Influences of head positions and bite opening on collapsibility of the passive pharynx

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Influences of head positions and bite opening on collapsibility of the passive pharynx. J Appl Physiol 97: 339–346, 2004. First published March 12, 2004; 10.1152/japplphysiol.00907.2003.—A collapsible tube surrounded by soft material within a rigid box was proposed as a two-dimensional mechanical model for the pharyngeal airway. This model predicts that changes in the box size (pharyngeal bony enclosure size anatomically defined as cross-sectional area bounded by the inside edge of bony structures such as the mandible, maxilla, and spine, and being perpendicular to the airway) influence patency of the tube. We examined whether changes in the bony enclosure size either with head positioning or bite opening influence collapsibility of the pharyngeal airway. Static mechanical properties of the passive pharynx were evaluated in anesthetized, paralyzed patients with sleep-disordered breathing based on M-mode ultrasonography. The box size was defined as the number of oxygen desaturation exceeding 4% from baseline. Severe sleep apnea was determined by oximetry data and anthropometric characteristics. The box size varied with head and mandibular positioning changes, which may be an influential factor of the pharyngeal airway patency. Cervical flexion with bite closure (neck flexion) decreases it. Bite opening with neutral neck position decreases the distance between the mentum and cervical column, which consequently increases the bony enclosure size, whereas cervical flexion with bite closure (neck flexion) decreases it. Bony opening increases the bony enclosure size without head positioning change. Accordingly, the purpose of this study was to evaluate influences of head positions and bite opening on static mechanical properties of the passive pharynx in anesthetized and paralyzed patients with SDB.

MATERIALS AND METHODS

Subjects and overnight oximetry. The study consisted of 24 male patients with SDB who were interested in undergoing uvulopalatopharyngoplasty and were scheduled to undergo endoscopic pharyngeal assessment to determine their indications for this procedure (4). All had histories of excessive daytime sleepiness, habitual snoring, and witnessed repetitive apnea. SDB was evaluated by a pulse oximeter (Pulsox-5; Minolta, Tokyo, Japan). All subjects were instructed to attach an oximetry finger probe before sleep and to remove the probe on awakening. Digital readings of arterial oxygen saturation (SaO₂) and pulse rate were stored every 5 s in a memory card. The stored data were displayed on a computer screen to check the quality of the recordings. The computer calculated oxygen desaturation index, defined as the number of oxygen desaturation exceeding 4% from the baseline, and the percent of time spent at SaO₂ <90%. Table 1 lists all nocturnal oximetry data and anthropometric characteristics. Although the oximetry evaluation alone does not clarify the nature of SDB, we believe that all patients can be safely diagnosed as having obstructive sleep apnea (OSA) on the basis of the oximetry results and the clinical symptoms (1).

Informed consent was obtained from all subjects after the aim and potential risks of the study were fully explained to each. The investigation was approved by the hospital ethics committee of our institution.

Preparation of the subjects. Each subject was initially premedicated with 0.5 mg of atropine and placed in the supine position on an operating table, where a modified tight-fitting nasal mask was attached. Care was taken to prevent air leaks from the mask, particularly by the inside edge of the bony structures and being perpendicular to the airway, corresponds to the box size of the mechanical model. The contribution of obesity and craniofacial anomaly such as a small maxilla and mandible to the increased collapsibility of the passive pharynx was well explained by the mechanical model (24).

Within one subject, the pharyngeal bony enclosure size varies with head and mandible positioning changes, which may be an influential factor of the pharyngeal airway patency. Cervical extension with bite closure (neck extension) increases the distance between the mentum and cervical column, which consequently decreases the bony enclosure size, whereas cervical flexion with bite closure (neck flexion) decreases it. Bite opening with neutral neck position decreases the distance between the mentum and cervical column, which consequently increases the bony enclosure size without head positioning change. Accordingly, the purpose of this study was to evaluate influences of head positions and bite opening on static mechanical properties of the passive pharynx in anesthetized and paralyzed patients with SDB.

A PATENT PHARYNGEAL AIRWAY IS CRUCIAL FOR STABLE BREATHING. Pharyngeal airway size is regulated by a precise interaction between neural regulation of pharyngeal airway dilator muscle activities (neural mechanisms) and structural properties of the pharyngeal airway (anatomical mechanisms) (7). Through elimination of the neural mechanisms by administration of a muscle blockade under general anesthesia, we demonstrated that closing pressures of the passive pharynx were distinctively higher in patients with sleep-disordered breathing (SDB) than in normal subjects (3). Furthermore, specific structural abnormalities, such as obesity and craniofacial anomaly, contributed to increased collapsibility of the passive pharynx (24).

