

Differences of Upper Airway Morphology According to Obesity: Study with Cephalometry and Dynamic MD-CT

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Objectives. We investigated difference of parameters of polysomnography, cephalometry and dynamic multi-detector computerized tomography (MD-CT) in wake and sleep states according to obesity.

Methods. We evaluated 93 patients who underwent polysomnography and cephalometry. MD-CT was performed in 68 of these 93 patients. Fifty-nine and 34 patients were classified as obese and non-obese, with obesity defined as BMI ≥ 25 . Cephalometry results were analyzed for 12 variables. Using the MD-CT, we evaluated dynamic upper airway morphology in wake and sleep states and divided the upper airway into four parts named as high retropalatal (HRP), low retropalatal (LRP), high retroglottal (HRG), and low retroglottal (LRG). A minimal cross sectional area (mCSA) and collapsibility index (CI) were calculated for each airway level.

Results. Diastolic blood pressure ($P=0.0005$), neck circumference ($P<0.0001$), and apnea-hypopnea index ($P<0.0001$) were statistically significantly different between the obese and non-obese group. Among 12 cephalometric variables, there was a significant difference in only the distance from mandibular plane to hyoid bone ($P=0.003$). There was statistical difference in CI of HRG and LRG in sleep state ($P=0.0449, 0.0281$) but no difference in mCSA in wake and sleep states.

Conclusion. The obese group had more severe sleep apnea than the non-obese group. We believe that the increased severity of apnea in the obese group may be have been due to increased collapsibility of the upper airway rather than decreased size of the upper airway.

Key Words. Snoring, Sleep apnea, CT scan, Obesity

INTRODUCTION

Apneas and hypopneas associated with obstructive sleep apnea syndrome (OSAS) are due to recurrent upper airway obstruction during sleep. The cause of upper airway obstruction involves both anatomic factors and pharyngeal collapsibility. The syndrome is associated with loud snoring, sleep fragmentation and excessive daytime sleepiness.

It has been reported that 4% of men and 2% of women in the United States are affected by obstructive sleep apnea (1). The incidence is similar in Korea (2).

It is well known that obesity is the most significant predisposing factor for OSAS. Previous studies have found a close connection between obesity and OSAS (3, 4). One study reported that weight loss is related to increased upper airway cross-sectional area, which may improve apnea-hypopnea index (AHI) (5).

Methods for viewing the obstructed area of the upper airway include cephalometry, computed tomography (CT), magnetic resonance imaging (MRI), nasopharyngoscope, fluoroscopy and electron beam tomography (EBT). These methods have their own advantages and disadvantages (6). Cephalometry is a useful tool to examine craniofacial and upper airway soft tissue morphology in OSAS patients, but only produces static two-dimensional

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images. Several studies have compared obese and non-obese patients using cephalometry (7-11), but the usefulness of their results was limited as the upper airway changes dynamically during sleep. We previously reported that EBT and multi-detector computerized tomography (MD-CT) were useful methods for effectively viewing dynamic changes of the upper airway (12).

The aim of this study was to identify the correlation between the obese and non-obese group by measuring apnea severity and anatomical features, using polysomnography, cephalometry, and dynamic MD-CT.

MATERIALS AND METHODS

Patients

Ninety-three OSAS patients who complained of snoring and sleep apnea were enrolled in this retrospective study. All subjects underwent both polysomnography and cephalometry and 68 underwent MD-CT study. Body weight and height were recorded and the body mass index (BMI) was calculated as follows: $BMI = \text{weight (kg)} / \text{height (m)}^2$. Based on the BMI value, the subjects were classified into two groups: non-obese ($BMI < 25$, $n=34$) and obese ($BMI \geq 25$, $n=59$). The patients who underwent MD-CT ($n=68$) were also classified as obese ($n=50$) or non-obese ($n=18$). The obese group consisted of 51 men and 8 women, with a mean age of 45 years. The non-obese group consisted of 24 men and 10 women, with a mean age of 41 years.

Polysomnography

One-night polysomnography (Beehive Millenium, Grass-Telefactor Inc., West Warwick, RI, USA) was performed in each patient and included electroencephalogram (EEG), bilateral electro-oculogram (EOG), submental electromyogram (EMG), nasal and oral airflow, electrocardiogram (ECG), chest and abdominal respiratory effort using inductive plethysmography, and oxygen saturation by finger oximetry. Apnea was defined as cessation of oronasal airflow for more than 10 seconds. Hypopnea consisted of a more than 50% decrease in airflow and with at least 4% decrease in oxygen saturation. Severity of OSA was judged in terms of AHI. The AHI was defined by the frequency of apnea and hypopnea episodes per hour during the total sleep time. We diagnosed OSA in cases where AHI exceeded 5 per hour.

