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Effects of Mandibular Position and Body Posture on Nasal Patency in Normal Awake Subjects

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ABSTRACT

The purpose of this study was to examine the changes in nasal patency induced by forward mandibular displacement or changes in body posture. Fifteen healthy adults participated in this study. To examine the influence of mandibular position, nasal resistance was recorded in intercuspal, middle, and maximum forward positions. To evaluate the effect of body posture, nasal resistance was recorded in the four postures of sitting erect, 30° and 60° dorsally reclined, and supine. The nasal patencies recorded in the middle and maximum forward mandibular positions were significantly higher than those recorded in the intercuspal position. Regarding the effect of body posture, the nasal patency showed a progressive decrease from the sitting erect position to the supine position. These results suggest that changes in mandibular position and body posture significantly affect nasal patency and that mandibular position and body posture should be considered basic information in the treatment of obstructive sleep apnea.

KEY WORDS: Nasal patency, Mandibular position, Body posture, Obstructive sleep apnea.

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INTRODUCTION Return to TOC

Obstructive sleep apnea (OSA) is a respiratory-related complication characterized by repetitive partial or complete obstruction of the upper airway during sleep.[1] Excessive daytime sleepiness caused by nocturnal sleep fragmentation is a common complaint of patients with OSA and interferes with daytime activities. The activities of the upper airway dilating muscles during sleep have recently attracted considerable attention as a cause of OSA, and the results of many recent studies have shed light on the pathogenesis of OSA.[2–8]

Based on full-night recordings of sleep stages and breathing rhythms, Zwillich et al[9] demonstrated that apneas, sleep arousals and awakenings, and changes in the sleep architecture occurred during nasal obstruction in normal men. Similar findings were reported by Taasan et al,[10] Olsen et al,[11] and Suratt et al.[12] These findings indicate that increased nasal resistance surely contributes to the occurrence of OSA and imply that controlling nasal patency is essential for the treatment of patients with OSA.

As a noninvasive modality for the treatment of OSA, the effect of an oral appliance that keeps the mandible in a forward position has recently been well documented.[13,14] However, there have been no studies regarding the effect of an oral appliance on nasal patency. Since the usefulness of an oral appliance for the treatment of OSA has been well recognized, it could be speculated that wearing an oral appliance might have a beneficial effect on nasal patency. If we could find a distinct pattern in nasal patency associated with wearing the oral appliance, it may help us to understand the working mechanism of the oral appliance from another perspective.

The first purpose of this study was to examine the change in nasal patency induced by forward mandibular displacement simulating the use of an oral appliance. On the other hand, there were a few studies that examined the effect of body posture on nasal patency.[15–18] However, none of the previous studies examined changes in nasal patency when the trunk of the body was reclined from sitting erect to supine positions in a stepwise manner. It has been reported that some OSA patients showed intraindividual variations in severities of symptoms depending on their body posture during sleep,[19–21] and the symptoms were generally the most severe in the supine position.[20,21] Since the deterioration of nasal patency is a proposed cause of OSA, it could be hypothesized that nasal patency might progressively decrease from the sitting erect position to the supine position. Thus the second purpose of this study was to examine the change in nasal patency related to changes in body posture.

MATERIALS AND METHODS Return to TOC

Subjects

Fifteen healthy adults (seven men and eight women) participated in this study (mean ± standard deviation; age, 25.8 ± 1.2 years; body mass index, 21.0 ± 2.9 kg/m^2). All participants had individual normal occlusion, and based on a careful clinical examination using simple questionnaires, they were confirmed to have no medical history of temporomandibular disorders, nasal obstruction, or sleep-disordered breathing such as snoring or apnea. Prior to the study, all of the subjects gave their informed consent to participate after receiving a full explanation of the study aim and design.

