Effect of Jaw Position on Forced Maximum Inspiratory Airflow in Normal Japanese Subjects and in Japanese Patients with Sleep Apnea Syndrome

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Purpose: The purpose of this study was to evaluate whether maximum forced inspiratory airflow changes occur by changing the jaw position in Japanese normal subjects and patients with obstructive sleep apnea (OSA) classified by their craniofacial features. ***Materials and Methods:*** The subjects included 8 male non-OSA subjects and 15 male patients with OSA whose conditions had been diagnosed with a polysomnographic recording. The OSA subjects were divided into 2 groups by means of a craniofacial (CF) score based on cephalometric variables: a high-score group (CF score ≥ 4) and a low-score group (CF score ≤ 3). A case-control design was utilized to assess group differences (control and 2 patient groups). Airflow changes were determined using a spirometer that assessed the velocity of airflow during forced inspiration. Maximum forced inspiratory airflow was measured in 4 positions in all patients. ***Results:*** All 3 groups had a significant decrease in their maximum forced inspiratory airflow upon reclining, and there were no significant group differences regarding the magnitude of this change. The OSA subjects returned to baseline measurements more than controls when the jaw was positioned forward, as the jaw was progressively advanced in high CF score subjects. ***Conclusion:*** This study suggested that a protrusive jaw position allows more inspiratory airflow to occur in OSA patients compared to controls, and this was significant in the patients with a high CF score. *Int J Prosthodont 2007;20:25–30.*

Obstructive sleep apnea (OSA) is a syndrome characterized by repeated collapse of the upper airway during sleep, resulting in recurrent episodes of apnea. OSA is a common syndrome that affects 1% to 4% of middle-aged men, and the prevalence increases with age up to 60 years. OSA is a complex and multifactorial problem, and some of the most commonly reported risk factors for OSA are morbid obesity, abnormalities of the craniofacial and upper airway structures, familial aggregation, smoking, hormonal differences, alcohol consumption, and nasal congestion at night.[1–10]

Most epidemiologic studies and clinical studies of OSA thus far have involved mainly Caucasians, and there is limited information on OSA in other racial groups.[11–15] Several recent studies have suggested that ethnicity may be an important risk factor in OSA.[7,9,16–28] In one study performed in Chinese patients, it was suggested that since increased body mass index (BMI) is a predictor of sleep-disordered breathing (SDB) and most Asians are not obese, then craniofacial abnormalities may be a more significant risk factor in Asians.[24] In fact, several studies have examined this issue and have supported the idea that craniofacial abnormalities might be a stronger predictor than BMI for OSA in Asians.[7,20,21]

Nasal continuous positive airway pressure (nCPAP) is a highly effective and safe treatment for OSA and is generally considered to be the current primary treatment for it, but a variety of other therapeutic approaches have also been proposed, including such

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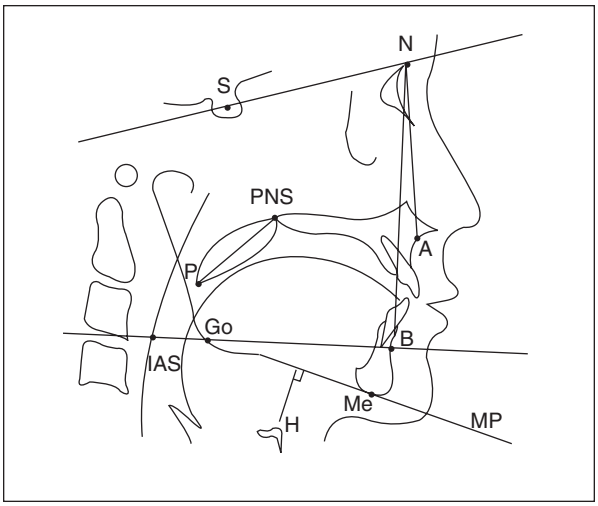

Fig 1 Cephalometric landmarks, reference lines, and abbreviations (from Yao et al[27]). A = point A; B = point B; Go = gonion; H = most anterosuperior point of the hyoid bone; IAS = inferior airway space; Me = menton; MP = mandibular plane; N = nasion; P = lowest point of the soft palate; PNS = posterior nasal spine; S = sella.

