

Management of Obstructive Sleep Apnea in Adults

Subjects: **Respiratory System**

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Obstructive sleep apnea (OSA) is a common disease that is often under-diagnosed and under-treated in all ages. The conventional diagnostic and therapeutic approaches for adults with OSA are usually based on clinical assessments, followed by polysomnography (PSG) or respiratory polygraphy (RPG) in a sleep lab or at home (HSAT: home sleep apnea testing), and treatment decisions. With this approach, it could be unadvisable for physicians to decide the treatment approach for OSA without evaluating AHI.

OSA

CPAP

Adults

personalized management

1. Overview

Continuous positive airway pressure (CPAP) therapy is an optimal treatment for adults with severe obstructive sleep apnea (OSA), as it has been demonstrated previously [\[1\]\[2\]\[3\]\[4\]](#). However, there are some patients who do not tolerate positive airway pressure (PAP) therapy. Patients with OSA-associated asthma or chronic obstructive pulmonary disease (COPD) as comorbidities (OLDOSA: obstructive lung disease and OSA) should have a personalized approach to devise an optimal treatment plan.

2. Personalization of OSA with Phenotype Approach

The phenotypic stepwise approach is one of the strategies to optimize treatment efficacy for patients with OSA. The definition of OSA phenotypes is described as the combination of disease features and clinical relevance of OSA (symptoms, response to treatment, health status, and quality of life) [\[5\]](#). Based on the clinical symptoms of each individual patient, physicians must have a personalized approach. Depending on the personalization of phenotypic characteristics of patients, the response to treatment of each individual patient is also different. Adherence to CPAP therapy might be more difficult with greater possibility of failure in patients with OSA complicated by anxiety disorders or depression. Although there are not enough data reported on symptom-based phenotypes to achieve a high therapeutic effect, the personalization of OSA management in adults should be performed, and based on the specific symptoms and populations.

The personalization of OSA management based on AHI may be considered as a corner stone for phenotype-based approaches, although AHI has an inaccurate reflection of OSA severity between men and women [\[6\]](#). However,

when analyzing the PSG to determine its correlation with cardiovascular morbidity and mortality, severe nadir hypoxemia might increase the risk of cardiovascular disease and death in patients with similar AHI. In addition, periodic leg movements have also been found to be associated with an increased risk of cardiovascular disease and death. Some studies have shown that the degree of hypoxia and the duration of hypoxia [7], or the duration of respiratory events [8] can replace the AHI when assessing the degree of OSA severity.

The personalization of OSA management based on the phenotype treatment response is also an approach that helps to achieve good efficacy. Prescribing CPAP therapy is the optimal treatment option for adult patients with severe or moderate symptomatic OSA because the majority of OSA patients tolerate this treatment well. Therefore, CPAP therapy focus on additional objectives such as lowering blood pressure, stabilizing blood glucose, and improving life expectancy [9]. There are a few patients with OSA who still have residual AHI despite optimal titration of CPAP pressure. This may be due to the development of central apnea after PAP treatment.

The personalization of OSA management needs more data on specific clinical and functional features of OSA to refine the description of OSA phenotypes. This would require the connection of multiple sleep data sources for the purpose of analyzing and synthesizing OSA-specific phenotypes.

3. Personalization of OSA with Function and Pathophysiological Approach

In its physiopathology, OSA is mainly due to the anatomical abnormalities of the laryngo-pharynx, inducing the narrow or complete obstruction of the upper airway during sleep. However, besides the anatomical factors, there are also non-anatomical factors that contribute to OSA severity and complicate the treatment of OSA, including oversensitivity to the ventilation control system, low threshold for respiratory stimulation, and poor tonus and responsiveness of pharyngeal muscles while sleeping [10][11][12]. These factors will influence the obstruction of the upper airway in OSA and also determine the tolerability, adherence, and detrimental response to the treatment with PAP [13][14][15].

The personalization of OSA management may help then to differentiate between OSA patients due to anatomical structure and those not due to anatomical abnormality. Thus, it might be unreasonable to grade the severity of OSA based on AHI alone because it limits the effectiveness of optimal therapy in patients with OSA [16].

