Association Between Nocturnal Bruxism and Gastroesophageal Reflux

Shouichi Miyawaki, DDS, PhD; Yuko Tanimoto, DDS; Yoshiko Araki, DDS; Akira Katayama, DDS; Akihito Fujii, DDS; Teruko Takano-Yamamoto, DDS, PhD

Department of Orthodontics and Dentofacial Orthopedics, Okayama University Graduate School of Medicine and Dentistry, Okayama, Japan

Study Objective: To examine the relationship between nocturnal bruxism and gastroesophageal reflux.

Design: Controlled descriptive study and double-blind, placebo-controlled, clinical study.

Setting: Portable pH monitoring, electromyography, and audio-video recordings were conducted during the night in the subjects' home.

Participants: Ten patients with bruxism and 10 normal subjects were matched for height, weight, age, and sex. They did not have symptoms of gastroesophageal reflux disease.

Intervention: Medication with a proton pump inhibitor (ie, a gastric–acid-inhibiting drug).

Measurements and Results: The bruxism group showed a significantly higher frequency of nocturnal rhythmic masticatory muscle activity (RMMA) episodes (mean ± SD: 6.7 ± 2.2 times per hour) and a higher frequency and percentage of time of gastroesophageal reflux episodes with a pH less than 4.0 and 5.0 (0.5 ± 0.9 and 3.6 ± 1.6 times per hour and 1.3% ± 2.5% and 7.4% ± 12.6%, respectively) than the control group (RMMA episodes: 2.4 ± 0.9 times per hour; gastroesophageal reflux episodes: 0.0 ± 0.0 and 0.1 ± 0.3 times per hour and 0.0% ± 0.0% and 0.0% ± 0.0%, respectively). In the bruxism group, 100% of the gastroesophageal reflux episodes with a pH less than 3.0 and 4.0 included both an RMMA episode and an electromyographic burst, the duration of which was approximately 0.5 to 1.0 seconds, probably representing swallowing of saliva. The majority of gastroesophageal reflux episodes with a pH of 4.0 to 5.0 also included both an RMMA episode and an electromyographic burst in the control and bruxism groups (100% ± 0.0% vs 70.7% ± 16.5%), again probably due to swallowing of saliva. The remaining minority of gastroesophageal reflux episodes with a pH of 4.0 to 5.0 contained only an electromyographic burst (swallowing of saliva). The frequency of RMMA episodes after the release of the medication from the proton pump inhibitor, which increased the gastric and esophageal pH, was significantly lower than that after administration of the placebo in the control and bruxism groups (1.0 ± 0.6 vs 1.9 ± 3.2 times per hour, and 3.7 ± 1.9 vs. 6.0 ± 2.2 times per hour, respectively).

Conclusions: Nocturnal bruxism may be secondary to nocturnal gastroesophageal reflux, occurring via sleep arousal and often together with swallowing. The physiologic link between bruxism and the increase in salivation needs to be investigated.

Key Words: Acid, bruxism, electromyogram, gastroesophageal reflux (GER), parafunction, proton pump inhibitor (PPI), rhythmic masticatory muscle activity (RMMA), sleep

Citation: Miyawaki S; Tanimoto Y; Araki Y et al. Association between nocturnal bruxism and gastroesophageal reflux. *SLEEP* 2003;26(7):888-92.

INTRODUCTION

SLEEP BRUXISM IS DEFINED AS A STEREOTYPED MOVEMENT DISORDER CHARACTERIZED BY TOOTH GRINDING DURING SLEEP AND HAS BEEN PLACED IN THE PARASOMNIA SECTION ACCORDING TO THE *INTERNATIONAL CLASSIFICATION OF SLEEP DISORDERS*.[1] Bruxism has also been considered to be a parafunction[2] because it afflicts approximately 10% of the general population, causing abnormal tooth wear, fracture, and hypersensitivity; masticatory muscle discomfort; and pain or temporomandibular disorders.[3,4] However, the mechanism that generates bruxism remains unknown.

In order to study sleep bruxism, Lavigne et al[5-7] focused their attention on rhythmic masticatory muscle activity (RMMA) during sleep. In patients with bruxism, approximately 90% or more of sleep bruxism episodes are RMMA episodes.[5-7] Rhythmic masticatory muscle activity is also commonly observed in normal healthy subjects, but its frequency in normal subjects is significantly lower than in patients with bruxism.[5-7] In addition, it has been recently reported that RMMA is secondary to micro-arousal[6,8,9]; RMMA often occurs in the supine position and is often associated with swallowing during sleep.[7]

Interestingly, among various phenomena occurring during sleep in humans, nocturnal gastroesophageal reflux (GER) has been found to be closely associated with sleep arousal such as micro-arousal,[10,11] supine position,[12] and swallowing for esophageal lubrication (ie, acid clearance).[11,13-15] Furthermore, sleep bruxism and GER are known to have several common features. For example, sleep bruxism and GER have been separately reported to be more prevalent in children than in adults[3,16]; bruxism and GER disease (GERD) also have separately been reported to be associated with obstructive sleep apnea,[17,18] smoking,[18,19] drinking of coffee[18,20] and alcohol,[18,21] and psychologic stress.[18, 22] In addition, the concordance rate for sleep bruxism in monozygotic twins has been reported to be higher than in dizygotic ones,[23] and a similar finding has also been reported for GERD. [24]

The purpose of this study was to test the hypothesis that nocturnal bruxism is associated with nocturnal GER by examining the relationship between RMMA and GER episodes and by performing a double-blind, placebo-controlled, clinical trial with a gastric–acid-inhibiting drug (ie, a drug commonly used for the treatment of GERD).

