

Anatomical Balance of the Upper Airway and Obstructive Sleep Apnea

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Background: Obesity and craniofacial abnormalities such as small maxilla and mandible are common features of patients with obstructive sleep apnea (OSA). The authors hypothesized that anatomical imbalance between the upper airway soft-tissue volume and the craniofacial size (rather than each alone) may result in pharyngeal airway obstruction during sleep, and therefore development of OSA.

Methods: Blind measurements of tongue cross-sectional area and craniofacial dimensions were performed through lateral cephalograms in 50 adult male patients with OSA and 55 adult male non-OSA subjects with various craniofacial dimensions.

Results: Maxillomandibular dimensions were matched between OSA and non-OSA groups. While the tongue was significantly larger in subjects with larger maxillomandible dimensions, OSA patients had a significantly larger tongue for a given maxillomandible size than non-OSA subjects. The hypothesis was also supported in subgroups matched for both body mass index and maxillomandible dimensions.

Conclusions: Upper airway anatomical imbalance is involved in the pathogenesis of OSA.

OBSTRUCTIVE sleep apnea (OSA) is a common disorder affecting 9% of women and 24% of men.¹ Obesity, a growing world-wide problem, may further increase prevalence and severity of OSA.² Although not conclusive, OSA is a potential risk factor for development of perioperative respiratory complications.³⁻⁵ Strategies of perioperative airway management of patients with OSA should be based on understanding of pathophysiology of upper airway obstruction.

In addition to obesity, craniofacial abnormalities such as small maxilla and mandible are common features of OSA patients.⁶⁻¹¹ We recently reported that obesity and craniofacial abnormalities synergistically increase pharyngeal airway collapsibility during general anesthesia and paralysis.¹² Structurally, the pharyngeal airway is surrounded by soft tissue such as the tongue, which is enclosed by bony structures such as the mandible and cervical vertebrae. Under suppression of pharyngeal dilator muscle contraction during sleep and anesthesia, the

anatomical balance between the soft-tissue volume inside the bony enclosure and the bony enclosure size seems to determine the pharyngeal airway size.^{12,13} Accordingly, the anatomical imbalance between upper airway soft-tissue volume and bony enclosure size may result in pharyngeal airway obstruction during sleep and anesthesia. That is, OSA patients may have excessive upper airway soft tissue for a given craniofacial dimension than non-OSA (control) subjects. Considering the heterogeneity of craniofacial characteristics and commonality of craniofacial abnormalities among OSA patients, this hypothesis must be tested in OSA and craniofacial dimension-matched non-OSA subjects including both normal and abnormal craniofacial characteristics.

Previous studies demonstrated increased tongue size in OSA patients compared with non-OSA subjects by two-dimensional cephalometry^{14,15} and three-dimensional computed tomography or magnetic resonance imaging assessments.^{16,17} However, subjects with craniofacial abnormalities were not included in the non-OSA control group, although OSA patients had craniofacial abnormalities in these studies.¹⁴⁻¹⁷ No previous study, to our knowledge, has matched craniofacial dimensions between OSA and non-OSA groups for comparison of tongue size and has assessed whether non-OSA subjects with craniofacial abnormalities have smaller tongue size than OSA patients with craniofacial abnormalities. In our sleep and difficult airway clinic, we often assess both craniofacial structures and breathing during sleep in subjects with craniofacial abnormalities because of suspicion or evidence of difficult tracheal intubation during induction of general anesthesia. In addition to potential non-OSA subjects with normal craniofacial characteristics, we included these subjects as part of potential non-OSA subjects for matching craniofacial dimensions between OSA and non-OSA groups and covering a wide range of craniofacial characteristics. Accordingly, we compared balance of craniofacial and tongue size between OSA patients and craniofacial dimension-matched non-OSA subjects.

Materials and Methods

The investigation was approved by the institutional review board of Graduate School of Medicine, Chiba University, Chiba, Japan. Informed consent was obtained from each subject after the aim and potential risks were fully explained to each.