surgical treatments as uvulopalatopharyngoplasty, tongue reduction or advancement, and chin-hyoid apparatus advancement; oral appliances; weight loss; medication; head and neck extension collars; and modification of sleep position.[29–32] These treatments have been evaluated for their indication, effectiveness, compliance, and side effects in various publications.[33–38] The oral appliance treatment approach has been effective in some patients with mild to moderate OSA.[33,34] In addition, oral appliances have recently been proven effective (although not as good as nCPAP) even in moderate to severe OSA patients who cannot tolerate or refuse nCPAP.[39,40] Oral appliances are also used after nasal and jaw surgery when adequate results are not achieved through surgery.[41] Several authors report discomfort or strain in the jaw muscles and/or the temporomandibular joint as one potential side effect of an oral device.[39,40,42–55] For these reasons, it is considered necessary to be able to adjust the device for optimal patient management.

The question of concern in this study is: "What is the jaw position at which most of the patients will respond and effectively treat their OSA?" Earlier, the authors evaluated the effect of jaw position and body posture on forced inspiratory airflow (considered an analog for airway caliber changes) in normal subjects and in patients with OSA.[56] This prior study revealed that the jaw protrusion position had a greater effect on maximum forced inspiratory airflow in OSA patients than in normal subjects. The earlier study did not have matched groups for age or BMI, and the craniofacial features of the studied groups were not measured or assessed. In the present study, subjects were matched by age, BMI, gender, and their response to a sleepiness questionnaire, and Japanese OSA patients and Japanese controls were measured and classified according to their craniofacial features. The different effects of jaw position on the maximum forced inspiratory airflow were also assessed. The null hypothesis was that these groups do not differ in regard to these parameters.

Materials and Methods

Subjects

This study involved 15 male patients with OSA who visited Kyushu Dental College Hospital to have an oral appliance fabricated for their OSA. They had received the diagnosis based on polysomnographic recordings. An AHI (Apnea-Hypopnea Index) was obtained from the polysomnographic recordings. The study also included 8 normal, nonsnoring, nonhypersomnolent male volunteers with no history of sleep respiratory disorder. The aim and potential risks of the study were fully explained to each subject, and informed consent was obtained from each according to the procedures and protocol established by Kyushu Dental College Hospital. Before the intervention, a clinical examination of the stomatognathic system, including measurements of mandibular mobility, palpation of the temporomandibular joint and masticatory muscles, and recordings of pain on jaw motion was performed. The Epworth Sleepiness Scale questionnaire was administered to all subjects. Any subject exhibiting symptoms of temporomandibular joint dysfunction, a history of respiratory disease, loss of molars, or occlusal dysfunction was excluded from this study; to eliminate any potential confounding effect of gender, women were also excluded.

Cephalometric Measurements

A lateral cephalogram was obtained from the 15 patients with OSA. A radiograph film holder was placed next to the left side of the head, and the cone of the radiography unit was 1.5 m from the subjects. Two cephalometric variables were measured: distance from hyoid (H) to mandibular plane (MP), and distance from sella (S) to nasion (N) (Fig 1). These measurements were chosen based on a publication that examined craniofacial form and SDB in Japanese patients.[27] Individual craniofacial (CF) scores were then calculated according to the method of Yao and colleagues.[27] This scoring method produces a number that increases when the S-N distance becomes shorter and H-MP becomes longer. Based on this score, the OSA subjects were divided into 2 groups: a high-score group (CF score ≥ 4) and a low-score group (CF score ≤ 3).

Table 1 Demographic Characteristics of Subjects

	Controls (n = 8)	Group A (n = 6)	Group B (n = 9)
Age	30.1 ± 8.7	43.0 ± 14.1	41.8 ± 13.4
BMI (kg/m^2)	22.2 ± 1.8	24.2 ± 2.1	23.9 ± 4.7
ESS	5.4 ± 2.1	8.0 ± 2.5	7.0 ± 4.1
MP (mm)	7.8 ± 1.9	8.8 ± 2.8	9.1 ± 2.5
AHI	NS	15.0 ± 8.8	23.00 ± 17.3

BMI = Body Mass Index; ESS = Epworth sleepiness scale; MP = maximum protrusion; AHI = Apnea-Hypopnea Index; NS = not significant.

