Sitting Posture Decreases Collapsibility of the Passive Pharynx in Anesthetized Paralyzed Patients with Obstructive Sleep Apnea

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is an independent risk factor for difficult and/or impossible mask ventilation during anesthesia induction. Postural change from supine to sitting improves nocturnal breathing in patients with OSA. The purpose of this study was to evaluate the effect of patient position on collapsibility of the pharyngeal airway in anesthetized and paralyzed patients with OSA. The authors tested the hypothesis that the passive pharynx is structurally less collapsible during sitting than during supine posture.

Method: Total muscle paralysis was induced with general anesthesia in nine patients with OSA, eliminating neuromuscular factors contributing to pharyngeal patency. The cross-sectional area of the pharynx was measured endoscopically at different static airway pressures. Comparison of static pressure–area plots between the supine and sitting (62° head-up) allowed assessment of the postural differences of the mechanical properties of the pharynx.

Results: Maximum cross-sectional area was greater during sitting than during supine posture at both retropalatal (median [10th–90th percentile]: 1.91 [1.52–3.40] vs 1.25 [0.65–1.97] cm²) and retroglossal (2.42 [1.72–3.84] vs 1.75 [0.47–2.35] cm²) airways. Closing pressure of the passive pharynx was significantly lower during sitting than supine posture. Differences of the closing pressures between the postures are 5.89 (3.73–11.6) and 6.74 (4.16–9.87) cm H₂O, at retropalatal and retroglossal airways, respectively, and did not differ between the pharyngeal segments.

Conclusions: Postural change from supine to sitting significantly improves collapsibility of pharyngeal airway in anesthetized and paralyzed patients with OSA.

What This Article Tells Us That Is New

❖ Although sitting posture improves symptoms in many patients with obstructive sleep apnea (OSA), whether it alters collapsibility of the pharyngeal airway during anesthesia is unknown

❖ In nine patients with OSA during general anesthesia with neuromuscular blockade, change from supine to sitting position significantly improved collapsibility of the pharyngeal airway

PHARYNGEAL airway obstruction impairs both spontaneous breathing and mechanical ventilation leading to severe hypoxemia during sleep or anesthesia. Presence of obstructive sleep apnea (OSA) is an independent risk factor for difficult and/or impossible mask ventilation during anesthesia induction. Establishment of airway management strategies for prevention or reversal of pharyngeal airway obstruction in anesthetized and paralyzed patients with OSA, therefore, is a significant task assigned to anesthesiologists responsible for patients' safety during perioperative periods.

Compared with supine posture, sitting posture is reported to decrease OSA frequency particularly in more obese patients with OSA, suggesting significant improvement of pharyngeal airway patency during sitting posture. However, no study has assessed postural changes of pharyngeal airway dimensions during sleep and anesthesia. In awake patients with OSA, previous studies demonstrated variable influences of sitting posture on the pharyngeal airway dimensions, possibly because of compensatory increase of the genioglossal muscle activity in response to the postural change from sitting to supine. Whereas significant decrease of upper airway closing pressure during sitting posture was reported in sleeping patients with OSA, pharyngeal muscle activity was not controlled during the closing pressure measurements in this...
study, and both structural and neural factors contributed to the influences of sitting posture on pharyngeal airway collapsibility.

We have developed a method for exclusively evaluating structural properties of each pharyngeal segment independently of the neural factors and successfully applied the methodology for assessing influences of various mechanical interventions on pharyngeal airway patency under general anesthesia. Accordingly, our primary purpose in this study was to test the hypothesis that sitting posture improves pharyngeal airway patency even under absence of neural mechanisms. We compared static pharyngeal mechanics of the passive pharynx during supine posture with those during sitting posture in anesthetized and paralyzed patients with OSA.

Materials and Methods

Subjects and Sleep Studies

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 (Graduate School of Medicine, Chiba University, Chiba, Japan).

