as the type and intensity of exercise,^{7,10,34,38,50} the muscle groups involved,^{2,4,31} and the physical environment in which the task is performed.^{6,9,15,17,20,48,63}

Given a specific task, however, the duration that a submaximal load can be supported varies widely among individuals. For example, Rudroff et al.⁵² found that the time to failure ranged from 216 to

914 s (mean \pm SD: 477 ± 276 s) when 8 men supported an inertial load that was 20% of maximal voluntary contraction (MVC) force with the elbow flexor muscles for as long as possible. Because the time to task failure is inversely related to the absolute target force,²² some of the variance in the time to failure among subjects is likely attributable to differences in strength due to the influence of contraction intensity on muscle perfusion.^{19,22,30,36} For example, the longer time to task failure observed in women relative to men is often a result of the women exerting a lower absolute force at the same percentage of the maximal force.^{22,57} The target force also influences the rate at which the electromyographic (EMG) activity increases during a sustained submaximal contraction,^{3,14,35,60,62} and this can limit the time to task failure as observed with variation in the type of load supported and task feedback during the fatiguing contraction,^{40,41,45,52} posture of the limb,⁵⁴ practice of the same task,²⁵ and several weeks of limb immobilization.⁵⁹ Furthermore, the rise in mean arterial pressure (MAP) can be insufficient to counteract the occlusion imposed by the contracting muscle and thereby hasten the arrival of task failure.^{13,19,23,30}

Abbreviations: aEMG, average electromyographic activity; ANOVA, analysis of variance; DBP, diastolic blood pressure; EMG, electromyography; HR, heart rate; MAP, mean arterial pressure; MVC, maximal voluntary contraction; SBP, systolic blood pressure

Key words: electromyogram; forearm posture; muscle fatigue; static exercise; target torque

Correspondence to: T. Rudroff; e-mail: thorsten.rudroff@colorado.edu

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The purpose of this study was to identify factors that could predict the differences between individuals in the time that a submaximal contraction could be sustained. Based on the critical role of the rate of increase in EMG activity in the involved muscles in accounting for the different times to failure across similar tasks,^{24,40,52} the hypothesis was that variation in time to failure among individuals is associated with differences in the rate of increase in EMG activity for the elbow flexor muscles. Some of these data have been presented previously in abstract form.⁵³

METHODS

Twenty healthy, normotensive adults (10 men, 25 ± 6 years) volunteered to participate in the study. The subjects reported no neurological disorders or cardiovascular diseases and no use of medications known to influence neurological function. Body mass (78.6 ± 10.4 and 72.4 ± 20.2 kg) and height (173 ± 8 and 172 ± 6 cm) were similar for men and women. The men and women were matched for strength in absolute terms (Nm) based on the torque exerted at the wrist during an MVC with the elbow flexor muscles. Each pair of subjects was matched within 5% of the MVC torque. Subjects provided written, informed consent before participating in the study and our institutional review board approved the protocol. The experimental design and procedures were similar to those described previously.^{52,53}

Each subject participated in the study on two occasions: an introductory session to become familiar with the equipment and procedures, and a session to sustain a submaximal contraction until failure. Subjects were seated upright in an adjustable chair with the right (dominant arm) shoulder joint flexed in the sagittal plane so that the upper arm was horizontal and the forearm was vertical and supinated (1.57 rad of flexion at the shoulder and elbow joints) (Fig. 1A). Nylon straps were placed over each shoulder and around the waist to stabilize and restrain the subject. The hand and forearm were placed in a modified wrist–hand–thumb orthosis (Orthoamerica, Newport Beach, California) and an inertial load equivalent to 20% of the peak force achieved during a maximal isometric contraction was suspended via a line-and-pulley system from the subject's wrist. The force under the elbow joint was measured with a transducer (Model ELW-D1-200 I; 0.27 mV/N; Entran, Hampton, Virginia). Elbow joint angle was measured with an electrogoniometer (Model XM110 and K100; Penny and Giles, Cwm-

