Effect of Head Elevation on Passive Upper Airway Collapsibility in Normal Subjects during Propofol Anesthesia

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ABSTRACT

Background: Head elevation can restore airway patency during anesthesia, although its effect may be offset by concomitant bite opening or accidental neck flexion. The aim of this study is to examine the effect of head elevation on the passive upper airway collapsibility during propofol anesthesia.

Method: Twenty male subjects were studied, randomized to one of two experimental groups: fixed-jaw or free-jaw. Propofol infusion was used for induction and to maintain blood at a constant target concentration between 1.5 and 2.0 μg/ml. Nasal mask pressure (P_N) was intermittently reduced to evaluate the upper airway collapsibility (passive P_{CRIT}) and upstream resistance (R_{US}) at each level of head elevation (0, 3, 6, and 9 cm). The authors measured the Frankfort plane (head flexion) and the mandible plane (jaw opening) angles at each level of head elevation. Analysis of variance was used to determine the effect of head elevation on P_{CRIT}, head flexion, and jaw opening within each group.

Results: In both groups the Frankfort plane and mandible plane angles increased with head elevation ($P < 0.05$), although the mandible plane angle was smaller in the free-jaw group (*i.e.*, increased jaw opening). In the fixed-jaw group, head elevation decreased upper airway collapsibility ($P_{CRIT} \sim -7$ cm H_2O at greater than 6 cm elevation) compared with the baseline position ($P_{CRIT} \sim -3$ cm H_2O at 0 cm elevation; $P < 0.05$).

Conclusion: Elevating the head position by 6 cm while ensuring mouth closure (centric occlusion) produces substantial decreases in upper airway collapsibility and maintains upper airway patency during anesthesia.

What We Already Know about This Topic

- The sniffing position improves pharyngeal airway patency. The beneficial effect of head elevation can be offset by simultaneous neck flexion and jaw opening.

What This Article Tells Us That Is New

- In spontaneously breathing nonobese persons, 6 cm head elevation with jaw closure is optimal for reducing pharyngeal collapsibility during propofol anesthesia.

THE maintenance of upper airway patency during anesthesia with spontaneous breathing is a critical issue because upper airway dilator muscle activity becomes significantly compromised in the absence of arousal responses.[1–3] During anesthesia or sleep, disturbances in upper airway neural control (*i.e.*, compensatory neuromuscular responses) may compromise pharyngeal patency.[4,5] Under these circumstances, mechanical upper airway properties predominate in governing the overall collapsibility of the airway. Thus, improving the mechanical characteristics of the airway during anesthesia is of prime importance to the maintenance of airway patency.

Jaw and head position are known to play an important role in the maintenance of upper airway patency[6,7] during anesthesia with spontaneous breathing, specifically head extension, flexion, and rotation.[6,8] Of the several positions, head elevation to a "sniff position" reduces upper airway

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Received from the Department of Clinical Physiology, Nagasaki University Graduate School of Biomedical Science, Nagasaki, Japan. Submitted for publication September 2, 2010. Accepted for publication April 7, 2011. Supported by institutional funding from Nagasaki University, Nagasaki, Japan; a grant from Suzuken Memorial Foundation, Osaka, Japan (to Dr. Moromugi); grant-in-aid No. 21592654 for scientific research from the Japanese Ministry of Education, Science, Sports and Culture, Tokyo, Japan (to Dr. Oi); and grant HL50381 (Mechanisms of Sleep Apnea in Severe Obesity) from the National Institutes of Health, Bethesda, Maryland (to Dr. Schwartz). Presented in part at the Annual Meeting of the American Society of Anesthesiologists, New Orleans, Louisiana, October 20, 2009.

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collapsibility and helps maintain airway patency during anesthesia and recovery.[9–15] Previous studies have suggested that head elevation using a 5- to 10-cm headrest can improve airway patency during the induction of anesthesia[9–15] in paralyzed subjects. However, opening of the jaw or accidental neck flexion can occur simultaneously with head elevation, which could attenuate the beneficial effects of head elevation.[6,16–19] Nevertheless, the effect of head elevation on passive upper airway collapsibility has not been quantified in anesthetized, spontaneously breathing subjects. No data are available to estimate the appropriate height to which the head should be increased to maintain upper airway patency during anesthesia and after surgery.

We hypothesized that the effect of head elevation on upper airway collapsibility might be impeded by jaw opening and neck flexion during propofol anesthesia. To address this hypothesis, we examined the effect of head elevation on passive upper airway collapsibility during propofol anesthesia in spontaneously breathing subjects. Specifically, we sought to establish the optimal height of head elevation for preventing upper airway obstruction by evaluating the influences of jaw opening and/or neck flexion on this effect on upper airway pressure-flow relationships.

