Risk factors and treatment for obstructive sleep apnea amongst obese children and adults

Mark Kohler

Discipline of Paediatrics, University of Adelaide, Women's and Children's Hospital, North Adelaide, South Australia, Australia

Correspondence to Mark Kohler, Discipline of Paediatrics, University of Adelaide, Women's and Children's Hospital, 72 King William Road, North Adelaide, SA 5006, Australia
Tel: +61 8 8161 8393; fax: +61 8 8161 7031; e-mail: mark.kohler@adelaide.edu.au

Current Opinion in Allergy and Clinical Immunology 2009, 9:4–9

Purpose of review

The purpose of this review is to summarize the evidence for the association between obesity and obstructive sleep apnea (OSA), as well as predisposing risk factors and treatment strategies for OSA amongst obese patients.

Recent findings

Recent findings highlight a number of factors including sex, age, upper airway structure and ethnicity, which may predispose patients to OSA when obese. Both invasive and noninvasive weight-reduction strategies also show positive signs of being an effective means to reduce or remediate OSA amongst obese adults and children.

Summary

In view of recent findings, a direct association between body mass and upper airway obstruction should be viewed with caution. Obesity may play a more significant role in the predisposition to OSA amongst particular subgroups of the population, such as adults, and those with particular craniofacial and upper airway morphology. Healthcare prioritization and requirements may be more substantial for such groups. Further, commonly used treatment methods for OSA (such as adenotonsillectomy for children and continuous positive airway pressure for adults) may be less effective for obese individuals. Weight-reduction strategies appear important for an optimal outcome, and such strategies may be more or less invasive depending on the severity of obesity, OSA or both, and other patient complications.

Keywords

adults, children, obesity, obstructive sleep apnea, risk, treatment

Curr Opin Allergy Clin Immunol 9:4–9

1528-4050

Introduction

The prevalence of overweight and obesity in the community continues to grow at an alarming rate despite a growing awareness of the significant morbidity associated with this condition. Amongst such morbidity, obesity is considered a major risk factor for upper airway obstruction during sleep, with increased body mass being associated with three to four-fold increase in severity of upper airway obstruction amongst adults [1,2]. These associations are particularly notable amongst male adults who display comparatively more central fat deposition [3,4]. By contrast to adults, the contribution of body mass to severity of upper airway obstruction during sleep in children is less clear and is likely to be dependent on a number of mediating factors including age, ethnicity and adenotonsillar hypertrophy [5••,6].

In the USA, the prevalence of obesity is reported to have increased from around 22% in adults between 1988 and 1994 to over 30% in 2003–2004 [7,8]. The prevalence of obesity amongst children has risen from around 10% between 1988 and 1991 to around 17% in 2003–2004 [8,9]. Similar trends have been observed in a number of other nations. Clinically significant sleep-related upper airway obstruction, or obstructive sleep apnea (OSA), is reported in 4% of male adults and 2% of female adults, with OSA present in 40% of obese adults and over 90% of morbidly obese adults [10–12]. Amongst children, the prevalence of OSA is estimated at 1–4% [13], whereas 20–30% of obese children demonstrate OSA [14–16]. It is not known whether the incidence of OSA is also increasing in line with rates of obesity. Nonetheless, the marked morbidity associated with both obesity and OSA, and the reported associations between conditions, are of increasing concern in the context of increased obesity amongst the community. A greater understanding of the nature of these associations will lead to better treatment and management plans. The purpose of this review is not to discuss in detail all that is currently known about these conditions or the association between them and related mechanisms. Rather, it is to discuss in general the reported risk for OSA that obesity presents across all ages and to draw particular attention to major

 DOI:10.1097/ACI.0b013e32831d8184

advances in knowledge that have been reported over the last 1–2 years.

This brief review of recent advances in knowledge of the risk for development of OSA amongst obese patients has identified two primary areas of interest, namely, a greater understanding of risk factors, which predispose obese individuals to develop OSA, and treatment effects amongst obese individuals with OSA. Both these topics are discussed for adults and children separately.