For OSA patients with upper airway obstruction, in more than 50% of them, it is anatomically multi-segmental and would require multi-level surgical intervention [17]. However, previous studies have advocated that multi-level surgery regarding the soft palate and the base of the tongue is safe and successful [18][19]. The use of DISE can demonstrate the level of obstruction at the base of the tongue and/or the epiglottis, and can predict the outcome of upper airway surgery [20][21]. With single-level palatal surgery, patient selection with DISE has attained better long-term outcomes [22]. Some suggestions for the personalized treatment of patients with OSA not due to anatomical abnormalities are: (1) patients with OSA and with low loop gain (ventilatory response/ventilatory disturbance < 1): an oral appliance can be used or upper airway surgery can be performed [23][24][25]; (2) patients with high loop gain

(ventilatory response/ventilatory disturbance > 1): respond to supplemental oxygen and are considered “responders” if their AHI is reduced by $\geq 50\%$ with oxygen therapy and otherwise considered “non-responders” [26][27][28]; (3) in patients with weak oropharyngeal muscle tone, stimulants can be used to increase upper respiratory muscle tone [29]. The role of myofunctional therapy in the treatment of OSA has been also emphasized in the recent recommendations as an adjunct in the management of OSA. These recommendations suggest that this treatment modality, consisting of exercises targeting oral and oropharyngeal structures, can lead to a reduction in the AHI and snoring, and can improve oxygenation during sleep and daytime sleepiness [30][31][32].

However, the identification of the non-structural OSA patients remains a challenge because it requires more advanced techniques such as magnetic resonance imaging (MRI) or the use of esophageal sensors in combination with CPAP therapy to assess upper airway narrowness. These techniques are invasive and expensive [14][33][34]. Therefore, it is necessary to differentiate patients who could use conventional and more readily available tests, such as PSG, RPG, or other non-invasive sleep monitoring devices which are non-invasive and easy to perform, from those who need more advanced forms of evaluation.

In the future, personalized medicine in OSA should rely on data from randomized controlled studies, and focus on the pathophysiological mechanisms and clinical phenotypes of patients with OSA. These multidirectional approaches also help to find out accurate therapies and relevant solutions to treat patients with OSA or to re-evaluate OSA patients with treatment failures. Therefore, the future diagnosis and treatment of OSA should coordinate these multidirectional approaches to solve the individual patient's problems related to OSA in long-term follow-up.

References

1. McARDLE, N.; Devereux, G.; Heidarnajad, H.; Engleman, H.M.; Mackay, T.W.; Douglas, N.J. Long-term Use of CPAP Therapy for Sleep Apnea/Hypopnea Syndrome. *Am. J. Respir. Crit. Care Med.* 1999, 159, 1108–1114.
2. Krieger, J.; Kurtz, D.; Petiau, C.; Sforza, E.; Trautmann, D. Long-Term Compliance with CPAP Therapy in Obstructive Sleep Apnea Patients and in Snorers. *Sleep* 1996, 19, S136–S143.
3. Engleman, H.M.; E Martin, S.; Douglas, N.J. Compliance with CPAP therapy in patients with the sleep apnoea/hypopnoea syndrome. *Thorax* 1994, 49, 263–266.
4. Hussain, S.F.; Irfan, M.; Waheed, Z.; Alam, N.; Mansoor, S.; Islam, M. Compliance with continuous positive airway pressure (CPAP) therapy for obstructive sleep apnea among privately paying patients- a cross sectional study. *BMC Pulm. Med.* 2014, 14, 188.
5. Zinchuk, A.V.; Gentry, M.J.; Concato, J.; Yaggi, H.K. Phenotypes in obstructive sleep apnea: A definition, examples and evolution of approaches. *Sleep Med. Rev.* 2017, 35, 113–123.

