Dose-Dependent Effects of Mandibular Advancement on Pharyngeal Mechanics and Nocturnal Oxygenation in Patients With Sleep-Disordered Breathing*

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Study objectives: To examine dose-dependent effects of mandibular advancement on collapsibility of the passive pharynx and sleep-disordered breathing (SDB).

Design: Prospective, randomized study.

Setting: University hospital.

Patients: Thirty-seven adult patients with SDB.

Interventions: Oral appliances with 2-, 4-, and 6-mm advancement of the mandible.

Measurements and results: Overnight oximetry was performed with and without oral appliances. Each 2-mm mandibular advancement coincided with approximately 20% improvement in number and severity of nocturnal desaturations. Percentages of patients producing a > 50% improvement rate of the number of desaturations were 25%, 48%, and 65% with use of oral appliances with 2-, 4-, and 6-mm mandibular advancement, respectively. Static pharyngeal mechanics were evaluated in six completely paralyzed patients with SDB under general anesthesia with and without the oral appliances. Advancement of mandibular position was found to produce dose-dependent closing pressure reduction of all pharyngeal segments. Normalization of nocturnal oxygenation was associated with negative closing pressure, especially at the velopharynx.

Conclusions: We conclude that improvement of both nocturnal oxygenation and pharyngeal collapsibility significantly depends on the mandibular position.

Key words: closing pressure; mandibular position; obstructive sleep apnea; oral appliance; pharynx

Abbreviations: Amax = maximum area; BMI = body mass index; CT90 = percent of time spent at SaO2 < 90%; OA = oral appliance; ODI = oxygen desaturation index; OP = oropharyngeal airway (retroglossal airway); OSA = obstructive sleep apnea; Paw = airway pressure; Pclose = estimated closing pressure; r2 = correlation coefficient of the regression; SaO2 = arterial oxygen saturation; SDB = sleep-disordered breathing; VP = velopharyngeal airway (retropalatal airway)

Sleep-related reduction of pharyngeal muscle activity manifests an abnormally high collapsible pharynx leading to pharyngeal narrowing and closure in patients with obstructive sleep apnea (OSA). A variety of therapeutic options capable of restoring patent airway is now available and developing although the success rate and patient compliance vary among treatments.1,2 Oral appliance (OA) appears to be a promising alternative to nasal continuous positive airway pressure therapy.2,3 Although many OAs in the literature are designed to move the mandible forward to reduce the severity of OSA, the extent of anterior displacement of the mandible was not well controlled, and evaluation of the dose-dependent effect of mandibular advancement on OSA severity was not performed. In addition, although dose-related improvement of pharyngeal collapsibility by mandibular advancement is expected, the precise mechanisms of its improvement have yet to be fully explored in the passive pharynx in which pharyngeal muscle activities are significantly reduced or abolished. Accordingly, the purpose of the present study is to test the hypothesis that both pharyngeal mechanics and severity of sleep-disordered breathing...
(SDB) improve in response to mandibular advancement in a dose-related fashion.

**MATERIALS AND METHODS**

**Subjects**

The study consisted of 43 adult patients with SDB treated with our OA. In addition to clinical symptoms suggesting SDB, such as daytime hypersomnia, loud snoring, and witnessed repetitive apneas, nocturnal oximetry (Pulsox 5; Minolta; Tokyo, Japan) indicated that oxygen desaturation index (ODI), defined as the number of oxygen dips > 4% from the baseline, was >10/h, and the percent of time spent at arterial oxygen saturation (SaO₂) <90% (CT₉₀) was >1% in all patients. After being informed of the various therapeutic options, including nasal continuous positive airway pressure, surgery, and OA, the patients were given a choice to participate in this study. Patients with inadequate dental anchoring structures (too few teeth) for the OA, temporomandibular joint dysfunction (pain during mandibular advancement or limitation of mouth opening), and past history of uvulopalatopharyngoplasty were excluded. The level of ODI determined by the nocturnal oximetry was not included in the exclusion criteria. Of the 43 patients, two desired uvulopalatopharyngoplasty, one preferred nasal continuous positive airway pressure, one produced significant improvement of nocturnal oxygenation by significant reduction of body weight, and two failed to construct their OA before completion of the study protocol. Consequently, nocturnal oxygenation evaluation with OA was performed in 37 patients with SDB. Anthropometric and oximetry variables are presented in Table 1. Six of these patients agreed to participate in the pharyngeal mechanics evaluation study under general anesthesia. Age, body mass index (BMI), and oximetry variables in these six patients did not differ from those of the remaining 31 patients. The aim and potential risks of the study were fully explained, and written informed consent was obtained from each subject. The investigation was approved by the hospital ethics committee.