Materials and Methods

Subjects

Twenty healthy male subjects were recruited, and a detailed clinical history was obtained. Subjects were excluded if they were overweight or obese (body mass index more than 25 kg/m^2), had a history of frequent or excessive snoring according to the bed-partner (greater than three times/week), had abnormal sleep patterns or reported excessive daytime sleepiness (Epworth Sleepiness Score more than 10), had significant medical disease (cardiopulmonary pathology) or other clinical history (allergy to anesthetic), or reported tobacco use or chronic alcohol or drug use. In addition, subjects were excluded if they had an anatomical deformation of the upper airway such as retrognathia or maxillary hypoplasia assessed by under conditions of jaw occlusion (normal overbite and overjet). The vertical occlusion condition was assessed by overbite, which indicates degree of overlap between upper incisor and lower incisor of 3 mm. The horizontal occlusion condition was assessed by overjet, which indicates horizontal distance between upper and lower incisor of 3 mm. All subjects had to have a Mallampati score of I or II and a thyromental distance longer than 60 mm. On enrollment in the study, subjects were randomly assigned to one of two groups (fixed-jaw or free-jaw); their demographic information is presented in table 1. The experimental protocol was approved by the Human Investigation Committee of the Nagasaki University School of Dentistry, and written informed consent was obtained from all subjects.

Table 1. Subject Demographics

	Fixed-jaw (n = 10)	Free-jaw (n = 10)
Age (yr)	24.2 ± 2.3	24.7 ± 2.6
Weight (kg)	62.3 ± 5.8	62.1 ± 5.2
Height (m)	1.73 ± 0.05	1.70 ± 0.02
BMI (kg/m^2)	20.8 ± 1.8	21.5 ± 1.5

Data are mean ± SD.
BMI = body mass index.

Experimental Techniques

Physiologic Measurements. All subjects underwent routine hemodynamic monitoring (systolic and diastolic blood pressure and pulse rate) and electrocardiogram, bilateral electrooculograms, electroencephalograms (C3-A2), and submental electromyogram to confirm anesthetic level. Electroencephalogram signals were processed by the Bispectral Index® monitor (Aspect Medical Systems, Natick, MA) to determine the depth of propofol anesthesia. Oxygen saturation (Spo_2) was measured by pulse oximetry. Airflow (V) was measured by a pneumotachometer (model 3830; Hans Rudolph, Kansas City, MO), and nasal pressure (P_N) was measured by a differential pressure transducer (model 1100; Hans Rudolph), both connected to the mask. To detect respiratory movement, both chest and abdominal movements were monitored with piezoelectric strain gauges.

A variable pressure device (ResMed, Bella Vista, Australia) was used to deliver constant P_N over the range from −15 to 15 cm H_2O. These measurements were displayed and stored simultaneously on a computer using a data acquisition device (Embla S7000 with Somnologica; Medcare, Broomfield, CO).

Propofol Anesthesia. No premedication was given. Propofol anesthesia was induced with intravenous propofol (Dipriva; Astra Zeneca, Alderley Park, Cheshire, United Kingdom), administered *via* a Diprifusor (Astra Zeneca) target-controlled infusion system (TCI pump TE-371; Terumo, Tokyo, Japan), which calculated the effect site concentration on the basis of a three-compartment pharmacokinetic algorithm.[1,20,21] The propofol target blood concentration was increased and kept constant between 1.5 and 2.0 μg/ml to obtain an adequate level of anesthesia (Bispectral Index® value between 40 and 60, no response to spoken command). At the conclusion of the measurements, all of the subjects were kept in the supine position until they showed a spontaneous emergence from anesthesia.

Evaluation of Passive Upper Airway Collapsibility (Passive P_{CRIT}). After the establishment of an adequate level of stable propofol anesthesia, the subjects initially breathed *via* the nasal mask at atmospheric pressure. P_N was then gradually increased to a holding pressure at which inspiratory airflow limitation was abolished ("passive state"), as previously described.[22,23] In brief, airflow limitation was defined as a plateau in the inspiratory airflow with continued respiratory effort. To establish the passive P_{CRIT}, P_N was rapidly lowered

