REVIEW ARTICLES

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Pathogenesis of Upper Airway Obstruction and Mechanical Intervention during Sedation and Sleep

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Upper airway patency is balanced by both oro-pharyngeal muscle activity and the intraluminal negative pressure caused by respiratory muscles during sleep and anesthesia. The mechanical upper airway properties may become the dominant factor governing upper airway collapsibility both during sleep and sedation due to the significant depression of consciousness and the impairment of neural mechanisms controlling compensatory neuromuscular responses. It is recognized that the pathogenesis of upper airway obstruction, due to alteration of consciousness during sleep and sedation, might be similar. Furthermore, the clinicians who manage obstructive sleep apnea patients should also be aware of the pathogenesis of upper airway obstruction during sleep. Anesthesiologists and surgeons who are responsible for airway management during procedures under sedation and the perioperative period should therefore be well versed with the physiological and pathophysiological mechanisms affecting upper airway patency. This review article presents the current understanding of mechanisms for maintaining upper airway patency during sleep and sedation based on the similarity of the pathophysiology governing upper airway patency. Possible mechanical interventions based on a quantitative analysis of upper airway collapsibility analyzing inspiratory flow limitation are also discussed.

Keywords: upper airway obstruction, sleep, sedation, obstructive sleep apnea, maxillofacial surgery

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INTRODUCTION

Upper airway patency depends on an appropriate balance between the dilating force of the pharyngeal muscles and the collapsing force of negative intraluminal pressure, which is generated by respiratory pump muscles. It is well accepted that maintenance of upper airway patency is a critical issue during sleep and sedation, because loss of consciousness may induce a depression of central respiratory output[1–3] by altering hypercapnic and hypoxic ventilatory drives, and it may decrease muscle contractility via cellular mechanisms by blocking sarcolemmal sodium channels.[4] Furthermore, it has been suggested that the neuromuscular activity of upper airway dilator muscles, such as the genioglossus and geniohyoid, may be affected by depression of hypoglossal motor neurons, which regulate tonic activation of these muscles. These influences, in association with depression of consciousness during sleep and sedation, may result in hypopnea and apnea due to upper airway obstruction. It has been suggested that the pathophysiology of upper airway obstruction might be similar in sleep and sedation.[5,6] Furthermore, the maintenance of mechanical upper airway properties may contribute significantly to upper airway patency. Therefore, understanding of the pathogenesis of upper airway obstruction may help establish clinical diagnostic and treatment methods in both sleep and sedation. Clinicians who are responsible for airway management during sleep and sedation should, therefore, be familiar with the physiological mechanisms influencing upper airway patency. This review article presents the current understanding of mechanisms for maintaining upper airway patency and discusses the developmental aspects of the mechanisms, based on a quantitative analysis of upper airway collapsibility using the concept of flow limitation. Furthermore, the similarity of the pathophysiology of upper airway obstruction between sleep and anesthesia based on the features of upper airway obstruction is also discussed. Lastly, the implications of the changes of upper airway patency by mechanical interventions during sleep and anesthesia are discussed.

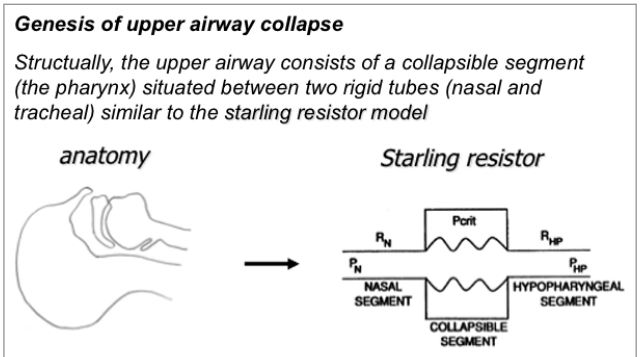
PATHOGENESIS OF UPPER AIRWAY OBSTRUCTION UNDER ANESTHESIA

The anatomical structure of the upper airway is characterized by a balanced combination of soft tissue components (tongue, soft palate, and pharyngeal mucosa) and bony structure components (maxilla, mandible, and vertebrae).[7] Upper airway patency is determined by precise interaction between the mechanical properties of the upper airway (anatomical mechanisms) and neural regulation of pharyngeal dilator muscle activity (neural mechanisms). Previously, the Neural Balance Model[8] and the Anatomical Balance Model[9] were introduced to understand how the upper airway is protected against upper airway obstruction during anesthesia.

