ELSEVIER

Clinical Neurophysiology 118 (2007) 999–1009

www.elsevier.com/locate/clinph

Effect of low-level clenching and subsequent muscle pain on exteroceptive suppression and resting muscle activity in human jaw muscles

Tetsurou Torisu [a,b], Kelun Wang [a], Peter Svensson [c], Antoon De Laat [d], Hiroyuki Fujii [b], Lars Arendt-Nielsen [a,*]

[a] *Center for Sensory-Motor Interaction (SMI), Laboratory for Human Experimental Pain Research, Aalborg University, Fredrik Bajers Vej 7 D-3, DK-9220, Aalborg, Denmark*

[b] *Division of Removable Prosthodontics and Management of Oral Function, Graduate School of Biomedical Sciences, Nagasaki University, Nagasaki, Japan*

[c] *Department of Clinical Oral Physiology, School of Dentistry, University of Aarhus, Aarhus, Denmark*

[d] *Cluster of Oral Physiology, Department of Oral and Maxillofacial Surgery, Catholic University Leuven, Leuven, Belgium*

Accepted 11 November 2006

See Editorial, pages 951–953

Abstract

Objective: To investigate the effects of muscle fatigue induced by low-level isometric jaw-clenching and subsequent glutamate-evoked muscle pain on the exteroceptive suppression (ES) response and resting electromyographic (EMG) activities in human jaw muscles.
Methods: The resting EMG activity and the ESs were recorded before (baseline), after low-level jaw-clenching (Post1), after subsequent glutamate or isotonic saline injections into the left masseter (Post2), and 60 min after the clenching (Post3) in 23 healthy volunteers.
Results: The late ES (ES2) showed more inhibition at Post1 compared with baseline ($P < 0.05$). It was less inhibited after both types of injections (Post2), and increased at Post3 again ($P < 0.05$) with no significant difference between the glutamate and isotonic saline sessions. The resting EMG activity increased at Post1 and Post2 ($P < 0.05$). The glutamate injection further increased the resting EMG activity in the injected muscle ($P < 0.01$).
Conclusions: Muscle fatigue influences inhibitory reflex pathways in jaw-closing muscles and subsequent acute muscle pain potentiates the local increase in the resting EMG activity of the painful muscle.
Significance: Muscle fatigue which can be observed in patients with oral dysfunctions may interact with nociceptive regulation and influence the clinical presentation of jaw symptoms and function.

Keywords: Muscle fatigue; Experimental pain; Exteroceptive suppression; Sustained contraction; Descending inhibition

1. Introduction

Parafunctional behaviors, especially jaw-clenching and grinding, have frequently been associated with temporomandibular disorders (TMD) (Olesen, 1988; Glaros et al., 1998; van der Meulen et al., 2006). Low-level parafunctional jaw-clenching was suggested to be a contributing factor for at least some types of TMD pain (Svensson et al., 2001; Glaros et al., 1998). However, these experimental data cannot be used directly to support a "vicious cycle" between muscle activity and pain, because there is little evidence to support that jaw-muscle fatigue or pain can lead to sustained muscle hyperactivity and reflex-mediated spasms in the muscles (Lund et al., 1991).

Intra- or peri-oral stimulation produces one or two successive suppressions of voluntary masseter and temporalis muscle activity in humans (Dubner et al., 1978; Cadden and Newton, 1988; Yu et al., 1973; Cruccu and

* Corresponding author. Tel.: +45 96358830; fax: +45 98154008.
E-mail address: LAN@smi.auc.dk (L. Arendt-Nielsen).

1388-2457/$32.00
doi:10.1016/j.clinph.2006.11.311

Romaniello, 1998). These have been termed the early and late exteroceptive suppressions (ES1 and ES2, respectively). ES1 and ES2 are generally assumed to be produced by activation of brainstem interneurons which inhibit the motoneurons of jaw-closing muscles (Cruccu et al., 1989; Cruccu and Ongerboer de Visser, 1999), and it has been suggested that ES responses can provide valuable information on brainstem pathology (Cruccu and Deuschl, 2000; Cruccu et al., 2005).