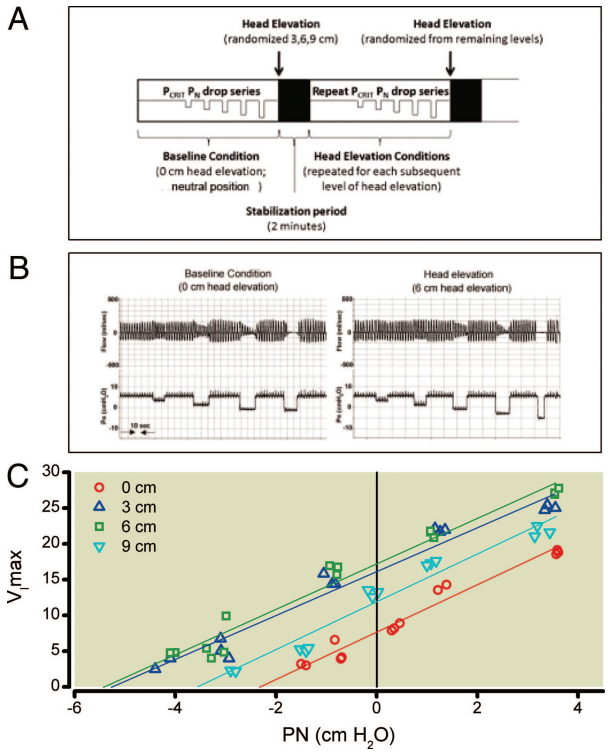

Fig. 1. A schematic diagram of the upper airway collapsibility measurements with head elevation (*A*). The raw data recording of upper airway collapsibility (passive P_{CRIT}) measurements with 6 cm head elevation in fixed-jaw condition in one subject is shown as nasal mask pressure (P_N) and Pneumotach airflow (Flow). P_N was abruptly reduced from an increased holding pressure to a level that induced inspiratory airflow limitation (flattened or nonsinusoidal shape in inflow). Subsequently, P_N was lowered in a stepwise fashion by 2 cm H_2O every five breaths until zero flow was obtained or Spo_2 reached a lower limit of 88–90%. Maximal inspiratory airflow ($V_I$max) was measured in the last three flow-limited inspirations at each level of nasal pressure. Note that P_N to obtain zero inspiratory airflow was lower in the 6 cm head elevation condition than the baseline condition (0 cm head elevation) (*B*). Example of pressure flow relationships in one subject in the fixed-jaw group at four levels of head elevation: 0 (*open red circle*), 3 (*open blue triangle*), 6 (*open green squares*) and 9 cm (*open inverted green triangle*) (*C*). The fit line for pressure-flow relationships was generated using linear regression analysis.

from the holding pressure to specific levels for five successive breaths (approximately 15–20 s) before being returned to the holding pressure (figs. 1A and B). A series of approximately 6–10 pressure drops (including P_N at lowest inflow or at zero flow: *i.e.*, airway occlusion) was obtained to generate a passive pressure-flow relationship for each level of head elevation. If any of the P_N drops were associated with transient arousal (Bispectral Index® more than 70) or an oxygen desaturation of less than 90%, P_N was returned to the holding pressure without reaching zero inflow, while subjects reestablished stable breathing, and then the series of pressure drops was continued.

Head Elevation. Each subject was positioned supine on a surgical bed with a motorized head platform (model EX-SP33, Takara Belmont Corp., Osaka, Japan). The vertical position of the head platform was set using a ruler positioned vertically and attached to the side of the surgical bed. The baseline head elevation condition was established when the head platform was at the same level as the bed (*i.e.*, 0 cm). Three-centimeter quanta (3, 6, and 9) of table elevation were used to examine the influence of head elevation on upper airway collapsibility.

Protocol

Each subject was asked to restrict food intake for 6 h before participating in the experimental measurements. Initially the monitoring sensors were attached to the subject and then were laid on a flat firm bed with no pillow. The subject's head was positioned in a neutral position with face straight up and with the individual's Frankfort plane angled at approximately 70–80 degrees to the horizontal plane of the bed (*i.e.*, without head extension or flexion). The initial Frankfurt plane and Mandible plane angle was then recorded using a custom-made protractor. The nasal mask was fitted over the subject's nose and checked for leaks by asking the subject to try to exhale through the mask while the airflow pathway was occluded. If air leaks were detected, the mask was repositioned and retested for leaks. To prevent air leaks, the subject's lips were sealed with flexible surgical tape without interfering with jaw opening.

The subjects were divided into two groups: fixed-jaw (n = 10) and free-jaw (n = 10). For the subjects in the fixed-jaw group, a chinstrap was applied to maintain centric occlusion of the teeth. For the subjects in the free-jaw group, free movement of the mandible was allowed without a chinstrap.

Once steady state anesthesia was attained, pressure-flow measurements were conducted at each of the four head elevation levels in random order at approximately 10-min intervals. On completion of the measurements, the anesthetic was withdrawn and subjects continued to be monitored while spontaneously emerging from anesthesia for 2 h after the study period.