Experimental procedures

The three mandibular positions that were used included the intercuspal position, the maximum forward position without pain or discomfort, and the anteroposterior position midway between these other positions (Figure 1). Bite registration in each position was accomplished using Paraffin wax (GC, Tokyo, Japan), and each mandibular position during the experiment was determined using bite wax. The bite wax provides retention to the bite position and holds the jaw forward. The lingual contour of the bite wax was trimmed so as not to disturb tongue movement. The vertical dimension was adjusted to be minimal for each anteroposterior position. In regard to body posture, the four positions defined were sitting erect, 30° dorsally reclined, 60° dorsally reclined, and supine.

Ten minutes after the subject was seated in a dental chair, the recording was started. A rhinomanometer (SR-11A: RION, Tokyo, Japan) was used to measure nasal resistance by the anterior method. The effect of changes in mandibular position on nasal patency recording was commenced in the intercuspal position followed by the middle and maximum forward positions. Throughout the recording session the body was held sitting erect and the head was laid on a headrest to keep both the body and head postures constant. On the other hand, the recording of the effect of body posture on nasal patency was started in the sitting erect position followed by the 30° and 60° reclined and supine positions. Throughout the recording session, each subject was instructed to close his or her mouth in the intercuspal position and to lay his or her head on a headrest constantly. Nasal resistance in each condition was measured three times at 1-minute intervals, and the mean of the three measurements was regarded as a representative value. The measurement was started 10 minutes after the subject changed mandibular position or body posture.

Data analysis

To standardize the nasal patency for each subject, a nasal-patency ratio was calculated. This ratio was defined as the ratio of the total nasal patency of each subject to the normal standard value. The nasal-patency ratio was automatically calculated by the rhinomanometer in the expiratory and inspiratory phases separately. The usefulness and validity of this method to evaluate nasal patency has previously been reported.[22] A two-way repeated analysis of variance (ANOVA) and a multiple comparison test (contrast) were used for the statistical analysis.

RESULTS Return to TOC

Effect of mandibular position on nasal patency

The results of the two-way repeated ANOVA indicated that neither gender alone nor gender interaction had any significant effects, and only mandibular position had significant effects in both the expiratory and inspiratory phases ($P < .01$). Accordingly, the following data analysis was performed on all subjects without considering gender.

Figure 2 and Table 1 show changes in the nasal-patency ratio induced by changes in mandibular position. In the expiratory phase, the nasal-patency ratio in the middle position was significantly higher than that in the intercuspal position ($P < .01$). The nasal-patency ratio in the maximum forward position was significantly greater than those in the middle ($P < .05$) and intercuspal ($P < .01$)

positions. Mean nasal-patency ratios in the intercuspal, middle, and maximum forward positions were 69.2%, 78.6%, and 83.9%, respectively.

On the other hand, in the inspiratory phase the nasal-patency ratio in the middle position was significantly greater than that in the intercuspal position ($P < .05$). Although the nasal-patency ratio in the maximum forward position was significantly greater than that in the intercuspal position, there was no significant difference in the nasal-patency ratio between the middle and maximum forward positions. Mean nasal-patency ratios in each mandibular position were 66.0, 71.4, and 75.9%, respectively.

Although a simple correlation coefficient was calculated to determine the relationship between the body mass index and the change in the nasal-patency ratio related to changes in mandibular position, no significant correlations were detected.

Effect of body posture on nasal patency

The results of the two-way repeated ANOVA indicated that gender had no significant effects on the change in the nasal-patency ratio related to changes in body posture, and only body posture had a significant effect in both the expiratory and inspiratory phases ($P < .01$). Therefore, all 15 subjects were analyzed as a single group.

Figure 3 and Table 2 show changes in the nasal-patency ratio induced by changes in body posture. Regarding the expiratory phase, the nasal-patency ratio tended to progressively decrease from the sitting erect position to the supine position, with significant differences in the nasal-patency ratio between all pairs of body postures ($P < .01$) except between the 30° and 60° reclined positions. Mean nasal-patency ratios in the sitting erect, 30° and 60° reclined, and supine positions were 72.7, 67.2, 65.3, and 52.6%, respectively. The greatest difference in the nasal-patency ratio was seen between the 60° reclined and supine positions (12.7%).

