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Modulation of the Stretch Reflex of Jaw-Closing Muscles in Different Modes and Phases of Respiration

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Kiyo Otani-Saito, DDS;[a] Takashi Ono, DDS, PhD;[b] Yasuo Ishiwata, DDS, PhD;[c] Takayuki Kuroda, DDS, PhD

ABSTRACT

The objective of this study was to investigate whether and how changes in the mode of respiration affect the electromyographic activity of human jaw-closing muscles. Fifteen men were examined in this study. A pair of surface electrodes was attached bilaterally to the masseter and anterior and posterior temporalis muscles for electromyographic recording. Respiratory movements of the chest wall and nasal airflow were recorded simultaneously. Recordings were performed with subjects in the sitting position during quiet nasal and oral respiration. The stretch reflex of jaw-closing muscles was elicited by randomly tapping the chin with an impulse hammer. In 11 subjects, we measured nasal resistance with a rhinomanometer. The amplitude of electromyographic activities of the masseter and anterior temporalis muscles during oral respiration was significantly less than that during nasal respiration, whereas that of the posterior temporalis muscle showed no significant difference between the different modes of respiration. Furthermore, the reduction in the amplitude of the electromyographic activity was more evident in the inspiratory phase during oral respiration. There was a significant positive correlation between the ratio of the reflex amplitude during inspiration in the 2 respiratory modes and nasal resistance for the masseter muscle, but not for the anterior temporalis muscle. These results suggest that the reflexive electromyographic activity of some human jaw-closing muscles is modulated during oral respiration.

KEY WORDS: Oral respiration, Stretch reflex, Jaw-closing muscle, Nasal resistance.

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INTRODUCTION Return to TOC

Many investigators have evaluated the effects of oral respiration on the neuromuscular system and craniofacial morphology.[1–13] In a chronic animal experiment, it has been shown that the tonic electromyographic (EMG) activity of the tongue and orbicularis oris muscles are markedly enhanced after blockage of nasal airflow.[5,6] Early adaptation to oral respiration recruits rhythmic EMG activity in pace with respiration in several orofacial muscles, including jaw-opening muscles. Furthermore, Harvold et al[7] demonstrated that the rhesus monkey with 2-year complete nasal obstruction develops a lower mandibular posture and spread lips to establish an oral passage for respiration, leading to an increase in the anterior facial height.

It is assumed that the mechanism for keeping the mandible in the lowered position during oral respiration can be explained by the increased EMG activity of jaw-opening muscles. However, the decreased EMG activity of jaw-closing muscles may also play a role. Indeed, in an acute experimental setting, Ono et al[11] showed that obstruction of the nasal airway in the cat significantly inhibited the tonic stretch reflex of the masseter muscle. Although this inhibition seemed to be more evident during inspiration, no quantitative analysis was performed. They suggested that the masseteric stretch reflex that involves monosynaptic projection of the afferent fibers from the muscle spindle to the trigeminal motor nucleus plays an important role in controlling the mandibular position.

On the basis of clinical observations, it is well accepted that many children who breathe through their mouths have a peculiar manifestation in the craniofacial region. This includes excessive anterior facial height, incompetent lip posture, steep mandibular plane, V-shaped maxillary arch, anterior open bite, and posterior cross bite.[1–4,8,9,12] These features may be related to so-called adenoid face or long-face syndrome. Woodside et al[4] demonstrated that adenoidectomy induced a change from mouth-open to mouth-closed breathing, and this change was accompanied by greater mandibular growth expressed at the chin, which was caused by a change in the direction of mandibular growth rather than by a change in the mandibular length.

Principato[12] demonstrated that many mouth-breathers have a risk of excessive eruption of molars, resulting in a clockwise rotation of the mandible and an anterior open bite. This also causes a disproportionate increase in the anterior facial height.[12] Recently, Vig[14] reported that oral respiration did not necessarily cause the typical long-face syndrome. She pointed out that one should consider other factors that contribute to nasorespiratory obstruction, as well as individual variations such as the degree, the age of onset, and the duration of such conditions.

