ORIGINAL ARTICLE

Inhibition of masseteric electromyographic activity during oral respiration

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Although the effects of oral respiration on the growth and development of craniofacial structure have been studied previously, little is known about how altered respiration affects the activity of the jaw-closing muscles. Obstruction of the nasal airway in the cat significantly inhibited the masseteric stretch reflex and discharges of masseteric motor units but did not affect the electromyographic activity of the diaphragm. This inhibition was greater during inspiration than during expiration. In addition, the amplitude of the masseteric monosynaptic reflex elicited by electrical stimulation of the mesencephalic trigeminal nucleus showed no significant change in association with the altered respiratory mode. These findings suggest that masseteric electromyographic activity is inhibited during oral respiration and that the γ-system is involved in this inhibition. (Am J Orthod Dentofacial Orthop 1998;113:518-25.)

The effects of oral respiration on the growth and development of craniofacial structure have been extensively studied for several decades.[1-3] Previous studies in rhesus monkeys have demonstrated that changes in craniofacial-muscle activity are induced by changes in the respiratory mode from the nasal to the oral pathway; this in turn results in the manifestation of different craniofacial structures.[4-6] In these studies, neuromuscular adaptation to experimental obstruction of the nasal airway was evaluated for 6 months. Vargervik et al.[7] found that the mandible was lowered and the mouth opened in animals after the nasal airway had been obstructed for 2 years. Tonic electromyographic (EMG) activity of one of the jaw-opening muscles (digastric muscle) and a synergist (geniohyoid muscle) was seen in some of these animals, but changes in the EMG activity of the jaw-closing muscles (e.g., masseteric and temporal muscles) were not recorded. It is possible that inhibition of the EMG activity of jaw-closing muscles is associated with oral respiration. A demonstration of acute changes in the function of the jaw-closing muscles caused by changes in the respiratory mode may not provide direct insight into abnormal patterns of the growth and development of craniofacial skeleton. However, knowledge regarding how the neuromuscular system adapts to factors that affect function in the craniofacial region is essential in the assessment of the long-term effects of such factors.

The masseteric stretch reflex, which is analogous to the spinal reflex in the spinal cord, has been identified in the craniofacial region. The central axonal terminals of the neurons in the trigeminal mesencephalic nucleus (MesV) provide monosynaptic projection onto trigeminal motoneurons.[8] Thus an afferent impulse from masseteric muscle spindles provides excitatory input to trigeminal motoneurons, and this reflex arc plays an important role in controlling mandibular position.[9] Because MesV neurons monosynaptically project to masseteric motoneurons, the changes in the amplitude of masseteric EMG activity elicited by electrical stimulation of the MesV in response to external disturbance can be measured to evaluate the excitatory level of masseteric motoneurons.

We hypothesized that the EMG activity of the jaw-closing muscles changes immediately in association with an altered respiratory mode from the nasal to the oral passage. We used the masseteric static stretch reflex to determine the levels of jaw-closing–muscle activity during oral and nasal respiration. We also discuss the putative mechanism underlying this neuromuscular adaptation to the altered respiratory mode.

MATERIAL AND METHODS

We performed experiments on six adult cats (five male, and one female) weighing 2.9 to 3.8 kg. The animals were pretreated with intramuscular chlorpromazine hydrochloride (2 mg/kg) and atropine sulfate (0.1 mg/kg). Thirty minutes later, they were anesthetized with intramuscular ketamine hydrochloride (20 mg/kg). After the

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trachea and right superficial radial vein were cannulated, halothane (1.0% to 2.0%) insufflation was started; it was continued throughout the surgical procedures. Thereafter, the animals were anesthetized with intravenous α-chloralose (initially 50 mg/kg),[10,11] supplemented with repeated doses (10 mg/kg/hr). In three animals, intercollicular decerebration was carried out under halothane anesthesia, which was then discontinued.

The animals were artificially ventilated throughout the recording session, and body temperature was maintained at 36° to 37° C with the use of a radiating lamp from above and an electric heater under the abdomen. The ECG was monitored continuously throughout the operation and during EMG recording sessions. No changes in the ECG were observed when electrical stimulation was applied or terminated. In addition, no motility was observed and no changes were observed in the size of the pupils when the skin was pinched.