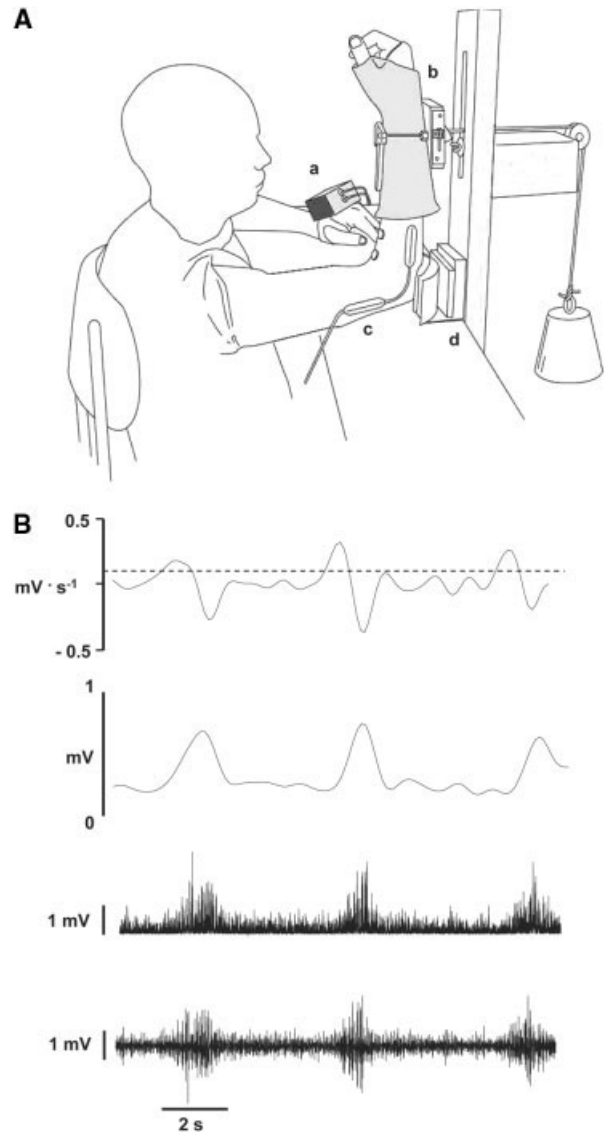


FIGURE 1. Experimental arrangement and EMG burst analysis. **(A)** Subjects were seated upright in an adjustable chair with the dominant (right) arm rotated forward so that the upper arm was horizontal and the forearm vertical. The right hand and forearm were placed in a modified wrist–hand–thumb orthosis. The fatiguing contraction involved supporting an inertial load that was suspended from the subject's wrist. (a) Blood pressure transducer, (b) accelerometers, (c) goniometer, and (d) elbow force transducer. **(B)** Identification of EMG bursts for the brachioradialis muscle during the fatiguing contraction. Records show the interference EMG (first trace from the bottom), the rectified EMG (second trace), the rectified EMG after it was low-pass filtered at 2 Hz (third trace), and the differentiated and normalized EMG (fourth trace). A burst was identified when the differentiated EMG signal exceeded a threshold set at $0.1 \text{ mV} \cdot \text{s}^{-1}$.

felinfach, Gwent, UK) that was securely fastened to the lateral aspect of the elbow joint, and the output was recorded and displayed on a monitor. Two uni-axial accelerometers (Model 7265A-HS; Endevco,

San Juan Capistrano, California) were mounted on the orthosis to measure acceleration of the hand in the frontal and sagittal planes.

EMG activity was measured with bipolar surface electrodes (Ag–AgCl, 8-mm diameter; 20-mm distance between electrodes) that were placed over the short and long heads of biceps brachii, brachioradialis, the lateral head of triceps brachii, and the anterior head of the deltoid muscle. The electrodes were attached distal to the innervation zone for each muscle.^{43,44} The EMG of the brachialis muscle was measured with an intramuscular bipolar electrode inserted ~3 cm proximal to the antecubital fossa. The electrode comprised two stainless-steel wires (100- μ m diameter) that were insulated with Formvar (California Fine Wire Co., Grover Beach, California). One wire in each pair had ~2 mm of insulation removed to increase the recording volume of the electrode. Reference electrodes were placed on a bony prominence at either the clavicle or the acromion. The EMG signal was amplified (1000 \times), band-pass filtered (13–1000 Hz; Coulbourn Instruments, Allentown, Pennsylvania), and recorded on a computer. Heart rate (HR) and MAP were monitored throughout the fatiguing contraction with an automated beat-by-beat blood pressure monitor (Finapres 2300; Ohmeda, Madison, Wisconsin), which was placed around the middle finger of the left hand. Each subject placed the non-exercising arm on a table that was located at the level of the subject's heart.

Experimental Protocol. Subjects were required to perform two tasks in the following order: (1) MVCs with the elbow flexor and extensor muscles and the anterior deltoid muscle; and (2) a sustained submaximal contraction with the elbow flexor muscles to failure.

MVC Force. The modified wrist–hand–thumb orthosis was rigidly attached to a force transducer and the subject increased the force from zero to maximum in 3 s; the maximal force was held for 2–3 s. Subjects were provided with strong verbal encouragement during this task. Subjects rested for 60–90 s between trials. Additional trials were performed when the peak MVC force in the two maximal trials differed by more than 5%. The greatest force achieved by each subject was taken as the MVC force and used as the reference value to calculate the target load for the submaximal contraction. The average electromyographic activity (aEMG) obtained during the fatiguing contractions was normalized to the maximal EMG obtained during the MVC.