RESPONSE TO ACUTE AND SUSTAINED PARTIAL UPPER AIRWAY OBSTRUCTION

Upper airway obstruction during sleep plays a pivotal role in the pathogenesis of obstructive sleep apnea[10] and is caused by structural defects and disturbances in neuromuscular

Figure 1—Mechanical analogue of upper airway consists of a two tube with a collapsible segment, upstream (nasal) and downstream (hypopharyngeal) segments.



The collapsible segment collapses only when tissue surrounding pressure exceeds intraluminal pressure. Under the conditions of airflow limitation, maximal flow (V_I max) is determined by the gradient between the upstream nasal pressure (P_N) and the P_{CRIT}, and the resistance (R_N) upstream as described in the equation, $V_I\,max = (P_N - P_{CRIT}) / R_N$. P_N = nasal pressure, P_{HP} = hypopharyngeal pressure, R_N = resistance in nasal segment, P_{HP} = resistance in hypopharyngeal segment.

control.[6] Upper airway obstruction can elicit neuromuscular responses that mitigate and/or compensate for the obstruction. Under conditions of upper airway obstruction (inspiratory airflow limitation), immediate responses in respiratory timing indices can help restore ventilation[11] and blunt disturbances in gas exchange.[12] Nevertheless, the impact of respiratory pattern responses on ventilation during periods of upper airway obstruction remains unclear. It has recently been suggested that the respiratory cycle, but not the respiratory rate, determines the individual's ability to compensate for inspiratory airflow limitation during sleep[13] and during propofol anesthesia,[5] and it may represent a quantitative phenotype for obstructive sleep apnea susceptibility.

It has also been indicated that the compensatory neuromuscular response to upper airway obstruction is partly intact during propofol anesthesia with spontaneous breathing.[5] Interestingly, it has been reported that there was a significant difference in the compensatory neuromuscular response to upper airway obstruction between male and female subjects during midazolam sedation.[14] In natural NREM sleep, there was a significant difference in the compensatory neuromuscular response to upper airway obstruction between OSA patients and healthy subjects.[6] It is easy to expect that patients with depressed neuromuscular activity, such as cerebral palsy patients, may have weaker effects of this function. Furthermore, similar to OSA, aging may affect the magnitude of the compensatory neuromuscular response to upper airway obstruction.

During natural sleep, when sustained partial obstruction or complete obstruction occurs, the obstructed upper airway is re-opened by a brief arousal response, resulting in the return of muscle tone.[15] During sedation, the decrease in muscle tone associated with reduction of consciousness is compounded by specific drug-induced inhibition of upper airway neural and muscular activity and suppression of protective arousal responses. This depression of the arousal reflex during sedation even more than during NREM sleep might increase upper airway obstruction, such that external mechanical intervention may be needed to overcome the obstruction. Furthermore, the role of chemoreceptors from carotid body and retroambiguus nucleus may be depressed during sedation and anesthesia.[16] Although the arousal response against sustained upper airway obstruction is a fundamental defensive mechanism in the compensatory neural system to maintain upper airway patency, this arousal response may be even more depressed as anesthetic depth increases.

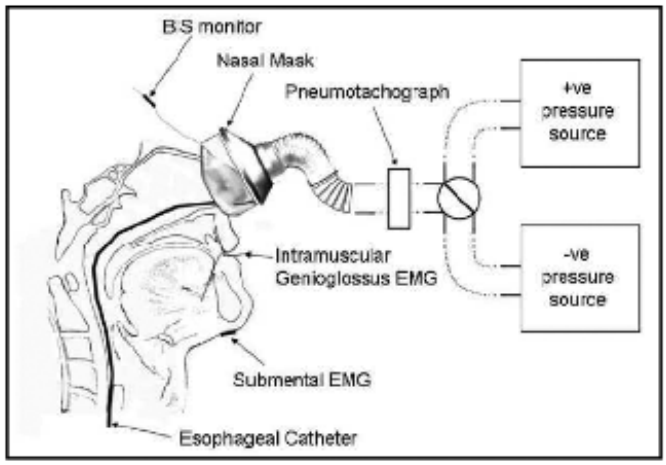
EVALUATION OF UPPER AIRWAY COLLAPSIBILITY

Concept of Flow Limitation and Critical Closing Pressure (P_{CRIT}) (Figures 1–4)

Upper airway collapsibility is evaluated by static imaging analysis via 2 dimension computerized tomography (CT) or magnetic resonance imaging (MRI), and 2 dimension cephalography X-ray, as well as dynamic analysis using the pressure-flow/volume relationship and pressure-cross-sectional area curves.