Table 2 Forced Inspiratory Flow (FIF_{25-75}) of 3 Groups in Upright and Supine Positions

Position/ amount of flow	Control (n = 8)	Group A (n = 6)	Group B (n = 9)
Upright			
0% F	100	100**	100**
Supine			
0% F	80.5 ± 10.5*	72.5 ± 10.0	76.3 ± 12.6
50% F	84.9 ± 8.7*	94.1 ± 10.0**	104.7 ± 21.8**
75% F	88.7 ± 12.9*	95.3 ± 14.3**	104.1 ± 15.2**

*Significant difference versus upright 0% F;**significant difference versus supine 0% F.

Spirometer

An electric spirometer (Chestgraph Jr, HI-101, Chest) was used to measure the middle portion (25%–75%) of each subject's maximum forced inspiratory flow (FIF_{25-75}). For all measurements, the interincisal opening was standardized at 12 mm with a customized mouth prop between the posterior teeth on one side. This distance was established based on the authors' prior study,[57] which was a pilot study of 5 control subjects that used different thicknesses of mouth prop to determine *(1)* which degree of separation was most comfortable for the subjects and *(2)* which thickness consistently allowed an FIF_{25-75} that was 90% or more of the value achieved for a maximum effort without a mouth prop. Based on these data, the amount of the interincisal opening at the anterior tooth region was standardized at 12 mm to avoid any dental restriction of airflow. To fabricate these bite-positioning blocks, individual plaster casts of both jaws were made and mounted into a centric occlusal record on an articulator.

A special jig was also fabricated to measure how much each patient could protrude the mandible. The jig was made from an acrylic resin block (18 × 30 × 12 mm) and fixed on the mandibular teeth. The habitual closure and maximum protrusive positions were marked on the jig, and then the 0% forward (F), 50% F, and 75% F positions were indicated. Each subject was given an individual set of bite-positioning blocks and a nose clip. Before each experimental session, the subjects were asked to practice making maximum inhalations and exhalations to familiarize themselves with the spirometric equipment and the wearing of the bite-positioning block. All subjects performed maximum FIF_{25-75} maneuvers with the 0% protrusive position in the upright position (upright 0% F) and 0%, 50%, and 75% protrusive positions in the supine position (supine 0% F, 50% F, 75% F). All supine measurements were performed with the subjects lying on a flat bed with the head supported by a pillow constructed of soft sponge material and designed to facilitate the subjects' natural head-to-body positions during their habitual supine sleep posture. Measurement of each posture and mandibular position was done 3 times by random assignment, accomplished using a randomization table.

Statistical Analysis

The patients were divided into 2 groups by CF score levels. Group A had a low CF score, and group B had a high CF score. These 2 groups were compared to the control subjects. The statistical significance of the differences in AHI between the patient groups was evaluated with the *t* test. The FIF_{25-75} data in the upright 0% protrusive position were used as a control, and all subsequent values were normalized to a percentage of this value. After testing with the Bartlett test, repeated-measures analysis of variance (ANOVA) was followed by the Tukey-Kramer test, used for multiple comparisons of FIF_{25-75} in different groups. As indicated, a 1-way ANOVA followed by the Tukey-Kramer test was used for comparisons of FIF in different mandibular positions. The statistical significance was established at $P < .05$. Statistical analysis was performed using a computer program (Stat View 5.0, SAS Institute).

Results

The patients were classified into groups by CF score; 6 fell into group A (CF score ≤ 3) and 9 fell into group B (CF score ≥ 4). Demographic data are shown in Table 1. In normal controls and group A patients, there was no supine jaw position in that exceeded FIF_{25-75} in the upright position. But, in group B patients, FIF_{25-75} in supine 50% F and supine 75% F exceeded that of the upright 0% F. By repeated-measures ANOVA, there were statistically significant main effects for the change in the FIF_{25-75} score with a change in jaw position ($P < .004$) and for the change in the FIF_{25-75} score by group ($P = .029$) (Table 2). When the position data were analyzed further, there were statistically significant differences between upright 0% F versus supine 0% F, upright 0% F versus supine 50% F, and upright 0% F