Risk factors for obstructive sleep apnea amongst obese adults

The association between obesity and OSA amongst adults is well established and a number of excellent reviews [17•,18•,19] have been recently published summarizing the evidence. The excess adipose tissue of obese individuals acts to reduce airway size and alter airway shape, increasing the susceptibility of the upper airway to collapse [20–23]. These obesity-associated changes may also partly explain sex and age effects. For example, the central adipose distribution typically found amongst obese men may increase risk amongst men, and increases in visceral fat deposition with age may also increase risk of OSA amongst older obese adults. A further predisposition for these interactions may occur amongst specific ethnicities.

Anthropometrics and upper airway anatomy

Location of fat deposition and upper airway structure remain important areas of research investigating risk for OSA. Refined or specific measures of adiposity may provide additional information useful for patient screening and determining risk. Recent results from both a clinic sample and a large community survey found that measures of truncal proportion were a better predictor of the likelihood of OSA than BMI and neck circumference; however, this relationship may only be present amongst men [24•,25]. Results from MRI of the upper airway amongst a smaller clinic sample found pharyngeal fat pad thickness was not predictive of OSA. However, adults with OSA demonstrated differences in airway shape, specifically, narrowing at the lower transversal level and increased pharyngeal muscle thickness [26].

A review of studies published prior to 2007 concludes that a low position of the hyoid bone is found amongst both obese and nonobese adults with OSA; however, obese patients demonstrate larger intermaxillary divergence (the angle between the palatal plane and the mandibular plane). Further, changes in soft-tissue dimensions of the upper airway, such as soft palate length and tongue width, were not associated with OSA amongst obese patients [27]. More recently, anatomical study [28] of the tongue found percentage of fat content was associated with BMI in both sexes and may contribute to OSA in obese adults due to posterior displacement of a large tongue. Similarly, a Japanese study [29] found that adults with OSA had larger tongues for a given maxillomandible size.

Ethnicity

The majority of studies investigating ethnic differences have been conducted in the USA and have generally shown African–American adults to be at greater risk of OSA compared with whites [30,31]. Also, obesity is associated with greater prevalence of OSA amongst American Indians and Hispanics [32]. Finally, OSA is more closely related to craniofacial changes than body mass amongst Asian as compared with white men [33]. The precise reasons for these ethnic differences have not been well defined. Amongst a large group of Japanese OSA patients, obesity was associated with OSA severity; however, contributed less amongst older patients (i.e. $\geq$55 years). OSA was also more severe amongst 'underweight' patients compared with normal-weight patients [34]. A large community survey [35] in Malaysia found BMI but not neck girth was independently associated with habitual snoring; however, truncal measurements were not reported.

Sex

Higher risk for OSA amongst male adults is consistently demonstrated [36,37]; however, studies do not provide enough evidence to determine whether such sex differences are evident across the entire spectrum of upper airway obstruction or amongst severely obese adults. Recordings of partial airway obstruction during sleep, in concordance with assessments of conventional apnea and hypopnea, show increasing severity with age amongst women and increasing severity with older age and increasing BMI amongst men. However, the association between partial upper airway obstruction and BMI was not significant amongst either sex [38•]. Recently, a high prevalence of OSA and a significant association between BMI and OSA severity has been shown amongst morbidly obese women [39].

Treatment of obstructive sleep apnea amongst obese adults

Treatment of OSA is complicated by obesity, and weight-loss strategies are emerging as effective treatment approaches, both alone and in combination with traditional techniques. Treatment for OSA amongst adults predominantly consists of forced ventilation [predominantly through use of continuous positive airway pressure (CPAP) or less commonly, biphasic positive airway pressure (BiPAP)] and weight loss in the case of obese patients. Successful treatment of OSA using dieting is low with only a 5–15% success rate reported over the first 8 years from treatment [40]. CPAP is

effective in minimizing OSA; however, it is burdensome and has poor long-term compliance, especially amongst more mild cases [41]. Recent evidence indicates that only regular CPAP use results in reduced visceral fat and associated metabolism [42•], suggesting that recovery from OSA is unlikely amongst obese patients with poor CPAP compliance. CPAP use in obese adults without significant weight reduction may also limit obesity-associated hypoventilation, despite reducing upper airway obstruction [43•]. Healthcare expense and risk for cardiovascular disease may not be substantially improved in such cases. Hence, surgical weight reduction and exercise have emerged as possible effective treatment alternatives or supplements to CPAP.

