Taiwanese Journal of Orthodontics

Volume 32 | Issue 1 Article 2

2020

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Recommended Citation

Hsieh, Shao-Chih; Chou, Szu-Ting; and Tseng, Yu-Chuan (2020) "Treatment Options for Obstructive Sleep Apnea," *Taiwanese Journal of Orthodontics*: Vol. 32 : Iss. 1 , Article 2.
DOI: 10.30036/TJO.202003_32(1).0002
Available at: https://www.tjo.org.tw/tjo/vol32/iss1/2

Review Article

Treatment Options for Obstructive Sleep Apnea

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Obstructive sleep apnea (OSA) is sleep-disordered breathing caused by the collapse of the upper airway. Because anatomical abnormalities of the narrow upper airway are undoubtedly the most critical cause of OSA, the available treatment methods for OSA mostly focus on these anatomical problems.

The collapse of the respiratory tract is associated with different levels of upper airway obstruction. The obstruction of the airway can be divided into three parts: the nasal cavity, retropalatal region, and retrolingual region. This article summarizes some common OSA treatment methods, their underlying theories, and their effect on the upper airway. ***(Taiwanese Journal of Orthodontics. 32(1): 17-24, 2019,2020)***

Keywords: obstructive sleep apnea; oral appliance; maxillary expansion; Powell-Riley surgical protocol.

INTRODUCTION

Obstructive sleep apnea (OSA) is a sleep disorder where breathing repeatedly stops or weakens because of upper airway collapse during sleep. This airway collapse results from anatomical abnormalities and low pharyngeal dilator muscle tone during sleep.[1] Upper airway collapse during sleep results in snoring, sleep fragmentation, and hypoxemia,[2] leading to symptoms such as excessive daytime sleepiness, inattention, memory loss, anxiety, and depression.[3] Thus, it may increase the incidence of car accidents. OSA may also lead to heart disease, high blood pressure, stroke, or type 2 diabetes.[4] This makes OSA a serious threat to the health and quality of life of patients.

Diagnosis

The diagnosis of OSA is based on physical examinations, a sleep test, and the medical history of a patient.

Polysomnography (PSG) is the standard sleep test for OSA. It involves electroencephalography, electrooculography, chin electromyography, electrocardiography, and measurements of the patient's airflow, oxygen saturation, respiratory effort, and heart rate.[2]

The most crucial piece of information in PSG is the Apnea-Hypopnea Index (AHI), defined as the number of apneas or hypopneas per hour of sleep. If the AHI> 5 for a patient who has signs and symptoms of disturbed sleep, the diagnosis of OSA is confirmed.[2]

Received: August 19, 2019 Revised: April 6, 2020 Accepted: April 16, 2020
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10.30036/TJO.202003_32(1).0002

The severity classification of OSA, defined by the American Academy of Sleep Medicine, is as follows: mild (if 5 ≤ AHI < 15), moderate (if 15 ≤ AHI ≤ 30), and severe (if AHI > 30).[2]

Site of upper airway obstruction

PSG can diagnose the severity of OSA, but it cannot distinguish the obstruction site in OSA. Oral examination,[5] fiberopticnasopharyngolaryngoscopy (with Müller's maneuver),[6] lateral cephalometric analysis, computed tomography, and magnetic resonance imaging[7] should be used to identify the site of obstruction.

The potential site of upper airway obstruction can be divided into three main areas: nasal cavity, retropalatal region, and retrolingual region (Table 1).[7]

The severity of OSA and the location of airway obstruction must be confirmed prior to treatment to establish a treatment baseline and make a reasonable treatment plan.

Treatment of OSA

Reduce the body weight

Obesity is a significant risk factor for OSA. Excessive adipose tissue in the upper respiratory tract accumulates in the tongue, pharyngeal wall, and soft palate and may increase the risk and severity of OSA.[8,9,10] Significant OSA is present in 40% of obese individuals and 70% of OSA patients are obese.[11]

Weight loss is an effective method for the non-surgical treatment of OSA.[12] Diet modification, medication, and surgical weight loss can improve the condition of patients with OSA.[12,13] Diet control and weight loss of 9%–20% can reduce AHI by 30%–75%.[14]