Cephalometry

A lateral cephalometric radiograph was obtained of each subject. Films were taken in a sitting position at a focus distance of 70 inches. Exposures were made at 70 kv at the end-expiratory phase during quiet breathing using CX90SP (Ashahi, Tokyo, Japan). Twelve standard bony and soft-tissue measurements, commonly reported to show changes in OSAS, were obtained. First, the following measurements were made: distance from

mandibular plane to hyoid bone (MP-H), distance from velum tip to pharyngeal wall parallel to Frankfurt horizontal line (PAS), posterior nasal spine to velum tip-soft palate length (SPL), condyle to gnathion-total mandibular length (Man TL), gonion to gnathion-mandibular body length (Man BL), and condyle to gonion-mandibular ramus height (Man Ht). The soft palate-soft palate thickness (SPT, widest measurement), pharyngeal length (PhL), the smallest anteroposterior dimension of the retropalatal space (RP), and the retroglossal space (RG) were also gauged. Finally, the sella-nasion-supradentale (SNA) and sella-nasion-infradentale (SNB) angles were measured.

Dynamic MD-CT

Scanning with MD-CT (Light speed ultra 16, GE medical systems, Milwaukee, WI, USA) was performed. First, the neck, designated from the hard palate to the epiglottis, was divided into 6 to 8 levels depending on the patient's neck length. Scanning was repeated 25 times at each level with an interval of 0.3 seconds. The scan time for each image was 0.4 seconds. Thereafter, Dormicum® (midazolam 0.1 mg/kg, Rocho, Seoul, Korea) was intravenously injected and filming repeated as above when the patient was upon onset of snoring.

The area from the hard palate to the tip of epiglottis of the upper airway was divided into two parts based on the end of the uvula and these two parts were subdivided into two equal parts; the resulting four areas were subsequently referred to as the high retropalatal (HRP), low retropalatal (LRP), high retroglossal (HRG), and low retroglossal (LRG).

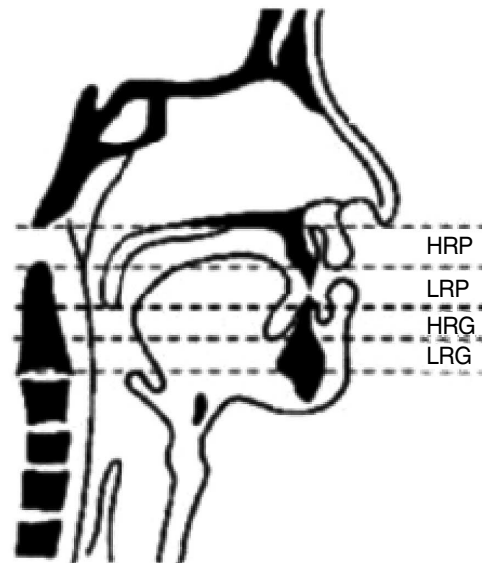


Fig. 1. Schematic lateral view of the upper airway. The site of obstruction is classified as one of four levels. The area from the inferior border of the hard palate to the inferior border of the uvula is classified as the retropalate level; the area from the inferior border of the uvula to the superior border of the epiglottis tip as the retroglossal level. Each of these two levels are further divided into 'high' and 'low' levels. HRP: high retropalatal area; LRP: low retropalatal area; HRG: high retroglossal area; LRG: low retroglossal area.

