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Excitability of the central masticatory pathways in patients with sleep bruxism

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HIGHLIGHTS

- An abnormal excitability in the central masticatory pathways in SB.
- Normal excitability of the corticobulbar pathways in SB.
- Reduced excitability of the brainstem reticular circuits in SB.

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ABSTRACT

Since sleep bruxism (SB) is defined as a stereotyped movement and mainly associated with rhythmic masticatory muscle activity, the aim of this study was to get a better understanding on the subcortical and cortical networks related to the excitability of the central masticatory pathways in SB patients. Of 26 SB patients (12 females and 14 males; mean age: 24.9 ± 4.0 years) and 30 normal subjects (18 females and 12 males; mean age: 24.1 ± 3.1 years) selected, the motor evoked potentials (MEPs) elicited by transcranial magnetic stimulation (TMS) in masseter muscles, and the masseter inhibitory reflex (MIR) elicited by magnetic stimulation with single and double-shock techniques were studied. The MEPs elicited by TMS were similar in both SB patients and normal subjects. As for the MIR elicited by single magnetic stimulation, the latency and duration of the early silent periods (SP1) between the two groups were similar; but in 5 patients the late silent periods (SP2) was absent, and this difference in the frequency of absence of the SP2 between SB patients and normal subjects was significant; with double-shock technique, the recovery of SP2 was significantly lower in SB patients compared to normal subjects. These results suggested an abnormal excitability of the central masticatory in SB patients; and it is also indicated that SB may be mainly under the influence of brainstem networks rather than that of cortical networks.

1. Introduction

Sleep bruxism (SB) is an oromandibular behavior that is defined as a stereotyped movement disorder occurring during sleep and characterized by tooth grinding and/or clenching [1]. In normal subjects, SB is reported by 8% of the adult population [27], and the consequences of SB include tooth wear, jaw pain, headaches, limitation of mandibular movement, as well as failures of dental prostheses and implants [14,25].

SB is mainly associated with rhythmic masticatory muscle activity (RMMA), which is probably an extreme manifestation of a masticatory muscle activity occurring during the sleep of most normal subjects, as RMMA is observed in 60% of normal sleepers with absence of grinding sounds [19]. A neuroimaging study suggested an association between bruxism and a dysfunction in the central regulation of jaw movements [22]. However, the etiological and pathophysiological mechanisms underlying SB are not completely understood. In the past, peripheral factors, like occlusal discrepancies and deviations in bony structures of the orofacial region, were considered the main causes for bruxism [24]. Nowadays, these factors are thought to play only a limited role, and recently obtained evidence have convergently suggested that the development of SB could result from pathological changes in central factors [15,20]. A polysomnography study suggested that the onset of rhythmic masticatory muscle activity and SB episodes during sleep were under the influences of brief and transient activity of the brainstem arousal [18]. Another magnetoencephalography study indicated that SB patients had significantly

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larger event-related desynchronization in the somatomotor area than healthy subjects [16]. It was also found that rhythmic jaw movements could induced by repetitive stimulation to the primary face motor cortex in nonhuman primates studies [13].

Accordingly it is assumed that SB patients might exhibit dysfunction of the motor-related subcortical and cortical networks that controlled orofacial motor behavior and masticatory neuromuscular system.

In the present study, with the aim of a better understanding on the subcortical and cortical networks related to the excitability of the central masticatory pathways in SB patients, the motor evoked potentials (MEPs) elicited by transcranial magnetic stimulation (TMS) in masseter muscles, and the masseter inhibitory reflex (MIR) elicited by magnetic stimulation with single and double-shock techniques of healthy subjects and SB patients were compared.

2. Materials and methods

2.1. Subjects

Subjects consisted of twenty six patients suffering from SB (12 females and 14 males; mean age: 24.9 ± 4.0 years) and thirty healthy volunteers (18 females and 12 males; mean age: 24.1 ± 3.1 years). Written informed consent was obtained from each subject, which was approved by the Ethics Committee of Tongji University.

All SB subjects were first selected based on: (1) a history of tooth grinding occuring at least three nights per week in the last six months; (2) a report of jaw muscle fatigue or tenderness in the morning; (3) the presence of abnormal tooth wear; (4) masseter muscle hypertrophy [30]. In four cases, the above clinical signs were not clear, and then the polygraphic research criteria was used: more than four SB episodes per hour of sleep or more than 25 SB bursts per hour of sleep, and two or more episodes with grinding noise over the sleeping period [1]. None of Subjects showed presence of neurological diseases, history of epilepsy, history of smoking, presence of craniofacial pain or temporomandibular joint problem or headaches. No subject used drugs that could affect the neuromuscular excitability or drugs associated with bruxism during, or 48 h prior to the evaluation. Female subjects were not examined during the menstrual period [31].