METHODS

Subjects

Ten patients with bruxism (Bruxism group: mean age, 27.0 years ± 7.0 years; 5 men and 5 women; mean height, 168.0 cm; mean weight, 59.1 kg) were recruited based on the following conditions: a history of tooth-grinding occurring more than 3 times a week for at least 6 months, jaw muscle fatigue or discomfort in the morning, and the presence of tooth wear.[1] They were then examined by use of an electromyogram (EMG) recording system along with a video recorder in their home. They were diagnosed as having bruxism according to 3 criteria: more than 4 bruxism episodes per hour, more than 25 EMG bursts per hour, and more than 2 tooth-grinding sounds per night.[25] The diagnosis remained con-

Disclosure Statement
No significant financial interest/other relationship to disclose.

Submitted for publication February 2003
Accepted for publication June 2003
Address correspondence to: Teruko Takano-Yamamoto, Department of Orthodontics and Dentofacial Orthopedics, Okayama University Graduate School of Medicine and Dentistry, 2-5-1 Shikata-Cho, Okayama 700-8525, JAPAN; Tel: +81(0) 86-235-6692; Fax: +81 (0) 86-235-6694; E-mail: t_yamamo@md.okayama-u.ac.jp

stant over time for all subjects.[26]

Ten healthy volunteers (Control group: mean age, 26.4 years ± 4.7 years; 6 men and 4 women; mean height, 165.0 cm; mean weight, 60.4 kg), without any of the aforementioned clinical findings, were also recruited as controls to be matched with the bruxism patients. They were also examined and found not to have bruxism, according to the same criteria.[25]

None of the above subjects had any history or signs of sleep or medical disorders. None were taking any medication influencing sleep, motor activity, gastric acid secretion, or salivary secretion nor did any have GERD symptoms such as heartburn. All subjects participated after providing informed written consent according to a protocol that had been reviewed and approved by the Institutional Review Board.

Equipment

For assessment of the nocturnal RMMA episodes, a portable telemeter EMG recording system (PC Card Recorder DR-C2, TEAC Instruments Corporation, Kawasaki, Japan) and disposable bipolar surface electrodes (12-mm diameter, blue sensor N-00-S, GE Market, Tokyo, Japan) were used. It is already known that both anterior temporal and masseter muscles show higher EMG bursts on each side during bruxism or RMMA episodes and that many electrodes and cables may restrict the sleeping position,[27] which is closely associated with the frequency of RMMA episodes.[7] In addition, it has been reported that a 1-channel portable EMG recorder allows full detection of RMMA episodes if the sampling frequency is equal to or more than 128 Hz or if the threshold is more than 10%.[28] Therefore, the EMG from the unilateral anterior temporal muscle, which is mostly responsible for the movement of the mandible,[29] was recorded at a sampling frequency of 1 kHz. The recording of the EMG was conducted on the side opposite to that on which facial contact with the pillow was more frequent in each subject. Audio-video recording of jaw movement was made for scoring RMMA episodes.[7,25]

To assess the nocturnal GER episodes, we adopted a conventional reliable method.[30,31] In terms of equipment, we used a standard 24-hour ambulatory portable esophageal pH-metric and manometric system, equipped with a disposable pH probe with a 2-mm external diameter and a manometric probe with a 4-mm external diameter (Micro Digitrapper 4Mb Flisuh Series, Zinetics 24 pH Catheter, and Konigsberg Solid State Catheter, respectively, Medtronic Functional Diagnostic A/S, Skovlunde, Denmark). Firstly, the position of the upper limit of the lower esophageal sphincter was manometrically defined. The position was then pH-metrically confirmed.[30] Finally, the pH electrode was passed through a nostril and placed 5 cm above the upper limit of the lower esophageal sphincter.[31] The sampling frequency was 4 Hz.

Data Recording

Each subject ate the same designated food and drank a 500-ml bottle of water 4 hours before going to bed. Smoking and drinking of alcohol and coffee were prohibited during the day of the experiment. The temporal muscle activity, pH in the lower esophagus, and audio-video data were recorded simultaneously in the home of each subject from approximately 11:00 PM until 7:00 AM or until the time when the subject awoke, on 2 successive nights. The first night was used for adaptation to the aforementioned equipment. Data from the second night were used for the diagnosis of bruxism and for study analysis. In order to discriminate RMMA episodes from other orofacial activities, we recorded baseline values prior to sleeping in the supine position for the following measurements: maximum voluntary clenching of the teeth for 1 second, tooth tapping, coughing, lateral and vertical head movements, and saliva swallowing.[7,25] All of these activities were repeated 3 times.

