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Pharyngeal Patency in Response to Advancement of the Mandible in Obese Anesthetized Persons

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Background: **During anesthesia in humans, anterior displacement of the mandible is often helpful to relieve airway obstruction. However, it appears to be less useful in obese patients. The authors tested the possibility that obesity limits the effectiveness of the maneuver.**

Methods: **Total muscle paralysis was induced under general anesthesia in a group of obese persons (n = 9; body mass index, 32 ± 3 kg^{-2}) and in a group of nonobese persons (n = 9; body mas index, 21 ± 2 kg^{-2}). Nocturnal oximetry confirmed that none of them had sleep-disordered breathing. The cross-sectional area of the pharynx was measured endoscopically at different static airway pressures. A static pressure–area plot allowed assessment of the mechanical properties of the pharynx. The influence of mandibular advancement on airway patency was assessed by comparing the static pressure–area relation with and without the maneuver in obese and nonobese persons.**

Results: **Mandibular advancement increased the retroglossal area at a given pharyngeal pressure, and mandibular advancement increased the retropalatal area in nonobese but not in obese persons at a given pharyngeal pressure.**

Conclusion: **Mandibular advancement did not improve the retropalatal airway in obese persons. (Key words: Airway: management; obstruction; mandibular advancement. Anesthesia: general.)**

LOSS of consciousness leads to pharyngeal narrowing or closure in association with reduction of upper airway muscle activities.[1,2] Forward displacement of the mandible improves such pharyngeal occlusion in comatose and anesthetized patients.[3,4] Recently, this procedure was successfully applied to treat obstructive sleep apnea (OSA).[5] We have demonstrated that anterior movement of the mandible widens the retropalatal airway and the retroglossal airway in anesthetized patients who have OSA.[6] However, it was also noted that the response to the maneuver varied among the patients and between the pharyngeal segments. This corresponds with our clinical experiences in which we often encounter a difficulty in the management of airway patency during induction of anesthesia. Safar *et al.*[4] reported that a combination of neck extension and elevation of the mandible was not effective to restore a clear upper airway in obese anesthetized persons. Body size appears to be a significant factor determining the response to mandibular advancement to maintain airway patency.

Both neuromuscular and anatomic factors influence pharyngeal patency.[7,8] Although mandibular advancement may modulate the neuromuscular and anatomic factors, the present study was designed to investigate effects of mandibular advancement on the intrinsic mechanical properties of the pharynx by eliminating neuromuscular factors. We induced total muscle paralysis under general anesthesia in a group of obese persons and in a group of persons who were not obese. Static mechanical properties of the pharynx were evaluated by the pressure-area relation of the pharyngeal segment, as we previously reported.[9] Accordingly, the purpose of the present study was to test the hypothesis that obese persons do not respond to mandibular advancement as effectively as nonobese persons do. Therefore we examined the influence of the mandibular advancement on pharyngeal patency by comparing the static pressure-area relation of the passive pharynx with and without the maneuver in obese and nonobese persons.

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Materials and Methods

Participants

We studied 9 obese and 9 age-matched nonobese persons without sleep-disordered breathing. Obese participants who had a body mass index >28 kg/m^2 were recruited from patients scheduled for elective surgery at Chiba University Hospital. Age-matched nonobese controls were also selected. Nocturnal disordered breathing was evaluated by a pulse oximeter (Pulsox-5; Minolta, Tokyo, Japan) in all participants. A computer calculated oximetry parameters, such as oxygen saturation index, which was defined as the number of oxygen desaturation exceeding 4% from the baseline, and the percentage of time spent at oxygen saturation <90% (CT_{90}). Patients who had oxygen desaturation indexes <5 and CT_{90} <1% were considered not to have sleep-disordered breathing and were included in the study.[10] None of them had clinical symptoms suggesting OSA. Individual characteristics and results of nocturnal oximetry are presented in table 1. The body mass indices were 31.3 ± 2.5 and 21.1 ± 2 kg^{-2} in obese and nonobese participants, respectively. Age and height did not differ between the groups. The aim and potential risks of the study were fully explained, and informed consent was obtained from each person. The investigation was approved by the hospital ethics committee.