At the end of the recording session, the animal was deeply anesthetized with pentobarbital sodium. We marked sites of the tips of stimulating and recording electrodes in the brainstem by passing a DC cathode current (20 μA for 20 seconds) through the electrodes. The animal was perfused transcardially with 0.9% saline solution followed by 10% formalin. Serial transverse frozen sections, 75 mm thick, were made of the brainstem; these were stained by the Klüver-Barrera method to verify the location of the electrolytic lesions.

A skin incision was made to expose the right masseteric muscle. A pair of enamel-coated fine steel wires (diameter, about 200 μm; interpolar distance, about 5 mm) were inserted into the right masseteric muscle, and the incision was sutured. The tip of enamel-coated fine steel wire electrode was bared 2 mm for mass EMG activity recording; however, it was not bared for motor unit recording. An incision was made in the skin over the right abdomen beneath the ribcage to expose the right diaphragm. A pair of enamel-coated fine steel wires, similar to those used in the right masseteric muscle, were inserted into the right diaphragm, and the incision was sutured. A small screw was fixed to the inferior edge of the mental symphysis. A piece of steel ligature wire (diameter, about 250 μm) was attached to the screw.

After the incision was sutured and lidocaine hydrochloride (1.0%) infiltrated to pressure points, the animal was mounted on a stereotaxic apparatus in a prone position. The spinal cord was transected at the C2 level. A midline incision was made in the skin over the cranium. To insert stereotaxically a concentric bipolar electrode (tip resistance, 200 kΩ at 1 kHz) into the right MesV for electrical stimulation to elicit the masseteric monosynaptic reflex, a circular piece of the right parietal bone was removed.

The left nostril of each animal was obstructed with a cone-shaped soft silicone plug. A similar plug containing a small plastic tube (length, about 4 mm; diameter, about 2 mm) was placed in the right nostril. Nasal airflow was monitored with a thermistor attached to the end of the plastic tube. To change the respiratory mode, a small cap was applied to the end of the plastic tube. To elicit the masseteric stretch reflex, passive jaw depression was applied manually to maximum opening in ramp-and-hold manner through the ligature wire attached to the screw implanted in the mental symphysis. The wire was then fixed to a hook on the table under the animal to keep the jaw maximally opened.

The EMG activities of the masseteric muscle and the diaphragm were amplified, bandpass-filtered, and integrated. In the record of the mass potential, a cross-sectional area of the integrated EMG activity for 1 second was randomly measured during both nasal and oral respiration. In the record of the masseteric motor-unit activity, the instantaneous firing frequency was measured during both nasal and oral respiration. When two or more masseteric motor units' activities were simultaneously recorded, the motor unit with the greatest amplitude was analyzed. The masseteric and diaphragm EMG activities during and after oral respiration were standardized to the masseteric and diaphragm EMG activities during nasal respiration immediately before oral respiration, respectively. Changes in the EMG activities of the masseteric muscle and the diaphragm during nasal and oral respiration were considered significant ($p < 0.05$) on ANOVA and Scheffe's test.

RESULTS

Changes in the masseteric and diaphragm EMG activities during oral respiration are shown in Fig. 1. As the respiratory mode changed from the nasal to the oral airway, a significant change was seen in masseteric EMG activity. Masseteric EMG activity gradually decreased nearly to the tonic level before the masseteric stretch reflex was elicited; tonic masseteric EMG activity disappeared and phasic EMG activity remained in the expiratory phase. On the other hand, diaphragm EMG activity showed an abrupt burst. However, the succeeding diaphragm EMG activity was nearly equal to that before the change in the respiratory mode. When the nasal airway was reopened, tonic masseteric EMG activity increased to the same level as before the nasal airway was obstructed, whereas diaphragm EMG activity was not affected by the change in the respiratory mode. When passive jaw-opening was discontinued, masseteric EMG activity decreased to the level before the masseteric stretch reflex was elicited, whereas no significant change was observed in diaphragm EMG activity.

