Electromyographic measures of muscle activation and changes in muscle architecture of human elbow flexors during fatiguing contractions

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Rudroff T, Staudenmann D, Enoka RM. Electromyographic measures of muscle activation and changes in muscle architecture of human elbow flexors during fatiguing contractions. J Appl Physiol 104: 1720–1726, 2008. First published March 20, 2008; doi:10.1152/japplphysiol.01058.2007.—The study compared changes in intramuscular and surface recordings of EMG amplitude with ultrasound measures of muscle architecture of the elbow flexors during a submaximal isometric contraction. Ten subjects performed a fatiguing contraction to task failure at 20% of maximal voluntary contraction force. EMG activity was recorded in biceps brachii, brachialis, and brachioradialis muscles using intramuscular and surface electrodes. The rates of increase in the amplitude of the surface EMG for the long and short heads of biceps brachiii and brachioradialis were greater than those for the intramuscular recordings measured at different depths. The amplitude of the intramuscular recordings from three muscles increased at a similar rate \((P = 0.13)\), as did the amplitude of the three surface recordings from two muscles \((P = 0.83)\). The increases in brachialis thickness \((27.7 \pm 5.7 \text{ to } 30.9 \pm 3.5 \text{ mm}; P < 0.05)\) and pennation angle \((10.9 \pm 3.5 \text{ to } 16.5 \pm 4.8^\circ; P = 0.003)\) were not associated with the increase in intramuscular EMG amplitude \((P > 0.58)\). The increase in brachioradialis thickness \((22.8 \pm 4.8 \text{ to } 25.5 \pm 3.4 \text{ mm}; P = 0.0075)\) was associated with the increase in the amplitude for one of two intramuscular EMG signals \((P = 0.007, r = 0.79)\). The time to failure was more strongly associated with the rate of increase in the amplitude of the surface EMG than for the intramuscular EMG, which suggests that the surface measurement provides a more appropriate measure of the change in muscle activation during a fatiguing contraction.

Ultrasound; elbow flexors; muscle fatigue; electromyogram

The progressive increase in the amplitude of the EMG during a sustained submaximal contraction \((1, 6, 15, 24, 33, 35)\) can vary among a group of synergist muscles \((14, 23, 34)\) due to differences in the mechanical action contributed by each muscle \((16, 44)\), the reflex pathways between the involved muscles \((22, 26, 27)\), and regional differences in motor unit activation within a muscle \((5, 25, 41, 43)\). Although EMG recordings can provide information about the relative intensity of muscle activation \((17)\), changes in recording conditions during a fatiguing contraction can confound the assessment of adjustments in muscle activation. For example, Mademli and Arampatzis (21) reported that some of the increase in the amplitude of the surface EMG for medial gastrocnemius during an isometric contraction sustained at 40% maximal voluntary contraction \((MVC)\) force was attributable to increases in fascicle length and pennation angle within the muscle. The purpose of the present study was to compare changes in intramuscular and surface recordings of EMG amplitude with selected architectural features of the elbow flexor muscles during a submaximal isometric contraction that was sustained as long as possible. Some of these data have been presented in abstract form \((36)\).

METHODS

Ten healthy men \((25 \pm 6 \text{ yr})\) participated in the study. The subjects reported no neurological disorders or cardiovascular diseases and no use of medications known to influence neurological function. Body mass was \(72 \pm 5 \text{ kg}\), and height was \(176 \pm 9 \text{ cm}\). Subjects provided written, informed consent before participating in the study, and the Human Subjects Committee at the University of Colorado approved the protocol, which was conducted in accordance with the Declaration of Helsinki. The experimental design and procedures were similar to previous studies \((15, 33–35)\).

Each subject participated in two sessions: 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 left (nondominant) arm abducted slightly and the elbow resting on a padded support. The left forearm was horizontal and neutral with the upper arm vertical. The hand and distal forearm were placed in a modified wrist-hand-thumb orthosis (Orthomax, Newport Beach, CA) that was secured to a force transducer (model JR-3 Force-Moment Sensor, 90.0 N/V JR-3; Woodland, CA; 900-N range). The target torque for the elbow flexor muscles was displayed on a 17-in. computer screen.