**Oral Appliance**

An OA was designed to advance the mandible to a prescribed distance from a centric occlusal position by securely anchoring both the maxillary and mandibular arches (Fig 1). Upper and lower teeth arches were fixed with a prescribed mandibular position, and a 5- to 8-mm opening between the upper and lower incisors allows oral breathing (nonadjustable one-piece type). After taking impressions of the upper and lower teeth and recording the centric occlusal position by wax bite, plaster models were fabricated by a heat-suction device (ES 2002; Erkopress; Erkodent Inc.), then cut away from the plaster models. For adaptation of the OAs, each patient wore two separate upper and lower appliances for the initial week, after which successive use of OA2, OA4, and OA6 was performed. Each appliance was used every night for at least a week, and was carefully adjusted to simultaneously increase comfort by relieving pressure on the teeth and avoid dislodgment of the appliance during sleep during the adaptation period. Although the aim of the study was not to evaluate side effects of the OA, excessive salivation and transient discomfort or pain of the temporomandibular joint for a brief time after awakening were commonly reported.

**Evaluation of Pharyngeal Mechanics**

Our endoscopic technique and evaluation of static pharyngeal mechanics are described in detail elsewhere.4–6 Briefly, each subject was placed in the supine position on an operating table, with the neck in the neutral position and with a modified nasal mask. General anesthesia was induced by IV administration of thiopental sodium (4 mg/kg), and IV injection of a muscle relaxant (vecuronium 0.2 mg/kg) produced complete paralysis throughout the experiment. Complete muscle paralysis was confirmed by intermittent electrical stimulation of the ulnar nerve. Anesthesia was maintained by inhalation of sevoflurane (2

<table>
<thead>
<tr>
<th>Variable</th>
<th>Median (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>49.0 (27.1 to 66.6)</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.65 (1.55 to 1.76)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>80.0 (59.1 to 116)</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>28.7 (23.0 to 40.0)</td>
</tr>
<tr>
<td>ODI, h⁻¹</td>
<td>26.0 (11.2 to 72.0)</td>
</tr>
<tr>
<td>CT₉₀, %</td>
<td>10.0 (1.2 to 58.8)</td>
</tr>
<tr>
<td>Mean nadir SaO₂, %</td>
<td>87.2 (78.0 to 91.8)</td>
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</table>

*CI = confidence intervals.
to 4%) while the subject was ventilated with positive pressure with the use of an anesthetic machine. SaO₂, ECG, and BP were continuously monitored. A slim endoscope (FB15H or FB10H; Pentax Inc; Tokyo, Japan; 3 and 4.5 mm outer diameter, respectively) was inserted through the nasal mask and nares without air leak. We placed the endoscope where the velopharyngeal airway (VP; retropalatal airway) or the oropharyngeal airway (OP; retroglossal airway) was in the images. A closed-circuit camera (ETV8; Nisso; Saitama, Japan) was connected to the endoscope to record pharyngeal images through video for later analysis.