On the other hand, it is well known that descending inhibition from mesencephalon and caudal brainstem plays an important role in endogenous pain control, and that stimulation of these regions can inhibit the responses in both nociceptive neurons and non-nociceptive neurons as well as the jaw-opening reflex in animals (Oliveras et al., 1974, 1975, 1977; Dostrovsky et al., 1982, 1983; Sessle and Hu, 1981). It is also suggested that central descending influences can modify jaw reflex mechanisms including ES responses (Dubner et al., 1978). Modifications in ES2 may reflect nociceptive inhibitory processes (Bär et al., 2003) although the functional implication is not fully understood. Indeed, the ES responses in human jaw muscles are influenced by numerous experimental and clinical conditions, for example, during ongoing experimental jaw-muscle pain (Wang et al., 1999; Svensson et al., 1999), headache (Bendtsen et al., 1996; Schoenen et al., 1987; Tataroglu et al., 2002), TMD (De Laat et al., 1985) and mental stress (Cadden et al., 1999; van der Glas et al., 2000) which could be mediated by presynaptic pathways (Wang et al., 1999). However, the effect of muscle fatigue on the ES responses is still unknown.

Jaw-muscle exercise is sometimes accompanied by muscle pain (Svensson et al., 2001; Glaros et al., 1998) and it has been reported that pain thresholds can be modified in relation to exercise pointing towards other aspects of the sensory-motor integration (Whiteside et al., 2004; Koltyn and Arbogast, 1998). Both muscle fatigue and pain are collateral effects of exercise (Koltyn, 2002; Svensson et al., 2001), and from a basic point of view both muscle pain (Graven-Nielsen and Mense, 2001) and fatigue (Kalezic et al., 2004; Garland, 1991; Brunetti et al., 2003; Pettorossi et al., 1999) are mediated through activation of group III and IV muscle afferent fibers. Therefore, muscle fatigue might also reduce ES response (Wang et al., 1999), and the modulation may be related with the level of descending inhibition.

There is a theoretical relationship between muscle fatigue, muscle pain and peripheral glutamate levels in the muscle because fatiguing and potentially painful exercises can evoke a release of glutamate (Lieber and Fridén, 2002; Gleeson et al., 1995; Omote et al., 1998). On the other hand, it is reported that increases in glutamate content and in the number of glutamate receptor in peripheral regions contribute to enhancement of pain (Carlton, 2001). Therefore, muscle pain evoked by direct glutamate injection was used in this study to investigate whether the excitatory amino acid glutamate has specific effects on ES responses under the condition with fatigue.

It has been reported that in muscle pain condition, muscle EMG activity decreases when the muscle is acting as an agonist and increases when it acts as an antagonist (Lund et al., 1991). However, there are few reports about resting muscle EMG activity of jaw muscles after muscle fatigue. At high levels of limb-muscle fatigue, fatigue reduces the monosynaptic reflex activities by presynaptic suppression of Ia excitation evoked by group III and IV afferents (Kalezic et al., 2004; Gandevia, 2001; Garland, 1991; Brunetti et al., 2003; Pettorossi et al., 1999). On the other hand, low-level fatigue has also been demonstrated to enhance the monosynaptic reflex activities (Pettorossi et al., 1999). Therefore, it is still unclear whether resting muscle EMG activity in jaw-closing muscles decreases or increases after a fatiguing condition induced by low-level jaw-clenching.

The aim of this study, therefore, was to investigate the effects of muscle fatigue induced by low-level sustained isometric jaw-clenching and subsequent glutamate-evoked experimental muscle pain on the ES response and resting EMG activity in healthy human and to investigate whether glutamate has specific effects on a fatiguing condition.

2. Materials and methods

2.1. Subjects

Twenty-three healthy volunteers, 11 women (25.5 ± 1.0 years) and 12 men (23.5 ± 0.9 years) (mean $\pm$ SEM), participated in this study. The subjects did not have signs or symptoms of TMD (Dworkin and LeResche, 1992), or daily headache (less than 12 days per year), or were aware of excessive parafunctional activities such as jaw-clenching. Four women reported to use oral contraceptives, but otherwise none of the subjects took any medication. Women were not tested in any particular phase of their menstrual cycle to exclude the effects of the cycle on the pain sensations (LeResche et al., 1997). The study was approved by the local Ethics Committee. All individuals gave their informed consent in accordance with the Helsinki Declaration, and understood that they were free to withdraw from the experiment at any time.

2.2. Experimental protocol

The experimental protocol is illustrated in Fig. 1. All subjects participated in two randomized sessions with glutamate (Glu) or isotonic saline (Iso) injections at least a one-week interval. To evoke muscle fatigue, the subject was asked to perform low-level sustained isometric jaw-clenching for 30 min at 10% maximum voluntary contraction force (MVCF) controlled with visual force-feedback (Svensson et al., 2001). After the end of the sustained jaw-clenching a glutamate (or isotonic saline) injection was carried out to induce muscle pain. The resting EMG activity, the EMG activity during light jaw-clenching, the ES responses and pressure pain threshold (PPT) at the left finger were recorded before the sustained jaw-clenching