Among these quantitative analyses of upper airway patency, determination of the airway pressure that causes airway collapse and inspiratory airflow limitation in patients with obstructive sleep apnea has been used extensively in sleep apnea research. The application of negative airway pressure to determine the collapsibility of the upper airway using pressure-flow relationships, as seen with flow limitation or complete obstruction, has been used during anesthesia and sleep. The concept of critical closing pressure (P_{CRIT}) arises from modeling the upper airway as a simple collapsible tube and generation of multipoint pressure flow (P-Q) relationships, which are then used to assess upper airway patency.[17] Schwartz et al.[18] indicated that the upper airway patency can be explained by a Starling resistor model (**Figure 1**), in which inspiratory flow limitation occurs once upper airway upstream pressure falls below a critical closing pressure (P_{CRIT}). It has been shown that P_{CRIT} (representing nasal pressure at zero flow, an index of upper airway collapsibility) and resistance (which reflects the degree of upper airway narrowing upstream to the site of collapse) are key elements governing upper airway patency. P_{CRIT} can be estimated for the quantitative evaluation of upper airway patency based on nasal pressure and maximum inspiratory airflow in laboratory and has been validated for clinical usage or a research tool, even though this is not easy to extract from ordinary respiratory parameters. It is worth mentioning the clinical significance of P_{CRIT}. In fact, a ~5-cm H_2O decrease in $P_{CRIT,}$ due to increased neuromuscular activity, has the same stabilizing effect as applying ~5 cm H_2O of continuous positive airway pressure (CPAP) in reversing upper airway obstruction in obstructive apnea patients. It was previously suggested that a

Figure 2—Diagram of the experimental setup.