Bariatric surgery

A recent review [44] of studies investigating the effectiveness of Bariatric surgery on OSA in association with weight reduction demonstrates great effectiveness. However, studies reviewed typically included small samples and only one study [45] included a follow-up period of more than 5 years. More recently, further evidence has been produced to support the effectiveness of Bariatric surgery in treatment of OSA amongst obese adults. Peluso and Vanek [46] report that gastric bypass surgery resolved OSA in 75% of obese patients over a 3.6–30.6-month follow-up period. Fritscher *et al.* [47] report resolved OSA in 25% of morbidly obese patients, and significant reduction in severity amongst 75% who had undergone gastric bypass surgery. In the same study, magnitude of weight loss was strongly associated with improved oxygen saturation indices. In a larger study, Haines *et al.* [48•] demonstrate reduced OSA severity and required CPAP pressure in association with reduced body mass over 6–42 months following gastric bypass surgery amongst 101 Bariatric patients. A second study [49] assessing changes in CPAP requirements following surgery found that none of 29 morbidly obese CPAP patients required CPAP 9 months postoperatively. In contrast, Lettieri *et al.* [50] found amongst a small predominantly female patient group that all but one patient experienced residual OSA 1 year after surgery despite significant reductions in severity. This reiterates the need for further research to determine specific sex-related risk factors for OSA.

Exercise

Lack of exercise has been previously associated with OSA severity independent of body mass [51]. In support of these findings, Quan *et al.* [52•] has shown that a minimum of 3 h of vigorous exercise each week significantly reduces the risk of OSA (odds ratio = 0.68, 95% confidence interval = 0.51–0.91). In this study, exercise appeared to provide more protection against OSA for men and obese patients. However, implementation of exercise programmes amongst obese OSA patients may be limited, as a recent work [53] has shown reduced exercise tolerance amongst obese OSA patients due to hypertensive response. OSA patients also show reduced intention and willingness to exercise. Smith *et al.* [54•] found that over 60% of 206 OSA patients had little intention or limited implementation of any planned exercise. Less than one-third of patients reported regular established exercise routines. In addition, patients were clustered into one of the four groups based on self-reported exercise motivation. On the basis of this clustering, 34% of OSA patients appear to be disengaged or unmotivated to exercise, with an additional 43% lacking resources to continue and establish regular exercise practices. Clearly, further research is needed to establish the efficacy of exercise as a potent OSA treatment amongst obese patients, and further work will be required to establish a best approach in combating physical and motivational limitations.

Risk factors for obstructive sleep apnea amongst obese children and adolescents

It is thought that obesity is linked with an increased prevalence of OSA amongst children [6] and adolescents [55]. Adenotonsillar hypertrophy may mediate this association [5••,6] and adenotonsillectomy is commonly the first line of treatment for both obese and nonobese child patients. Unfortunately, the effectiveness of adenotonsillectomy appears to be reduced amongst obese children [56,57••,58–60], adding to the suggestion of obesity being an independent risk for OSA such as seen amongst adults. During the past 1–2 years, the interaction of adenotonsillar hypertrophy, OSA and obesity amongst children has been investigated in more detail, and an emerging literature has indicated other potential risk modulators of the association between body mass and OSA severity.

Adenotonsillar hypertrophy

Enlargement of the adenoids and tonsils is thought to lead to more severe upper airway obstruction when in combination with obesity. Recently, Xu *et al.* [61] showed that obesity and adenotonsillar hypertrophy were significant independent risk factors for OSA. It has also been shown that compared with normal-weight children, obese children have a relatively high frequency of tonsillar enlargement [62], potentially predisposing a higher number of obese children to upper airway obstruction.

