Research Paper

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The effect of propranolol on sleep bruxism: hypothetical considerations based on a case study

T.T. Sjöholm[1] DDS PhD, I. Lehtinen[2] MD PhD and S.J. Piha[3] MD PhD

[1]Institute of Dentistry, [2]Department of Physiology and [3]Department of Clinical Physiology, University of Turku, Finland

Correspondence and reprint requests: T.T. Sjöholm DDS, Institute of Dentistry, University of Turku, 20520 Turku, Finland
Tel: (+358) 21 63381. Fax: (+358) 21 6338356

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The purpose of this study was to investigate the effect of the β-adrenergic antagonist, propranolol, on the nocturnal masseter muscle activity of a heavy sleep bruxist. Three all-night polysomnographic registrations were performed with bilateral masseter muscle EMG recordings. The first night study served as the baseline night, the second night registration was performed after total sleep deprivation and the third night registration was made with propranolol. Sleep deprivation decreased the masseter contraction (MC) index by 61% and propranolol by 72% when compared to the level of the baseline night. This preliminary observation is in line with our hypothesis suggesting a link between autonomic regulation of circulation and rhythmic activation of masticatory muscles, especially when associated with body movements during sleep.

Keywords: sleep bruxism; masseter contraction (MC) episode; rhythmic jaw movements (RJM); MC-index; propranolol

Introduction

The rhythmic pattern of masseter electromyograph (EMG) activity during sleep has been emphasized as being characteristic of sleep bruxism.[1] The strongest rhythmic masseter contraction (MC) episodes occur in association with large body movements during transition from delta sleep to a lighter sleep stage.[2,3] During these episodes heavy bruxists show forces which are close to their maximal clenching.[4,3] Such parasomnias as enuresis, somnambulism, and sleep terrors also occur during transitions from delta sleep to lighter sleep stages. The hypothesis of the presence of an 'arousal disorder' has been suggested as a cause of these parasomnias as well as of sleep bruxism.[5,2] However, the rhythmicity and high forces typical of bruxism cannot be explained solely on the basis of this hypothesis. Furthermore, rhythmic jaw movements have also been observed in asymptomatic controls.[3]

We hypothesized that rhythmic masticatory muscle activity is an autonomic reflex, that works as a kind of auxiliary pump in helping to maintain the homeostasis of brain circulation. To study our hypothesis, sleep deprivation (the patient stayed awake one night and one day before the study night) was selected as a way to decrease nocturnal motor activity.[6] Propranolol, the β-adrenergic receptor blockade, was used to reduce sympathetic activity.[7]

Subject and methods

Clinical history

A 25-year-old healthy female bruxist (height 158.5 cm and weight 50 kg) who fulfilled the criteria for a chronic bruxist was selected for our study.[8] The subject had no other regular medications, except a contraceptive pill (Minucet). The state-trait anxiety inventory[9] did not show any impairment of psychosocial functioning (the test value was 38). Informed consent was obtained. The study was approved by the Ethical Committee of the Medical Faculty of the University of Turku.

Set-up for sleep recordings

Polygraphic recordings were analysed by an experienced neurophysiologist (as a blind observer) using the method by Rechtschaffen and Kales (1968)[10] with right and left masseter EMG recordings. The following variables for masseter muscle activity were used:[11,3]

1. the frequency of MC episodes;
2. the mean relative amplitude of MC episodes;
3. bursts (single contractions) per hour;
4. MC index (burst × mean relative amplitude);
5. the frequency of rhythmic jaw movement (RJM) episodes;
6. the distribution of body movement activity detected by sensitive movement sensor (SCSB-mattress); different categories were used for single body movements and for those occurring in series of movements (with less than 60 s, but more than 5 s between movements); body movement occurred either alone or simultaneously with MC activity;
7. the distribution of single MC episodes and those occurring in series of episodes (with less than 60 s, but more than 5 s low tonic activity between MC episodes).

The categories were:
a) single (isolated) event
b) series of two events
c) series of three events
d) series of four events.

Study design
The first study night served as the baseline without any treatment. The second recording was made two months later, after one night (total) sleep deprivation. The sleep deprivation was carried out using a supervisor who ensured that the subject stayed awake through the whole night and day previous to the recording night (the recovery night). The third recording night was 12 months after the second study night when propranolol hydrochloride (Inderal) was given at a dose of 2.8 mg/kg. Some of the propranolol (40 mg p.o.) was given the night before the third study night. More propranolol (60 mg) was given p.o. before the beginning of sleep at 22.48 h and a 40 mg p.o. booster dose four hours later at 03.00 h.

