



# Interpretation of EMG integral or RMS and estimates of “neuromuscular efficiency” can be misleading in fatiguing contraction

Todor I. Arabadzhiev<sup>a,\*</sup>, Vladimir G. Dimitrov<sup>b</sup>, Nonna A. Dimitrova<sup>a</sup>, George V. Dimitrov<sup>a</sup>

<sup>a</sup> Centre of Biomedical Engineering, Bulgarian Academy of Sciences, Acad. G. Bonchev Str., Bl. 105, Sofia 1113, Bulgaria

<sup>b</sup> Institute of Biophysics, Bulgarian Academy of Sciences, Sofia, Bulgaria

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## ABSTRACT

In occupational and sports physiology, reduction of neuromuscular efficiency (NME) and elevation of amplitude characteristics, such as root mean square (RMS) or integral of surface electromyographic (EMG) signals detected during fatiguing submaximal contraction are often related to changes in neural drive. However, there is data showing changes in the EMG integral ( $I_{\text{EMG}}$ ) and RMS due to peripheral factors. Causes for these changes are not fully understood. On the basis of computer simulation, we demonstrate that lengthening of intracellular action potential (IAP) profile typical for fatiguing contraction could affect EMG amplitude characteristics stronger than alteration in neural drive (central factors) defined by number of active motor units (MUs) and their firing rates. Thus, relation of these EMG amplitude characteristics only to central mechanisms can be misleading. It was also found that to discriminate between changes in RMS or  $I_{\text{EMG}}$  due to alterations in neural drive from changes due to alterations in peripheral factors it is better to normalize RMS of EMG signals to the RMS of M-wave. In massive muscles, such normalization is more appropriate than normalization to either peak-to-peak amplitude or area of M-wave proposed in literature.

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## 1. Introduction

Failure to maintain the required or expected force is referred to as muscle fatigue. For many years there has been an interest in determining the relative contribution of central and peripheral factors to muscle fatigue (Edwards et al., 1995). The finding of linear relation between integrated electromyographic activity ( $I_{\text{EMG}}$ ) and tension in *soleus* muscle before and after fatiguing contraction (Edwards and Lippold, 1956) has attracted attention of explorers to these measures of muscle activity. Moreover, the slope of this relation depended on the level of fatigue. The authors suggested that the progressive increase of electrical activity during continuous isometric contraction of a given submaximal strength is due to recruitment of additional motor units (MUs) necessary to compensate for the decrease of contraction force occurring in the fatigued muscle fibers.

Later, the quantity of the so-called neural energy (Viitasalo and Komi, 1977, 1978), total electrical signal sent from the central nervous system to muscle (Enoka, 1988), changes in the central command (Viitasalo and Komi, 1977, 1978) or in the central drive to motoneurons (Linssen et al., 1993; van der Hoeven et al., 1993; Barton and Hayes, 1996; Carpentier et al., 2001) were often estimated by  $I_{\text{EMG}}$  or root mean square (RMS) of EMG or by their

changes. The authors considered that  $I_{\text{EMG}}$  was the best variable to describe the intensity of neural effect during maintained activity. In line with this opinion, in occupational and sports physiology, the neural adaptation following strength/power training or muscle unloading is estimated on the base of changes in  $I_{\text{EMG}}$  (Moritani and deVries, 1979; Hakkinen and Komi, 1983; Häkkinen, 1994; Higbie et al., 1996; Hortobágyi et al., 1996; Aagaard et al., 2000; Häkkinen et al., 2001, 2003; Aagaard et al., 2002; Deschenes et al., 2002; Sale, 2003; Moore et al., 2004; Gabriel et al., 2006; Seynnes et al., 2007).