Ethnicity

A large number of studies investigating the association between obesity and OSA amongst children include cohorts of varied ethnicities without ethnic stratification of results (see [5••] for a more comprehensive review). An independent association between African–American ethnicity and OSA has been reported amongst children

[57••,63], potentially biasing results of studies containing large proportions of African–American children. Further evidence that ethnicity may indeed mediate the association between obesity and OSA was shown by Rudnick *et al.* [64•], who found that African–American children who were obese were most likely to have OSA. Amin *et al.* [57••] recently found that after adjusting for the other variables, African–American ethnicity was a greater predictor than body mass and body mass growth velocity for recurrence of OSA following adenotonsillectomy. Studies [61,65,66] of Chinese children consistently report significant association between body mass and OSA, whereas the few studies [67–69] of white and Hispanic children suggest little or no association. A recent study [70] amongst a group of severely obese Bangladeshi, Afro-Caribbean and white children found ethnicity was not predictive of OSA and BMI within the morbidly obese range was not related to OSA severity. Combined, these findings suggest body mass may interact with ethnicity only up until a point, after which the impact of morbid obesity on pulmonary function supersedes any contribution related to ethnic variation.

Age

Reductions in upper airway tone and responsiveness with age have been reported amongst children [71–73], and suggest a mass effect due to obesity on the upper airway during sleep may be a greater risk for OSA amongst older children and adolescents. A recent review [5••] has highlighted the fact that around two-thirds of studies amongst children with mean age more than 10 years report a significant effect of obesity on OSA as compared with around one-third amongst children with mean age less than 10 years. Two studies have recently investigated the impact of age on the association between obesity and OSA amongst children. The first, amongst snoring white Australian children aged 4–12 years, found body mass, but not age, was a significant but weak predictor of upper airway obstruction severity [68]. The second study, amongst snoring Greek children aged 1–15 years, found risk of OSA was higher amongst younger children ($\leq$6 years) irrespective of body mass; however, older children (>6 years) who were obese were twice as likely to have OSA as compared with nonobese children of the same age [74•]. Clearly, further studies are required to examine age or developmental changes in risk for OSA due to obesity which better account for other potential influences such as adenotonsillar hypertrophy and ethnicity.

Treatment of obstructive sleep apnea amongst obese children and adolescents

Adenotonsillectomy is considered the treatment of choice for OSA during childhood, particularly when associated with adenotonsillar hypertrophy; however, the impact of traditional and alternative treatment strategies amongst obese children with OSA is another area of recent research growth. Consistent with rates reported in previous studies [57••,58–60], Mitchell and Kelly [58] found 76% of obese children as compared with 28% of normal-weight children with OSA still demonstrated OSA after adenotonsillectomy. More recently, Amin *et al.* [57••] demonstrated not only obesity but also growth velocity were predictive of OSA recurrence following adenotonsillectomy. In contrast, a recent study [75] amongst Greek children found obesity, postoperative weight gain and adenotonsillar hypertrophy were not predictive of residual OSA. A distinguishing feature of the results from this study was the low success rate of adenotonsillectomy in 'curing' OSA for both obese and nonobese patients. However, when a higher cutoff for OSA severity was used, the success rates were dramatically increased for both weight groups. Although there may be ethnic differences in susceptibility to OSA and response to treatment, normative data from various ethnic groups are also lacking and may reveal differing 'normal' values compared with a predominantly Anglo-Caucasian literature. Higher baseline values of respiratory event frequency amongst Greek children could explain the discrepancy between results in this recent study and previous findings.

The encouraging findings from weight-loss treatment approaches amongst adults may also prove important in management of OSA amongst obese children. Davis *et al.* [76] found that 20–40 min of daily aerobic exercise over a 13-week period lead to reductions in parent reports of snoring amongst 100 overweight children. Recently, the effect of gastric bypass surgery on sleep amongst 19 obese adolescents was examined. Results show severity of OSA and body mass were significantly improved at around 1 year following surgery, as was sleep efficiency and frequency of arousals [77•].

Central sleep apnoea

Investigation of nonobstructive sleep breathing disorders in the context of child obesity has lagged behind the adult research; however, some new findings suggest this will be an important area of future study. Two articles have recently examined the association between central apnoea and body mass amongst children. The first [14] of these found a relatively high prevalence of central sleep apnoea amongst overweight and obese children. The second article [68] found frequency of central apnoeas was significantly associated with increasing body mass and younger age. A recent review [5••] also reports unpublished results of increasing frequency of central hypopnoea with obesity.