Preparation of the Participants

Figure 1 illustrates experimental settings. Each participant was initially premedicated with 0.5 mg atropine. Studies were performed with the participants in a supine position on an operating table, with the neck in a comfortable neutral position. A modified tight-fitting nasal anesthetic mask was used. Care was taken to prevent air leaks in the mask, particularly when the airway was pressurized above 20 cm H_2O. Participants' mouths were closed by chin straps. General anesthesia was induced by intravenous administration of thiopental sodium (4 mg/kg). Intravenous administration of muscle relaxant (0.2 mg/kg vecuronium) induced complete paralysis throughout the experiment. Anesthesia was maintained by inhalation of sevoflurane (2–4%) while the participants were ventilated with positive pressure using an anesthetic machine. Oxygen saturation, electric activity of the heart, and blood pressure were continuously monitored. A slim endoscope (3-mm outer diameter; FB10X; Pentax, Tokyo, Japan) was inserted through the modified nasal mask and the naris. The tip of the scope was placed at the upper airway to visualize the velopharynx (retropalatal space) and the oropharynx (retroglossal space). A closed-circuit camera (ETV8; Nisco, Saitama, Japan) was connected to the endoscope, and the pharyngeal images were recorded on a videotape. Airway pressure (P_{AW}) readings, measured by a water manometer, were recorded simultaneously on the videotape. The experiment was completed before the start of any surgical procedure.

Experimental Procedure

To determine the pressure–area relation of the pharynx, the anesthetic machine was disconnected from the nasal mask. The latter was in turn connected to a pressure-control system capable of accurately manipulating P_{AW} from −20 to 20 cm H_2O in a stepwise manner. Cessation of mechanical ventilation resulted in apnea due to complete muscle paralysis. The P_{AW} was immediately increased to 20 cm H_2O by dilating the airway. While the participants remained apneic for 2–3 min, P_{AW} was gradually reduced from 20 cm H_2O to a velopharyngeal closing pressure in a stepwise manner. The latter represented the pressure at which complete closure of the velopharynx occurred, as evident on the video screen (figure 2). The apneic test was stopped when oxygen saturation decreased to <95%. This procedure of experimentally induced apnea allowed construction of the pressure–area relation of the visualized pharyngeal segment. Measurements were made for the velopharyngeal and the oropharyngeal airways with and without mandibular advancement. The maneuver was achieved manually by a skilled anesthesiologist, who placed the forefingers and the second fingers behind the angle of the mandible while placing the thumbs on the maxilla, so that forward motion of the mandible could be applied. The mouth was kept closed and care was taken to maintain a neutral neck position during the procedure. The participants were manually ventilated for at least 1 min before and after the apneic test. Distance between the tip of the endoscope and the narrowing site was measured with a wire passed through the aspiration channel of the endoscope.

Data Analysis

To convert the image on the monitor to an absolute value of cross-sectional area of the pharynx, magnification of the imaging system was estimated for every 1-mm distance between the tip of the endoscope and an object in a range of 10–30 mm. The pharyngeal image on the monitor at a determined P_{AW} was outlined on a tracing paper (50 g/m^2). The paper was cut along