Data Analysis

Upper Airway Pressure-flow Relationship. At each level of P_N, breaths were evaluated for the presence of inspiratory airflow limitation, as previously described.[22–25] Inspiratory flow limitation was defined as the presence of a flattened or nonsinusoidal appearance on the inspiratory inflow signal[26,27] that ended abruptly with a return to nonflow limited breaths with sinusoidal shape when the P_N was increased to the holding pressure. Breaths that were associated with arousal were excluded from analysis. Maximal inspiratory airflow ($V_I$max) was measured in the last three flow-limited inspirations at each level of nasal pressure, as previously described,[22] and used to define the corresponding P_N *versus* $V_I$max relationship (fig. 1C). Least-squares linear regression

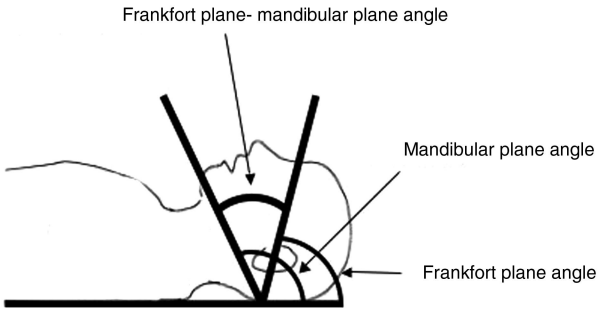

Fig. 2. Diagram of experimental protocol and indication of Frankfort plane angle and mandible plane angle.

was used to generate the pressure-flow relationship[28] and fit with the following equation:

$$\dot{V}_I\text{max} = (P_N - P_{CRIT})/R_{US}$$

where P_{CRIT} was the critical closing pressure (P_N at zero flow) and R_{US} was the resistance of the portion of the airway upstream of the site of collapse.

Measurement of Neck Flexion (Frankfurt Plane) and Jaw Opening (Mandible Plane). The Frankfort plane was defined as a plane passing through the inferior margin of either orbit (orbitale) and the upper margin of each ear canal (porion) (fig. 2). The degree of head flexion was assessed by measuring the angle between the Frankfort plane and the horizontal plane of the bed. The mandible plane was defined as a plane passing through the lower border of the ramus of the mandible from the edge of the mandible angle and tip of the mandible. The Frankfurt plane angle was used to indicate head position: a decrease showed head flexion and an increase signified head extension. The degree of jaw opening was defined by the change of angle between Frankfort plane and mandible plane relative to the occluded condition. Each plane angle was directly measured with reference to the horizontal plane using a custom-made protractor placed alongside the subject's head and mandible before and after changing of the head height. These angles were reconfirmed using a protractor on a photograph of the lateral view, which was taken with the subject in each head position.

Sample Size Analysis. Before the experiment, we calculated the sample size estimation using a statistical tool (StatMate2; GraphPad Software, LaJolla, CA) to determine how much difference in passive P_{CRIT} is clinically significant. Estimates of mean and SD values for passive P_{CRIT} by head extension during midazolam anesthesia were 4.3 ± 1.4 cm H_2O from data obtained in a previous study in our laboratory.[8] Based on the performance characteristics of repeated measurements of P_{CRIT},[29] we estimated that a sample size of 10 subjects in each group (fixed-jaw and free-jaw) would have 90% power to detect a difference in means of passive P_{CRIT} of from 3 to 5 cm H_2O using a nonpaired *t* test with a 0.05 two-sided significance level.

Statistical Analysis. All statistical analyses were performed using Prism5 (GraphPad Software) to test a two-tailed hypothesis. To examine the effect of head elevation on the primary outcomes variables (passive P_{CRIT}, Frankfort plane angle; and mandible plane angle), we used ANOVA for repeated measurement. To examine whether there was a difference in the effect of head elevation between the fixed-jaw and free-jaw groups, we used two-way ANOVA for repeated measures. When significant differences were detected, a *post hoc* protected Dunnett test was used to isolate the differences. Secondary outcomes analysis was performed on R_{US} and the difference in angle between Frankfort plane and mandible plane using the Mann–Whitney test (data not normally distributed). Statistical significance was assumed for $P < 0.05$. The data are presented as mean ± SD unless otherwise noted.

Table 2. Experimental Variables

	Fixed-jaw	Free-jaw
Propofol (μg/ml)	1.75 ± 0.11	1.78 ± 0.10
BIS value (au)	48.8 ± 5.6	45.3 ± 1.4
Baseline Spo_2 (%)	97.1 ± 0.7	97.5 ± 0.5
Lowest Spo_2 (%)	90.8 ± 1.3	89.6 ± 1.5

Data are mean ± SD.
au = arbitrary units; BIS = Bispectral Index®; Spo_2 = oxygen saturation.

Results

The demographic and anthropometric characteristics of the subjects in the fixed-jaw and free-jaw groups are shown in table 1. There were no group mean differences in the age, height, weight, or body mass index (all $P > 0.3$; *t* tests). The experimental conditions for each of the groups were similar in the level of mean target blood concentration of propofol, Bispectral Index® value, and baseline and lowest Spo_2 (all $P > 0.2$; *t* tests; table 2).

