The Effect of Tensor Veli Palatini Stimulation on Upper Airway Patency

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Objective: To evaluate the effect of selective electrical stimulation of the tensor veli palatini muscle on upper airway patency.

Methods: Pressure-flow relationships were evaluated, in a feline isolated upper airway preparation, to determine the role of the soft palate musculature on airflow dynamics. The tensor veli palatini muscles were selectively stimulated while monitoring upper airway collapsibility (critical pressure), maximal inspiratory airflow, and the nasal resistance upstream to the flow-limiting site.

Results: Tensor veli palatini stimulation resulted (mean ± SEM) in an increase in maximal inspiratory airflow from 74 ± 13 mL/s to 93 ± 18 mL/s (P = .04). The increase in maximal inspiratory airflow was associated with a decrease in critical pressure from −2.3 ± 1.7 cm H2O to −4.7 ± 2.7 cm H2O (P = .01) and an increase in nasal resistance from 32.4 ± 24.3 cm H2O · L−1 · s−1 to 50.8 ± 29.7 cm H2O · L−1 · s−1 (P = .02).

Conclusions: Tensor veli palatini stimulation decreases upper airway collapsibility and is likely an integral component in maintaining airway patency. However, the effects of the isolated tensor veli palatini muscles are less significant than those seen previously with physiologic stimuli such as hypercapnia. These findings suggest that upper airway patency, although contributed to by the tensor veli palatini, requires the coordinated activation of palatopharyngeal muscles to adequately influence upper airway collapsibility.

METHODS

Institutional guidelines regarding animal experimentation were followed, and all experiments were approved by the Animal Use Committee of the Johns Hopkins University School of Medicine, Baltimore, Md. The study was performed in 5 supine male cats, weighing 2 to 2.5 kg, premedicated with an intramuscular injection of xylazine hydrochloride (3 mg/kg) and anesthetized with ketamine hydrochloride (50 mg/kg). Blood pressure was monitored through a femoral arterial line and maintained with isotonic sodium chloride solution given through a femoral vein. The cranial was exposed, and midcollicular decerebration was performed.

A feline isolated upper airway preparation was used as previously described. To prepare the isolated upper airway, the cervical trachea was transected and the distal end was cannulated with an endotracheal tube (5 mm inner diameter), through which the animal breathed spontaneously. A rigid cannula (3 mm inner diameter, 2 cm in length) was inserted into the proximal trachea and secured at the level of the aryepiglottic folds. To monitor respiration, an esophageal balloon was inserted into the lower esophagus. During the collection of data, the tongue was allowed to prolapse into the mouth and the lips were sutured shut with a purse-string suture. The cat’s head was fixed in place at an angle of approximately 50° to 60° from the horizontal plane. The epiglottis was left undisturbed in its normal anatomic position.

A surgical technique was devised to isolate the tensor palatini muscles using an approach through a midline cervical incision. Care was taken to avoid injury to the nerves, blood vessels, and mucosa. Dissection was carried out superiorly, anterolaterally to the carotid sheath, to the level of the skull base where the pterygoid hamulus was identified by palpation. The tensor veli palatini muscle was identified as it turned over the hook of the hamulus and spread into an aponeurosis for insertion at the midline raphe. The vertical portion of the tensor veli palatini was isolated by blunt dissection circumferentially around the muscle, between the scaphoid fossa and pterygoid hamulus (Figure 1). Latex finger cots were placed under the tensor veli palatini muscles to isolate the muscles and to ensure selective electrical stimulation of the tensor veli palatini muscles.

To electrically stimulate the tensor veli palatini muscles, Teflon-coated fine-wire needle electrodes were placed into the bellies of the 2 tensor veli palatini muscles and connected to a constant current electrical stimulator (Transcutaneous Electrical Nerve Stimulator; Medtronic, Inc, Minneapolis, Minn). The tensor veli palatini muscles were stimulated at 50 Hz with a supramaximal voltage and a pulse duration of 40 microseconds. Selective stimulation of the tensor veli palatini muscles was confirmed by visual inspection. The muscle bellies were noted to visibly contract and a marked tensing of the soft palate was observed with stimulation.

A vacuum source was applied to the proximal trachea to generate inspiratory flow through the isolated upper airway. Pressure-flow recordings were made during the inspiratory phase of the respiratory cycle of the spontaneously breathing cats, as previously described, to determine the V_{Imax} as well as P_{crit} and R_{n}. The tensor veli palatini muscles were then electrically stimulated just prior to an inspiratory effort, and the pressure-flow recordings were repeated during maximal stimulation. This series of measurements was repeated 5 to 6 times for each condition in each of the 5 decerebrated cats. The measurements were reproducible for each condition in each cat.

Data are presented as means ± SEMs for each measurement at each condition. A 1-way analysis of variance (Minitab Inc, State College, Penn) was used to compare the effects of stimulation with baseline. A significance level of P<.05 was used for all comparisons.

