

Effect of Propofol Anesthesia and Continuous Positive Airway Pressure on Upper Airway Size and Configuration in Infants

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Background: Infants are prone to obstruction of the upper airway during general anesthesia. Continuous positive airway pressure (CPAP) is often used to prevent or treat anesthesia-induced airway obstruction. The authors studied the interaction of propofol anesthesia and CPAP on airway caliber in infants using magnetic resonance imaging.

Methods: Nine infants undergoing elective magnetic resonance imaging of the brain were studied. Head position was standardized. Spin echo magnetic resonance images of the airway were acquired at the level of the soft palate, base of the tongue, and tip of the epiglottis. Four sets of images were acquired in sequence: (1) during light propofol anesthesia at an infusion rate of $80 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, (2) after increasing the depth of propofol anesthesia by administering a bolus dose (2.0 mg/kg) and increasing the infusion rate to $240 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, (3) during continued infusion of $240 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ propofol and application of $10 \text{ cm H}_2\text{O}$ CPAP, and (4) after removal of CPAP and continued infusion of $240 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ propofol.

Results: Increasing depth of propofol anesthesia decreased airway caliber at each anatomical level, predominantly due to anteroposterior narrowing. Application of CPAP completely reversed the propofol-induced decrease in airway caliber, primarily by increasing the transverse dimension.

Conclusions: Airway narrowing with increasing depth of propofol anesthesia results predominantly from a reduction in anteroposterior dimension, whereas CPAP acts primarily to increase the transverse dimension. Although airway caliber during deep propofol anesthesia and application of CPAP was similar to that during light propofol anesthesia, there were significant configurational differences.

INCREASING depth of general anesthesia is associated with changes in upper airway size and configuration that predispose to soft tissue obstruction of the airway. Infants depend importantly on neural mechanisms for the maintenance of airway patency and may be more vulnerable to anesthesia-induced airway obstruction than are

older children and adults.^{1,2} Continuous positive airway pressure (CPAP) is an effective and commonly used intervention that improves upper airway patency and can prevent or treat anesthesia-induced airway obstruction.³⁻⁵ However, the precise effects of general anesthesia on airway size and configuration in infants and how these are modified by application of CPAP are unknown.

Magnetic resonance imaging (MRI) can accurately determine upper airway cross-sectional area and has been used to assess the effects of sedation and general anesthesia on airway caliber.^{4,6,7} We have previously used this imaging technique to evaluate the effect of increasing depth of propofol anesthesia on upper airway caliber and configuration in children.⁷ The aim of the current study was to evaluate the effect of propofol anesthesia on upper airway caliber in infants. Given the airway dependence on neural mechanisms in infants² and the sensitivity of these mechanisms to propofol,⁸ we hypothesized that airway narrowing with increasing depth of propofol anesthesia would be greater in infants than previously reported for older children. In addition, we sought to determine the degree to which the propofol-induced airway narrowing in infants is reversed by application of CPAP ($10 \text{ cm H}_2\text{O}$). Therefore, we evaluated the interaction between propofol and CPAP on upper airway caliber in infants who were scheduled to undergo elective MRI of the brain.

Materials and Methods

With approval from the Hospital for Sick Children Research Ethics Board (Toronto, Ontario, Canada), written informed parental consent was obtained to study nine unpremedicated infants, aged 5-12 months, who had American Society of Anesthesiologists physical status of I or II and were scheduled to undergo elective MRI of the brain. Excluded were infants who had pathology of the upper airway, gastroesophageal reflux, craniofacial anomalies, or increased intracranial pressure and those who were born prematurely or weighed 130% or more of ideal body weight. In addition, failure to maintain a patent airway with the head in the neutral position was considered an exclusion criterion during the study.

