Magnetic Resonance Imaging of the Upper Airway

Effects of Propofol Anesthesia and Nasal Continuous Positive Airway Pressure in Humans

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Background: Anesthetic agents inhibit the respiratory activity of upper airway muscles more than the diaphragm, creating a potential for narrowing or complete closure of the pharyngeal airway during anesthesia. Because the underlying mechanisms leading to airway obstruction in sleep apnea and during anesthesia are similar, it was hypothesized that anesthesia-induced pharyngeal narrowing could be counteracted by applying nasal continuous positive airway pressure (CPAP).

Methods: Anesthesia was induced in ten healthy volunteers (aged 25–34 yr) by intravenous administration of propofol in 50-mg increments every 30 s to a maximum of 300 mg. Magnetic resonance images of the upper airway (slice thickness of 5 mm or less) were obtained in the awake state, during propofol anesthesia, and during administration of propofol plus 10 cm nasal CPAP.

Results: Minimum anteroposterior diameter of the pharynx at the level of the soft palate decreased from 6.6 ± 2.2 mm (SD) in the awake state to 2.7 ± 1.5 mm (P < 0.05) during propofol anesthesia and increased to 8.43 ± 2.5 mm (P < 0.05) after nasal CPAP application. Anteroposterior diameter of the pharynx at the level of the dorsum of the tongue increased from 7.9 ± 3.5 mm during propofol anesthesia to 12.9 ± 3.6 mm (P < 0.05) after nasal CPAP. Pharyngeal volume (from the tip of the epiglottis to the tip of the soft palate, assuming this space to be a truncated cone) significantly increased from 2,437 ± 1,008 mm³ during propofol anesthesia to 5,847 ± 2,827 mm³ (P < 0.05) after nasal CPAP application.

Conclusions: In contrast to the traditional view that relaxation of the tongue causes airway obstruction, this study suggests that airway closure occurs at the level of the soft palate. Application of nasal CPAP can counteract an anesthesia-induced pharyngeal narrowing by functioning as a pneumatic splint. This is supported by the observed reduction in anteroposterior diameter at the level of the soft palate during propofol anesthesia and the subsequent increase in this measurement during nasal CPAP application. (Key words: Airway management. Anesthetics, intravenous: propofol. Measurement techniques: magnetic resonance imaging. Ventilation: nasal continuous positive airway pressure.)

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terior displacement of the tongue. However, recent work in anesthetized humans has challenged this view, implicating the soft palate or epiglottis as the site of obstruction.

In the last decade, a vast amount of knowledge has been accumulated about the mechanics and forces involved in maintaining airway patency. Most of this information was gleaned from patients with obstructive sleep apnea syndrome. The mechanisms that lead to airway obstruction in obstructive sleep apnea and during anesthesia demonstrate physiologic similarities, including reduced tonic activity of the upper airway musculature, reduced lung volume, a considerable reduction in the critical pressure for airway closure, and failure of phasic activation of the upper airway musculature to precede diaphragm activity.

Continuous positive airway pressure applied to the nose (nasal CPAP) has been shown to abolish snoring, increase hemoglobin oxygen saturation, and restore airway patency in obstructive sleep apnea syndromes. It has been proposed that nasal CPAP restores the patency of the narrowed collapsible pharyngeal airway by creating a positive transmural pressure that functions as a pneumatic splint. This "physical pressure" splint prevents upper airway occlusion by pushing the soft palate and the tongue forward and away from the posterior pharyngeal wall.

We hypothesized that anesthesia-induced pharyngeal narrowing similarly could be counteracted by applying nasal CPAP. Nasal CPAP is produced by a high flow system that delivers a continuous stream of gas mixture into a sealed nasal mask. We performed magnetic resonance imaging scans of the upper airway on healthy, nonanesthetized subjects lying supine with the head in neutral position. The scan was repeated after propofol anesthesia, with and without application of nasal CPAP, to determine whether upper airway morphology changes with nasal CPAP therapy.