Relations between force and  $I_{\text{EMG}}$  were also used for estimating different kinds of “efficiency”. Lenman (1959) and DeVries (1968) used the gradient of the relationship [Force/ $I_{\text{EMG}}$ ] to characterize “efficiency of electrical activity (EEA)”. Mills and Willison (1987) considered that the ratio [EMG/Force] gives a measure of the efficiency of the coupling, that is how much EMG is required to produce a certain force in different conditions. Milner-Brown et al. (1986) and Miller et al. (1987) introduced the term “neuromuscular efficiency” (NME) to designate the ratio [Force/ $I_{\text{EMG}}$ ] at 50% of muscular voluntary contraction (MVC). The authors also considered that the reduction in NME with fatigue indicated that more MUs were recruited to generate the same amount of force compared with the non-fatigued muscle. Deschenes et al. (2002) considered the ratio between the mean force and RMS of each EMG burst as a reliable measure of the NME. Gauthier et al. (1996) and more recently Castaingts et al. (2004) as well as Nicolas

\* Corresponding author. Tel.: +359 2 979 36 10; fax: +359 2 872 37 87.

E-mail address: [tosho@clbme.bas.bg](mailto:tosho@clbme.bas.bg) (T.I. Arabadzhiev).

et al. (2005, 2008) attributed variations of the NME with fatigue and time-of-day also to changes in the neural drive. Kallenberg and Hermens (2006) noted that surface EMG parameters are commonly used to assess the muscle activation level that is imposed by the central nervous system. Thus, there is a general belief that  $I_{\text{EMG}}$  or RMS changes reflect only neural drive alteration. This is irrespective of the fact that the amplitude characteristics of surface EMG signals could increase (Cobb and Forbes, 1923; Bigland-Ritchie et al., 1986a; Löscher et al., 1996; Søgaard et al., 2006), decrease (Stephens and Taylor, 1972; Bigland-Ritchie, 1981) or remain almost unchanged (Merton, 1954; Thomas et al., 1989) with fatigue.

In addition, there are facts pointing to peripheral factors inducing changes in amplitudes of motor unit potentials (MUPs) (Gydiakov et al., 1976). It was also shown that changes in the profile of intracellular action potential (IAP) (the spatial distribution of IAP along muscle fiber membrane) affect amplitudes of single muscle fiber potentials (SMFP) and MUPs (Dimitrova, 1973; Dimitrov and Dimitrova, 1977, 1979; Dimitrova and Dimitrov, 2002, 2003, 2006). As the generation of extracellular potentials in a muscle as a volume conductor is a spatial problem, just the IAP profile determined the generated potentials. One characteristic of the IAP profile is its length defined by the product of IAP duration and muscle fiber propagation velocity (MFPV).

In addition, during a submaximal voluntary contraction, changes in strictly muscle electrical characteristics, M-wave and MUP, could resemble changes in the  $I_{\text{EMG}}$  (Viitasalo and Komi, 1977, 1978). Gandevia (2001) noted that activity-induced changes in the SMFP seriously limit usage of the surface-recorded EMG as a measure of voluntary activation of motoneurons. Hamlin and Quigley (2001a,b) concluded that muscle weakness and increased EMG and [EMG/Force] ratio after eccentric exercise could not be attributed to central fatigue. Farina et al. (2004) and Keenan et al. (2005) also noted that although the amplitude characteristics of the surface EMG increased monotonically with the neural drive to a muscle, the changes in the average rectified or RMS values of EMG and in the NME might not rigorously reflect altered neural drive because of the cancellation of positive and negative phases of MUPs. Consequently, for correct interpretation of the obtained experimental results, it is necessary to discriminate changes in EMG integral or RMS due to alterations in central factors, from those due to changes in peripheral factors (IAP and MFPV).

Although the effect of IAP and MFPV on the extracellular potential duration and amplitude is distance dependent (Dimitrova, 1973; Dimitrov and Dimitrova, 1977, 1979; Dimitrova and Dimitrov, 2002, 2003, 2006), it is natural to assume that the effect should be similar on both EMG and M-wave recorded by the same electrodes. In this sense, the estimations based on the ratio between RMS or integral of interference EMG and M-wave amplitude (Haughton et al., 1994; Löscher et al., 1996; Kent-Braun, 1999; Zory et al., 2005) seem to be perspective for rejecting contribution of the changes in peripheral factors and thus, for distinguishing contribution of the changes in central factors only. However, the evaluations of the results obtained with such estimates vary from: “the use of M-wave normalization for surface EMG amplitude values with either peak-to-peak amplitude or M-wave area, may be the best normalization technique available at this time” (Weir et al., 2006) to “studies using RMS normalized to M-wave amplitude, and, more generally, surface EMG as an index of voluntary drive, should be interpreted with caution” (Place et al., 2007). What is the cause for such differences?