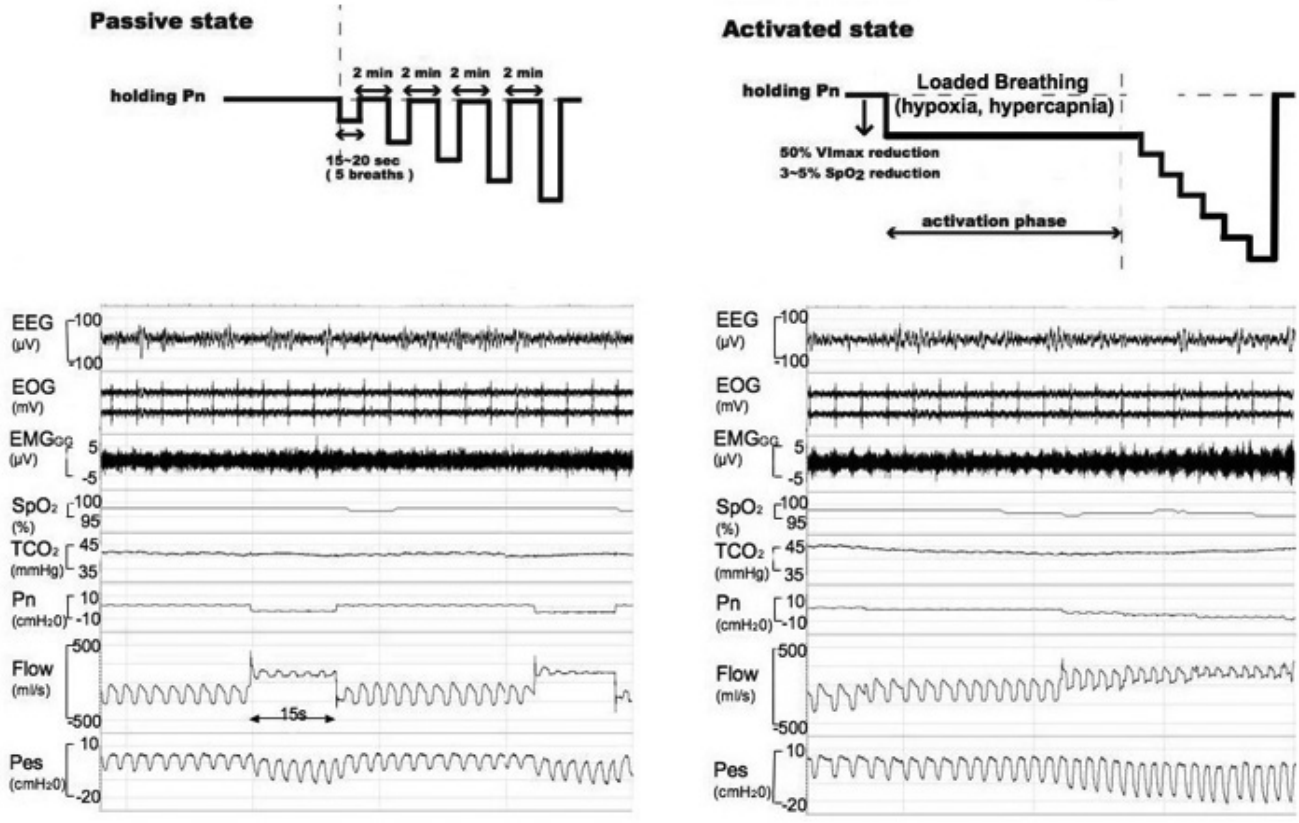


Maximally inspiratory flow (Vlmax)
Nasal pressure (Pn)
Esophageal pressure (Peso)
Inductance plethymogram (SUM)
Electroencephalograms (EEG: C3-A2)
Electrooculograms (EOG)
Submental electromyograms (EMG)
$ETCO_2$, SpO_2, BP

Hoshino Y. Respir Physiol Neurobiol 2009;166(1):24–31.

A nasal mask attached to a pneumotachograph is connected via tubing to either a positive (+ve) or negative (−ve) pressure source. Electromyography of the genioglossus muscle (EMG_{GG}) was recorded using fine wire intramuscular electrodes positioned percutaneously. Respiratory effort was determined by an esophageal pressure transducer-tipped catheter that was inserted via the nares. The bispectral index (BIS), electroencephalography, and submental surface EMG were recorded to monitor depth of anesthesia.

Figure 3—A schematic of the experimental protocol for producing upper airflow obstruction.



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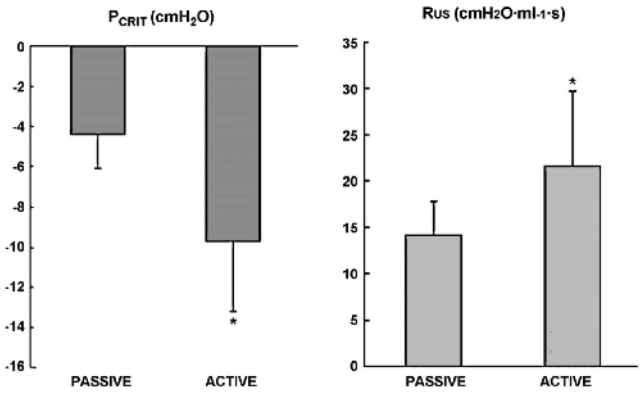
The polysomnographic recordings include the electroencephalogram (EEG), electro-oculogram (EOG), intramuscular genioglossus electromyogram (EMG_{GG}), nasal mask pressure (P_N), pneumotach airflow (V = Flow), esophageal pressure (*P*eso), and impedance plethysmography (RESP). A stable unobstructed breathing pattern was initially maintained at a positive holding pressure. Thereafter, P_N was lowered by 2 cm H_2O steps until a quasi-steady state flow-limited breathing pattern associated with a 40% to 50% reduction in V_I max (partial obstruction) was achieved. Subsequently, P_N was lowered in a stepwise fashion by 2 cm H_2O every 5 breaths, until zero flow of complete obstruction associated with an increase in respiratory negative pressure was obtained or SpO_2 reached a lower limit of 88% to 90%.

Figure 4—The change of compensatory neuromuscular response during propofol anesthesia.



Hoshino Y. Respir Physiol Neurobiol 2009;166(1):24–31.

The analysis of critical closing pressure (P_{CRIT}) and upper airway resistance (R_{US}) is shown. P_{CRIT} is calculated as a value of nasal pressure at zero flow by a linear regression analysis between maximum inspiratory airflow and nasal pressure.

change in P_{CRIT} of ~5 cm H_2O due to neuromuscular activity is clinically relevant,[6] since this represents the magnitude of the response required to convert either obstructive apneic events to less severe hypopneic events or hypopneic events to stable breathing. Because the P_{CRIT} measurements can be clinically relevant for evaluating upper airway collapsibility in patients during anesthesia and sleep, this method might be useful for investigating the pathophysiology of upper airway obstruction occurring during monitored anesthesia care. The advantage of this model is that it gives a global measure of upper airway collapsibility that includes both the structural and neuromuscular factors that determine upper airway collapsibility. More recently, advanced methods for evaluating both the mechanical properties and the compensatory neuromuscular responses to upper airway obstruction were predicted.[5,6] The most recent paper revealed that the P_{CRIT} can be assessed by the analysis software (P_{CRIT} Analysis Software in a numerical computing enviroment with 4th generation programming language: PAS) to streamline P_{CRIT} analysis using quantitative airflow measurement data in clinical cohorts study.[19]