Cessation of mechanical ventilation resulted in apnea because of complete muscle paralysis. Using a simple pressure-control system consisting of a blower, voltage slider, and water manometer, airway pressure (Paw) was immediately increased, then slowly reduced in steps of 1 cm H₂O from 20 cm H₂O to VP closing pressure (Pclose), ie, the pressure at which the VP was observed to close completely. SaO₂ remained >95% throughout the apneic test, which lasted for 2 to 3 min in all subjects. The apneic tests were repeated without OA (control), and with OA2, OA4, and OA6. The order of the evaluating conditions was randomly selected by each subject. Measurements were made for the VP and OP, which allowed construction of the static pressure–area relationship of the visualized pharyngeal segment for each condition. The study was terminated with the administration of atropine (1 mg) and neostigmine (2 mg) to reverse muscle paralysis.

The measured cross-sectional area of each pharyngeal segment was plotted as a function of Paw. Accuracy of the cross-sectional area measurements was reported to be within 8%. 4–6 Maximum area (Amax) was determined as the mean value of the highest three Paw (18, 19, and 20 cm H₂O). As reported previously, the pressure/area relationship of each pharyngeal segment was denoted as the exponential function

\[ A = A_{\text{max}} - b \times e^{-k \times \text{Paw}} \]

where \( b \) and \( k \) are constants and \( A \) is cross-sectional area. This curve-fitting analysis allowed us to estimate \( P^{\prime} \text{close} \) from the following equation for each pharyngeal segment

\[ P^{\prime} \text{close} = \ln(b) \times k^{-1} \]

although the OP closure was not usually observed during the apneic tests. We consider that both \( k \) values and \( P^{\prime} \text{close} \) represent collapsibility of the pharynx, whereas the former characterizes the shape of the curve and the latter determines the position of the exponential curve.

Evaluation of Nocturnal Oxygenation

After acclimatization to the OAs, effects of each OA on nocturnal oxygenation were assessed by home overnight oximetry. All subjects were instructed to attach a finger probe of the oximeter to check quality of the recordings. In addition to ODI and CT₉₀, mentioned above, the mean nadir SaO₂ was calculated by averaging the nadir SaO₂ values of all desaturation events. Overnight oximetry was repeated for eight consecutive nights with and without the OAs, and each condition was evaluated at two-night intervals. The order of evaluation was randomly selected by each patient. Oximetry was repeated when patients reported poor quality of sleep and possible dislodging of the device during the study. Mean values of the two-night recordings for each condition were used for analysis.

Statistical Analysis

Statistical significance of effects of dose-dependent mandibular advancement on nocturnal oximetry variables and mechanical variables of the pharynx were assessed by Friedman repeated measures analysis of variance on ranks. Dunnett’s method was used for comparison between the control condition and other states. Correlation between variables was performed by Spearman rank order test. Results are expressed as median (95% confidence intervals). A \( p < 0.05 \) was considered to be significant.

RESULTS

Dose-Dependent Effects of Mandibular Advancement on Static Pharyngeal Mechanics

Pressure–area relationships of the passive pharynx with or without the OAs were satisfactorily fitted by exponential functions with reasonably high regression coefficient values. Table 2 presents the effects of mandibular advancement on mechanical variables obtained by the fitted pressure–area curves of the VP and OP. No statistical significance was evident in VP and OP \( k \) values and Amax at the oropharynx. Increases in Amax at the VP and decreases in \( P^{\prime} \text{close} \) at the VP and OP significantly depended on the extent of mandibular advancement. Positive median values of \( P^{\prime} \text{close} \) became negative with OA4 at the VP and OA2 at the OP. A 6-mm advancement of the mandible resulted in a reduction of \( P^{\prime} \text{close} \) by 5.5 cm H₂O at the VP and by 4.8 cm H₂O at the OP.

VP \( P^{\prime} \text{close} \) was higher than that of the OP in four of six control patients and in five of six patients with OAs. Figure 2 demonstrates the relationship between higher \( P^{\prime} \text{close} \) and ODI for each condition in these six patients. In all the data points, ODI was significantly associated with the higher \( P^{\prime} \text{close} \) values, indicating that improvement of collapsibility of the passive pharynx, mainly at the VP level, resulted in improvement of nocturnal oxygenation. When application of the oral devices reduced the higher \( P^{\prime} \text{close} \) below atmospheric pressure, ODI decreased to <10/h. By contrast, ODI remained >10/h when the higher \( P^{\prime} \text{close} \) remained above atmospheric pressure even with OAs.

