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Muscle force up to 50% of maximum does not affect cutaneous silent periods in thenar muscles

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Abstract

Objective: The cutaneous silent period (CSP) is a spinal inhibitory reflex mediated by A-delta fibers. The amount of muscle contraction has been reported to influence CSPs, but this has not been confirmed in studies applying clinically feasible stimulus parameters.
Methods: Fourteen healthy subjects underwent comparison of CSPs while contracting the target muscle at different levels ranging from 10% to 50% of the maximum force, which was continuously monitored with a force transducer. Rectified surface electromyographic (EMG) recordings were obtained from abductor pollicis brevis muscle following ipsilateral recurrent noxious digit 2 and digit 5 stimulation.
Results: Group average CSP onset and end latency, CSP duration, and the magnitude of EMG suppression were not influenced by volitional muscle contraction ranging from 10% to 50% of the maximum. In contrast, background EMG levels increased significantly with the amount of muscle force.
Conclusions: CSPs prove to be robust nociceptive cutaneomuscular reflexes, which are – in contrast to previous reports – not significantly influenced by clinically easily applicable amounts of muscle force.
Significance: Non-dependence on the amount of volitional target muscle activation over a range from 10% to 50% of individual maximum force levels renders CSPs particularly suitable for clinical use in patients with peripheral and spinal disorders affecting the A-delta fiber system.

Keywords: A-delta fibers; Cutaneous silent period; Exteroceptive suppression; Muscle force; Normal values

1. Introduction

Exteroceptive electromyographic (EMG) suppression in small hand muscles following noxious digital nerve stimulation has been termed cutaneous silent period (CSP) (Uncini et al., 1991; Leis et al., 1992). The CSP is a spinal inhibitory reflex mainly mediated by A-delta fibers (Uncini et al., 1991; Leis et al., 1992; Kofler, 2003). Upper limb CSPs represent the inhibitory part of a complex defensive reflex mechanism serving to protect the hand from harmful stimuli (Inghilleri et al., 1997; Leis et al., 2000; Kofler, 2003; Kofler et al., 2004). Concomitant excitatory withdrawal flexor reflexes operate in parallel to retract the hand from the source of the noxious stimulus (Floeter et al., 1998; Rossi et al., 2003). Similar reflexes have recently been described in the lower limb (Svilpauskaite et al., 2006). Painful stimuli are necessary to effectively elicit both inhibitory and excitatory reflex components, resulting in an efficient strategy of the central nervous system by simply "turning off" and "turning on" muscle synergies (Leis et al., 2000). Additionally, low threshold afferents have

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been implicated in CSP generation (Serrao et al., 2001), particularly in certain finger–muscle combinations, e.g. index finger and thenar muscles (Kofler, 2003). However, different spinal circuitry seems to be involved in mediating separate inhibitory reflexes elicited by high- and low-threshold afferents (Serrao et al., 2001; Floeter, 2003).

An inverse relationship has been suggested between the amount of voluntary muscle activation and EMG suppression during the CSP (Uncini et al., 1991; Shefner and Logigian, 1996; Serrao et al., 2001). Hence EMG suppression should increase with less muscle activation, but the opposite has been observed in patients with centromedullary cervical spinal cord lesions, who also presented with mild paresis due to corticospinal tract involvement (Štetkárová et al., 2001; Kofler et al., 2003). Influence of force on EMG suppression, if present, would critically limit the usefulness of CSP testing in patients with paresis, who are investigated for suspected A-delta fiber system dysfunction, since they may experience difficulty in maintaining constant high levels of muscle contraction.

In our experience, however, muscle force does not seem to be such a critical factor (Kofler and Poustka, 2005). We therefore assessed and quantified the influence of muscle force on various CSP parameters in a systematic study applying different levels of voluntary muscle contraction, as well as different stimulation intensities and sites likely to be used in the clinical setting, as they are innervated by different peripheral nerves and roots.

