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Mild Obstructive Sleep Apnea Syndrome Should Not Be Treated

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The diagnosis of obstructive sleep apnea (OSA) generally requires a presenting symptom or symptoms and the demonstration of apneas and/or hypopneas per hour of sleep (AHI) on a formal sleep study. The diagnosis of mild OSA is not well defined but in a 1999 report of the American Academy of Sleep Medicine (AASM), definitions of mild OSA by symptoms of sleepiness and AHI were proposed. These definitions will be used in this review.

Mild symptoms were an unwanted sleepiness or involuntary sleep episodes occurring during activities that require little attention. Examples included sleepiness that is likely to occur while watching television, reading, or traveling as a passenger. Symptoms produce only minor impairment of social or occupational function. Mild levels of AHI were 5 to 15 and moderate were 15 to 30.[1]

Although there are many studies that address treatment of OSA, relatively few focus on mild OSA. In addition, there are several potential treatment modalities. For purposes of this review, continuous positive airway pressure (CPAP) will be the main treatment comparator. Interventions such as attempted weight loss, sleep position, sleep hygiene, etc. will be considered part of usual care and not specific treatment of OSA.

In 2006, The AASM published evidence-based practice parameters (recommendations) on the use of CPAP in the treatment of OSA. The recommendation for mild OSA was as follows[2]:

"CPAP is recommended for the treatment of mild OSA (Option). This recommendation as an option is based on mixed results in 2 Level I and 3 Level II outcome studies in patients with mild OSA. An option is a patient-care strategy, which reflects uncertain clinical use." "The term option implies either inconclusive or conflicting evidence or conflicting expert opinion."

The evidence review[3] that supported the above recommendation concluded as follows:

"The sole study that examined change in blood pressure associated with treatment in milder OSA using a tablet placebo failed to show differences between CPAP treatment and placebo. The 2 Level II studies that evaluated the impact of CPAP versus placebo on heart rate produced conflicting results. Therefore, the impact of CPAP treatment on cardiovascular risk and associated organ dysfunction in milder OSA is unknown." and "The 3 Level I studies and 3 Level II studies that were restricted to patients with mild to moderate OSA found that CPAP reduced AHI but did not improve objective sleepiness or blood pressure. Conflicting results were found for subjective measures of sleepiness, neurobehavioral performance, mood and quality of life. Thus, it remains unclear whether CPAP has utility across outcomes for this level of disease severity." Of note, the Level I and II studies were randomized controlled trials but the design (usually a greater sample size) of Level I studies provided stronger evidence than Level II studies.

In summary, none of AHI, objective sleepiness, systemic blood pressure, subjective sleepiness, neurobehavioral performance, mood, or quality of life were obviously improved by CPAP in mild to moderate OSA.

The review concluded further "There are no Level I or II studies that have examined the efficacy or effectiveness of CPAP treatment in OSA patient with an AHI < 5. There have been several Level III studies as described in a large review paper that have examined the use of CPAP in Upper Airway Resistance Syndrome (with an AHI <5) and in subjects with an AHI <10. There is insufficient evidence to draw conclusions regarding the efficacy and/or effectiveness of CPAP treatment in this population."

Apart from the difficulty in documenting improvement with CPAP in mild OSA, the issue of acceptance and adherence should be taken into account. For example, a randomized study of the diagnosis of OSA by either polysomnography or home portable monitor followed by autotitrating PAP treatment (APAP) was performed.[4] Of 32 patients undergoing polysomnography with an AHI less than 10 per hour of sleep, 10 had improved quality of life at 4 weeks and 4 continued to adhere with APAP for 3 or more months. The comparable figures for diagnosis by home monitoring were 69, 18 and 3. In summary, patients with mild OSA are not usually improved by PAP treatment and even if improved are not likely to be adherent.