Changes in masseteric motor-unit activity during oral respiration are shown in Fig. 2. When the jaw was passively opened, motor-unit activity was induced and two masseteric motor units fired toni-

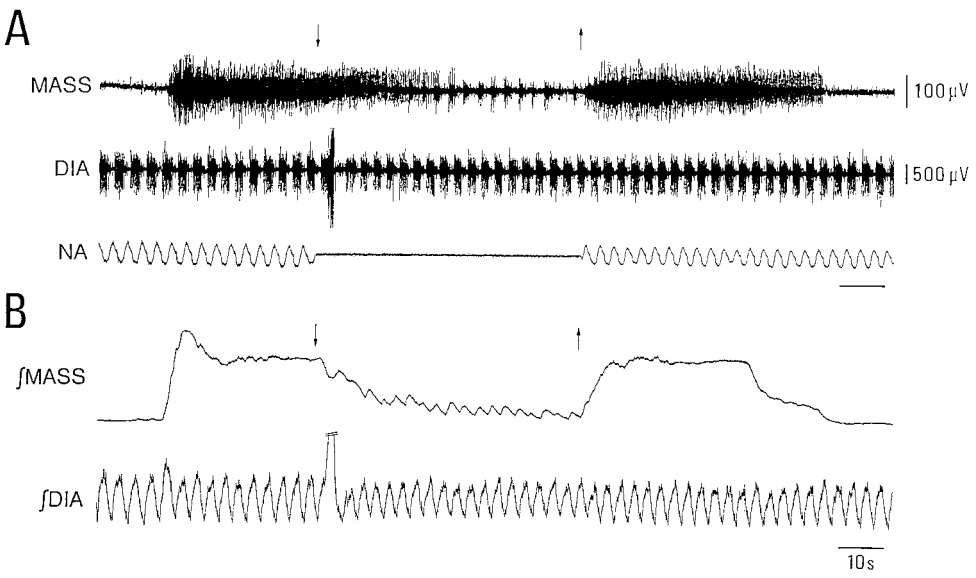

Fig. 1. Masseteric EMG activity before, during, and after oral respiration. *Down and up arrows* indicate onset and offset of oral respiration, respectively. The masseteric stretch reflex was elicited by opening the jaw. **A,** simultaneous record of masseteric and diaphragmatic EMG activities, and nasal airflow. **B,** same record as in **A**. The masseteric and diaphragmatic EMG activities are full-wave rectified and integrated. *MASS,* masseteric EMG activity; *DIA,* diaphragmatic EMG activity, *NA,* nasal airflow, *∫MASS,* rectified and integrated masseteric EMG activity, *∫DIA,* rectified and integrated diaphragmatic EMG activity.

cally, but no significant changes were observed in diaphragm EMG activity. When the nasal airway was obstructed, the instantaneous firing frequency of the masseteric motor unit with greater amplitude gradually decreased. The masseteric motor unit became silent approximately 30 seconds after the onset of oral respiration. During oral respiration, no significant changes were observed in diaphragm EMG activity. When the nasal airway was reopened, the masseteric motor unit resumed tonic firing, but no significant change was observed in diaphragm EMG activity.

Fig. 3 shows the changes in masseteric EMG activity, the instantaneous firing frequency of the masseteric motor unit and diaphragm EMG activity before, during, and after oral respiration. Masseteric EMG activity significantly decreased during oral respiration (Fig. 3A). However, masseteric EMG activity significantly increased when the nasal passage was reopened. Masseteric EMG activity was significantly less during oral respiration than before or after oral respiration, whereas there was no significant difference between the values before and after oral respiration. Likewise, when the nasal passage was obstructed, the number of masseteric motor-unit discharges decreased significantly (Fig. 3B). However, the instantaneous firing frequency of the masseteric motor-unit discharge increased significantly when the nasal passage was reopened. The instantaneous firing frequency of the masseteric motor unit was significantly less during oral respiration than before or after oral respiration. In addition, the instantaneous firing frequency of the masseteric motor unit was significantly less after oral respiration than before oral respiration. Fig. 3C shows the changes in diaphragm EMG activity before, during, and after oral respiration. We noted no significant difference among the diaphragm EMG activities before, during, and after oral respiration.