The same trend for changes in the nasal-patency ratio was observed for the inspiratory phase. Mean nasal-patency ratios in each body posture were 65.9% in the sitting erect position, 61.8% in the 30° reclined position, 59.0% in the 60° reclined position, and 48.0% in the supine position. As in the expiratory phase, the greatest difference in the nasal-patency ratio was seen between the 60° dorsally reclined and supine positions (11.0%).

Although a simple correlation coefficient was calculated to determine the relationship between the body mass index and the change in the nasal-patency ratio related to changes in body posture, no significant correlations were found.

DISCUSSION Return to TOC

The nasal-patency ratio used in this study showed remarkable interindividual variations from 20% to 120%. This may have been because the criteria for subject selection did not include subjective and/or objective symptoms regarding ongoing nasal airway obstruction. It has been demonstrated that the air temperature and humidity affect nasal resistance.[23] However, since these variables were held constant during recording in the present study, any changes in nasal resistance were considered to be due to changes in the mandibular position or body posture. Therefore, such extreme interindividual variations could be accepted because this study attempted to examine changes in nasal patency on an individual basis.

It is well known that nasal resistance changes in a cyclical manner from side to side throughout the day and night, and this is generally referred to as the nasal cycle.[24] Thus the nasal cycle should be considered when evaluating unilateral nasal resistance. Furthermore, it has also been reported that total nasal resistance varies very little despite wide variations in unilateral nasal resistance during the nasal cycle.[24] Therefore, total nasal resistance, instead of unilateral nasal resistance, was measured in the present study.

In the present study, male and female subjects were regarded as a single group. Some previous studies reported a gender difference in the response of oropharyngeal structures to orthognathic intervention,[25] or that men were predisposed to sleep-disordered breathing.[26] However, in the present study gender did not appear to have any effect on the change in nasal patency caused by changes in mandibular position or body posture. Although the reason for this lack of a difference is unknown, data from male and female subjects were pooled and analyzed as a single group.

Although some disadvantages of the anterior rhinomanometric method are indicated, this form of rhinomanometry has been used extensively because this method is simple and associated with less discomfort to subjects compared to the posterior rhinomanometry. Indeed, this method may be acceptable for the assessment of major changes in total nasal resistance,[27] and therefore the anterior rhinomanometry was employed in the present study.

Effect of mandibular position on nasal patency

Recently, oral appliances have been accepted as an effective conservative treatment option for patients with OSA,[12,13] and several studies have examined the working mechanism of this appliance.[28–33] These studies have demonstrated that the upper airway dimension in the velopharyngeal and oropharyngeal regions is increased as a result of forced forward positioning of the mandible induced by wearing an oral appliance. These structural changes could contribute to preventing upper airway obstruction during sleep.

Although many previous studies have examined the effect of head or mandibular postural changes on the upper airway,[34–38] there has been no study regarding the change in nasal patency related to forward displacement of the mandible imitating the use of an oral appliance for the treatment of OSA. The results of this study showed that the nasal-patency ratio progressively increased following forward positioning of the mandible. Previous studies, which examined the effect of nasal obstruction on respiratory function during sleep, have demonstrated that normal respiration was disturbed and the frequency of apnea and hypopnea were increased by nasal obstruction.[9–12] Although the exact reason for this relationship is unknown, there are at least two possible explanations. One possibility is that nasal obstruction during sleep causes a reflex change in the tone of upper airway–dilating muscles due to the loss of stimulation of the nasal mucosa.[11,39] Another possibility is that the increased upper airway resistance resulting from nasal obstruction could create greater negative pressure during inspiration, which would lead to inspiratory collapse of the upper airway.[11]

If forward positioning of the mandible increases nasal patency, nasal breathing will be promoted, the volume of air through the nasal passage will be augmented, and greater stimulation of the nasal mucosa can be expected. In addition, increased nasal patency could reduce the inspiratory negative pressure within the upper airway. Therefore it can be deduced that an oral appliance will not only dilate the upper airway through passive displacement of the soft palate and tongue following forward positioning of the mandible, but also should induce a nasal reflex and reduce inspiratory negative pressure through changes in nasal patency.