Table 1. Individual Characteristics and Results of Nocturnal Oximetry

Subject	Sex	Age (yr)	Height (m)	Weight (kg)	BMI (kg/m^2)	ODI (h^{-1})	CT_{90} (%)
Obese group							
1	M	43	1.73	85.0	28.4	4.2	0
2	M	18	1.75	107.0	34.9	3.7	0
3	M	31	1.72	93.0	31.4	1.6	0
4	M	37	1.70	98.0	33.9	4.4	0
5	F	49	1.47	72.0	33.3	2.1	0
6	M	34	1.75	86.4	28.2	5.0	0
7	M	58	1.55	69.0	28.7	1.4	0
8	F	49	1.63	85.0	32.0	1.2	0
9	M	35	1.58	78.0	31.2	3.9	0
Mean		39	1.65	85.9	31.3	3.1	0
SD		12	0.10	12.2	2.5	1.5	0
Nonobese group							
10	M	58	1.61	60.8	23.5	0.3	0
11	F	46	1.51	48.2	21.1	1.0	0
12	M	42	1.65	60.0	22.0	3.4	0
13	M	41	1.73	65.0	21.7	1.2	0
14	M	26	1.75	55.5	18.1	0.4	0
15	M	25	1.61	52.0	20.1	0.8	0
16	M	46	1.65	58.0	21.3	2.2	0
17	M	27	1.65	65.0	23.9	0.4	0
18	F	42	1.64	50.0	18.6	0	0
Mean		39	1.64	57.2*	21.1*	1.1*	0
SD		11	0.07	6.2	2.0	1.1	0

BMI = body mass index; ODI = oxygen desaturation index defined as the number of oxygen desaturation exceeding 4% from the baseline; CT_{90} = the percent of time spent at Sp_{O_2} less than 90%.

* $P < 0.01$ *versus* obese group.

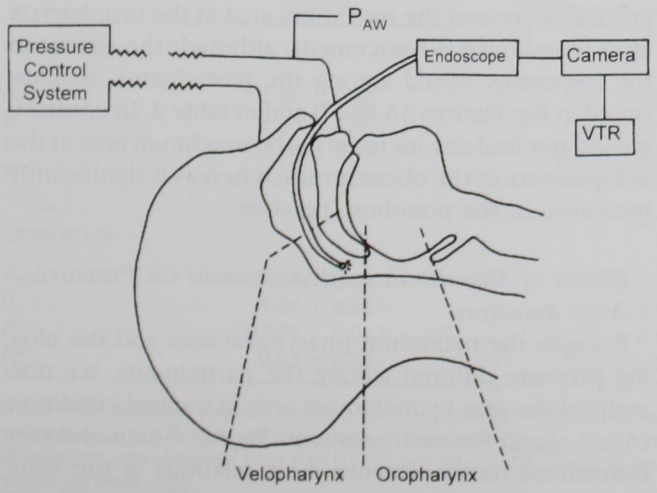

Fig. 1. Experimental settings. P_{AW}, airway pressure; VTR, videotape recorder.

the trace, and the weight of the paper was measured (ER120A scale; AND, Tokyo, Japan). The weight was converted to square centimeters of the pharyngeal area according to the magnification. For a constant distance, the area measurements were validated to be accurate within 8% ($-0.1 \pm 4.6\%$; range, +6.5 to -7.6%) by known-diameter tubes (4 to 9 mm inner diameter).

The measured luminal cross-sectional area was plotted as a function of P_{AW}. At high values of P_{AW}, the cross-sectional area became relatively constant and maximum area (A_{max}) was determined as mean values of measured area at the three highest P_{AW}s (18, 19, and 20 cm H_2O). As reported previously,[9] the pressure-area relation of each pharyngeal segment was fitted by an exponential function, $A = A_{max} - B \cdot \exp(-K \cdot P_{AW})$, where B and K are constants. A nonlinear least-squares technique was used to fit the curves, and the quality of the fitting was provided by coefficient R^2 (SigmaPlot version 2.0;