On arrival in the induction room, nitrous oxide (70%) in oxygen was administered, and a peripheral intravenous catheter was inserted. Nitrous oxide was discontinued immediately after insertion of the catheter. Anesthesia was induced using 2.0 mg/kg propofol and 10

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$\mu\text{g}/\text{kg}$ glycopyrrolate, and a continuous infusion of propofol was started at a rate of $80\text{--}100\ \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ using a syringe pump. Oxygen was administered *via* a facemask while infants were transported to the MRI room. An MRI-compatible device was used to monitor arterial oxygen saturation, exhaled carbon dioxide tension, respiratory rate, heart rate, and arterial blood pressure. Ventilation was spontaneous throughout the study and subsequent imaging of the brain. All patients were studied in the supine position, with the head position standardized such that the angle between the horizontal plane of the MRI table and a line connecting the tragus of the ear and the lateral corner of the eye was 110° .⁶

Magnetic resonance images were acquired using a 1.5-T MRI scanner with maximal gradient strength and slew rates of $22\ \text{mT}/\text{m}$ and $120\ \text{mT} \cdot \text{m}^{-1} \cdot \text{ms}^{-1}$, respectively (Signa Echospeed; General Electric Healthcare, Milwaukee, WI), and a quadrature head coil. A T1-weighted three-plane gradient echo localizer image (echo time = $1.6\ \text{ms}$; repetition time = $4.5\ \text{ms}$; 30° flip angle; 5-mm slice thickness; number of excitations = 1; 5 slices/plane; and 256×128 matrix) was performed to identify the midline and allow selection of subsequent axial images. An axial/oblique T2-weighted multishot fast spin echo pulse sequence (echo time = $30\ \text{ms}$; variable repetition time; 3-mm slice thickness; number of excitations = 0.5; field of view = $16 \times 9.6\ \text{cm}$; echo train length = 16; and 192×192 matrix) was used to acquire images at three anatomical levels in the upper airway: the soft palate, the base of the tongue, and the tip of the epiglottis. No attempt was made to acquire images at a fixed point in the respiratory cycle. The acquisition time was approximately 0.5 s for each image.

Acquisition of upper airway images commenced after the infant was motionless on the MRI table for approximately 5 min. Four sets of images were acquired in sequence: (1) during light propofol anesthesia at an infusion rate of $80\ \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, (2) after increasing the depth of propofol anesthesia by administering a bolus dose of $2.0\ \text{mg}/\text{kg}$ and increasing the infusion rate to $240\ \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, (3) during application of $10\ \text{cm H}_2\text{O}$ CPAP and continued infusion of $240\ \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ propofol, and (4) after removal of CPAP and continued administration of $240\ \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ propofol. These infusion rates of propofol encompass the dose range used clinically for pediatric patients undergoing ambulatory procedures.⁹ The interval between acquisition of each set of airway images was 3–5 min. CPAP was applied using an Ayre's T-piece breathing circuit connected to a 1-l reservoir bag (Vital Signs, Totowa, NJ), an MRI-compatible positive end-expiratory pressure valve (Ambu, Copenhagen, Denmark), an airway pressure gauge (Siemens, Munich, Germany), and a transparent facemask that was held in place using a head strap (fig. 1). Care was taken to ensure that application of the

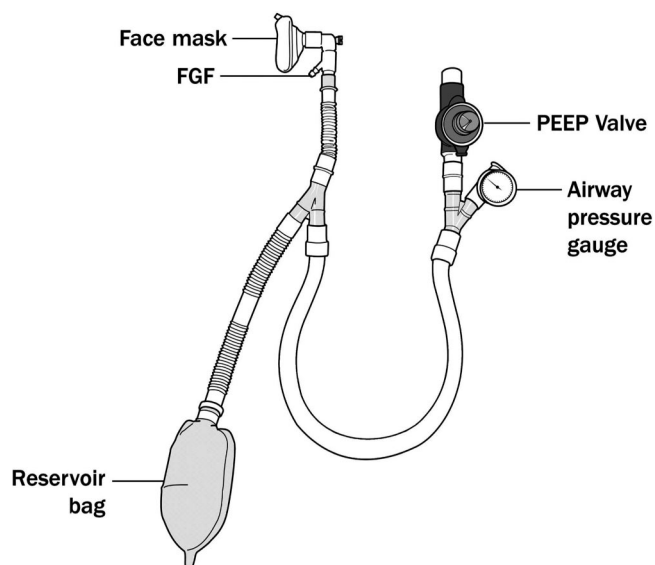


Figure 1

Fig. 1. Circuit diagram. FGF = fresh gas flow; PEEP = positive end-expiratory pressure.