Next, these subjects participated in a double-blind, placebo-controlled, clinical trial to examine the effect of 1 tablet (10 mg) of rabeprazole (sodium rabeprazole, Esai Co., Ltd, Tokyo, Japan) on nocturnal RMMA. This gastric–acid-inhibiting drug is a relatively new proton pump inhibitor (PPI). Simultaneous recording of temporal muscle activity and audio-video data was performed in the home of each subject on 2 separate nights from 11:00 PM until 7:00 AM, or until the time when the subject awoke. Smoking and drinking of alcohol and coffee were prohibited during the day of the experiment. Each subject ate the same designated food and drank a 500-ml bottle of water 4 hours before going to bed. On 1 of the nights, a PPI tablet was given to each subject 2 hours before sleep. On the other night, a placebo tablet was given to each subject. The order of administering the PPI and placebo was randomly determined. The washout period was 1 week. Both subjects and experimenters were blind to the contents of the tablets of inactive and active medication, which were identical in appearance. Prior to sleeping, baseline values were also recorded. In this study, a single dose of medication at night was adopted because all subjects were healthy adults without GERD symptoms and because the administration of a single dose of rabeprazole before sleep is known to significantly increase the intragastric pH.[32] The time medication schedule was determined based upon the pharmacokinetics of the drug (ie, the concentration of this drug in the blood reaches its highest value approximately 2 hours after the medication is ingested).[33] In fact, according to a preliminary study, the frequency and percentage of time of GER episodes decreases after the administration of such a single dose.

Data Analysis

The first and last hour of data were excluded from the analysis because, according to audio-video data and interviewing of the subjects, there was a possibility that subjects were awake during this time. The EMG data were full-wave rectified and averaged with a moving interval of 1 millisecond and a window time of 19 milliseconds.[34] The muscle activity during sleep was normalized by the amplitude during the maximum voluntary clenching of teeth in each subject. The RMMA episodes were scored based on research criteria that have been previously reported.[25] The research criteria were as follows: EMG bursts with amplitudes that were more than 10% of the amplitude during maximum voluntary clenching of the teeth were selected.[5] The phasic type of bruxism episode corresponded to at least 3 bursts of 0.25- to 2.0-seconds duration with less than 3.0-seconds separation, and the tonic type of bruxism episode corresponded to a single burst lasting more than 2.0 seconds. The mixed type of bruxism episode consisted of both phasic and tonic types. Phasic and mixed types of bruxism episodes were selected as RMMA episodes.[5-7] We used the frequency and duration per episode of RMMA episodes as parameters because it was reported that the frequency remained constant in the range of coefficient of variation of 25% over time for all subjects[26] and that the duration per episode also remained constant.[5-7]

Episodes of GER were scored by using a conventional method.[30,31] In this study, a pH of 5.0 was also adopted as a cut-off value for this episode in addition to a pH of 3.0 and 4.0 because all participants in this study were healthy adults without GERD symptoms.[35,36] The minimum duration for GER episodes was defined as 30 seconds.[37] We used the number of GER episodes per hour (ie, frequency) and the percentage of time of GER episodes because these parameters have been reported to be reproducible and reliable.[30,31] The time relationship between RMMA and GER episodes was analyzed. Both nocturnal RMMA and GER episodes were automatically detected by means of a bruxism and gastroesophageal reflux analyzing software (MTS50011, Medical Try System, Tokyo, Japan). The RMMA episodes were confirmed from audio-video data.[7,25]

Statistical Analyses

For all data, normality testing was performed using the χ^2 test. If a hypothesis that the distribution of data showed a normal one was rejected ($P<0.05$), we used a nonparametric test; otherwise, a parametric test was used. For comparisons between groups, the unpaired *t*-test or Mann-Whitney U test was used according to the data distribution. For compar-

isons within groups, the paired *t*-test or Wilcoxon rank-sum test was also used. Probability levels of $P < 0.05$ were considered statistically significant. Tests of significance were calculated using statistical analysis software (StatView, SPSS, Chicago, IL, USA).

RESULTS

The bruxism group showed a significantly higher frequency of nocturnal RMMA episodes and a higher frequency and percentage of time of GER episodes with a pH less than 4.0 and 5.0 than did the control group. However, there was no significant difference in the GER episodes with a pH less than 3.0 between the groups because subjects in both groups showed a low frequency of GER episodes with a pH less than 3.0. There were no significant differences in the recording time and duration per episode of nocturnal RMMA episodes between the 2 groups (Table 1).

In 5 bruxism patients having GER episodes with a pH less than 3.0 or 4.0, 100% of the GER episodes with a pH less than 3.0 and 4.0 included both an RMMA episode and an EMG burst with a duration of approximately 0.5 to 1.0 seconds, as shown in Figure 1. All control subjects and 5 patients with bruxism had no GER episodes with a pH less than 3.0 and 4.0. The majority of GER episodes in the control and bruxism groups with a pH of 4.0 to 5.0 also included both an RMMA episode and an EMG burst with a duration of approximately 0.5 to 1.0 second (100 ± 0.0 % vs 70.7 ± 16.5 %). The remaining minority of GER episodes with a pH of 4.0 to 5.0 contained only an EMG burst with a duration of about 0.5 to 1.0 seconds.

Approximately 60% of the RMMA episodes occurred during GER episodes with a pH less than 5.0 in 2 control subjects and 9 patients with bruxism (57.2 ± 40.4 % vs 65.1 ± 17.1 %), although the GER episodes with a pH less than 5.0 occupied up to only 7% of the sleeping time. Almost all of the remaining RMMA episodes in both groups occurred when the esophageal pH decreased rapidly, as shown in Figure 2.