PATIENT FACTORS PREDISPOSING TO UPPER AIRWAY OBSTRUCTION

Patient Position during the Procedure

Supine Position

The supine posture predisposes to upper airway obstruction, as the effects of gravity increase the extra-luminal compressive forces exerted by the tongue, soft palate, and related structures, resulting in narrowing of the retropalatal and retrolingual spaces.[20]

Head Down Posture:

The table tilt with head down position is recognized as being unfavorable, because of loss of longitudinal tension on the upper airway and fluid displacement into the upper airway region. Fluid displacement from the lower body to upper airway regions may increase upper airway collapsibility. Shepard et al. suggested that fluid accumulation in soft tissues surrounding the upper airway may increase pharyngeal collapsibility in patients with OSA.[21] It has also been shown that ~375 mL of fluid displacement from the legs by lower body positive pressure increases upper airway collapsibility by about 7.6 ± 1.9 cm H_2O in healthy, non-obese men while awake.[22] More recent studies have shown that fluctuation of estrogen and progesterone levels is coupled to fluid shifts from the vascular into the interstitial fluid compartments, causing edema.[23–25]

Neck Flexion

Neck flexion reportedly decreases pharyngeal size and increases passive P_{CRIT} in anesthetized patients.[9,26] Walsh et al.[26] reported that neck flexion with 10-degree deviation from the neutral position produced a 4.9 ± 3.1 cm H_2O increase in passive P_{CRIT}. Head elevation with a pillow seems to dose-dependently improve pharyngeal patency, although the possibility of simultaneous neck flexion would attenuate the beneficial effects of head elevation.[27] Accidental neck flexion may easily occur during surgical procedures in the oro-pharyngeal region.

Bite (Mouth) Opening

It is essential to keep the mouth open during oral-maxillofacial surgical procedures and dental treatment. However, as has been previously reported, this may cause obstruction.[28,29] Mouth opening decreases the space enclosed by the maxilla, mandible, and cervical vertebrae and increases the soft tissue volume inside the bony box, similar to that with neck flexion, since the mandibular movement with mouth opening is essentially the same as that which occurs with neck flexion.[9,28] Accordingly, the resultant increase in passive P_{CRIT} is predictable by the possible anatomical imbalance during mouth opening. In fact, passive P_{CRIT} increased significantly by 5.1 cm H_2O with the mouth open.[28] Mouth opening may change the vector force direction of the pharyngeal dilator muscles. Obviously there are high risks of increased upper airway collapsibility by existence of large tonsils[30–32] and macroglossia.[33,34]

Mechanical Displacement of the Tongue

Clinically, the tongue is frequently manipulated during surgical procedures in the mouth, with compression by instruments (tongue retractor) to avoid accidental injury by surgical instruments or to maintain the surgical view. In contrast to the beneficial effect of tongue protrusion on upper airway collapsibility[35] during sleep, downward displacement (retrograde) of the tongue in supine position might increase upper airway collapsibility during sleep.[36,37] Although the effect of tongue displacement on upper airway collapsibility during anesthesia has not been well established, we should be aware of the potential risk of surgical procedures in the oro-pharyngeal region.

Patients' Individual Anatomical Factors

It is well recognized that obesity, micrognathia, macroglossia and maxillary hypoplasia, acromegaly, Down syndrome, Pierre-Robin syndrome, or other craniofacial abnormalities

are major anatomical risk factors for upper airway obstruction. The degree of obstruction depends on the anatomical abnormalities in the pharynx. Furthermore, sex and age may other factors for controlling upper airway patency.

Recently, we demonstrated that female patients in the luteal phase of their menstrual cycle had an increased passive P_{CRIT} during propofol anesthesia. This conceivably reflects the development of pharyngeal edema due to the effect of sex hormones.[38] Based on evidence that there is a significant increase in edema formation in the upper airway region during the late-luteal phase in premenstrual dysphoric disorders,[39] we speculated that upper airway collapsibility may be significantly increased by edema formation[40] in the premenstrual phase, especially when there is a reduction of neuromuscular activity during shifts in progesterone level. We believe that our findings may provide new insight into the management of sedation in pregnant women, since they have much higher estrogen levels and significant upper airway edema.

Several studies have shown that surface tension[41–43] and saliva production[44] are important factors controlling upper airway patency, because surface tension is important for re-opening closed upper airways. These studies demonstrated that reduction of the surface tension in the upper airway mucosa by one-third can reduce the difference between the opening pressure and the closing pressures of the upper airway by 2 cm H_2O. Furthermore, our recent study found that an increase of 100-nM phosphatidylcholine decreases surface tension of saliva by ~17 mN/m. Surface tension may be increased in Sjögren syndrome.[45] Kirkness et al.[42] revealed that changes in surface tension significantly reflect the changes in upper airway opening pressure without affecting the upper airway closing pressure in humans. Deformation of the upper airway by negative transmural pressure during inspiration alters the activity of upper airway mechanoreceptors, causing a reflex increase in upper airway muscle activity. There may be significant influence of inflammation of pharyngeal tissue and neuropathic changes in tissue on reduction of reactivity in maintaining upper airway patency.

