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Original paper

EMG frequency during isometric, submaximal activity: a statistical model for biceps brachii

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The purpose of this study was to develop a statistical model to describe the electromyography (EMG) signal frequency changes during a submaximal isometric contraction. Thirty subjects performed a 30-second isometric contraction of the biceps brachii muscle at 80% of the maximal voluntary isometric force. Surface EMG electrodes recorded electrical activity of the biceps brachii. Zero-Crossing-Rate was calculated to identify EMG frequency shifts. The mean frequencies for every one-second period were used to calculate a linear relationship between frequency and time. A significant relationship ($p < 0.05$) between slope and initial frequency value was identified. The model described EMG frequency changes during submaximal effort of biceps brachii up to 15 seconds. The prediction error was 9.8%. Modifying this equation to initial values of frequency of each participant decreased prediction error to 7.2%. These results demonstrate that despite individual differences between subjects it is possible to derive single equation that describes EMG alterations during submaximal, isometric contractions across a homogeneous group of people.

Key words: *electromyography, biomedical modelling, muscle fatigue, zero-crossing-rate*

1. Introduction

Muscle fatigue is a phenomenon that reduces one's ability to perform physical activity. Formerly, muscle fatigue had been identified as a "failure" point during a contraction when skeletal muscle can no longer generate a required level of force [1]. Recently, the concept of fatigue is rather viewed as a progressive process that starts at the beginning of neuromuscular activity and continues throughout an entire contraction [2]. Muscle fatigue, therefore, is a continuum of physiological events that ranges from minimal initial adaptations to larger final responses when the muscle can no longer contract sufficiently. Under this paradigm, fatigue can be analyzed by observing developing or progressive mechanical and physiological changes that are present before exhaustion [3].

Many physiological changes are associated with the continuous process of muscle fatigue. The increased imbalance between K^+ and Na^+ ions affects the propagation of depolarization waves along the muscle fibers [4]. Limiting the dispersion of action potentials can crucially affect Ca^{2+} release, which will then lead to impairment of the contracting mechanisms within myofilaments. Other factors such as a decrease in pH, or the presence of phosphate ions and other metabolites may also reduce the Ca^{2+} release [5]. Intramuscular pressure rises throughout muscle contraction. Even low-level muscle activation can entirely occlude blood flow in the muscle,

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which reduces the energy supply. Moreover, this process obstructs the removal of metabolic by-products [6], [7]. During isometric contractions, blood flow in the muscle is limited and sustained submaximal contractions cause prolonged, localized ischemia which accelerates the fatiguing process [8]. Measurements of metabolite accumulation can be used as an indicator of muscle fatigue; however, these techniques are often invasive (e.g., muscle biopsy, blood drawing). Therefore this approach is not practical and may pose difficulties during routine measurement of muscle fatigue.

Lactate level or associated pH changes can decrease the action potential velocity (APV), one of the major factors associated with the electromyographic (EMG) frequency alterations [9]–[15]. Other factors such as changes in motor unit synchronization and recruitment [16]–[19] can affect the amplitude and frequency domain of the EMG signals. Ischemia, which can occur even at low levels of muscle contractions [20], leads to distinct metabolite built up and therefore causes the frequency of EMG signals to shift to lower values [8]. Since these physiological responses affect the depolarization characteristics of muscle fibres and since EMG signal characteristics are sensitive to changes in membrane depolarization, EMG signals may be sensitive to these physiological responses. Thus it may be possible to indirectly quantify the continuous physiological process of muscle fatigue through the observation of EMG signal characteristics. EMG frequency shifts have been widely used to examine muscle fatigability [21]–[24]. DEDERING et al. [25] proposed an EMG frequency protocol, based on the initial and final EMG values and their rate of change over time to assess the index of back muscle fatigability. FARINA et al. [26] studied the optimal electrode location to investigate the manifestation of muscle fatigue of the upper trapezius muscle. These authors suggested that proper electrode placement can provide accurate evidence of muscle fatigue. However, these studies are based on subject-specific changes of the EMG signals. That is, individual slopes of the EMG frequency shifts were used to distinguish the fatigability of different muscles. To date no broadly applicable EMG model predicting muscle fatigue across test subjects has been developed.