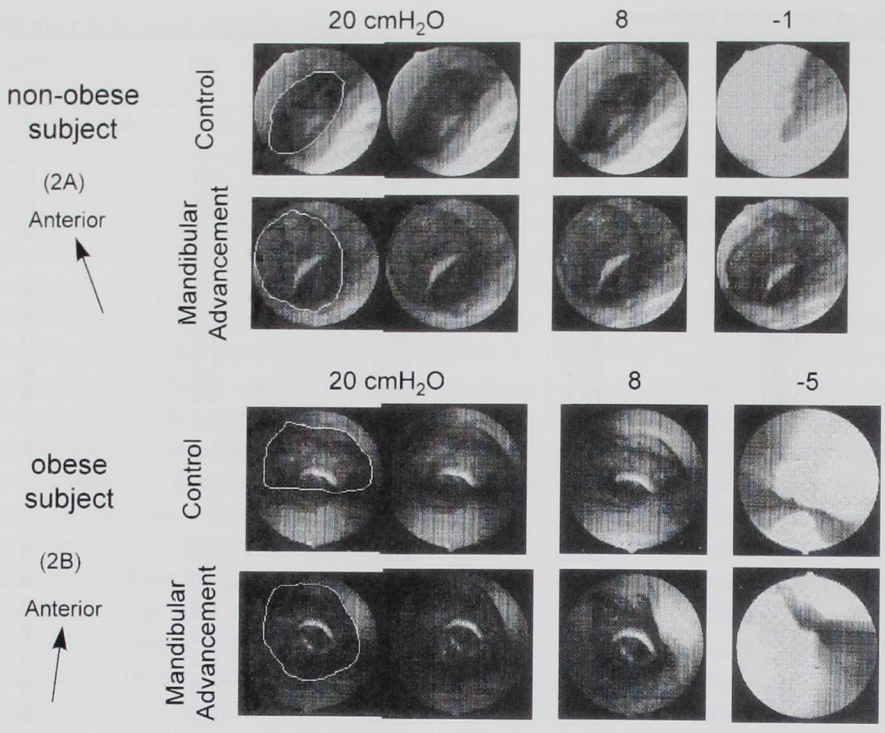

Fig. 2. Representative velopharyngeal images in nonobese (*A*) and obese (*B*) persons.

Jandel Scientific Software, San Rafael, CA). A regressional estimate of closing pressure (P'_{close}) was calculated from the following equation for each pharyngeal segment: $P'_{close} = \ln(B/A_{max})K^{-1}$. The value of K described the shape of the curve independent of maximum area. Stiffness of the pharyngeal segment is considered to increase with decreasing K values.

Statistical Analysis

All values are expressed as means ± SD, except in the figures. Statistical analysis was performed using a paired *t* test to examine the effect of mandibular advancement in the same group, and an unpaired *t* test was used to compare parameters between the groups. A probability value <0.05 was considered significant.

Results

Figure 2 shows representative velopharyngeal images in a nonobese (figure 2A) and in an obese (figure 2B) patient. Mandibular advancement increased the cross-sectional area of the velopharynx for all the airway pressures, and the airway was open widely even at the control closing pressure in the nonobese patient. In contrast, the same maneuver did not improve airway patency in the obese person. It is noteworthy that mandibular advancement improved the anterior-posterior dimension, but lateral narrowing is prominent in the obese patient.

Effects of Mandibular Advancement on Maximum Pharyngeal Area

All the participants, both in the obese and nonobese groups, increased the maximum area at the oropharynx after mandibular advancement, although the extent of the responses varied among the participants, as illustrated in the Figures 3A and B and in table 2. In contrast, we did not find any increase in the maximum area at the velopharynx in the obese group, whereas it significantly increased in the nonobese persons.

Effects of Mandibular Advancement on Pressure-Area Relations

Because the maximum pharyngeal area and the closing pressure differed among the participants, we normalized the area by maximum area in control condition ($A_{max - control}$) for each segment. Figure 4 demonstrates normalized mean pressure-area relations at the velopharynx (figure 4A and C) and the oropharynx (figure 4B and 4D) with (closed symbols) and without mandib-

