Editorial

Rajagopal T.P.* Safreena M.**
*Professor and Head, **Assistant Professor, Department of Pulmonary Medicine,
Institute of Chest Diseases, Government Medical College, Kozhikode.

A healthier way to sleep: Tackling obesity in Obstructive Sleep Apnoea

The sleep industry is booming as increased awareness amongst the public and medical personnel is leading to the diagnosis of sleep apneas in large numbers. Although Continuous Positive Airway Pressure (CPAP) remains the standard of care for Obstructive Sleep Apnea (OSA), a multitude of oral appliances as well as upper airway surgical procedures are available as treatment options. However, it is a matter of concern that the reversible and preventable factors causing OSA are inadequately addressed even as CPAP is widely prescribed.

The theme of the 2014 World Sleep Day is "*Restful Sleep, Easy Breathing, Healthy Body*"[1]. This is a three-in-one message highlighting the preventable risk factors for obstructive sleep apnea. These simple, yet effective treatment strategies include weight reduction, smoking cessation and avoidance of alcohol and other respiratory depressants. Measures to improve sleep hygiene should also be advocated, including avoidance of caffeine and other stimulants, a regular sleep-wake schedule, environmental measures to promote a comfortable undisturbed sleep, and avoidance of daytime napping.

Obesity is a major (and perhaps the leading) risk factor for obstructive sleep apnoea[2]. The growing rates of obesity also contributes to increasing prevalence of OSA[3]. About two-thirds of patients with OSA are obese. Weight gain represents a high risk for the further progression of OSA. Data from the Wisconsin Sleep Cohort study, a large population-based study aimed to determine the natural history of cardiopulmonary effects of sleep-disordered breathing and its prevalence, indicate that a one standard deviation increase in the body mass index (BMI) is associated with a 4-fold increase in risk for sleep disordered breathing and that each percentage change in weight is associated with an approximate mean change in the Apnea-Hypopnea Index (AHI) of 3%[4].

There are several mechanisms by which obesity could result in OSA, and these may act synergistically[5]. It is proposed that increased peripharyngeal fat deposition results in mechanical loading that offsets the maintenance of airway patency by the dilator muscles and that this increase in collapsibility is particularly prominent during sleep when there is a reduction in neuromuscular activity. Pharyngeal collapsibility may be further accentuated by obesity related reduction in functional residual capacity (FRC) and subsequent decrease in tracheal traction on the pharynx. Finally, a self-perpetuating cycle may develop in which sleep disruption leads to increased appetite (especially for calorie-rich high carbohydrate foods), reduced activity levels, further weight gain, and increased severity of OSA. Weight loss results in a reduction in pharyngeal fat pads and structural changes that increases upper airway volume.

Currently, weight loss is not recommended as the sole therapy for OSA. Instead, it is to be used in conjunction with other treatments such as CPAP[6,7,8]. Intensive weight-loss interventions help reduce AHI scores, improve OSA symptoms and are associated with health benefits other than for OSA. Data from a population-based study shows that a 10% reduction in weight was associated with a 26% decrease in the AHI[9]. Weight reduction may lower the effective nasal CPAP pressure required to control OSA and thereby potentially improve compliance. However, obese patients should not expect a complete cure of their OSA but could achieve improvements in severity of disease. OSA persists in many patients despite major weight loss, and use of CPAP should be anticipated.

The decision of how aggressively to treat patients and which modalities to use is determined by the patient's risk status, their abilities and desires, and by what resources are available. Thus weight loss strategies vary and can mainly be classified into lifestyle (including caloric restriction, behavioural therapy, and exercise), pharmacologic and surgical treatments[10].

Obesity is fundamentally a disease of energy imbalance. Hence all patients must learn how and when energy is consumed (diet), how and when energy is expended (exercise), and how to incorporate this information into their daily life (behaviour therapy). Lifestyle management has been shown to result in a modest (typically 3-5 kg) weight loss compared to no treatment or usual care[10]. In comprehensive lifestyle interventions, overweight and obese individuals are typically initiated on a diet designed to induce an energy deficit of 500 kcal/day from the patient's habitual diet. This requires a dietary intake of 1200-1500 kcal/day for females and 1500-1800 kcal/day for males. This goal can be accomplished by choosing smaller portion sizes, eating more fruits, vegetables and whole grain cereals and using meal replacements. Patients are also typically prescribed increased aerobic physical activity (such as brisk walking) for 150 min/week (30 min/day on most days of the week). Higher levels of physical activity, approximately 200 to 300 min/week, are recommended to maintain lost weight or minimize weight regain in the long term (>1 year). Comprehensive lifestyle interventions also provide a structured behaviour change program that includes regular self monitoring of food intake, physical activity, and weight[10,11].