Dose-Dependent Effects of Mandibular Advancement on Nocturnal Oxygenation

Figure 3 illustrates overnight recordings of SaO₂ for a patient with and without OAs. As clearly shown in the figure, the number and severity of repetitive desaturations decreased with advancing mandibular position.

Table 3 summarizes dose-dependent effects of mandibular advancement on oximetry variables of 37 patients. Although the response to mandibular ad-
Factors Influencing Improvement of Nocturnal Oxygenation

Median improvement rate of ODI with OA6 was 0.62, which did not significantly correlate with any anthropometric and oximetry variables in the control condition. Figure 2. Relationship between highest $P'_{close}$ and ODI for each condition. The highest $P'_{close}$ was identical to VP $P'_{close}$ in all except one patient. Correlation coefficient was obtained for all the data points. Note that improvement of highest $P'_{close}$ was significantly associated with improvement of ODI.

Table 2—Dose-Dependent Effects of Mandibular Advancement on Static Pharyngeal Mechanics*

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>OA2</th>
<th>OA4</th>
<th>OA6</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Velopharynx</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$A_{max}, \text{cm}^2$</td>
<td>0.89 (0.49 to 1.95)</td>
<td>1.41 (0.55 to 1.86)</td>
<td>1.41 (0.77 to 2.31)</td>
<td>1.82 (0.66 to 1.94)</td>
</tr>
<tr>
<td>$b$</td>
<td>1.23</td>
<td>1.3</td>
<td>1.12</td>
<td>1.05</td>
</tr>
<tr>
<td>$k$</td>
<td>0.12</td>
<td>0.17</td>
<td>0.15</td>
<td>0.15</td>
</tr>
<tr>
<td>$P'_{close, \text{cm H}_2\text{O}}$</td>
<td>2.2 (0.09 to 0.26)</td>
<td>0.11 (0.11 to 0.21)</td>
<td>$-1.71^\dagger$ (0.08 to 0.21)</td>
<td>$-3.31^\dagger$ (0.07 to 0.25)</td>
</tr>
<tr>
<td>$r^2$</td>
<td>0.95</td>
<td>0.96</td>
<td>0.95</td>
<td>0.91</td>
</tr>
<tr>
<td></td>
<td>0.85 to 0.99</td>
<td>0.88 to 0.97</td>
<td>0.88 to 0.98</td>
<td>0.82 to 0.96</td>
</tr>
<tr>
<td><strong>Oropharynx</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$A_{max}, \text{cm}^2$</td>
<td>1.48 (1.09 to 4.46)</td>
<td>2.84 (0.91 to 4.74)</td>
<td>2.57 (1.52 to 5.90)</td>
<td>2.58 (1.36 to 5.44)</td>
</tr>
<tr>
<td>$b$</td>
<td>2.37</td>
<td>2.2</td>
<td>1.73</td>
<td>1.37</td>
</tr>
<tr>
<td>$k$</td>
<td>0.16</td>
<td>0.15</td>
<td>0.14</td>
<td>0.16</td>
</tr>
<tr>
<td>$P'_{close, \text{cm H}_2\text{O}}$</td>
<td>1.3 (0.12 to 0.20)</td>
<td>$-1.21$ (0.09 to 0.21)</td>
<td>$-2.41^\dagger$ (0.11 to 0.18)</td>
<td>$-3.51^\dagger$ (0.08 to 0.18)</td>
</tr>
<tr>
<td>$r^2$</td>
<td>0.95</td>
<td>0.94</td>
<td>0.94</td>
<td>0.93</td>
</tr>
<tr>
<td></td>
<td>0.92 to 0.99</td>
<td>0.88 to 0.98</td>
<td>0.88 to 0.96</td>
<td>0.84 to 0.99</td>
</tr>
</tbody>
</table>

*a, b = constants obtained by fitting the pressure–area relationship of the velopharynx to an exponential function (see Methods); OA2, OA4, OA6 = oral appliances with 2-, 4-, and 6-mm mandibular advancements. Data are median (95% confidence intervals).