Submaximal Contraction. The subject maintained a constant arm position for as long as possible while supporting an inertial load that was equal to 20% of the MVC force with the elbow flexor muscles. The net muscle torque was determined as the product of the weight of the load attached at the wrist and the moment arm from the load location to the elbow joint (lateral epicondyle of the humerus to the radial styloid process). The submaximal contraction was terminated when one or more of the following criteria were met for at least 5 s, despite strong verbal encouragement: (1) pronation of the forearm; (2) deviation of elbow joint angle from 1.57 rad by 0.2 rad; or (3) removal of the elbow from the underlying transducer. Displacement of the forearm from the supinated position was evaluated visually and feedback was provided to the subjects by the same investigator for all experiments. The duration that the task could be sustained was denoted as the “time to failure of the task.”

Data Analysis. All data collected during the experiments were recorded on a computer and analyzed off-line using the Spike2 data analysis system (Cambridge Electronic Design, Cambridge, UK). The force, position, and blood pressure signals were digitized at 200 samples/s and the EMG signals were recorded at 2000 samples/s. HR and MAP were quantified as 15-s averages at 10% intervals of contraction duration. The blood pressure signal was analyzed to determine mean systolic blood pressure (SBP), mean diastolic blood pressure (DBP), and the number of pulses per second. MAP was calculated as $MAP = DBP + \frac{1}{3}(SBP - DBP)$. The fluctuations in hand acceleration during the sustained contraction were characterized as the average SD of the resultant acceleration derived from the measurement of acceleration in the forward–backward (sagittal plane) and side-to-side (frontal plane) directions. The SD of the resultant acceleration was determined for each 20% interval of the contraction duration.

The maximal EMG of the elbow flexor muscles and triceps brachii were determined as the average rectified EMG value over a 0.5-s interval that was centered at the peak MVC force. The EMG activity during the sustained contraction was quantified by averaging the rectified EMG (aEMG) over intervals that corresponded to 20% of task duration. The EMG of the anterior deltoid was normalized to the maximal EMG obtained for the muscle when the seated subject performed a maximal isometric shoulder flexion with the arm extended horizontally and the forearm pronated.³²

The bursts of EMG activity for each muscle during the sustained contraction were determined with the method developed by Hunter et al.^{24–26} (Fig. 1B). The rectified EMG signal was smoothed with a low-pass filter at 2 Hz (surface EMG) or 3.8 Hz (intramuscular EMG), differentiated, and divided by the average rectified signal EMG. The start of a burst was defined as the time at which the EMG increased by $>0.1 \text{ mV}\cdot\text{s}^{-1}$ (surface EMG) or $>0.26 \text{ mV}\cdot\text{s}^{-1}$ (intramuscular EMG). These values corresponded to 3 SDs above the mean differentiated EMG signal based on 50 samples when the absolute EMG signal displayed minimal bursting activity during the initial period of the fatiguing contraction. The end of a burst was defined as the time when the EMG declined in amplitude to a value less than or equal to that at the start of the burst. Alternatively, when the EMG signal did not return to the starting value before a subsequent burst, the end of the burst was defined as the time at which the differentiated, filtered EMG signal was most negative prior to the next burst. The start of each burst was required to be greater than 2 s from the onset of the preceding burst, which resulted in an upper detection limit for the burst frequency of 0.5 Hz.^{24–26} Burst amplitude, frequency, and duration were quantified as averages over intervals that corresponded to 20% of task duration. The amplitude of the EMG burst was quantified as the average amplitude of the rectified, low-pass-filtered (2 Hz), and linear-detrended EMG signal.

Statistical Analysis. The dependent variables were HR, MAP, target torque, SD of hand acceleration, average EMG activity, and the frequency, duration, and amplitude of aEMG bursts. Two-factor analyses of variance (ANOVAs; gender \times time) with repeated measures on time were used to compare the dependent variables of HR, MAP, and SD of acceleration between men and women. A mixed, three-factor ANOVA (muscle \times time \times gender) with repeated measures on time and muscle was used to compare the dependent variables of aEMG and the frequency, duration, and amplitude of aEMG bursts of the short and long heads of biceps brachii, brachioradialis, brachialis, triceps brachii, and anterior deltoid during the fatiguing contraction between the two genders. Paired *t*-tests (independent and dependent) with Bonferroni corrections were used as post hoc analyses to test differences among pairs of means when appropriate.

Multiple, linear regressions and the associated partial correlations (*r*) were performed to examine the contribution of each independent variable (initial values of aEMG activity, frequency, and amplitude of aEMG bursts of each muscle and target

torque) to the time to task failure. The initial values corresponded to those obtained from the first 20% of the contraction. The associated partial correlation coefficients were used to identify the unique contribution of each independent variable to the time to task failure. In addition, the associations between two variables were examined with bivariate regression analyses and were performed between the following variables: (1) rate of increase in the normalized aEMG for the elbow flexor muscles, initial SD of hand acceleration, and time to task failure; (2) rate of increase in the normalized aEMG for the long head of biceps brachii, target torque, initial aEMG burst frequency for the long head of biceps brachii, and rate of increase in MAP; and (3) the initial normalized amplitude of brachioradialis aEMG and initial SD of hand acceleration.