facemask and head strap caused no posterior mandibular displacement. The mouth, visualized through the facemask, was seen to be closed in all patients. The infants breathed oxygen (30%) in air throughout the study and subsequent imaging of the brain. During the latter, the infusion of propofol was titrated at the discretion of the anesthesiologist.

All images were stored on computer and subsequently analyzed by a single investigator who was blinded to the depth of anesthesia and application of CPAP. To prevent observer bias, each image was assigned a random code number and presented for analysis in random order. After image magnification ($\times 3$), the upper airway cross-sectional area, anteroposterior dimension, and transverse dimension were determined using image-analysis software (General Electric Advantage Workstation 4.2; General Electric Medical Systems, Milwaukee, WI). At each anatomical level, measurements were obtained in triplicate, and average values were calculated.

Statistical Analysis

Data are expressed as mean \pm SD. We estimated *a priori* the sample size needed to detect a difference of at least $30\ \text{mm}^2$ in cross-sectional area at the level of the minimum area (soft palate), assuming an SD similar to that found in older children⁷ and a two-tailed α of 0.05. For the study to have the ability to detect such a difference at a power of 0.8, we estimated that a sample size of nine infants would be needed. One-way repeated-measures analysis of variance with the Student-Newman-Keuls *post hoc* test was used for multiple comparisons. $P < 0.05$ was considered statistically significant.

Table 1. Patient Demographics and Diagnoses

Patient	Sex	Age, months	Weight, kg	Diagnosis
1	F	12	10.0	Seizure disorder
2	F	7	7.1	Post embolization vein of Galen
3	F	5	8.0	Post resection neuroblastoma
4	F	7	8.7	Seizure disorder
5	M	11	10.0	Vascular malformation lower lip and cheek
6	M	11	8.5	Mild developmental delay
7	F	5	6.6	Giant congenital nevus scalp
8	M	10	8.6	Meningomyelocele
9	M	8	9.6	Sensorineural hearing loss, before cochlear implant

Results

The demographics of the nine infants are shown in table 1. Mean age and weight were 8.4 ± 2.7 months and 8.6 ± 1.2 kg, respectively. Mean airway cross-sectional area was least at the level of the soft palate, increasing in the order soft palate < tip of the epiglottis < base of the tongue. With increasing depth of propofol anesthesia, comparable reductions in airway cross-sectional area resulted predominantly from reductions in anteroposterior dimension and were completely reversed by application of CPAP (figs. 2–4).

Soft Palate

Increasing depth of propofol anesthesia decreased airway cross-sectional area at the level of the soft palate by 60% ($P < 0.001$; fig. 2). The decrease in cross-sectional area resulted from a 50% reduction in anteroposterior dimension ($P < 0.001$) and a 30% decrease in transverse dimension ($P < 0.05$). Application of CPAP completely reversed the propofol-induced reduction in cross-sectional area ($P < 0.001$), due primarily to an 84% increase in transverse dimension ($P < 0.001$). Application of CPAP resulted in a small increase in anteroposterior dimension that did not achieve statistical significance.

Base of the Tongue

Cross-sectional area at the level of the base of the tongue decreased by 58% ($P < 0.01$) with increasing depth of anesthesia, due to reductions in anteroposterior and transverse dimensions of 43% ($P < 0.001$) and 29% ($P < 0.01$), respectively (fig. 3). Application of CPAP reversed the reduction in cross-sectional area ($P < 0.01$), due primarily to a 76% increase in transverse dimension

($P < 0.001$) and to a small increase in anteroposterior dimension that did not achieve statistical significance.