Cohort studies of mortality with CPAP treatment suggest that only those patients with an AHI greater than 30 or an AI greater than 20 have a reduced mortality. For example, in a retrospective analysis of OSA only those patients with an AI greater than 20 had substantial mortality over 9 years and those patients also benefited from treatment with CPAP or tracheostomy.[5] More recently, a prospective cohort study indicated that patients with a previous

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transient ischemic attack or stroke and OSA and an AHI equal to or greater than 30 had a reduced mortality compared to those with an AHI of 5 to 30.[6] Another prospective nonrandomized study[7] included OSA untreated with CPAP, OSA treated with CPAP, simple snorers and healthy controls. This study provided evidence that CPAP was effective in preventing cardiovascular events or death in OSA patients with an AHI greater than 30 but ineffective in those with an AHI less than 30. The rates of events and deaths were statistically similar in all groups except those with an AHI greater than 30 untreated with CPAP. This group had an increased incidence of cardiovascular events and mortality.

The question may be raised about other modalities to treat mild OSA. As mentioned, "conservative" or medical therapy was considered to be usual care. Such care may be effective in improving symptoms in patients with OSA. A recent review and practice parameter indicated that weight loss, positional therapy in patients with supine OSA, and nasal corticosteroids in patients with allergic rhinitis may be effective.[8] The use of CPAP has been compared to a dental appliance (also called mandibular advancement device, mandibular advancement splint, or mandibular repositioning appliance) and found to be superior, particularly with respect to AHI.[9] A dental appliance has been compared to surgery (uvulopalatopharyngoplasty) and found to be superior.[10] This suggests a hierarchy of effectiveness of CPAP > dental appliance > surgery. Since CPAP is not effective in treating mild to moderate OSA, it is unlikely that dental appliances or surgery would also be effective.

In summary, the benefits of CPAP compared to usual care with respect to daytime sleepiness, symptoms, cardiovascular risk, quality of life and mortality are minimal or nonexistent in patients with mild obstructive sleep apnea and the adherence to PAP at three months appears inadequate.

I conclude that first line treatment of mild obstructive sleep apnea should be medical. CPAP and other modalities such as a dental appliance or surgery should be reserved for failed treatment in highly selected cases.

REFERENCES

1. The Report of an American Academy of Sleep Medicine Task Force Sleep–related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. Sleep 1999;22:667-89
2. Kushida CA, Littner MR, Hirshkowitz M, et al. Practice parameters for the use of continuous and bilevel positive airway pressure devices to treat adult patients with sleep-related breathing disorders. An American Academy of Sleep Medicine Report. Sleep 2006;29:375-80.
3. Gay P, Weaver T, Loube D, Iber C, et al. Evaluation of positive airway pressure treatment for sleep related breathing disorders in adults. A Review by the Positive Airway Pressure Task Force of the Standards of Practice Committee of the American Academy of Sleep Medicine. Sleep 2006;29:381-401.
4. Whitelaw WA, Brant RF, Flemons WW. Clinical usefulness of home oximetry compared with polysomnography for assessment of sleep apnea. Am J Respir Crit Care Med 2005;171:188-93.
5. He J, Kryger MH, Zorick FJ, Conway W, Roth T. Mortality and apnea index in obstructive sleep apnea. Experience in 385 male patients. Chest 1988;94:9-14.
6. Parra O, Arboix A, Montserrat JM, Quinto L, Bechich S, Garcia-Eroles L. Sleep-related breathing disorders: impact on mortality of cerebrovascular disease. Eur Respir J. 2004;24:267-72.
7. Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. Lancet 2005;365(9464):1046-53.
8. Morgenthaler TI, Kapen S, Lee-Chiong T, et al. Practice parameters for the medical therapy of obstructive sleep apnea. Standards of Practice Committee of the American Academy of Sleep Medicine. Sleep 2006;29:1031-5.
9. Barnes M, McEvoy RD, Banks S, et al. Efficacy of positive airway pressure and oral appliance in mild to moderate obstructive sleep apnea. Am J Respir Crit Care Med 2004;170:656-64.
10. Walker-Engstrom ML, Tegelberg A, Wilhelmsson B, Ringqvist I. 4-year follow-up of treatment with dental appliance or uvulopalatopharyngoplasty in patients with obstructive sleep apnea: a randomized study. Chest 2002;121:739-46.