†p < 0.05 vs control.

Vancement was variable among the patients, all three variables representing the number and severity of nocturnal desaturations significantly improved with OAs. ODI in the control condition significantly decreased with OA2, OA4, and OA6, and each 2-mm mandibular advancement was associated with approximately 20% improvement of ODI. Reduction of CT$_{90}$ appeared to be linear to the extent of mandibular advancement, and 2-mm increment of the mandibular position coincided with approximately 20% improvement of CT$_{90}$. Although mean nadir $SaO_2$ significantly decreased with OAs, the response appears not to be linear.

Figure 4 (left) demonstrates dose-dependent increases in percentages of responders to the OAs. Percentages of responders, defined as those with $>50\%$ improvement of ODI, were 25%, 48%, and 65% for the use of OA2, OA4, and OA6, respectively. Considering the six patients who dropped out as possible nonresponders to the OAs, the percentage of responders with OA6 seems to range between 56% and 65%. When responders were defined as those with ODI $<10/h$, the percentage of responders to OA6 remained at up to 50%, and dose dependency was evident. ODI improvement rate by OAs is exhibited in Figure 4 (right), clearly indicating dose dependency in the improvement rate. Median 50% improvement rate was achieved by OA4.
condition. ODI in the control condition significantly correlated with BMI ($r^2 = 0.347$, $p < 0.05$), and evidence of correlation was even present in OA6 ($r^2 = 0.331$, $p < 0.05$). Improvement rate with OA6 did not significantly correlate with BMI or control; however, patients with decreased ODI of < 10/h with OA6 (n = 18) were significantly less obese than those who did not respond to OA6 (n = 19; BMI, 27.8 vs 31.1 kg/m², respectively; $p < 0.05$). Similarly, the former had less severe nocturnal desaturation compared with the latter (ODI, 18.2 vs 40.7/h, respectively; $p < 0.001$; CT₉₀, 4.8 vs 21.6%, respectively; $p < 0.05$). These results indicate that normalization of nocturnal oxygenation significantly depends on body size and severity of nocturnal desaturation before treatment whereas the improvement rate is not determined by these factors.

**Table 3—Dose-Dependent Effects of Mandibular Advancement on Oximetry Variables**

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>OA2</th>
<th>OA4</th>
<th>OA6</th>
</tr>
</thead>
<tbody>
<tr>
<td>ODI, h⁻¹</td>
<td>26</td>
<td>17.3†</td>
<td>14.7†</td>
<td>10.8†</td>
</tr>
<tr>
<td></td>
<td>(11.2 to 72.0)</td>
<td>(2.6 to 70.1)</td>
<td>(2.9 to 58.9)</td>
<td>(2.3 to 48.6)</td>
</tr>
<tr>
<td>CT₉₀, %</td>
<td>6.4†</td>
<td>3.5†</td>
<td>2†</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(1.2 to 58.7)</td>
<td>(0 to 46.1)</td>
<td>(0 to 35.4)</td>
<td>(0 to 31.2)</td>
</tr>
<tr>
<td>Mean nadir SaO₂, %</td>
<td>87.2</td>
<td>89.2†</td>
<td>89.5†</td>
<td>89.6†</td>
</tr>
<tr>
<td></td>
<td>(78.0 to 91.8)</td>
<td>(80.0 to 92.6)</td>
<td>(78.1 to 93.4)</td>
<td>(81.3 to 92.6)</td>
</tr>
</tbody>
</table>

*Data are median (95% confidence intervals).
†$p < 0.05$ vs control.