Tip of the Epiglottis

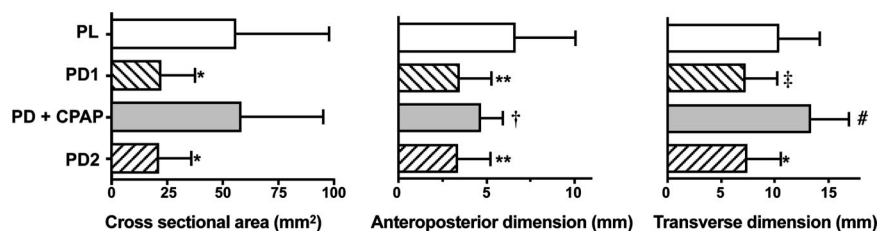
Cross-sectional area at the level of the tip of the epiglottis decreased by 51% ($P < 0.01$) with increasing depth of anesthesia, resulting from reductions in anteroposterior and transverse dimensions of 38% ($P < 0.001$) and 21% ($P < 0.05$), respectively (fig. 4). Application of CPAP completely reversed the reduction in cross-sectional area ($P < 0.05$), primarily by increasing the transverse dimension by 58% ($P < 0.001$). In all localizer images, the tip of the epiglottis was located caudal to the base of the tongue.

Removal of CPAP restored airway cross-sectional area and dimensions to values obtained before application of CPAP (figs. 2–4). Therefore, upper airway cross-sectional area during deep anesthesia and application of CPAP was similar to that during light anesthesia at each anatomical level. These changes in airway caliber are depicted in representative images at the level of the soft palate (fig. 5).

Respiratory Variables

Increasing depth of propofol anesthesia decreased the mean respiratory rate from 26 breaths/min (range, 22–33 breaths/min) to 21 breaths/min (range, 18–25 breaths/min). Arterial oxygen saturation remained unchanged during the study ($\geq 96\%$). There was a small increase in exhaled carbon dioxide tension with increasing depth of anesthesia (41 ± 4 vs. 46 ± 3 mmHg), with no further change resulting from application of CPAP. No infant demonstrated stridor, paradoxical chest wall motion, or

Fig. 2. Upper airway cross-sectional area, anteroposterior dimension, and transverse dimension at the level of the soft palate in infants. Values are mean \pm SD. PL = light propofol anesthesia (see Materials and Methods for propofol infusion rate); PD1 = deep propofol anesthesia; PD + CPAP = deep propofol anesthesia with application of 10 cm H₂O continuous positive airway pressure (CPAP); PD2 = deep propofol anesthesia after removal of CPAP. * $P < 0.001$ versus PL and PD + CPAP. ** $P < 0.001$ versus PL. † $P < 0.01$ versus PL. ‡ $P < 0.01$ versus PL and $P < 0.001$ versus PD + CPAP. # $P < 0.01$ versus PL and $P < 0.001$ versus PD1 and PD2.



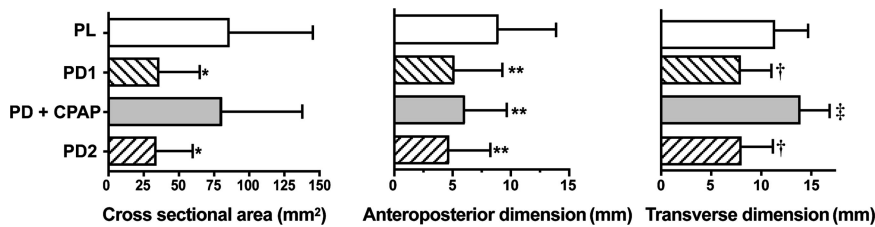


Fig. 3. Upper airway cross-sectional area, anteroposterior dimension, and transverse dimension at the level of the base of the tongue in infants. Values are mean \pm SD. PL = light propofol anesthesia (see Materials and Methods for propofol infusion rate); PD1 = deep propofol anesthesia; PD + CPAP = deep propofol anesthesia with application of 10 cm H₂O continuous positive airway pressure