It has been reported that the greatest airflow resistance in the normal airway occurs at the nasal valve.[40–42] Warren et al,[43] Hairfield and Warren,[44] and Drake et al[45] measured the smallest cross-sectional area of the nasal passage (ie, nasal valve size) during quiet respiration in individuals with and without cleft. The nasal valve is located in the region between the upper and lower lateral cartilages and the pyriform aperture just beyond the anterior ends of the inferior turbinates. Hairfield et al[46] reported that the nasal airway was an active participant in the breathing process, rather than a passive conduit of the airflow that can be attributed to nasal valve function. Moreover, it was suggested that the nasal valve size could be easily modified by a variety of factors, eg, mucosal swelling of the inferior turbinates can diminish valve size and the anterior portion of the turbinates can impinge on the valve.[44] The anterior migration of the mandible may affect the nasal airway, including the nasal valve, and result in the increased nasal patency.

Since the upper airway negative pressure is generated in the inspiratory phase, the inspiratory nasal patency seems to be particularly important in considering upper airway obstruction. There were no significant changes in the nasal-patency ratio during inspiration between the middle and maximum forward mandibular positions. This finding suggests that although forward positioning of the mandible to the middle position could be useful for preventing OSA, further forward positioning might not yield an additional benefit for the treatment of OSA.

Effect of body posture on nasal patency

A few studies have examined the relationship between changes in body posture and nasal patency. Rundcrantz[15] demonstrated an increase in nasal resistance when lying down horizontally, and the change in rhinitic patients was greater than that in normal subjects. In the present study, a significant reduction in the nasal-patency ratio was demonstrated with a change from the sitting erect position to the supine position, whereas there was no significant difference between the 30° and 60° reclined positions. The greatest difference in the nasal-patency ratio was seen between the 60° reclined and supine positions.

The physiological mechanism of the posture-related change in nasal patency can be explained as follows. The stroke volume and cardiac minute volume increase with a change from sitting to the supine posture, which will result in an increase in blood pressure.[47] Next, a baroreceptor-mediated reflex will be evoked to depress the blood pressure by a decrease in the heart rate and the dilation of peripheral blood vessels.[47] Such dilation of peripheral blood vessels in the nasal region will lead to swelling of the nasal mucosa and result in a decrease in nasal patency.[48] Furthermore, the nasal region, which is positioned superiorly in the sitting erect position, will be aligned in the same horizontal level as the heart in the supine position, and the pressure difference corresponding to the distance between the heart and nasal region will also dilate the blood vessels in the nasal mucosa.

It has been demonstrated that changing from the upright to the supine position causes a decrease in functional residual capacity and the upper airway size, including the pharyngeal region, which was not affected by the change in functional residual capacity in healthy subjects.[19] Hoffstein et al[49] reported similar findings in that a change in functional residual capacity was not apt to be associated with a large decrease in pharyngeal size in obese subjects without OSA. Based on these previous reports and the fact that the pharynx is a vulnerable region of the airway that is susceptible to postural changes, it can be considered that position-dependent changes in lung volume might not have a significant effect on the size of the nasal airway or nasal patency.

In this study, the most remarkable differences in both the expiratory and inspiratory nasal-patency ratios were recorded between the 60° reclined and supine positions. Based on this finding, only a 30° postural change from the supine position is needed to dramatically affect nasal patency and the severity of OSA. Moreover, there were no significant differences in the nasal-patency ratio between the 30° and 60° reclined positions. This might mean that further improvement cannot be expected beyond the 30° reclined position even if the patient assumes a more upright posture. Hence when the body posture is considered a method for treating OSA, it will be reasonable to observe the symptoms of OSA patients in the 30° dorsally reclined position.

In this study, the effects of mandibular position and body posture on nasal patency were examined in young and slim subjects without OSA during wakefulness, and it remains unclear whether the same effect is seen in middle-aged and obese patients with OSA during sleep. Furthermore, one should note that the effects of mandibular protrusion seen in the upright position cannot necessarily be extrapolated to the supine position or to sleep.

