Genesis of sleep bruxism: Motor and autonomic-cardiac interactions

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1. Definition and recognition of sleep bruxism

Sleep bruxism (SB) is defined as a stereotyped movement disorder occurring during sleep and characterized by tooth grinding (TG) and/or clenching. SB should be distinguished from the daytime-awake bruxism that is mainly related to “stress/anxiety” reactivity and expressed as a jaw muscle clenching habit/tic. In the presence of medical disorders, medication or drug use, TG is described as secondary or iatrogenic. In normal subjects, SB-TG is considered to be.

Abstract
This is a short review paper presenting hypothesis to explain the mechanism that may be involved in the genesis of sleep bruxism (SB). In humans, SB is a repetitive sleep movement disorder mainly characterized by rhythmic masticatory muscle activity (RMMA) at a frequency of 1 Hz and by occasional tooth grinding. Until recently, the mechanism by which RMMA and SB episodes are triggered has been poorly understood. It is reported that during light sleep, most SB episodes are observed in relation to brief cardiac and brain reactivations (3–15 s) termed “micro-arousals”. We showed that RMMA are secondary to a sequence of events in relation to sleep micro-arousals: the heart (increase in autonomic sympathetic activity) and brain are activated in the minutes and seconds, respectively, before the onset of activity in suprahyoid muscles and finally by RMMA in jaw closing masseter or temporalis muscles. In non-human primate study, we have shown that the excitability of cortico-bulbar pathways is depressed during sleep; no rhythmic jaw movements (RJM) are observed following intracortical microstimulation (ICMS) of cortical masticatory area (CMA) during sleep compared to the quiet awake state.

The above results suggest that the onset of RMMA and SB episodes during sleep are under the influences of brief and transient activity of the brainstem arousal—reticular ascending system contributing to the increase of activity in autonomic-cardiac and motor modulatory networks.
primary and is reported by 8% of the adult population. Its prevalence decreases with age from 14% in childhood to 3% in the elderly; no gender difference is observed.

The consequences of SB may be tooth destruction, temporomandibular joint and muscle pain or jaw lock, temporal headaches and cheek-biting (worse if xerostomia). The odds ratio (OR) of reporting temporomandibular disorders or chronic myofascial pain of masticatory muscles, when clenching and/or grinding are concomitant, have been estimated at between 4.2 and 8.4. Up to 65% of SB patients of all ages report headaches.

The sleep laboratory diagnosis of SB requires that it be associated with TG noise. The noise made by the TG can greatly disturb the sleep of bedroom partners. The following major risk factors have been shown to exacerbate SB-TG: (1) smoking, caffeine and heavy alcohol drinking; (2) type A personality—anxiety; (3) sleep disorders such as snoring (OR: 1.4), sleep apnea (OR: 1.8) or periodic limb movements (concomitant in 10%).

Clinically, SB is diagnosed following report by the sleep partner or parent of recent/frequent TG (this is the most reliable criterion), the presence of tooth wear or jaw muscle hypertrophy, and awareness of jaw clenching while awake.

The sleep laboratory diagnosis of SB requires that it be distinguished from other oromandibular activity during sleep (e.g., oromandibular myoclonus, swallowing, coughing, grunting, tooth tapping, vocalization, etc.) that may represent up to 30% of all oral activities during sleep. Episodes of jaw muscle contractions are scored as moderate to severe SB if more than four EMG events per hour of sleep are noted. The final diagnosis is made if at least two SB episodes per night are associated with TG noise.

2. Hypotheses on genesis of SB

Various hypotheses have been proposed to explain SB: changes in dental occlusion but no strong evidence-based data support this hypothesis; stress and anxiety has also been suggested; involvement of the dopaminergic system that remains to be confirmed since in most randomised experimental trials with dopaminergic medications (e.g., l-dopa, bromocriptine), the onset of SB episodes is only marginally reduced. During wakefulness, dopamine has a role in the execution of movement and in maintaining vigilance; during sleep the dopaminergic system is probably minimally active at the exception of brief period of arousal related movements such as periodic limb movements.