6. Butler, M.P.; Emch, J.T.; Rueschman, M.; Sands, S.A.; Shea, S.A.; Wellman, A.; Redline, S. Apnea–Hypopnea Event Duration Predicts Mortality in Men and Women in the Sleep Heart Health Study. *Am. J. Respir. Crit. Care Med.* 2019, 199, 903–912.
7. Azarbarzin, A.; A Sands, S.; Stone, K.L.; Taranto-Montemurro, L.; Messineo, L.; I Terrill, P.; Ancoli-Israel, S.; Ensrud, K.; Purcell, S.; White, D.P.; et al. The hypoxic burden of sleep apnoea predicts cardiovascular disease-related mortality: The Osteoporotic Fractures in Men Study and the Sleep Heart Health Study. *Eur. Hear. J.* 2019, 40, 1149–1157.
8. Montesi, S.B.; Edwards, B.; Malhotra, A.; Bakker, J.P. The Effect of Continuous Positive Airway Pressure Treatment on Blood Pressure: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *J. Clin. Sleep Med.* 2012, 8, 587–596.
9. Castro-Grattoni, A.L.; Torres, G.; Martínez-Alonso, M.; Barbé, F.; Turino, C.; Sánchez-De-La-Torre, A.; Cortijo, A.; Duran-Cantolla, J.; Egea, C.; Cao, G.; et al. Blood pressure response to CPAP treatment in subjects with obstructive sleep apnoea: The predictive value of 24-h ambulatory blood pressure monitoring. *Eur. Respir. J.* 2017, 50, 1700651.
10. Capdevila, O.S.; Gozal, L.; Dayyat, E.; Gozal, D. Pediatric Obstructive Sleep Apnea: Complications, Management, and Long-term Outcomes. *Proc. Am. Thorac. Soc.* 2008, 5, 274–282.
11. Garg, R.K.; Afifi, A.M.; Garland, C.B.; Sanchez, R.; Mount, D.L. Pediatric Obstructive Sleep Apnea: Consensus, Controversy, and Craniofacial Considerations. *Plast. Reconstr. Surg.* 2017, 140, 987–997.
12. Bazzano, L.A.; Hu, T.; Bertisch, S.M.; Yao, L.; Harville, E.W.; Gustat, J.; Chen, W.; Webber, L.S.; Redline, S. Childhood obesity patterns and relation to middle-age sleep apnoea risk: The Bogalusa Heart Study. *Pediatr. Obes.* 2016, 11, 535–542.
13. Sands, S.A.; Eckert, D.J.; Jordan, A.S.; Edwards, B.A.; Owens, R.L.; Butler, J.P.; Schwab, R.J.; Loring, S.H.; Malhotra, A.; White, D.P.; et al. Enhanced Upper-Airway Muscle Responsiveness Is a Distinct Feature of Overweight/Obese Individuals without Sleep Apnea. *Am. J. Respir. Crit. Care Med.* 2014, 190, 930–937.
14. Eckert, D.J.; White, D.P.; Jordan, A.S.; Malhotra, A.; Wellman, A. Defining phenotypic causes of obstructive sleep apnea. *Identif. Nov. Ther. Targets. Am. J. Respir. Crit. Care Med.* 2013, 188, 996–1004.
15. Owens, R.L.; Edwards, B.A.; Eckert, D.J.; Jordan, A.S.; Sands, S.A.; Malhotra, A.; White, D.P.; Loring, S.H.; Butler, J.P.; Wellman, A. An Integrative Model of Physiological Traits Can be Used to Predict Obstructive Sleep Apnea and Response to Non Positive Airway Pressure Therapy. *Sleep* 2015, 38, 961–970.

16. Azarbarzin, A.; Sands, S.A.; Taranto-Montemurro, L.; Marques, M.D.O.; Genta, P.R.; Edwards, B.A.; Butler, J.; White, D.P.; Wellman, A. Estimation of Pharyngeal Collapsibility During Sleep by Peak Inspiratory Airflow. *Sleep* 2016, 40, zsw005.
17. Kotecha, B.T.; Hannan, S.A.; Khalil, H.M.; Georgalas, C.; Bailey, P. Sleepnasendoscopy: A 10-year retrospective audit study. *Eur. Arch. Otorhinolaryngol.* 2007, 264, 1361–1367.41.
18. MacKay, S.G.; Carney, A.S.; Woods, C.; Antic, N.; McEvoy, R.D.; Chia, M.; Sands, T.; Jones, A.; Hobson, J.; Robinson, S. Modified uvulopalatopharyngoplasty and coblation channeling of the tongue for obstructive sleep apnea: A multi-centre Australian trial. *J. Clin. Sleep Med.* 2013, 9, 117–124.42.
19. Pang, K.P.; Siow, J.K.; Tseng, P. Safety of multilevel surgery in obstructive sleep apnea: A review of 487 cases. *Arch. Otolaryngol. Head Neck Surg.* 2012, 138, 353–357.43.
20. Vicini, C.; Montevecchi, F.; Campanini, A.; Dallan, I.; Hoff, P.T.; Spector, M.E.; Thaler, E.; Ahn, J.; Baptista, P.; Remacle, M.; et al. Clinical outcomes and complications associated with TORS for OSAHS: A benchmark for evaluating an emerging surgical technology in a targeted application for benign disease. *ORL J. Otorhinolaryngol. Relat. Spec.* 2014, 76, 63–69.
21. Koutsourelakis, I.; Safiruddin, F.; Ravesloot, M.; Zakyntinos, S.; de Vries, N. Surgery for obstructive sleep apnea: Sleep endoscopy determinants of outcome. *Laryngoscope* 2012, 122, 2587–2591.
22. Yngkaran, T.; Kanaglingam, J.; Rajeswaran, R.; Georgalas, C.; Kotecha, B. Long-term outcomes of laser-assisted uvulopalatoplasty in 168 patients with snoring. *J. Laryngol. Otol.* 2006, 120, 932–938.
23. Edwards, B.A.; Andara, C.; Landry, S.; Sands, S.A.; Joosten, S.A.; Owens, R.L.; White, D.P.; Hamilton, G.S.; Wellman, A. Upper-Airway Collapsibility and Loop Gain Predict the Response to Oral Appliance Therapy in Patients with Obstructive Sleep Apnea. *Am. J. Respir. Crit. Care Med.* 2016, 194, 1413–1422.
24. Joosten, S.A.; Leong, P.; Landry, S.A.; Sands, S.A.; Terrill, P.I.; Mann, D.; Turton, A.; Rangaswamy, J.; Andara, C.; Burgess, G.; et al. Loop Gain Predicts the Response to Upper Airway Surgery in Patients with Obstructive Sleep Apnea. *Sleep* 2017, 40, 1–10.
25. Li, Y.; Ye, J.; Han, D.; Cao, X.; Ding, X.; Zhang, Y.; Xu, W.; Orr, J.; Jen, R.; Sands, S.; et al. Physiology-Based Modeling May Predict Surgical Treatment Outcome for Obstructive Sleep Apnea. *J. Clin. Sleep Med. JCSM: Off. Publ. Am. Acad. Sleep Med.* 2017, 13, 1029–1037.
26. Wellman, A.; Malhotra, A.; Jordan, A.S.; Stevenson, K.E.; Gautam, S.; White, D.P. Effect of oxygen in obstructive sleep apnea: Role of loop gain. *Respir. Physiol. Neurobiol.* 2008, 162, 144–151.