We studied nine consecutive male patients with OSA who were interested in undergoing uvulopalatopharyngoplasty as a treatment for their OSA and were scheduled to have endoscopic assessment of their pharyngeal mechanics to determine whether they were favorable candidates for this procedure. Exclusion criteria in this study included (1) difficulty in performing mandible advancement and head extension, (2) presence of clinical symptoms suggesting chronic heart failure, (3) presence of pulmonary aspiration risk, and (4) presence of a beard, which potentially may cause a mask leak. All had histories of excessive daytime sleepiness, habitual snoring, and witnessed repetitive apnea. Sleep disordered breathing was evaluated by a pulse oximeter (Pulsiox-5; Minolta, Tokyo, Japan) at home. All subjects were instructed to attach an oximetry finger probe before sleep and to remove the probe on awakening. After checking quality of the recordings of arterial oxygen saturation (SpO2), oximetry variables were calculated by computer software. Although the oximetry evaluation alone does not clarify the nature of sleep-disordered breathing, we believe that all nine patients can be safely diagnosed as having OSA based on the oximetry results and the clinical symptoms. OSA diagnosis was confirmed by standard full polysomnography in seven patients with OSA. Recordings include bilateral electroencephalograms, bilateral electrooculograms, submental electromyogram, leg electromyograms, electrocardiogram, airflow measurement with a thermistor at the mouth and nose, thoracoabdominal wall motions, SpO2, snoring over a microphone, and body position. Apnea was defined as absence of airflow for more than 10 s. Hypopnea was determined upon an apparent reduction of airflow for more than 10 s with reduction of SpO2 by more than 4% from the baseline. Apneic events were classified as obstructive, mixed, and central, and the apnea-hypopnea index was calculated as the total number of the obstructive or mixed apnea and hypopnea events per hour of sleep.

Preparation of the Subjects

Each subject was initially premedicated with 0.5 mg atropine. Studies were performed with the subjects in either supine position or sitting at a 62° angle on an adjustable medical stretcher trolley, with the neck in a comfortable neutral position. The sitting angle was chosen because it was the most upright position safe for an anesthetized and paralyzed patient without restraint equipment. A modified tight-fitting nasal mask was used. Care was taken to prevent air leaks from the mask, particularly when the airway was pressurized above 20 cm H2O. Use of a chin strap maintained contact of the upper and lower incisors and eliminated air leaks through the mouth. Air leaks through the mask and mouth were detected by inadequate increase in the airway pressure (Paw) and manual palpation. General anesthesia was induced by intravenous infusion of propofol (2 mg/kg) and intravenous injection of a muscle relaxant (vecuronium 0.2 mg/kg). General anesthesia with total paralysis was maintained by continuous infusion of propofol (6–10 mg·kg−1·h−1) while the subject was ventilated through the nasal mask with positive pressure through an anesthetic machine. Complete paralysis was confirmed by no responses to train-of-four stimulations at the ulnar nerve. SpO2 and an electrocardiogram were continuously monitored, and blood pressure was noninvasively measured every 5 min. A slim endoscope (3 mm OD; FB10X; Pentax, Tokyo, Japan) was inserted through a 10-mm diameter hole in a modified nasal mask and into a naris (fig. 1). A modified silicone rubber plug with continuous bubbles (15 mm OD; SILICOSEN type L; Shin-Etsu Polymer Co., Ltd., Tokyo, Japan) tightly plugged the hole and held the endoscope to prevent leakage around them. The tip of the scope was placed at the upper airway to visualize the retropalatal airway space (airway space behind the soft palate) and the retroglossal airway space (airway space behind the base of the tongue). A closed-circuit camera (ETV8; Nisco, Saitama, Japan) was connected to the endoscope, and the pharyngeal images were recorded on a videocamera.
tape. Reading of $P_{AW}$, measured by a water manometer, was simultaneously recorded on the videotape.

**Experimental Procedures**

To determine the pressure-area relation of the pharynx, the anesthetic machine was disconnected from the nasal mask. The latter was in turn connected to a pressure-control system capable of accurately manipulating $P_{AW}$ from +20 to −20 cm H$_2$O in a stepwise fashion. Cessation of mechanical ventilation resulted in apnea caused by complete muscle paralysis. $P_{AW}$ was immediately increased up to 20 cm H$_2$O, dilating the airway. While the subject remained apneic for 2 to 3 min, $P_{AW}$ was gradually reduced from 20 cm H$_2$O to a closing pressure ($P'_{close}$) 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. In this experimental setting, the retroglossal $P_{AW}$ was not reduced below the retropalatal $P_{AW}$. $SpO_2$ was maintained above 95% during the apneic tests. This procedure of experimentally induced apnea allowed construction of the pressure-area relation 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. Measurements were made for the retropalatal and retroglossal airways with patients lying supine and sitting at a 62° angle. Care was taken to maintain the neutral neck position throughout the procedure, particularly when the patient was in the sitting position, although we did not measure the head angle. After measurements of the static pharyngeal mechanics, lung volume changes from the supine to the sitting position were measured with a spirometer connected to a tightly fitted facemask at atmospheric pressure. Patent airway was maintained during the lung volume measurement by triple airway maneuver (mandible advancement, neck extension, and mouth opening) with the use of two hands. Airway opening and absence of mask leak were confirmed by progressive increase of spirometer tracing in response to the postural change.

