

O71- Neuromuscular fatigue development during intermittent electrical stimulation of the triceps surae in spinal cord-injured patients.

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The aim of the present study was to examine neuromuscular fatigue development under intermittent electrical stimulation (ES) applied to complete spinal cord-injured subjects. The *triceps surae* was fatigued using a 30-Hz ES protocol composed of three series of five trains. Spinal excitability, muscle excitability and muscle contractile properties were tested before and after every five-train series. Torque evoked by ES significantly decreased throughout the protocol ($P < 0.001$). This decrease was accompanied by an enhancement of muscle excitability ($P < 0.001$), while spinal excitability was not modified by the protocol. The mechanical response was significantly altered during the ES protocol ($P < 0.05$). The results indicated high fatigue development that could be attributed to alterations taking place distally to the muscle membrane.

Keywords: muscle excitability, spinal excitability, contractile properties

INTRODUCTION

Electrical stimulation (ES) is one of the rare techniques permitting the contraction of paralyzed muscles and is used in the rehabilitation context as a technique to supplement for lost functions or to partially counteract the deleterious adaptations that skeletal muscle undergoes after Spinal Cord Injury (SCI). The motor unit recruitment order under ES is different from that observed for voluntary contractions (Jubeau et al, 2007) and metabolic stress is sharply increased (Vanderthommen et al, 2003), leading to marked and early fatigue development. It is widely acknowledged that neuromuscular fatigue can develop at many sites along the pathway from brain to muscle. The multi-factorial character of fatigue has been observed even under ES, applied in healthy subjects, under both high (Boerio et al, 2005) and low (Papaïordanidou et al, 2010) frequency stimulation. Rapid fatigue development is a limiting factor of ES and one of the major reasons why this technique has not yet gained widespread acceptance by clinicians. Dealing with neuromuscular fatigue is thus important for an efficient clinical application of ES and an essential first step is the understanding of the physiological mechanisms of this phenomenon. This should include both muscular and neural mechanisms' investigation. However, fatigue of SCI subjects has until now been studied as a peripheral phenomenon (study of muscle excitability and contractile properties), without taking under consideration spinal excitability's involvement in the development of neuromuscular fatigue. The aim of the present study was to examine the time course of neuromuscular fatigue components, both at the peripheral and spinal levels, under low frequency intermittent ES in persons with complete SCI.

METHODOLOGY

Thirteen complete SCI patients (ASIA A, lesion at the cervical and thoracic level) volunteered to participate in the study, approved by the Nîmes, France, ethics committee for human protection (2008-A00068-47/1). Subjects visited the research laboratory of the Propara Center on two occasions, separated by 2-3 days. During their first visit, the stimulation intensities were identified. A recruitment curve was drawn in order to identify the intensities at which the maximum M-wave (M_{max}) and H reflex (H_{max}) were obtained. The intensity at which maximum torque was evoked by ES (T_{max}) was also determined. On their second visit, the experimental session started with verification of the aforementioned stimulation intensities. Then, the fatiguing intermittent ES protocol was performed (3 x 5 trains, 30 Hz, 450 μ s, 2 s on-2 s off, at an intensity evoking 50% of T_{max}), with neuromuscular tests taking place before (pre) and after every five trains of stimulation (post5, post10, post15). Neuromuscular tests consisted of one train eliciting T_{max} and three twitches delivered at the intensity of the

maximal M-wave and three twitches at the intensity of the maximal H reflex. Torque and electromyographic activity (EMG) of the *triceps surae* muscle were continuously and synchronously recorded during the two sessions. All variables recorded before, during and after the ES protocol were tested using a one-way (time) repeated measures ANOVA [before ES (pre), after five trains (post5), after 10 trains (post10), and after 15 trains (post15)].

RESULTS

Torque developed during the stimulation trains and T_{\max} significantly decreased throughout the protocol ($P < 0.001$). This decrease was significant from post5 and continued for the entire ES session. M_{\max} amplitude increased significantly during the protocol compared with pre values ($P < 0.001$). The amplitude of H_{\max} and H_{\max}/M_{\max} ratio were not significantly modified by the intermittent ES protocol ($P = 0.84$ and $P = 0.2$, respectively). The amplitude of the mechanical response significantly decreased during the protocol ($P < 0.05$). The decrease, although evident from post5, became significant at post15.

DISCUSSION

The aim of the present work was to examine the development of neuromuscular fatigue during intermittent low frequency ES applied to muscles paralyzed by SCI. Main results showed rapid development of neuromuscular fatigue, evident from the first ES bout, which was accompanied by significant alterations in muscle contractile properties, despite enhanced muscle excitability. Spinal excitability was preserved throughout the protocol. The substantial fatigue observed during this study was accompanied by an increase in M_{\max} amplitude, which is an index of the effectiveness of neuromuscular transmission and impulse propagation in muscle fibers (Hicks and McComas, 1989), proving enhanced muscle excitability. The preserved H_{\max} and H_{\max}/M_{\max} ratio throughout the experimental session showed that spinal factors were not implicated in the development of neuromuscular fatigue under this specific intermittent ES protocol. Muscle mechanical response was altered at the end of the protocol, which is consistent with contractile fatigue development. The amplitude of the mechanical response was decreased, giving evidence of impaired cross-bridge force-generating capacity. Increased inorganic phosphate (Pi) is thought to be one of the major causes of peripheral muscle fatigue. Although its direct effect on cross-bridge function is probably rather small in mammalian muscle at physiological temperatures (Allen, Lamb and Westerblad, 2008), increased Pi can reduce filament sensitivity to Ca^{2+} and hence impact force production (Millar and Homsher, 1990). These results give evidence of purely peripheral fatigue development when intermittent ES is applied to paralyzed muscles, caused by impairment of muscle contractile properties, and allow a better understanding of the time-course of electrically induced fatigue.

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