Continuous positive airway pressure

Continuous positive airway pressure (CPAP) is the first-line treatment for moderate to severe OSA.[2,15,16,17] It provides pressure to open the airway that collapses when sleeping, which can effectively reduce AHI and maintain the blood oxygen concentration in the patient.[18,19,20] It is largely proven to be efficacious in reducing sleepiness, blood pressure, and cardio/cerebrovascular risk.[21]

CPAP is suitable for patients with mild, moderate, and severe OSA and is effective regardless of the site of airway obstruction.[22] When patients use CPAP, they can eliminate apneas and hypopneas, and the blood oxygen concentration can be maintained in the normal range.[20,23]

However, adherence to CPAP has always been a problem.[19] On average, 10.7% of patients cannot tolerate CPAP. The average duration of nighttime use is only 4.7 h.[24]

Table 1. *The potential sites of upper airway obstruction and their causes.*

Obstruction site	Cause
Nasal	septal deviation, turbinate hypertrophy, alar collapse or sinonasal masses
Retropalatal	posteriorly elongated palate and uvula, tonsil and adenoid hypertrophy
Retrolingual	macroglossia, retrognathia and micrognathia, inferior displacement of hyoid

10.30036/TJO.202003_32(1).0002

The overall CPAP non-adherence rate was reported to be 34.1%, which may not allow CPAP treatment to reach the therapeutic threshold, thus casting a doubt on its efficacy.[24]

Oral appliances

The two main types of oral appliances (OAs) are mandibular repositioning appliances and tongue retaining devices.[2]

The function of an OA is to advance the lower jaw or tongue to help opening the upper airway; in essence, OA is more effective for patients with prevalent retrolingual obstruction and retrognathia.[25,26,27] On the other hand, nasal obstruction may reduce patients' tolerance to the OA and have a negative influence on treatment response. Increased nasal resistance is associated with a higher proportion of oral breathing during sleep.[28] Because the mouth opening may be limited by OAs, mouth breather (nasal obstruction) tend not to tolerate the OA.[29] Higher nasal resistance also associated with pharyngeal and lingual collapse, reduce the effect of OA.[29] It was reported that the non-responders to OAs have higher nasal resistance than the responders.[25] Thus, nasal obstruction should be treated before using OA.

OA has been suggested for patients with mild (SIGN guideline[30]) to moderate OSA and for those who cannot tolerate CPAP therapy or prefer alternate therapy.[31,32,33] OA has also been recommended to alleviate simple snoring.[31] The mean reduction in AHI by OA was 13.60 events/h.[31]

Maxillary expansion

A high-arched palate is a risk factor for OSA.[34] The palate is the roof of the mouth and the floor of the nasal cavity. A high-arched palate represents a narrow nasal cavity (increased nasal resistance) and a narrow dental arch (decreased space for the tongue). Increased nasal resistance and a lack of palatal space for the tongue result in oral breathing and low tongue posture.[35] Ultimately, the downward and backward displacement of the tongue indirectly causes retrolingual obstruction.[35]

Maxillary expansion is primarily used to improve OSA by correcting a high-arched palate. It could extend the expansion to the nasal cavity, reduces nasal obstruction, and improves tongue position in oral breathing.

Maxillary expansion for pediatric OSA

Pediatric OSA is classified as mild if $1 \leq AHI < 5$, moderate if $5 \leq AHI \leq 10$, and severe if $AHI > 10$.[36] The most common cause of pediatric OSA is the enlargement of tonsils and/or adenoids.[37,38] The modalities of treatments for pediatric OSA are adenotonsillectomy, CPAP, weight loss, and medications.[36,39,40]

Rapid maxillary expansion (RME) is another option for treating pediatric OSA. RME stretches and widens the maxilla and nasal floor by distraction osteogenesis to reduce nasal obstruction.[41,42] It is suitable for children and adolescents with a narrow palate and dental crossbite.[42,43] RME creates more space for the tongue to raise, thus indirectly enlarging the pharyngeal airway.[44] After RME treatment, the mean reduction in AHI was 6.86 events/ h.[45]

Maxillary expansion for adult OSA

Maxillary expansion can also help adult patients reduce AHI and OSA symptoms.[35] However, an adult maxillary bone has ceased the growth and the midpalatal suture is closed. Thus, RME achieves only dental expansion in adults and does not effectively enlarge the palatal bone. Therefore, it has traditionally been possible to rely on surgically assisted rapid palatal expansion (SARPE) to achieve maxillary expansion in adults. SARPE improves transverse discrepancy with a tooth-borne expander with horizontal and vertical osteotomy. It also improves OSA symptoms and decreases AHI by 56%.[46]

