Respiratory function of velopharyngeal constrictor muscles during wakefulness in normal adults

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Launois, Sandrine H., Judy Tsui, and J. Woodrow Weiss. Respiratory function of velopharyngeal constrictor muscles during wakefulness in normal adults. J. Appl. Physiol. 82(2): 584–591, 1997.—The levator veli palatini (LVP) and the superior pharyngeal constrictor (SPC) influence velopharyngeal patency and soft palate position, but their behavior during respiration is incompletely characterized. To further clarify their respiratory function, we recorded electromyographic activity (EMG) in the LVP and the SPC in awake normal subjects breathing orally. EMG data were obtained in six subjects for the LVP and in nine subjects for the SPC. EMG activity and timing and ventilation were measured during isocapnic hypoxia and hyperoxic hypercapnia. Phasic EMG activity was inconsistently present during unstimulated oral breathing. Timing of EMG phasic activity was variable for both muscles. Peak LVP activity was mainly or exclusively expiratory in three of six subjects, Peak SPC activity was mainly or exclusively inspiratory in five of nine subjects. With chemostimulation, recruitment of phasic activity was observed in the LVP in four of six subjects and in the SPC in five of nine subjects. Tonic activity increased in four of six subjects for the LVP and in three of nine subjects for the SPC. However, the response was alinear, and intersubject as well as breath-to-breath variability was substantial. In conclusion, LVP and SPC are characterized by the high inter- and intrasubject variability of EMG activity, timing of activation, and response to chemostimulation.

METHODS

Subjects

Fourteen normal volunteers were enrolled in the study. In 10 subjects, electrode position was confirmed physiologically, and data from these subjects are reported below. EMG activity was recorded in five men and five women (mean age 28.3 ± 5.7 yr, mean body mass index 22.2 ± 2.1 kg/m²) (Table 1). None had a history of respiratory disease, snoring, or sleep disorder, and none had undergone tonsillectomy. Subjects were not taking any medication and had no symptoms of upper airway infection at the time of the study. All but one (subject 4) were naive subjects. The study was approved by the Committee on Clinical Investigations of the Beth Israel Hospital, and written informed consent was obtained from the subjects before the study.

Measurements

Respiration. Subjects were connected to a bag in a box via a unidirectional breathing circuit. Route of breathing was fixed to oral breathing through a mouthpiece connected to the breathing circuit. Nose clips were used to prevent nasal airflow. Airflow was measured with a wedge spirometer (Med Science 500), and the flow signal was integrated to obtain volume (R815A Respiratory integrator, Hewlett-Packard). Arterial oxygen saturation (SaO2) was monitored with a pulse oximeter (Ohmeda 3700) by using a finger probe. End-tidal PCO2 (PETCO2) was measured at the mouth by a mass spectrometer (MGA-1100, Perkin-Elmer Medical Instruments).

EMG. LVP and SPC EMGs were recorded from bipolar fine-wire electrodes (Teflon-coated stainless steel wire, 0.08
mm, Medwire). A 4% lidocaine spray was applied to the oropharynx and the oral side of the soft palate. LVP electrodes were inserted by using a 1.5-in. 24-gauge sterile needle bent at the distal third to a 30° angle and attached to an empty 12-ml syringe. While the subject sustained the sound “aah,” the needle tip was inserted laterocranioposteriorly ~10 mm in the “levator dimple” (8). SPC electrodes were inserted by using a 0.75-in. 23-gauge sterile needle bent at midpoint to a 90° angle and attached to an empty 12-ml syringe. The needle was inserted ~2 mm under the mucosal surface of the posterior pharyngeal wall, midway between the level of the soft palate margin and the level of the tongue base (9). After removal of the placement needles, wires were taped securely to the face and attached to copper spring clips soldered to the free ends of amplifier lead-in wires. A grounding electrode was placed on the forehead. EMG signals were preamplified and band-passed filtered between 30 and 1,000 Hz with battery-powered differential amplifiers (Grass P15D) and further amplified (Tektronix TM 504) before being full-wave rectified and electronically integrated with a leak time constant of 150 ms. Once in place, the electrodes did not cause any discomfort.