**Discussion**

Major findings of this study are that (1) step-advancement of mandibular position resulted in dose-dependent reduction of closing pressure of the passive pharynx, (2) successful improvement of nocturnal oxygenation appeared to be achieved when the OA reduced the closing pressure of the passive pharynx below atmospheric pressure, and (3) each 2-mm mandibular advancement coincided with approximate 20% improvement of the number and severity of nocturnal desaturations.

**Limitation and Design of the Study**

Because nocturnal oximetry was performed without EEG monitoring, information regarding sleep staging while wearing the OA is unknown, and
improvement of oxygenation with OAs reported here is possibly a result of increased awakening time stimulated by OAs or an oximetry probe. To minimize the artificial improvement of nocturnal oxygenation, at least a 4-week acclimatization period was set aside before performing nocturnal oximetry. The mandible was advanced 2 mm weekly while monitoring the patient’s acceptance to the advancement. Furthermore, because the oximetry was performed at home without attendance, potential dislodgment of the OA might have influenced the results of the nocturnal oximetry. In the absence of an intraoral compliance-monitoring chip, it is unknown what amount of time the OA was actually seated with the patient asleep, which would have been a more accurate measurement instead of total monitoring time, for appropriate evaluation of the therapeutic efficacy of the OA.

Effects of OAs on upper airway size have been evaluated by lateral cephalometry and pharyngeal endoscopy during wakefulness in previous studies. Because pharyngeal muscles actively contract during wakefulness, especially in patients with OSA, the pharyngeal size measured in these studies may not accurately represent airway dimensions during sleep, in which pharyngeal closure or narrowing occurs because of significant reduction of pharyngeal dilator muscle activities. It is our belief that the restoration mechanism of pharyngeal patency by oral devices can only be explored under elimination of the pharyngeal muscle activities. Our unique evaluation method for static mechanics of the passive pharynx was previously reported. Because administration of muscular blockade produces complete elimination of neuromuscular factors influencing pharyngeal patency, the intrinsic mechanical properties of the pharynx is represented by the pressure–area relationship of the pharynx. Accordingly, this is the first precise documentation of the mechanical influence of an oral device on the passive pharyngeal airway configuration.

Although the paralyzed pharynx does offer an appropriate model for assessing its intrinsic mechanical properties, pharyngeal behavior in this condition may differ from that during natural sleep. Therefore, the model may not allow investigation of all the mechanisms causing OSA and does not predict the severity of apnea during sleep. Furthermore, it does not take into account both possible chemoreflex modulation of the upper airway muscle activity and the effects of spontaneous respiration. Recent electromyographic recordings, however, demonstrated that genioglossus activities decrease with mandibular advancement during sleep, which suggests little contribution of pharyngeal muscles in restoring pharyngeal patency with mandibular advancement. Furthermore, our study did not assess the influence of the OA on breathing route. The dynamic influence of mandibular advancement on breathing during sleep needs to be examined in a future study.

**To What Extent Should the Mandible Be Advanced for Treatment of SDB?**

Therapeutic effect of oral devices has been reported with mandibular advancement of 3 to 7 mm from the neutral position or >75% of the maximum protrusive position. These studies, however, only evaluated the effect of OAs at a single mandibular position per patient, and did not elucidate whether repositioning of the mandible resulted in an increase or decrease in device efficacy. In our
study, it was demonstrated that improvement of nocturnal oxygenation depended on the extent of forward mandibular displacement and that 20% improvement of nocturnal oxygenation can be expected for each 2-mm anterior mandibular movement. A 50% improvement of nocturnal oxygenation by OA suggests that the therapeutic range of mandibular advancement appears to be > 4 mm, in agreement with previous reports. However, it should also be noted that obese patients with severe nocturnal desaturation did not normalize oxygenation with a 6-mm mandibular advancement, although the improvement rate did not significantly differ from that of less obese patients. Accordingly, obese patients with severe nocturnal desaturation may not be appropriate candidates for OA therapy.18

Mechanical Action of OA on Pharyngeal Airway

Previous cephalometric analyses indicated that oral devices capable of advancing the mandible increased the posterior airway space, particularly at the soft palate level in patients with OSA.7–9 In increased the posterior airway space, particularly at oral devices capable of advancing the mandible.

