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COMMENTARY

From Couch Potato to Gym Junkie—CPAP May Not Be the Answer

Commentary on Batool-Anwar et al. Impact of CPAP on activity patterns and diet in patients with obstructive sleep apnea (OSA). J Clin Sleep Med 2014;10:465-472.

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Obesity is a common cause of obstructive sleep apnea (OSA). Ample evidence of this is provided from longitudinal research and the OSA-minimizing effect of weight loss.[1] However several lines of evidence suggest the relationship may be bi-directional and that OSA may be obesogenic. For example, OSA patients are known to have anabolic hormone deficiency,[2] increased leptin resistance, and increased levels of the appetite-stimulating hormone ghrelin compared to similarly obese people without OSA.[3] Patients with OSA are poorly motivated to exercise with studies, even suggesting a negative association between OSA severity and physical activity levels after adjusting for BMI.[4-6] The combined effects of hormone imbalance and reduced energy expenditure would be expected to result in a positive energy balance. However, contrary to this, there are also several mechanisms that could result in negative energy balance in OSA including increased work of breathing against an obstructed airway during sleep and sleep fragmentation, which, in some severe cases, may be significant.[7,8] Ultimately the state of energy balance in OSA is likely to influence weight change with a positive balance promoting weight gain and a negative balance promoting weight loss. In support of the weight promoting effects of OSA, a recent study showed that OSA patients were resistant to weight loss compared to people with similar levels of central obesity without OSA.[9] However, surprisingly when OSA is treated with continuous positive airway pressure (CPAP), some studies—including the largest randomized controlled trial to date, the Apnea Positive Pressure Long-term Efficacy Study (APPLES)[10]—have reported a paradoxical weight gain.

It is therefore timely that in the this issue of the *Journal of Clinical Sleep Medicine* the APPLES investigators have now examined levels of physical activity and dietary intake to explore potential mechanisms for the previously observed weight gain. In this APPLES sub-study, Batool-Anwar et al.[11] used self-reported physical activity from the Arizona Activity Frequency Questionnaire and dietary intake using a food frequency questionnaire. They found no changes in either physical activity domains or dietary patterns with CPAP compared to sham. These overall findings that CPAP does not change physical activity and/ or dietary intake are in agreement with other studies including one randomized controlled study.[12,13] Interestingly in this study which comprised nearly twice as many men as women, the lack of change in physical activity or dietary habits appeared to be largely influenced by male gender. In fact women showed a modest increase in recreational activity with CPAP. Regardless, these overall findings then raise the question of why body weight might increase following treatment of OSA. The answer may lie in studies that have specifically assessed relative changes in lean and fat mass with OSA treatment rather than changes in BMI/body weight which does not quantify either. Recent short-term randomized studies have consistently shown that fat mass does not change with CPAP.[14,15] However, over longer periods of observation with CPAP, lean body mass has been shown to increase.[14,16] The net effect is for an overall increase in body mass, although other studies have shown that long-term CPAP may also eventually reduce fat mass.[17] In this context, both sarcopenia and abdominal obesity in older men have both been shown to increase the risk of cardiovascular and all-cause mortality, with all-cause mortality being highest in sarcopenic obese men.[18] Hence long-term treatment of OSA during aging may ultimately preserve lean mass but also potentially reduce fat mass, thereby reducing overall mortality risk. Regardless of these findings, the study by Batool-Anwar et al. suggests that the mechanisms that would underpin any increase in lean mass and/ or a decrease in fat mass in the long term do not appear to be influenced by changes in diet or physical activity. They are instead more likely to be directly influenced by as yet unknown mechanisms directly related to alleviation of OSA.

Several limitations to this study may have influenced the results. The CPAP compliance was relatively low at 4 hours and may be insufficient to influence activity or diet. However this usage is not dissimilar to many other studies where positive metabolic and hormonal effects of CPAP have been shown. A further limitation may be the short length of CPAP treatment which was 4 months. Lean muscle and insulin sensitivity improvements have been reported but only after 6 months treatment.[14] Finally, the lack of gold-standard measures to assess physical activity (accelerometry) and dietary intake (food diaries) as well as a direct measure of energy expenditure

(room calorimetry or doubly labelled water) may have resulted in inaccurate assessment of these outcomes.

Despite the limitations, this study does add to our knowledge of the potential role of CPAP to alter behaviors including physical activity and diet, which subsequently impact on obesity and its complications. More information will be obtained by future longer-term randomized controlled studies that utilize gold-standard methods of measuring energy expenditure and balance including assessing the influence of gender. The clinical message is that clinicians cannot assume patients with sleep apnea and obesity will lose weight and become more active by simply using a CPAP machine. Weight loss needs to be a parallel strategy in such patients.

CITATION

Hoyos CM, Phillips CL, Grunstein RR. From couch potato to gym junkie—CPAP may not be the answer. *J Clin Sleep Med* 2014;10(5):473-474.

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