The frequency of RMMA episodes after administration of the PPI medication, which increased the gastric and esophageal pH, was significantly lower than the frequency after the administration of the placebo medication in the control and bruxism groups. Figure 2 shows an example in which RMMA episodes were less frequently observed, along with a lower frequency of GER episodes after the PPI medication than after the placebo medication. There were no significant differences in the recording time and duration per episode of RMMA between placebo and PPI medication in either group (Table 2).

DISCUSSION

In this study, we deleted the first and last hour of the data because we did not examine the sleep stage[38] and because the subjects might have been awake just after going to bed and just before awakening. In fact, it seemed that all the subjects showed stable sleep in their homes, with eyes closed during the analysis time period, irrespective of the pH probe through their nostril and administration of the PPI medication. Therefore, we consider that we were able to collect reliable data during sleep.

The bruxism patients with a higher frequency of RMMA episodes, compared with the control subjects, showed a significantly higher percentage of time of nocturnal GER episodes with a pH less than 4.0, but the mean value (1.3% ± 2.5%) was within the normal range.[30] Furthermore, control subjects had no GER episodes with a pH less than 4.0. It is known that patients with GERD (eg, esophagitis) show a high percentage of time of GER episodes with a pH less than 4.0.[30,31,39] It has been reported that the cut-off value for the diagnosis of GERD is 8.8 %.[30] Therefore, neither bruxism patients nor control subjects were diagnosed as having GERD in the present study, and none showed symptoms of GERD.

Many previous epidemiologic studies have demonstrated that patients with GERD often show excessive tooth wear,[40] which is also a characteristic of sleep bruxism.[41] According to a recent case report, a young adult who complained of extreme tooth hypersensitivity, tooth wear, and bruxism was diagnosed as having GER by a gastroenterologist.[42]

In this study, we have revealed for the first time the causal relationship between bruxism (RMMA) and GER. The main results include the following. Most GER episodes, particularly episodes with a lower pH, contained both an RMMA episode and an EMG burst with a duration of approximately 0.5 to 1.0 seconds, probably representing saliva swallowing.[7,43] Also, RMMA episodes more frequently occurred during GER episodes than during other sleeping time. The frequency of RMMA episodes decreased significantly after the administration of the PPI medication, which increased the gastric and esophageal pH. Furthermore, it is known that nocturnal GER, in which chemoreceptors in the esophagus are stimulated by acid or hydrogen ions,[44] often causes sleep arousal such as micro-arousal (ie, a rapid short shift in electroencephalogram frequency characterized by theta, alpha, or fast-frequency waves during sleep).[10-12,45,46] It is also known that sleep arousal often causes

Table 1—Nocturnal rhythmic masticatory muscle activity and gastroesophageal reflux episodes*

Variables	Control group (n=10)	Bruxism group (n=10)
Recording time (h)	6.1 ± 0.9	6.0 ± 1.6
RMMA episode		
Frequency (times/h)	2.4 ± 0.9	6.7 ± 2.2‡
Duration per episode (s)	10.4 ± 2.2	11.7 ± 3.8
GER episode		
pH < 3.0		
Frequency of episodes (times/h)	0.0 ± 0.0	0.1 ± 0.3
Percentage of time (%)	0.0 ± 0.0	0.7 ± 1.6
pH < 4.0		
Frequency of episodes (times/h)	0.0 ± 0.0	0.5 ± 0.9†
Percentage of time (%)	0.0 ± 0.0	1.3 ± 2.5†
pH < 5.0		
Frequency of episodes (times/h)	0.1 ± 0.3	3.6 ± 1.6‡
Percentage of time (%)	0.0 ± 0.0	7.4 ± 12.6‡

*Data are given as mean ± SD; RMMA indicates rhythmic masticatory muscle activity; GER, gastroesophageal reflux.
† $P<0.05$; ‡ $P<0.01$ (Unpaired *t*-test or Mann-Whitney U test)