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Study Subjects

Two groups of adult male subjects (control group: 55 non-OSA subjects; OSA group: 50 OSA patients) were selected for this study after matching age, height, and maxillomandible dimensions. Exclusion criteria for both groups included presence of hypertrophied tonsils, acromegalics, and difficulty in identifying tongue silhouette on the cephalogram. The subjects were limited to men in this study because of possible craniofacial differences between sexes. OSA patients with an apnea-hypopnea index less than 20 episodes per hour were not included in the OSA group.

Obstructive sleep apnea patients were recruited from our clinic for sleep-disordered breathing. Because craniofacial abnormalities are common in OSA patients,^{11,12} we considered that recruitment of potential non-OSA subjects from the general adult population would fail to match their craniofacial dimensions for those of OSA patients. Accordingly, 55 non-OSA male subjects were selected from three different populations. Because craniofacial abnormalities are known to be associated with difficult tracheal intubation,¹⁸ 17 male patients with suspicion or evidence of difficult tracheal intubation during anesthesia induction (Cormack and Lehane grade greater than 2 with laryngeal pressure) were included. Twenty-three male subjects who were referred to our clinic for preoperative airway assessment and were confirmed for absence of sleep-disordered breathing were also included. Furthermore, data from 15 non-OSA subjects previously reported were reanalyzed as control subjects with normal craniofacial characteristics.¹²

Sleep Studies

Diagnosis of OSA was confirmed by standard full polysomnography, which was performed at one of the two local sleep laboratories (Komagamine Sleep Respiratory Center in Tokyo or Pulmonary Department of Sannou Hospital Medical Center in Chiba). Recordings include bilateral electroencephalograms, bilateral electrooculograms, submental electromyogram, leg electromyograms, electrocardiogram, airflow measurement with a thermistor at the mouth and nose, thoracoabdominal wall motions, oxygen saturation (SpO_2), snoring over a microphone, and body position. Apnea was defined as absence of airflow for more than 10 s. Hypopnea was determined upon an apparent reduction of airflow for more than 10 s with reduction of SpO_2 by more than 4% from the baseline. Apneic events were classified as obstructive, mixed and central, and the apnea-hypopnea index was calculated as the total number of the obstructive or mixed apnea and hypopnea events per hour of sleep.

Nocturnal oximetry by a pulse oximeter (Pulsox-5 or Pulsox-3i; Minolta, Tokyo, Japan) and careful assessment of clinical symptoms were performed to exclude patients with potential sleep-disordered breathing from the control group. All potential control subjects were in-

structed to attach an oximetry finger probe before sleep and to remove the probe upon awakening. Digital readings of SpO_2 and pulse rate were stored every 5 s in the oximeter. The stored data were displayed on a computer screen to check quality of the recordings. The oxygen desaturation index, defined as the number of oxygen desaturation exceeding 4% from the baseline, the percentage of time spent at oxygen saturation less than 90%, the mean nadir of oxygen desaturations, and lowest SpO_2 were calculated by the computer. Nocturnal oximetry was repeated twice, and only subjects who presented normal oximetry—defined as an oxygen desaturation index $<5\ h^{-1}$, the percentage of time spent at oxygen saturation less than 90% $< 1\%$, and absence of daytime sleepiness—were included in the control group.¹⁸ Thirteen habitual snorers without daytime sleepiness met the normal oximetry criteria and were included in the control group.

Cephalometric Assessments

A lateral cephalometric radiograph was obtained for each patient in the upright position with natural head posture using a pair of earpieces (Chiba University Hospital). Before exposure, the subject was instructed to close the jaw in a natural occlusive position and to breathe quietly. The radiograph was taken at the end of expiration. The exposure parameters were arranged to clearly visualize bony landmarks.

Blind cephalogram analyses were made by an investigator (S.T.). Cephalometric parameters reflecting position and size of the maxilla and mandible were selected. As illustrated in figure 1, the following 10 cephalometric variables were measured and calculated for craniofacial characterization and estimation of tongue size.

Relative position of the mandible to the maxilla: ANB angle = angle between the line from A (subspinale: the deepest point on the anterior surface of the maxillary alveolar bone) to N (nasion) and the line from B (supramentale: the deepest point on the anterior surface of the mandibular alveolar bone) to N.