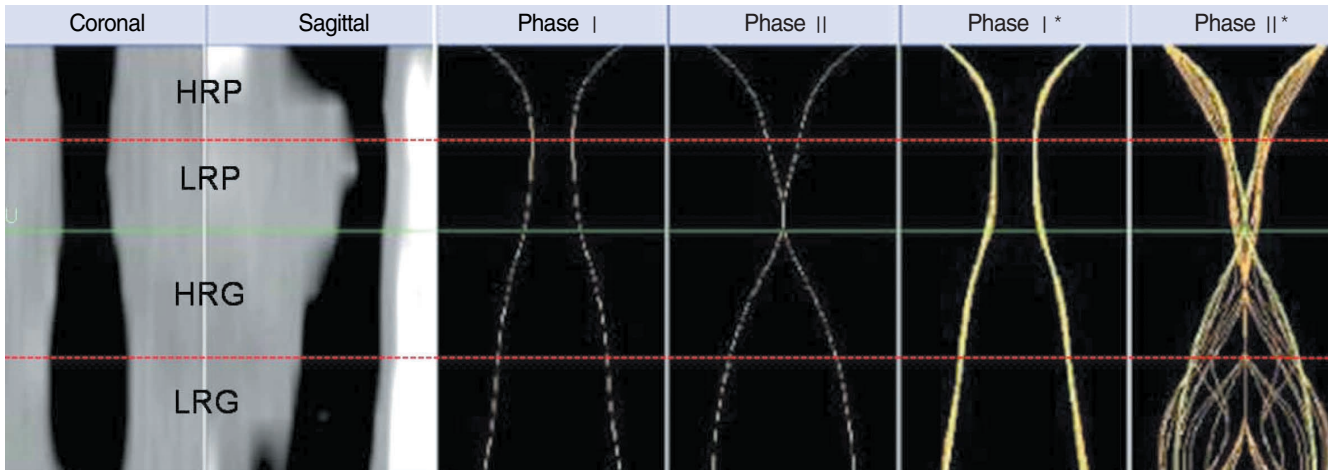


Fig. 2. Simulated longitudinal display of the oropharynx showing the results of multi-detector computerized tomography in wake and sleep states. coronal: coronal scout view of oropharynx; sagittal: sagittal scout view of oropharynx; phase I: minimal cross-sectional area (mCSA) of oropharynx in wake state; phase II: mCSA of oropharynx in sleep state; phase I*: dynamic imaging of mCSA according to respiration in wake state; phase II*: dynamic imaging of mCSA according to respiration in sleep state; HRP: high retropalatal area; LRP: low retropalatal area; HRG: high retroglossal area; LRG: low retroglossal area. The U indicates uvular levels of oropharynx.

Table 1. Demographic and sleep data for obese and non-obese patients with obstructive sleep apnea

	Obese (n=59)	Non-obese (n=34)	P-value
BP			
Systolic	132.9±15	128.3±15.8	0.1666
Diastolic	83.2±9.7	75.4±10.4	0.0005*
NC			
Sitting	41±2.8	36.6±3.2	<0.0001*
Lying	41.9±2.9	37.4±3.1	<0.001*
AHI			
Supine	48.7±23.3	23.9±20.7	<0.0001*
Non-supine	23.4±20.8	8.6±16.9	0.0016*
Total	38.2±20.6	17.5±16.6	<0.0001*

*P<0.05.

BP: blood pressure; NC: neck circumference; AHI: apnea-hypopnea index.

roglossal (HRG), and low retroglossal (LRG) areas (Fig. 1). With the computerized data obtained from MD-CT, the maximal and minimal cross sectional area (MCSA and mCSA) of each of these four areas was measured and the degree of movement of the upper airway according to respiration was calculated by the collapsibility index $[CI=(MCSA - mCSA)/MCSA \times 100]$.

Changes of the cross sectional area of the upper airway were visualized by a newly developed computer program which drew a simulated diagram of the longitudinal view using cross-sectional area data (Fig. 2).

Statistic analysis

Statistical analysis was performed using the SPSS ver. 15.0 (SPSS Inc., Chicago, IL, USA). The difference in demographic, polysomnographic, cephalometric and MD-CT data was analyzed using univariate analysis (parametric unpaired *t*-tests).

Table 2. Cephalometric comparison between obese and non-obese patients with obstructive sleep apnea

	Obese	Non-obese	P-value
SNA (°)	83.6±4.6	84.8±4.9	0.2511
SNB (°)	81.4±4.0	82.0±4.2	0.5307
PAS (mm)	12.6±3.2	11.3±3.0	0.0883
SPL (mm)	41.6±4.0	40.1±5.1	0.2137
SPT (mm)	10.9±2.0	10.6±2.4	0.6115
MP-H (mm)	20.5±6.9	15.5±7.4	0.003*
RP (mm)	7.5±2.6	7.0±2.5	0.3987
RG (mm)	10.6±3.3	9.6±2.9	0.1413
Man TL (mm)	125±6.6	120±21	0.1218
Man Ht (mm)	68.2±5.6	67.8±6.4	0.7666
Man BL (mm)	80.1±5.2	78.3±5.6	0.1568
PhL (mm)	61.1±8.0	62.1±12.0	0.6456

*P<0.05.

SNA: sellanasion-supradentale; SNB: sella-nasion-infradentale; PAS: posterior airway space; SPL: soft palate length; SPT: soft palate thickness; MP-H: mandibular plane to hyoid bone; RP: retropalatal; RG: retroglossal; Man TL: mandibular total length; Man Ht: mandibular ramus height; Man BL: mandibular body length; PhL: pharyngeal length.