Experimental Protocol

Subjects were studied on a single occasion while they were awake. They were asked to refrain from caffeinated beverages 12 h before the study. After EMG electrode placement, subjects were seated in a specially designed chair that fixes body and head position. Correct electrode position was ascertained by observing bursts of EMG activity while the subject swallowed and produced a sustained “aah.” Furthermore, several bursts of activity were observed during the production of two test sentences: “I do not think so” and “The book belongs to my father” (7). The experimental protocol started at least 30 min after application of topical anesthesia. EMG responses to hyperoxic hypercapnia and to isocapnic hypoxia were assessed (21, 22). Two consecutive trials were performed for each chemostimulation, separated by 10-min rest periods. Electrical zero and system zero (defined by shorting the amplifier inputs) were verified immediately before and after each trial. Before the study was terminated and the electrodes were removed, the soft palate and oropharynx were inspected to confirm that all wires had remained in place.
had reached 48 Torr \((P = 0.0001)\). LVP tonic activity increased significantly with \(\text{PETCO}_2\) in subjects 1 and 10 \((P = 0.0001)\) and remained unchanged in subjects 2, 3, and 8. In subject 5, tonic activity increased between 45 and 54 Torr, decreased sharply when phasic activity started to decline, and remained stable until the end of the challenge. Phasic LVP EMG activity increased with hypoxia in subjects 3, 8, and 10 \((P \leq 0.02)\) and decreased in subjects 1 and 5 \((P < 0.001)\) (Fig. 2). In subject 2, progressive hypoxia was accompanied by an increase in phasic LVP activity during the first trial and a decrease in activity during the second trial. LVP tonic activity increased significantly with progressive hypoxia in subjects 3, 8, and 10 and decreased in subjects 1 and 5. In subject 2, LVP tonic activity followed the same pattern as the phasic activity: it increased with hypoxia during the first trial and decreased during the second trial (Fig. 3).

### SPC Response to Chemical Stimulation

SPC phasic activity increased or appeared with increasing \(\text{PETCO}_2\) in five of nine subjects (subjects 1, 6, and 8–10) (Fig. 4). In subjects 2, 4, and 7, SPC phasic activity remained unchanged with progressive hypercapnia. In subject 5, phasic activity progressively increased until \(\text{PETCO}_2\) reached 52–54 Torr; it then decreased sharply and started to rise again at the end of the trial. SPC tonic EMG activity increased in response to progressive hypercapnia in subjects 6, 8, and 10. It decreased significantly in subject 2 \((P = 0.0001)\) and remained unchanged in the remaining five subjects (subjects 1, 4, 5, 7, and 9) (Fig. 5). In subjects 6–8 and 10, phasic SPC activity significantly increased with hypoxia \((P \leq 0.003)\). In subject 5, progressive hypoxia was accompanied by a significant decrease in phasic SPC EMG \((P = 0.0001)\). In the remaining three subjects, hypoxia did not significantly affect SPC phasic activity. With hypoxia, SPC tonic activity increased in subjects 6, 8, and 9. Progressive hypoxia was accompanied by a decrease in SPC tonic activity in subject 2. In subject 5, tonic activity was stable from 100 to 94%, dropped abruptly at \(\sim 94\%\), and then remained stable.

### Table 2. Phase pattern of EMG peak activity: effect of chemostimulation

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
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<td>20</td>
<td>6</td>
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<td>0</td>
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<tr>
<td>1</td>
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<td>100</td>
<td>86</td>
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<td>100</td>
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<td>38</td>
</tr>
<tr>
<td>Superior pharyngeal constrictor</td>
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<td>NA</td>
</tr>
<tr>
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</table>

A, normal \(\text{PETCO}_2\), normal \(\text{SaO}_2\); B, mild hypercapnia, normal \(\text{SaO}_2\); C, moderate hypercapnia, normal \(\text{SaO}_2\); D, normal \(\text{PETCO}_2\), mild hypoxia; E, normal \(\text{PETCO}_2\), moderate hypoxia; NA, no phasic or biphasic EMG activity.