REFERENCES Return to TOC

1. Kryger MH, Roth T, Dement WC. *Principles and Practice of Sleep Medicine*. London: W.B. Saunders; 1994.

2. Sauerland EK, Harper RM. The human tongue during sleep: electromyographic activity of the genioglossus muscle. *Exp Neurol.* 1976; 51:160–170. [PubMed Citation]

3. Guilleminault C, Hill MW, Simmons FB, Dement WC. Obstructive sleep apnea: electromyographic and fiber optic studies. *Exp Neurol.* 1978; 62:48–67. [PubMed Citation]

4. Remmers JE, deGroot WJ, Sauerland EK, Anch AM. Pathogenesis of upper airway occlusion during sleep. *J Appl Physiol: Respirat Environ Exerc Physiol.* 1978; 44:931–938. [PubMed Citation]

5. Jeffries B, Brouillette RT, Hunt CE. Electromyographic study of some accessory muscles of respiration in children with obstructive sleep apnea. *Am Rev Respir Dis.* 1984; 129:696–702. [PubMed Citation]

6. Wiegand DA, Latz B, Zwillich CW, Wiegand L. Geniohyoid muscle activity in normal men during wakefulness and sleep. *J Appl Physiol.* 1990; 69:1262–1269. [PubMed Citation]

7. Mezzanotte WS, Tangel DJ, White DP. Waking genioglossal electromyogram in sleep apnea patients versus normal controls (a neuromuscular compensatory mechanism). *J Clin Invest.* 1992; 89:1571–1579. [PubMed Citation]

8. Tangel DJ, Mezzanotte WS, Sandberg EJ, White DP. Influences of NREM sleep on the activity of tonic vs. inspiratory phasic muscles in normal men. *J Appl Physiol.* 1992; 73:1058–1066. [PubMed Citation]

9. Zwillich CW, Pickett C, Hanson FN, Weil JV. Disturbed sleep and prolonged apnea during nasal obstruction in normal men. *Am Rev Respir Dis.* 1981; 124:158–160. [PubMed Citation]

10. Taasan V, Wynne JW, Cassisi N, Block AJ. The effect of nasal packing on sleep-disordered breathing and nocturnal oxygen desaturation. *Laryngoscope.* 1981; 91:1163–1172. [PubMed Citation]

11. Olsen KD, Kern EB, Westbrook PR. Sleep and breathing disturbance secondary to nasal obstruction. *Otolaryngol Head Neck Surg.* 1981; 89:804–810. [PubMed Citation]

12. Suratt PM, Turner BL, Wilhoit SC. Effect of intranasal obstruction on breathing during sleep. *Chest.* 1986; 90:324–329. [PubMed Citation]

13. Schmidt-Nowara W, Lowe A, Wiegand L, Cartwright R, Perez-Guerra F, Menn S. Oral appliances for the treatment of snoring and obstructive sleep apnea: a review. *Sleep.* 1995; 18:501–510. [PubMed Citation]

14. Schmidt-Nowara W. Recent development in oral appliance therapy of sleep disordered breathing. *Sleep Breath.* 1999; 3:103–106. [PubMed Citation]

15. Rundcrantz H. Postural variations of nasal patency. *Acta Otolaryngol.* 1969; 68:435–443.

16. Cole P, Haight JSJ. Posture and nasal patency. *Am Rev Respir Dis.* 1984; 129:351–354. [PubMed Citation]

17. Kase Y, Hilberg O, Pedersen OF. Posture and nasal patency: evaluation by acoustic rhinometry. *Acta Otolaryngol.* 1994; 114:70–74.

18. Meysman M, Vincken W. Effect of body posture on spirometric values and upper airway obstruction indices derived from the flow-volume loop in young nonobese subjects. *Chest.* 1998; 114:1042–1047. [PubMed Citation]

19. Fouke JM, Strohl KP. Effect of position and lung volume on upper airway geometry. *J Appl Physiol.* 1987; 63:375–380. [PubMed Citation]

20. Cartwright RD, Lloyd S, Lilie J, Kravitz H. Sleep position training as treatment for sleep apnea syndrome: a preliminary study. *Sleep.* 1985; 8:87–94. [PubMed Citation]

21. McEvoy RD, Sharp DJ, Thornton AT. The effects of posture on obstructive sleep apnea. *Am Rev Respir Dis.* 1986; 133:662–666.