Ultrasonography. A B-mode ultrasonographic apparatus (Sonolayer SSH0140A, Toshiba) with a 7.5-MHz linear-array probe \((38\text{-mm scanning length})\) was positioned on the skin to measure selected features of the muscle architecture: the thickness of the long head of biceps brachii, brachialis, and brachioradialis muscles, and the pennation angle of the fascicles in brachialis \((\text{Fig. } 1)\). Minimal pressure was applied to the scanner against the skin so that deformation of the tissues was minimized. Ultrasound measurements were performed before and after the fatiguing contraction in the absence of the EMG electrodes. The utility of the resting measurements is based on the assumption that the reactive hyperemia immediately after task failure provides an index of the changes in the volume conductor during the fatiguing contraction. To ensure the same location for these measurements, landmarks were drawn on the skin overlying the muscles. The thickness of biceps brachii and brachialis was measured by placing the transducer on the anterior aspect of the arm in a sagittal plane and proximal to the crease of the elbow. The transducer was moved slightly in a lateral direction to ensure a clear image of the muscle boundaries and the peristosteal reflection from the humerus. Brachioradialis was measured by placing the transducer over the muscle belly just distal to the crease of the elbow joint and parallel to the longitudinal axis of the forearm. Two ultrasound measurements were made for brachialis: the thickness between the superficial border of the muscle and the humeral surface \(2 \text{ cm}\) from the left-hand edge of the image, and the angle of pennation between the most clearly visualized fascicle and its insertion into the humeral surface. The reliability of the pennation angle measurement for brachialis with
ultrasound is high (12). The thickness of the long head of biceps brachii and brachioradialis muscles was determined as the distance between the lower and upper boundaries. The ultrasound analyses were made offline with digitizing software (Scion Image, National Institutes of Health).

EMG recordings. Surface EMG was measured with bipolar electrodes (Ag-AgCl, 8-mm diameter; 20-mm distance between electrodes) that were placed over the short and long heads of biceps brachii and brachioradialis and the lateral head of triceps brachii. The electrodes were attached on the distal portion of the muscle between the innervation zone and the junction between the muscle and tendon (23). Ultrasound measurements were performed at the same location.

The EMG activity of the long head of biceps brachii, brachialis, and brachioradialis was also measured with intramuscular bipolar wire electrodes. The wire electrodes were inserted between the pair of surface electrodes. The intramuscular electrodes comprised two stain-

![Diagram of muscle anatomy](image)

**Fig. 1.** Representative ultrasonography of intramuscular EMG depth. Ultrasound image (left) of the relaxed long head of biceps brachii and brachialis muscles with the transducer placed on the anterior aspect of the arm in a sagittal plane and proximal to the crease of the elbow. Right shows the parameters measured for each muscle: a, thickness of the long head of biceps brachii; b, thickness of brachialis; α, pennation angle for fascicles in brachialis muscles. Depth of the electrodes: 1 = 1/3a, 2 = 1/3b, 3 = 2/3b.

![Graph of EMG recordings](image)

**Fig. 2.** Representative data obtained during the fatiguing contraction at 20% maximal voluntary contraction (MVC). The interference EMG of the elbow flexor muscles measured by intramuscular (first 5 traces from top) and surface recordings (next 4 traces) increased progressively throughout the contraction. The average force (bottom trace) remained constant, but the fluctuations in the flexion-extension direction increased in amplitude toward the end of contraction. The contraction was sustained for 3.9 min.
less steel wires (100-μm diameter) that were insulated with Formvar (California Fine Wire, Grover Beach, CA). The recording area comprised the cut ends of the wires. A disposable 25-gauge hypodermic needle was used to insert the wires into the muscle bellies at an angle that was parallel to the ultrasound probe. The needle was removed after the wires reached a depth that was determined by ultrasound measurements; the depths were ½ of the muscle cross-section for biceps brachii, and ½ and ⅔ of muscle thickness for brachialis (Fig. 1) and brachioradialis, the latter ~3 cm distal from the elbow crease.

Reference electrodes for each bipolar pair were placed on a bony prominence at either the clavicle or the acromion. The EMG signals were amplified (2,000 X), band-pass filtered (13–1,000 Hz; Coulbourn Instruments, Allentown, PA), and recorded on a computer. Digitizing rate was 2,000 samples/s for the EMG and 200 samples/s for force.

