Myo-electric fatigue manifestations revisited: power spectrum, conduction velocity, and amplitude of human elbow flexor muscles during isolated and repetitive endurance contractions at 30% maximal voluntary contraction

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Summary. A brief survey of the literature on manifestations of myo-electric fatigue has disclosed a surprisingly sharp conflict between early studies, focusing on neuro-motor regulatory mechanisms, and more recent studies which stress the determinant influence of local metabolism and skewed homeostasis. Favoured explanations concerning changes in the electromyographic (EMG) spectrum were synchronization/grouping of motor unit (MU) firing and conduction velocity (CV) decreases of the action potential propagation. The notion of mutual exclusivity interwoven with these theories prompted us to reinvestigate the EMG of moderate level, static endurance contraction. Ten men in their twenties performed isometric elbow flexion (elbow angle 135°) at 30% maximal voluntary contraction (MVC), and the surface EMG of the brachioradialis (BR) and biceps brachii (BB) muscles was recorded. Initially the CV – determined by cross-correlation – was 4.3 m·s⁻¹ (BR) and 4.6 m·s⁻¹ (BB). At exhaustion the CV of the BR muscle had declined by 33%, roughly twice the decrease of the BB CV. Substantially larger relative median frequency (f_m) reductions of 50% (BR) and 43% (BB) were found. Simultaneously, the root-mean-square amplitudes grew by 150% (BR) and 120% (BB). All changes during contraction reached the same level of significance (P<0.001, both muscles). From the largely uniform relative increases in f_m and CV during the last 4 min of a 5-min recovery period, variations in CV were suggested to produce equivalent shifts in f_m. The gradually increasing discrepancies between relative decreases in f_m and CV during contraction presumably reflected centrally mediated regulation of MU firing patterns (notably synchronization). After the 5-min recovery another 11 endurance contractions were executed, separated by 5-min intervals. The series of contractions reduced the endurance time to one-third of the 153 s initially sustained, while the terminal CV recordings increased by 1.0 (BR) and 0.6 (BB) m·s⁻¹, and the terminal f_m increased by 24 (BR) and 14 (BB) Hz. The relative CV decreased in direct proportion to the endurance time and the f_m decreases varied with the CV; the findings did not support a causal link between CV decrease (signifying impaired fibre excitability) and the force failure of exhaustion.

Key words: Moderate level static contraction – Repetitive endurance loading – Localized muscle fatigue – Conduction velocity – Myo-electric power spectrum

Introduction

Piper (1909, 1912) has investigated the surface electromyogram (EMG) of the forearm finger flexors during strong, semi-static, voluntary contraction. He described two distinct features of the EMG signal of fatigue, i.e.: 1. That the number of “Hauptwellen” (main waves) per s in the EMG decreased from around 50 (the Piper rhythm) to 25–35

2. That, approaching exhaustion, some recordings showed silent phases of absolutely no EMG activity alternating with bursts of myo-electric oscillations.

Cobb and Forbes (1923) have confirmed and extended Piper's findings. They found that powerful static, and dynamic, endurance contractions of the wrist flexors lowered the frequency of the “main waves” in the EMG by 20% and 9%, respectively, while simultaneously increasing their peak-to-peak amplitude by roughly 80%. Edwards and Lippold (1956) have also documented fatigue induced growth in the surface EMG amplitude; as to the raw EMG changes accompanying amplitude increases Scherrer and Bourguignon (1959) have stressed explicitly “the rhythmical sinusoidal form which these events tend to adopt; ... the Piper rhythm occurs with particular clarity far more readily than in un-fatigued muscle”.

Substantial progress was achieved in the field of frequency analysis of the human EMG by the introduction of automatic spectrometers (Walton 1952), digital com-
The EMG, Kogi and Hakamada (1962a, b) have established evidence for an increased synchronization (tendency for grouping) of the motor unit (MU) discharges, which in addition showed a marked increase in irregular fluctuations in their interpulse intervals. A more pronounced synchronization of MU firing with fatigue had already been demonstrated by Buchtal and Madsen (1950) and Lippold et al. (1960), and until the early 1970s spectral compression and synchronization generally were assumed to be causally interrelated (Lloyd 1971), although Lippold et al. (1960) had suggested that the EMG of fatigue might also reflect changes in conduction velocity (CV) and membrane potential of muscle fibres.