Materials and Methods

This study was approved by the institutional Review Board and written, informed consent was obtained from each subject. Ten healthy volunteers (5 female, 5 male) between the ages of 25 and 34 were included in this study. Persons with a history of smoking, tobacco chewing, sleep disturbances, gastroesophageal reflux, upper airway pathology, and those weighing more than 20% of their ideal body weight were excluded from the study.

On the morning of the study, the subjects were brought to the magnetic resonance imaging suite after a regular night of sleep. They did not ingest any caffeine, nicotine, or food for eight hours before the study. A 20-G intravenous cannula was inserted into a dorsal vein of the left hand. Hemoglobin oxygen saturation was monitored continuously with a Nellcor N-100 pulse oximeter (Nellcor, Hayward, CA), and electrocardiogram was monitored by standard surface electrodes. Respiration was monitored by observing chest wall movement and by using a precordial stethoscope. A nasal cannula was applied with 2 L oxygen flow. Throughout the study period, the subjects lay supine, with their heads on a headrest maintained in a neutral position and their mouths closed.

Anesthesia was induced by intravenous administration of 50 mg propofol every 30 s to a maximum dose of 300 mg. After each incremental dose of propofol, the eyelash reflex was assessed and administration of anesthetic was continued until the reflex disappeared. Airway obstruction was judged to be present if breathing became noisy, or if the breathing pattern became abnormal. When one of these end points was reached, no more propofol was given for 2 min. After 2 min, a propofol infusion was started at a rate of 75 µg·kg⁻¹·min⁻¹ until the study was completed.

During propofol anesthesia, 10 cm H₂O nasal CPAP was applied via a tight-fitting nasal mask with the mouth closed for 10 min.

Magnetic Resonance Imaging

Each volunteer was placed inside the magnetic resonance imaging scanner and instructed to breathe normally with the mouth closed while lying supine with the head in the neutral position resting on a circular pillow. Imaging of the upper airway was performed on a 1.5-T magnet, using an anterior surface coil (General Electric Medical Systems, Milwaukee, WI). T₁-weighted images (TR 500 ms; TE 25 ms) were obtained in all three planes with a slice thickness of 5 mm or less (sagittal and axial: 5 mm/no skip; coronal: 3 mm/no skip) and a matrix of 256 × 192; total acquisition time did not exceed 80 s. Images were obtained at the following intervals: awake state, during propofol anestheisia, and after addition of 10 cm H₂O nasal CPAP.
Fig. 1. Magnetic resonance imaging parameters. (A) Midline sagittal T1-weighted images. (1) Minimum anteroposterior diameter at soft palate (diameter SP); (2) minimum anteroposterior diameter of dorsum of the tongue (diameter T); (3) minimum anteroposterior diameter of dorsum of tongue at level of epiglottis (diameter E). (B) Axial T1-weighted image at level of uvula. (C) Axial T1-weighted image at level of epiglottis.

The following measurements were made by two independent radiologists and the results were averaged: minimum anteroposterior (AP) diameter of the pharynx at the level of the dorsum of the tongue (diameter T); minimum AP diameter of the pharynx at the level of the tip of the epiglottis (diameter E); and minimum AP diameter of the pharynx at the level of the soft palate (diameter SP, figs. 1A–1C). Airway volume was calculated from the tip of the epiglottis to the tip of the soft palate, assuming this space to be a truncated cone. No attempt was made to obtain images at a fixed point in the respiratory cycle.

Statistical Evaluation

Statistical comparisons were made with the help of a computer-driven software program (Systat 5.0, Systat Inc., Evanston, IL). Measurements made during propofol anesthesia with the subjects breathing spontaneously and during nasal CPAP application were compared with measurements made during awake conditions using Student-Newman-Keuls test for paired observations. All values were expressed as means ± standard deviation. A P value < 0.05 was considered significant.

Table 1. Demographic Data

<table>
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<th>Patient No.</th>
<th>Sex</th>
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<th>Weight (kg)</th>
<th>Propofol (mg)</th>
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</table>
Results

Five men (aged 28–34 yr) and five women (aged 25–33 yr) were studied. Patient demographic data and amount of propofol administered to each subject are given in Table 1: representative sagittal T1-weighted images are shown in Figures 2A–2C.