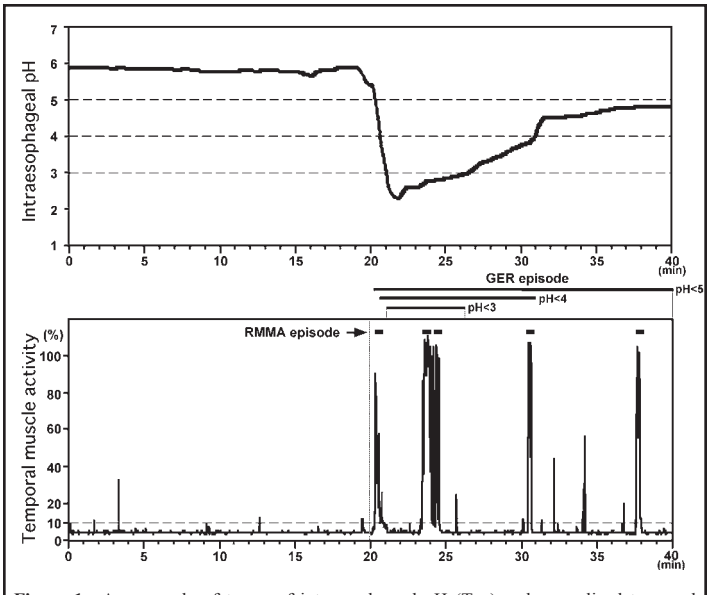

Figure 1—An example of traces of intraesophageal pH (Top) and normalized temporal muscle activity (Bottom) measured in a bruxism patient for 40 minutes, starting approximately 3 hours after the subject went to bed. The cut-off values to determine the gastroesophageal reflux (GER) episode were a pH of 3.0, 4.0, and 5.0 (upper dashed lines), and the cut-off value to determine the rhythmic masticatory muscle activity (RMMA) episode was 10% (lower dashed line). During the first 20 minutes with a stable high pH in the esophagus, no RMMA episodes were observed. On the other hand, during the last 20 minutes containing a GER episode, 5 RMMA episodes and other high electromyographic bursts were observed. Audio-video data confirmed that the subject was not awake during the analysis period. The sampling frequency of 4 Hz for the pH and 1 kHz for the electromyogram was reduced to 1 Hz to draw these traces.

RMMA[6,8,9] and swallowing during sleep.[11,13-15] It was recently reported that a swallowing event often occurs during nocturnal RMMA episodes, particularly during the last part of the episode.[7] Therefore, it is suggested that nocturnal bruxism may be secondary to nocturnal GER, occurring via sleep arousal, often together with swallowing.

It is generally considered that gastric acid in the esophagus due to GER during sleep is mainly cleared by primary peristalsis, caused by saliva swallowing, and secondary peristalsis that is considered to be important during sleep[47] because the swallowing frequency during sleep decreases to approximately one tenth of that during wakefulness.[48-50] Saliva swallowing is important in lubricating the esophagus, pharynx, and mouth[11,13-15] because saliva contains both bicarbonate ions, which play an important role in neutralization of acid,[51] and epidermal growth factor, which is important in the esophageal protective mechanism.[52] Furthermore, it has been shown that salivary flow and bicarbonate ion concentration increase with decreasing esophageal pH, particularly when esophageal pH is less than 1.8.[53] The results of the present study showed that RMMA episodes were frequently observed with decreasing esophageal pH, particularly when esophageal pH is less than 3.0 or 4.0, often together with possible saliva swallowing. Therefore, the increase in salivation and RMMA may be associated with each other, but the physiologic link remains to be elucidated.

Acid stimulation in the esophagus,[54] gustatory stimulation by acid in the mouth,[55] and pressure stimulation of mechanoreceptors in the periodontium around the teeth during mastication have all been well established as causes of stimulation of salivary secretion during wakefulness.[56-59] In fact, previous epidemiologic studies have shown that GERD frequently occurs in older persons[60,61] who have lost much sensation via periodontal mechanoreceptors due to the loss of a large number of teeth.[61,62] The threshold of pressure stimulation to periodontal mechanoreceptors of teeth has been reported to be approximately 5% of the comfortable chewing force.[60,62] The cut-off value of EMG amplitude for RMMA is known to be 10% of the amplitude during maximum voluntary clenching of the teeth.[5] Therefore, there is a possibility that the pressure stimulation of periodontal mechanoreceptors caused by RMMA may stimulate salivary secretion during sleep as well as during wakefulness. However, it remains unknown as to whether RMMA stimulates the salivary secretion during sleep.

According to the results of previous randomized, double-blind, placebo-controlled, clinical trials of the effect of medicine on sleep bruxism,

Table 2—Nocturnal rhythmic masticatory muscle activity episodes after administration of placebo and proton pump inhibitor medication*

Variables	Control group (n=10)	Bruxism group (n=10)
Placebo medication		
Recording time (min)	5.5 ± 0.9	6.0 ± 0.9
Frequency (times/h)	1.9 ± 3.2†	6.0 ± 2.2†‡
Duration per episode (s)	9.4 ± 2.8	10.2 ± 4.4
PPI medication		
Recording time (min)	5.8 ± 0.9	5.9 ± 0.9
Frequency (times/h)	1.0 ± 0.6†	3.7 ± 1.9†‡
Duration per episode (s)	9.4 ± 3.8	9.2 ± 4.4

*Data are given as mean ± SD.
†$P<0.01$ (Paired t-test or Wilcoxon rank sum test)
‡$P<0.01$ (Unpaired t-test or Mann-Whitney U test)