(CPAP); PD2 = deep propofol anesthesia after removal of CPAP. * $P < 0.01$ versus PL and $P < 0.01$ versus PL and $P < 0.001$ versus PD + CPAP. † $P < 0.01$ versus PL. ‡ $P < 0.01$ versus PL.

other clinical evidence of upper airway obstruction during the study or subsequent imaging of the brain.

Discussion

This study shows that increasing depth of propofol anesthesia resulted in a relatively uniform reduction in caliber throughout the entire infant pharyngeal airway. This reduction in caliber was predominantly the result of a decrease in anteroposterior dimension. Application of intraluminal positive pressure, by increasing primarily the transverse airway dimension, resulted in a uniform increase in cross-sectional area that was of sufficient magnitude to reverse completely the propofol-induced reduction in airway caliber.

The methods used in the current study are fundamentally identical to those used in our previous study looking at the effect of propofol anesthesia on upper airway caliber in older children. In that study, we found that the degree of propofol-induced airway narrowing varied throughout the pharyngeal airway, with the greatest narrowing occurring in the hypopharynx at the level of the epiglottis (28%) and the least at the level of the soft palate (17%).⁷ By contrast, in the current study, the degree of propofol-induced airway narrowing was relatively uniform and considerably greater (51–60%), averaging more than double that found in older children. Although propofol infusion rates were similar in the two studies, age-related pharmacokinetic differences such as a larger volume of distribution in relation to body weight would tend to decrease the pharmacodynamic effect in infants. Despite these pharmacokinetic differences, the reduction in airway caliber was substantially greater in infants than in children, suggesting that the response of the pharyngeal airway to increasing depth of anesthesia changes with maturation.

Comparing the two studies, the greatest difference in airway collapsibility between infants and older children was found at the level of the soft palate. This was also the site of the minimum airway in infants, suggesting that the vulnerability of the infant airway to anesthesia-induced obstruction might reside predominantly at the level of the soft palate. Although the site of obstruction within the infant airway is unclear, recent studies in adults have challenged the time-honored view that obstruction occurs only in the oropharynx due to posterior displacement of the tongue¹⁰ and suggest that airway closure can occur at the level of the soft palate and epiglottis.^{8,11–14} The current findings, together with those of our previous study,⁷ provide evidence for the greater sensitivity of the infant airway to anesthesia-induced narrowing and suggest that this sensitivity might reside primarily at the level of the soft palate.

The mechanism underlying the vulnerability of the infant airway to anesthesia-induced obstruction remains speculative. Upper airway caliber is determined by the interaction between intrinsic mechanical properties of the pharynx (anatomical mechanisms) and pharyngeal dilator muscle activity (neural mechanisms).¹⁵ In this study, the anatomical mechanisms remained constant throughout the study, suggesting that the neural mechanisms that acted to maintain airway patency in the lightly anesthetized state were subsequently depressed by increasing depth of propofol anesthesia. Indeed, Eastwood *et al.*⁸ recently showed in adults that neural mechanisms, as assessed using the electromyogram activity of the genioglossus, are suppressed by increasing depth of propofol anesthesia in a dose-related fashion. In general, anesthetics suppress pharyngeal muscle activity more so than activity of the diaphragm and chest wall muscles.^{16,17} The resulting muscle imbalance is associated with an increase in airway resistance¹⁸ and is presumed