Maxillomandible dimensions and lower face cage:

1. Maxilla length (Cd-A) = distance between the medial condylar point of the mandible (Cd) and point A
2. Mandible length (Cd-Pog) = distance between Cd and pogonion (Pog: the most prominent point of the anterior surface of the mandibular symphysis in respect to the mandibular plane)
3. Developmental direction of the mandible: angle between Cd-A and Cd-Pog (angle A-Cd-Pog)
4. Axial component of the mandible length (Cd'-Pog) = mandible length projected to Cd-A line calculated as $Cd-Pog \cdot \cos(A-Cd-Pog)$
5. Longitudinal component of the mandible length (Cd-Cd') = mandible length projected to vertical line to Cd-A calculated as $Cd-Pog \cdot \sin(A-Cd-Pog)$

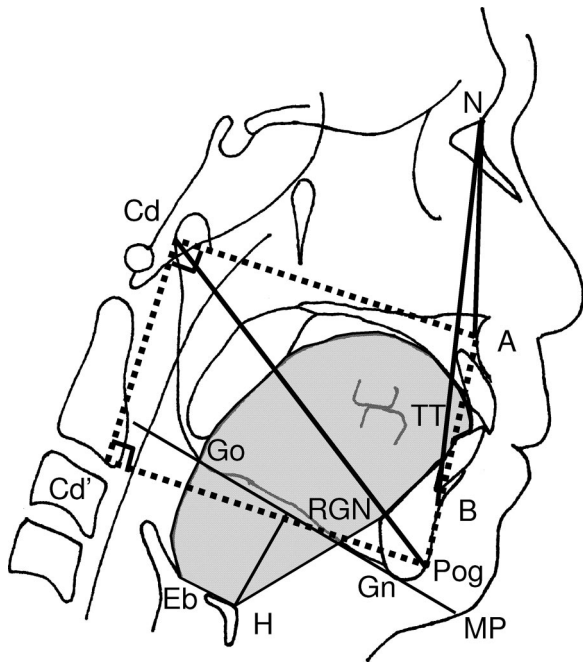


Fig. 1. Definitions of cephalometric variables. A = subspinale; B = supramentale; Cd = medial condylar point of the mandible; Cd' = a point that Pog projects on the perpendicular line to the Cd-A line at the Cd point; Eb = base of epiglottis; Go = gonion; Gn = gnathion; H = hyoid bone; MP = mandibular plane; N = nasion; Pog = pogonion; RGN = retrognathion; TT = tongue tip. *Shaded area* indicates tongue cross-sectional area. Lower face cage was defined as a trapezoid formed by Cd-A-Pog-Cd' (dotted lines).

6. Size of lower face cage—cross-sectional area (LF-CSA; dotted trapezoid in fig. 1): CSA of a trapezoid enclosed by Cd-A-Pog-Cd' for assessment of bony enclosure size of the upper airway.

Hyoid position: distance MP-H for evaluation of position of the hyoid bone = perpendicular distance from anterosuperior point of the hyoid bone (H) to mandibular plane (MP).

Tongue size: cross-sectional area (T-CSA: shaded area in fig. 1) outlined by the dorsum of the tongue surface and lines that connect TT (tongue tip), RGN (retrognathion), H, and Eb (base of epiglottis).

Assessments of Upper Airway Anatomical Balance. Difference of the upper airway anatomical balance was assessed by comparison of the tongue size between craniofacial dimension-matched control and OSA groups. Because of wide range of craniofacial dimensions, the upper airway anatomical balance was also assessed in each of the following three subgroups with different craniofacial dimensions: small LF subgroup (LF-CSA ≤ 65 cm²), intermediate LF subgroup (65 cm² < LF-CSA < 71 cm²), and large LF subgroup (LF-CSA ≥ 71 cm²). Furthermore, the tongue size was compared between body mass index (BMI) and craniofacial dimension-matched control and OSA subgroups.