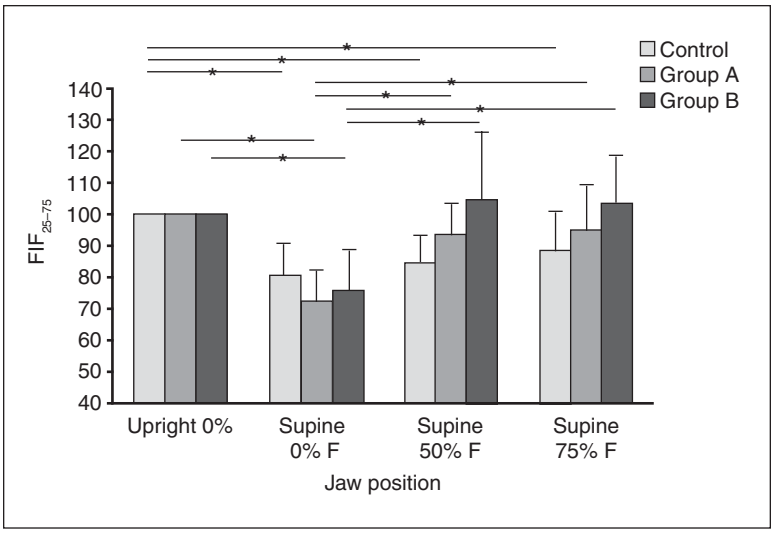

Fig 2 Forced inspiratory flow ($FIF_{25–75}$) in control subjects and in groups A and B (*$P < .05$).

versus supine 75% F in controls, and in the patient groups there were statistically significant differences between upright 0% F versus supine 0% F, supine 0% F versus supine 50% F, and supine 0% F versus supine 75% F (Fig 2).

Discussion

The changes in the controls in $FIF_{25–75}$ when lying supine in the current study were almost identical to what was reported in our earlier study,[57] namely, the control subjects experienced a diminished $FIF_{25–75}$ in the supine position (80.5%) versus the upright position (100%). Moreover, as the jaw was advanced in this group, there was a return toward the 100% level. The control group in the previous study had a BMI of 21 and a maximum protrusive motion of 7.9 mm compared to the current controls, who had a BMI of 22.2 and 7.8 mm of protrusive jaw motion. The biggest difference between the current study and the prior study is with regard to the change in the $FIF_{25–75}$ scores in the OSA subjects. Because the jaw was advanced in our previous study, the return to baseline was similar to what was seen in the control group (returned to 92%). In the present study, the OSA subjects also returned to baseline moreso than controls, as the jaw was progressively advanced in group B subjects.

This above-baseline return could be explained by several factors. First, the OSA subjects in the previous study were substantially more obese and had a much higher level of apnea than the OSA subjects in the present study. In the earlier study, the OSA subjects had a BMI of 31.6, whereas the current OSA subjects had a BMI between 23 and 24. Second, if the upright $FIF_{25–75}$ 0% score was a true maximum effort, it should not have been possible to have a greater than 100% response in the supine position, so it may be that our OSA subjects did not make a maximum effort.

Of course, any and all of these reasons could explain the greater-than-baseline response to jaw advancement in the supine position in the OSA groups. Another question investigated in the present study was whether the subjects with a larger CF score would have a substantially different $FIF_{25–75}$ response than subjects with a low CF score. Because CF scoring was not available for the controls, this means that comparisons can be made only between groups A and B. The magnitude and pattern of the changes in these 2 groups were not greatly divergent, with the most evident change being 10% at the $FIF_{25–75}$ 50% position, and in fact there was a statistically significant difference between groups A and B in this position ($P < .05$). It would be easy to speculate that because the $FIF_{25–75}$ increased more at the 50% level for patients with OSA, especially in the subjects with a higher CF score, this might mean that less mandibular advancement is necessary in subjects with this CF profile. However, such a conclusion would need to be tested using an adjustable mandibular-positioning device and polysomnographic recording of AHI in the different positions. This speculation is supported by a Scandinavian study that reported that, in mild to moderate OSA patients, a starting jaw position should not be more than 50% protrusive.[58]

There are some limitations to the present research. First, the sample size was very small, so further investigation is needed using a larger sample population. Second, the subjects of this study had a large age range, so age may have influenced the result. Third, because only cephalometry was used in this analysis, information about the upper airway form is limited to this. Fourth, bias may have occurred, because all measurements were done by one person.

As stated earlier, there are inherent limitations in using spirometric measurements of airflow as an analog of airway caliber. These include: *(1)* the subjects were awake during all measurements; *(2)* maximum forced inspiratory flow is not natural breathing, and while it can assess the effect of jaw position on airflow, it does not assess the collapsibility of the airway during sleep; *(3)* the amount of incisal opening was standardized at 12 mm, which is a larger opening than typically occurs with an oral appliance for apnea.