RESULTS

Figure 2 presents the results of the bilateral tensor veli palatini muscle stimulation for the 5 cats. The results are presented as V_{Imax} (top panel), P_{crit} (middle panel), and R_{n} (bottom panel) plotted before and after electrical stimulation. With stimulation of the bilateral tensor veli palatini muscles, V_{Imax}, the maximal inspiratory inflow, increased from 74 ± 13 mL/s to 93 ± 18 mL/s (P = .04). The increase in V_{Imax} was associated with a decrease in P_{crit} from −2.3 ± 1.7 cm H_{2}O to −4.7 ± 2.7 cm H_{2}O (P = .01). The upstream nasal resistance, R_{n}, increased from 32.4 ± 24.3 cm H_{2}O · L^{-1} · s^{-1} to 50.8 ± 29.7 cm H_{2}O · L^{-1} · s^{-1} (P = .02).
In recent years, uvulopalatopharyngoplasty has become the most commonly performed surgical therapy for obstructive sleep apnea. Unfortunately, there remains a 50% failure rate for the surgery, with few selection criteria identified to predict a positive outcome. Consequently, a great deal of research has been focused on the palate, and particularly the tensor veli palatini muscle, to help understand its role in the pathogenesis of obstructive sleep apnea. In dog models, the tensor veli palatini has been shown to have inspiratory phasic activity that is augmented by hypercapnia, hypoxemia, and negative upper airway pressure. In humans, investigators have shown the tonic activity of the tensor veli palatini decreases with the onset of sleep, and the augmented tensor veli palatini activity seen with negative upper airway pressure is attenuated during sleep. Thus, it has been postulated that a loss of tensor veli palatini activity may lead to upper airway collapse in patients with obstructive sleep apnea. Nevertheless, the influences of changes in tensor veli palatini muscle activity on airflow dynamics are not well understood.

We have used a feline isolated upper airway model to examine the neuromuscular mechanisms modulating maximal airflow through the upper airway. This model reproduces the condition of pharyngeal airflow obstruction and inspiratory flow limitation that characterizes human sleep-disordered breathing. The model has further allowed us to determine the effect of individual muscles on V$_{\text{Imax}}$, and whether changes observed in V$_{\text{Imax}}$ were due to changes in pharyngeal collapsibility (P$_{\text{crit}}$) or resistance (R$_n$) upstream to the site of obstruction. In the present study, we used this model to investigate the role played by the tensor veli palatini muscles in modulating the severity of upper airway obstruction. Our results indicate that bilateral stimulation of the tensor veli palatini muscles leads to an increase in V$_{\text{Imax}}$. The increase in V$_{\text{Imax}}$ is associated with a decrease in P$_{\text{crit}}$, reflecting a decrease in palatal collapsibility at the site of airflow obstruction, and a concomitant increase in R$_n$. We conclude that tensor palatini stimulation increased V$_{\text{Imax}}$ by decreasing collapsibility, but the increase in V$_{\text{Imax}}$ was offset somewhat by a concomitant increase in the resistance upstream to the site of airflow collapse.

One advantage of our experimental approach was that methods were developed to stimulate the tensor veli palatini muscles selectively. This allowed us to determine the influence of the tensor veli palatini muscles on pharyngeal airflow dynamics without altering the activity of the other upper airway muscles. The tensor veli palatini muscles neither elevate nor depress the soft palate. Rather, their mechanism of action tenses the palate in a lateral direction. Accordingly, we can attribute observed decreases in P$_{\text{crit}}$ to increased tension within the soft palate itself. This finding is consistent with previous observations suggesting that P$_{\text{crit}}$ is influenced by changes in tension in the pharyngeal wall. In fact, earlier studies suggest such an increase in pharyngeal wall tension is associated with an increase in R$_n$. Thus, the increase in R$_n$ that we observed in this study suggests that significant dilatation of the upper airway does not occur with tensor veli palatini muscle stimulation. Therefore, we believe stimulation of the tensor palatini muscles decreases P$_{\text{crit}}$ by increasing airway wall tension rather than by dilating the airway.

Although we found that V$_{\text{Imax}}$ and P$_{\text{crit}}$ changed significantly with tensor veli palatini muscle stimulation, we believe these changes were modest compared with those responses observed with previous experiments in this model. For example, we have shown hypercapnia produces large changes in both V$_{\text{Imax}}$ and P$_{\text{crit}}$, presumably because of its more generalized stimulation of upper airway neuromuscular activity. With the cat breathing a hypercapnic air mixture, a 4.5-cm H$_2$O decrease in P$_{\text{crit}}$ was observed, whereas with tensor veli palatini muscle stimulation, the mean decrease in P$_{\text{crit}}$ was 2.4 cm H$_2$O. Similarly, selective stimulation of the genioglossus muscle produced even larger decreases in P$_{\text{crit}}$, with an average decrease of 12.0 cm H$_2$O, suggesting that other muscles may play a greater role in the control of pharyngeal collapsibility. It appears, there-
fore, that activation of several muscles is responsible for the changes in Pcrit seen during hypercapnia. This is not surprising given the anatomic complexity of the upper airway. Moreover, we and others have previously demonstrated that length-tension relationships of specific upper airway muscles may play a role in modulating pharyngeal collapsibility. Therefore, we suggest that the coordinated activation of the palatopharyngeal muscles helps achieve an airway configuration that maximally increases upper airway tension and decreases pharyngeal collapsibility.

In summary, selective bilateral electrical stimulation of the tensor veli palatini muscles leads to a decrease in collapsibility in an isolated upper airway feline model. We speculate the decreased collapsibility is related to increased tension of the palate when the tensor veli palatini muscles are electrically stimulated. Selective stimulation of the tensor veli palatini muscles, however, results in airflow dynamics changes that are only modest by comparison to those produced by more generalized neuromuscular stimulation. This suggests that other upper airway muscles are also involved in the modulation of upper airway collapsibility. These data also suggest other sites in the airway may be the locus of airway collapse, and the effect of the palatal tensing may be less than that required for therapeutic effect in the treatment of obstructive sleep apnea. This work might be applied to uvulopalatopharyngoplasty failure. From this, one might infer that palatal stiffening does not completely alleviate flow obstruction. Additional work should be conducted to determine if stimulation of the tensor veli palatini muscles would be a predictor of good outcome following uvulopalatopharyngoplasty.

Accepted for publication March 11, 1999.

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REFERENCES