Statistics

Statistical analyses were performed by using a computer software (SigmaStat for Windows Version 3.11; Systat Software, Inc., Point Richmond, CA). Statistical differences between the control and OSA groups were assessed by Mann-Whitney rank sum test. Comparison between the subgroups was performed by Kruskal-Wallis one-way analysis of variance on ranks. All pairwise comparisons were performed by the Dunn method. A value of $P < 0.05$ was considered to be significant. All values are expressed as median (10th/90th percentiles).

Results

Verification of Matching Craniofacial Dimensions between the Groups

Anthropometric characteristics and results of the sleep study are presented in table 1. Age and height did not

Table 1. Anthropometric Characteristics and Results of Sleep Study

	CF Matched		CF and BMI Matched	
	Control	OSA	Control	OSA
n	55	50	13	13
Age, yr	49 (26/71)	48 (34/68)	52 (31/70)	49 (35/66)
Height, m	1.7 (1.6/1.8)	1.7 (1.6/1.8)	1.7 (1.6/1.8)	1.7 (1.6/1.8)
Weight, kg	63 (54/78)	82 (69/112)*	73 (57/81)	73 (64/88)
BMI, kg/m ²	22 (20/26)	28 (25/37)*	25 (23/28)	25 (23/28)
Neck circumference, cm	38 (35/40)	42 (40/45)*	38 (36/42)	41 (39/44)*
AHI, episodes/h	NA	63 (32/93)	NA	56 (32/90)
4% ODI, episodes/h	1 (0/3)	37 (21/77)*	1 (0/3)	34 (16/58)*
CT ₉₀ , %	0 (0/1)	22 (4/57)*	0 (0/0)	6 (3/23)*
Nadir SpO ₂ , %	92 (89/94)	86 (79/90)*	92 (89/94)	89 (84/90)*
Lowest SpO ₂ , %	88 (79/93)	64 (40/80)*	90 (84/93)	75 (53/84)*

Values are median (10th/90th percentiles).

* $P < 0.05$ vs. control.

AHI = apnea-hypopnea index; BMI = body mass index; CF = craniofacial dimension; CT₉₀ = percentage of time spent at oxygen saturation less than 90%; NA = not applicable; nadir SpO₂ = mean nadir of oxygen desaturations; 4% ODI = oxygen desaturation index (number of episodes per hour that oxygen saturation declined by more than 4% from the baseline); OSA = obstructive sleep apnea; SpO₂ = oxygen saturation.

Table 2. Results of Cephalometric Analyses

	CF Matched		CF and BMI Matched	
	Control	OSA	Control	OSA
n	55	50	13	13
ANB angle, °	5 (2/8)	5 (1/10)	4 (1/8)	6 (2/9)
Maxilla length: Cd-A, mm	96 (91/101)	96 (92/101)	97 (92/102)	96 (92/100)
Mandible length: Cd-Pog, mm	125 (116/131)	125 (115/132)	126 (116/131)	123 (118/131)
Mandible direction: A-Cd-Pog, °	33 (29/37)	34 (30/38)	33 (30/35)	33 (30/38)
Cd'-Pog, mm	105 (97/111)	103 (93/114)	108 (99/111)	103 (95/113)
Cd-Cd', mm	68 (60/74)	69 (62/77)	66 (59/73)	68 (61/76)
Lower face cage: LF-CSA, cm ²	68 (60/77)	68 (61/78)	66 (60/75)	66 (63/73)
Tongue size: T-CSA, cm ²	34 (31/40)	39 (35/44)*	34 (31/40)	39 (36/41)*
Hyoid position: MP-H, mm	16 (8/24)	24 (13/32)*	16 (6/20)	26 (11/31)*

Values are median (10th/90th percentiles). See figure 1 for definitions of the cephalometric variables.

* $P < 0.05$ vs. control.

BMI = body mass index; CF = craniofacial dimension; CSA = cross-sectional area; LF = lower face; OSA = obstructive sleep apnea; T = tongue.

differ between the groups. OSA patients were more obese and had larger neck circumference than control subjects. Polysomnogram revealed the presence of severe OSA accompanied with severe desaturations in OSA patients. Absence of sleep-disordered breathing was confirmed by few episodes of nocturnal desaturations in control subjects.