Since we can assume that physiological changes due to muscle fatigue show similar trends among subjects and they are reflected in the EMG signals, we hypothesize that it is possible to model muscle fatigue across a homogeneous group of subjects using frequency characteristics of the electromyographic signals. Therefore, the purpose of this study was to create a single mathematical equation that will describe EMG signal frequency alterations caused by a fatiguing submaximal isometric contraction in a group of subjects. This resulting model could be useful in creating a marker of muscle fatigue generalized to homogeneous muscle groups based on surface EMG methodology.

2. Methods

2.1. Subjects

Thirty healthy, right-handed, untrained male subjects (mean age of 22.2 ± 2.7 years, mean height of 177.9 ± 5.9 cm, and mean mass of 74.8 ± 8.2 kg) volunteered to participate in this study. All subjects were physically active and had no history of upper limbs injuries. Following a detailed explanation of the purpose and design of the study, each subject signed an informed consent form approved by University of Physical Education in Wrocław, Poland.

2.2. Experimental procedure

The fatigue protocol for this experiment consisted of 30-s isometric, submaximal (80% MVC) muscle contractions of biceps brachii. Prior to the fatigue protocol, each subject completed a warm up on a Biodex dynamometer including 10 full-range elbow movements at a constant angular velocity of 60°/s. Following the warm up period, subjects performed three maximal isometric voluntary contractions of elbow flexors and extensors (MVC), each 3 s in duration, separated by 1-min rest intervals. The mean of the two highest values was taken as 100% torque production during MVC. This value served to calculate the subject-specific 80% MVC. Subjects were given a 10-min rest period before engaging in the fatigue protocol. Participants were asked to generate 80% of MVC of elbow flexor torque for 30 s, while the EMG was recorded. Visual feedback of torque values, presented as bar plots, were provided to all subjects to enable them to maintain a relatively steady torque. Six of the thirty subjects recruited could not maintain the required torque level during the 30-s fatiguing protocol. They were therefore eliminated from the study and the analysis was performed on twenty-four subjects.

2.3. Torque measurement

Dynamometric measurements of torque were conducted on a Biodex Multi-Joint System 3 Pro (Biodex Medical Systems, NY, USA). Participants were seated with their right upper extremity in 15° of shoulder adduction and 90° of elbow flexion. The dynamometer's axis was aligned with the medial-lateral axis of the right elbow joint (lateral epicondyle of the humerus was taken as a point of reference). Dynamometer lever was attached to the distal part of the forearm, a few centimeters above the wrist. Stabilization straps were used as per the recommendation of the procedure manual to minimize extraneous movement of the trunk and lower extremities. Torque signal was stored at a sampling frequency of 100 Hz.

2.4. Surface EMG recording

Bipolar, disposable, pre-gelled Ag/AgCl surface electrodes were placed on the belly of the biceps brachii, at 1/3 length of the line between the fossa cubit and medial acromion, with 20-mm inter-electrode distance. The electrodes were 10 mm in diameter. Additionally, the EMG signals from triceps brachii were recorded during the task from nineteen subjects to identify the existence of the co-activation. Electrodes were placed at the middle of the line connecting the posterior crista of the acromion and the olecranon and lateral to that line. The exact placement of the electrodes followed the recommendations of the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM) [27]. To avoid motion artifacts, cables were fixed to the arm using tape. The reference electrode was placed on the olecranon process of the opposite arm. EMG signals were preamplified, band-pass filtered (10–500 Hz), sampled at 1 kHz with a gain of 1000 (common mode rejection ratio of 115 dB, noise of 4.5 μV RMS) using AMT-8 Octopus System (Bortec Biomedical Ltd, Canada).

2.5. EMG signal analysis

Recorded raw EMG signals were partitioned into 1-s time periods. Signal frequency was calculated for every epoch using the zero-crossing-rate (ZCR) [28]. ZCR has been widely used to study muscle fatigue [29]–[32], and was shown to provide similar results to other muscle fatigue indices like mean and median frequency [33], [34].

To obtain the activation of the triceps brachii muscle, the root mean square (RMS) of the signal amplitude was calculated with bin width of 0.3 s. Subsequently, the RMS was normalized to the RMS of EMG signals recorded during the MVC. All signals were processed by a custom-developed program, written in C++.