The strength of the association is reported as the squared Pearson product-moment correlation coefficient (r^2), which indicates the proportion of variance for the criterion variable (typically referred as the dependent variable; e.g., time to failure) that is accounted for by its linear relation with the predictor variable (typically referred as the independent variable; e.g., target torque).²¹ The goodness-of-fit of the model, which indicates how well the linear combination of the predictor variables (e.g., initial burst frequency of the long head of biceps brachii, initial burst amplitude of brachioradialis) predicted the criterion variable (e.g., time to failure), is reported as the squared multiple correlation (R^2). The relative importance of the predictors was estimated with the partial correlations (*r*), which provide the correlation between the dependent variable and an independent variable when the linear effects of the other independent variables in the model have been removed from both.²¹ A positive sign of the partial correlation indicates that the predictor and the criterion are positively related, whereas a negative sign indicates that they are inversely related.

A significance level for all statistical tests was set at $P < 0.05$, except when modified by the Bonferroni correction. Data are reported as means \pm SD within the text, and displayed as means \pm SE in the figures. All regression analyses were performed with SPSS software (version 13.0; SPSS, Chicago, Illinois).

RESULTS

The average target torque was $17.3 \pm 3.7 \text{ Nm}$ (range: 11.7–24.1 Nm) and the time to task failure was $567 \pm 353 \text{ s}$ (range: 176–1411 s). The subjects terminated the task due to a combination of the inability to maintain both the supinated forearm position and

Table 1. Average EMG (% MVC) at five time-points during the fatiguing contraction.

	10%	30%	50%	70%	90%
Biceps brachii, long	13 ± 5	14 ± 5	15 ± 6	16 ± 6	25 ± 10
Biceps brachii, short	12 ± 6	13 ± 7	15 ± 8	17 ± 8	27 ± 17
Brachioradialis	13 ± 5	13 ± 6	14 ± 6	16 ± 7	28 ± 12
Brachialis	21 ± 6*	21 ± 6*	22 ± 7*	23 ± 7*	32 ± 13*
Triceps brachii	5 ± 2	5 ± 2	5 ± 3	6 ± 3	7 ± 5
Anterior deltoid	11 ± 10	11 ± 11	11 ± 11	13 ± 16	15 ± 11

* $P < 0.05$ with brachioradialis and biceps brachii (short and long).

required elbow angle. Men and women exhibited similar target torques (17.4 ± 3.9 and 17.3 ± 3.6 Nm, respectively; $P > 0.05$) and times to task failure (521 ± 347 and 614 ± 372 s, respectively; $P > 0.05$).

Changes in EMG Activity, MAP, HR, and SD of Acceleration.

As reported previously, aEMG amplitude and burst frequency increased progressively during the fatiguing contraction for all subjects.^{22–26,40,52} The amplitude of the aEMG and the amplitude and frequency of aEMG bursts did not differ for men and women for all muscles (gender main effect, in all instances $P > 0.336$), across time (gender \times time interaction, in all instances $P > 0.716$), or across muscles (gender \times muscle interaction, all $P > 0.103$; gender \times muscle \times time interaction, in all instances $P > 0.120$). Therefore, the data were collapsed across gender. The amplitude of the aEMG and the amplitude and frequency of aEMG bursts increased with time (time main effect: $P < 0.001$) (Tables 1 and 2). The mean burst duration for the elbow flexor muscles during the fatiguing contractions was 4.3 ± 5.9 s and did not change across the fatiguing contractions. There was no difference in burst duration between men and women. Furthermore, the burst duration was similar for all muscles, including: short head of biceps brachii (4.8 ± 5.5 s); long head of biceps brachii (4.5 ± 5.5 s); brachioradialis (5.3 ± 8.1 s); and brachialis (3.2 ± 3.9 s). The values for burst duration were averaged across the entire time course of the fatiguing contraction.