2.2. Preparation

TMS-evoked MEPs and magnetic-evoked MIR were recorded from masseter muscles using surface electrodes. The reference electrode was placed 2 cm below the mandible angle and the active electrode was placed over the lower third of the muscle belly, with an interelectrode distance of approximately 4 cm [9]. The ground electrode was placed over the forehead. Skin impedance was lower than 15 kΩ. The signals were amplified and filtered (20–5000 Hz) using an amplifier (KeyPoint, Medtronic, Dantec).

Subjects were seated in the upright position on a comfortable chair. They activated their masseters by clenching in the intercuspal position at approximately 30% of maximal voluntary contraction for the MEPs studies and approximately 80% for the MIR studies, with the aid of visual feedback of the electromyographic (EMG) activity of the masster muscles on the computer screen.

2.3. TMS-evoked MEPs

TMS was performed by a MagPro X100 stimulator (Medtronic, Dantec) with a figure-of-eight coil (external diameter: 8 cm). The coil was placed over the face area of the motor cortex of the right hemisphere, at an angle of 120° relative to the parasagittal plane and the handle pointing forwards and laterally; the optimal spot for masseter activation was carefully searched into an area 4–10 cm lateral to the vertex and 0–4 cm frontal to the bi-auricular line; these area and coil orientation were previously found to be optimal for TMS to elicit masseter muscle the largest MEP with the lowest threshold [11]. In this position, the TMS activated the presumptive cortico-bulbar descending fibers that produced a long-latency and a low-amplitude response in the contralateral MEP(c-MEP); and also directly excited the ipsilateral trigeminal root, as evidenced by appearance of a short-latency response in the ipsilateral masseter EMG(r-MEP). The active motor threshold (AMT) was defined as the minimum stimulus intensity that induced a contralateral MEP greater than 0.1 mV in peak to peak amplitude for 5 out of 10 consecutive stimuli [29]. In each case, MEPs were assessed by using 130% of the AMT, the latency and amplitude over 3 trials for the c-MEP, the latency of the r-MEP, and the central conduction time (CCT) were also measured; the CCT was calculated by subtracting the latency of the r-MEP from that of the c-MEP.

2.4. MIR elicited by magnetic stimulation

MIR was elicited by magnetic stimulation with single and double-shock techniques [9]. The intensity of magnetic stimuli evoking the MIR were at 40–60% of maximum output of stimulator; this intensity elicited a complete MIR, which was composed of 2 separate silent periods (SPs), an early silent periods (SP1) and a late silent periods (SP2). With the coil placed at mental level in the midline position, the MIR was obtained by the single stimuli at a series of 6 trials.

MIR recordings also allow the excitability of a reflex circuit assessed by studying its recovery cycle. The recovery cycle was obtained by delivering double-shock technique at interstimulus interval (ISIs) of 100, 200, 300, 400, 500, and 600 ms; a series of 6 trials were repeated at each ISIs. Subjects were asked to relax the muscles (their teeth were not clenched) for 15 s between each magnetic stimuli and rest after each trial, to avoid fatigue.

Onset and end of SPs were taken at the intersection of the rectified and averaged signal and a line indicating 80% of the background EMG level [8], then the frequency of SPs and the latency and duration of the SPs under single stimuli, the recovery of the SP2 at each ISIs under paired stimuli were analyzed. The recovery of the SP2 was measured as the area of the response to the second stimuli (test) in percentage of the area of the response to the first stimuli (conditioning), and the area of the SPs was automatically computed with the software of KeyPoint.

2.5. Statistical analysis

The latency of the r-MEP, the duration of the SP1 of the MIR obtained from magnetic single stimuli between the two groups were compared by independent-samples *t* test; the latency of the c-MEP, the CCT between the two groups were compared by approximate *t* test; the amplitude of the c-MEP, the latency of the SP1, and the recovery of the SP2 for each ISI between the two groups were compared by the non-parametric Wilcoxon test; the frequency of absence of the SP2 between the two groups evoked by magnetic single stimuli was compared by chi-square test. The statistical significance threshold was set at the a-error level of 0.05 (two-tailed test). Statistical software SPSS17 was employed for statistical processing.

3. Results

3.1. TMS-evoked MEPs

Both the contralateral and ipsilateral masseter muscles evoked a MEP in all normal subjects and SB patients during bilateral biting. The results of the MEPs are shown in Table 1.There were no statistically significant differences between the patients and the