2.6. Statistical analysis

A time series analysis was used to process the ZCR data. Coefficients of the mathematical model were assessed using the least squares linear regression (equation (1)) [35]:

$$\hat{f}(t) = \beta_1 \cdot t + \beta_0 + \varepsilon\,, \tag{1}$$

where:

$\hat{f}(t)$ – frequency in each time period,

β_0, β_1 – regression coefficients,

ε – residuals,

t – time period.

The adequacy of the model was verified by: 1) the test of individual regression coefficients (using t-test), 2) the test of significance of regression (using F-Snedecor test) and 3) the assumption of the least squares method. The significance level was set at $\alpha = 0.05$. Random pattern of residuals was observed justifying the application of the least squares method. The assumption of normality of residuals and their homoscedasticity was tested and confirmed using the Durbin–Watson test. Calculated values were verified by computing mean error of prediction, using in every second the following equation:

$$\overline{E}_i = \frac{\sum_{j=1}^{n} \frac{|x_i - \hat{x}_i|}{x_i} \cdot 100\%}{n}\,, \tag{2}$$

where:

$\overline{E}_i$ – mean prediction error,

j – j-th subject,

n – amount of subjects,

i – i-th time period,

x_i – measured data,

$\hat{x}_i$ – calculated data.

An error of prediction below 10% was deemed acceptable [36]. All statistical analyses were performed using Statistica 6.0 (StatSoft inc., OK, USA).

3. Results

3.1. Group analysis

The mean MVC torque for the elbow flexors across subjects was 64.7 ± 12 Nm. Autocorrelation of residuals was observed at the experimental time point of 16 s and beyond. For that reason, only the data from 0 to 15 s was used for analyses. Regression analysis revealed an excellent fit, $R^2 = 0.96$ (equation 3), while simultaneously satisfying all linear regression assumptions (homoscedastidy, absence of autocorrelation and normal distribution of residuals). The results of this analysis are shown in table 1 and the resultant regression equation was

$$\hat{f}_g(t) = -3.24 \cdot t + 190.23 , \quad (3)$$

where:

$\hat{f}_g(t)$ – group frequency in each time period,

t – time.

Table 1. Results of group linear regression analysis for mean ZCR values. DW – Durbin–Watson test results. All results are significant at level $p < 0.05$

R^2	Error	B_0	β_1	DW
0.96	2.93	190.23	–3.24	1.48

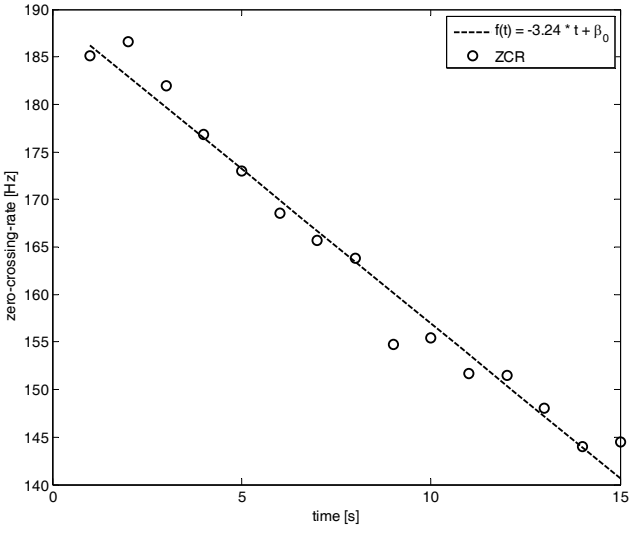

Fig. 1. Mean frequency values during the contraction and their approximation with the linear function. Dashed lines represent 95% confidence intervals

This equation provides the mathematical model

$$\hat{f}_g(t) = -3.24 \cdot t + \beta_0 , \quad (4)$$

where:

$\beta_0 = \frac{Z_{n1} + Z_{n2}}{2}$,

Z_{n1} – ZCR in 1 s,

Z_{n2} – ZCR in 2 s.

Predicted frequency values were calculated from the first two seconds of the EMG signal using equation (4). Differences between modelled and measured values in each time period were then assessed (equation (2)). Model verification showed that mean error ($\overline{E}_g$) increased with time, reaching maximal value of 9.8% at 15 seconds (figure 1). Detailed results are shown in table 3.