Results of cephalometric analyses are presented in table 2. Relative position of the mandible to the maxilla and maxillomandibular dimensions were heterogeneous, ranging widely in both the control and OSA groups, and did not differ between the groups. The size of the lower face cage did not differ between the groups, verifying matching craniofacial dimensions and bony enclosure size surrounding the upper airway between the groups.

Tongue Size and Upper Airway Anatomical Balance

Compared with the craniofacial dimension-matched non-OSA subjects, a significantly larger tongue was evident in OSA patients, indicating anatomical imbalance surrounding the upper airway (table 2). Results indicate a more caudal location of the larger tongue in OSA

patients, expanding from the lower face cage as evident from a significantly longer MP-H distance. These results are clearly demonstrated in figure 2, which shows non-OSA and OSA subjects with equivalent lower face size but different tongue CSA.

Subgroup Analyses Based on Lower Face Cage Size

Results of subgroup cephalometric analyses are presented in table 3. While the maxillomandibular dimensions were significantly larger in the larger LF subgroups by the definitions, the dimensions did not differ between non-OSA and OSA patients within each of the subgroups, indicating successful matching of craniofacial dimensions in the subgroups. While the tongue was larger in subjects with larger lower face cage, the tongue was significantly larger in OSA patients than non-OSA subjects by 3–5 cm², indicating excessive upper airway soft tissue for a given craniofacial size in OSA patients (fig. 3). Noticeably, the tongue of non-OSA subjects with larger lower face cage did not differ from that of OSA patients with smaller lower face cage, suggesting little significance of absolute tongue size and significance of imbalance of tongue size and lower face cage for development

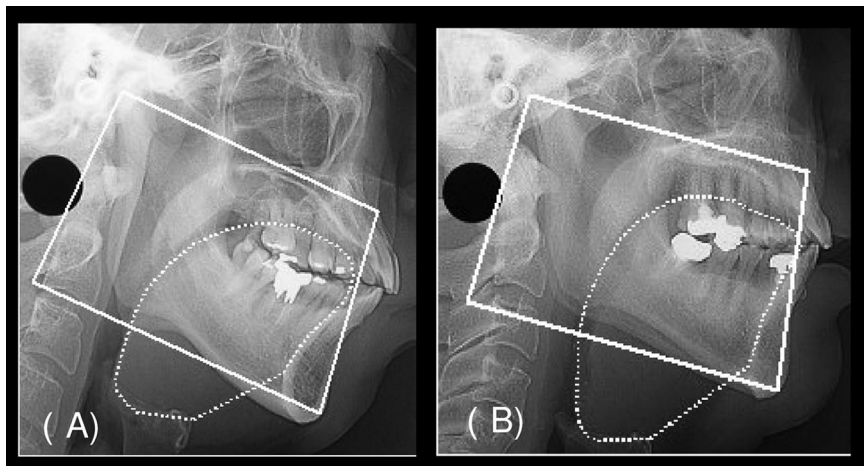


Fig. 2. Representative cephalograms illustrating interaction between craniofacial dimensions and tongue size for obstructive sleep apnea development. The obstructive sleep apnea patient (A) had equivalent size of the lower face cage (78 vs. 77 cm²) but had larger tongue cross-sectional area (41.5 vs. 35.0 cm²) compared with the non-obstructive sleep apnea subject (B).