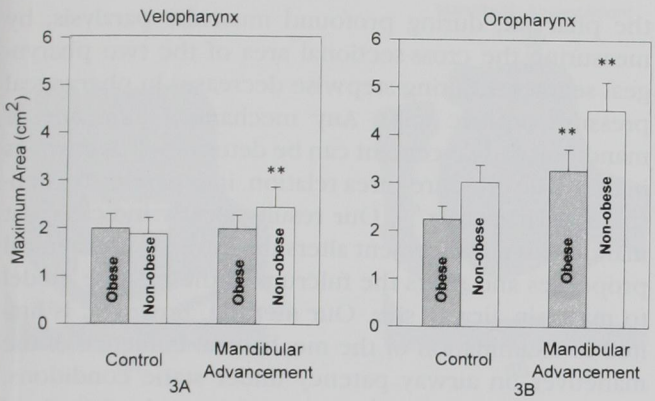

Fig. 3. Effects of mandibular advancement on maximum area at the velopharynx (*A*) and at the oropharynx (*B*). ** = $P < 0.05$ vs control. Values are means ± SE.

ular advancement (open symbols) for the obese group (upper panel) and the nonobese group (lower panel). The difference between P_{AW} and velopharyngeal closing pressure ($P_{close - VP}$) was used as the independent variable on the abscissa. As clearly shown in the figures, the maneuver shifted the pressure-area curves above those of controls at the oropharynx both in the obese and nonobese groups, whereas the velopharyngeal pressure-area curves were shifted only in the nonobese persons. The extent of the upward shift did not differ between the groups at the oropharynx.

Results of the Curve-fitting Analysis

Pressure-area relations were reasonably well fitted by the exponential function. Table 2 shows the means of the fitted exponential function. It is noteworthy that the constant K values significantly decreased at the velopharynx in the nonobese group. This indicates that the mandibular advancement increases stiffness of the velopharyngeal segment when it is effective.

Closing pressure was estimated from the fitted exponential function for each segment. Figure 5 presents the effects of mandibular advancement on the estimated closing pressure for the velopharynx (figure 5A) and the oropharynx (figure 5B). At the oropharynx, the closing pressure significantly decreased after the maneuver in both the obese and nonobese groups. The estimated closing pressure at the velopharynx significantly decreased after mandibular advancement only in the nonobese group, whereas any improvement in the parameter was not observed in the obese group.

Discussion

We found that mandibular advancement shifted upward the pressure-area relations of the oropharynx while increasing the maximum area and decreasing the closing pressure at this segment regardless of the body habitus, and mandibular advancement failed to improve

Table 2. Results of the Exponential Curve Fitting

	Obese (n = 9)		Nonobese (n = 9)	
	Control	Mandibular Advancement	Control	Mandibular Advancement
Velopharynx				
A_{max}	1.99 ± 0.92	1.98 ± 0.88	1.88 ± 1.03	2.45 ± 1.28*
B	1.35 ± 0.64	1.55 ± 0.91	1.35 ± 0.81	1.09 ± 0.82
K	0.134 ± 0.053	0.103 ± 0.023	0.137 ± 0.049	0.089 ± 0.042†
R^2	0.94 ± 0.04	0.93 ± 0.02	0.95 ± 0.04	0.93 ± 0.03
Oropharynx				
A_{max}	2.24 ± 0.81	3.27 ± 1.35*	3.02 ± 1.07	4.53 ± 1.78*
B	1.24 ± 0.53	1.86 ± 2.01	2.00 ± 1.64	1.41 ± 0.93
K	0.129 ± 0.043	0.121 ± 0.038	0.137 ± 0.043	0.112 ± 0.063
R^2	0.93 ± 0.03	0.89 ± 0.09	0.93 ± 0.03	0.88 ± 0.11

B and K are constants obtained by fitting the pressure/area relationship of each pharyngeal segment to an exponential function. $A = A_{max} - B^*\exp(-K^*P_{AW})$, where A, A_{max}, and P_{AW} denote cross-sectional area of the pharynx, maximum area of the pharynx, and airway pressure. A_{max} was determined as mean values of measured area at highest three P_{AW} (18, 19, and 20 cmH_2O). Quality of the fit is provided by coefficient R^2. K characterizes shape of the exponential curve. Values are mean ± SD.