Changes in the amplitude of the masseteric monosynaptic reflex elicited by electrical stimulation of MesV during oral respiration are shown in Fig. 4. Obstruction of the nasal airway reduced masseteric EMG activity, and reopening of the nasal airway elicited recovery of masseteric EMG activity, whereas no significant changes were observed in diaphragm EMG activity during this procedure (Fig. 4A). Furthermore, no significant changes were observed in the amplitude of the masseteric monosynaptic reflex during oral respiration (Fig. 4B). In-

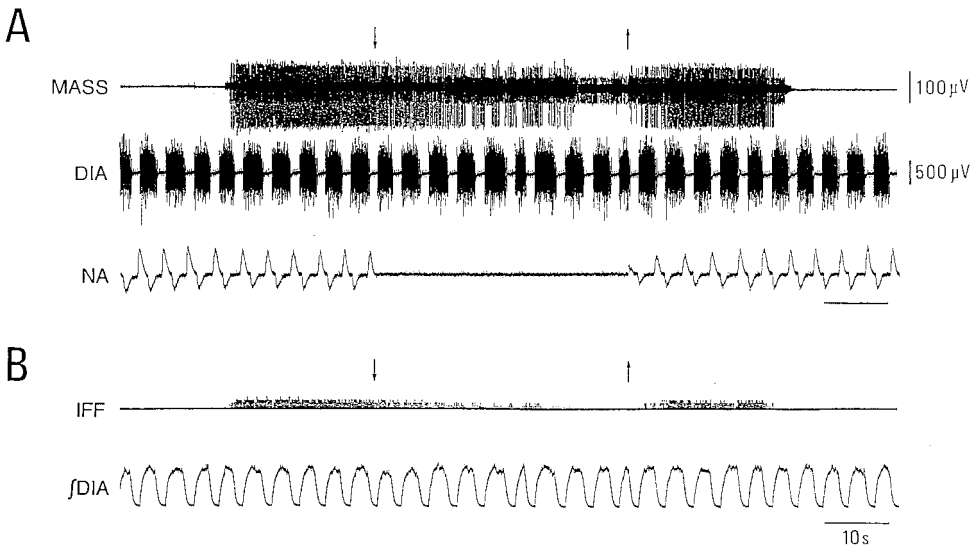

Fig. 2. Spike potentials of the masseteric motor units before, during, and after oral respiration. *Down and up arrows* indicate onset and offset of oral respiration, respectively. The masseteric stretch reflex was elicited by opening the jaw. **A,** Simultaneous record of spike potentials of the masseteric motor unit, the diaphragmatic EMG activity, and nasal airflow. **B,** same record as in **A**. The instantaneous firing frequency of spike potentials of the masseteric motor unit with the greatest amplitude is calculated. The diaphragmatic EMG activity is full-wave rectified and integrated. *MASS,* masseteric EMG activity; *DIA,* diaphragmatic EMG activity; *NA,* nasal airflow, *IFF,* instantaneous firing frequency; *∫DIA,* rectified and integrated diaphragmatic EMG activity.

deed, averaging of the masseteric monosynaptic reflex ($n = 20$) revealed no significant difference in the amplitude before, during, and after oral respiration (Fig. 4C).

DISCUSSION

The results of our study show that the masseteric stretch reflex was significantly inhibited during oral respiration. On the other hand, diaphragm EMG activity was not affected by changes in the respiratory mode. Thus oral respiration preferentially inhibits masseteric EMG activity. In addition, the amplitude of the masseteric monosynaptic reflex elicited by electrical stimulation of the MesV was not affected by changes in the respiratory mode.