2. Materials and methods

Fourteen healthy subjects (6 men, 8 women; mean age 39.9 ± 7.6 years) without history of neurological or psychiatric disorders granted informed consent for multiple recordings of CSPs. The dominant side was investigated in 13 right-handed and 1 left-handed subjects. They were seated in a comfortable chair with the forearm resting upon an adjustable arm support. The elbow joint was maintained at a 90° angle with the hand held in a slightly supinated position and with the fingers extended. The subjects were then asked to perform a thumb abduction as if "hitch-hiking", but the movement was blocked by a force transducer consisting of a piezo-electrical element ("Interface" by MFG, Scottsdale, AZ, USA) mounted over the arm support. Routine electrodiagnostic equipment (Viking IV, Nicolet Biomedical, Madison, Wisconsin, USA) was used in all experiments.

Averaged rectified electromyographic (EMG) recordings were obtained from thenar muscles with surface electrodes attached in a belly-tendon fashion. Maximum voluntary thumb abduction was determined three times over 5 s with the force transducer and was monitored both by visual feedback of the applied force in kiloponds on a display and by audio feedback of the EMG signal.

Electrical stimulation with constant current square waves of 0.5 ms duration was applied to the tip of either digit 2 (D2) or digit 5 (D5) at a stimulation rate of 0.7 Hz, and sensory thresholds were established in each subject as previously described (Kofler, 2003).

CSPs were recorded in thenar muscles during four separate experimental sessions: following D2 stimulation with an intensity of 10 times individual sensory threshold (experiment 1, D2-10ST), followed by stimulation with an intensity of 20 times sensory threshold (experiment 2, D2-20ST); and following D5 stimulation with an intensity of 10 times sensory threshold (experiment 3, D5-10ST), followed by stimulation with 20 times sensory threshold (experiment 4, D5-20ST). Stimulation with 20ST was chosen because this was previously estimated to be close to individual pain thresholds (Kofler, 2003), or mildly painful based on values of 4.5 ± 2.4 on a visual analogue scale ($0 =$ no pain; $10 =$ most severe pain) (Kofler, 2004). Stimulation with 10ST was selected to seek a potential influence of muscle force on EMG suppression when applying non-nociceptive stimuli. Experiments 3 and 4 were performed after a break of several hours ($n = 7$), or on another day ($n = 7$), in order to avoid muscle fatigue.

In each experiment 1–4, subjects were asked to maintain constant isometric thumb abduction with either 10%, 20%, 30%, or 50% of their maximum voluntary contraction force (MVC) as monitored by audiovisual feedback. Subjects were encouraged to interrupt the recording whenever they felt tired; however, no formal attempt was undertaken to quantify muscle fatigue. The sequence of different muscle forces was randomized and balanced across subjects within each experimental condition.

Each CSP recording included two times 25 responses with sweeps of 500 ms, filtered (30–10,000 Hz), amplified, rectified, averaged, and superimposed to ensure reproducibility. CSP parameters were assessed as previously published (Kofler, 2003; Kofler et al., 2004). Briefly, a CSP was defined by a drop in rectified EMG activity at appropriate latencies below a projection line indicating 80% of the mean baseline EMG level during a 100 ms period preceding the stimulus. Measurements were taken of CSP onset and end latency, CSP duration, and index of suppression (i.e. average rectified EMG amplitude during the CSP divided by the average rectified baseline EMG amplitude $\times 100$).

EMG activity frequently interrupted the CSP due to an excitatory transcortical long-loop reflex (LLR) thereby separating an earlier (I1) from a later (I2) phase of EMG inhibition (Kofler, 2003). CSP duration was then measured from the beginning of I1 to the end of I2. Occasionally, a large LLR was superimposed over the beginning of the CSP, thereby rendering it impossible to measure the true CSP onset, and seemingly delaying its onset. Such a finding was termed "late-onset CSP", in order to differentiate it from "early-onset CSPs" with the onset of EMG suppression consistent with the uncompromised presence of an I1 phase (Kofler, 2003). "Late-onset CSPs" were excluded from statistical analysis, in order not to contaminate the results with "artificially" delayed CSP onset values.