### Table 3. Phase pattern of EMG peak activity: overall trend

<table>
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<th>Subject No.</th>
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<td>65.2</td>
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<td>10</td>
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<td>77.2</td>
</tr>
</tbody>
</table>

SPC, superior pharyngeal constrictor; LVP, levator veli palatini.

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### Timing of EMG Phasic Activity

LVP peak activity occurred mainly or exclusively during inspiration in subject 1 and during expiration in subject 2. SPC peak activity occurred mainly or exclusively during inspiration in subject 8 and during expiration in subjects 6 and 10. In all other cases, phase-pattern timing varied with the sample considered. However, we did not observe any trend that could suggest that the nature or intensity of the chemostimulation explained the variability in timing (Table 2). When the five samples were averaged, LVP peak activation was mainly inspiratory in one-half of the subjects, and SPC peak activation was mainly expiratory in five of nine subjects (Table 3).

In all subjects, and for both muscles, a biphasic discharge pattern was observed occasionally for a single breath. In subject 8, a consistent biphasic pattern was noted in the LVP during hypoxia but not during hypercapnia. In subject 3, LVP was phasically active during expiration and inspiration during the first hypoxic challenge. With progressive hypoxia, inspiratory activity increased, whereas expiratory bursts of EMG activity disappeared (Fig. 1).

### LVP Response to Chemical Stimulation

Subjects 1–3 and 10 showed a significant increase in LVP phasic activity as \(\text{PETCO}_2\) rose \((P < 0.01)\). In subject 5, phasic activity was present in the LVP at the beginning of the trials and progressively disappeared as \(\text{PETCO}_2\) increased. In subject 8, LVP phasic activity increased significantly with hypercapnia once \(\text{PETCO}_2\)
at this lower level until the end of the hypoxic challenge. In subjects 4, 7, and 10, SPC tonic activity did not change notably with hypoxia.

**DISCUSSION**

In these normal awake subjects, we found that, while seldom present during quiet oral breathing, phasic activation of the LVP and SPC appeared during progressive isocapnic hypoxia and hyperoxic hypercapnia. However, in our subjects, respiratory activity in these muscles was characterized by marked inter- and intra-subject variability. EMG responses to chemostimulation were brisk and linear in some individuals and weak or a linear in others. In some subjects, phasic and tonic activity disappeared despite progressive hypoxia and hypercapnia. Some subjects responded to one stimulus but not to the other. Variability was also observed in the phase pattern: most subjects displayed activity during both inspiration and expiration.

Although nonneuromuscular factors influence pharyngeal cross-sectional area, palatal dilator muscle activity is considered to be the main determinant of velopharyngeal patency throughout the respiratory cycle (16). Those dilator muscles are thought to counteract intraluminal subatmospheric pressure by producing phasic inspiratory activity, although the role of tonic activity has recently been stressed (26). Constrictor velopharyngeal muscles have received little attention. However, there is evidence to suggest that their participation in velopharyngeal mechanics should be carefully examined. Velopharyngeal constrictor muscles consist of the LVP and the SPC. Activation of the LVP moves the soft palate rostrally and posteriorly, allowing velopharyngeal closure during swallowing and phonation (7). SPC contraction narrows the velopharynx, and its palatal insertion pulls the soft palate posteriorly (7). Phasic activity has been demonstrated in the SPC during quiet breathing during wakefulness in a previous study (9). LVP respiratory-related activity has also been recorded in normal subjects during quiet breathing (3, 18, 28). Our results confirm the presence of respiratory activity in both muscles. In most previous studies, LVP and SPC respiratory activity appeared to be expiratory (3, 8, 23). However, recent preliminary reports suggest that timing of phasic activity is more variable than previously reported (25, 27). In the

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**Fig. 1.** Levator veli palatini (LVP) EMG activity during isocapnic hypoxia [end-tidal PCO₂ (PETCO₂) = 37 Torr; arterial O₂ saturation (Sao₂) = 78 - 77%] in subject 3. This segment illustrates variability of EMG phasic activity timing with respect to respiratory cycle. Downward deflexion in flow signal indicates inspiration (Insp). Exp, expiration.