Structurally, the pharyngeal airway is surrounded by soft tissue such as the tongue, which is enclosed by bony structures such as the mandible and cervical vertebrae (Fig. 1). Consequently, a collapsible tube surrounded by soft material within a rigid box was proposed as a two-dimensional mechanical model for the pharyngeal airway (24). Pharyngeal bony enclosure size, anatomically defined as cross-sectional area bounded

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when the airway was pressurized above 20 cmH2O. General anesthesia was induced and maintained by intravenous infusion of propofol, and intravenous injection of a muscle relaxant (vecuronium 0.2 mg/kg) produced complete paralysis throughout the experiment while the subject was ventilated with positive pressure through an anesthetic machine. SaO2, electrocardiogram, and blood pressure were continuously monitored. The tip of a slim endoscope (FB10X, Pentax, Tokyo, Japan, 3 mm OD) was inserted through the modified nasal mask and the naris down to the upper airway to visualize the velopharynx (retropalatal airway), and the oropharynx (retroglossal airway). A closed-circuit camera (ETV8, Nisco, Saitama, Japan) was connected to the endoscope, and the pharyngeal images were recorded on a videotape. Reading of airway pressure (Paw), measured by a water manometer, was simultaneously recorded on videotape.

**Experimental procedures.** To determine the pressure-area relationship of the pharynx, after disconnection from the anesthetic machine the nasal mask was connected to a pressure-control system capable of accurately manipulating Paw from -20 to 20 cmH2O in a stepwise fashion. At cessation of mechanical ventilation of the subject under complete muscle paralysis, apnea resulted. Paw was immediately increased up to 20 cmH2O to dilate the airway and then gradually reduced, within a 2- to 3-min span, from 20 cmH2O to the closing pressure of the retropalatal airway in a stepwise fashion. The latter represented the pressure at which complete closure of the retropalatal airway occurred, as evident on the video screen. The apneic test was terminated when SaO2 fell below 95%. This procedure of experimentally induced apnea allowed construction of a pressure-area relationship of the visualized pharyngeal segment. The subject was manually ventilated for at least 1 min before and after the apneic test. Distance between the tip of the endoscope and the narrowing site was measured with a wire passed through the aspiration channel of the endoscope.

Each patient participated in either the head position study (n = 13) or the bite opening study (n = 11) (Fig. 2). In the head position study, in addition to the control measurement (head and neck in neutral position with bite closed by a chin strap), the apneic tests were repeated during both neck extension (neck maximally extended by placing cushions under the shoulders with bite closed by a chin strap) and neck flexion (neck maximally flexed by placing cushions under the head with bite closed by a chin strap) in seven patients, whereas the tests were performed either during only neck extension (n = 4) or neck flexion (n = 2) in the remaining six patients. The control measurement was performed once per patient. In the bite opening study, the bite was widened by inserting a mouthpiece between the upper and lower incisors in head and neck in the neutral position, producing a 15-mm distance between the incisors (Fig. 2). The apneic test was initiated immediately after establishment of each experimental condition. On the completion of the experiment time span of 30–60 min, atropine (0.02 mg/kg) and neostigmine (0.04 mg/kg) were administered to reverse muscle paralysis.

**Data analysis.** To convert the monitor image to an absolute value of the pharyngeal cross-sectional area, magnification of the imaging system was estimated at 1.0-mm interval distances between the endoscopic tip and the object in the range of 5–30 mm. At a defined value of Paw, the image of the pharyngeal lumen was traced and pixels included in the area were counted (SigmaScan version 2.0, Jandel Scientific Software, San Rafael, CA). The pixel number was converted to pharyngeal cross-sectional area according to the distance-magnification relationship. Using known-diameter tubes, we tested the accuracy of the cross-sectional area measurements. For constant distance, the measured areas were systematically deviated from actual areas (Fig. 3); the largest known area tested