Conclusion

In this study, the effects of jaw position on FIF_{25-75} in a supine posture were measured for OSA and control subjects. Essentially, the decrease in the FIF_{25-75} 0% forward jaw position from the upright to the supine body position was the same in controls and in OSA subjects. Moreover, with protrusive motion, all OSA subjects demonstrated a return toward baseline in their FIF score. The OSA patients were separated into 2 subgroups (high and low CF scores); patients with a high CF score demonstrated a full return to baseline with jaw protrusion, while patients with a low CF score did not reach baseline. The magnitude of this return was actually greater than that seen in the control subjects. In the subgroup with the higher CF score, the return to and above baseline was largest. This may be related to the CF form of the subjects, or it may simply be that these patients had larger protrusive motion than the control group. Additional research is needed that controls for these factors.

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References

1. Young T, Skatrud J, Peppard P. Risk factors for obstructive sleep apnea in adults. JAMA 2004;291:2013–2016.
2. Partinen M, Guilleminault C, Quera-Salva MA, Jamieson A. Obstructive sleep apnea and cephalometric roentgenograms. Chest 1988;93:1199–1205.
3. Ferguson KA, Ono T, Lowe AA, Ryan CF, Fleetham JA. The relationship between obesity and craniofacial structure in obstructive sleep apnea. Chest 1995;108:375–381.
4. Tangugsorn V, Krogstad O, Espeland L, Lyberg T. Obstructive sleep apnea: A canonical correlation of cephalometric and selected demographic variables in obese and nonobese patients. Angle Orthod 2001;71:23–35.
5. Liao YF, Chuang ML, Huang CS, Tsai YY. Upper airway and its surrounding structures in obese and nonobese patients with?sleep-disordered breathing. Laryngoscope 2004;114:1052–1059.
6. Nelson S, Hans M. Contribution of craniofacial risk factors in increasing apneic activity among obese and nonobese habitual snores. Chest 1997;111:154–162.
7. Yu X, Fujimoto K, Urushibata K, Matsuzawa Y, Kubo K. Cephalometric analysis in obese and nonobese patients with obstructive sleep apnea syndrome. Chest 2003;124:212–218.
8. Mortimore IL, Marshall I, Wraith PK, Sellar RJ, Douglas NJ. Neck and total body fat deposition in nonobese and obese patients with sleep apnea compared with that in control subjects. Am J Respir Crit Care Med 1998;157:280–283.
9. Sakakibara H, Tong M, Matsushita K, Hirata M, Konishi S, Suetsugu S. Cephalometric abnormalities in non-obese and obese patients with obstructive sleep apnoea. Eur Respir J 1999;13:403–410.
10. Watanabe T, Isono S, Tanaka A, Tanzawa H, Nishino T. Contribution of body habitus and craniofacial characteristics to segmental closing pressure of the passive pharynx in patients with sleep-disordered breathing. Am J Respir Crit Care Med 2002;165:260–265.
11. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993;328:1230–1235.
12. Lavie P, Herer P, Peled R, et al. Mortality in sleep apnea patients: A multivariate analysis of risk factors. Sleep 1995;18:149–157.
13. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: A population health perspective. Am J Respir Crit Care Med 2002;165:1217–1239.
14. Kripke DF, Ancoli-Israel S, Klauber MR, Wingard DL, Mason WJ, Mulaney DJ. Prevalence of sleep-disordered breathing in ages 40-60 years: A population-based survey. Sleep 1997;20:65–76.
15. Bixler EO, Vgontzas AN, Have TT, Tyson K, Kales A. Effects of age on sleep apnea in men: I. Prevalence and severity. Am J Respir Crit Care Med 1998;157:144–148.
16. Schmidt-Nowara WW, Coultas DB, Wiggins C, Skipper BE, Samet JM. Snoring in a Hispanic-American population. Risk factors and association with hypertension and other morbidity. Arch Intern Med 1990;150:597–601.
17. Kripke DF, Ancoli-Israel S, Klauber MR, Wingard DL. U.S. population estimate for disordered sleep breathing: High rates in minorities. Sleep Res 1995;24:268.
18. Ancoli-Israel S, Klauber MR, Stepnowaky C, Estline E, Chinn A, Fell R. Sleep-disordered breathing in African-American elderly. Am J Respir Crit Care Med 1995;152:1946–1949.
19. Redline S, Tishler PV, Hans MG, Tosteson TD, Strohl KP, Apry K. Racial differences in sleep-disordered breathing in African-Americans and Caucasians. Am J Respir Crit Care Med 1997;155:186–192.
20. Ong KC, Clerk AA. Comparison of the severity of sleep-disordered breathing in Asian and Caucasian patients seen at a sleep disorders center. Respir Med 1998;92:843–848.
21. Li KK, Powell NB, Kushida C. A comparison of Asian and White patients with obstructive sleep apnea syndrome. Laryngoscope 1999;109:1937–1940.
22. Li KK, Kushida C, Powell NB, Riley RW, Guilleminault C. Obstructive sleep apnea syndrome: A Comparison between Far East Asian and White men. Laryngoscope 2000;110:1689–1693.
23. Liu Y, Lowe AA, Zeng X, Fu M, Fleetham JA . Cephalometric comparisons between Chinese and Caucasian patients with obstructive sleep apnea. Am J Orthod Dentofacial Orthop 2000;117:479–485.
24. Ip MS, Lam B, Lauder IJ, et al. A community study of sleep-disordered breathing in middle-aged Chinese men in Hong Kong. Chest 2001;119:62–69.
25. Cakirer B, Hans MG, Graham G, Aylor J, Tishler PV, Redline S. The relationship between craniofacial morphology and obstructive sleep apnea in Whites and in African-Americans. Am J Respir Crit Care Med 2001;163:947–950.