The minimum diameter T and diameter E remained unchanged after propofol anesthesia. The tongue base did not contact the posterior pharyngeal wall in any of our study subjects. In awake supine subjects, with the mouth closed, the tongue contacted the soft palate, implying a nasal breathing route. Diameter SP decreased from 6.6 ± 2.2 mm (SD) in awake state to 2.7 ± 1.5 mm (P < 0.05) during propofol anesthesia and increased to 8.4 ± 2.5 mm (P < 0.05) after nasal CPAP application. Diameter T increased from 7.9 ± 3.5 mm during propofol anesthesia to 12.9 ± 3.6 mm (P < 0.05) after application of nasal CPAP. Pharyngeal volume increased significantly from 2,437 ± 1,008 mm³ during propofol anesthesia to 5,847 ± 2,827 mm³ (P < 0.05) after nasal CPAP application.

Four subjects demonstrated evidence of upper airway obstruction, i.e., audible snoring, which resolved with nasal CPAP application.

Discussion

Our results indicate that the airway obstruction during propofol anesthesia occurs at the level of the soft palate, not the tongue. We also demonstrated that airway patency could be restored by the application of nasal CPAP.

Our findings are remarkably similar to those of Nandi et al., who used lateral radiography to assess the changes in airway geometry in patients anesthetized with thiopental. In their series, apparent radiographic occlusions of the airway occurred most consistently at the level of the soft palate and manual traction of the tongue failed to relieve nasopharyngeal obstruction. Abernathy et al. also demonstrated inconsistent tongue movement during thiopental and propofol anesthesia. These findings, in conjunction with our current observation, suggest that the tongue is unlikely to be an important cause of airway obstruction in anesthetized patients. Unlike Nandi and Abernathy, we used a magnetic resonance imaging technique, which produces high-resolution images with superb soft tissue contrast and is well suited to examine the posterior pharyngeal wall, epiglottis, and soft palate. Further investigations of the posterior border of the tongue with magnetic resonance imaging are needed. However, because of the nature of the scanner, we could not directly evaluate the tongue. Therefore, the influence of the tongue on airway morphology could not be assessed in our study.

The soft palate, being a dynamic structure, plays a critical role of a gatekeeper in respiratory function. The soft palate is maintained in a resting position by the tongue. The posterior pharynx (oral cavity) is maintained against the tongue (nasal cavity) by gravity. The position (oral or nasal breathing) is determined by the position of the tongue, which is determined by the mechanism by which the soft palate is displaced anteriorly against the posterior pharynx. In a condition of narrowing of the airway, the soft palate, a finding suggestive of sleep apnea, moves to the minimum AP position of the soft palate. The resistance at the palatal level is similar to that of the soft palate, with obstructive sleep apnea. The mechanism of the collapse of the soft palate is not clear. Another cause of obstruction is the posterior displacement of the soft palate against the posterior pharynx. This is a dynamic process and is not static during inspiration. The optimal position of the soft palate is not known. The soft palate may move in both directions to maintain a clear airway. During application of nasal CPAP, the soft palate moves against the back of the tongue, increasing the width of the airway. This movement is not seen in the nasal airway, which is not affected by nasal CPAP. The position of the soft palate is not affected by nasal CPAP. The position of the soft palate is determined by the position of the tongue. The tongue moves in response to the pressure of the nasal CPAP. The movement of the tongue is not affected by nasal CPAP. The movement of the tongue is not affected by nasal CPAP.
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the posterior border of the tongue is seen more clearly with magnetic resonance imaging than ultrasonography. However, because of the technical limitations of the scanner, we could not obtain real-time images. Therefore, the influence of the respiratory cycle on the airway morphology could not be ascertained in the current study.