**Data Analysis**

The technique and accuracy of conversion of the monitor pharyngeal image to an absolute value of cross-sectional area have been reported previously. In short, magnification of the imaging system was estimated at 1.0-mm interval distances between the endoscopic tip and the object (1-cm$^2$ grid) in range of 5–30 mm, producing a relation between distance and pixels corresponding to 1 cm$^2$. At a defined value of $P_{AW}$, the image of the pharyngeal lumen was traced and counted pixels included in the area (SigmaScan version 2.0; Systat Software, Inc, San Jose, CA). The pixel number was converted to the pharyngeal cross-sectional area according to the distance-magnification relation. Using tubes of known diameter, we tested the accuracy of our cross-sectional area measurements. For a constant distance, the measured areas were systematically deviated from actual areas. The largest known area tested (0.95 cm$^2$) was underestimated by 11% because of image deformation at outer image area, and the smallest known area tested (0.03 cm$^2$) was overestimated by 13% because of reduction in image resolution. The measured luminal cross-sectional area ($A$) was plotted as a function of $P_{AW}$. We defined $P'_{close}$ as the pressure corresponding to the zero area. At high values of $P_{AW}$, relatively constant cross-sectional areas were revealed; therefore, maximum area ($A_{max}$) was determined as the mean value of the highest three $P_{AW}$ (18, 19, and 20 cm H$_2$O). As reported previously, the pressure-area relation of each pharyngeal segment was fitted by the following exponential function: $A = A_{max} - B \times \exp(K \times P_{AW})$, where $B$ and $K$ are constants. A nonlinear least-squares technique was used for the curve fitting, and the quality of the fitting was provided by coefficient $r^2$ (SigmaPlot version 2.0; Systat Software, Inc.).

A regressive estimate of $P_{close}$ which corresponds to an intercept of the curve on the $P_{AW}$ axis, was calculated from the following equation for each pharyngeal segment: $P'_{close} = \ln(B/A_{max}) \times K^{-1}$. The shape of the pressure-area relation was described by the value of $K$. When the pressure-area relation is curvilinear, compliance of the pharynx, defined as a slope of the curve, varies with changes in $P_{AW}$. Therefore, a single value of compliance calculated for a given $P_{AW}$ does not represent collapsibility of the pharynx for entire ranges of $P_{AW}$. By contrast, the $K$ value represents a rate of changes in the slope of the curve. When the $K$ value is high, small reduction of $P_{AW}$ results in significant increase in compliance, leading to remarkable reduction in cross-sectional area. Accordingly, collapsibility of the pharynx increases with increasing $K$ value. We suggest that both $P'_{close}$ 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**

Our study indicates that a maximum SD of our primary variable, the $P'_{close}$ of patients with OSA, is 2.8 cm H$_2$O. Neill et al. found a difference of 4.3 cm H$_2$O of upper airway closing pressure between supine and sitting (30° upper body elevation). Because the effect of 62° upper body elevation was assessed in this study, we expected that the $P'_{close}$ difference between the positions would be greater than 5 cm H$_2$O. Appropriate sample size was determined to be seven or more for detecting the difference assuming $\alpha = 0.05$ (two tailed) and 80% power (SigmaStat 3.1; Systat Software, Inc.). All values are expressed by median (10th–90th percentiles). Wilcoxon signed rank test was used for comparison of static mechanics variables between the supine and sitting positions (SigmaStat 3.1). Mann–Whitney rank sum test was used for comparison of the static mechanics variables between the pharyngeal segments. Spearman rank-order test was performed for correlation analyses between $P'_{close}$ differences between the positions and anthropometric and sleep.
Values are presented as median (10th–90th percentile). Polysomnogram for AHI measurements was performed in only 7 patients. AHI = apnea hypopnea index, defined as number of apnea and hypopnea per hour of sleep; BMI = body mass index; CT90 = percentage of time spent with SpO2 less than 90%; ∆LV = lung volume change in response to the position change from the supine to the sitting position; Lowest SpO2 = lowest SpO2 value among the desaturation events; ODI = oxygen desaturation index, defined as number of desaturations exceeding greater than 4% per hour of monitoring; SpO2 = oxygen saturation measured by pulse oximetry.

study data (SigmaStat 3.1). $P$ less than 0.05 (two-tailed) was considered significant.