26. Udwadia ZF, Doshi AV, Lonkar SG, Singh CI. Prevalence of sleep-disordered breathing and sleep apnea in middle-aged urban Indian men. Am J Respir Crit Care Med 2004;169:168–173.
27. Yao M, Tachibana N, Okura M, et al. Relationships of craniofacial morphology and Body Mass Index with sleep-disordered breathing in Japanese men. Laryngoscope 2004;114:1838–1842.
28. Lam B, Ip MS, Tench E. Craniofacial profile in Asian and White subjects with obstructive sleep apnoea. Thorax 2005;60:504–510.
29. Cartwright RD. Effect of sleep position on sleep apnea severity. Sleep 1984;7:110–114.
30. Cartwright RD, Ristanovic R, Diaz F. A comparative study of treatments for positional sleep apnea. Sleep 1991;14:546–552.
31. Jokic R, Klimaszewski A, Sridhar G, Fitzpatrick MF. Continuous positive airway pressure requirement during the first month of treatment in patients with severe obstructive sleep apnea. Chest 1998;114:1061–1069.
32. Itasaka Y, Miyazaki S, Ishikawa K, Togawa K. The influence of sleep position and obesity on sleep apnea. Psychiatry Clin Neurosci 2000;54:340–341.
33. Ferguson KA, Ono T, Lowe AA, Keenan SP, Fleetham JA. A randomized crossover study of an oral appliance vs nasal-continuous positive airway pressure in the treatment of mild-moderate obstructive sleep apnea. Chest 1996;109:1269–1275.
34. Clark GT, Blumenfeld I, Yoffe N, Peled E, Lavie P. A crossover study comparing the efficacy of continuous positive airway pressure with anterior mandibular positioning device on patients with obstructive sleep apnoea. Chest 1996;109:1477–1483.
35. Ferguson KA, Ono T, Lowe AA, Al-Majed S, Love LL, Fleetham JA. A short-term control trial of an adjustable oral appliance for the treatment of mild to moderate obstructive sleep apnea. Thorax 1997;52:362–368.
36. Walker-Engstrom ML, Wilhelmsson B, Tegelberg A, Dimenas E, Ringqvist I. Quality of life assessment of treatment with dental appliance or UPPP in patients with mild to moderate obstructive sleep apnea. A prospective randomized 1-year follow-up study. J Sleep Res 2000;9:303–308.
37. Walker-Engstrom ML, Tegelberg A, Wilhelmsson B. 4-year follow-up treatment with dental appliance or uvulopalatopharyngoplasty in patients with obstructive sleep apnea. Chest 2002;121:739–746.
38. Randerath WJ, Heise M, Hinz R, Ruehle KH. An individually adjustable oral appliance vs continuous positive airway pressure in mild-to-moderate obstructive sleep apnea syndrome. Chest 2002;122:569–575.
39. O'Sullivan RA, Hillman DR, Mateljan R, Pantin C, Finucane KE. Mandibular advancement splint: An appliance to treat snoring and obstructive sleep apnea. Am J Respir Crit Care Med 1995;151:194–198.
40. Bloch KE, Iseli A, Zhang JN, et al. A randomized, controlled crossover trial of two oral appliances for sleep apnea treatment. Am J Respir Crit Care Med 2000;162:246–251.
41. Millman RP, Rosenberg CL, Carlisle CC, Kramer NR, Kahn DM, Bonitati AE. The efficacy of oral appliance in the treatment of persistent sleep apnea after uvulopalatopharyngoplasty. Chest 1998;113:992–996.