The upper-airway dilator muscles and the diaphragm are controlled by the central nervous system through similar mechanisms.[12] However, several reports have documented that the upper-airway dilator muscles and the pump muscle (i.e., the diaphragm) respond differently to external stimulation, including chemical stimulation,[13,14] airway occlusion,[15] and respiratory loading.[16] These studies confirmed that the responses of the upper-airway muscles to respiratory and nonrespiratory stimulation were different from those of the diaphragm. The main function of the masseteric muscle is to close the jaw during speech, mastication, and deglutition. Although the masseteric muscle shows no respiratory-related activity during quiet breathing,[17,18] it shows inspiratory activity in response to inspiratory loading.[17,19] Hollowell and Suratt[17] demonstrated that the masseteric muscle showed respiratory activity when inspiratory resistive loads were applied to the pharynx. In our study, inhibition of both masseteric EMG activity and masseteric motor-unit activity was more evident in the inspiratory phase than in the expiratory phase during oral respiration. Unless contraction of the masseteric muscle is inhibited in the inspiratory phase of oral respiration, the inspiratory resistance of the upper airway might be increased, possibly predisposing the upper airway to collapse. In this sense, the masseteric muscle acts as a respiratory muscle, although it may not substantially contribute to respiration if the nasal airway is maintained. The masseteric muscle may function as an accessory respiratory muscle when the nasal airway is threatened.

Basner et al.[20] showed greater EMG activity in the genioglossus muscle during nasal respiration

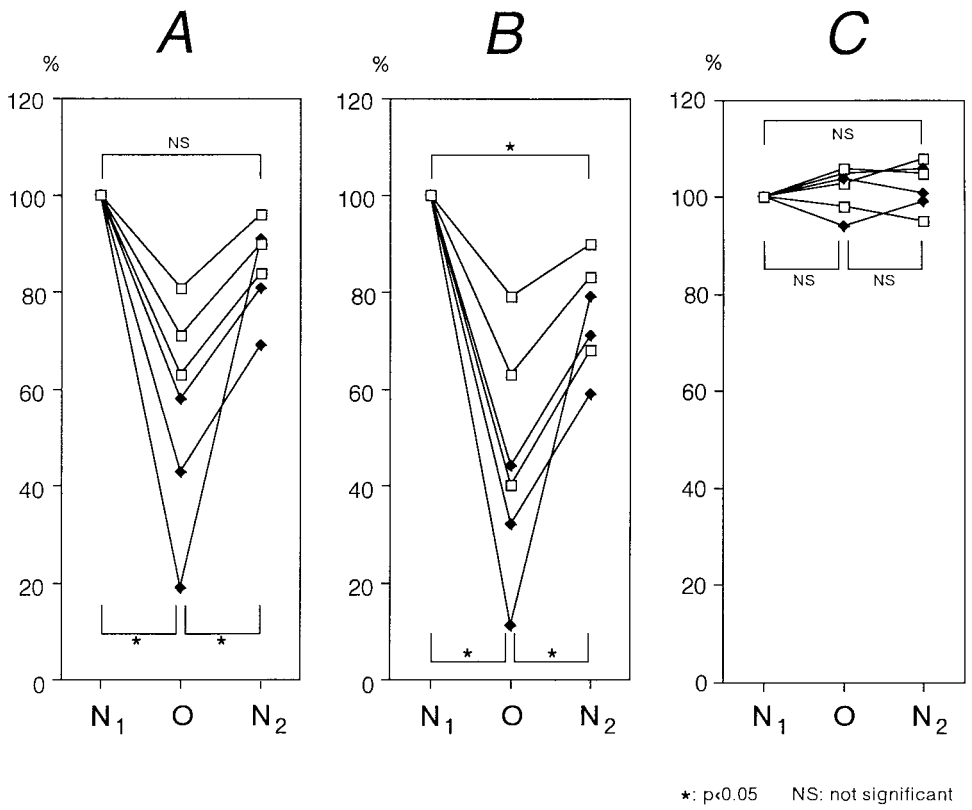

Fig. 3. A, Changes in masseteric EMG activity before, during and after oral respiration. Values are standardized to the values before oral respiration. **B,** Changes in instantaneous firing frequency of the masseteric motor unit before, during and after oral respiration. **C,** Changes in diaphragmatic EMG activity before, during, and after oral respiration. □ and ■ denote values from anesthetized and decerebrate animals, respectively. *N1,* nasal respiration immediately before oral respiration; *O,* oral respiration; *N2,* nasal respiration immediately after oral respiration. *$p < 0.05$; *NS,* not significant.

