

MODELLING THE ELECTROMYOGRAM: FROM CELL TO SYSTEM

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INTRODUCTION

The surface electromyogram (EMG) is a highly complex, structured signal. It contains a wealth of information on both the state of the muscle and on the central nervous system control patterns that govern its activation. Identifying the information that can be extracted reliably from the EMG signal and determining the optimal recording and analysis methods to achieve this, remains a challenge.

Computational modeling can help to shed light on some of these questions. Modeling provides a framework to formulate understanding of the processes involved and allows the effects of variables, which may not be accessible experimentally, to be examined. For over three decades, models of varying complexity have been used to explore the surface EMG signal, typically focusing on a given physical or physiological component [1,2]. Traditionally, muscle fibers have been simulated to lie within relatively simple volume conductors with the action potential represented by a dipole or tripole approximation.

In this paper, we describe a model of the surface EMG signal that aims to capture the underlying physiology and physics from the level of the ion channels in the sarcolemma and transverse tubular system to the motoneuron recruitment and firing patterns that control the behavior of the neuromuscular system. The model incorporates volume conduction properties of the surrounding inhomogeneous tissues, enabling capacitive effects within the tissue and the role of the surface electrode to be quantified. We illustrate how the model may be used to examine how factors such as subcutaneous fat tissue and the surface electrode shape the EMG signal, and to explore phenomena including EMG cross-talk, the possible role of oscillations within the neuromuscular system and the interaction between muscle fiber conduction velocity and inter-pulse interval.

METHODS

The model is comprised of three components: the bioelectric source, the volume conductor representing surrounding tissues and the model of excitation of the motoneuron pool.

The transmembrane action potential is simulated using a model of the propagating muscle fiber action potential that captures the behavior of the ionic currents and the sodium potassium pump in both the sarcolemma and within the transverse tubular system. Temperature scaling of the membrane is incorporated to enable action potential behavior to be simulated across a range of temperatures. The volume conductor in which the muscle fibers are situated is simulated using the finite element method, to enable complex geometries and inhomogeneous tissues, including the surface EMG electrode and electrical double layer at the electrode-skin interface, to be incorporated [3]. The firing times and recruitment profiles of each motor unit are determined using a model of the motoneuron pool in which

each motoneuron is represented by a single compartment threshold-crossing model. Each motoneuron receives three inputs: one common to all motoneurons which determines the level of activation of the muscle; a common oscillatory input, also applied to all motoneurons, and an independent random membrane noise component [4].

Non-propagating start-up and end effects are incorporated by means of current compensation at the neuromuscular and musculotendonous junctions, respectively. The motor unit action potential is calculated as the sum of the action potentials from all constituent fibers of the motor unit and the EMG signal is the sum of the potential detected at the electrode from all motor units.

RESULTS AND DISCUSSION

The EMG model predicts a range of behaviors consistent with *in vivo* and *in vitro* experimental data. These include variations due to accumulation of extracellular potassium during sustained contraction and an interaction between muscle fiber conduction velocity and interpulse interval [5,6].

The electrical properties of fat, skin and bone are observed to alter the shape of the surface motor unit action potentials [7], while the electrode and electrical double layer effectively act to average the potential at the skin surface beneath [8]. Simulation of common presynaptic inputs to the motoneuron pool predict a strong correlation between low-frequency common drive and 0-5 Hz motor unit coherence and between short-term motor unit synchronization and 15-30 Hz coherence, with fluctuations in force more highly correlated with low-frequency oscillatory drives than higher frequency inputs associated with synchronization [3]. Both broad-band common motoneuron inputs and EMG cross-talk can result in significant coherence across a range of frequencies, making it difficult to distinguish between the two when cross-talk may be present [9].

The model has been applied clinically in the development of novel control paradigms for myoelectric prostheses and monitoring upper airway muscle activity in obstructive sleep apnoea. Modeling the system from the ion-channel level through to nervous system drives will enable the model to be applied to examine changes occurring at the muscle level across a wide range of neuromuscular disorders.

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