We hypothesized that the EMG activity of jaw-closing muscles decreased during oral respiration to open the mouth and enhance the oral airflow. To ascertain this hypothesis, we used the stretch reflex as an index of the jaw-closing-muscle activity, because it plays an important role in determining the mandibular position and simply represents the excitability of trigeminal motoneurons. Thus, the aim of this study was to examine whether and how the EMG activity of human jaw-closing muscles is modulated when nasal airflow is restricted.

MATERIALS AND METHODS

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Fifteen skeletal Class I men, 26.0 ± 1.07 years old (mean ± standard deviation [SD]) participated in this study. Each subject had a normal occlusion, and none showed symptoms of neuromuscular or temporomandibular disorders. All of the subjects were in good health and did not have a cold or allergic rhinitis. The subjects did not open their mouths when they breathed objectively. In addition, none of the subjects reported that they breathed through the mouth subjectively. Informed consent was obtained from each subject before the study.

A pair of surface electrodes was attached bilaterally to the masseter and anterior and posterior temporalis muscles at the point of maximum thickening during clenching and in line with the muscle fibers for EMG recording. A reference electrode was attached to the earlobe. Respiratory movements of the chest wall and nasal airflow were simultaneously recorded by an inductance band (TR-751T; Nihon-Kohden, Tokyo, Japan) and a thermistor (TR-762T; Nihon-Kohden, Tokyo, Japan), respectively. Subjects were seated comfortably in a straight-backed chair with a headrest, and mandibular position was maintained in an almost identical resting position during both nasal and oral respiration.

Before the investigation, background EMG activity was recorded during quiet nasal and oral respiration. During oral respiration, the nasal passage was completely blocked by placing a nose clip on both nostrils. The subject was instructed to close his eyes during data recording. The stretch reflex of jaw-closing muscles was elicited by tapping the midsagittal region of the chin with an impulse hammer (GK-3100; Ono Sokki, Kanagawa, Japan), the tip of which was covered with rubber (Figure 1A). Downward and backward taps were given randomly, but the intertap interval was always more than 1 second. The respiratory mode was changed from nasal to oral respiration, and this procedure was sequentially repeated 3 times in each subject. In each respiratory mode, we tapped the chin 30–40 times and thus obtained a total of 90–120 reflexive EMG activities for each respiratory mode.

To eliminate any possible anxiety and fatigue effects and examiner's bias, we randomly chose 30–40 waves of reflexive EMG activities in each respiratory mode as judged from the waveform of the chest-wall movements. This produced 15–20 waves in each respiratory phase in a given respiratory mode. While sampling, reflexive EMG activities elicited during transient periods (approximately 500 ms) of respiration were discarded. In 11 subjects, we measured nasal resistance by the anterior-nozzle method with a rhinomanometer (MPR-3100; Nihon-Kohden, Tokyo, Japan) immediately before and after EMG recording (3 times each). The mean nasal resistance was calculated for each of these subjects.

EMG signals were amplified, band-pass filtered at 30 Hz to 1 kHz, and full-wave rectified. After conversion of the EMG signals through an analog-to-digital converter (Maclab/8s; ADinstruments, Castle Hill, Australia), they were stored in a personal computer for data analysis. The trigger for starting the sampling process was the onset of stimulation with the impulse hammer. The amplitude of the stimulation was measured from the baseline to the peak in the waveform of the stimulation. We evaluated the amplitude as the difference between the minimum and the maximum of the first phase in the rectified reflex activity. We evaluated the latency as the time from the onset of stimulation to the onset of the reflex (Figure 1B). Each of these onsets was determined as the time of the response corresponding to the baseline plus 3 SD of the mean. We compared the mean value of the amplitude and latency between the 2 modes of respiration and among different phases of respiration.

In the statistical analysis, we first confirmed that there were no significant differences among variances by using both the F test and Kolmogorov-Smirnov test. The amplitudes of the stimulation artifact during nasal and oral respiration were compared by using the unpaired *t*-test in each subject. A paired *t*-test was performed to identify any significant differences in background EMG activity, mean amplitudes, and latencies of the reflex between nasal and oral respiration. A 1-way repeated analysis of variance and contrasts were used to compare mean amplitudes among different respiratory modes. The Spearman rank test was used to determine the correlation between the ratio of the inspiratory EMG amplitude of jaw-closing muscles during oral to nasal respiration and the nasal resistance. All procedures were performed with commercially available statistical software (StatView 5.0; Hulinks, Tokyo, Japan).