3.2. Individual analysis

Linear regression analysis (equation (1)) was performed independently for each subject. Autocorrelation of the residuals was observed in three cases, providing a basis for their exclusion from the individual analysis (see table 2). The minimum and maximum values of the determination coefficient were $R^2 = 0.47$ and $R^2 = 0.93$, respectively.

There was a statistically significant relationship ($p < 0.05$) between coefficients β_1 and β_0 (equations (1) and (5)). The value of the correlation coefficient was $r = –0.72$.

$$\beta_1 = -0.03 \cdot \beta_0 + 2.59 . \quad (5)$$

The adjusted mathematical model, based on relationship between β_1 and β_0, was formulated to describe EMG frequency:

$$\hat{f}_i(t') = (2.59 - 0.03 \cdot \beta_0') \cdot t' + \beta_0' , \quad (6)$$

where:

$\hat{f}_i(t')$ – individual frequency in each time period,

$\beta_0' = \frac{Z_{n1} + Z_{n2}}{2}$,

Z_{n1} – ZCR in 1 s,

Z_{n2} – ZCR in 2 s,

$t' = t - 1$.

Calculated data were verified for every time epoch (equation (2)). Results identified a time-dependent increase in the error of prediction, reaching a maximum value of 7.2% at 15 s (table 3). Figure 2 shows a graphical representation of measured frequencies calculated from the model during fatiguing task.

Table 2. Results of individual linear regression analysis of ZCR shifts. Durbin–Watson test results (DW). All results are significant at level $p < 0.05$. DW values marked with an asterisk (*) identify presence of residual autocorrelation

Subject	R^2	Sd. error	β_0	β_1	DW
1	0.82	7.85	176.67	–3.65	1.81
2	0.93	4.51	201.11	–3.54	2.05
3	0.52	8.20	163.42	–1.84	1.93
4	0.75	6.38	181.92	–2.38	1.93
5	0.58	5.14	158.13	–1.30	1.74
6	0.62	10.05	158.70	–2.75	1.51
7	0.57	12.13	175.79	–3.03	1.31*
8	0.76	14.27	215.44	–5.50	1.83
9	0.84	9.25	206.37	–4.62	2.28
10	0.47	10.80	188.69	–2.21	2.00
11	0.74	8.08	195.75	–2.96	1.76
12	0.72	13.24	210.83	–4.58	2.13
13	0.59	12.14	213.95	–3.14	2.20
14	0.84	8.45	172.58	–4.21	2.84
15	0.73	10.69	244.73	–3.83	1.60
16	0.54	9.01	165.61	–2.09	2.58
17	0.67	8.16	163.66	–2.51	1.71
18	0.80	12.05	225.71	–5.11	1.76
19	0.84	6.41	191.85	–3.14	2.72
20	0.57	14.76	223.70	–3.70	2.03
21	0.71	8.96	182.30	–3.00	1.00*
22	0.60	8.33	155.55	–2.21	0.86*
23	0.76	10.30	213.98	–3.99	2.28
24	0.51	11.71	179.27	–2.58	1.48

The amplitude analysis of EMG signals from triceps brachii revealed subject's specific activity of that muscle during thirty seconds of submaximal, isometric contractions of biceps brachii. In some cases, the amplitude of the signal decreased, remained at the constant level or even increased (see figure 3).

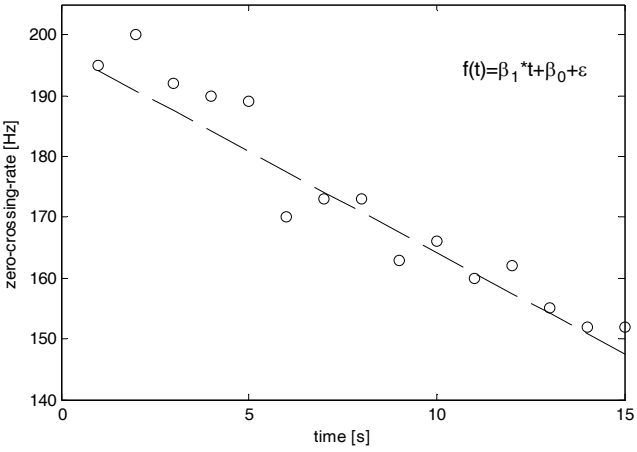

Fig. 2. Example of the individual linear regression analysis result for subject A. Circles represent measured EMG frequency. Dashed line represents EMG signal frequency calculated from the model (7)