The role of anti-obesity drugs in the treatment of OSA is not clear. The current guidelines on obesity recommend adjunctive pharmacotherapy for patients with a BMI of 30 kg/m^2 or with a BMI of 27 kg/m^2 who also have concomitant obesity-related risk factors or diseases and for whom dietary and physical activity therapy has not been successful[10,11]. Orlistat inhibits gastrointestinal lipase thus reducing fat absorption, but its use in OSA needs to be studied in clinical trials[8]. Sibutramine, a serotonin and noradrenaline reuptake inhibitor, has shown mixed results in patients with OSA, but has been withdrawn from the market due to concerns regarding safety[8]. Rimonabant, a cannabinoid receptor antagonist, has also been withdrawn. Lorcaserin and Phentermine-topiramate are FDA approved medications for obesity[8].

Bariatric surgery can be considered for patients with BMI of 40 kg/m^2 or more, or between 35 and 39.9 kg/m^2 in the presence of significant co morbidities[10,11]. In addition, all nonsurgical methods should have been employed with failure to achieve or maintain adequate weight loss over at least 6 months. Surgical weight loss functions by reducing caloric intake, and depending on the procedure, macronutrient absorption. The two commonly performed procedures are Laparoscopic Adjustable Silicone Gastric Banding (LASGB) and Roux-en-Y

Gastric Bypass (RYGB). For patients who undergo LASGB, there are no intestinal absorptive abnormalities other than mechanical reduction in gastric size and outflow. These procedures are generally effective in producing an average weight loss of approximately 30-35% of total body weight that is maintained in nearly 60% of patients at 5 years. In general, surgical weight loss interventions have shown greater improvements in AHI when compared to dietary treatments alone.

Obesity is a strong causal factor for sleep-disordered breathing, and because of the ongoing obesity epidemic, the prevalence of OSA is on the rise. Guidance for weight loss should be part of the treatment for all overweight and obese patients with OSA[11,12]. Despite the untoward and negative outcomes associated with obesity, achieving weight loss can be quite challenging. Comorbid medical illnesses associated with obesity and symptoms of OSA including hypersomnia and fatigue may affect weight loss goals and efforts[8]. However, the most difficult aspect of managing obesity is the long-term sustenance of the weight loss achieved. A multidisciplinary team approach to weight reduction, aiming at realistic and sustainable weight loss is required to optimize clinical results.

References

1. World Association of Sleep Medicine. Available from: URL: http://worldsleepday.org
2. Young T, Peppard PE, Taheri S. Excess weight and sleep-disordered breathing. Journal of Applied Physiology 2005; 99 (4): 1592–1599.
3. Peppard PE, Young T, Barnet JH et al. Increased Prevalence of Sleep-Disordered Breathing in Adults. American Journal of Epidemiology 2013; 177(9):1006–1014.
4. Young T, Palta M, Dempsey J, et al. Burden of sleep apnea: Rationale, design, and major findings of the Wisconsin Sleep Cohort Study. Wisconsin Med J. 2009; 108(5): 246–249.
5. Cowan CD, Livingston E. Obstructive Sleep Apnoea Syndrome and Weight Loss: Review. Sleep Disorders 2012; Article ID 163296: DOI: 10.1155/2012/163296.
6. Qaseem A, Dallas P, Owens DK et al. Diagnosis of Obstructive Sleep Apnea in Adults: A Clinical Practice Guideline from the American College of Physicians. Ann Intern Med 2014; 161: 210-220.
7. Epstein LJ, Kristo D, Strollo PJ Jr et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. J Clin Sleep Med 2009; 5(3): 263-276.
8. Jones SF, Chebbo A. Weight Loss in the Management of Obstructive Sleep Apnea. Sleep Med Clin 2013; 8 (4): 527–542.
9. Peppard PE, Young T, Palta M, et al. Longitudinal study of moderate weight change and sleep disordered breathing. JAMA 2000; 284: 3015–21.
10. Friedman M. Sleep Apnoea and Snoring: Surgical and Non-Surgical Therapy. 1st ed. Saunders; 2009. p. 51-59.
11. Jensen MD, Ryan DH, Apovian CM et al. 2013 AHA/ACC/TOS guideline for the Management of overweight and obesity in adults: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and The Obesity Society. J Am Coll Cardiol 2014; 63: 2985–3023.
12. Capies SM, Gami AS, Somers VK. Obstructive Sleep Apnoea. Ann Intern Med 2005; 142: 187-97.