Mechanical Action of OA on Pharyngeal Airway

Previous cephalometric analyses indicated that oral devices capable of advancing the mandible increased the posterior airway space, particularly at the soft palate level in patients with OSA.7–9 In contrast, videofluoroscopy of the pharynx during wakefulness demonstrated that mandibular advancement significantly increased anteroposterior diameter and cross-sectional area only at the OP.10 Variability of the sites of action among these studies can be accounted for by uncontrolled upper airway dilator muscle activities. Response of the pharyngeal airway, already dilated by active contraction of the upper airway muscles, to mandibular advancement may not necessarily agree with that of the atonic pharynx examined in this study.

From a consideration of anatomic arrangement of the upper airway, our results suggest several mechanisms for restoration of pharyngeal patency by mandibular advancement.4,6 Improvement of OP patency by forward mandibular displacement may be accomplished by anterior movement of the tongue base connected to the anterior inferior lingual portion of the mandible at the genioglossus muscles. Further, this tongue displacement would decrease the external pressure to the soft palate produced by posterior movement of the tongue base or stiffen the VP through the palatoglossal arch, which connects the tongue base to the lateral wall of the soft palate. This speculation may be supported by our finding that closing pressure at the VP as well as the oropharynx significantly decreased with mandibular advancement. Unfortunately, the influence of mandibular advancement on the airway patency at the level of the tip of the epiglottis (hypopharyngeal airway) was not systematically evaluated in this study. Because distance between the mandible and the hyoid bone is reported to decrease with an OA,11 the roles of hypopharyngeal airway patency on functioning mechanisms of the OA are necessary to be elucidated in the future.

Who Can Be Treated by OA?

The most collapsible site of airway is located at the VP in patients with OSA during sleep, whereas the OP, although to a lesser extent, also narrows.5 The entire pharynx, including the VP and the OP, therefore, needs to be enlarged for effective treatment with the OA. In accordance with our previous report,4 the therapeutic range of mandibular advancement altered intrinsic mechanical properties of the VP as well as those of the OP. In addition, significant improvement of nocturnal oxygenation was achieved only in patients whose closing pressure, mostly at the VP, was decreased below atmospheric pressure by the OA as shown in Figure 2. Considering our previous report that normal subjects have subatmospheric closing pressure of the passive pharynx and patients with SDB have pressure above atmospheric pressure,5 the OA appears to approximate the mechanical properties of the passive pharynx of patients with SDB to those of normal subjects. Our results strongly suggest that the key to successful treatment of SDB is improvement of the VP patency. Because patients with severe desaturations during sleep are likely to have higher closing pressure at the VP as we have previously reported,5 failure of normalization of nocturnal oxygenation by OA in this group of patients can possibly be explained by insufficient improvement of the VP closing pressure by mandibular advancement. Further, poor response to the OA in obese patients with SDB may be caused by a lack of mechanical influence of mandibular advancement on the VP as we previously reported.6 Eveloff et al11 reported that the soft palate length significantly differed between responders and nonresponders to the OA, which suggests the importance of VP response. Presence of severe OP and hypopharyngeal narrowing may be an alternative explanation for the poor responses to the OAs. Increased soft tissue elasticity of the tongue possibly limits mechanical transmission of the mandibular advancement force to the base of the tongue. Although further studies are necessary to establish a selection criteria for an OA, evaluation of mechanical alteration of the passive airway, particularly at the VP, by anterior movement of the mandible could provide useful information for the selection.

Conclusion

Improvement of both nocturnal oxygenation and pharyngeal collapsibility significantly depends on the
position of the mandible advanced by oral devices. Successful treatment of SDB by oral devices is likely to be associated with body size and baseline severity of SDB. Reduction of pharyngeal closing pressure, especially at the VP, appears to be the key for mandibular advancement treatment.

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