The soft palate, being strategically located between the tongue and the posterior pharyngeal wall, plays the role of a gatekeeper in regulating airflow through the nose or mouth. In awake, supine persons, the position of the soft palate is maintained either posteriorly against the posterior pharynx (oral breathing route), anteriorly against the tongue (nasal breathing route), or in a middle position (oral or nasal route). In our subjects lying supine with the mouth closed, the soft palate was lying anteriorly against the tongue, implying a nasal route of breathing. In all our volunteers, obstruction or narrowing of the airway occurred at the level of the soft palate, a finding supported by reduction in the minimum AP diameter of the pharynx at the level of the soft palate. The remarkably consistent obstruction at the palatal level is similar to that found in patients with obstructive sleep apnea syndrome. The exact mechanism by which the soft palate might occlude the pharynx is not clear. Anesthesia-induced inhibition of tensor-veli-palatine activity may result in relaxation and posterior displacement of the soft palate toward the pharynx. This leads to increased oropharyngeal airflow resistance and negative transmural pressure during inspiration, which leads to airway occlusion.

Application of nasal CPAP restored the patency of the pharyngeal airway in our study population. This observation is supported by the reduction in diameter SP during propofol anesthesia and the increase in diameters T, E, SP, and volume after nasal CPAP application. During application of nasal CPAP, the pressurized flow of air is delivered into the nares but usually does not escape through the mouth. The soft palate moves forward against the back of the tongue, sealing off the oral route in most but not all patients. Previous studies have shown that nasal CPAP is an effective nonsurgical treatment for obstructive, mixed, and central apnea. Incremental increases of nasal CPAP in patients with obstructive sleep apnea convert complete obstructive events to partial obstruction, as indicated by snoring and then elimination of snoring completely, maintaining a patent airway throughout the respiratory cycle. In our study subjects, snoring and an obstructive breathing pattern were completely eliminated after the application of nasal CPAP. We arbitrarily applied 10 cm of H2O nasal CPAP in all study subjects, restoring airway patency. We did not measure the critical pressure necessary to prevent airway occlusion. The critical level of nasal CPAP depends on the individual patient, site of airway obstruction, bulk of the soft palate, and the degree of respiratory effort. A recent study by Kuna and associates measured upper airway cross-sectional area in normal subjects with computerized tomographic scans during the application of varying nasal CPAP. The increase in airway area with nasal CPAP was primarily caused by lateral extension of the airway. Our findings are in keeping with those of Kuna’s study (fig. 3C).

The relevance of our observations in the clinical care of anesthetized patients is as follows: Commonly applied maneuvers to relieve airway obstruction, such as extension of the head, elevation of the occiput, and anterior displacement of mandible, are designed to displace the tongue and epiglottis away from the posterior pharyngeal wall. Whether a similar maneuver could restore airway patency in the presence of airway obstruction at the level of the soft palate is not definitely known. However, a preliminary clinical observation by Gallaway suggested that head extension accompanied by forward thrust of the lower jaw and flexion of the neck by use of a pillow was the most effective maneuver to relieve airway obstruction at the level of the soft palate. This maneuver often is successful in opening the airways of anesthetized patients. Our finding that obstruction occurred consistently at the level of the soft palate suggests that insertion of the nasopharyngeal airway may restore airway patency in patients anesthetized with propofol. This effect could be attributed to the oropharyngeal airway permitting airflow through the mouth rather than the nose.

The simplicity of nasal CPAP and its ease of application with equipment available in the operating room make it an ideal maneuver for prompt relief of airway obstruction in anesthetized patients. It is tempting to speculate that application of nasal CPAP may serve as an adjunct to maintain airway patency during propofol anesthesia. However, caution should be exercised before it is recommended for routine use because nasal CPAP has the potential for causing gastric distention and possible regurgitation and aspiration of gastric contents. Further, in the presence of a floppy epiglottis, nasal CPAP application may push the epiglottis within the glottis inlet, resulting in total airway obstruction.

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Our results are consistent with the hypothesis that application of nasal CPAP can counteract anesthetic-induced pharyngeal narrowing by functioning as a pneumatic splint. In contrast to the traditional view that relaxation of the tongue causes airway obstruction, this study suggests that during propofol anesthesia, airway closure occurs at the level of the soft palate. Our conclusions are limited to propofol anesthesia because previous studies have demonstrated that other anesthetic agents attenuate genioglossus activity even at minimal anesthetic concentrations. Further studies are required to test whether these findings hold true for other anesthetic agents.

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References

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