* $P < 0.01$ *versus* control.

† $P < 0.05$ *versus* control.

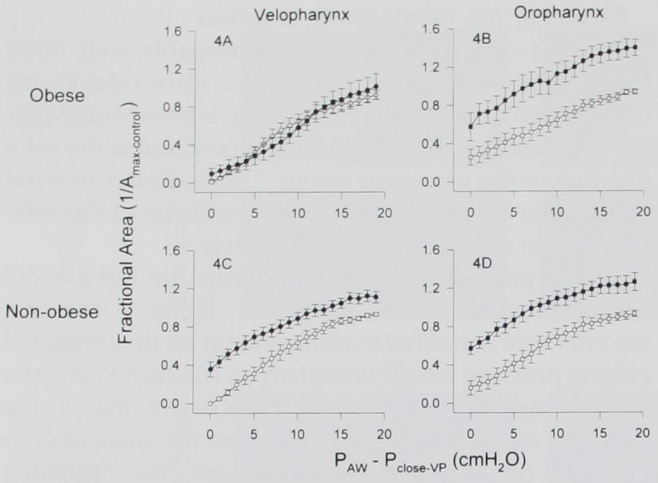

Fig. 4. Influences of mandibular advancement on normalized mean pressure–area relations of the pharynx. (*A*) Velopharyngeal responses in obese persons. (*B*) Oropharyngeal responses in obese persons. (*C*) Velopharyngeal responses in nonobese persons. (*D*) oropharyngeal responses in nonobese persons. Open squares, control condition; closed squares, mandibular advancement. $A_{max-control}$, maximum area without mandibular advancement; P_{AW}, airway pressure; $P_{close-VP}$, velopharyngeal closing pressure without mandibular advancement. Values are means ± SE.

velopharyngeal airway patency in obese persons but was effective in nonobese persons.

Design and Limitation of the Study

Balance between outward forces developed by pharyngeal dilating muscles and inward forces resulting from negative luminal pressure determines the size of the pharyngeal lumen during inspiration.[1,2,7] Intrinsic mechanical properties of the pharynx act as a fulcrum in this balance model. Therefore, pharyngeal patency is determined by complicated interaction among the three factors. A key feature of loss of consciousness such as sleep and general anesthesia is reduction of pharyngeal muscle activities leading to pharyngeal narrowing or closure.[1,2,11] The airway closure induces various neuromuscular responses such as hypoxic and hypercapnic stimulation and upper airway reflexes. These sequential events modulate the pharyngeal muscle activities and luminal pressure. It is difficult to distinguish the relative contribution of each factor to the airway narrowing and closure. Many previous studies examined the site of pharyngeal closure without controlling these factors.[3,4,12–15] We eliminated variation in muscle activities by effecting muscular blockade under general anesthesia. We assessed the mechanical properties of the pharynx, during profound muscular paralysis, by measuring the cross-sectional area of the two pharyngeal segments during stepwise decreases in pharyngeal pressure (apneic test). Any mechanical influence of mandibular advancement can be determined as changes in the static pressure–area relation, independent of neuromuscular factors.[6–9] Our results clearly indicate that mandibular advancement alters the intrinsic mechanical properties and shifts the fulcrum of the balance model to maintain airway size. Our method, however, is limited to examination of the mechanical influence of the maneuver on airway patency under static conditions. Mandibular advancement may increase the length of the upper airway muscles, such as the genioglossus, to augment the contractility of these muscles. These neuromuscular mechanisms should be explored in the future. There may be a potential bias in our measurements of the pharyngeal area because we traced the pharyngeal lumen on the images without controlling for mandibular advancement.