Table 3. Results of Subgroup Cephalometric Analyses

	Small LF Subgroup		Intermediate LF Subgroup		Large LF Subgroup	
	Control	OSA	Control	OSA	Control	OSA
n	20	12	17	19	18	19
ANB angle, °	4 (1/7)	6 (2/10)	4 (2/8)	5 (-1/10)	5 (2/9)	5 (2/8)
Maxilla length: Cd-A, mm	94 (91/99)	95 (91/100)	96 (92/99)	95 (91/100)	99 (93/108)†	97 (92/103)
Mandible length: Cd-Pog, mm	120 (113/127)	117 (111/127)	123 (119/129)	121 (118/131)	130 (123/134)†‡	129 (124/138)†‡
Mandible direction: A-Cd-Pog, °	30 (29/37)	34 (27/36)	34 (31/39)†	33 (30/38)	34 (32/39)†	35 (32/38)
Cd'-Pog, mm	102 (95/110)	97 (90/113)	103 (97/111)	102 (94/113)	108 (99/113)	105 (98/116)††
Cd-Cd', mm	61 (57/65)	63 (57/68)	69 (66/72)†	67 (65/73)	72 (69/76)†‡	74 (69/79)†‡
Lower face cage: LF-CSA, cm ²	61 (57/66)	62 (57/64)	68 (66/71)†	67 (65/70)†	74 (71/80)†‡	74 (72/82)†‡
Tongue size: T-CSA, cm ²	33 (29/37)	36 (33/39)*	34 (31/39)	39 (35/43)*†	37 (31/41)†	41 (38/45)*†‡
Hyoid position: MP-H, mm	16 (7/21)	25 (14/34)*	17 (8/25)	27 (20/34)*	16 (10/26)	22 (11/31)

Values are median (10th/90th percentiles). See figure 1 for definitions of the cephalometric variables.

* *P* < 0.05 vs. control. † *P* < 0.05 vs. small lower face (LF) subgroup. ‡ *P* < 0.05 vs. intermediate LF subgroup.

CSA = cross-sectional area; OSA = obstructive sleep apnea; T = tongue.

of OSA. Interestingly, MP-H distance did not significantly differ between non-OSA and OSA in the large LF subgroup.

BMI-matched Subgroup Analysis

By selecting BMI-matched non-OSA subjects and OSA patients from the LF-CSA matched subgroups, we succeeded in matching BMI and craniofacial dimension between 13 non-OSA and 13 OSA patients (table 1 and 2). Neck circumference was longer in the OSA subgroup than in the non-OSA subgroup. A larger tongue and a more caudal location of the hyoid were evident in OSA patients.

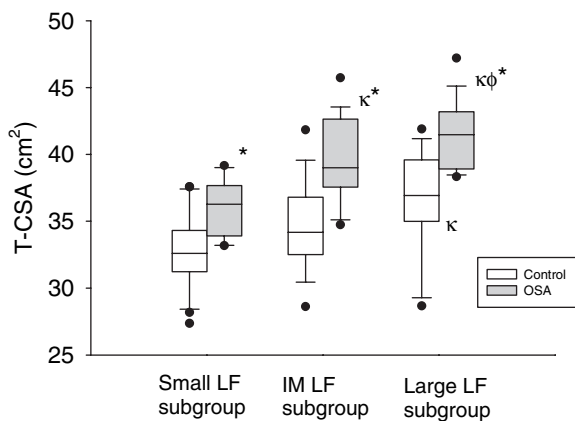


Fig. 3. Box plots showing differences of the tongue cross-sectional area (T-CSA) between non-obstructive sleep apnea subjects (control) and patients with obstructive sleep apnea (OSA) for each of three subgroups defined based on the lower face cage (LF) cross-sectional area (CSA). Lower and upper boundaries indicate 25th and 75th percentages. A solid line within the box marks the median, and vertical lines indicate the 10th and 90th percentages. Solid circles are outliers. Definitions of the subgroups: small LF subgroup (LF-CSA ≤65 cm²), intermediate (IM) LF subgroup (65 cm² < LF-CSA < 71 cm²), large LF subgroup (LF-CSA ≥71 m²). * *P* < 0.05 versus control. κ *P* < 0.05 versus small LF subgroup. φ *P* < 0.05 versus IM LF subgroup.

Discussion

This is the first study that compares tongue size between craniofacial dimension-matched non-OSA subjects and OSA patients, including wide range of craniofacial characteristics from normal to abnormal. While the tongue was significantly larger in subjects with larger maxillomandible dimensions, OSA patients had a significantly larger tongue for a given maxillomandible size, suggesting that the anatomical imbalance surrounding the upper airway contributes to development of OSA.