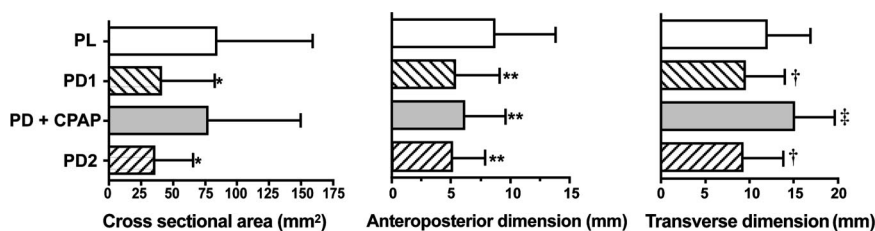


Fig. 4. Upper airway cross-sectional area, anteroposterior dimension, and transverse dimension at the level of the tip of the epiglottis in infants. Values are mean \pm SD. PL = light propofol anesthesia (see Materials and Methods for propofol infusion rate); PD1 = deep propofol anesthesia; PD + CPAP = deep propofol anesthesia with application of 10 cm H₂O continuous positive airway pressure

(CPAP); PD2 = deep propofol anesthesia after removal of CPAP. * $P < 0.01$ versus PL and $P < 0.05$ versus PD + CPAP. ** $P < 0.001$ versus PL. † $P < 0.05$ versus PL and $P < 0.001$ versus PD + CPAP. ‡ $P < 0.01$ versus PL.

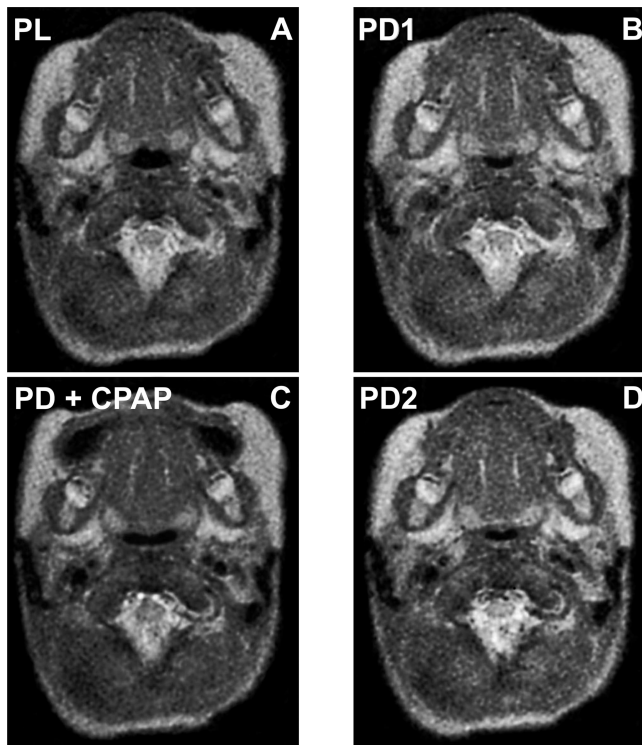


Fig. 5. Representative axial/oblique magnetic resonance images at the level of the soft palate. Four sets of images were acquired in sequence: (A) during light propofol anesthesia at an infusion rate of $80 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (PL), (B) after increasing the depth of propofol anesthesia by administering a bolus dose of 2.0 mg/kg and increasing the infusion rate to $240 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ (PD1), (C) during application of 10 cm H₂O continuous positive airway pressure (CPAP) and continued infusion of $240 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ propofol (PD + CPAP), and (D) after removal of CPAP and continued administration of $240 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ propofol (PD2).

to be an important factor predisposing the upper airway to obstruction. Moreover, developmental differences in the sensitivity of pharyngeal muscles have been reported in animals, with suppression being greater in infant than adult animals.^{19,20} Infants have a relatively collapsible upper airway²¹ and depend importantly on neural mechanisms for maintaining the patency of the structurally collapsible upper airway.² With maturation, airway collapsibility decreases as indicated by a reduction in pharyngeal airway closing pressures.²² Accordingly, the greater propofol-induced airway narrowing observed in infants can be attributed both to relatively underdeveloped anatomical mechanisms and to enhanced sensitivity of neural mechanisms to propofol.