There was no difference in mean holding pressure required to abolish flow limitation between the fixed-jaw (6.3 ± 1.4 cm H_2O) and free-jaw (6.9 ± 0.9 cm H_2O; $P = 0.14$) groups. For all subjects, the average number of pressure drops in each experimental condition was 8 (range, 6–10). The range of P_N applied across all conditions was 6.3 ± 1.4 cm H_2O to −3.9 ± 2.6 cm H_2O in the fixed-jaw group and 6.9 ± 0.9 cm H_2O to −0.8 ± 1.8 cm H_2O in the free-jaw group.

Effect of Head Elevation on Upper Airway Collapsibility

Figure 3 shows the mean passive P_{CRIT} at each level of head elevation (0, 3, 6, and 9 cm). In the baseline condition (0 cm head elevation) and 3 cm head elevation, P_{CRIT} was similar between the fixed-jaw and free-jaw groups ($P = 0.41$ and $P = 0.34$, respectively; two-way ANOVA; table 3). There was significant difference of P_{CRIT} between the fixed-jaw and free-jaw groups at 6 ($P = 0.006$; two-way ANOVA; table 3) and 9 cm head elevation ($P = 0.02$; two-way ANOVA; table 3). In the fixed-jaw group, there was a significant decrease in

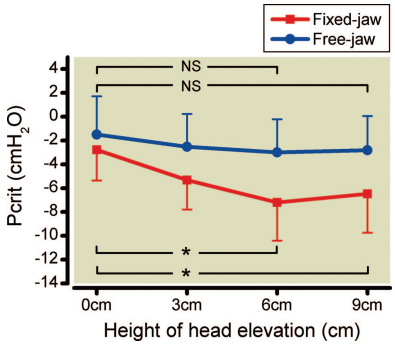

Fig. 3. The change of mean upper airway collapsibility (passive P_{CRIT}) at four different heights of head elevation in the fixed-jaw and free-jaw conditions. * $P < 0.05$ *versus* neutral position (0 cm head elevation). NS = nonsignificance with $P > 0.05$.

P_{CRIT} (less collapsible) with 6 cm or more head elevation ($P < 0.0001$; one-way ANOVA). In the free-jaw group, there was no change in P_{CRIT} at any level of head elevation ($P > 0.2$; one-way ANOVA). In the baseline condition R_{US} in the fixed-jaw group (37.6 ± 15.5 cm $H_2O \cdot ml^{-1} \cdot s$) was not different from the free-jaw group (49.6 ± 14.6 cm $H_2O \cdot ml^{-1} \cdot s$; $P > 0.3$) two-way ANOVA). R_{US} did not change with head elevation in either group ($P > 0.1$; one-way ANOVA).

Effect of Head Elevation on Head Flexion and Jaw Opening

In the baseline condition, the Frankfort angle was similar in the fixed-jaw (78 ± 1 degree) and free-jaw groups (77 ± 1 degree; $P = 0.53$; two-way ANOVA). Every 3-cm head elevation increment increased the Frankfort angle (head flexion) by approximately 3 degrees in both groups ($P < 0.0001$; one-way ANOVA; fig. 4A). In the baseline condition, the mandibular angle was similar in the fixed-jaw (120 ± 1 degree) and the free-jaw groups (121 ± 1 degree; $P = 0.77$; two-way ANOVA). Each progressive 3-cm head elevation increment increased the mandibular angle by approximately 2 degrees in the fixed-jaw group and by approximately 6 degrees in the free-jaw group (*i.e.*, there was significant jaw opening with head elevation in the free-jaw group; figs. 4B and C). Figures 5A and B shows the degree of jaw opening evaluated by the change of δ angle between the Frankfort plane and mandible plane relative to the occluded condition. Each progressive 3-cm head elevation increment increased the δ angle by approximately 2–3 degrees in the free-jaw group. There was a significant increase in δ angle with 9 cm of head elevation ($P = 0.004$; one-way ANOVA) compared with the baseline condition.

Table 3. Effect of Head Elevation on Upper Airway Collapsibility (P_{CRIT})

Elevation (cm)	Fixed-jaw	Free-jaw	*P* Value
Baseline (0)	−2.8 ± 2.6	−1.5 ± 3.2	0.41
3	−5.3 ± 2.5	−2.5 ± 2.8	0.34
6	−7.2 ± 3.2	−3.0 ± 2.8	0.006
9	−6.5 ± 3.3	−2.8 ± 2.8	0.02

Values are P_{CRIT}; data are mean ± SD. *P* values are fixed-jaw group compared with free-jaw group using two-way ANOVA.