EFFECT OF SEDATION ON UPPER AIRWAY COLLAPSIBILITY (P_{CRIT})

It has been reported that the upper airway tends to get obstructed during sedation. Changes in upper airway patency during sedation appear to vary with the agents used, which include intravenous anesthetics (propofol) and sedative drugs (midazolam).[46,47] Midazolam and propofol are common anesthetic agents administered to provide anxiolysis, sedation, and amnesia during interventional procedures due to their rapid onset and limited duration of action. Although the effects of midazolam and propofol anesthesia are believed to be equivalent in terms of upper airway patency[47] during moderate levels of monitored anesthesia care, upper airway collapsibility dose-dependently increases as depth of anesthesia increases with each anesthetic agent.[46,48] Norton et al.[47] suggested that midazolam and propofol anesthesia have the same propensity for causing upper airway obstruction with mild to moderate sedation, based on an analysis using dynamic negative airway pressures. Using P_{CRIT} analysis (**Figure 1**), we confirmed that upper airway mechanical properties are similar with midazolam (mean value of passive P_{CRIT} = −5.1 cm H_2O) and propofol (mean value of passive P_{CRIT} = −4.4 cm H_2O) even during deeper stages of anesthesia.[5,28] This value of passive P_{CRIT} during anesthesia is similar to the mean value of passive P_{CRIT} values (−4.5 ± 3.0 cm H_2O) in normal subjects during natural sleep.[6] Therefore, we concluded that tonic neuromuscular activity of upper airway dilator muscles is relatively intact during monitored anesthesia care with midazolam and propofol, and the upper airway mechanical properties are the same as those during sleep. Interestingly, Eikermann et al.[49] showed that ketamine is a respiratory stimulant that abolishes the coupling between loss of consciousness and upper airway dilator muscle dysfunction over a wide dose range. They also indicated that ketamine might help stabilize upper airway patency during anesthesia. During sedation or hypnotic-induced sleep, repeated measurements of upper airway collapsibility appear to have less variability, suggesting that arousal and alterations in posture contribute to mechanical alterations in upper airway properties.[50]

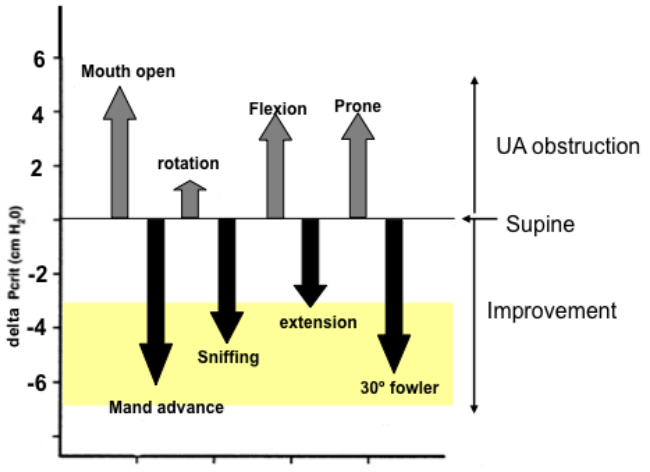
Recently, there appears to be a clinical advantage to use dexmedetomidine (DEX) for procedural sedation in pediatric patients and OSA patients.[51,52] In contrast to other sedative agents, DEX can provide better sedative properties similar to natural NREM sleep, without major respiratory depression. Therefore, DEX has recently been recommended for sedation during procedural sedation with local anesthesia for children and OSA patients.

Topical anesthesia of the upper airway mucosa, which greatly reduces this reflex response,[53,54] causes an increase in upper airway resistance and, thus decreases airflow during sleep. Berry et al. suggested that topical lidocaine applied to the nasal trigeminal area and hypopharynx-laryngeal area markedly induced airflow limitation due to reduction of the amount of phasic activity of the genioglossus electromyogram[53] during NREM sleep. A previous study[54] also showed that topical anesthesia might increase pharyngeal resistance during stage 1 sleep and at the wake to sleep transitions due to elimination of upper airway mucosal mechanoreceptors. Although the effect of local anesthesia on upper airway collapsibility during anesthesia has not been understood, the influence of local anesthesia in the oro-pharyngeal region may further modify sensitivity to negative airway pressure and chemical reflexes, increasing the patients' risk during monitored anesthesia care.

