

Short Communication

*Corresponding author

Ana Paula Hermont, PhD, MSc, BDS

Department of Pediatric Dentistry and
Orthodontics

Faculty of Dentistry

Universidade Federal de Minas Gerais –

Av. Antônio Carlos 6627

Belo Horizonte

MG 31270-901, Brazil

Tel. +5531997618587

E-mail: anapaulahermont@gmail.com

Volume 2 : Issue 1

Article Ref. #: 1000EPOJ2109

Article History

Received: December 12th, 2016

Accepted: January 4th, 2017

Published: January 6th, 2017

Citation

Guimarães MLR, Hermont AP. Obstructive sleep apnea and oral appliances. *Epidemiol Open J.* 2017; 2(1): 16-18. doi: [10.17140/EPOJ-2-109](https://doi.org/10.17140/EPOJ-2-109)

Copyright

©2017 Hermont AP. This is an open access article distributed under the Creative Commons Attribution 4.0 International License (CC BY 4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Obstructive Sleep Apnea and Oral Appliances

Maria de Lourdes Rabelo Guimarães, BDS, DMD¹; Ana Paula Hermont, PhD, MSc, BDS^{2*}

¹Faculty of Dentistry, Universidade Federal de Minas Gerais, Belo Horizonte, Brazil;

Benvinda de Carvalho, 105/101, Santo Antônio, Belo Horizonte, Minas Gerais, Brazil

²Department of Pediatric Dentistry and Orthodontics, Faculty of Dentistry, Universidade

Federal de Minas Gerais – Av. Antônio Carlos 6627, Belo Horizonte, MG 31270-901, Brazil

Central sleep apnea is a sleep-related breathing disorder characterized by repetitive cessation or decrease of both airflow and respiratory effort during sleep.¹ Conversely, in obstructive sleep apnea (OSA) there is a respiratory effort against a closed upper airway evidencing that patients who suffer from OSA have anatomic/functional deficits in the upper airways.² When associated with clinical signs and symptoms such as excessive daytime sleepiness, cognitive deficit, decreased quality of life (QoL) and increased cardiovascular morbidity it is called obstructive sleep apnea syndrome (OSAS).³⁻⁵

In clinical practice it is noticeable that rarely do patients suffer from pure OSAS, the majority of them exhibits some proportion of central and/or mixed events. The coexistence of two types of apnea indicates more complex underlying disorders than just increased resistance in the upper airway. In fact, unstable respiratory control has been implicated in the pathogenesis of both central and obstructive sleep apnea.²

Oral appliances (OA) are recommended exclusively as a treatment option for OSAS. In the literature, 3 large groups of OA are found: tongue retaining devices, anterior mandibular repositioning devices and lifting devices of the soft palate and uvula. Combined therapy using intraoral positive-pressure devices can also be used.⁶ Nowadays, the category of anterior mandibular repositioning devices is by far the most common type of OA in use.⁷

Many different device designs have contributed towards the misunderstanding related to the success of the treatment. In order to correct these deficiencies, a consensus conference was held to develop an evidence-based definition of an effective OA for the treatment of sleep-related breathing disorders and to establish a standard reference for research and clinical practice. Thus, the American Academy of Dental Sleep Medicine (AADSM) has determined that the OA terminology refers to mandibular advancement devices because they are the most effective and widely used in clinical practice. However, this definition will be re-evaluated and revised as new methods and evidences become available. Innovations have been released in OSA treatments, including advances in relation to OA treatment.⁸⁻¹¹

Although OA are indicated exclusively for the treatment of OSA, a study published in 1991 showed two cases of patients with central apnea treated with this device.¹² The pathophysiology of central sleep apnea syndrome has not yet been fully clarified, but obstruction of the upper airway may be one of the causes.¹³ This relationship between central sleep apnea and partial obstruction of the upper airway was suggested in a study in which a continuous positive airway pressure device (CPAP), administered to improve respiratory load during sleep, eliminated central sleep apnea in patients who had narrow upper airways, but it had no positive effect on central apnea in patients with brainstem lesions.¹⁴

Central sleep apnea emergent to the treatment of OSA has been described, especially after therapy with positive airway pressure (PAP) device. However, it is important to be aware that central apneas can also arise by treatment with OA.¹⁵ Thomas et al¹⁶ has used OA

in patients with complex apnea and who are intolerant to PAP with reasonable success. According to these authors, residual sleep apnea may occur when using OA and it requires adjuvant therapy. Once OA are less likely to induce hypocapnia and are also less effective in treating obstructions, the researcher has used “cocktails” in the treatments including OA. He has used a combination with acetazolamide, or a benzodiazepine, or supplemental oxygen.

In our clinical routine we have observed that the polysomnography of patients with OSA often does not exhibit central and mixed events in the reports. We believe it happens due to the fact that scores of respiratory events in patients with sleep apnea have traditionally been inclined to an obstructive phenotype, although the recent 2007 update of the AASM guidelines established some criteria to quantify central hypopneas and short sequences of periodic breathing/cheyne-stokes respiration.¹⁷

Scoring guidelines for respiratory events predict that central hypopneas should not be marked in the presence of flow limitation, but obstruction is a common feature of central events.¹⁶ Direct visualization of the upper airway commonly shows collapse in the nadir of the cycle, even in polysomnography of the “central” disease. Narrowing of the pharynx on expiration occurs during central hypocapnic hypopnea, supporting the concept that the presence of flow limitation by itself cannot be used to distinguish between obstructive and central hypopnea.¹⁸⁻²⁰

We consider that scoring central and mixed events in polysomnography is important not only in the baseline assessment of sleep-related breathing disorders, but it should also be taken into account by the criteria used to estimate the therapies success.