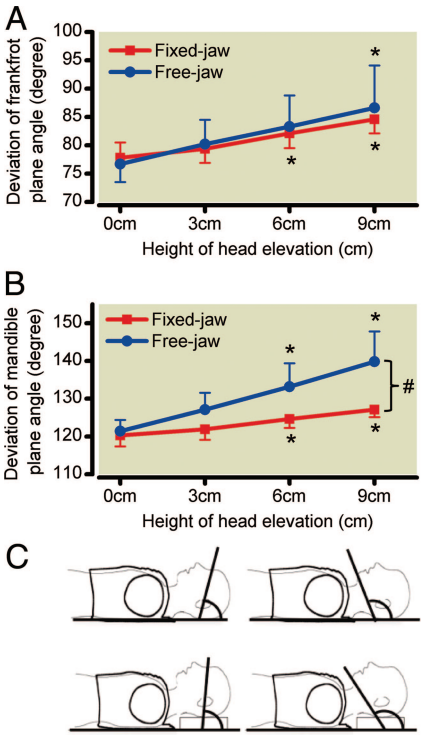

Fig. 4. The mean deviation of the Frankfort plane angle (*A*) and the mandible plane angle (*B*) from neutral position (0 cm head elevation) in the fixed-jaw and free-jaw groups. * $P < 0.05$ *versus* neutral position (0 cm head elevation). The condition main effect (# $P < 0.05$ fixed-jaw *vs.* free-jaw). Diagram of head elevation associated with increase in Frankfort plane angle and mandible plane angle from neutral position (0 cm head elevation) (*C*).

Discussion

The major finding of this study is that head elevation significantly reduces passive upper airway collapsibility during spontaneous breathing during propofol anesthesia. This study also indicates that the beneficial effect of head elevation on collapsibility is limited by opening of the jaw and by the degree of neck flexion associated with head elevation. There may be a critical threshold of head elevation for maintaining upper airway patency beyond which the beneficial effect is offset by head flexion and mouth opening. The estimated optimal head elevation appeared to be approximately 6 cm in our subjects when centric dental occlusion was maintained. Our findings suggest that manipulating the head position while ensuring mouth closure (centric occlusion) produces substantial decreases in upper airway collapsibility and maintains upper airway patency during anesthesia induction and recovery.

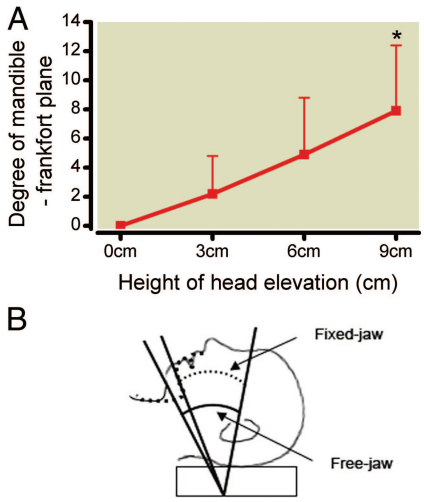

Fig. 5. The mean deviation of δ angle between Frankfort plane and mandible plane relative to the occluded condition in the free-jaw group. * $P < 0.05$ *versus* baseline position (0 cm head elevation) (*A*). Diagram of fixed-jaw and free-jaw position associated with increase in δ angle between Frankfort plane angle and mandible plane angle (*B*).

Validation of Passive Upper Airway Collapsibility

Our study follows previous work demonstrating substantial increases in pharyngeal collapsibility[30] in spontaneously breathing patients during sedation and anesthesia.[31,32] We recently reported that the precision of repeated measurements of passive P_{CRIT} is approximately 1 cm H_2O during hypnotic induced sleep, which would be similar to the findings of the current study.[29] Although anesthesia can produce elevations in upper airway collapsibility,[1,33,34] we found that the passive P_{CRIT} (−2.8 cm H_2O) in the neutral position during propofol anesthesia is comparable with that reported in a previous study of propofol anesthesia by Walsh *et al.* (−0.4 cm H_2O)[6] and is higher than the passive P_{CRIT} found during natural nonrapid eye movement stage 2 sleep (passive P_{CRIT} = −4.5 cm H_2O) in normal subjects.[30,35] It has been shown that neuromuscular responses to negative airway pressure and upper airway obstruction during propofol anesthesia were significantly depressed in spontaneously breathing rat[36] and human subjects.[1] In addition, we previously demonstrated that defects in upper airway mechanical properties and compensatory neuromuscular responses to upper airway obstruction are necessary for the development of sustained obstruction, based on measurements of passive P_{CRIT} and active P_{CRIT} (under conditions of increased genioglossus muscle activity) during propofol anesthesia.[3] Although we still do not fully understand the mechanisms of upper airway obstruction during anesthesia, anatomical imbalance in the pharyngeal airway, lung volume reduction caused by positional change, and breathing instability associated with alteration of inspiratory airflow during anesthesia may contribute significantly to the development and deterioration of sustained upper airway obstruction. We acknowledge the existence of neural mechanisms in our experimental model that are not present in other passive airway models used with general anesthesia and complete paralysis.[34] However, we think our experimental model may be suitable for assessing mechanical properties and can provide an estimate of the mechanical properties of the pharynx when neuromuscular activity in upper airway dilator muscles is minimal.[1]