The resting, initial, and final values, and the rates of increase for MAP, HR, and SD of hand acceleration did not differ for men and women (gender \times main effect, $P > 0.336$), or across time (gender \times time interaction, $P > 0.41$). Therefore, the data were collapsed across gender. The resting values for MAP and HR were 80 ± 9 mm Hg and 76 ± 14 bpm, respectively. Both parameters increased progressively during the sustained submaximal contraction and achieved mean increases at failure of $35 \pm 16\%$ for MAP and $27 \pm 16\%$ for HR (time main effect, $P < 0.001$). The rates of increase in MAP and HR were associated with time to failure ($r^2 = 0.57$, $P < 0.0001$ and $r^2 = 0.52$, $P = 0.0003$, respectively), whereas the magnitude of change in MAP and HR was not associated with time to failure ($r^2 = 0.16$, $P = 0.08$ and $r^2 = 0.11$, $P = 0.16$, respectively). This finding indicates that subjects achieved a similar rise in MAP and HR; however, the increase was more rapid in those subjects who had a shorter time to failure. The initial SD of hand acceleration was 0.19 ± 0.13 m/s² and this increased to 0.53 ± 0.37 m/s² at failure (time main effect, $P < 0.001$) (Table 3).

Factors That Contributed to the Time to Failure. As predicted by the hypothesis, differences among subjects were related to the rate of increase in aEMG of the elbow flexor muscles. However, only the rate of increase in aEMG for the long head of the biceps brachii was linearly associated with differences in the time to failure across subjects, and the relation was

Table 2. Burst frequency (bursts.min⁻¹) and burst amplitude (% MVC) at five time-points during the fatiguing contraction.

	Burst frequency (bursts.min ⁻¹)					Burst amplitude (% MVC)				
	10%	30%	50%	70%	90%	10%	30%	50%	70%	90%
Biceps brachii, long	4 ± 4	4 ± 4	7 ± 5	11 ± 6	15 ± 7	11 ± 5	12 ± 6	15 ± 8	18 ± 9	23 ± 9
Biceps brachii, short	2 ± 4	3 ± 4	7 ± 4	10 ± 4	14 ± 7	12 ± 7	14 ± 8	17 ± 9	21 ± 11	27 ± 14
Brachioradialis	4 ± 3	5 ± 5	8 ± 5	14 ± 7	17 ± 8	12 ± 5	13 ± 6	16 ± 8	21 ± 9	29 ± 12
Brachialis	5 ± 5	6 ± 7	8 ± 7	10 ± 7	13 ± 9	21 ± 9*	21 ± 8*	26 ± 11	26 ± 10	38 ± 30
Triceps brachii	1 ± 1	2 ± 1	2 ± 2	5 ± 5	6 ± 5	4 ± 2	4 ± 2	4 ± 2	5 ± 3	6 ± 3
Anterior deltoid	3 ± 4	2 ± 3	2 ± 3	3 ± 3	6 ± 8	5 ± 3	5 ± 4	10 ± 21	10 ± 15	14 ± 21

* $P < 0.05$ with brachioradialis and biceps brachii (short and long).

Table 3. Mean arterial pressure (MAP), heart rate, and acceleration SD at five time-points during the fatiguing contraction.

	10%	30%	50%	70%	90%
MAP (mm Hg)	80 ± 9	87 ± 8	94 ± 9	99 ± 10	104 ± 11
Heart rate (bpm)	76 ± 14	80 ± 14	85 ± 14	88 ± 14	93 ± 15
Acceleration SD (m/s ²)	0.19 ± 0.1	0.19 ± 0.1	0.21 ± 0.1	0.25 ± 0.1	0.53 ± 0.4

moderate ($r^2 = 0.51$, $P < 0.0001$). Rather, the relation between the rate of increase in aEMG for the long head of biceps brachii and time to failure was best fitted with an exponential decay function ($r^2 = 0.74$, $P = 0.0004$), which suggested that other factors also influenced the time to failure. The rate of change in aEMG for the short head of the biceps brachii ($r^2 = 0.049$, $P = 0.347$), brachialis ($r^2 = 0.23$, $P = 0.06$), and brachioradialis ($r^2 = 0.01$, $P = 0.63$) were not associated with differences in the time to failure across subjects.

Multifactorial regression analysis was used to improve the prediction of time to failure; the predictor variables included the initial (first 20% of contraction) and maximal values for burst frequency and aEMG amplitude for the elbow flexor muscles, heart rate, mean arterial pressure, SD of hand acceleration, and target torque. The prediction of time to failure improved significantly with a multiple linear regression function ($R^2 = 0.78$, $P < 0.001$; Fig. 2A) that included the target torque, the initial burst frequency in the aEMG in the long head of biceps