[PubMed Citation]

22. Naitoh Y, Shirato M. Comparison of rhinomanometric values expressed by rate—validity and normal range. *J Otolaryngol Jpn.* 1989; 92:1884–1894. [PubMed Citation]

23. Salman SD, Proctor DF, Swift DL, Evering SA. Nasal resistance: description of a method and effect of temperature and humidity changes. *Ann Otol.* 1971; 80:736–743. [PubMed Citation]

24. Principato JJ, Ozenberger JM. Cyclical changes in nasal resistance. *Arch Otolaryngol.* 1970; 91:71–77. [PubMed Citation]

25. Nakagawa F, Ono T, Ishiwata Y, Kuroda T. Morphologic changes in the upper airway structure following surgical correction of mandibular prognathism. *Int Adult Orthod Orthognath Surg.* 1998; 13:299–306. [PubMed Citation]

26. Block AJ, Boysen PG, Wynne JW, Hunt LA. Sleep apnea, hypopnea and oxygen desaturation in normal subjects: a strong male predominance. *N Engl J Med.* 1979; 300:513–517. [PubMed Citation]

27. Cole P. Rhinomanometry: practice and trends. *Laryngoscope.* 1989; 99:311–315. [PubMed Citation]

28. Bonham PE, Currier GF, Orr WC, Othman J, Nanda RS. The effect of a modified functional appliance on obstructive sleep apnea. *Am J Orthod Dentofac Orthop.* 1988; 94:384–392. [PubMed Citation]

29. Schmidt-Nowara WW, Meade TE, Hays MB. Treatment of snoring and obstructive sleep apnea with a dental orthosis. *Chest.* 1991; 99:1378–1385. [PubMed Citation]

30. Menn SJ, Loube DI, Morgan TD, Mitler MM, Berger JS, Erman MK. The mandibular repositioning device: role in the treatment of obstructive sleep apnea. *Sleep.* 1996; 19:794–800. [PubMed Citation]

31. Ferguson KA, Love LL, Ryan F. Effect of mandibular and tongue protrusion on upper airway size during wakefulness. *Am J Respir Crit Care Med.* 1997; 155:1748–1754. [PubMed Citation]

32. Lowe AA, Sjoholm TT, Ryan CF, Fleetham JA, Ferguson KA, Remmers JE. Treatment, airway and compliance effects of a titratable oral appliance. *Sleep.* 2000; 23: S. 172–S178.

33. Liu Y, Zeng X, Fu M, Huang X, Lowe AA. Effects of a mandibular repositioner on obstructive sleep apnea. *Am J Orthod Dentofac Orthop.* 2000; 118:248–256. [PubMed Citation]

34. Weber ZJ, Preston CB, Wright PG. Resistance to nasal airflow related to changes in head posture. *Am J Orthod.* 1981; 80:536–545. [PubMed Citation]

35. Liistro G, Stanescu D, Dooms G, Rodenstein D, Veriter C. Head position modifies upper airway resistance in men. *J Appl Physiol.* 1988; 64:1285–1288. [PubMed Citation]

36. Hellsing E. Changes in the pharyngeal airway in relation to extension of the head. *Eur J Orthod.* 1989; 11:359–365. [PubMed Citation]

37. Johnson LM, Arnett GW, Tamborello JA, Binder A. Airway changes in relation to mandibular positioning. *Otolaryngol Head Neck Surg.* 1992; 106:143–148. [PubMed Citation]

38. Solow B, Skov S, Ovesen J, Norup PW, Wildschiodtz G. Airway dimensions and head posture in obstructive sleep apnea. *Eur J Orthod.* 1996; 18:571–579. [PubMed Citation]

39. Wynne JW. Obstruction of the nose and breathing during sleep. *Chest.* 1982; 82:657–658. [PubMed Citation]

40. De Wit CA, Kapteyn TS, van Bochove W. Some remarks on the physiology, the anatomy and the radiology of the vestibulum and the isthmus nasi. *Int Rhinol.* 1965; 3:37–42.