Influence of Optimal Head Elevation on Passive Upper Airway Collapsibility

This study quantifies the influence of head elevation on passive upper airway collapsibility in spontaneously breathing subjects during propofol anesthesia. P_{CRIT} significantly decreased at 6 and 9 cm head elevation compared with the neutral position when the jaw was closed (fig. 3). Nevertheless, no additional changes in P_{CRIT} were observed beyond 6 cm elevation, suggesting that an optimal head position exists for the maintenance of airway patency, provided that jaw opening is prevented. Moreover, P_{CRIT} fell by 4.4 cm H_2O, suggesting a substantial reduction in passive mechanical properties of the pharynx.[3,5] Reductions in P_{CRIT} of this magnitude are equivalent to the stabilizing effect of applying nearly 5 cm H_2O of continuous positive airway pressure to reverse upper airway obstruction during anesthesia. Such decreases in P_{CRIT} also approximate the magnitude of the response required to convert obstructive events to less severe hypopneic events or hypopneic events to stable breathing during anesthesia and sleep,[6,8] suggesting head elevation as a source of variability in sleep apnea severity throughout the night.

Although we found the optimal height of head elevation to be approximately 6 cm during anesthesia with spontaneous breathing, it is difficult to estimate exactly what height each patient requires under different clinical situations because there are no quantitative data to help determine the optimal height of head elevation. However, previous studies performed in paralyzed subjects may provide useful information to predict the suitable height. Adnet *et al.* also indicated that the sniffing position (head increased by approximately 8 cm, with the face straight up, achieved by placing cushions under the head) might be hypothetically obtained by flexing the neck on the chest and elevating the head approximately 7–10 cm with a pad under the occiput in a magnetic resonance imaging study.[15] They concluded the underlying mechanism of cervical extension by head elevation with jaw closure produced an increase in the distance between the mentum and cervical column.[37] Isono *et al.* provided additional information in a study of general anesthesia with paralysis, suggesting that the sniffing position improves patency of the passive pharyngeal airway in patients with obstructive sleep apnea.[11] In that study, they postulated that an increase in bony enclosure size and improvement of anatomical balance contribute to maintaining upper airway patency during anesthesia. Therefore, we speculate that a similar mechanism may explain an improvement of upper

airway collapsibility by head elevation with jaw closure during propofol anesthesia with spontaneous breathing.

Upper airway mechanical properties are affected by the type and dose of anesthetic. It is well recognized that a reduction of tonic activity of dilator muscles may be directly affected by the depth of anesthesia and the different anesthetic agent used.[1,2] It has been reported that passive P_{CRIT} differs depending on the anesthetic used[8,36,38] because such agents likely differ in the ways they depress tonic dilator muscle activity during anesthesia. For example, midazolam and propofol had similar dose-dependent effects on upper airway mechanical properties.[1,39] Eikermann *et al.* suggested that pentobarbital increases the respiratory activity of the genioglossus muscle in a dose-dependent manner while impairing diaphragmatic function in anesthetized rats.[40] Thus, it is possible that the optimal height of head elevation varies, depending on the type and dose of anesthetic. A recent study by Herbstreit *et al.*[41] suggested that the residual effects of neuromuscular blockade increase airway collapsibility and blunt genioglossus muscle activity in response to negative pharyngeal pressure. This finding strongly suggests that the optimal height of head elevation in the recovery unit may depend on the time course of recovery from neuromuscular blockade and anesthetic agents.

It should be mentioned that the lack of change in R_{US} reported for both the conditions in our experiment may reflect a relatively constant airway size at the different amounts of head elevation.[22] The most likely explanation for this finding is that the upper airway segment upstream from the site of collapse may not influence the effects of head elevation on upper airway collapsibility resulting from depression of dilator muscle activity associated with propofol anesthesia. The velopharyngeal segment of the upper airway is particularly prone to collapse and has been found to be the predominant flow-limiting site during sedation[33,38,42] and anesthesia.[34] In the current study, we conclude that head elevation appears to modulate the collapsibility of the pharynx without altering the caliber or patency of the upstream segment.

Influence of Neck Flexion and Jaw Opening on Upper Airway Collapsibility

Our data also show that neck flexion occurs as the head elevation is increased. Head elevation with a pillow appears to improve the pharyngeal patency in a height-dependent manner, but previous studies suggest that concomitant neck flexion is likely to attenuate the beneficial effects[11,13] on pharyngeal size and passive P_{CRIT} in anesthetized patients.[6,18] Walsh *et al.*[6] reported that neck flexion with a 10-degree deviation from the neutral position produced a 4.9-cmH_20 increase in passive P_{CRIT} during propofol anesthesia, suggesting that the 4.3-degree change in neck flexion induced by head elevation produced an offset in P_{CRIT} of approximately 2.1 cm H_2O.