We aimed at testing the reliability of NME assessment, and the possibility of discriminating the contribution of changes in central factors during fatigue to amplitude characteristics of surface interference EMG.

## 2. Methods

NME is usually assessed during contractions at submaximal strength when amplitude characteristics of surface interference EMG signals increase with fatigue. Therefore, in our simulations of surface EMG at various stages of fatigue, block of neuromuscular propagation and loss of muscle fiber excitability were not taken into account. We studied the following EMG amplitude characteristics: root mean square ( $\text{RMS}_{\text{EMG}}$ ), integral ( $I_{\text{EMG}}$ ), and their normalized characteristics. Normalization was performed in respect of different M-wave amplitude characteristics: peak-to-peak amplitude ( $P-P_M$ ), root mean square ( $\text{RMS}_M$ ), or integral ( $I_M$ ). Thus, the normalized amplitude characteristics were:  $\text{RMS}_{\text{EMG}}/P-P_M$ ;  $I_{\text{EMG}}/P-P_M$ ;  $\text{RMS}_{\text{EMG}}/\text{RMS}_M$ ; and  $I_{\text{EMG}}/I_M$ . Both integral and RMS were calculated for the whole simulated periods (2 s for interference EMG signals and 250 ms for M-waves).

### 2.1. Simulation of muscle morphology and recording conditions

The muscle was assumed to have a circular area (20 mm diameter) and comprise a total of 125 MUs grouped in 4 MU types – slow-twitch resistant to fatigue (S), fast-twitch resistant to fatigue (FR), fast-twitch intermediate (FIN), and fast-twitch fatigable (FF). Territories of the MUs had circular shapes. They were spread randomly within the muscular area, with uniform distribution (Stålberg and Dioszeghy, 1991). Adjacent MU territories overlapped. The main parameters of MUs grouped by type can be seen in Table 1. Muscle fibers were also distributed uniformly within each MU territory. The mean length of the muscle fibers was 123 mm with longer semi-length of 63 mm, i.e. end-plates positions were asymmetrical with respect to fiber-ends, like in human *m. biceps brachii*. Fiber ends were scattered normally within 18 mm range. The overall width of the end-plate region was 30 mm, whereas for a distinct MU it was set to 10 mm. Maximal desynchronization (differences in the time of activation of fibers of the same MU due to different motoneuron terminal branches lengths and different synaptic delay) for all fibers was set to 750  $\mu\text{s}$ .

The recording electrode was bipolar, with 10 mm inter-pole distance. The center of the bipolar electrode was located above the longer semi-length, 30 mm away from the middle of the end-plate region. The distance between the muscle axis and electrode was 10, 15, 20, 25 or 30 mm in anisotropic volume conductor ( $K_{\text{an}} = 5$ ), simulating different conditions with respect to the tissue surrounding the muscle. The smallest distance (10 mm) corresponded to electrode positioned at the boundary of muscle territory like in the cases of subcutaneous recording. Other distances corresponded to surface recordings from a muscle with fat layer of different thickness.

### 2.2. Simulation of peripheral fatigue

We simulated an initial stage preceding fatigue (stage 0) and four consecutive stages of muscle fatigue, associated with changes in peripheral factors: IAP and MFPV. The IAP time courses under initial and consecutive stages of fatigue are depicted in Fig. 1. They are based on experimental results obtained by Hanson and Persson (1971). Similar changes of IAP shapes with fatigue were reported by Hanson (1974) for human and rat muscle fibers. In the time domain, the IAP shape was described (Arabadzhiev et al., 2008) by the following analytic function:

$$\text{IAP}(t) = A_1 \cdot [(A_3 \cdot t^3 + A_2 \cdot t^2 - A_4 \cdot A_5 \cdot t - A_5) \cdot e^{-A_4 \cdot t} + A_5] \cdot e^{-A_6 \cdot t} \quad (1)$$

The coefficient values used for the computation of IAP under different stages of fatigue are given in Table 2. Muscle fibers in all MUs

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