The level of statistical significance was defined as *P*<0.05.

RESULTS

Demographic and sleep data

The demographic and polysomnographic data for each group are summarized in Table 1. The mean blood pressure (BP), neck circumference (NC), and AHI in the obese group were higher than those found in the non-obese group. Statistically significant differences were observed in BP, NC, and AHI between the obese

Table 3. Minimal cross sectional area (mCSA) at four levels of upper airway during the wake and sleep states between obese and non-obese patients with obstructive sleep apnea

	Obese (n=50)	Non-obese (n=18)	P-value
Wake (mm ³)			
HRP	255.6 ± 154.4	276.8 ± 118.4	0.6004
LRP	87.5 ± 52.4	101.7 ± 48.0	0.3197
HRG	215.5 ± 101.3	192.6 ± 89.1	0.4017
LRG	278.5 ± 142.3	250.8 ± 120.4	0.4662
Volume	16,146.2 ± 5,725.9	15,180.5 ± 4,094.7	0.5146
Sleep (mm ³)			
HRP	176.8 ± 138.0	211.1 ± 122.9	0.3499
LRP	17.4 ± 29.5	23.7 ± 22.3	0.4138
HRG	50.5 ± 103.5	53.1 ± 57.5	0.9193
LRG	90.9 ± 77.3	114.2 ± 80.0	0.2821
Volume	7,636.8 ± 4,624.4	8,350.1 ± 3,655.4	0.5577

HRP: high retropalatal area; LRP: low retropalatal area; HRG: high retroglottal area; LRG: low retroglottal area.

and the non-obese group, except for systolic BP.

Cephalometric measurements

In comparison to the non-obese group, the obese group had higher PAS, SPL, SPT, MP-H, RP, RG, Mn TL, and Mn Ht values. However, only the MP-H value of the obese group was statistically significant higher ($P=0.003$) (Table 2).

Dynamic MD-CT

All sleep state mCSA measurements in the obese group were lower than those in the non-obese group. However, there was no statistical difference in wake or sleep state mCSA measurements (Table 3). During the wake state, there was no statistical difference in CI values. During the sleep state, however, statistical difference was seen in HRG, LRG, volume and overall CI values ($P=0.0449$, 0.0281 , 0.0107 , and 0.0209 , respectively) (Table 4).

DISCUSSION

The pathogenesis of OSA is known to be very complex and is not yet fully understood. The major pathophysiology is sleep-related collapse of the upper airway (13), with anatomic factors and pharyngeal collapsibility known to play a part.

Several methods have been employed to locate regions of upper airway obstruction: cephalometry, fiberoptic nasopharyngoscopy, fluoroscopy, CT and MRI (6). Each of these methods has several disadvantages. Cephalometry produces only two-dimensional static images and allows anteroposterior dimension measurement, but not lateral dimension measurement. While MRI can provide a cross-sectional image of organs, the required data acquisition time is long, with resultant motion artifacts due to

Table 4. Collapsibility index (CI) at four levels of upper airway during the wake and sleep states between obese and non-obese with obstructive sleep apnea

	Obese	Non-obese	P-value
Wake (%)			
HRP	26.8 ± 16.9	21.2 ± 17.1	0.235
LRP	43.5 ± 25.3	35.1 ± 20.5	0.2135
HRG	26.3 ± 19.6	20.9 ± 15.7	0.2963
LRG	26.6 ± 21.2	29.2 ± 26.6	0.683
Volume	17.7 ± 13.0	19.6 ± 16.5	0.6206
Overall CI	30.8 ± 15.6	26.6 ± 9.4	0.2878
Sleep (%)			
HRP	52.6 ± 26.2	45.4 ± 20.8	0.2946
LRP	85.0 ± 18.5	75.6 ± 15.0	0.0586
HRG	85.9 ± 19.3	74.9 ± 20.1	0.0449*
LRG	80 ± 18.5	67.7 ± 23.1	0.0281*
Volume	62.6 ± 19.8	48.5 ± 18.3	0.0107*
Overall CI	75.9 ± 16.2	65.9 ± 12.2	0.0209*

* $P<0.05$.