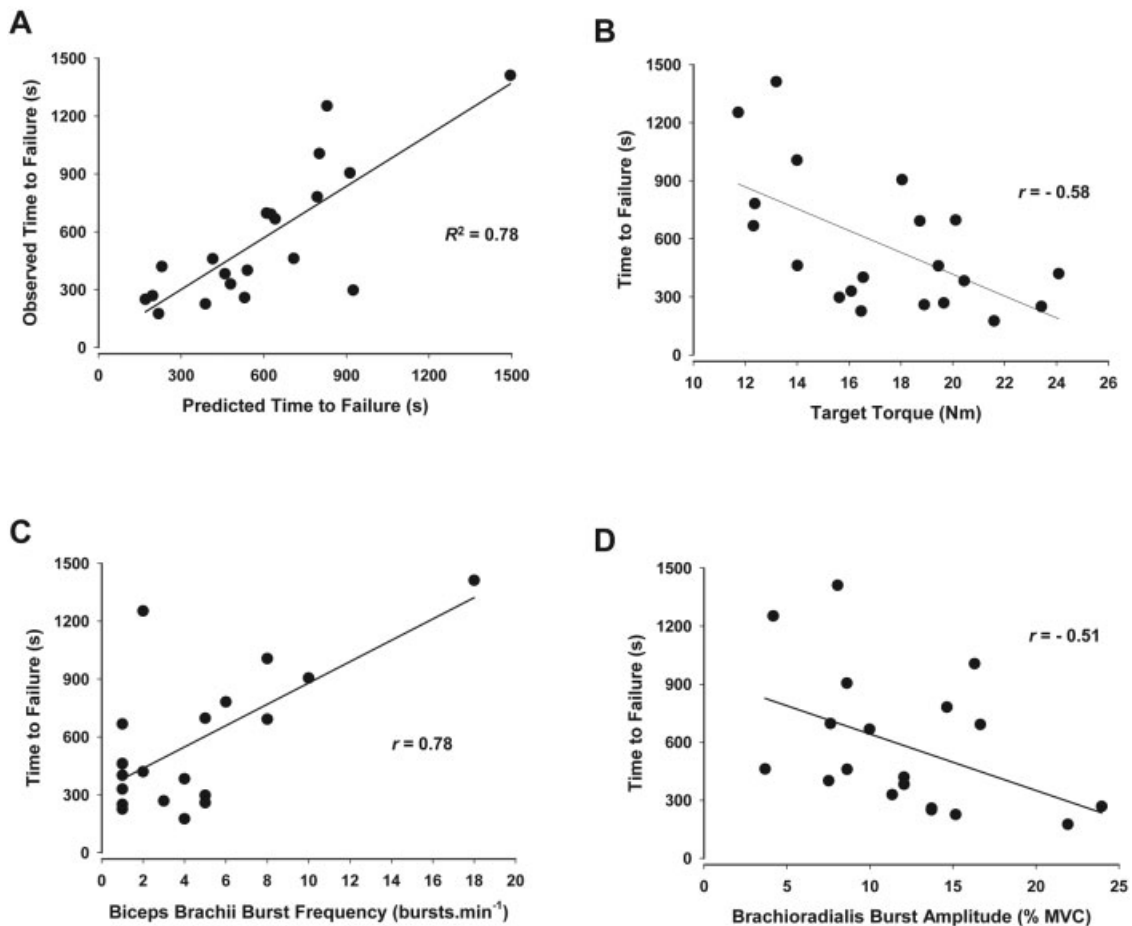


FIGURE 2. Prediction of time to failure. (A) The time to task failure was strongly predicted by the linear combination of the initial frequencies of the aEMG bursts in the long head of biceps brachii, the initial aEMG burst amplitudes for brachioradialis, and the target torque. (B) Subjects with greater target torques exhibited briefer times to failure. (C) Subjects with greater initial aEMG burst frequencies for the long head of the biceps brachii experienced longer times to failure. (D) Subjects with greater initial aEMG burst amplitudes in brachioradialis had briefer times to failure.

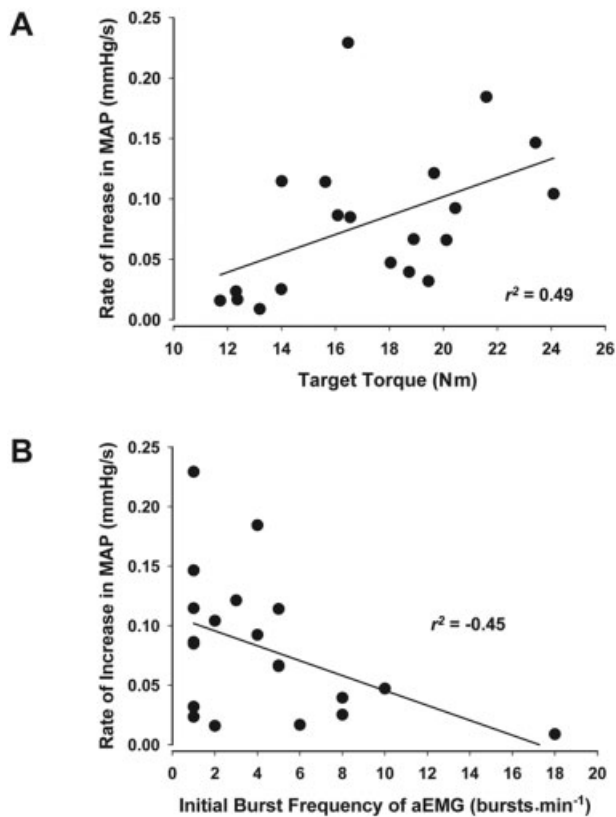


FIGURE 3. Target torque and the initial burst frequency of the aEMG for the long head of the biceps brachii influenced the time to failure by changing the rate of increase in MAP. The rate of increase in MAP was positively associated with the target torque (A), and negatively associated with the initial burst frequency of the long head of biceps brachii (B).