RESULTS Return to TOC

Figure 2 illustrates a typical simultaneous record of chest-wall movement, nasal airflow, the timing of stimulation applied by the impulse hammer, and EMG activities of the masseter and anterior and posterior temporalis muscles during nasal and oral respiration. Reflexive EMG activities in the anterior and posterior temporalis muscles are elicited shortly after stimulation, though some of them are barely visible (Figure 2).

The reflexive EMG activities during nasal respiration appeared to be greater than those during oral respiration. Modulation of the reflexive EMG activities of jaw-closing muscles is demonstrated in Figure 3. There was a marked reduction in the amplitude of the EMG activities of the masseter and the anterior and posterior temporalis muscles during oral respiration. The amplitude of the stimulation artifact showed no significant difference between nasal and oral respiration.

Differences in the mean amplitude of reflexive EMG activities between the 2 respiratory modes are summarized in Figure 4. The mean amplitude of reflexive EMG activities was significantly decreased during oral respiration compared with that during nasal respiration in both the masseter and anterior temporalis muscles ($P < .01$). However, the mean amplitude of the reflexive EMG activity in the posterior temporalis muscle showed no significant difference between nasal and oral respiration. To identify the respiratory phase in which the reduction in the amplitude of EMG activity was more evident, we compared the mean amplitude of reflexive EMG activities of jaw-closing muscles in each respiratory phase during nasal and oral respiration (Figure 5). In the masseter muscle, there was a significant reduction ($P < .01$) in the mean amplitude in the inspiratory phase during oral respiration compared with those in the inspiratory and expiratory phases during nasal respiration and that in the expiratory phase during oral respiration. In the anterior temporalis muscle, there was a significant reduction in the mean amplitude in the inspiratory phase during oral respiration compared with those in the inspiratory ($P < .05$) and expiratory ($P < .01$) phases during nasal respiration.

Differences in the mean latency of reflexive EMG activities between the 2 respiratory modes are summarized in Figure 6. In contrast to the changes in the mean amplitude, there were no significant differences in the latency of reflexive EMG activities of the masseter and anterior temporalis muscles between nasal and oral respiration.

Furthermore, we compared the mean latency of reflexive EMG activities of the masseter and anterior temporalis muscles among each respiratory phase during nasal and oral respiration to examine any effect of respiratory phase on the latency (Figure 7). Although there were no significant differences in the latency of the masseter and anterior temporalis muscles, the latency of reflexive EMG activity of the masseter ($P = .073$ between inspiratory EMG activity during oral and nasal respiration) and anterior temporalis ($P = .079$ between inspiratory EMG activity during oral inspiration and expiratory EMG activity during nasal expiration) muscles in the inspiratory phase during oral respiration tended to increase when compared with those of other respiratory phases or modes.

Figure 8 shows the relationship between the ratio of the EMG amplitude of the masseter muscle in the inspiratory phase during oral respiration to that during nasal respiration and the mean nasal resistance. There was a significant positive correlation between these 2 variables for the masseter muscle ($P < .05$), whereas there was no significant correlation between these 2 variables for the anterior temporalis muscle.

DISCUSSION Return to TOC

This study showed that the magnitudes of the stretch reflex of both the masseter and anterior temporalis muscles were significantly modulated during oral respiration, especially in the inspiratory phase. This suggests that a change in the respiratory mode is accompanied by a phase-linked change in the EMG activity of some jaw-closing muscles.