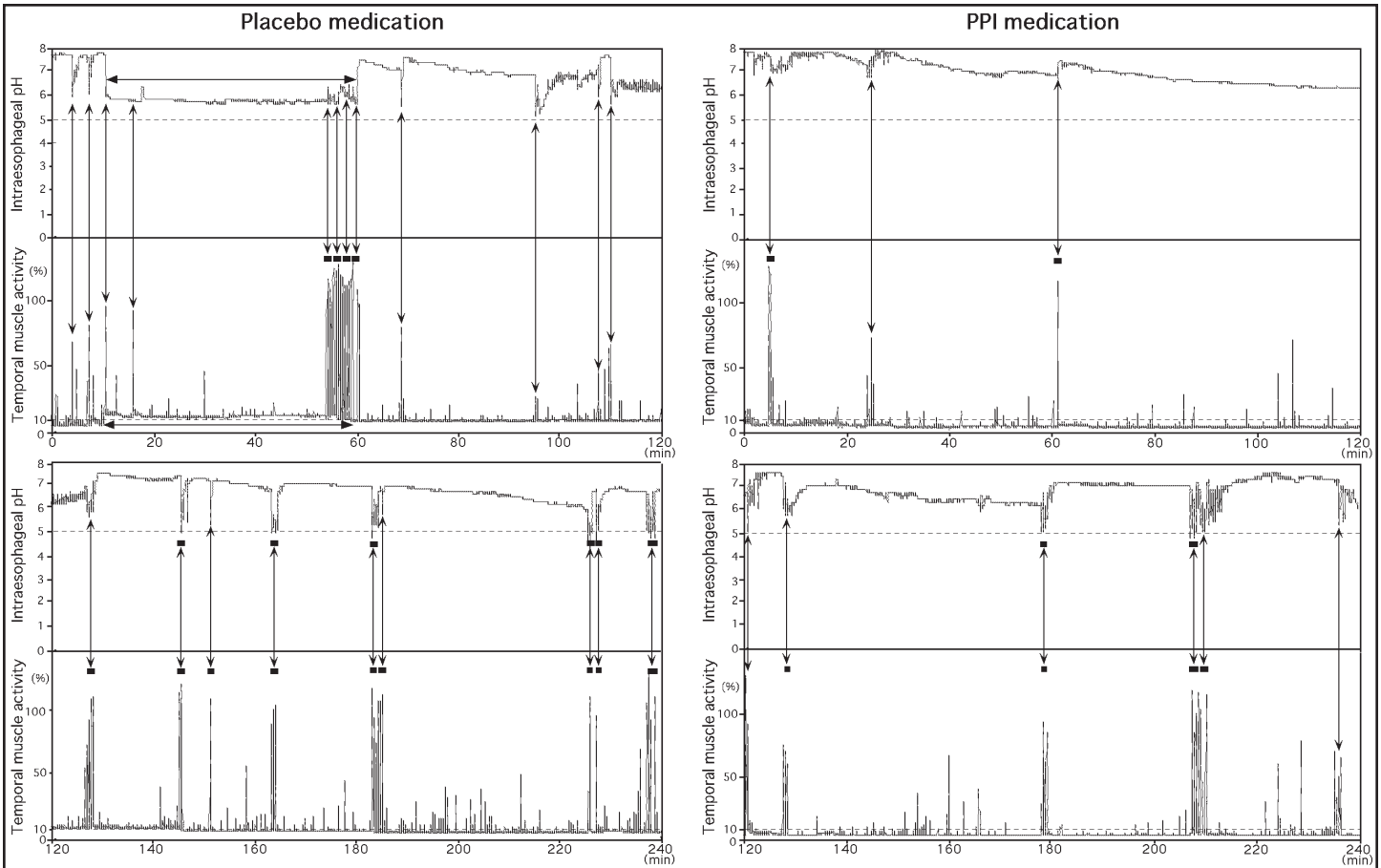

Figure 2—An example of traces of intraesophageal pH and normalized temporal muscle activity for 4 successive hours (excluding the first hour after going to bed) in a subject. Left: after placebo medication, Right: after administration of a proton pump inhibitor (PPI) (ie, a gastric–acid-inhibiting drug) medication. The rectangular marks show nocturnal gastroesophageal reflux (GER) episodes with a pH less than 5.0 (upper dashed line) and rhythmic masticatory muscle activity (RMMA) episodes. The frequency of GER and RMMA episodes with PPI medication were lower than those with placebo medication. Irrespective of the drug treatment, high electromyographic (EMG) bursts such as an RMMA episode and relatively low EMG bursts, probably representing saliva swallowing, occurred during the GER episode or when the intraesophageal pH decreased rapidly (see vertical arrows). During the time period from approximately 10 to 60 minutes with a continuous low intraesophageal pH, many EMG bursts with more than 10% amplitude (lower dashed line) including 4 RMMA episodes were observed with the administration of a placebo medication (see horizontal arrows). Audio-video data confirmed that the subject was not awake during the analysis period. The sampling frequency of 4 Hz for the pH and 1 kHz for the EMG was reduced to 1 Hz to draw these traces.

it has been reported that the use of tricyclic antidepressant amitriptyline is not effective[63] but that the catecholamine precursor L-dopa is effective.[64] However, in more than 50% of cases examined in that study, the rate of decrease in the frequency of bruxism episodes was less than the day-to-day variability (ie, 25% coefficient of variation).[26] In the present study, the mean rate of decrease in the frequency of bruxism episodes with the administration of the PPI medication was approximately 40% (ie, higher than the variability).[5] Therefore, we consider that a PPI (ie, a gastric–acid-inhibiting drug) may be a more effective drug to reduce the frequency of bruxism episodes than L-dopa.

ACKNOWLEDGEMENT

We thank Professor Gilles J. Lavigne for helpful comments on this project.

