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COMMENTARY

Too Little, but Still Great?

Commentary on Roche et al. Obstructive sleep apnea and sleep architecture in adolescents with severe obesity: effects of a 9-month lifestyle modification program based on regular exercise and a balanced diet. *J Clin Sleep Med*. 2018;14(6):967–976.

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Childhood obesity has reached pandemic proportions, representing a global health care crisis across the world.[1] Since 1980 obesity in the United States has more than tripled among children and quadrupled among teens to 17% and 20.5% respectively.[2,3] There has been a 10-fold increase in obesity in children and adolescents worldwide.[4] Along with increasing obesity overall in children, there has been a concomitant increase in severe obesity, with 2% of children in the United States now meeting criteria for class 3 obesity.[5] Childhood obesity is responsible for an increase in risk for type 2 diabetes, asthma, high blood pressure, high cholesterol, depression, obstructive sleep apnea (OSA) and obesity in adulthood.[6] OSA is a common comorbidity in obese children, as up to 61% of obese children also have OSA.[7] Additionally, both childhood obesity[8] and OSA[9] are linked to cardiac dysfunction in children, which may lead to future heart failure as adults.

In morbidly obese patients, dietary modification and aerobic exercise have been recommended as first line therapies for weight loss and an adjunct therapy for treating OSA.[10,11] Two prior studies measuring the effect of weight loss interventions on OSA in obese adolescents and young adults have shown a decrease in OSA prevalence by 60% and 67%,[12,13] under adult criteria (apnea-hypopnea index [AHI] ≥ 5 events/h). Verhulst et al. performed a study using a benchmark of an AHI ≥ 2 events/h to define OSA in younger obese adolescents.[14] In this study, despite an average weight loss of 24 kg after the 5-month weight loss intervention, OSA was still present in 62% of participants.[14] Another 12-week exercise intervention demonstrated no change in AHI at the end of the program, although there was an increase in sleep duration.[15]

In this issue of the *Journal of Clinical Sleep Medicine*, Roche et al. evaluated the effects of a rigorous long-term weight loss program in extremely obese adolescents.[16] Twenty-four extremely obese (body mass index 42.37 ± 7.72 kg/m^2) adolescents (mean age of 14.7 ± 1.3 years) underwent a 9-month intensive residential weight loss program. The program included cardiorespiratory training for 45–60 minutes five times a week, dietary modification with a 2300–2500 kcal balanced diet, nutritional counseling and behavioral therapy. OSA, defined as an obstructive apnea-hypopnea index (OAHI) ≥ 2 events/h, was present in 58% of subjects. The intervention was highly effective in both improving cardiorespiratory fitness as well as significant weight reduction. Surprisingly, there was no effect of weight loss on OAHI despite significant weight loss (11.1 kg, $P < .0001$) among participants who completed the 9-month program. Despite no improvement in sleep-disordered breathing, there were improvements in sleep duration (increased 34 minutes) and slightly increased REM sleep.

The current study was limited by the unexpected low severity of OSA in these extremely obese adolescents, with a mean OAHI of only 3.7 ± 2.6 events/h among participants with OSA. Given that essentially all participants had only mild-moderate OSA under pediatric criteria (mild OSA under adult criteria); a floor effect may have been seen, with only limited room for improvement in OSA. It may be beneficial for a future study to focus on obese adolescents with more severe OSA. An additional limitation was the absence of CO_2 monitoring. Given the marked obesity in the study, obesity hypoventilation syndrome may have been present, but was not assessed in this study. Inclusion of CO_2 monitoring in future studies may provide a more thorough evaluation of sleep-disordered breathing, beyond only AHI. Finally, future studies would benefit from including measures of cardiac function including blood pressure monitoring and echocardiography. Given that both OSA and obesity are associated with cardiac dysfunction and hypertension, this would provide valuable information on the dynamics between cardiac dysfunction, obesity, OSA and cardiorespiratory fitness. Improved cardiorespiratory fitness is associated with improved cardiac function in obese children,[17,18] but no prior studies have also evaluated comorbid OSA.

Roche et al. should be commended for conducting such a rigorous weight loss program and scientific study in a group of extremely obese adolescents. Unfortunately, even with such a rigorous program, after 9 months, there were no changes in OSA severity in this population. As the authors point out, this is likely due to the marked degree of initial obesity in this population, as the mean body mass index after intervention was still greater than 35 kg/m^2 and the weight loss, while significant, may have been too little. It is possible that with continued weight loss, OSA may resolve. However, it appears that interim

treatment of OSA may be needed while persistent weight loss over the course of a year or more may lead to resolution of OSA. It is noteworthy that despite no improvement in OSA, sleep architecture and sleep duration both improved with a diet and exercise intervention in this group of extremely obese adolescents. The increase in sleep duration may be particularly important for future weight reduction, given the known relationship between short sleep and obesity.[19] Additionally, physical fitness was dramatically improved. Participants had a 14% reduction in body mass index, along with a 26 cm reduction in waist circumference and significant improvement in aerobic fitness. This suggests that while continued weight loss is needed to improve OSA, there are more immediate great benefits that come from a balanced weight loss program in extremely obese children.

CITATION

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