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**Table 1. Anthropometric characteristics and results of nocturnal oximetry**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Median (25–75 percentiles)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>47.0 (38.0–54.5)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>77.7 (69.0–82.8)</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.72 (1.66–1.77)</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26.4 (23.7–28.4)</td>
</tr>
<tr>
<td>ODI, h⁻¹</td>
<td>27.7 (20.8–49.5)</td>
</tr>
<tr>
<td>CT₉₀, %</td>
<td>12.0 (2.0–23.1)</td>
</tr>
<tr>
<td>Nadir SaO₂, %</td>
<td>87.9 (84.9–90.3)</td>
</tr>
<tr>
<td>Lowest SaO₂, %</td>
<td>72.0 (61.0–80.5)</td>
</tr>
</tbody>
</table>

Values are medians (25–75 percentiles) of all patients (n = 24). BMI, body mass index; ODI, oxygen desaturation (SaO₂) index defined as number of desaturations exceeding >4% per hour; CT₉₀, percent of time spent SaO₂ <90%; Nadir SaO₂, mean of the nadir SaO₂ values in all desaturation events; Lowest SaO₂, a lowest SaO₂ value among the desaturation events.
(0.95 cm²) was underestimated by 11% because of image deformation of the outer image area, and the smallest known area tested (0.03 cm²) was overestimated by 13% because of reduction of the image resolution (5).

The measured luminal cross-sectional area was plotted as a function of Paw. The closing pressure was defined as pressure corresponding to the zero area. At high values of Paw, relatively constant cross-sectional areas were revealed; therefore, maximum area \( A_{\text{max}} \) was determined as the mean value of highest three Paw (18, 19, and 20 cmH₂O). The pressure-area relationship of each pharyngeal segment was fitted by an exponential function, \( A = A_{\text{max}} - B \times \exp(-K \times \text{Paw}) \), where \( B \) and \( K \) are constants. A nonlinear least square technique was used for the curve fitting, and the quality of the fitting was provided by the coefficient \( R^2 \) (SigmaPlot version 2.0, Jandel Scientific Software, San Rafael, CA). A regressive estimate of closing pressure (\( P_{\text{close}} \)), which corresponds to an intercept of the curve on the Paw axis, was calculated from the following equation for each pharyngeal segment: \( P_{\text{close}} = \ln(B/A_{\text{max}})K^{-1} \). The shape of the pressure-area relationship was described by the value of \( K \). When pressure-area relationship is curvilinear, compliance of the pharynx defined as a slope of the curve varies with changes in Paw; therefore, a single value of compliance calculated for a given Paw does not represent collapsibility of the pharynx for the entire Paw ranges. In contrast, \( K \) represents the rate of changes in the slope of the curve; therefore, when \( K \) is high, a small reduction in Paw results in a significant increase in compliance, leading to remarkable reduction in cross-sectional area. Consequently, collapsibility of the pharynx increases with increasing \( K \). We suggest that both \( P_{\text{close}} \) and \( K \) values represent collapsibility of the pharynx, whereby the former determines the position of the exponential curve and the latter characterizes the shape of the curve.

**Statistical analysis.** All values are expressed by median (25–75 percentiles). The Wilcoxon’s signed-rank test was used for comparison between the control and other conditions. Linear regression analysis was performed between observed and estimated closing pressures. \( P < 0.05 \) was considered to be significant.

**RESULTS**

**Effects of neck extension on static pharyngeal mechanics.** Changes in static mechanical variables before and during neck extension are presented in Fig. 4. Neck extension approximately doubled \( A_{\text{max}} \) and significantly decreased both \( K \) and \( P_{\text{close}} \) at the oropharynx. In addition to the significant influ-
ences on the oropharyngeal segment, $P'_{\text{close}}$ also significantly decreased during neck extension. Notably, there were tendencies of increase in $A_{\text{max}}$ ($P = 0.054$) and decrease in $K$ ($P = 0.067$) at the velopharynx, as shown in Fig. 4, although these are not statistically significant. The results indicate that neck extension dilates and stiffens the velopharyngeal and oropharyngeal airway, improving pharyngeal airway patency.

Effects of neck flexion on static pharyngeal mechanics. Changes in static mechanical variables before and during neck flexion are presented in the Fig. 5. Neck flexion significantly decreased $A_{\text{max}}$ and increased $P'_{\text{close}}$ at the oropharynx. Velopharyngeal $P'_{\text{close}}$ also increased during neck flexion. $K$ did not change in response to neck flexion.