HRP: high retropalatal area; LRP: low retropalatal area; HRG: high retroglottal area; LRG: low retroglottal area.

breathing and swallowing. Cephalometry, MRI and CT are performed in the wake state. The meaningfulness of their results is therefore limited as the upper airway is known to change dynamically during sleep. Fluoroscopy (14), EBT (15), and MD-CT (12), on the other hand, are used for dynamic radiographic examination of the upper airway. The disadvantages of fluoroscopy are that, as with cephalometry, the airway is shown in only two dimensions with measurement possible in the anteroposterior dimension, but not the lateral dimension. Furthermore, fluoroscopic examination leads to significant radiation exposure. In the case of EBT, Ye et al. (15) showed it to have the potential to provide information quickly and with no effect on upper airway dynamics. Comparing EBT and MD-CT, we previously reported that EBT and MD-CT were efficient modalities for dynamic upper airway mechanics, and that the results of MD-CT were similar to those of EBT (12). Additionally, MD-CT had superior resolution and was less expensive in terms of maintenance than EBT.

Obesity is one of the most common predisposing factors in the development and progression of OSA. Previous studies have found a close connection between obesity and OSA (3, 4). Fat pads in the pharyngeal wall are increased in obese patients with OSA, causing narrowing of the pharyngeal lumen (16, 17). Static pharyngeal size modulated by dynamic loading of the airway due to the weight of fatty tissue of the neck may also contribute to the pathogenesis of OSA (18). One study reported weight loss to be related to increase in upper airway cross-sectional area, and that this could improve AHI (5). Consistent with these results, the demographic data from our study shows that the obese group was more affected by OSA than the non-obese group. In another study, 50% of sleep apnea was accompanied

by hypertension (HTN), and 30% of HTN was associated with sleep apnea (19). We found positive association between obesity and hypertension and inferred that there was an interrelationship between hypertension and sleep apnea. The NC was significantly associated with apnea and snoring (20), so larger neck circumference may be associated with greater severity of OSA. The NC of obese patients was statistically significant higher compared to non-obese patients in our study. In another study, the obese patients were shown to have higher AHI values, and a correlation between NC and AHI (21). Our results were consistent with these findings.

It was reported that in both non-obese and obese OSA patients, skeletal changes were often evident in cephalometric values (8). Displacement of the hyoid bone may contribute to the pathogenesis of OSA (21). Ferguson et al. (11) found that increased MP-H is related to increased neck circumference. The hyoid bone is important as an anchorage for the tongue muscle, and increased MP-H distance is the result of increased soft tissue and fat deposition in the tongue. A previous study has reported that OSA patients showed a significant correlation between an increased MP-H distance and a higher AHI (22). However, another study reported that the difference in the position of the hyoid bone in non-obese and obese OSA patients was not significant, and that in obese patients upper airway soft tissue enlargement may play a more important role in the development of obstructive sleep apnea, whereas in non-obese patients, bony structure discrepancies may be the dominant contributing factors for obstructive sleep apnea (10). In our study, inferior shift of the hyoid bone, reflected by increased MP-H, was significantly associated with obesity. This suggests that displacement of the hyoid bone may contribute to OSA.

Two mechanisms for upper airway occlusion during sleep have been put forward: a neural hypothesis and an anatomic theory (23). The neural hypothesis involves decreased neural output to pharyngeal dilator muscles during sleep, leading to muscle hypotonia. The anatomic theory submits that upper airway collapsibility may be affected by soft tissue and body facial anatomy: fat deposits around the pharynx, hypertrophied adenoids and tonsils, macroglossia, retrognathia and micrognathia. Several studies using CT scanning have reported that upper airway collapse occurred at the oropharyngeal level, and that oropharyngeal narrowing at the level of the uvula, soft palate, and retrolingual space was the principle site of obstruction in OSA (24-26). Also, CT scanning during wakefulness may not predict the development of airway obstruction as the upper airway moves dynamically during respiration. In this study, the MD-CT was carried out in the wake and sleep states. We found that retroglottal area (HRG, LRG) in the obese group (wake or sleep states) was not narrower compared with that of the non-obese group, suggesting that anatomical narrowing is not an important factor aggravating OSA in the obese group. However, the CI of HRG and LRG was significantly increased in the obese

group, suggesting that the neural hypothesis is a more important causative factor of OSA.

Sexual differences may have affected these results. Among the 68 patients who underwent MD-CT, 57 were men, and 11 were women. We therefore also compared the obese and non-obese groups after excluding female patients. The resulting data (not shown) was similar to the data which included female patients (data not shown).

The limitation of this study was the use of medications such as midazolam to induce sleep. Sleep inducing medications are known to influence upper airways physiologically and dynamically (27). Furthermore, we were unable to observe the whole period of sleep.

In conclusion, the obese group had more severe sleep apnea than the non-obese group. Furthermore, sleep apnea in obese patients may be aggravated by increased collapsibility of the upper airway rather than decreased size of the upper airway.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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