Mechanical Influence of Mandibular Advancement on Pharyngeal Patency

Recent evidence indicates that the most collapsible site of the airway is located at the velopharynx in anesthetized patients while the oropharyngeal airway, although to a lesser extent, also narrows.[13,14] Effective mandibular advancement, therefore, should widen the entire pharynx, including the velopharynx and the oropharynx, as we found in our healthy, nonobese participants. The results are compatible to our previous report

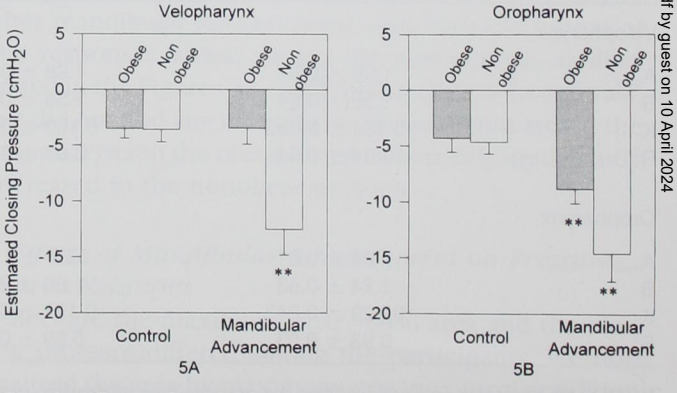

Fig. 5. Effects of mandibular advancement on estimated closing pressure at the velopharynx (*A*) and the oropharynx (*B*). The estimated closing pressure was calculated from $\ln(B/A_{max})K^{-1}$, where B and K are constants in a fitted exponential function and A_{max} is the maximum pharyngeal area. $^{**}P < 0.01$ *vs.* control. Values are means ± SE.

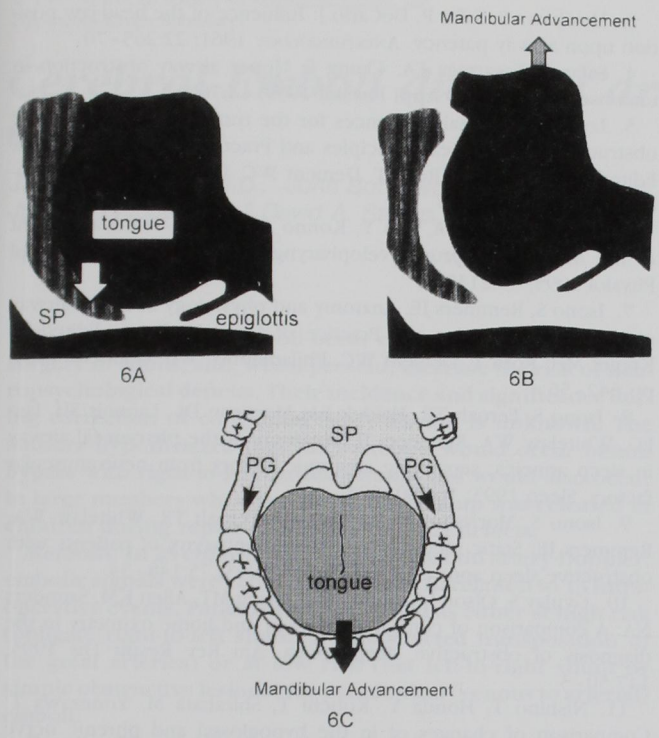

Fig. 6. A diagram explaining possible mechanisms for improvement of velopharyngeal airway patency by mandibular advancement. The gravitational effect of the tongue on the anterior wall of the soft palate (*A*) may be reduced by anterior movement of the mandible (*B*). Alternatively, anterior displacement of the tongue may stiffen the lateral wall of the soft palate through the paratoglossal arch (*C*). SP, soft palate; PG, palatoglossal arch.