Effects of bite opening on static pharyngeal mechanics. Changes in static mechanical variables before and during bite opening are presented in the Fig. 6. Bite opening significantly decreased $A_{\text{max}}$ and increased $P'_{\text{close}}$ at the oropharynx. Velopharyngeal $P'_{\text{close}}$ also increased during bite opening. $K$ did not change in response to bite opening. Notably, the pattern of changes in the pharyngeal mechanics during bite opening is similar to that during neck flexion.

Comparison between observed and estimated closing pressures. Figure 7 demonstrates the correlation between $P'_{\text{close}}$ and observed $P_{\text{close}}$ (the highest airway pressure at which complete closure of either the retropalatal or retroglossal airway was seen on the video screen) at the primary site of closure for all experimental conditions. Most data points are located below the identity line. A linear relationship between the variables ($\text{observed } P_{\text{close}} = -0.56 + 1.0 \times P'_{\text{close}}, R^2 = 0.966$) was obtained form a linear regression analysis. Accordingly, the $P'_{\text{close}}$ is significantly greater than the observed $P_{\text{close}}$ approximately by 0.5 cmH$_2$O on average.

DISCUSSION

Major findings in this study are as follows: 1) neck extension decreased closing pressures of the velopharynx...
and oropharynx and increased maximum oropharyngeal airway size, 2) neck flexion and bite opening increased closing pressures of the velopharynx and oropharynx and decreased maximum oropharyngeal airway size, and 3) oropharyngeal airway compliance decreased during neck extension. These findings support the concept of the mechanical model presented in Fig. 1 to predict upper airway patency and collapsibility of the passive pharynx.

**Design and limitations of the study.** Although many previous studies have reported significant influences of neck positions (9, 10, 13, 16, 17, 25) and mouth opening on pharyngeal airway patency (11), this is the first study that purely evaluates regional structural changes of airway collapsibility by mechanical interventions under the elimination of neural mechanisms. Because techniques in obtaining closing pressures, study population, and amount of structural changes by the interventions differ between previous studies and this study, it is inappropriate to compare the amount of changes in closing pressures between the studies. However, it should be noted that the amount of increase in the closing pressure with bite opening in the active pharynx during sleep (3 cmH2O) (23) is never smaller than that obtained in the passive pharynx (2–2.7 cmH2O), suggesting no recruitment of the pharyngeal dilator muscles for compensation of the structural pharyngeal narrowing with bite opening during sleep. In contrast, the fact that bite opening did not influence upper airway collapsibility during wakefulness (23) strongly suggests the presence of neural compensatory mechanisms for the structural detrimental effects of bite opening during wakefulness.

Because of the systematic error of cross-sectional area measurement in our experimental setting, as shown in Fig. 3, the pressure-area relationship obtained in this study may systematically deviate from a true relationship. A true $A_{\text{max}}$ is considered to be $\sim 10\%$ greater than the measured $A_{\text{max}}$. Because $A_{\text{max}}$ error mainly results from deformation of the endoscopic image at the outer area, statistical $A_{\text{max}}$ differences before and during mechanical interventions may be valid. In contrast, $P_{\text{close}}$ may be minimally influenced by the measurement error due to the nature of the exponential curve. We consider that the significant discrepancy between observed $P_{\text{close}}$ and estimated $P_{\text{close}}$ demonstrated in Fig. 7 is a result of cross-sectional measurement error, but rather of discontinuity of airway pressure changes (1-cmH2O step changes) during the apneic test.

**Mechanical model of the pharyngeal airway.** Assuming that neck extension increases the pharyngeal bony enclosure size, the two-dimensional model presented in Fig. 1 predicts

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Fig. 5. Changes in static mechanical variables in response to neck flexion (NF) at the velopharynx (A) and oropharynx (B). Each line represents a different patient. Lower and upper boundaries of the box indicate 25th and 75th percentages. Solid line within the box marks the median, and vertical lines indicate the 90th and 10th percentages. *$P < 0.05$ vs. control.
reduction of pressure surrounding the collapsible tube (P_tissue) and increase of the airway size for a given pressure inside the collapsible tube (P_lumen). In contrast, neck flexion and bite opening increase P_tissue and decrease the airway size, with the assumption that these interventions decrease the pharyngeal bony enclosure size. In fact, the results of this study confirmed these predictions, although this does not indicate that change in the bony enclosure size is the only predominant mechanism for the observed changes in pharyngeal airway collapsibility during the mechanical interventions.