More studies are needed to better evaluate and clarify the relationship between obstructive, central and mixed events and the impact of OA on the treatment of these sleep-related respiratory events.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

REFERENCES

1. American Academy of Sleep Medicine. *International Classification of Sleep Disorders*. 3rd ed. Darien, IL, USA; American Academy of Sleep Medicine; 2014.
2. Xie A, Bedekar A, Skatrud JB, Teodorescu M, Gong Y, Dempsey JA. The heterogeneity of obstructive sleep apnea (predominant obstructive vs pure obstructive apnea). *Sleep*. 2011; 34(6): 745-750. doi: [10.5665/SLEEP.1040](https://doi.org/10.5665/SLEEP.1040)
3. Barbé, Pericas J, Munoz A, Findley L, Anto JM, Agusti AG.

Automobile accidents in patients with sleep apnea syndrome. An epidemiological and mechanistic study. *Am J Respir Crit Care Med*. 1998; 158(1): 18-22. doi: [10.1164/ajrccm.158.1.9709135](https://doi.org/10.1164/ajrccm.158.1.9709135)

4. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. *N Engl J Med*. 2005; 353: 2034-2041. doi: [10.1056/NEJMoa043104](https://doi.org/10.1056/NEJMoa043104)

5. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep*. 1999; 22: 667-689.

6. SD Smith. Oral appliances in the treatment of obstructive sleep apnea-hypopnea syndrome. *RCOE*. 2002; 7(4): 391-402.

7. Hoffstein V. Review of oral appliances for treatment of sleep-disordered breathing. *Sleep Breath*. 2007; 11(1): 1-22. doi: [10.1007/s11325-006-0084-8](https://doi.org/10.1007/s11325-006-0084-8)

8. Scherr SC, Dort LC, Almeida FR. Definition of an effective oral appliance for the treatment of obstructive sleep apnea and snoring: A report of the American Academy of Dental Sleep Medicine. *J Dent Sleep Med*. 2014; 1(1): 39-50. Web site. <http://www.aadsm.org/resources/pdf/consensusReport.pdf>. Accessed December 11, 2016.

9. Singh GD, Keropian B, Pillar G. Effects of the full breath solution appliance for the treatment of obstructive sleep apnea: A preliminary study. *Cranio*. 2009; 27(2): 109-117. doi: [10.1179/crn.2009.017](https://doi.org/10.1179/crn.2009.017)

10. Ngiam J, Kyung HM. Microimplant-based mandibular advancement therapy for the treatment of snoring and obstructive sleep apnea: A prospective study. *Angle Orthod*. 2012; 82(6): 978-984. doi: [10.2319/071311-449.1](https://doi.org/10.2319/071311-449.1)

11. Dort L, Remmers J. A combination appliance for obstructive sleep apnea: The effectiveness of mandibular advancement and tongue retention. *J Clin Sleep Med*. 2012; 8(3): 265-269. doi: [10.5664/jcsm.1910](https://doi.org/10.5664/jcsm.1910)

12. Farrow SJ. Successful treatment of central sleep apnea with an oral prosthesis. *Chest*. 1991; 100(5): 1461-1462. doi: [10.1378/chest.100.5.1461](https://doi.org/10.1378/chest.100.5.1461)

13. Bradley TD, McNicholas WT, Rutherford R, Popkin J, Zamel N, Phillipson EA. Clinical and physiologic heterogeneity of the central sleep apnea syndrome. *Am Rev Respir Dis*. 1986; 134(2): 217-221. Web site. <http://www.atsjournals.org/doi/abs/10.1164/arrd.1986.134.2.217>. Accessed December 11, 2016.

14. Guilleminault C, Quera-Salva MA, Nino-Murcia G, Partinen M. Central sleep apnea and partial obstruction of the upper airway. *Ann Neurol*. 1987; 21(5): 465-469. doi: [10.1002/](https://doi.org/10.1002/)

[ana.410210509](#)

15. Mohan A, Henderson J, Mador MJ. Mandibular advancement device-emergent central sleep apnea can resolve spontaneously: A case report. *J Clin Sleep Med*. 2016; 12(1): 137-138. doi: [10.5664/jcsm.5414](https://doi.org/10.5664/jcsm.5414)

16. Thomas RJ. Alternative approaches to treatment of central sleep apnea. *Sleep Med Clin*. 2014; 9(1): 87-104. doi: [10.1016/j.jsmc.2013.10.008](https://doi.org/10.1016/j.jsmc.2013.10.008)

17. Berry RB, Budhiraja R, Gottlieb DJ, et al. Rules for scoring respiratory events in sleep: Update of the 2007 AASM Manual for the Scoring of Sleep and Associated Events. Deliberations of the Sleep Apnea Definitions Task Force of the American Academy of Sleep Medicine. *J Clin Sleep Med*. 2012; 8(5): 597-

619. doi: [10.5664/jcsm.2172](https://doi.org/10.5664/jcsm.2172)

18. Badr MS, Toiber F, Skatrud JB, Dempsey J. Pharyngeal narrowing/occlusion during central sleep apnea. *J Appl Physiol (1985)*. 1995; 78(5): 1806-1815. Web site. <http://jap.physiology.org/content/78/5/1806.long>. Accessed December 11, 2016.

19. Thomas RJ, Tamisier R, Boucher J, et al. Nocturnal hypoxia exposure with simulated altitude for 14 days does not significantly alter working memory or vigilance in humans. *Sleep*. 2007; 30(9): 1195-1203.

20. Sankri-Tarbichi AG, Rowley JA, Badr MS. Expiratory pharyngeal narrowing during central hypocapnic hypopnea. *Am J Respir Crit Care Med*. 2009; 179(4): 313-319. doi: [10.1164/rccm.200805-741OC](https://doi.org/10.1164/rccm.200805-741OC)