Limitations of the Study

Our structural analysis was two-dimensional and did not include the whole upper airway structure possibly involved in development of OSA, indicating a major methodologic drawback for investigation of OSA pathophysiology, unlike sophisticated three-dimensional analyses.^{16,17} Nevertheless, it was surprising to find upper airway anatomical imbalance despite the methodologic limitation. Although our approach may have an advantage for clinical applicability and usefulness over three-dimensional analyses, future studies including volumetric three-dimensional analyses are necessary to provide conclusive evidence for upper airway anatomical imbalance as an OSA pathogenesis.

Another major limitation is the potential bias in selecting non-OSA control subjects. Polysomnography would have been preferable to exclude OSA from potential control subjects, because subjects with craniofacial abnormalities known to be associated with OSA were actively included.⁶⁻¹¹ However, control subjects were recruited from three different populations and do not represent a true non-OSA population. However, recognizing this potential bias, we invited subjects with difficult tracheal intubation to increase the number of control subjects with craniofacial abnormalities.¹⁹ In reality, it is difficult to obtain numerous control subjects with craniofacial abnormalities from the general adult popu-

lation to match craniofacial dimensions between the groups and test our hypothesis.

The study population was limited to Japanese adult men. Because of the differences of BMI and craniofacial characteristics between sex and race, our results may not be completely applicable to females and other races. White OSA patients are more obese and have larger maxillomandible dimensions than Asian OSA patients,^{20,21} agreeing with the upper airway anatomical balance concept across races. However, obesity may contribute to the development of OSA by reduction of lung volume in addition to excessive upper airway soft tissue.²² Differences of respiratory chemosensitivity²³ and reflex control of the upper airway muscles²⁴ among the subjects were not assessed and controlled in this study, which may have produced a significant overlap of tongue size between non-OSA and OSA for a given craniofacial dimension clearly evident in figure 3.

Significance of Anatomical Imbalance for Development of OSA

As indicated by significant association between tongue size and lower face cage, absolute tongue size alone may contribute to development of OSA less than the relative tongue size representing balance between the amount of soft tissue and bony enclosure size surrounding the upper airway. Shelton *et al.*²⁵ reported that both mandible enclosure size and body weight are important determinants of the number of OSA episodes. We extended their concept and confirmed that anatomical balance between the amount of soft tissue inside the maxillomandibular enclosure and the bony enclosure size determines the pharyngeal airway size.^{12,13,26} Anatomical imbalance is not usually evident during wakefulness and pharyngeal airway patency is maintained mainly by neural compensatory mechanisms in OSA patients.²⁴ The anatomical balance model presented in figure 4 may be helpful for conceptualizing and understanding the interaction be-

tween neural and anatomical mechanisms, while it ignores numerous factors that may influence pharyngeal airway patency.^{12,13,27}

Structural Interaction Surrounding the Pharyngeal Airway

Although heterogeneous craniofacial features among humans may be determined by both genetic and environmental influences, development of craniofacial structures is generally accomplished by 20 yr of age, when prevalence of OSA is low.²⁸ Prevalence of both obesity and OSA increases during middle age.²⁹ Because one of the two anatomical balances, *i.e.*, bony enclosure size, in figure 4 is relatively constant during adulthood, our results suggest that OSA could develop in subjects with excessive upper airway soft tissue caused possibly by being overweight.

Our results completely agree with those recently reported by Schwab *et al.*,¹⁷ who performed magnetic resonance imaging three-dimensional volumetric measurements of soft tissue surrounding the upper airway in both control and OSA subjects. After statistical adjustments for sex, race, age, craniofacial size, and parapharyngeal fat, they estimated significant differences of tongue and total upper airway soft tissue between control and OSA subjects by 17 cm³ and 21 cm³, respectively. It should be noted that the difference is close to the 5-cm² difference of tongue CSA between control and OSA subjects found in this study, assuming both maxillary and mandibular arch width to be approximately 3–5 cm.³⁰ In addition to confirming their results, we first demonstrated interaction between the craniofacial structures and soft tissue surrounding the pharynx for determining OSA development in craniofacial dimension-matched non-OSA and OSA groups.