Reduction of pharyngeal airway compliance during neck extension. One notable finding in this study is reduction of the oropharyngeal K value, i.e., reduction of the oropharyngeal airway wall compliance, during neck extension. This can be interpreted as flattening of the “tube law” curve of the pharyngeal airway in the two-dimensional mechanical model (Fig. 1). However, the mechanisms causing the tube law change are not clearly presented by the model. In anesthetized dogs, van de Graaff (22) previously demonstrated increase in the tracheal tension decreased upper airway resistance. Recently, Thut et al. (21) found significant changes of critical closing pressure during head positioning in association with changes in upper airway length in cats with isolated upper airways. In anesthe-
This is the alternative mechanism. The dilator muscles also need to be included in the model as an additional condition like natural sleep, the role of pharyngeal airway longitudinal forces (Fig. 8). Furthermore, in a nonparalyzed condition like natural sleep, the role of pharyngeal airway dilator muscles also needs to be included in the model as an alternative mechanism.

Fig. 8. Advanced 3-dimensional mechanical model of the pharyngeal airway explaining the results of this study. Neck extension (left) possibly produces increase in box size and displacement of soft tissue into the box in addition to airway lengthening, resulting in decreasing $P_{\text{tissue}}$ and increasing longitudinal force. In contrast, neck flexion and bite opening (right) may produce reduction of box size and displacement of the soft tissue out of the box, resulting in increasing $P_{\text{tissue}}$.

Regional differential effects of mechanical interventions. This is the first study that demonstrates that neck extension, neck flexion, and bite opening influence airway collapsibility at both velopharynx and oropharynx. Because tongue musculature originates from and is enclosed by the mandible, changes in the mandibular position by mechanical interventions should result in displacement of the tongue base toward the same direction; therefore, it is not surprising to find significant influences of mechanical interventions on the oropharyngeal airway patency. In contrast, soft tissue at the level of the velopharynx is not enclosed by the mandible and there is no direct structural connection between the mandible and soft palate, which implies less or no influence of the mandibular position changes on velopharyngeal airway patency. Nevertheless, we found significant influences by mechanical interventions on the velopharyngeal patency. Structurally, the dorsum of the tongue is in close apposition with the anterior wall of the soft palate and is enclosed by the maxilla, suggesting mechanical interaction between the tongue and soft palate as we recently reported (6). Changes in the mandibular position may displace the tongue soft tissue at the level of the velopharynx, resulting in changes in the amount of soft tissue enclosed by the maxilla and therefore the $P_{\text{tissue}}$ at this segment. Soft tissue interaction along the airway during a variety of mechanical interventions needs to be evaluated in future studies.

Clinical implications. Cranio-cervical extension with a forward head posture is reported to be common in severe OSA patients during wakefulness (14, 19). Our results suggest that head posture compensates for increased collapsibility of the pharyngeal airway in these patients. Head position during sleep is mainly determined by the height and design of the pillow. Although use of a higher pillow often flexes the neck, which is not advantageous for airway maintenance, placement of a higher pillow with maintenance of the head straight up (sniffing position) produced higher cervical extension and improvement of pharyngeal airway patency in sedated children (18). Recently, Kushida et al. (8) found significant improvement of SDB in mild OSA patients with use of a pillow that promoted neck extension, whereas the improvement was not evident in severe OSA patients. Their finding agrees with our observation that 3 of 11 patients presented $P_{\text{close}}$ above atmospheric pressure even after neck extension, whereas neck extension significantly decreased $P_{\text{close}}$. Accordingly, neck extension alone may not completely establish patent airway and normalize breathing in severe OSA patients.

Miyamoto et al. (12) reported that the total time spent with mouth opening during sleep was greater in OSA patients than in normal subjects, in accordance with previous observations by Hollowell and Suratt (2). They further reported that bite opening progressively increased during the apneic period and decreased at termination of apnea (12). Our results suggest that bite opening partly contributes to the occurrence and persistence of obstructive apnea; moreover, bite closure partly contributes to reestablishment of the patent airway. Furthermore, bite closure with the use of oral appliances can contribute to efficacy of oral appliances for treatment of SDB, although a recent clinical investigation did not support this (15).

We conclude that head positions and bite opening influence collapsibility of the passive pharynx in patients with SDB. The
observations are well explained by a mechanical model for the pharyngeal airway (a collapsible tube surrounded by soft material within a rigid box).

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