than during oral respiration; this disappeared with nasal anesthesia in conscious human subjects. They suggested that nasal airflow has an excitatory effect on conscious upper-airway–muscle activity. Nasal airflow has a similar excitatory effect on respiration during sleep,[21] although sleep itself has an inhibitory effect on skeletal muscle tone. Mathew et al.[16] demonstrated that the inspiratory activity of the genioglossus muscle was increased by nasal obstruction, although there was no significant change in diaphragm EMG activity. Because augmented genioglossus EMG activity was observed in the first respiratory phase immediately after obstruction of the isolated upper airway, they suggested that upper-airway mechanoreceptors rather than chemoreceptors were involved in this reflex. In our study, the decrease in masseteric EMG activity occurred during the first breath immediately after the nasal airway was obstructed (Fig. 1). Therefore it is likely that this response is mediated through mechanoreceptors, which respond faster than the detection of significant changes in arterial blood-gas concentrations by chemoreceptors.[22]

Because the masseteric muscle usually shows no measurable respiratory-related changes in its EMG activity,[17,18] we used the masseteric stretch reflex to quantify the change in masseteric EMG activity induced by an altered respiratory mode. Monosynaptic reflex activation of the masseteric muscle by MesV electrical stimulation follows a neuronal pathway similar to that of the masseteric stretch reflex. It is likely that the γ-system is involved in the inhibition of masseteric EMG activity during oral respiration. First, masseteric EMG activity was inhibited even though the magnitude of the inputs from the masseteric muscle spindles was constant before and after oral respiration. This suggests that the magnitude of the inputs from the masseteric muscle

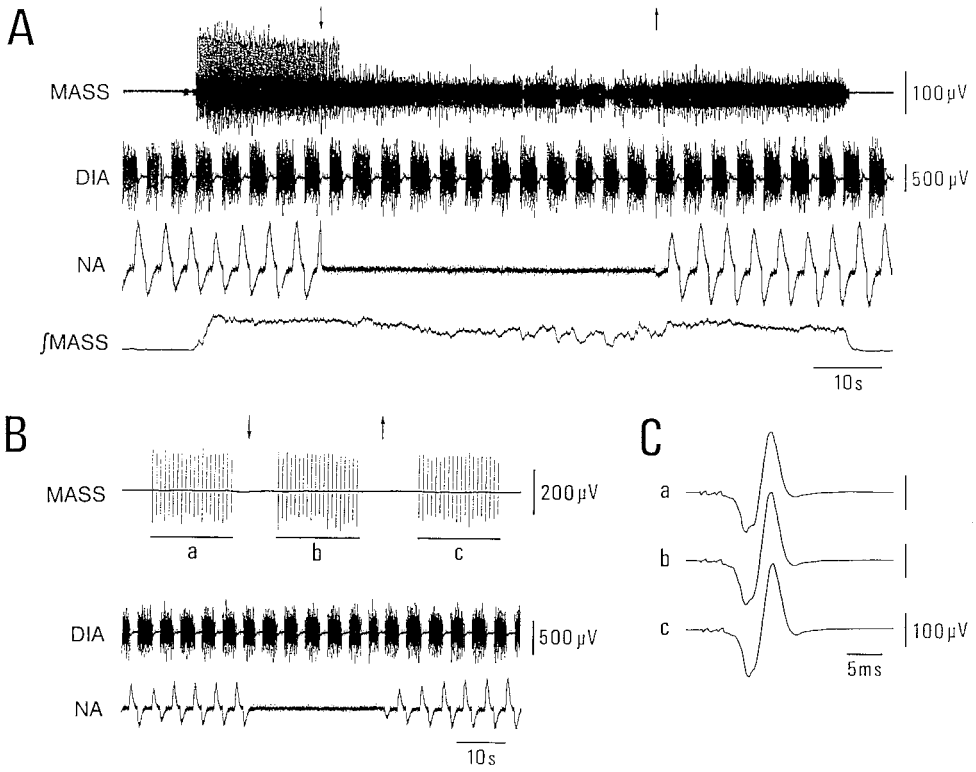

Fig. 4. Effects of oral respiration on masseteric monosynaptic reflex. **A,** Simultaneous record of masseteric and diaphragmatic EMG activities, and nasal airflow. **B,** Simultaneous record of the masseteric monosynaptic reflex, diaphragmatic EMG activity, and nasal airflow. **C,** Changes in the amplitude of the masseteric monosynaptic reflex shown in **B** (a) before, (b) during, and (c) after oral respiration. *Down and up arrows* indicate onset and offset of oral respiration, respectively. *MASS,* masseteric EMG activity; *DIA,* diaphragmatic EMG activity; *NA,* nasal airflow; *∫MASS,* rectified and integrated masseteric EMG activity.