We also evaluated the effect of concomitant jaw opening on passive P_{CRIT}, based on the changes in the Frankfort and mandibular plane angles with head elevation, and found that there were no significant differences in the change in the Frankfort plane angle between the fixed-jaw and free-jaw conditions. In contrast, there was a significantly greater change in the mandibular plane angle in the free-jaw position relative to the fixed-jaw positions. These findings suggest that head elevation is associated with some degree of jaw opening, which increases in a height-dependent manner with head elevation.

The mechanism underlying the change in upper airway collapsibility associated with head flexion and jaw opening might relate to a decrease in tracheal length and traction by flexion[6] and displacement of the tongue by jaw opening.[18,24] These anatomical influences promote the alteration of extraluminal surrounding tissue pressure in the upper airway segment and increase upper airway collapsibility by changing the transmural pressure.

Possible Limitations of the Current Study

There are several limitations in this study. First, we did not evaluate responses to head elevation in obese subjects in this study. Levitan *et al.*[14] describes the use of a single standard pillow size, which did not always provide optimal cervical flexion for all subjects because of modest variations in weight, head circumference, and length of the neck. We suspect that obese individuals might require more head elevation than do normal-weight individuals to produce a comparable amount of head elevation because of greater amounts of body fat on the back and shoulders. Although the issue was not addressed in the current study, we also suspect that the optimal head elevation might be greater in large-built subjects or those of taller stature. Second, we acknowledge that head elevation is not the only method of optimizing airway patency clinically, and we did not evaluate other methods for maintaining airway patency. Adnet *et al.*[37] reported that the sniffing position with head elevation offered no appreciable advantage over simple head extension for improvement of glottic visualization during direct laryngoscopy. Therefore, it would be useful to compare head elevation with other methods, such as simple head extension. Third, it is possible that we have underestimated the influence of jaw opening because the surgical tape placed over the subject's lips to prevent air leakage through the mouth may have limited the opening to some extent. Fourth, our findings cannot be generalized to the population at large despite that our study design was powered to detect both clinically and statistically significant differences in a relatively homogeneous cohort study.

Clinical Implications

The findings of this study have substantial clinical implications. First, we observed that the change in the mandibular plane angle was greater than the change in the Frankfort plane angle, suggesting that head elevation is associated with jaw opening, which is likely to be an important independent determinant of upper airway patency. Preventing jaw open-

ing (*e.g.*, with a chin strap or a cervicomandibular support collar[43]) can help to maximize the benefits of head elevation. It is known that maintaining the sniffing position by a handling maneuver by the anesthesiologist is one of the best options for maintenance of upper airway patency. However, we suggest that head elevation with jaw closure may offer an alternative method that does not require direct handling. These considerations also have implications for optimizing head and neck posture for fiberoptic intubation and during magnetic resonance imaging of sedated patients in determining the best use of pillows and optimal head position.

Second, these findings may have implications for positional therapy for obstructive sleep apnea (OSA) patients during sleep and anesthesia. Because the passive collapsibility of the upper airway during propofol anesthesia is similar to passive airway collapsibility during nonrapid eye movement sleep,[3,33] our passive P_{CRIT} data simulate the mechanical influences of head posture during sleep. Head position during sleep is mainly determined by appropriate height and placement of the pillow under the head. Our data clearly show that excessive pillow height promotes head flexion associated with jaw opening. Such changes during sleep could lead to the development or worsening of upper airway obstruction in OSA patients. It has been suggested that producing head extension with a cervical pillow may improve mild but not severe OSA.[44] It is possible that jaw opening during head extension might offset any potential improvement in upper airway patency. We speculate that the combination of head elevation and head extension with jaw closure can decrease upper airway collapsibility during sleep. Additional study is needed to test the appropriate combination of head posture and jaw position during sleep in OSA patients.

Third, our findings may help in estimating the risk of airway management during the postoperative period in OSA patients. In this study, we found that the maximum improvement induced by head elevation in the fixed-jaw condition was a passive P_{CRIT} of approximately 4.3 cm H_2O at 6 cm head elevation. We speculate that if patients have greater airway collapsibility with a passive P_{CRIT} of greater than 5 cmH_20, for example in OSA or obese patients, head elevation may not be effective in maintaining upper airway patency. Therefore, host factors may influence the optimal height and clinical efficacy of head elevation, depending on the degree of jaw opening and neck flexion.

Conclusion

This study indicates that head elevation is useful in the maintenance of airway patency during anesthesia and that there may be a critical threshold in the degree of elevation for this maneuver to be effective. Jaw opening and neck flexion can attenuate these beneficial effects. We suggest that the optimal height of head elevation in normal-weight subjects during propofol anesthesia with spontaneous breathing is approximately 6 cm with jaw closure. This information is directly relevant to managing upper airway patency during anesthesia with spontaneous breathing and recovery from it.

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