42. Clark GT, Arand D, Chung E, Tong D. Effect of anterior mandibular positioning on obstructive sleep apnea. Am Rev Respir Dis 1993;147:624–629.
43. Eveloff SE, Rosenberg CL, Carlisle CC, Millman RP. Efficacy of a Herbst mandibular advancement device in obstructive sleep apnea. Am J Respir Crit Care Med 1994;149:905–909.
44. Marklund M, Franklin KA, Sahlin C, Lundgren R. The effect of a mandibular advancement device on apneas and sleep in patients with obstructive sleep apnea. Chest 1998;113:707–713.
45. Pancer J, Al-Faifi S, Al-Faifi M, Hoffstein V. Evaluation of variable mandibular advancement appliance for treatment of snoring and sleep apnea. Chest 1999;116:1511–1518.
46. Clark GT, Sohn J, Hong CN. Treating obstructive sleep apnea and snoring: Assessment of anterior mandibular positioning device. J Am Dent Assoc 2000;131:765–771.
47. Henke KG, Frantz DE, Kuna ST. An oral elastic mandibular advancement device for obstructive sleep apnea. Am J Respir Crit Care Med 2000;161:420–425.
48. Marklund M, Franklin KA, Persson M. Orthodontic side-effect of mandibular advancement devices during treatment of snoring and sleep apnea. Eur J Orthod 2001;23:135–144.
49. Mehta A, Qian J, Petocz P, Daredeliler MA, Cistulli PA. A randomized, controlled study of a mandibular advancement sprint for obstructive sleep apnea. Am J Respir Crit Care Med 2001;163:1457–1461.
50. Marklund M, Sahlin C, Stenlund H, Persson M, Franklin KA. Mandibular advancement device in patients with obstructive sleep apnea :long-term effects on apnea and sleep. Chest 2001;120:162–169.
51. Yoshida K. Influence of sleep posture on response to oral appliance therapy for sleep apnea syndrome. Sleep 2001;24:538–544.
52. Rose EC, Staats R, Schulte-Monting J, Jonas IE. Treatment of obstructive sleep apnea with the Karwetzky oral appliance. Eur J Oral Sci 2002;110:99–105.
53. Pitsis AJ, Darendeliler MA, Gotsopoulos H, Petocz P, Cistulli PA. Effect of vertical dimension on efficacy of oral appliance therapy in obstructive sleep apnea. Am J Respir Crit Care Med 2002;166:860–864.
54. De Almeida FR, Bittencourt LR, De Almeida CIR, Tsuiki S, Lowe AA, Tufik S. Effects of mandibular posture on obstructive sleep apnea severity and the temporomandibular joint in patients fitted with an oral appliance. Sleep 2002;25:507–513.
55. Robertson C. Cranial base consideration between apnoeics and non-apnoeic snores, and associated effects of long-term mandibular advancement on condylar and natural head position. Eur J Orthod 2002;24:353–361.
56. Tsuda H, Masumi S. Evaluation of effective jaw position and body posture for oral appliance of patients with obstructive sleep apnea syndrome. J Jpn Prosthodont Soc 2005;49:736–743.
57. Masumi S, Nishigawa K, Williams A, Yan-Go FL, Clark GT. Effect of jaw position and posture on forced inspiratory airflow in normal subjects and patients with obstructive sleep apnea. Chest 1996;109:1484–1489.
58. Tegelberg A, Walker-Engstrom ML, Vestling O, Wilhelmsson B. Two different degree of mandibular advancement with a dental appliance in treatment of patients with mild to moderate obstructive sleep apnea. Acta Odontol Scand 2003;61:356–362.