27. Sands, S.A.; Edwards, B.A.; Terrill, P.I.; Butler, J.P.; Owens, R.L.; Taranto-Montemurro, L.; Azarbarzin, A.; Marques, M.; Hess, L.B.; Smales, E.T.; et al. Identifying obstructive sleep apnoea patients responsive to supplemental oxygen therapy. *Eur. Respir. J.* 2018, 52, 1800674.
28. Xie, A.; Teodorescu, M.; Pegelow, D.F.; Teodorescu, M.C.; Gong, Y.; Fedie, J.E.; Dempsey, J.A. Effects of stabilizing or increasing respiratory motor outputs on obstructive sleep apnea. *J. Appl. Physiol.* 2013, 115, 22–33.
29. Taranto-Montemurro, L.; Sands, S.A.; Edwards, B.A.; Azarbarzin, A.; Marques, M.; de Melo, C.; Eckert, D.J.; White, D.P.; Wellman, A. Desipramine improves upper airway collapsibility and reduces OSA severity in patients with minimal muscle compensation. *Eur. Respir. J.* 2016, 48, 1340–1350.
30. Mediano, O.; González Mangado, N.; Montserrat, J.M.; Alonso-Álvarez, M.L.; Almendros, I.; Alonso-Fernández, A.; Barbé, F.; Borsini, E.; Caballero-Eraso, C.; Cano-Pumarega, I.; et al. Spanish Sleep Network. *Int. Consens. Doc. Obstr. Sleep Apnea. Arch. Broconeumol.* 2022, 58, 52–68.
31. Chang, J.L.; Goldberg, A.N.; Alt, J.A.; Ashbrook, L.; Auckley, D.; Ayappa, I.; Bakhtiar, H.; Barrera, J.E.; Bartley, B.L.; Billings, M.E.; et al. International consensus statement on obstructive sleep apnea. *Int. Forum Allergy Rhinol.* 2022.
32. Duarte, R.L.M.; Togeiro, S.M.G.P.; Palombini, L.O.; Rizzatti, F.P.G.; Fagundes, S.C.; Magalhães-da-Silveira, F.J.; Cabral, M.M.; Genta, P.R.; Lorenzi-Filho, G.; Clímaco, D.C.S.; et al. Brazilian Thoracic Association Consensus on Sleep-disordered Breathing. *J. Bras. Pneumol.* 2022, 48, e20220106.
33. Wellman, A.; Eckert, D.; Jordan, A.; Edwards, B.; Passaglia, C.; Jackson, A.C.; Gautam, S.; Owens, R.L.; Malhotra, A.; White, D.P. A method for measuring and modeling the physiological traits causing obstructive sleep apnea. *J. Appl. Physiol.* 2011, 110, 1627–1637.
34. Wellman, A.; Edwards, B.; Sands, S.; Owens, R.L.; Nemat, S.; Butler, J.; Passaglia, C.; Jackson, A.C.; Malhotra, A.; White, D.P. A simplified method for determining phenotypic traits in patients with obstructive sleep apnea. *J. Appl. Physiol.* 2013, 114, 911–922.

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