brachii, and the initial burst amplitudes in the aEMG for brachioradialis. The equation for the predicted time was: $1232 + (-35.6 \times \text{target torque}) + (49.8 \times \text{initial burst frequency in the long head of biceps brachii}) + (-20.3 \times \text{initial amplitude of brachioradialis})$. Figure 2B–D shows the individual associations between these predictors and the time to failure. Target torque and the initial aEMG burst amplitudes in brachioradialis were negatively associated with time to failure (partial $r = -0.58$, $P = 0.014$ and partial $r = -0.51$, $P = 0.035$, respectively; Fig. 2B and D), whereas the initial burst frequency in the aEMG for the long head of biceps brachii was positively associated with time to failure (partial $r = 0.78$, $P < 0.0001$; Fig. 2C).

The influence of the target torque and initial aEMG burst frequency for the long head of biceps brachii on the time to failure can be explained by their associations with the rate of increase in MAP. Both variables influenced the time to failure by changing the rate of increase in MAP. The target

torque was positively associated with the rate of increase in MAP ($r^2 = 0.49$, $P < 0.03$; Fig. 3A), whereas the initial burst frequency for the long head of biceps brachii was negatively associated with the rate of MAP increase ($r^2 = -0.45$, $P < 0.049$; Fig. 3B). Thus, subjects with a greater target torque experienced a greater rate of increase in MAP and a briefer time to failure, and subjects with greater initial burst frequencies in the aEMG for the long head of biceps brachii exhibited a slower rate of increase in MAP and longer times to failure.

The activity of the brachioradialis muscle, however, appeared to be related to the time to failure by influencing the fluctuations in hand acceleration. The initial aEMG amplitude in brachioradialis was associated with the initial SD of hand acceleration ($r^2 = 0.26$, $P = 0.026$; Fig. 4A), which was negatively associated with the time to failure ($r^2 = -0.45$, $P < 0.0001$; Fig. 4B). These results indicate that subjects with greater initial aEMG burst amplitudes in bra-

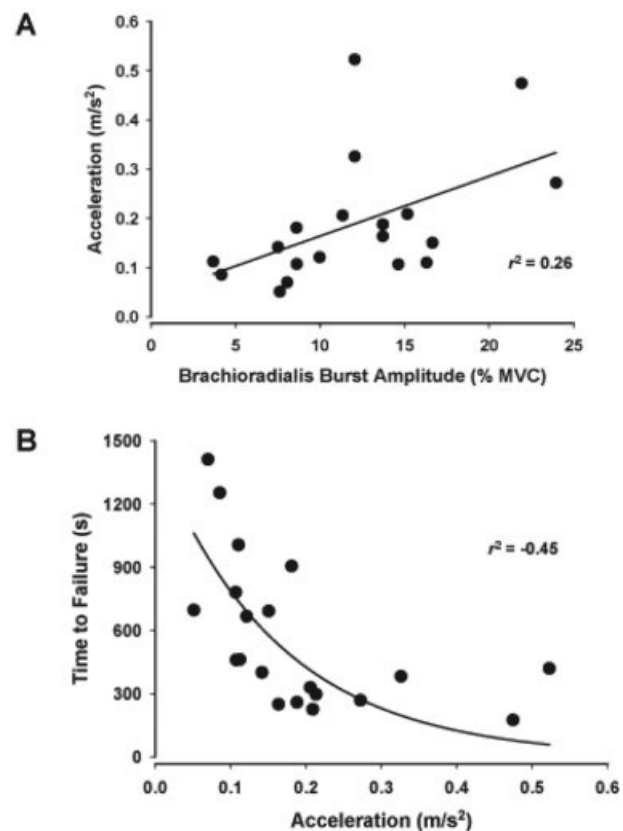


FIGURE 4. Initial aEMG burst amplitude for brachioradialis was positively associated (A) with the initial hand acceleration and the initial hand acceleration was negatively associated (B) with the time to failure. These results indicate that subjects with greater initial aEMG burst amplitudes in brachioradialis and SDs of hand acceleration had briefer times to failure.