Experimental protocol. MVC torque was measured by the subject increasing the torque from zero to maximum in 3 s and holding the maximal torque for ~3 s. Subjects were provided with strong verbal encouragement during the maximal contraction. There was a rest period of 60–90 s between trials. Additional trials were performed when the peak torques for two of the three trials were not within 5% of each other. The greatest torque achieved during the MVC task was used to determine the target torque for the submaximal contraction. The average EMG amplitudes obtained during the fatiguing contractions were normalized to the maximal EMG values measured during the MVC performed before the fatiguing contraction.

Subjects sustained a submaximal contraction at a target of 20% MVC torque until failure. 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 criteria for task termination were 1) an inability to sustain the torque within 5% of the target value for >5 s or 2) removal of the elbow from the underlying transducer for >5 s without correction, despite strong verbal encouragement. The duration that the task could be sustained was denoted as the time to failure of the task. Immediately after task failure, subjects performed an additional MVC.

Data analysis. All data collected during the experiments were recorded on a computer and analyzed offline using the Spike2 data analysis system (Cambridge Electronic Design, Cambridge, UK). The maximal EMG of the elbow flexor muscles was determined as the average rectified EMG (aEMG) value over a 0.5-s interval that was centered at the peak MVC torque. The EMG activity during the fatiguing contractions was quantified by averaging the rectified EMG over the first and last 30 s of task duration and over 30-s intervals centered at 20, 40, 60, and 80% of task duration. EMG values were normalized to the average EMG obtained during the initial MVC.

Statistical analysis. The dependent variables were target torque, ultrasound measurements, and average normalized EMG amplitude. A two-factor ANOVA (muscle × time) was used to compare the dependent variables of aEMG for the short and long heads of biceps brachii, brachioradialis, brachialis, and triceps brachii during the fatiguing contraction. The rate of change in aEMG of each muscle during the contraction was quantified by the slope of an exponential fit to the data for individual trials. The coefficient e^r was used as an indicator of the exponential rate of increase ([t] = exp(at + b)). Paired t-tests (independent and dependent) with Bonferroni corrections were used as post hoc analyses to test differences among pairs of means when appropriate. Changes in muscle thickness and pennation angle were tested by using paired t-tests. The association between selected variables was examined with regression analyses: 1) change of brachialis thickness and pennation angle; 2) changes in muscle thickness and EMG activity of long head of biceps brachii, brachialis, and brachioradialis; and 2) the exponential rates of increase in the surface and intramuscular EMG amplitudes and the time to task failure. The degree of linear relation is reported as the Pearson correlation coefficient (r).

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 ± SD within the text and tables, and they are displayed as means ± SE in the figures. All statistical analyses were performed with SPSS software (SPSS version 13.0).

RESULTS

This study compared changes in the EMG amplitude of the elbow flexor muscles measured at different depths in selected muscles with the changes in muscle architecture during a
sustained submaximal contraction performed at 20% of MVC torque. The average target torque was 16 ± 2.8 N·m (range: 11.3 to 20.1 N·m), and the time to task failure was 6.5 ± 1.9 min (range: 3.4–10 min). MVC torque declined from 78.7 ± 14.1 to 65.3 ± 14.3 N·m at task failure, which corresponded to a decrease of 17.1% (P = 0.009). EMG amplitude increased progressively during the fatiguing contraction for all subjects, as indicated by the representative data shown in Fig. 2. There was a main effect for time for the amplitude of all surface and intramuscular EMG recordings (P < 0.001) (Fig. 3, Table 1).

As quantified by the exponential coefficient $e^a$ (Table 1), the rate of increase in EMG amplitude for the long head of biceps brachii was greater for the surface EMG recording compared with its intramuscular signal (P = 0.009). Similarly, the rate of increase in surface EMG amplitude of the brachioradialis muscle was greater for the surface EMG than for both intramuscular recordings (P < 0.024). The rates of increase in surface EMG amplitude for the long and short heads of biceps brachii and brachioradialis muscles were similar (P = 0.833), as were the rates for the intramuscular EMGs at $1/3$ and $2/3$ muscle depths for the brachialis muscle (P = 0.24). The rates of increase in the amplitude of the intramuscular signals were similar for all five recordings (P = 0.11).