of patients with OSA.[6] Although the mechanisms of the actions of mandibular advancement on pharyngeal patency are still speculative, we suggest that there might be several possible mechanical interactions in the pharynx, considering the anatomic arrangement of the upper airway.[6] Because the tongue directly connects to the mandible, forward displacement of the mandible should move the base of the tongue anteriorly and increase the retroglossal airspace. Mandibular advancement never failed to improve airway patency at the oropharynx, probably because of this well-established mechanism. Although the action of mandibular advancement on the velopharynx is complicated, two mechanisms are possible. First, anterior movement of the tongue may decrease the gravitational effect on the soft palate if we assume that the base of the tongue opposes the anterior wall of the soft palate (figures 6A and B). This mechanism appears to be less likely because Nandi *et al.*[13] found that tongue retraction failed to restore the velopharyngeal closure in anesthetized persons. Rowley *et al.*[16] also found little improvement of air flow dynamics by tongue displacement in anesthetized cats. Second, forward displacement of the mandible may decrease collapsibility of the velopharynx. Because the lateral wall of the soft palate anatomically connects to the base of the tongue through the palatoglossal arch, mandibular advancement possibly stretches the soft palate through the mechanical connection, stiffening the velopharyngeal segment (figure 6C). This corresponds well with our observation that mandibular advancement significantly decreased the velopharyngeal closing pressure and the K values.

The action of mandibular advancement on the velopharynx was weak in obese persons. This finding corresponds closely not only with the description of Safar *et al.*[4] but also with our clinical experiences. Mandibular advancement improved oropharyngeal patency to a similar extent in obese and nonobese persons, and therefore relief of the gravitational influence of the tongue on the soft palate is expected to be identical among these persons. Velopharyngeal patency was improved in the nonobese but not in the obese participants. Again, this also suggests that the gravitational influence of the tongue on the soft palate is rather small, although this line of speculation needs to be tested further by examining mechanical effects of body positions. The mandible of the obese participants might not have been advanced as much as in the nonobese ones. The criticism may be valid because we did not measure the amount of the forward displacement of the mandible. This mechanism is, however, unlikely because the oropharyngeal airway increased to a similar extent in obese and nonobese participants. Although the following explanation is speculative now and needs further study, the observed failure to restore velopharyngeal airway patency in response to the mandibular advancement in obese persons can be explained by a rather redundant palatoglossal arch, which mechanically connects the soft palate and the base of the tongue, as illustrated in figure 6C. Obesity is associated with a reduction in lung volume, which appears to modulate the collapsibility of the upper airway through tracheal traction.[17-19] In obese persons, the longitudinal tracheal traction may be little transmitted to the pharynx, resulting in the redundant pharyngeal wall. Accordingly, the stretching forces by mandibular advancement to the palatoglossal arch may not be integrated enough to stiffen the velopharyngeal wall and to increase the velopharyngeal cross-sectional area in obese persons. Our observation

may be similar to the findings of Rowley *et al.*,[16] who examined the effect of trachea and tongue displacement on upper airway air flow dynamics in a feline model and found that tracheal displacement augmented the increase in air flow and reduction of collapsibility of the upper airway with tongue retraction.

Clinical Implications of the Study

Advancement of the mandible never failed to increase the retroglossal area, even in obese persons. Therefore, the oral route is preferable for breathing during induction of anesthesia, especially in obese persons. This may partly explain why mouth-to-mouth breathing is more effective than mouth-to-nose breathing in resuscitation.[20] Although positive-pressure ventilation is expected to increase pharyngeal area in accordance with the pressure-area relations, the increment is smaller in obese persons, indicating less effectiveness of the ventilation. It is obvious that airway maintenance during spontaneous breathing is more difficult in obese persons because negative collapsing forces developing during inspiration. Our findings may have several clinical implications for OSA treatment and for airway management during anesthesia. Although mandibular advancement with a dental appliance is one promising therapeutic option for patients with OSA, we have not established a selection criteria for the therapy.[5] Lack of mechanical influence of mandibular advancement on the velopharyngeal airway possibly accounts for poor responsiveness to the dental appliance, as Eveloff *et al.*[21] have speculated.

In conclusion, forward displacement of the mandible did not improve the patency of the velopharynx in obese persons, whereas this maneuver improved oropharyngeal airway patency both in obese and nonobese persons. Mandibular advancement appeared to have a different mechanical effect on the velopharynx and the oropharynx.

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