spindles was decreased not as a result fatigue of the masseteric muscle fibers but rather to inhibition of the intraspindle muscle fibers innervated by masseteric γ-motoneurons. Fatigue may not have a significant effect on the mass EMG potential but may have a significant effect on motor unit discharge, depending on the type of the motor unit. Second, the amplitude of the masseteric monosynaptic reflex showed no significant change during oral respiration, indicating that the excitability of the masseteric α-motoneurons did not change significantly. Thus neither excitatory nor inhibitory potentials affected the masseteric α-motoneurons during oral respiration. Intercollicular decerebration induces augmented EMG activity of extensors by way of γ-motoneurons,[23] which also increases masseteric EMG activity. Therefore the effect of oral respiration on masseteric EMG activity would be evident in decerebrated animals if the γ-system is involved in the inhibition of masseteric EMG activity during oral respiration.

The masseteric muscle consists of several compartments containing different types of muscle fibers that respond differently to various environmental demands, including different types of food and different modes of respiration.[24,25] In Fig. 2, two masseteric motor units' activities were simultaneously recorded. The activity of masseteric motor unit with greater amplitude was inhibited, whereas that of the masseteric motor unit with lesser amplitude was not influenced during oral respiration. Because the mass EMG activity consists of integrated activities of several motor units, changes in activity of a motor unit more or less affect the mass EMG activity. We did not identify the type of masseteric muscle fiber influenced during oral respiration. If oral respiration persisted, masseteric EMG activity would be inhibited on a long-term basis, which would in turn produce a new equilibrium among the muscles attached to the mandible. Navarro et al.[26] recently demonstrated that the mandibular plane angle opened along with a clock-

wise rotation of the mandible after deprivation of masseteric muscle contraction in the rat.

From the standpoint of relationship between muscle function and oral respiration, our findings may have some clinical implications. First, it is well-known that many children with large adenoids breathe through the mouth.[27] Their specific facial expression suggests lack of activity of the masseteric muscle. Some children do not start to breathe through the nose even after adenoidectomy.[27] Is the masseteric stretch reflex permanently influenced if the condition has lasted for a long time during a certain period of development? Long-term experiments such as ours could answer the question. Second, sleep-disordered breathing—including snoring and obstructive sleep apnea—has been paid more attention by orthodontists as the use of oral appliances in the treatment of patients with obstructive sleep apnea has been introduced.[28-32] Some patients with obstructive sleep apnea are mouth-breathers during sleep even when they can keep their mouth shut while awake. Even when more or less extensive surgery (e.g., nasal reconstruction, uvulopalatopharyngoplasty, bimaxillary advancement)[33] is performed both in the throat and nose, it is common for the patient to continue breathing through the mouth during sleep, and the upper-airway obstruction persists. Is a stimulation of the masseteric muscle effective in activating the muscle spindle afferents to the MesV when the mouth is opened during sleep? Could the enhanced MesV efferents consequently increase the tongue-protruding (e.g., genioglossus) muscle activity by way of the trigemino-hypoglossal reflex arc? If this is the case, a stimulation of the masseteric muscle may be helpful in moving the tongue forward to prevent upper-airway obstruction in patients with obstructive sleep apnea. Because oral respiration has been shown to be one of the important external influences on the growth and development of craniofacial structure, further studies are needed to assess the clinical relevance of our findings.

CONCLUSIONS

Obstruction of the nasal airway (i.e., oral respiration) in the cat significantly inhibited the masseteric stretch reflex and discharges of masseteric motor units but did not affect the EMG activity of the diaphragm. In addition, the amplitude of the masseteric monosynaptic reflex elicited by electrical stimulation of the mesencephalic trigeminal nucleus showed no significant change in association with the altered respiratory mode. Our findings suggest that masseteric electromyographic activity is inhibited during oral respiration and that the γ-system is involved in this inhibition.

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