HOW CAN UPPER AIRWAY PATENCY BE MAINTAINED? (Figures 5, 6)

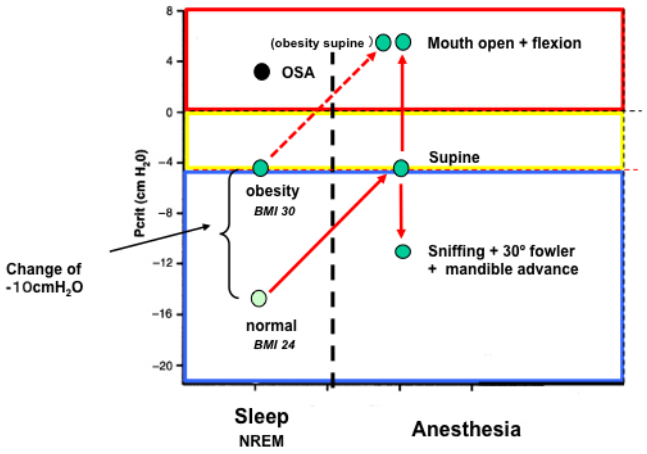
It is fundamentally impossible to keep the mouth closed during a procedure in order to maintain upper airway patency by avoiding the effect of mouth opening on the increase in upper airway collapsibility. Therefore, we should establish another mechanical intervention to maintain upper airway patency. How can we minimize the risk of upper airway obstruction during a procedure?

Figure 5—Effects of head and upper body position on upper airway collapsibility during sedation.



Opening of the mouth, rotation of the neck, neck flexion, and prone positioning cause upper airway obstruction. In contrast, mandible advancement, sniffing position, neck extension, and the 30-degree Fowler position decrease upper airway collapsibility.

Figure 6—Predicted effects of mechanical intervention on changes in critical closing pressure (P_{CRIT}) in obese patients during sedation.



During NREM sleep, the P_{CRIT} is higher in obese patients (BMI 30 kg/m^2) than that in normal subjects (BMI 24 kg/m^2) and lower than that in moderate OSA patients. If obese patients are managed under sedation in the supine position, upper airway collapsibility may increase, with higher P_{CRIT} values than during sleep. Positional change of mouth opening may further increase upper airway collapsibility with higher P_{CRIT} values. Mechanical intervention by the attending clinician, in the form of maintaining the patient's head and neck in the 30-degree fowler position or sniffing position, might improve upper airway collapsibility. Furthermore, simultaneous mandibular advancement may further improve upper airway collapsibility, similar to that during NREM sleep in normal subjects.

Effects of Mandible Advancement on Upper Airway Patency

Previously, we found that mandibular advancement significantly decreased P_{CRIT} to −13.3 ± 3.2 cm H_2O ($p < 0.05$ vs. the centric position), but it did not significantly affect upstream airway resistance (Rua) calculated by equation of V_I max = (P_N − P_{CRIT}) / Rua (22.1 ± 6.3 cm H_2O/L/s) during midazolam sedation.[55] In this study, we evaluated upper airway collapsibility in three different mandibular positions, centric occlusion position, incisors aligned position, and mandible advancement position (75% of the subject's maximum possible protrusion without any excessive discomfort and pain). Briefly, three different types of rigid-type custom mandible appliance were made during awake condition at different experimental day prior to experimental sedation condition. Three mandibular appliances with centric occlusion position, incisors aligned position, and mandible advancement position (75% of the subject's maximum possible protrusion) was constructed of clear acrylic resin and 1-mm polyethylene plate (Erkodur; Erkodent Inc.; Pfalzgrafenweiler, Germany) for each subject in reference to previous study by Tsuiki et al.[56] This study indicated that mandibular advancement in the incisor-aligned position can decrease both upper airway collapsibility by changes of P_{CRIT} and resistance during midazolam sedation, and that maximal mandible advancement (maximal comfortable protrusion) may not be necessary for the preservation of upper airway patency. We found that mandibular advancement produced isolated decreases in P_{CRIT}, indicating a decrease in collapsibility at the flow-limiting site.[28] Moreover, this evidence indicates that mandibular advancement should ameliorate sleep apnea if P_{CRIT} falls by 5 to 10 cm H_2O. In patients in whom moderate upper airway obstruction predominates, more modest degrees of mandibular advancement (possibly 25% to 50% of the patient's maximum possible protrusion) should be clinically effective, since decreases in P_{CRIT} of only 3 to 5 cm H_2O are required to relieve airflow obstruction during sleep and sedation. We have also suggested that the degrees of mandibular advancement can be titrated progressively to relieve obstruction in patients with partial or complete upper airway occlusion during sleep,[57] possibly due to the allowing muscle fibers adaptation. However, it should be noted that mandible advancement with mouth opening might alter the respiratory phase resetting during swallowing and the timing of swallowing in relation to the respiratory cycle phase. This finding indicates that mandible re-positioning may strongly affect coordination between nasal breathing and non-nutritive swallowing by altering respiratory parameters and by inhibiting movement of the tongue-jaw complex.[58]