REFERENCES

1. Thorpy MJ. International classification of sleep disorders: diagnostic and coding manual. Rochester, MN: American Sleep Disorders Association; 1997.
2. American Academy of Orofacial Pain. Orofacial Pain: Guidelines for Assessment, Classification, and Management. Chicago: Quintessence; 1996:223-68.
3. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep 1994;17:739-43.
4. Lavigne GJ, Manzini C. Bruxism. In: Kryger MH, Roth T, Dement W, eds. Principles and Practice of Sleep Medicine. Philadelphia: WB Saunders; 2000:773-85.
5. Lavigne GJ, Rompre PH, Poirier G, Huard H, Kato T, Montplaisir JY. Rhythmic masticatory muscle activity during sleep in humans. J Dent Res 2001;80:443-8.
6. Kato T, Rompre P, Montplaisir JY, Sessle BJ, Lavigne GJ. Sleep bruxism: an oromotor activity secondary to micro-arousal. J Dent Res 2001;80:1940-4.
7. Miyawaki S, Lavigne GJ, Mayer P, Guitard F, Montplaisir JY, Kato T. Association between sleep bruxism, swallowing-related laryngeal movement, and sleep positions. Sleep 2003;26:461-5.
8. Macaluso GM, Guerra P, Di Giovanni G, Boselli M, Parrino L, Terzano MG. Sleep bruxism is a disorder related to periodic arousals during sleep. J Dent Res 1998;77:565-73.
9. Kato T, Montplaisir JY, Guitard F, Sessle BJ, Lund JP, Lavigne GJ. Evidence that experimentally induced sleep bruxism is a consequence of transient arousal. J Dent Res 2003;82:284-8.
10. Freidin N, Fisher MJ, Taylor W, et al. Sleep and nocturnal acid reflux in normal subjects and patients with reflux oesophagitis. Gut 1991;32:1275-9.
11. Orr WC, Robinson MG, Johnson LF. The effect of esophageal acid volume on arousals from sleep and acid clearance. Chest 1991;99:351-4.
12. Vandenplas Y, Hauser B. Gastro-oesophageal reflux, sleep pattern, apparent life threatening event and sudden infant death. The point of view of a gastro-enterologist. Eur J Pediatr 2000;159:726-9.
13. Helm JF. Esophageal acid clearance. J Clin Gastroenterol 1986;8:5-11.
14. Kahrilas PJ. Esophageal motor activity and acid clearance. Gastroenterol Clin North Am 1990;19:537-50.
15. Orr WC, Johnson LF, Robinson MG. Effect of sleep on swallowing, esophageal peristalsis, and acid clearance. Gastroenterology 1984;86:814-9.
16. Kawahara H, Dent J, Davidson G. Mechanisms responsible for gastroesophageal reflux in children. Gastroenterology 1997;113: 399-408.
17. Foresman BH. Sleep-related gastroesophageal reflux. J Am Osteopath Assoc 2000;100:7-10.
18. Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. Chest 2001;19:53-61.
19. Pandolfino JE, Kahrilas PJ. Smoking and gastro-oesophageal reflux disease. Eur J Gastroenterol Hepatol 2000;12:837-42.
20. Pehl C, Pfeiffer A, Wendl B, Kaess H. The effect of decaffeination of coffee on gastro-oesophageal reflux in patients with reflux disease. Aliment Pharmacol Ther 1997;11:483-6.
21. Bujanda L. The effects of alcohol consumption upon the gastrointestinal tract. Am J Gastroenterol 2000;95:3374-82.
22. Bradley LA, Richter JE, Pulliam TJ, et al. The relationship between stress and symptoms of gastroesophageal reflux: the influence of psychological factors. Am J Gastroenterol 1993;88:11-9.
23. Hublin C, Kaprio J, Partinen M, Koskenvuo M. Sleep bruxism based on self-report in a nationwide twin cohort. J Sleep Res 1998;7:61-7.
24. Cameron AJ, Lagergren J, Henriksson C, Nyren O, Locke GR 3rd, Pedersen NL. Gastroesophageal reflux disease in monozygotic and dizygotic twins. Gastroenterology 2002;122:55-9.
25. Lavigne GJ, Rompre PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res 1996;75:546-52.
26. Lavigne GJ, Guitard F, Rompre PH, Montplaisir JY. Variability in sleep bruxism activity over time. J Sleep Res 2001;10:237-44.
27. Metersky ML, Castriotta RJ. The effect of polysomnography on sleep position: possible implications on the diagnosis of positional obstructive sleep apnea. Respiration 1996;63:283-7.
28. Gallo LM, Lavigne G, Rompre P, Palla S. Reliability of scoring EMG orofacial events: polysomnography compared with ambulatory recordings. J Sleep Res 1997;6:259-63.
29. Moller E. Action of the muscles of mastication. Vol.1. In: Kawamura Y, eds. Frontiers of Oral Physiology. Basel: Karger; 1974:121-58.
30. Schindlbeck NE, Heinrich C, Konig A, Dendorfer A, Pace F, Muller-Lissner SA. Optimal thresholds, sensitivity, and specificity of long-term pH-metry for the detection of gastroesophageal reflux disease. Gastroenterology 1987;93:85-90.
31. Kahrilas PJ, Quigley EM. Clinical esophageal pH recording: a technical review for practice guideline development. Gastroenterology 1996;110:1982-96.
32. Williams MP, Pounder RE. Review article: the pharmacology of rabeprazole. Aliment Pharmacol Ther 1999;13: 3-10.
33. Swan SK, Hoyumpa AM, Merritt GJ. Review article: the pharmacokinetics of rabeprazole in health and disease. Aliment Pharmacol Ther 1999;13:11-7.
34. Miyawaki S, Ohkochi N, Kawakami T, Sugimura M. Changes in masticatory muscle activity according to food size in experimental human mastication. J Oral Rehabil 2001;28:778-84.