**Results**

Endoscopic measurements of static pressure–area relations of the retropalatal and retroglossal airways were successfully performed in both the supine and sitting positions in all patients. As listed in table 1, anthropometric characteristics and sleep study data varied among the patients with OSA. Median values of these variables indicate that they were middle-aged, overweight patients with moderate to severe OSA.

Table 1. Anthropometric Characteristics and Results of Sleep Studies

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>52.0 (43.6–57.8)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>76.0 (61.6–87.0)</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.65 (1.59–1.79)</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27.9 (23.8–31.9)</td>
</tr>
<tr>
<td>ODI</td>
<td>47.5 (12.7–56.0)</td>
</tr>
<tr>
<td>CT90, %</td>
<td>18.7 (3.8–45.2)</td>
</tr>
<tr>
<td>Nadir SpO2, %</td>
<td>86.2 (76.0–89.5)</td>
</tr>
<tr>
<td>Lowest SpO2, %</td>
<td>67.0 (33.8–75.0)</td>
</tr>
<tr>
<td>AHI, hr⁻¹</td>
<td>42.2 (9.6–78.5)</td>
</tr>
<tr>
<td>∆LV, ml</td>
<td>330 (140–564)</td>
</tr>
</tbody>
</table>

Table 2 presents results of Spearman correlation analyses between $P'_{close}$ position differences and lung volume change.

**Fig. 2.** Series of endoscopic images of retropalatal (A) and retroglossal (B) airways during supine and sitting postures at various airway pressures ($P_{AW}$) in a patient with obstructive sleep apnea. Note that the cross-sectional area significantly increased for a given airway pressure at both retropalatal and retroglossal airways.

positions are 5.89 (3.73–11.6) cm H$_2$O and 6.74 (4.16–9.87) cm H$_2$O at retropalatal and retroglossal airways, respectively, and were not different in the pharyngeal segments. Median lung volume increase in response to the position change from the supine to the sitting position was 330 (140–564) ml.

**Table 2. Static Mechanics of the Retropalatal and the Retroglossal Airways in Supine and Sitting Positions**

<table>
<thead>
<tr>
<th>Retropalatal airway</th>
<th>Supine</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>$A_{max}$, cm$^2$</td>
<td>1.25 (0.65–1.97)</td>
<td>1.91 (1.52–3.40)**</td>
</tr>
<tr>
<td>$B$</td>
<td>1.74 (0.88–7.06)</td>
<td>1.17 (0.68–1.92)*</td>
</tr>
<tr>
<td>$K$</td>
<td>0.16 (0.11–0.22)</td>
<td>0.18 (0.09–0.26)</td>
</tr>
<tr>
<td>$r^2$</td>
<td>0.95 (0.92–0.97)</td>
<td>0.97 (0.92–0.99)</td>
</tr>
<tr>
<td>$P'_{close}$ cm</td>
<td>2.20 (0.84–6.12)</td>
<td>−3.47 (−8.51–1.32)**</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Retroglossal airway</th>
<th>Supine</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>$A_{max}$, cm$^2$</td>
<td>1.75 (0.47–2.35)</td>
<td>2.42 (1.72–3.84)*</td>
</tr>
<tr>
<td>$B$</td>
<td>1.84 (1.20–3.70)</td>
<td>1.01 (0.65–2.19)**</td>
</tr>
<tr>
<td>$K$</td>
<td>0.21 (0.15–0.26)</td>
<td>0.15 (0.12–0.23)</td>
</tr>
<tr>
<td>$r^2$</td>
<td>0.97 (0.91–0.99)</td>
<td>0.97 (0.83–0.99)</td>
</tr>
<tr>
<td>$P'_{close}$ cm</td>
<td>2.67 (−2.22–5.02)</td>
<td>−5.31 (−9.70–1.60)**</td>
</tr>
</tbody>
</table>