Results

Table 1 shows the sleep data of the study nights. The percentage distribution of sleep stages was within the normal range.[12] The subject reported no side effects with propranolol.

The results of treatment effects on masseter muscle activity are shown in the upper panel of Table 2. Although the frequency of MC episodes decreased both after sleep deprivation and with propranolol, the total number of MC episodes indicated that there was a marked change only with propranolol. The percentage decrease in the MC index when compared with first night values was 61% after sleep deprivation, and 72% with propranolol. The percentage decrease in MC episodes with RJMs was 50% and 67%, respectively.

The middle panel of Table 2 demonstrates the distribution of body movements and the lower panel shows the distribution of MC episodes. The longer the series of events was, the less activity was observed with propranolol (see Figure 1). In conclusion, with propranolol, the content of MC activity clearly decreased and the number of series of MC episodes was smaller.

The mean duration of intervals between the MC episodes that occurred in pattern form was 21.4 s (SEM 3.8) during the baseline night, 16.6 s (SEM 2.8) after sleep deprivation and 27.3 s (SEM 3.5) with propranolol.

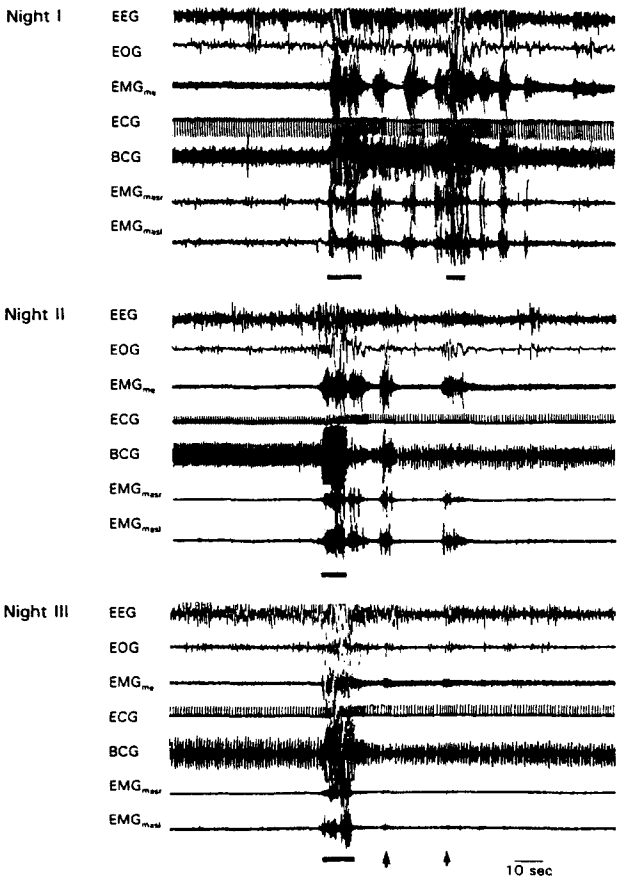

Figure 1. Three examples of the transition stage of sleep from different nights showing the effect of postural change on masseter muscle activity. All the examples are taken from the end of the first third of the sleep. Night I = baseline night, night II = after one night sleep deprivation and night III = with propranolol. EEG = electroencephalogram (C3-A2), EOG = electro-oculogram, EMGme = electromyogram of mental muscle, ECG = electrocardiogram, BCG = ballistocardiogram from static charge-sensitive bed, EMGmasr = right masseter muscle EMG, EMGmasl = left masseter muscle EMG. Horizontal black bars indicate the site of body movements. Black arrows demonstrate the site of possible treatment effect. Paper speed is 1.5 mm/s. Note that the effect of postural change causes similar rhythmic changes in masseter activity, except with propranolol when the latter components after sleep movement remain small

Table 1. Sleep data

	Night I (baseline)	Night II (deprivation)	Night III (propranolol)
Time in bed (TIB)	437 min	608 min	490 min
Total sleep time (TST)	436.2 min	605.2 min	474.7 min
Number of awakenings (AW)	–	–	2
AW	–	–	15.3 min
Sleep latency (SL)	0.8 min	2.8 min	0.2 min
REM latency (RLA)	150 min	163 min	158 min
Number of REM periods	4	5	3
Stages 1 and 2 (% of TST)	71.5%	57.8%	66.3%
Stages 3 and 4 (% of TST)	12.8%	21.0%	18.6%
REM (% of TST)	15.5%	21.2%	15.1%

Table 2. Variables of masseter muscle activity per TST and the distribution of single events and events that occur in pattern form