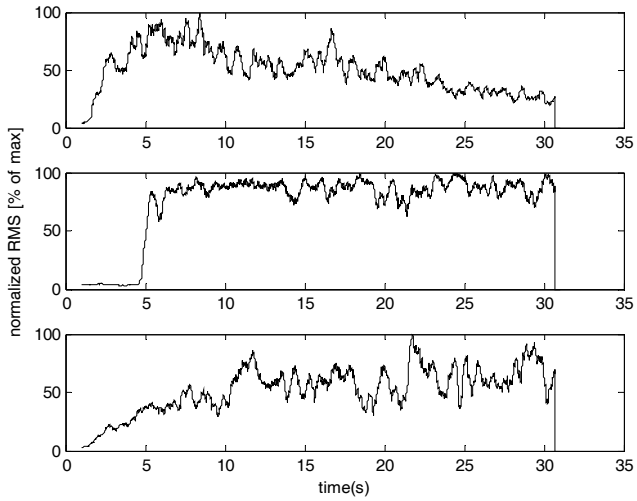

Fig. 3. Subject B (top graph) – RMS of the EMG shows decrease of triceps brachii activity during the task. Subject C (middle graph) – relatively constant RMS during the task. Subject D (bottom graph) – RMS of the EMG increases during the task

Table 3. Mean prediction errors and their standard deviations (SD) in each second for group $\overline{E}_g$ and individual models $\overline{E}_i$. Error was determined between calculated and measured data

	Experimental time periods (s)														
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
$\overline{E}_g$ (%)	4.54	4.60	4.67	4.76	4.91	5.15	5.40	5.67	5.98	6.45	7.00	7.63	8.31	9.04	9.80
SD $\overline{E}_g$ (%)	2.72	2.75	2.89	3.11	3.36	3.58	3.88	4.27	4.70	5.00	5.29	5.57	5.87	6.20	6.58
$\overline{E}_i$ (%)	4.44	4.38	4.32	4.34	4.36	4.46	4.62	4.79	4.97	5.20	5.51	5.83	6.20	6.68	7.19
SD $\overline{E}_i$ (%)	2.74	2.75	2.82	2.83	2.90	2.94	2.96	3.05	3.23	3.42	3.59	3.85	4.15	4.38	4.68

4. Discussion

Based on the results of both group and individual analyses, a constant decrease of EMG frequencies during a sustained, submaximal isometric contraction was observed (see equations (4) and (6)). Similar linear trends were reported by other authors [10], [11], [37]–[40]. However, the novelty of our approach is that we are able to develop one linear equation for a group of multiple subjects.

Since metabolite concentration increases linearly during sustained muscle contractions [41], the strong, linear characteristic of the change in the EMG frequency pattern observed during the experiment can support the hypothesis of their strong relationship with physiological changes during the fatigue process. Some studies presented theoretical models, describing the connection between the slowing of APV and EMG frequency decline [10], [12], [42]. The regression equation presented in this study describes frequency changes for all subjects and suggests that physiological processes that affect EMG signal characteristics have a common, inter-subject tendency.

Initial frequency values (β_0) were calculated from first two seconds of the electrical activity measurement (see equations (4) and (6)). High variability of the frequency values at the beginning of the EMG recording was observed. We did not control the speed of torque production at the start of the contraction. Subjects therefore generated force with different gradients during the torque initiation phase. SBRICCOLI et al. [43] suggest that the order of muscle fiber activation can differ, depending on contraction speed. Authors of that study found a significant relation between contraction speeds and motor unit recruitment strategies. Different recruitment of type I and type II muscle fibers could significantly affect the frequency content of the EMG signals measured at the beginning of the isometric contraction presented in this study. Moreover, elbow flexor muscles do not respond homogeneously to different contraction speeds. KULIG et al. [44] showed that during fast elbow flexion, the biceps brachii was more active compared with the brachialis, while the brachialis was recruited more during slow flexion. Since different force–time gradients can cause altered agonist coactivation, the biceps brachii may have been activated to various levels among the subjects during the initial two-second interval. Therefore, lack of the force ramping unification would increase the variability of electromyographic signal frequencies at the beginning of the EMG recording. To minimize frequency deviations, we decided to use the mean frequency from first two seconds of measurements as the initial frequency for each subject.