Values are presented as median (10th–90th percentile). Quality of the fit is provided by coefficient $r^2$. $A = A_{max} - B \times \exp(-K \times P_{AW})$, where $A$ and $P_{AW}$ denote cross-sectional area of the pharyngeal airway and airway pressure; $A_{max}$ = maximum cross-sectional area; $B = constant$ obtained by fitting the pressure-area relationship of each pharyngeal airway to an exponential function; $K = constant$ obtained by fitting the pressure-area relationship of each pharyngeal airway to an exponential function; $P'_{close}$ = estimated closing pressure calculated by $\ln(B/A_{max})K^{-1}$, ** $P < 0.05$, 0.01 vs. supine position, respectively.
Mechanisms of Pharyngeal Airway Patency Improvement during Sitting Position

Our results agree with the previous studies examining influences of sitting posture on pharyngeal collapsibility. Using nasal occlusion technique, Neill et al. found improvement of upper airway closing pressure from 0.3 ± 2.4 cm H$_2$O (supine) to −4.0 ± 3.2 cm H$_2$O (30° head elevation) in sleeping patients with OSA. By measuring pressure-flow relationship in non-OSA subjects under midazolam sedation, Ikeda et al. found significant reduction of the critical closing pressure from −8.2 ± 5.2 cm H$_2$O (supine) to −13.3 ± 4.9 cm H$_2$O (30° head elevation). These studies did not assess pharyngeal segments responding to the postural changes, and the neuromuscular factors were not controlled in their experimental conditions. Although our study does not completely address the mechanisms by which sitting posture improves pharyngeal airway patency, we have confirmed the results of the previous studies and evidenced significant contribution of structural factors to the mechanisms by eliminating the neuromuscular factors. We consider two possible structural mechanisms that operate near pharyngeal airway (local structural mechanism) and from a distance (structural mechanism from a distance) for development of pharyngeal obstruction.

Patients with OSA have significantly larger soft tissue volume surrounding the pharyngeal airway for a given maxillomandibular enclosure size, resulting in upper airway anatomical imbalance. The soft tissues are not uniformly distributed within the maxillomandibular enclosure. The larger mass of the soft tissues, such as the tongue, anteriorly overrides on the pharyngeal airway wall while the patient is in the supine posture. In addition, the excessive anterior soft tissues are able to be displaced through the submandible region. Accordingly, postural changes of direction of gravity acting on the soft tissues may significantly influence the anatomical balance. In fact, we previously demonstrated that lateral posture significantly improved pharyngeal airway patency. As illustrated in figure 4, a relatively larger vector of gravity perpendicular to the airway in the supine posture is divided into perpendicular and vertical components in the sitting posture. The perpendicular component of the gravity decreases during sitting posture and effective mass acting on the airway possibly decreases, possibly improving upper airway anatomical balance. In addition, gravity vertical to the airway created during sitting posture may displace the anterior soft tissue out of the maxillomandibular enclosure through the submandible region, improving anatomical imbalance. This longitudinal gravity may also increase longitudinal tension of the pharyngeal airway wall, stiffening the airway. In fact, Tsuiki et al. found significant elongation of the pharyngeal airway during the postural change from supine to sitting. Furthermore, the postural change significantly alters venous blood distribution, decreasing total soft tissue volume inside the maxillomandibular enclosure. Redolfi et al. recently demonstrated importance of fluid

**Fig. 3.** Changes of closing pressures (P'$_{close}$) at the level of retropalatal and retroglossal airways in response to postural change from supine to sitting. Each line represents a different subject. Note that the P'$_{close}$ at both pharyngeal segments decreased below the atmospheric pressure during sitting position in all patients with obstructive sleep apnea.

**Discussion**

We found that the postural change from supine to sitting enlarged both retropalatal and retroglossal airways and decreased P'$_{close}$ at both pharyngeal segments by approximately 6 cm H$_2$O in completely paralyzed and anesthetized patients with OSA. The results clearly demonstrate that structural properties of the passive pharynx improve while patients are in the sitting posture.

**Table 3.** Results of Spearman Correlation Analyses between P'$_{close}$ Difference in the Supine and Sitting Positions, and Lung Volume Change during the Position Change, Anthropometric, and Sleep Study Variables

<table>
<thead>
<tr>
<th>P'$_{close}$ Difference</th>
<th>Retropalatal</th>
<th>Retroglossal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung volume change</td>
<td>−0.650</td>
<td>−0.317</td>
</tr>
<tr>
<td>Age</td>
<td>0.151</td>
<td>0.077</td>
</tr>
<tr>
<td>BMI</td>
<td>0.267</td>
<td>0.000</td>
</tr>
<tr>
<td>ODI</td>
<td>−0.900*</td>
<td>−0.650</td>
</tr>
<tr>
<td>AHI</td>
<td>−0.786*</td>
<td>−0.607</td>
</tr>
</tbody>
</table>

Values are correlation coefficients. * $P < 0.05$.