Neck Extension and Chin Lift

Isono et al. reported that neck extension significantly decreased closing pressure of the velopharynx and oropharynx.[9] They observed an approximately 3.5-cm H_2O reduction in passive P_{CRIT} in the velopharynx and oropharynx and suggested that neck extension significantly decreases compliance of the oropharyngeal airway wall. Previous studies reported that the chin lift caused widening of the entire pharyngeal airway during propofol sedation.[59,60] They also suggested that the improvement in airway collapsibility during the chin lift is caused by a combination of increased tension of the pharyngeal muscles and forward movement of the muscles attached to the mandible. A previous study also confirmed that drug-induced

sleep endoscopy completed with a simulation bite approach for the prediction of the outcome of treatment of obstructive sleep apnea with mandibular repositioning appliances.[61]

Sniffing Position (Head Elevation)

Placing the head in the "sniffing position"[62] (lower cervical flexion, upper cervical extension with full extension of head on neck) increases longitudinal tension on the upper airway and decreases its collapsibility. Similar to neck extension, the sniffing position increases the distance between the mentum and cervical column, consequently increasing the space enclosed by the maxilla, mandible, and cervical vertebrae. This possibly results in a predictable reduction in passive P_{CRIT} due to improvement of mechanical factors in the sniffing position, although no information is available on changes in soft tissue volume in the pharynx. Recently Kobayashi et al. found a significant reduction in passive P_{CRIT} by a mean value of 4.3 cm H_2O in response to 6-cm head elevation during propofol anesthesia with spontaneous breathing.[63] They demonstrated that the optimal height of head elevation in normal-weight subjects under propofol anesthesia with spontaneous breathing through the closed mouth was approximately 6.0 cm.

Lateral Position

Boudewyns reported that P_{CRIT} fell from 1.8 cm H_2O in the supine position to –1.1 cm H_2O (delta 2.9 cm H_2O) in the lateral recumbent position.[64] Another study found that the upper airway of a spontaneously breathing child who was deeply sedated with propofol widened in the lateral position.[65]

Head Rotation

We previously demonstrated that head rotation decreased upper airway collapsibility in adult subjects during midazolam sedation.[66] However, we concluded that the therapeutic effect was insufficient to maintain upper airway patency. A previous study observed that passive P_{CRIT} (–2.8 cm H_2O) increased significantly when the head was rotated, compared to P_{CRIT} (–4.4 cm H_2O) in the supine condition (delta 1.6 cm H_2O) in pediatric patients, indicating a significant increase in pharyngeal airway collapsibility in the head rotated position.[67]

Upper Body Elevation (Sitting Position)

A previous study[68] found that a 30-degree elevation of the upper body resulted in an improvement of upper airway collapsibility compared with both the supine and lateral positions, as seen by measuring upper airway closing pressures in patients with obstructive sleep apnea. They reported that a 30-degree elevation caused a 4.3-cm H_2O decrease in P_{CRIT}, while adopting the lateral position caused a 1.4-cm H_2O decrease in P_{CRIT} relative to the supine position. In our previous study,[66] we found a 5.4-cm H_2O decrease in closing pressure after 30-degree upper body elevation compared to the supine position. A previous study indicated that postural change from supine to sitting positions enlarged both retropalatal and retroglossal airways and decreased P_{CRIT} in both pharyngeal segments by approximately 6 cm H_2O in completely paralyzed and anesthetized patients with OSA.[69] They postulated that this improvement can be due to mechanical interaction between the thorax and upper airway, such that caudal movement of the larynx with increasing lung volume results in secondary stiffening and dilatation of the pharynx.[69]

CONCLUSION

Mechanical upper airway anatomy may become the dominant factor governing upper airway collapsibility during sleep and sedation due to the significant impairment of neural mechanisms controlling compensatory neuromuscular responses. It is, therefore, important to understand the effectiveness of mechanical interventions and develop a systematic approach to evaluating the factors that contribute to maintenance of upper airway patency during sleep and sedation.

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