The tooth-borne expander of SARPE acts on the teeth, which renders the device's force unable to be directly transmitted to the midpalatal suture. This in turn causes side effects from the dental expansion, resulting in a relapse rate of SARPE of up to 64%.[47]

Maxillary expansion by distraction osteogenesis is a technique involving the use of a bone-borne expander with horizontal and vertical osteotomy. Mini-implants are

placed in the maxillary bone along the midpalatal suture to deliver the force exerted by the expander, and Le Fort I osteotomy and vertical osteotomy are subsequently performed to open the midpalatal suture and reduce the resistance of maxillary expansion.[48]

Miniscrew-assisted RPE (MARPE) is another non-surgical treatment option involving the complete or partial use of a bone-borne expander. Bone screws are used to deliver force directly from the expander to the maxillary bone to split the midpalatal suture. MARPE has been reported to expand the nasal cavity and the upper half of the pharynx, thus reducing upper airway resistance. Therefore, MARPE may also be an effective treatment option for OSA.[49] The success rate of MARPE for young adults range from 84.2% (mean age= 20.1 years)[50] to 86.96% (mean age = 20.9 years).[51] However, the failure rate seems to increase with age, it's about 15% in the patients' 20s and 50% in the 30s.[52]

Surgical treatment

The Powell–Riley surgical protocol is a logical two-phase surgical treatment protocol for OSA.[6] This protocol emphasizes the need to clearly diagnose the obstruction site of the upper airway. Surgery can be performed to target the site of anatomical obstruction, thus avoiding wide range of surgery.

Phase I surgery

Conservative operation on soft tissue does not change the patient's facial bones or the occlusion. Table 2 illustrates the surgery for each upper airway obstruction site.

The overall success rate of phase I surgery is 61%, ranging from 42% to 75% depending on the severity of the disorder.[6,53]

Phase II surgery

After phase I surgery, the obstruction of the retrolingual area (base of the tongue) may still not be completely treated. Subsequent treatment options can be phase II surgery or other alternative treatment modalities including tracheotomy, nasal CPAP,[6] and OA.

The second stage of surgery involves maxillomandibular advancement osteotomy, which reduces the collapse of the retrolingual airway by creating more space for the tongue in the oral cavity;[6] tongue reduction can be used as adjuvant therapy.[6]

The success rate of phase II surgery is more than 90%,[53] whereas the success rate of phase II surgery that is subsequent to phase I treatment is more than 95%.[44]

Table 2. *Conservative surgery for each obstruction site.*

Obstruction site	Phase I surgery
Nasal	Septoplasty, turbinate reduction, nasal valve grafting masses
Retropalatal	Tonsillectomy, uvulopalatopharyngoplasty (UPPP) or uvulopalatal flap (UPF)
Retrolingual	Genioglossus advancement, hyoid myotomy and suspension of hyoid

Taiwanese Journal of Orthodontics. 2020, Vol. 32. No. 1
10.30036/TJO.202003_32(1).0002

CONCLUSION

Many treatment options are available for OSA. However, no treatment solution covers every situation. Detailed diagnosis and clear identification of the upper airway obstruction guide the choice of correct treatment.

Basing on severity, OA can be selected as the first-line treatment for mild OSA. For patients with moderate to severe OSA, the first-line of treatment is CPAP. Surgery can be used to treat the patients who have more severe OSA, poor response to previous treatment, or are unable to cope with conservative treatment.

With regard to the patient's airway obstruction site, CPAP and weight loss are not site-specific treatment options, OAs are more effective for patients with retrolingual obstruction. Palatal expansion can help to reduce the nasal resistance. Surgical treatment should be administered according to the site of airway obstruction, thus improving airway anatomy with a good therapeutic effect.

