



Presynaptic inhibition mechanisms may subserve the spinal excitability modulation induced by neuromuscular electrical stimulation

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ABSTRACT

This study aimed at deciphering the origins of spinal excitability modulation that follows neuromuscular electrical stimulation (NMES). Ten participants (age: 24.6 ± 4.2 years) performed 2 randomized NMES sessions on plantar flexors with frequencies of stimulations of 20 or 100 Hz (pulse width: 1 ms) at 20% of maximal voluntary contraction (MVC). Before and after each session, the posterior tibial nerve was stimulated to record H-reflex of soleus (SOL), gastrocnemius medialis (GM) and gastrocnemius lateralis (GL). D1 presynaptic inhibition was assessed by conditioning H reflex with prior common peroneal nerve stimulation. Resting H-reflex of SOL decreased after both protocols, but in a greater extent following the 100 Hz session (100 Hz: $-34.6 \pm 7.3\%$, 20 Hz: $-17.1 \pm 3.8\%$; $P = 0.002$), accompanied by an increase of presynaptic inhibition ($+22 \pm 5.8\%$ at 100 Hz vs. $+8 \pm 3.7\%$ at 20 Hz, $P < 0.001$). GM and GL spinal excitability and presynaptic inhibition were also altered after NMES, but in a similarly extent after 20 Hz and 100 Hz protocols. Neuromuscular fatigue following a single session of NMES involves spinal presynaptic circuitry, even at low stimulation frequency. The spinal sensitivity to NMES seems also muscle dependent.

1. Introduction

Neuromuscular electrical stimulation (NMES), consisting to evoke contractions by applying an electrical current over the muscles via surface electrodes, is a successful method to enhance muscle force (Bax et al., 2005) or to maintain contractile activity in paralyzed or immobilized muscles (Sheffler and Chae, 2007). It is now well established that NMES does not only activate the muscle by an efferent pathway (direct activation), but also induces a depolarization of afferent fibers, mainly Ia, leading to muscle activation via spinal loop (indirect activation). Muscle fatigue induced by a NMES session can be associated with neural changes at spinal level (Butler and Thomas, 2003; Duchateau and Hainaut, 1993; Grosprêtre et al., 2017; Papiordanidou et al., 2014). For example, it has been shown that H-reflex amplitude, which reflects the efficiency of Ia-alpha motoneuronal transmission, is drastically decreased following a train of 20 s of high frequency NMES applied over the triceps surae (Wegrzyk et al., 2015). Moreover, the fatigue-induced H-reflex depression has been shown to strongly match the decrease of the electrically evoked torque (Gueugneau et al., 2016). Even if the Ia-motoneuronal transmission can be altered by changes of motoneurons' excitability itself, H-reflex depression may also be explained by alterations of primary afferent discharge. This includes : (i)

changes in the excitability threshold of the sensory axons leading to a decreased synaptic inputs to motoneurons and (ii) increased presynaptic inhibitions due to homosynaptic postactivation depression of the Ia terminals (HPAD) (Hultborn et al., 1987) and primary afferent depolarizing interneurons (PAD) (Rudomin and Schmidt, 1999). However, the contribution of the presynaptic inhibition mechanisms to the alteration of the Ia-alpha motoneuron transmission efficiency following NMES has never been investigated.

The purpose of the present study was to investigate the changes in spinal circuitry properties following a NMES session applied to the triceps surae muscles. Specifically, the presynaptic circuitry was tested by conditioning H-reflexes from the triceps surae with a prior stimulation of the tibialis anterior Ia afferent projecting on the PAD interneurons. Indeed, the use of a conditioning-test interval of ~ 21 ms specifically acts on the Ia-Mn α transmission and thus allow to precisely track modulations of the presynaptic pathway (Achache et al., 2010; Faist et al., 1996). The forthcoming results should improve our knowledge of spinal mechanisms associated to neuromuscular fatigue following a NMES session. Because both stimulation intensity and frequency of NMES may influence the spinal circuitry response (Grosprêtre et al., 2017; Lagerquist and Collins, 2010; Martin et al., 2016; Neyroud et al., 2014; Wegrzyk et al., 2015), two different NMES

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Nomenclature		M_{atH}	amplitude of the sub-maximal M-wave that precedes the H-reflex
<i>List of abbreviations</i>		M_{max}	maximal M-wave amplitude
GM	gastrocnemius medialis muscle	MVC	maximal voluntary contraction
GL	gastrocnemius lateralis muscle	NMES	neuromuscular electrical stimulation
H_{cond}	conditioned H-reflex with prior common peroneal nerve	PAD	primary afferent depolarization
HPAD	homosynaptic post-activation depression	SOL	soleus muscle
H_{test}	unconditioned H-reflex	TA	tibialis anterior muscle

protocols performed at similar total force–time integral, i.e. similar muscle work, were compared (20 Hz with high stimulation intensity vs. 100 Hz with low stimulation intensity). It was hypothesized that both stimulation protocols would alter triceps surae H-reflex, due to an increase in Ia presynaptic inhibition, but in a greater extent for 100 Hz compared to 20 Hz NMES.