35. Irvin TT, Perez-Avila C. Diagnosis of symptomatic gastroesophageal reflux by prolonged monitoring of lower esophageal pH. Scand J Gastroenterol 1977;12:715-20.
36. Rosen SN, Pope CE 2nd. Extended esophageal pH monitoring. An analysis of the literature and assessment of its role in the diagnosis and management of gastroesophageal reflux. J Clin Gastroenterol 1989;11:260-70.
37. Orr WC. Gastrointestinal disorders. In: Kryger MH, Roth T, Dement WC, eds. Principles and Practice of Sleep Medicine. Philadelphia: WB Saunders; 2000:1113-22.
38. Rechtschaffen A, Kales A, eds. A manual of standardized terminology, techniques, and scoring system for sleep stages of human subjects. Los Angeles: Brain Information Service/ Brain Research Institute, UCLA; 1968.
39. Kasapidis P, Xynos E, Mantides A, et al. Differences in manometry and 24-H ambulatory pH-metry between patients with and without endoscopic or histological esophagitis in gastroesophageal reflux disease. Am J Gastroenterol 1993;88:1893-9.
40. Bartlett DW, Evans DF, Smith BG. The relationship between gastro-oesophageal reflux disease and dental erosion. J Oral Rehabil 1996;23:289-97.
41. Khan F, Young WG, Daley TJ. Dental erosion and bruxism. A tooth wear analysis from south east Queensland. Aust Dent J 1998;43:117-27.
42. Stephan AD. Diagnosis and dental treatment of a young adult patient with gastroesophageal reflux: a case report with 2-year follow-up. Quintessence Int 2002;33:619-26.
43. Gay T, Rendell JK, Spiro J. Oral and laryngeal muscle coordination during swallowing. Laryngoscope 1994;104:341-9.
44. DeVault KR. Acid infusion does not affect intraesophageal balloon distention-induced sensory and pain thresholds. Am J Gastroenterol 1997;92:947-9.
45. Orr WC, Johnson LF. Responses to different levels of esophageal acidification during waking and sleep. Dig Dis Sci 1998;43:241-5.
46. Cohen JA, Arain A, Harris PA, et al. Surgical trial investigating nocturnal gastroesophageal reflux and sleep (STINGERS). Surg Endosc 2003;17:394-400.
47. Holloway RH. Esophageal body motor response to reflux events: secondary peristalsis. Am J Med 2000;108: 20S -6.
48. Lear CSC, Flanagan JB Jr, Moorrees CFA. The frequency of deglutition in man. Arch Oral Biol 1965;10:83-99.
49. Lichter I, Muir RC. The pattern of swallowing during sleep. Electroencephalogr Clin Neurophysiol 1975;38:427-32.
50. Rudney JD, Ji Z, Larson CJ. The prediction of saliva swallowing frequency in humans from estimates of salivary flow rate and the volume of saliva swallowed. Arch Oral Biol 1995;40:507-12.
51. Mandel ID. The functions of saliva. J Dent Res 1987;66:623-7.
52. Sarosiek J, McCallum RW. Do salivary organic components play a protective role in health and disease of the esophageal mucosa? Digestion 1995;56:32-7.
53. Dutta SK, Matossian HB, Meirowitz RF, Vaeth J. Modulation of salivary secretion by acid infusion in the distal esophagus in humans. Gastroenterology. 1992;103:1833-41.
54. Garrett JR, Proctor GB. Control of salivation. In: Linden RWA, ed. The scientific basis of eating. taste, smell, mastication, salivation and swallowing and their dysfunctions. Frontiers of Oral Biology. Basel: Karger; 1998;9:135-55.
55. Shannon IL. The biochemistry of human saliva in health and disease. In: NH Rowe, ed. Salivary glands and their secretion. Ann Arbor: University of Michigan Press; 94-121.
56. Anderson DJ, Hector MP. Periodontal mechanoreceptors and parotid secretion in animals and man. J Dent Res 1987;66:518-23.
57. Hector MP, Linden RW. The possible role of periodontal mechanoreceptors in the control of parotid secretion in man. Q J Exp Physiol 1987;72:285-301.
58. Jensen Kjeilen JC, Brodin P, Aars H, Berg T. Parotid salivary flow in response to mechanical and gustatory stimulation in man. Acta Physiol Scand 1987;131:169-75.
59. Dodds MW, Hsieh SC, Johnson DA. The effect of increased mastication by daily gum-chewing on salivary gland output and dental plaque acidogenicity. J Dent Res 1991;70:1474-8.
60. Anderson DJ, Hector MP, Linden RW. The effects of unilateral and bilateral chewing, empty clenching and simulated bruxism, on the masticatory-parotid salivary reflux in man. Exp Physiol 1996;81:305-12.
61. Heading RC. Epidemiology of oesophageal reflux disease. Scand J Gastroenterol Suppl 1989;168:33-7.
62. Gaengler P, Goebel G, Kurbad A, Kosa W. Assessment of periodontal disease and dental caries in a population survey using the CPITN, GPM/T and DMF/T indices. Community Dent Oral Epidemiol 1988;16:236-9.
63. Mohamed SE, Christensen LV, Penchas J. A randomized double-blind clinical trial of the effect of amitriptyline on nocturnal masseteric motor activity (sleep bruxism). Cranio 1997;15:326-32.
64. Lobbezoo F, Lavigne GJ, Tanguay R, Montplaisir JY. The effect of catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. Mov Disord 1997;12:73-8.