REFERENCE

1. Fenik VB, Penzel T, Malhotra A. Editorial: anatomy of upper airway and neuronal control of pharyngeal muscles in obstructive sleep apnea. Front Neurol. 2019; 10:733. doi: 10.3389/fneur.2019.00733.
2. Epstein LJ, Kristo D, Strollo PJ, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clinical Sleep Medicine. 2009; 5(3):263-76.
3. Al-Barrak M, Shepertycky MR, Kryger MH. Morbidity and mortality in obstructive sleep apnea syndrome 2: Effect of treatment on neuropsychiatric morbidity and quality of life. Sleep and Biological Rhythms. 2003; 1(2):65-74. doi: 10.1046/j.1446-9235.2003.00008.x.
4. Jean-Louis G, Zizi F, Clark LT, Brown CD, McFarlane SI. Obstructive sleep apnea and cardiovascular disease: role of the metabolic syndrome and its components. J Clin Sleep Med. 2008; 4(3):261-72. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2546461/. Accessed July 6, 2019.
5. Sawanyawisuth K, Ruangsri S, Jorns TP, Puasiri S, Luecha T, Chaithap C. Which oropharyngeal factors are significant risk factors for obstructive sleep apnea? An age-matched study and dentist perspectives. Nature and Science of Sleep. 2016; 8:215-19. doi: 10.2147/NSS.S96450.
6. Carvalho B, Hsia J, Capasso R. Surgical therapy of obstructive sleep apnea: a review. Neurotherapeutics. 2012; 9(4):710-16. doi: 10.1007/s13311-012-0141-x.
7. Powell NB. Contemporary surgery for obstructive sleep apnea syndrome. Clin Exp Otorhinolaryngology. 2009; 2(3):107-14. doi: 10.3342/ceo.2009.2.3.107.
8. Arens R, Sin S, Nandalike K, et al. Upper airway structure and body fat composition in obese children with obstructive sleep apnea syndrome. Am J Respir Crit Care Med. 2011; 183(6):782-7. doi:10.1164/rccm.201008-1249OC.
9. Schellenberg JB, Maislin G, Schwab RJ. Physical findings and the risk for obstructive sleep apnea. The importance of oropharyngeal structures. Am J Respir Crit Care Med. 2000; 162 (2 Pt 1):740-8. doi: 10.1164/ajrccm.162.2.9908123.
10. Pack AI: Sleep Apnea : Pathogenesis, Diagnosis and Treatment. 2nd Ed. London, Informa Healthcare, 2012; p. 13.
11. Vgontzas AN, Tan TL, Bixler EO, et al. Sleep Apnea and Sleep Disruption in Obese Patients. Archives of Internal Medicine. 1994; 154(15):1705-11. doi: 10.1001/archinte.1994.00420150073007.
12. McFarlane SI. Obstructive sleep apnea and obesity: implications for public health. Sleep Med Disord Int J. 2017; 1(4):93-99. doi: 10.15406/smdij.2017.01.00019.
13. Hudgel DW, Patel SR, Ahasic AM, et al. The role of weight management in the treatment of adult obstructive sleep apnea. An official american thoracic society clinical practice guideline. Am J Respir Crit

Care Med. 2018; 198(6):e70-e87. doi: 10.1164/rccm.201807-1326ST.