From the regression analysis, we noticed increased fluctuation in the ZCR data above 15 s of the contraction. Due to these irregularities, assumptions of the linear regression analysis were not fulfilled. We are not aware of any physiological processes within an individual muscle, which would appear at this particular time and differentiate subjects. This variability between subjects may be explained by different neuromuscular control strategies during highly fatigable sustained contractions. Torque data recorded during the experiment represents force production from all muscles in the elbow flexor group, not only biceps brachii. It is reasonable that other muscles became more active to compensate for the force reduction by the fatigued biceps. This shift in torque production among the elbow flexors would result in periods of reduced force and EMG in the biceps brachii and also variation in biceps force and EMG during the sustained contraction. Furthermore, variation in biceps force would affect blood flow occlusion which would secondarily affect the EMG signal. Therefore, variability of the EMG frequency alterations after 15 s between subjects would depend on subject specific strategies of agonist activation. In this study, we did not collect the EMG signals from other elbow flexors, thus a more profound analysis of this process was not possible.

Altered activation and force production in the antagonist triceps brachii may also have affected the EMG frequency pattern after the initial 15-s period. The analysis of the electrical activity of the triceps brachii showed different activation patterns of that muscle among subjects during the prolonged, isometric biceps brachii contraction. The co-contraction of the antagonist muscle limits the use of the dynamometer to monitoring the maintenance of steady flexor torque production. The dynamometer recorded net torque values generated by elbow flexors and extensors. Increased activity of antagonist muscles leads to increased force production by elbow flexors when the constant level of net torque production is required. Different activation strategies of the triceps brachii (see figure 3) during the experiment led to irregular force production in the biceps brachii among subjects. Processes such as the force control strategy described above would intensify through the contraction interval and manifest when muscle fatigue reached an excessive level. The different co-activation strategies used by individual subjects after 15 s of contraction may partially explain why data from only first 15 s of measurement showed a linear trend over time.

The results of this study indicate that dynamics of the EMG frequency changes are proportional to the initial frequency values. MANNION and DOLAN [45] suggest that initial frequency values provide information about the distribution of the muscle fiber types recruited at the beginning of the contraction. This relationship suggests that the slope of the EMG frequency changes is related to muscle fiber distribution in the muscle. Individual analysis revealed a significant relationship between regression coefficients β_1 (slope) and β_0 (initial frequency) (see equation (5)). After applying this modification to the created model (equation (6)), the accuracy of calculated data improved as validated by a decrease in the mean error of prediction. Slow twitch muscle fibers contain more mitochondria and a higher hemoglobin concentration, which makes them less fatigable. During the high level, sustained isometric contraction, some type II fibers could have been deactivated due to the fatigue process. This may have led to a situation where the majority of the EMG signal comes from slow twitch fibers that generate lower frequency electromyographic signals. Thus, the more fast-twitch fibers activated at the beginning of the contraction (higher intercept values), the more pronounced alterations of the signal frequency would occur (steeper slope).

In conclusion, our study proves that despite individual differences among subjects it is possible to derive one equation to describe EMG alterations during submaximal, sustained, isometric contractions across a homogeneous group of people. The heuristic model showed a strongly linear characteristic of the EMG frequency changes under well-controlled conditions of a fatiguing protocol. Statistical, linear regression analysis was applied to create a mathematical model which was adopted to calculate changes in the frequency content of the EMG signals recorded during submaximal, sustained biceps brachii contractions. Mean prediction error between collected and calculated data was less than 10%. Model modification, based on a revealed relation between initial frequencies and a slope, decreased the error of the calculated data and moreover enabled us to calculate the frequency values up to 15 s, using data from first two seconds of measurement. Initial frequency values can be used to predict EMG alterations in the frequency domain up to 15 s. However, the accuracy of prediction should be validated by comparing calculated data with collected EMG signals that were not used to create the model (i.e., from a new group of subjects). The results of this study may be helpful in using surface electromyography to monitor muscle fatigue process. The resultant mathematical model may also lead to improved control algorithms in EMG-driven devices. Modelling the changes in the control signal could significantly increase the functionality of such devices.

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