**Fig. 2.** Individual LVP phasic EMG responses to isocapnic hypoxia. EMG activity is expressed as %maximum respiratory activity. Data from 2 trials are combined.
present study, phasic activity in both muscles was inspiratory, expiratory, biphasic, or variable. LVP and SPC activation are likely to affect velopharyngeal geometry or mechanics at any point during the respiratory cycle. As the main palatal muscle, LVP regulates route of breathing by modifying soft palate position (3, 7, 27). During oral breathing, inspiratory contraction of the LVP could represent active facilitation of oral airflow. Alternatively, the LVP could be activated during inspiration in response to caudal tug of the soft palate. With increasing respiratory efforts during chemical stimulation, EMG activity would increase to counteract passive stretching. If biomechanical action of the SPC is considered alone, the benefit of inspiratory contraction is not apparent. Data obtained with various preparations suggest, however, that the net mechanical effect of a muscle contraction strongly depends on the activity of other upper airway muscles (1, 5). Synergistic action of the SPC with the LVP, palatoglossus, or palatopharyngeus could, therefore, also tend to decrease resistance to oral airflow. In addition to inspiratory activity, our subjects also demonstrated expiratory activation of the

Fig. 3. Individual LVP tonic EMG responses to isocapnic hypoxia. EMG activity expression and trial data are as described in Fig. 2.

Fig. 4. Individual superior pharyngeal constrictor (SPC) phasic EMG responses to hyperoxic hypercapnia. EMG activity expression and trial data are as described in Fig. 2.
LVP and SPC. Expiratory airflow is, in part, regulated by laryngeal and hypopharyngeal resistance, determined by the combination of abductor muscle relaxation and adductor muscle contraction (5, 15). Further modulation of expiratory flow could be achieved by activation of pharyngeal constrictors. Last, activation of constrictor muscles during expiration may affect velopharyngeal air space by altering the length of dilator muscles and modifying their mechanical action. Contraction of these muscles during expiration decreases the velopharyngeal area, thus affecting inspiratory dynamics. Indeed, it has recently been demonstrated that during wakefulness, minimal airway size is seen at the end of expiration in normal subjects and sleep apnea patients (24). Even in the absence of phasic activation, these muscles could affect the size of the upper airway by the level of their tonic activity. However, in the absence of a biomechanical model of upper airway muscles, the effect of constrictor muscle activation on inspiratory mechanics cannot be predicted.

The variability exhibited by LVP and SPC respiratory activity (variable levels of activation during quiet breathing, amplitude and direction of the response to chemostimulation, timing with respect to the respiratory cycle) raises concerns about the validity of our recordings. We believe, however, that technical difficulties are not likely to account for this variability. Electrodes were inserted following a standard method (8, 9), and preparatory cadaver dissections confirmed that our technique was adequate. Furthermore, the presence of EMG activity during voluntary activation ensured that the wire electrodes were in place. Correct placement of the electrodes was also verified visually at the end of the study, although speech maneuvers were not repeated. We cannot, therefore, exclude the possibility that a wire may have been partially dislodged in some patients. Electrical activity from contiguous muscles is unlikely to explain our findings. At the retropalatal level, the SPC is the sole muscle of the posterior pharyngeal wall. To record from the LVP, electrodes are inserted in the levator dimples. In this area of the velum, fibers from the TVP are tendinous and other palatal muscles insert more laterally (palatoglossus, palatopharyngeus) or more posteriorly (musculus uvulae) than the LVP. We are, therefore, confident that electrodes were properly inserted and that the variability of our data reflects variability in LVP and SPC activity from subject to subject.