14. Strobel RJ, Rosen RC. Obesity and weight loss in obstructive sleep apnea: a critical review. Sleep. 1996; 19(2):104-15. doi: 10.1093/sleep/19.2.104.
15. Giles TL, Lasserson TJ, Smith B, White J, Wright JJ, Cates CJ. Continuous positive airways pressure for obstructive sleep apnea in adults. Giles T, ed. Cochrane Database of Systematic Reviews. July 2006. doi: 10.1002/14651858.cd001106.pub3.
16. Turino C, de Batlle J, Woehrle H, et al. Management of continuous positive airway pressure treatment compliance using telemonitoring in obstructive sleep apnea. Eur Respir J. 2017; 49(2):1601128. doi: 10.1183/13993003.01128-2016.
17. Stasche N. Selective indication for positive airway pressure (PAP) in sleep-related breathing disorders with obstruction. GMS Curr Top Otorhinolaryngol Head Neck Surg. 2006; 5:Doc06.
18. Toukh M, Pereira EJ, Falcon BJ, et al. CPAP reduces hypercoagulability, as assessed by thromboelastography, in severe obstructive sleep apnea. Respir Physiol Neurobiol. 2012; 183(3):218-23. doi: 10.1016/j.resp.2012.06.022.
19. Boyd SB, Walters AS. Effectiveness of treatment apnea-hypopnea index: a mathematical estimate of the true apnea-hypopnea index in the home setting. J Oral Maxillofac Surg. 2013; 71(2):351-7. doi: 10.1016/j.joms.2012.05.009.
20. Borak J, Cieślicki JK, Koziej M, Matuszewski A, Zieliński J. Effects of CPAP treatment on psychological status in patients with severe obstructive sleep apnea. J Sleep Res. 1996; 5(2):123-7. doi:10.1046/j.1365-2869.1996.d01-60.x.
21. Lombardi C, Caravita S, Parati G. Central sleep apnea during continuous positive airway pressure therapy in obstructive sleep apnea patients: from the compliance to adaptation, maladaptation and reflexes. J Thorac Dis. 2017; 9(11):4152-56. doi: 10.21037/jtd.2017.09.116.
22. Demko BG. Ten misconceptions that dentists have about treating obstructive sleep apnea. J Dent Sleep Med. 2018; 5(3):90-103. doi:10.15331/jdsm.7036
23. Sanders MH. Nasal CPAP effect on patterns of sleep apnea. Chest. 1984; 86(6):839-44. doi: 10.1378/chest.86.6.839
24. Rotenberg BW, Murariu D, Pang KP. Trends in CPAP adherence over twenty years of data collection: a flattened curve. J Otolaryngol Head Neck Surg. 2016; 45(1):43. doi: 10.1186/s40463-016-0156-0.
25. Zeng B, Ng AT, Qian J, Petocz P, Darendeliler MA, Cistulli PA. Influence of nasal resistance on oral appliance treatment outcome in obstructive sleep apnea. Sleep. 2008; 31(4):543-7. doi: 10.1093/sleep/31.4.543.
26. Sutherland K, Vanderveken OM, Tsuda H, et al. Oral appliance treatment for obstructive sleep apnea: an update. J Clin Sleep Med. 2014; 10(2):215-27. doi: 10.5664/jcsm.3460.
27. Basyuni S, Barabas M, Quinnell T. An update on mandibular advancement devices for the treatment of obstructive sleep apnea hypopnea syndrome. J Thorac Dis. 2018; 10(S1):S48-S56. doi: 10.21037/jtd.2017.12.18.
28. McLean HA, Urton AM, Driver HS, et al. Effect of treating severe nasal obstruction on the severity of obstructive sleep apnea. Eur Respir J. 2005; 25(3):521-7. doi: 10.1183/09031936.05.00045004.
29. Yaremchuk K and Wardrop PA: Sleep Medicine. San Diego, CA, Plural Pub Inc, 2011. Print; Ch.18: pp 271-82.
30. Scottish Intercollegiate Guidelines Network. Management of Obstructive Sleep Apnea/Hypopnea Syndrome in Adults: A National Clinical Guideline. Edinburgh: Scottish Intercollegiate Guidelines Network; 2003.
31. Ramar K, Dort LC, Katz SG, et al. Clinical practice guideline for the treatment of obstructive sleep apnea

and snoring with oral appliance therapy: an update for 2015. J Clin Sleep Med. 2015; 11(7):773-827. doi: 10.5664/jcsm.4858