41. Bridger GP. Physiology of the nasal valve. *Arch Otolaryngol Head Neck Surg.* 1970; 92:543–553.

42. Hinderer KH. Surgery of the valve. *Int Rhinol.* 1970; 8:60–67.

43. Warren DW, Lehman MD, Hinton VA. Analysis of simulated upper airway breathing. *Am J Orthod.* 1984; 86:197–206. [PubMed Citation]

44. Hairfield WM, Warren DW. Dimensions of the cleft nasal airway in adults: a comparison with subjects without cleft. *Cleft Palate J.* 1989; 26:9–13.

45. Drake AF, Davis JU, Warren DW. Nasal airway size in cleft and noncleft children. *Laryngoscope.* 1993; 103:915–917. [PubMed Citation]

46. Hairfield WM, Warren DW, Hinton VA, Seaton DL. Inspiratory and expiratory effects of nasal breathing. *Cleft Palate J.* 1987; 24:183–189.

47. Detweiler DK. Regulation of systemic and pulmonary circulation. In: Brobeck JR, ed. *Best and Taylor's Physiological Basis of Medical Practice,* 9th ed. Baltimore: Williams & Wilkins; 1973: 3–189.

48. Hasegawa M, Saito Y. Postural variations in nasal resistance and symptomatology in allergic rhinitis. *Acta Otolaryngol.* 1979; 88:268–272.

49. Hoffstein V, Zamel N, Phillipson EA. Lung volume dependence of pharyngeal cross-sectional area in patients with obstructive sleep apnea. *Am Rev Respir Dis.* 1984; 130:175–178. [PubMed Citation]

TABLES Return to TOC

TABLE 1. The Nasal-patency Ratios in Each Mandibular Position (Mean ± SD)

	Intercuspal	Middle	Maximum Forward
Expiratory phase	69.2 ± 26.5	78.6 ± 30.0	83.9 ± 36.2
Inspiratory phase	66.0 ± 23.8	71.4 ± 25.4	75.9 ± 31.0

TABLE 2. The Nasal-patency Ratios in Each Body Posture (Mean ± SD)

	Erect	30° Reclined	60° Reclined	Supine
Expiratory phase	72.7 ± 24.3	67.2 ± 23.3	65.3 ± 22.5	52.6 ± 21.8
Inspiratory phase	65.9 ± 21.1	61.8 ± 20.0	59.0 ± 18.8	48.0 ± 17.8

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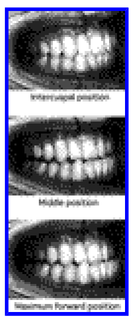

Click on thumbnail for full-sized image.

FIGURE 1. Three mandibular positions used in this study

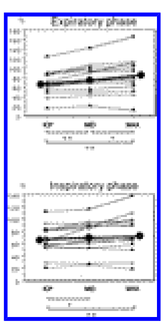

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FIGURE 2. Effect of mandibular position on nasal patency. ICP indicates intercuspal position; MID, middle position; MAX, maximum forward position; triangle, female subjects; circle, male subjects (***P* < .01, **P* < .05). Larger solid black circles indicate mean values, and the vertical axis represents the nasal-patency ratio

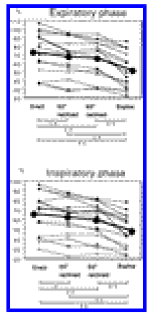

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FIGURE 3. Effect of body posture on nasal patency. Erect indicates sitting erect position; 30° reclined, 30° dorsally reclined position; 60° reclined, 60° dorsally reclined position; supine, supine position; triangle, female subjects; circle, male subjects (***P* < .01, **P* < .05). Larger solid black circles indicate mean values, and the vertical axis represents the nasal-patency ratio

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