Methodological considerations

Because jaw-closing muscles usually exhibit no respiratory-related EMG activity during quiet breathing,[15,16] we used the stretch reflex as an index of the excitability of trigeminal motoneurons. Several previous studies have used the stretch reflex to evaluate the excitability of these motoneurons.[17–27] These studies showed that reflexive EMG activity could be influenced by various factors, including afferents from periodontal ligaments[19] and the region of the temporomandibular joint,[19,28] tapping,[20] head position,[20] sex,[25,29] and anxiety.[25,27] With regard to the influence of tapping, it is known that the intensity of stimulation is directly related to reflexive EMG activity.[20] In our study, we tried to reiterate chin tapping in terms of the intensity and direction. Although we did not use the way of automated mechanical tapping,[27] there were virtually no significant differences in the intensity of the tapping force given to the subject's chin between nasal and oral respiration (Figure 3). Regarding the effect of sexual dimorphism, all of our subjects were men who had no complaints of pain in the temporomandibular joint or other symptoms. Furthermore, we instructed each subject to keep his head resting firmly on the headrest and to maintain his mandible in the rest position to eliminate reflexive effects of periodontal receptors. In addition, the subjects were told to close their eyes during chin tapping to help reduce stress and anxiety. In fact, both the amplitude of the stretch reflex measured by raw EMG signals and latency in this study are comparable with those in previous studies.[5,18,26] Thus, we believe that our study is free from the above factors that can affect reflexive EMG activity, and it is highly likely that the difference in the respiratory mode is responsible for the change in reflexive EMG activity in our study.

In our study, anterior rhinomanometry was used to measure nasal resistance. This technique is less complex and cumbersome than other techniques such as posterior rhinomanometry[30] and SNORT (simultaneous nasal and oral respirometric technique).[31,32] The mean nasal resistance in 11 subjects in our study was 0.285 ± 0.009 Pa/cm^3/s, and this is comparable to values in previous studies in which anterior rhinomanometry was used for subjects without nasorespiratory complications.[30,33]

Modulation of reflexive EMG amplitude among different modes or phases of respiration

The conditions in this study are not directly comparable to physiological conditions in so-called mouth-breathers, because the mandible was in an almost identical position during nasal and oral respiration and the nasal passage was completely blocked during oral respiration. However, knowledge regarding how neuromuscular adaptation occurs in response to an altered respiratory mode is essential. No significant differences were found in the background EMG activity of jaw-closing muscles between nasal and oral respiration (Figure 9), indicating that there were no differences in the resting excitability of relevant motoneurons between nasal and oral respiration in our experimental setting. The amplitudes of reflexive EMG activity of both the masseter and anterior temporalis muscles were significantly decreased during oral respiration. Because the background EMG activity showed no significant differences between the respiratory modes, it is likely that changes in the respiratory mode may affect the threshold of muscle spindles, probably by reducing the excitability of γ-motoneurons, thereby indirectly changing the excitability of trigeminal α-motoneurons via Ia afferent fibers. This assumption is supported by a study performed by Ono et al,[11] who suggested that the γ-system is involved in the inhibition of masseter EMG activity during oral respiration in the cat. Although it is not clear if this is the case in humans, it is evident that oral respiration affected the human trigeminal neuromuscular system. Bishop et al[20] reported that the jaw jerk showed no differences between phases of quiet nasal breathing, which is consistent with our study. Reflexive EMG activities of the masseter and anterior temporal muscles were significantly decreased during the inspiratory phase of oral respiration compared with other respiratory modes and phases. This indicates that it may be necessary to reduce jaw-closing muscle EMG activity to decrease inspiratory resistance through the oral passage and to avoid upper-airway obstruction in the inspiratory phase of oral respiration. A previous finding that jaw-closing muscles exhibited their peak EMG amplitude during expiration supports this assumption.[5]

Differences in responses among the masseter and anterior and posterior temporalis muscles

During oral respiration, there was a significant decrease in the amplitude of reflexive EMG activity in both the masseter and anterior temporalis muscles, whereas there were no significant changes in that of the posterior temporalis muscle. It is unknown why the EMG amplitude of the posterior temporalis muscle showed no significant changes. However, it is possible that the functional differences of jaw-closing muscles may play a role; the masseter muscle consists of 2 parts. The superficial layer acts dominantly in the incisal edge-to-edge position, whereas the deep layer acts dominantly in the retrusive position.[34,35] The temporalis muscle chiefly controls the jaw position against gravity.[36] The anterior and posterior temporalis muscles pull the mandible superoanteriorly and superoposteriorly, respectively. A recent study also reported the heterogenous EMG activities among human jaw-closing muscles.[37]