AHI = apnea hypopnea index, defined as number of apnea and hypopnea per hour of sleep; BMI = body mass index; ODI = oxygen desaturation index, defined as number of desaturations exceeding greater than 4% per hour of monitoring.
distribution on the pharyngeal airway maintenance. Pae et al.\(^7\) reported significant reduction of the tongue volume during the sitting posture supporting this possibility. These are speculative with little evidence and need to be tested in the future studies.

Recent studies suggest significant involvement of lung volume changes in development of OSA. Heinzet et al.\(^26\) demonstrated that a 0.77-l lung volume increase during sleep in obese patients with OSA decreased the apnea hypopnea index by approximately half. Our group found significant reduction of retropalatal \(P'_{\text{close}}\) in response to 0.7-l lung volume increase in anesthetized and paralyzed patients with OSA.\(^{13}\) Direct association between \(P'_{\text{close}}\) improvement and body mass index suggested greater lung volume dependence in obese patients with OSA. Therefore, in this study, we measured lung volume change from supine to sitting posture to examine potential contribution of the lung volume to the observed \(P'_{\text{close}}\) change as an alternative structural mechanism operating at a distance from the pharyngeal airway. However, we failed to find significant association between the \(P'_{\text{close}}\) improvement and lung volume changes in response to the postural change. This may not necessarily mean that the lung volume mechanism is unimportant in the postural improvement of the pharyngeal airway. Only one OSA patient with body mass index greater than 30 kg/m\(^2\) was included in this study, and his lung volume increased by only 100 ml during sitting posture. The absolute lung volume changes in this study were unexpectedly small compared with those in previous studies, possibly because of a different degree of head elevation and positioning of the lower legs.\(^{27}\) In particular, the total number of subjects tested in this study (n = 9) is small; therefore, the absence of a relation between the \(P'_{\text{close}}\) improvement and lung volume changes should be cautiously interpreted. Future studies need to examine contribution of the lung volume to postural changes of pharyngeal collapsibility in morbidly obese patients with OSA.

**Clinical Implications**

Induction of general anesthesia places patients at risk for both respiratory and circulatory derangements. The supine posture with the head in the sniffing position is a current standard for anesthesia induction. Compared with the supine posture, head-up posture significantly prolonged the apnea tolerance period in obese patients.\(^{28}\) Valenza et al.\(^{27}\) clearly demonstrated that the beach chair position and application of positive end expiratory pressure improved lung mechanics and oxygenation in obese patients. This study further demonstrated significant increase of the pharyngeal airway size and improvement of pharyngeal collapsibility in patients with OSA. It is noteworthy that the sitting posture successfully reduced the pharyngeal closing pressure below the atmospheric pressure in all patients with OSA, indicating that the sitting posture is the most effective mechanical intervention among the other postural interventions.\(^{29}\) Taken together, respiratory function during anesthesia induction is best maintained by placing the patient in sitting posture with the head in the sniffing position while applying positive end expiratory pressure and the triple airway maneuver with two hands.\(^{30}\) Despite these respiratory advantages, the sitting posture potentially decreases cerebral blood flow as a result of induced hypotension.\(^{31,32}\) Accordingly, the beneficial effects of the sitting posture during anesthesia induction must be weighed against hemodynamic derangements particularly in patients with OSA and cardiovascular comorbidities, and the patient should be returned to the supine posture immediately after successful placement of an endotracheal tube.

In conclusion, postural change from supine to sitting significantly enlarged pharyngeal cross-sectional area and decreased closing pressures at both retropalatal and retroglossal airways in anesthetized and paralyzed patients with obstructive sleep apnea. Sitting may be an advantageous posture compared with supine posture during induction of anesthesia in these patients for airway maintenance. The possible value of the sitting position during general anesthesia induction should be investigated further in obese patients with OSA.

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**References**

1. Isono S: Obstructive sleep apnea of obese adults: Pathophysiology and perioperative airway management. *Anesthesiology* 2009; 110:908–21

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