32. Marklund M, Carlberg B, Forsgren L, Olsson T, Stenlund H, Franklin KA. Oral appliance therapy in patients with daytime sleepiness and snoring or mild to moderate sleep apnea. JAMA Intern Med. 2015; 175(8):1278-85. doi: 10.1001/jamainternmed.2015.2051.
33. Kryger MH, Roth T, Dement WC: Principles and Practice of Sleep Medicine. Philadelphia, PA, Elsevier, 2017:p. 1131.
34. Lee YC, Eun YG, Shin SY, Kim SW. Prevalence of snoring and high risk of obstructive sleep apnea syndrome in young male soldiers in Korea. J Korean Med Sci. 2013; 28(9):1373-7. doi: 10.3346/jkms.2013.28.9.1373.
35. Abdullatif J, Certal V, Zaghi S, et al. Maxillary expansion and maxillomandibular expansion for adult OSA: a systematic review and meta-analysis. J Craniomaxillofac Surg. 2016; 44(5): 574-8.
36. Roland PS, Rosenfeld RM, Brooks LJ, et al. Clinical practice guideline: polysomnography for sleep-disordered breathing prior to tonsillectomy in children. Otolaryngol Head and Neck Surg. 2011; 145(1 suppl):S1-15. doi: 10.1177/0194599811409837.
37. Suen JS, Arnold JE, Brooks LJ. Adenotonsillectomy for treatment of obstructive sleep apnea in children. Arch Otolaryngol Head Neck Surg. 1995; 121(5):525-30.
38. Chang SJ and Chae KY. Obstructive sleep apnea syndrome in children: epidemiology, pathophysiology, diagnosis and sequelae. Korean J Pediatr. 2010; 53(10):863-71. doi:10.3345/kjp.2010.53.10.863.
39. Ahn YM. Treatment of obstructive sleep apnea in children. Korean J Pediatr. 2010; 53(10):872-9. doi:10.3345/kjp.2010.53.10.872.
40. Rubinstein BJ and Baldassari CM. An update on the management of pediatric obstructive sleep apnea. Curr Treat Options in Peds. 2015; 1(3):211-23. doi:10.1007/s40746-015-0022-8.
41. Villa MP, Rizzoli A, Miano S, Malagola C. Efficacy of rapid maxillary expansion in children with obstructive sleep apnea syndrome: 36 months of follow-up. Sleep Breath. 2011; 15(2):179-84. doi: 10.1007/s11325-011-0505-1.
42. Ramires T, Maia RA, Barone JR. Nasal cavity changes and the respiratory standard after maxillary expansion. Braz J Otorhinolaryngol. 2008; 74(5):763-69. doi: 10.1016/S1808-8694(15)31388-4.
43. McNamara JA, Lione R, Franchi L, et al. The role of rapid maxillary expansion in the promotion of oral and general health. Prog Orthod. 2015; 16(1):33. doi: 10.1186/s40510-015-0105-x.
44. Guilleminault C: Sleep Medicine, An Issue of Medical Clinics of North America- E-Book. The Clinics: Internal Medicine, Elsevier Health Sciences, 2010; 94(3). eBook: 9781455700424
45. Machado-Júnior AJ, Zancanella E, Crespo AN. Rapid maxillary expansion and obstructive sleep apnea: a review and meta-analysis. Med Oral Patol Oral Cir Bucal. 2016; 21(4):e465-9. doi: 10.4317/medoral.21073.
46. Vinha PP, Eckeli AL, Faria AC, Xavier SP, de Mello-Filho FV. Effects of surgically assisted rapid maxillary expansion on obstructive sleep apnea and daytime sleepiness. Sleep Breath. 2016; 20(2):501-8. doi: 10.1007/s11325-015-1214-y.
47. Chamberland S and Proffit WR. Short-term and long-term stability of surgically assisted rapid palatal expansion revisited. Am J Orthod Dentofacial Orthop. 2011; 139(6):815-22. doi: 10.1016/j.ajodo.2010.04.032.
48. Yoon A, Guilleminault C, Zaghi S, Liu SY. Distraction osteogenesis maxillary expansion (DOME) for adult obstructive sleep apnea patients with narrow maxilla and nasal floor. Sleep Med. June 2019. doi: 10.1016/j.sleep.2019.06.002.
49. Hur JS, Kim HH, Choi JY, Suh SH, Baek SH.

Investigation of the effects of miniscrew-assisted rapid palatal expansion on airflow in the upper airway of an adult patient with obstructive sleep apnea syndrome using computational fluid-structure interaction analysis. Korean J Orthod. 2017; 47(6):353-64. doi: 10.4041/kjod.2017.47.6.353.

50. Park JJ, Park YC, Lee KJ, Cha JY, Tahk JH, Choi YJ. Skeletal and dentoalveolar changes after miniscrew-assisted rapid palatal expansion in young adults: A cone-beam computed tomography study. Korean J Orthod. 2017; 47(2):77-86. doi: 10.4041/kjod.2017.47.2.77.
51. Choi SH, Shi KK, Cha JY, Park YC, Lee KJ. Nonsurgical miniscrew-assisted rapid maxillary expansion results in acceptable stability in young adults. Angle Orthod. 2016; 86(5):713-20. doi: 10.2319/101415-689.1.
52. Shin H, Hwang CJ, Lee KJ, Choi YJ, Han SS, Yu HS. Predictors of midpalatal suture expansion by miniscrew-assisted rapid palatal expansion in young adults: A preliminary study. Korean J Orthod. 2019; 49(6):360-71. doi: 10.4041/kjod.2019.49.6.360.
53. Riley RW, Powell NB, Guilleminault C. Obstructive sleep apnea syndrome: a surgical protocol for dynamic upper airway reconstruction. J Oral Maxillofac Surg. 1993; 51(7):742-7. doi: 10.1016/s0278-2391(10)80412-4.