Another important factor determining airway caliber is the effect of lung volume on the upper airway.²³ Upper airway caliber increases as lung volume increases. This effect is maximal at total lung capacity and may be attributed to a progressive increase in airway wall tension caused by traction of the lung on the upper airway, a reflex mediated by intrathoracic stretch receptors, or both.²³ A proportionately greater reduction in lung volume and hence airway caliber might occur in infants

compared with older children because infants have an exceptionally compliant chest wall and greater sensitivity of respiratory muscles to anesthetics. The relative roles of changes in upper airway muscle activity and lung volume remain to be established.

Our results are consistent with studies in infants showing that application of CPAP increased pharyngeal dimensions and reduced inspiratory inward movement of the lateral pharyngeal wall.^{24,25} CPAP has been hypothesized to enlarge the upper airway by acting as a pneumatic split or by increasing upper airway wall tension through an increase in lung volume or both. In the current study, we also evaluated the relative contributions of anteroposterior and lateral dimensional changes to the observed changes in cross-sectional area. The disproportionate decrease in anteroposterior dimension with increasing depth of propofol anesthesia is consistent with data from older children⁸ and is presumably a gravitational effect unmasked by loss of upper airway muscle tone or wall tension. The relative noncompliance of the anteroposterior dimension with application of CPAP also likely reflects gravitational influences restricting movement of the anterior airway wall and has been reported for adults.²⁴ In contrast to the relatively uniform airway distension noted in the current study, the airway distension with application of CPAP in adults was greatest in the hypopharynx, intermediate in the oropharynx, and least in the nasopharynx,³ suggesting that the regional compliance of the infant airway differs from that of the adult.

Magnetic resonance imaging yields high-resolution representations of upper airway anatomy, allowing evaluation of the effects of anesthesia on airway configuration. In the current study, we used fast spin echo pulse sequences to acquire airway images without the influence of any type of airway instrumentation. Head position was standardized, and glycopyrrolate was given to all infants to prevent accumulation of secretions, which could reduce image quality and affect upper airway collapsibility by altering mucosal surface forces.²⁶ A limitation of our study is that it was not possible to acquire baseline data in the awake state because infants will not lie motionless for the duration of imaging. Therefore, our data likely underestimate the magnitude of propofol-induced airway narrowing because they were referenced to measurements obtained in the lightly anesthetized state. A second limitation is the lack of data at varying levels of CPAP, although levels greater than 10 cm H₂O are used infrequently in clinical practice. Third, the relatively high respiratory rate in infants prevented us from acquiring images at a specific point in the respiratory cycle. Airway narrowing resulting from negative intraluminal pressure during inspiration could potentially enhance the propofol-induced narrowing^{7,24}; however, airway caliber decreased during inspiration on average by approximately 10% in anesthetized children,⁷ which is

small relative to the magnitude of change observed with propofol and CPAP in the current study.

Our results support the view that infants depend importantly on neural mechanisms for airway maintenance and are more prone to anesthesia-induced airway obstruction than older children. Airway narrowing with increasing depth of anesthesia renders the airway susceptible to collapse for several reasons.²⁷ Clinically, measures should be taken to anticipate and prevent airway obstruction when anesthetizing infants and to minimize residual anesthetics before extubation of the trachea. The application of CPAP, together with the use of various airway maneuvers, may be crucial to help maintain airway patency in anesthetized infants.

In summary, increasing depth of propofol anesthesia resulted in a reduction in the caliber of the entire pharyngeal airway in spontaneously breathing infants. The reduction in airway caliber is more than double that reported previously for older children, suggesting that infants are more sensitive to anesthesia-induced airway narrowing than are older children. Increasing depth of propofol anesthesia decreased primarily the anteroposterior airway dimension, whereas CPAP acted to increase primarily the transverse dimension. Consequently, although airway caliber during deep anesthesia and application of CPAP was similar to that during light anesthesia, there were substantial configurational differences.

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