There was no difference (P = 0.43) in the thickness of the subcutaneous tissue at the recording sites for the long head of biceps brachii (3.84 ± 1.19 mm) and brachioradialis (3.49 ± 1.44 mm). The thickness of the brachialis and brachioradialis muscles after the fatiguing contraction increased by 10 ± 14% and 11 ± 11%, respectively (P < 0.05). There was no change in the thickness of biceps brachii at the distal location of the measurement (P = 0.65; Table 2). The increase in thickness of brachioradialis was associated with the increase in the amplitude of the deep intramuscular brachioradialis EMG (P = 0.007, r = 0.79; Fig. 4B), but it was not associated with either the intramuscular EMG recording at $1/3$ muscle depth (P = 0.58) or the surface EMG recording (P = 0.26). The pennation angle of the brachialis muscle increased by 31 ± 17% (P = 0.003) and was associated with the increases in brachialis thickness (P = 0.012, r = 0.75; Fig. 4A). The changes in brachialis thickness and pennation angle were not related to the increase in the amplitude of its intramuscular signals (P > 0.56). The greater increase in amplitude for the surface EMG compared with the intramuscular signal was not related to the thickness of the subcutaneous tissue for the long head of either biceps brachii (P = 0.28) or brachioradialis (P > 0.41).

There were significant associations between the time to task failure and the exponential rates of increase in surface EMG amplitudes for the short (r = −0.63, P = 0.049) and long (r = −0.69, P = 0.028) heads of biceps brachii and brachioradialis (r = −0.85, P = 0.002) (Fig. 5). In contrast, the associations with the time to failure were not significant between the $e^a$ in intramuscular EMG amplitudes for the long head of biceps brachii (r = 0.04, P = 0.92), brachioradialis at the $1/3$ (r = 0.23, P = 0.57) and $2/3$ depths (r = 0.28, P = 0.5), and brachialis at the $1/3$ (r = −0.38, P = 0.31) and $2/3$ depths (r = −0.49, P = 0.17).

**DISCUSSION**

This study is the first to demonstrate that the noninvasive surface recording is more strongly correlated with the capacity of the individual to sustain a fatiguing contraction than the invasive intramuscular recording. Furthermore, the study is the first to compare two noninvasive techniques (surface EMG and ultrasound) and one invasive technique (intramuscular EMG) with the intramuscular recordings performed at different muscle depths. The main finding of this study was that the rate of increase in the amplitude of the surface EMG for the elbow flexor muscles during a sustained submaximal contraction was greater than that for the concurrently recorded intramuscular signal. The rates of increase in EMG amplitude were not

**Table 1. Average EMG at six time points and its rate of increase during the fatiguing contraction**

<table>
<thead>
<tr>
<th></th>
<th>Start</th>
<th>20% MVC</th>
<th>40% MVC</th>
<th>60% MVC</th>
<th>80% MVC</th>
<th>End</th>
<th>$e^a$</th>
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<tr>
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<td>15 ± 5</td>
<td>18 ± 6</td>
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<td>33 ± 11</td>
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<tr>
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<td>17 ± 4</td>
<td>20 ± 4</td>
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<td>33 ± 10</td>
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<td>16 ± 12</td>
<td>20 ± 16</td>
<td>21 ± 16</td>
<td>26 ± 16</td>
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<td>21 ± 8</td>
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<td>28 ± 9</td>
<td>34 ± 11</td>
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<td>8 ± 5</td>
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<td>1.0013 ± 0.0007</td>
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Values are means ± SD. Average EMG values are given as % maximal voluntary contraction (MVC). $e^a$, rate of increase. *P < 0.05 compared with intramuscular.
statistically different for any of the five intramuscular recordings from three of the elbow flexor muscles or for any three surface recordings from two of these muscles. Although the difference in the rates of increase in the intramuscular and surface signals was associated with an increase in muscle thickness for brachioradialis, there was no such association for the long head of biceps brachii. The time to task failure was strongly associated with the rate of increase in the amplitude of the surface EMG recordings than the intramuscular EMG recordings.