2.1. Descending influences from cortex during sleep

Another hypothesis on genesis of RMMA during sleep is that, conversely to waking state, cortico-bulbar influences are not dominant during sleep. The top-down circuits seem to be partially de-activated during sleep to preserve the so-called sleep continuity. The following evidences support this hypothesis in the physiopathology of SB: (1) a specific increase in the cortical activity, over the motor cortex, precedes most limb movements. This brief and large deflection of brain wave activity is termed pre-motor potential. We have found that RMMA are not preceded by such pre-motor cortical potential. This suggests that SB is not generated by a clear pattern of cortical activation. (2) We also found, during sleep of SB patients, that SB episodes are not associated with the so-called cortical K complexes that are electroencephalographic (EEG) marker of sudden changes in brain endogenous activity or secondary to sounds influences. (3) In order to better understand the “basis” of the genesis of RJM, we directly tested if cortico-bulbar pathways remain active during sleep of primates (macaca fascicularis). We observed that the intracortical microstimulation (ICMS) threshold did not evoke RMMA (or a rhythmic jaw movements = RJM) responses from the cortical masticatory area (CMA) during light non-REM sleep (stages 1 and 2 in humans) in comparison to the quiet awake state. However, as soon as the animal wakes up, there is a rapid return of RJM at ICMS threshold levels comparable to pre-sleep. These preliminary data suggest that the geneses of RJM or RMMA during sleep are probably not directly under the influence of the cortical network as seen during wake state.

2.2. Sleep micro- arousal: brain and autonomic reactivation toward arousal

It is possible that the sudden onset of RMMA during sleep is occurring in brief time windows at which the brain is switching from sleep to an aroused state. These periods are termed micro-arousal which is defined as 3–15 s abrupt shifts in EEG activity accompanied by a rise in heart rate and muscle tone. Micro-arousals (MA) tends to recur 8–15 times per hour of sleep in young healthy subjects.

To better understand the role of MA in sleep it is important to revise how sleep in initiated and maintained. To initiate non-REM (rapid eye movement) sleep, a massive inhibition of GABA on brain arousal ascending system is needed to reverse the influences of arousal related orexin/hypocretin, from the hypothalamus, and on acetylcholine, noradrenaline, histamine and serotonin brain networks. Moreover, from the onset of sleep, a reduction in muscle tone to a clear hypotonia or a near limb paralysis in the REM sleep stages is observed. It is further suggested that the reduction of muscle tone during REM sleep is under the influences of noradrenergic neurons of the pedunculopontine tegmentum (PPT) neurons and of GABA and glycine inhibition on both brainstem and spinal cord motor-neurons.

SB tends to occur in relation to recurrent MA within the so-called cyclic alternating pattern or CAP. As described above MA are characterized by a repetitive rise in heart and brain activity within sleep and are thought to reflect a natural process that acts as a sensor for maintaining body homeostasis and as a protective sentinel during sleep. We explored the role of MA, as a physiological state that may increase the probability of initiating an episode of SB, with the use of a sensory vibrator during sleep. Experimentally induced RMMA related MA were followed by TG in over 70% of trials in SB patients only and not in control subjects. Our laboratory has further demonstrated that the onset of SB is related to a sequence of physiological activations in relation to the MA (see Fig. 1):

(1) A rise in sympathetic cardiac activity around 4 min before RMMA.
Then, our data suggest that the episodic and transient reactivation of arousal ascending system may be associated to the sudden apparition of RMMA in relation to “permissive” excitation or disinhibition (inhibition of inhibition) on some motor trigeminal neuron or interneuron of the central pattern generator (CPG) network responsible of RMMA. The rhythm of jaw movements generated durinb bruxism is at 1 Hz frequency. Also, in contrast to mastication, sleep-related RMMA are characterized by co-contraction of opening and closing jaw muscles and these purposeless movements rarely last more than 8 s.\textsuperscript{20,46}

### 3. Conclusion

The above results suggest that the onset of RMMA and SB episodes during sleep are under the influence of the brainstem arousal—reticular ascending system contributing to the increase of activity in motor and autonomic-cardiac neuronal networks.

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### References

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