However, the speculations concerning the explanation of the compression of the EMG spectrum with fatigue were tremendously influenced by two publications by the Gothenburg group (Mortimer et al. 1970; Lindström et al. 1970). Through spectral dip analysis, the CV of muscle action potentials (AP) was proved to be reduced by 20% to 40% as a consequence of heavy static loading of human elbow flexor muscles (65%–75% of the maximal voluntary contraction (MVC); Lindström et al. 1970). From the conduction time of compound action potentials in cat gastrocnemius and soleus muscles, ischaemia and electrical stimulation was found to produce a reduction of CV similar to the above (Mortimer et al. 1970). Perfusion with de-oxygenated dextran partly (gastrocnemius muscle) or entirely (soleus muscle) abolished the CV decrease. The power spectrum changes with fatigue were considered to arise secondarily to a lowered CV; a metabolic by-product (lactic acid) was thought to affect adversely the propagation capability of muscle fibre membranes.

The linking of EMG spectrum variation to instantaneous changes in the metabolic state brought about by ongoing or previous activity of muscles produced a wealth of studies on myo-electric spectral parameters and fatigue over the next two decades. Yet, the dominating effects of CV changes and lactic acid on the frequency domain modifications have lately been questioned: 1. The CV has appeared not to be the sole contributor to spectral compression in the EMG of fatigue (Broman et al. 1985; Zwarts et al. 1987; Hägg 1991, 1992). The median frequency ($f_m$) decrease of moderate and low-level loading has depended only partly on CV changes (Krogh-Lund and Jørgensen 1991, 1992). 2. Power spectrum variation in fatigue and recovery would appear to follow a time course which weakens the lactic acid hypothesis. While the changes in $f_m$ and pH during static loading have seemed well correlated (Vestergaard-Poulsen et al. 1992), the pH in human muscles have remained near the final low level for 2 min or more at the end of intense, exhausting static and dynamic contractions (Miller et al. 1987; Boska et al. 1990; Juel et al. 1990), in contrast to the substantial $f_m$ restitution which has been found after just 1 min of postexhaustion inactivity (Broman et al. 1985; Krogh-Lund and Jørgensen 1991, 1992). Following a series of intermittent medium load contractions Roy et al. (1991) have reported complete $f_m$ restitution without any detectable accompanying pH recovery.

Although CV decrease only partially dependent on the contraction level and the muscle in question accounts for the spectral compression with fatigue, it is still of interest to ascertain the agent causing CV slowing. Bigland-Ritchie et al. (1979) have hypothesized cation exchanges over the muscle fibre membrane in particular K⁺ accumulation in the extracellular space to explain both the CV decrease and the force failure of fatigue. Evidence in favour of this point of view has subsequently emerged. Fatiguing maximal contractions of the knee extensors of three patients, who were unable to produce lactate due to myophosphorylase deficiency, have provoked a marked decrease in the mean frequency ($f$) of the EMG spectrum (Mills and Edwards 1984). Accumulated extracellular K⁺ due to limited energy resources for the maintenance of membrane integrity was thought to cause the $f$ lowering.

Saltin et al. (1981), Sjøgaard et al. (1985), and Sjøgaard (1988) have convincingly demonstrated voluntary contractions (static as well as dynamic) to bring about a continuous release of K⁺ via the venous blood drained from the active muscles, thus indirectly establishing proof for a load level dependent, and at times considerably elevated, K⁺ concentration in the interstitium of the working muscles. Saltin, Sjøgaard and colleagues have – in line with Bigland-Ritchie et al. (1979) – ascribed the reduction in force generation capacity with fatigue to the K⁺ loss from active muscle fibres. Notwithstanding that it has been found that in vitro K⁺ accumulation in the external medium has reduced the force of electrically stimulated mouse muscles (Juel 1988, his Table 3), the twitch response of single frog fibres on the other hand has been found to be nearly doubled in 14 mM K⁺ Ringer solution (Lännergren and Westerblad 1986), when compared with normal Ringer (2.5 mM K⁺). In addition, no in vivo studies have established the suggested inter-relationship between the extracellular K⁺ accumulation, membrane potential variation, and force failure (and restitution) of fatigue and recovery.

The objectives of the present study were twofold: 1. Our brief survey of the possible causes underlying fatigue induced EMG spectrum changes made further investigation of the EMG of the elbow flexors during a moderate load level (30% MVC) endurance contraction seem reasonable to see if the $f_m$ variation reflected changes in the CV. Moreover, it was thought that the existence of a predictive interdependency be-