Although the maxillomandible bony enclosure and the cranial base limit soft-tissue expansion, the submandibular area does not have the bony structure of this region, thereby permitting caudal expansion of the excessive soft

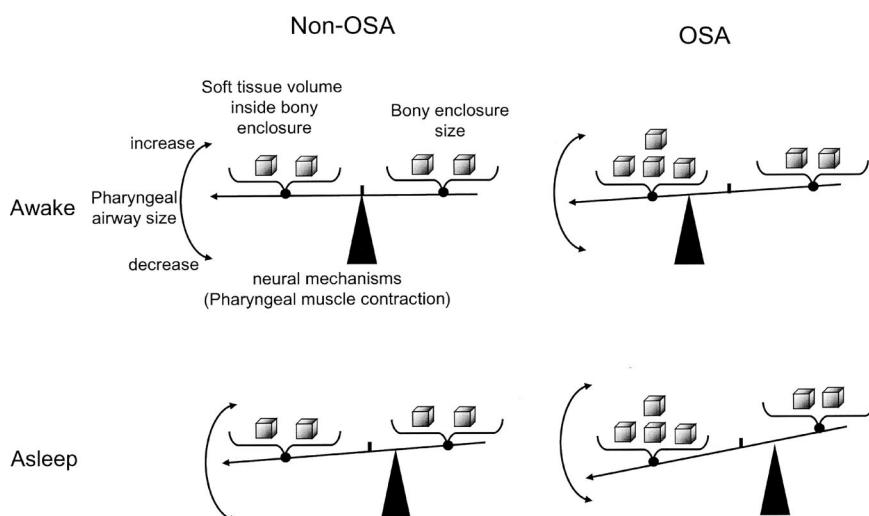


Fig. 4. Schematic model explaining pharyngeal airway patency, showing soft-tissue volume inside bony enclosure and bony enclosure size on either side of fulcrum that represents magnitude of pharyngeal muscle contraction, *i.e.*, neural mechanisms. Interaction between the anatomical balance and neural mechanisms determines pharyngeal airway size. Increased neural mechanisms can compensate the anatomical imbalance in obstructive sleep apnea (OSA) patients during wakefulness. When the neural mechanisms are suppressed during sleep and anesthesia, pharyngeal airway severely narrows because of the anatomical imbalance in OSA patients.

tissue. The hyoid bone was demonstrated to be located more caudally in OSA patients in accord with previous studies.^{11,12} Because the human hyoid bone has no connection to other bones and is mobile, caudal expansion of soft tissue resulting from excessive soft tissue within the maxillomandible enclosure may be responsible for shifting the hyoid bone caudally. It is our belief that caudal displacement of the hyoid bone reflects upper airway anatomical imbalance in OSA patients.

Clinical Implications

While a variety of physical examinations are proposed as potential predictors of OSA, modified Mallampati score, thyromental angle, and cricomental space are reported to be better differentiating variables of OSA and non-OSA.^{31,32} The results of this study provide a pathophysiologic explanation for the usefulness because these assess relative excessiveness of soft tissue surrounding the upper airway and agree with the anatomical balance model. Furthermore, our cephalometric analysis could serve as an alternative preoperative predictor by measuring both maxillomandibular dimension and tongue CSA in addition to position of the hyoid bone.

Within one subject, the pharyngeal bony enclosure size varies with head and mandible positioning changes, possibly improving the anatomical imbalance observed in OSA patients. Head extension, the sniffing position, and mandible advancement increase the distance between the mentum and the cervical column increasing the bony enclosure size. We previously demonstrated significant improvement of pharyngeal closing pressures in response to these position changes in anesthetized and paralyzed OSA patients.^{13,26,33} Furthermore, the sitting position may improve the anatomical balance by displacing the excessive soft tissue outside the bony enclosure through the submandibular space. The upper airway anatomical balance concept may provide the base for establishing strategies for perioperative airway management of OSA patients.

This study demonstrated that OSA patients have excessive soft tissue for a given craniofacial dimension. An upper airway anatomical balance model was proposed for better understanding the pathophysiology of pharyngeal obstruction and OSA.

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