Changes in the latency of reflexive EMG activity

Unfortunately, we could barely measure the latency of the reflexive EMG activity in the posterior temporalis muscle, because the responses were too small to determine the onset according to our definition (Figure 1B). Thus, we compared only the latency of the masseter and anterior temporalis muscles in different modes of respiration. There were no differences in the latency of the reflexive EMG activity in the masseter and anterior temporalis muscles between nasal and oral respiration. Most previous studies[17–27] on the stretch reflex of the jaw-closing muscles have compared the amplitude, latency, or duration as indices among different subjects and conditions. Interestingly, only the amplitude changed with different modes of respiration; the latency did not. This may be partly explained by the definition of the latency in our study, which is the time lag between the onset of stimulation and the onset of the reflex. In the masseter and anterior temporalis muscles, a value of 3 SD of the baseline EMG activity is greater during nasal than oral respiration (data not shown). This indicates that the latency of reflexive EMG activity during nasal respiration may be prolonged compared with oral respiration according to our definition. In addition, the latency may show few or no changes if a reduction in the firing frequency of recruited motor units occurred.

Relationship between changes in EMG amplitude and nasal resistance

The difference in EMG amplitude of the masseter and anterior temporalis muscles between the 2 respiratory modes may have been mainly caused by a reduction of the EMG amplitude in the inspiratory phase during oral respiration (Figure 5). Therefore, we studied the possible correlation between nasal resistance and the change in the masseter and anterior temporalis muscles in the inspiratory phase of the 2 respiratory modes.

We found a significant correlation between nasal resistance and the rate of change of the masseter EMG amplitude in the inspiratory phase of the 2 respiratory modes. This implies that when the breathing route is changed from the nasal to oral passage, a subject with greater nasal resistance tends to show less change in masseter EMG activity during inspiration, whereas a subject with less nasal resistance tends to show a greater change. Therefore, the excitability of masseter motoneurons in a subject with greater nasal resistance might have already been lower than that in a subject with less nasal resistance because of respiratory inputs even during nasal respiration. Thus, the masseter motoneurons of a subject with greater nasal resistance may be resistant (ie, aplastic) to the inspiratory-related central or peripheral inputs associated with a change in the respiratory mode. On the contrary, these motoneurons in a subject with less nasal resistance may be susceptible (ie, plastic) to such perturbation. One of the reasons why there was no significant correlation between nasal resistance and the rate of change in the EMG amplitude of the anterior temporalis muscle in the inspiratory phase of the 2 respiratory modes may be the lower reduction of the EMG amplitude in this muscle compared with that in the masseter muscle.

CONCLUSION Return to TOC

On the basis of the findings of this study, it is suggested that the reflexive EMG activity of some human jaw-closing muscles (ie, the excitability of corresponding motoneurons) is modulated in the inspiratory phase during oral respiration and that nasal resistance might affect the degree of such modulation.

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[a] Graduate student, Maxillofacial Orthognathics, Graduate School, Tokyo Medical and Dental University, Tokyo, Japan.

[b] Assistant Professor, Maxillofacial Orthognathics, Graduate School, Tokyo Medical and Dental University, Tokyo, Japan.

[c] Part-time lecturer, Maxillofacial Orthognathics, Graduate School, Tokyo Medical and Dental University, Tokyo, Japan.

[d] Professor, Maxillofacial Orthognathics, Graduate School, Tokyo Medical and Dental University, Tokyo, Japan.

Corresponding author: Kiyo Otani-Saito, DDS, Maxillofacial Orthognathics, Graduate School, Tokyo Medical and Dental University, 5-45 Yushima l-chome, Bunkyo-ku, Tokyo, 113-8549 Japan (E-mail: k-ohtani.mort@tmd.ac.jp).

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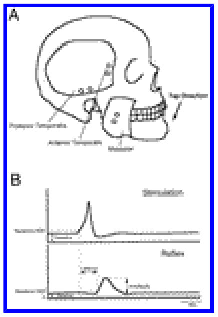

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FIGURE 1. (A) Schematic drawing of the spatial relationship between the direction of chin-tapping and relevant jaw-closing muscles. (B) Definition of the amplitude and latency of reflexive electromyographic activity

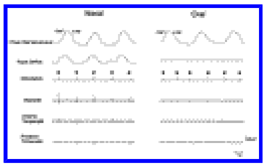

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FIGURE 2. Simultaneous records of electromyographic (EMG) activities of jaw-closing muscles during nasal and oral respiration. The vertical bar represents 25 μV for EMG activities, and the horizontal bar represents 1 s. Dots denote the timing of chin tapping. Nasal indicates nasal respiration; oral, oral respiration; insp, inspiration; and exp, expiration