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DISCUSSION

Although the rate of increase in aEMG activity and the frequency of EMG bursts of all muscles increased during sustained fatiguing contraction,^{24–26} our hypothesis was only partially supported as the time to failure was associated with the rate of increase in EMG activity of a subset of the muscles involved in the task. Specifically, only the rate of increase in aEMG for the long head of biceps brachii was related to the time to failure. This relation was non-linear and best fitted with an exponential decay function due to the influence of other factors on the time to failure. Multifactorial regression analysis indicated that variation in the time to failure among individuals was associated with the frequency of EMG bursts in the long head of biceps brachii during the first 20% of the contraction, the amplitude of EMG bursts in the brachioradialis during the first 20% of the contraction, and the target torque. Hence, subjects with greater initial bursting of the long head of biceps brachii, lower activity of the brachioradialis muscle, and lower target torque were able to sustain the submaximal contraction for a longer time.

Contributing Factors to the Time to Task Failure.

Only the rate of increase in aEMG for the long head of biceps brachii was related to the time to failure. Although both heads of the biceps brachii contribute to the supination and flexion torques about the elbow joint, the long head appears to provide significant postural support at the shoulder joint, especially when the arm is in an elevated position.⁵⁶ Therefore, the specific limb posture may have influenced the relative significance of the activity in the long head of biceps brachii.

As known from previous studies^{24–26} the rate of change in EMG activity of the elbow flexor muscles is associated with the time to task failure. Our results extend these findings. It is suggested that target torque and initial burst frequencies in the long head of biceps brachii are among multiple factors that contributed to the time to failure by influencing the rate of increase in MAP. Greater target torques increased MAP, whereas greater initial burst frequencies in the long head of biceps brachii resulted in a slower rate of increase in MAP. As expected from previous studies,^{22,42,58} subjects with lower absolute target torques were able to support the submaximal inertial load for a longer time. Accordingly, the differences among subjects in the time to failure was

influenced by the absolute target torque, and the significant association for MAP suggests that the mechanisms involved in maintaining muscle perfusion did vary substantially across subjects. Although Hunter et al.²⁷ found no association between differences in the time to task failure and the increase in MAP between men and women, our study indicated that differences in the increase in MAP can contribute to variation in the time to failure across subjects.

Although we did not measure blood flow, studies^{39,55} have shown that greater absolute forces are associated with increased intramuscular pressures, occlusion of blood flow, accumulation of metabolites, heightened metaboreflex responses, and impairment of oxygen delivery to the muscle. In addition, activation of the pressor response, as measured by the rate of increase in MAP, is inversely related to the time to task failure.^{13,58} Subjects with lower target forces demonstrated a reduced rate of increase in MAP (pressor response) and therefore a longer time to task failure, likely due to less mechanical resistance of blood supply to the active muscles and a reduced buildup of metabolites within the muscle.

Sustained submaximal contractions are characterized by the presence of bursting activity in the interference EMG signal.^{22,23,26,29,39,40} This bursting activity may involve motor unit substitution and rotation,³⁷ which has been observed in long-duration fatiguing contractions,^{1,61} that would enable the fatiguing motor units to undergo brief amounts of rest during the sustained contraction. Subjects with greater initial burst activity demonstrate a slower rate of increase in MAP, which suggests that the transient recruitment of higher-threshold motor units was more gradual. Accordingly, subjects who had greater initial burst frequencies in the long head of biceps brachii also had a lower rate of increase in MAP and were able to sustain the task for a longer duration.

Although the EMG recordings obtained in our study could not determine whether the EMG burst involved either the addition, substitution, or rotation of motor units, the progressive increase in burst amplitude suggests that the compensation involved either the recruitment of larger motor units, the activation of motor units that were closer to the recording electrode,^{33,45} or an increase in the coincidence of motor unit discharge times. Furthermore, the burst duration did not change over the course of the fatiguing contraction for any of the muscles, which indicates that either additional motor units were not recruited and remained active or the discharge rate of the involved motor units remained the same.

The third predictor of the time to failure was the burst amplitude of brachioradialis muscle during the first 20% of the contraction. Due to its anatomical arrangement, the brachioradialis muscle exerts a pronation moment when the forearm is supinated and a supination moment when the forearm is pronated.⁴⁶ Further, afferent feedback from the branch of the radial nerve that innervates brachioradialis inhibits the discharge of single motor units in biceps brachii.^{47,51} Moreover, the concept of signal-dependent noise²⁸ indicates that greater increases in the fluctuations in motor output are associated with greater excitation of the motor neuron pool.^{8,12,16} Thus, the rate of increase in excitation of the motor unit pool for brachioradialis varied across subjects and was associated with differences in the time to task failure. Taken together, the findings indicate that differences among individuals in the time to failure of a sustained, submaximal contraction with the elbow flexor muscles can be explained by relative activity in some muscles (long head of biceps brachii and brachioradialis) and the magnitude of the target torque.

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