Previous studies have shown that the development of muscle fatigue during a submaximal contraction is typically accompanied by a gradual augmentation of the EMG signal (1, 6, 15, 33–35), suggesting an increase in motor unit activity to compensate for the progressive reduction in the capacity of the contractile proteins to sustain force (1, 3). The increase in EMG amplitude during a sustained, submaximal contraction is largely due to the recruitment of larger motor units as the muscle becomes progressively fatigued (6, 10, 24, 31). The main finding in the present study was that the exponential rate of increase in EMG amplitude was greater for the surface recordings compared with the intramuscular signals. Potential explanations for the greater rate of increase in EMG amplitude for the surface recordings include differences in the recording volume of the respective electrodes, changes in the recording conditions during the fatiguing contraction, and differences in the summation of the motor unit potentials at the two recording locations.

The first possible explanation is that the surface electrodes detected a greater proportion of the motor unit activity and that the intramuscular recordings represented more local changes (4, 5, 18, 19, 29, 43). However, the similar rates of increase for
the two intramuscular signals recorded in each of brachialis and brachioradialis suggest that the smaller recording volumes adequately represented the total motor unit activity during the fatiguing contraction. Rather, another factor is likely responsible for the different rates of increase in EMG amplitude for the surface and intramuscular recordings.

A second possible explanation is a difference in the conduction of the muscle fiber action potentials through the volume of tissues interposed between the source and each set of electrodes. Many factors not related to the activation of the muscle fibers can influence an interference EMG, including the properties of the volume conductor (7, 8, 11). This includes such properties as the shape of the volume conductor, thickness of the subcutaneous tissue, amount of cross talk from nearby muscles, and conductivities of the tissues.

When an individual sustains a submaximal isometric contraction to task failure, the shape of the volume conductor changes due to the shortening of muscle fibers and the gradual occlusion of muscle perfusion (28, 37, 39). Changes in the shape of the volume conductor can be estimated by measuring muscle thickness, which increases as contraction force increases up to moderate levels (13) and during the first part of a sustained, high-force contraction (38). Because the difference in the rates of increase in EMG amplitude for the surface and intramuscular recording was greater during the latter part of the fatiguing contraction in the present study (Fig. 3), the difference between the two signals was likely not due an effect of fiber shortening on the recordings at the two locations. Consistent with this interpretation, the rate of change in EMG amplitude did not parallel the change in either fascicle length or pennation angle in medial gastrocnemius when the plantar flexor muscle sustained an isometric contraction at 40% MVC torque until failure (21).

To assess the influence of interrupting blood flow, the reactive hyperemia (9, 20, 42) was estimated by measuring muscle thickness immediately after the fatiguing contraction. The results of the present study suggest that the change in the shape of the volume conductor as estimated by the changes in muscle architecture after task failure cannot account for the difference between the surface and intramuscular signals because the amplitude of the surface EMG increased more rapidly than that for the intramuscular EMG for the long head of biceps brachii, even though there was no change in muscle thickness at the recording site. Furthermore, the rate of increase in the amplitude of the two intramuscular recordings was similar and less than that for the surface signal for brachioradialis, yet only the rate of increase in the amplitude of the deep intramuscular recording was related to the increase in the thickness of brachioradialis.

Another property of the volume conductor that could contribute to a difference in the rate of increase in amplitude of the two EMG signals is the thickness of the subcutaneous tissue. Both experimental and simulation studies demonstrate that the recording volume for surface electrodes will increase with the thickness of the subcutaneous tissue (2, 7, 40). However, the present study found that the greater increase in the amplitude of the surface EMG for the long head of biceps brachii and brachioradialis was not associated with differences in thickness of the subcutaneous tissue above the recording sites in these two muscles across subjects.

The third possible explanation for the different rates of increase in EMG amplitude for the surface and intramuscular recordings is factors that influence the summation of the motor unit potentials for each interference signal. Although the amplitude of the extracellularly recorded muscle fiber potential decreases with an increase in the distance to the recording electrode (30, 32), the relative decline in amplitude at the more distant recording locations is less during sustained activation (30). As a consequence of these relations, the decrease in the amplitude of muscle fiber action potentials and the summed interference signals are likely greater at recording locations closer to the source. Accordingly, regression analyses used in the present study indicated that only the rates of increase in surface EMG amplitude were associated with time to failure of the fatiguing contraction, whereas the rates of increase in intramuscular EMG amplitudes were not associated with time to failure.

In summary, the rate of increase in surface EMG amplitude provides a measure of the change in muscle activation during a fatiguing contraction that is more strongly associated with the duration that the contraction can be sustained than does the rate of change in the amplitude of an intramuscular recording.

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GRANTS

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