2. Method

Ten healthy participants (7 males and 3 females; age: 24.6 ± 4.2 years old; height: 1.76 ± 0.1 m; mass: 73.1 ± 10.8 kg), with no history of neurological or muscular disorder, gave written informed consent to perform three experimental sessions. The participants committed not to engage in any unusual training or exercise program during the whole duration of the study and were told to avoid any intense exercise prior and after each session. The experimental design of the study was approved by the Regional Ethics Committee (CPP EST A00064-49) and conducted in conformity with the latest version of the Declaration of Helsinki.

2.1. Experimental design

Each participant took part in one familiarization and two experimental sessions conducted to test two NMES protocols with different stimulation frequencies and intensities: 20 Hz (high stimulus intensity) and 100 Hz (low stimulus intensity). The sessions were separated by 2–7 days and randomly performed. The first session lasted (20–30 min) and was performed to familiarize the participants with electrical stimuli at both nerve and muscle levels. The two following experimental sessions lasted about 2 h, and were both designed as follow (see also Fig. 1).

After skin preparation and electrode positioning, subjects sat on an isokinetic dynamometer (Biodex System 3, Shirley, NY). Dynamometer axis was aligned with the external malleolus of the right leg. Subjects were placed with hip and knee joints at 90° ($180^\circ =$ full extension), and ankle joint at 90° (angle between the leg and the sole of the foot). The trunk was stabilized by two crossover shoulder harnesses.

PRE-measurements. Recruitment curves were first established to determine nerve stimulation intensities needed to evoke H-reflexes and M-waves responses of triceps surae muscles. Presynaptic inhibition was assessed at rest by conditioning H-reflex stimulations with prior stimulations of the nerve of antagonist muscles. Then, after a warm-up of 8–10 submaximal contractions, participants were asked to perform two maximal voluntary contractions (MVC) of 2–3 s separated by at least 1 min rest. For each session, the intensity of NMES was determined to evoke a torque corresponding to 20% of MVC.

NMES PROTOCOL. Each session consisted in evoking intermittent contractions of the triceps surae muscle (6 s ON/6 s OFF) at constant NMES intensity during the whole session. Torque signal was recorded continuously during the protocol and integrated on line by a homemade electronic device, providing instantaneously the torque time integral value during the protocol. Independently of the protocol tested, 40 contractions were always evoked in the first session. For the second session, in order to keep constant muscular solicitation, the total torque-time integral was matched with the value obtained during this first session (Grosprêtre et al., 2017).

POST-measurements. Immediately after the end of the last train, one maximal M wave, four conditioned and unconditioned H-reflexes were elicited at rest in randomized order. After those neuromuscular assessments at rest, one MVC was subsequently performed to account for global fatigue.

2.2. Electromyographic recordings

EMG activity was recorded from the three triceps surae muscles (Soleus, SOL; Gastrocnemius Medialis, GM; Gastrocnemius Lateralis, GL) and from one muscle of the tibial compartment (tibialis anterior, TA). After shaving and dry-cleaning the skin with alcohol to keep low impedance ($< 5 \text{ k}\Omega$), EMG signals were obtained by using two silver-chloride surface electrodes (8mm diameter, center-to-center distance: 2 cm) placed over the muscle bellies. The common reference electrode was placed in a central position between the stimulation and recording sites. Electrode placements were marked on the skin with indelible ink to ensure same positioning between sessions.

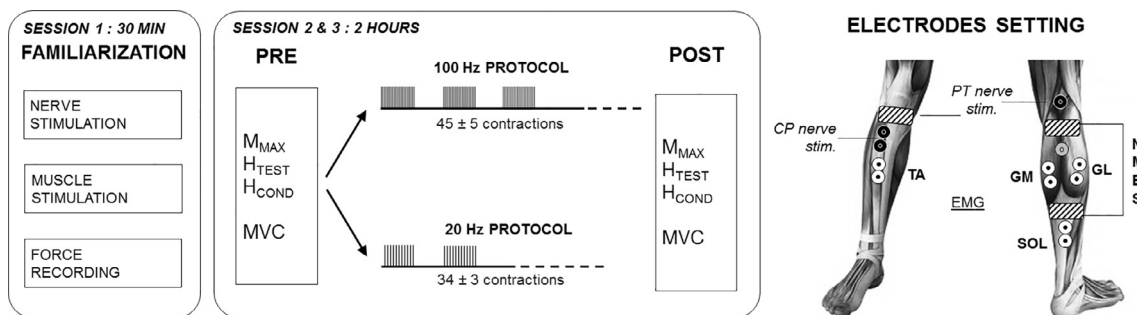


Fig. 1. Diagram of the experimental protocol and electrodes (recording and stimulating) positioning. MVC: Maximal Voluntary Contraction. Mmax: maximal M-wave. Htest: unconditioned H-reflex. Hcond: conditioned H-reflex. CP: Common Peroneal nerve. PT: Posterior Tibial nerve. TA: Tibialis Anterior. GM: Gastrocnemius Medialis. GL: Gastrocnemius Lateralis. SOL: Soleus. NMES: Neuromuscular Electrical Stimulation. White circles depict EMG recording electrodes, black circles stimulating electrodes and grey circle the reference electrode. All responses were evoked at rest randomly before MVC assessment.

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