	Night I (baseline)	Night II (deprivation)	Night III (propranolol)
Frequency of MC episodes/h	9.1	6.3	4.7
Relative amplitude of MC episode	0.9	0.8	1.0
Bursts hour	79.2	35.1	20.1
MC index	71.3	28.1	20.1
Frequency of RJM episodes/h	4.5	2.3	1.5
Body movement activity			
Single body movement	54	56	49
Series of two body movements	20	15	10
Series of three body movements	8	6	2
Series of four body movements	6	1	1
Total	88	78	62
MC episodes			
Single MC episodes	37	42	27
Series of two MC episodes	17	14	9
Series of three MC episodes	7	6	1
Series of four MC episodes	5	1	1
Total	66	63	38

Discussion

Propranolol reduced the MC index and the frequency of RJM episodes close to the level reported in asymptomatic controls.[3] This can be partly explained by the inhibition of the increased sympathetic activity or 'fight or flight' response especially associated with large body movements.[7] Sleep deprivation reduced nocturnal motor activity, probably showing the lowest range that can be achieved without medication. The effect was even stronger with propranolol when compared with the sleep deprivation (see Figure 1). The first night showed the basic frequencies of masseter activity, but the result may represent a higher activity level typical after a stress situation (the first night effect). Gross body movements occur quite consistently throughout the night with characteristic patterns in the same sleeper from night to night.[13] Even when exposed to environmental noises during sleep, the rate of body movement remains generally unchanged. Movements are temporally redistributed to the proximity of the noise, without any increase in their total number.[14] Only sleep deprivation appears to reduce the rate of nocturnal movements during recovery nights.[6]

Hypothetical considerations

The autonomic nervous system maintains homeostasis and co-ordinates responses to external stimuli.

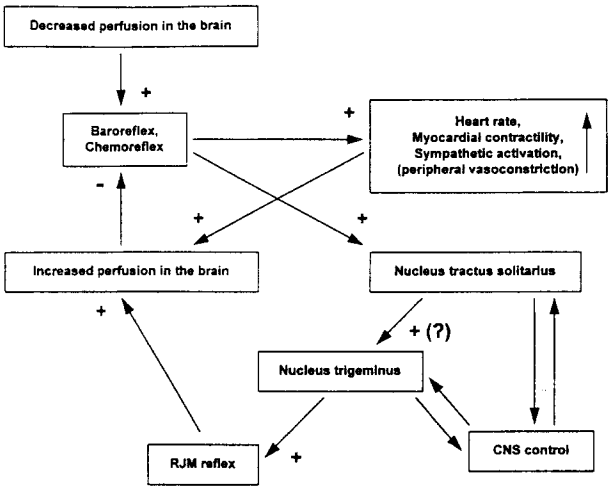

Figure 2. Hypothetical regulation of masticatory muscle rhythmic activity (RJMs) during sleep

Hence, we speculate that masticatory muscles may have a physiological function that is controlled by the autonomic nervous system. Bruxists have increased sympathetic activity prior to sleep bruxism.[1,2] Our resent observations with the cardiovascular reflex test (Valsalva manoeuvre, deep breathing and standing up) showed that conscious bruxists have disturbed autonomic control of peripheral blood circulation and this probably predispose bruxists more frequently to stronger MC episodes during sleep.[15] Muscle nerve sympathetic activity (MSA) occurs pulse-synchronously, consisting of intermittent bursts of vasoconstrictor impulses which are time-locked to the cardiac rhythm and occur predominantly during reductions in blood pressure.[16] Rhythmic jaw movements occur at 1 Hz frequency.[1] According to our hypothesis, RJMs could function as an 'auxiliary pump' aiming for short-term regulation of cerebral blood flow (see Figure 2). This is especially evident when there is an abrupt need to increase cerebral circulation. For instance, during delta sleep, the brain's metabolic rate of O_2 is reduced by 20% when compared to other sleep stages or wakefulness.[17] Furthermore, the estimated metabolic rates of glucose and blood flow are reduced in non-REM sleep compared with wakefulness.[18] This could explain why the strongest 'pumping' activity occurred in transition from delta sleep to a lighter sleep stage in association with body movements.[3]

Although we did not use any direct measurement of autonomic activity during sleep, the positive effects of propranolol and sleep deprivation are in agreement with our hypothesis of autonomic reflex. The hypothesis provides an explanation for the rhythmicity and the high relative amplitudes of masseter EMG during sleep. Further studies, however, are needed firstly to confirm this preliminary result and secondly, to establish the importance of medication as possible alternative treatment for sleep bruxism.

Acknowledgements

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