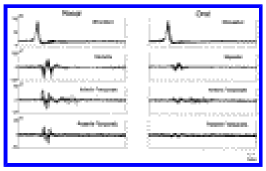

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FIGURE 3. Comparisons of reflexive electromyographic (EMG) activities of jaw-closing muscles in different respiratory modes. Raw EMG activities of the right masseter and anterior and posterior temporalis muscles during nasal and oral respiration were superimposed 30 times

at the onset of stimulation. The horizontal bar represents 10 ms. Nasal indicates nasal respiration; oral, oral respiration; and AU, arbitrary unit

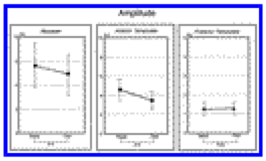

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FIGURE 4. Comparisons of electromyographic amplitudes during nasal and oral respiration. In this and following figures, means and standard errors are indicated by symbols and solid bars, respectively. Filled circles indicate the masseter muscle; filled squares, anterior temporalis muscle; and filled triangles, posterior temporalis muscle. Nasal indicates nasal respiration; oral, oral respiration; and AU, arbitrary unit. **P< .01. N.S. indicates not significant

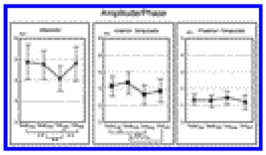

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FIGURE 5. Comparisons of reflexive electromyographic (EMG) amplitudes among different respiratory modes and phases. Nasal$_{insp}$ indicates inspiratory EMG activity during nasal respiration; nasal$_{exp}$, expiratory EMG activity during nasal respiration; oral$_{insp}$, inspiratory EMG activity during oral respiration; oral$_{exp}$, expiratory EMG activity during oral respiration; and AU, arbitrary unit. *P < .05, **P < .01

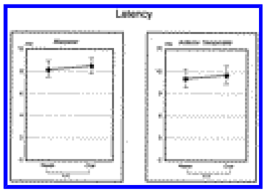

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FIGURE 6. Comparisons of reflexive electromyographic latencies during nasal and oral respiration. There were no significant changes in latencies associated with changes in the respiratory mode. N.S. indicates not significant; nasal, nasal respiration; and oral, oral respiration

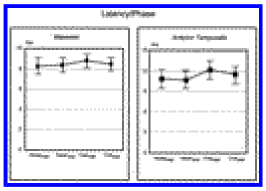

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FIGURE 7. Comparisons of reflexive electromyographic (EMG) latencies among different respiratory modes and phases. There were no significant changes in latencies associated with changes in respiratory modes or phases. Nasal$_{insp}$ indicates EMG latency in the inspiratory phase during nasal respiration; nasal$_{exp}$, EMG latency in the expiratory phase during nasal respiration; oral$_{insp}$, EMG latency in the inspiratory phase during oral respiration; and oral$_{exp}$, EMG latency in the expiratory phase during oral respiration

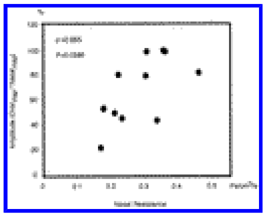

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FIGURE 8. Relationship between nasal resistance (abscissa) and the ratio of the electromyographic (EMG) amplitude of the masseter muscle in the inspiratory phase during oral respiration to that during nasal respiration (ordinate) for 11 subjects. ρ denotes Spearman's rank-difference correlation coefficient. Nasal_{insp}, inspiratory EMG activity during nasal respiration; Oral_{insp}., inspiratory EMG activity during oral respiration; *Pa,* pascal

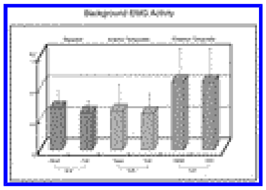

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FIGURE 9. Comparisons of mean background electromyographic (EMG) activities of jaw-closing muscles during nasal and oral respiration. There were no significant differences in background EMG activity associated with changes in the respiratory mode. Standard errors are indicated by solid bars. Abbreviations: Nasal, nasal respiration; Oral, oral respiration; AU, arbitrary unit