Although it has not been reported for the alae nasi, the tensor palatini, or the genioglossus, intersubject variability in EMG activity has been observed in an animal preparation for the geniohyoid (30) and in normal human subjects for the thyroarytenoid (15), the arytenoid (14), and, to a lesser degree, for the cricothyroid (31). Like these muscles, the SPC and the LVP, as highly specialized muscles active in numerous nonrespiratory functions, are under strong behavioral control. It is, however, unexpected to find such a degree of intersubject variability during chemostimulation, when low behavioral influences would be anticipated. In the medulla, pharyngeal motoneurons are located in an area overlapping respiratory centers (4), and respiratory influences on the pharyngeal motoneurons are suggested by respiratory-related electrical activity recorded in pharyngeal muscles. However, suprapontine influences are likely to be predominant in the regulation of these motoneurons, which are involved in voluntary activities such as speech produc-

Fig. 5. Individual SPC tonic EMG responses to hyperoxic hypercapnia. EMG activity expression and trial data are as described in Fig. 2.
tion or swallowing. Discomfort due to the mouthpiece, the noseclips, or hyperventilation may have resulted in behavioral control overriding automatic control of palatal musculature, allowing some subjects to modify the position of their palate. Indeed, during a different experimental protocol, we have observed the effect of discomfort caused by a thin nasopharyngoscope on the LVPEMG activity in subject 10: while he was complaining of discomfort, tonic activity was near maximum value; after adjustment of the scope and relief of discomfort, tonic activity decreased sharply by 75% (unpublished observations). Such abrupt changes in tonic activity decreased, phasic activity was "unmasked" (Fig. 6). It is, therefore, possible that phasic response to chemostimulation was obscured by high tonic activity in some subjects. Last, tongue and jaw position were not fixed during the course of the experiment. Given the close interactions between pharyngeal muscles, movements of either of these structure are likely to modify the activity of the SPC and LVP (13).

Non-rapid-eye-movement (NREM) sleep provides an opportunity to examine the relative contribution of suprapontine influences and "peripheral" or anatomic factors (jaw or tongue position, for instance) to the variability in muscle activity. There are, however, few data available at the present time. In the only study of SPC activity during sleep, Sauerland et al. (23) observed the persistence of SPC phasic activity in NREM but also noted that this activity could be intermittent in some (but not all) subjects. Tangel et al. (29) reported a consistent decrease in peak LVP activity during NREM sleep, although the amplitude of the decrease was somewhat variable. The issue of possible EMG variability was not addressed in either study, and an experimental protocol aimed at investigating how vigilance affects EMG variability may yield important information.

Variable phase pattern was observed in our subjects for the SPC and the LVP. Similar findings have been reported in animal models and normal adults. In dogs, pharyngeal constrictors are active during expiration but can exhibit inspiratory activity as well (11). In decerebrate artificially ventilated cats, glossopharyngeal motoneurons display inspiratory discharge only, whereas both inspiratory and expiratory bursts are present in the pharyngeal branches of the vagus (4). In normal subjects, during progressive hypercapnia, posterior cricoarytenoid inspiratory activity can be prolonged and persists after the beginning of expiration (2). Previous studies did not show any variability in the relationship of LVP or SPC activity to the respiratory cycle (3, 9, 18, 23, 28). Differences in species, body position, route of breathing, or chemical drive could account for the discrepancy among studies. The significance of shifts in phase pattern of pharyngeal muscles remains unclear.

In conclusion, velopharyngeal constrictors display respiratory activity and respond to chemostimulation. Their electrical activity is remarkably variable, and we suggest that this variability results from strong behavioral influences on pharyngeal motoneurons supplying these muscles. Additional studies of velopharyngeal muscle activity during wakefulness and sleep may provide further evidence of the role of these suprapontine influences.

At this stage, we can only speculate on how the variability that we observed in EMG activity may affect velopharyngeal function. This variability in velopharyngeal musculature activity is likely to contribute to the absence of a consistent relationship between palatal muscle activity and velopharyngeal patency that our laboratory has reported recently in normal awake adults (17). If one assumes that velopharyngeal muscles determine velopharyngeal patency, variable EMG activation would require multiple interaction patterns between local and regional muscles to maintain velopharyngeal patency while behavioral and anatomical factors fluctuate.

Last, the findings of this study should encourage future investigators to take variable EMG activity into account when investigating palatal muscle contribution to transpalatal resistance and velopharyngeal narrowing during sleep.

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