As early as in 1976, recommendations were being made for weight reduction to combat obesity hypoventilation

syndrome (OHS) amongst children [78]. Such recommendations appear to have been either ignored or forgotten. The recent increase in prevalence of childhood obesity should prompt new research investigating prevalence and treatment efficacy for OHS.

Conclusion

Research to date strongly suggests obesity is a major contributor to the pathogenesis and morbidity of OSA across all age groups. Contributing factors in the association of obesity and OSA are also becoming evident and predominantly include age, upper airway structure and ethnicity. Traditional forms of treatment amongst adults and children are increasingly being recognized as inadequate amongst obese patients. Weight-reduction surgery and exercise have emerged as potential effective treatment options amongst adults, either independently or in combination with other treatment approaches. Initial studies indicate these same treatment strategies may be useful amongst an increasingly obese paediatric population.

Acknowledgements

Dr Kurt Lushington for his constructive comments and the National Health and Medical Research Council (NHMRC).

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 79).

1 Peppard PE, Young T, Palta M, *et al.* Longitudinal study of moderate weight change and sleep-disordered breathing. JAMA 2000; 284:3015–3021.

2 Young T, Palta M, Dempsey J, *et al.* The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993; 328:1230–1235.

3 Dancey DR, Hanly PJ, Soong C, *et al.* Gender differences in sleep apnea: the role of neck circumference. Chest 2003; 123:1544–1550.

4 Legato MJ. Gender-specific aspects of obesity. Int J Fertil Womens Med 1997; 42:184–197.

5 •• Kohler MJ, van den Heuvel CJ. Is there a clear link between overweight/obesity and sleep disordered breathing in children? Sleep Med Rev 2008; 12:347–361.

This is a comprehensive review addressing age and ethnicity as modulators of the association between obesity and OSA amongst children.

6 Verhulst SL, Van Gaal L, De Backer W, Desager K. The prevalence, anatomical correlates and treatment of sleep-disordered breathing in obese children and adolescents. Sleep Med Rev 2008; 12:339–346.

7 Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960–1994. Int J Obes Relat Metab Disord 1998; 22:39–47.

8 Ogden CL, Carroll MD, Curtin LR, *et al.* Prevalence of overweight and obesity in the United States, 1999–2004. JAMA 2006; 295:1549–1555.

9 Troiano RP, Flegal KM, Kuczmarski RJ, *et al.* Overweight prevalence and trends for children and adolescents. The National Health and Nutrition Examination Surveys, 1963 to 1991. Arch Pediatr Adolesc Med 1995; 149:1085–1091.

10 Valencia-Flores M, Orea A, Castano VA, *et al.* Prevalence of sleep apnea and electrocardiographic disturbances in morbidly obese patients. Obes Res 2000; 8:262–269.

11 Punjabi NM, Sorkin JD, Katzel LI, *et al.* Sleep-disordered breathing and insulin resistance in middle-aged and overweight men. Am J Respir Crit Care Med 2002; 165:677–682.

12 Strohl KP, Redline S. Recognition of obstructive sleep apnea. Am J Respir Crit Care Med 1996; 154 (2 Pt 1):279–289.

13 Lumeng JC, Chervin RD. Epidemiology of pediatric obstructive sleep apnea. Proc Am Thorac Soc 2008; 5:242–252.

14 Verhulst SL, Schrauwen N, Haentjens D, *et al.* Sleep-disordered breathing in overweight and obese children and adolescents: prevalence, characteristics and the role of fat distribution. Arch Dis Childhood 2007; 92:205–208.

15 Rosen CL. Clinical features of obstructive sleep apnea hypoventilation syndrome in otherwise healthy children. Pediatr Pulmonol 1999; 27:403–409.

16 Stepanski E, Zayyad A, Nigro C, *et al.* Sleep-disordered breathing in a predominantly African-American pediatric population. J Sleep Res 1999; 8:65–70.

17 • Pillar G, Shehadeh N. Abdominal fat and sleep apnea: the chicken or the egg? Diabetes Care 2008; 31 (Suppl 2):S303–S309.

This review provides an excellent summary of anatomical, neuromuscular and hormonal changes associated with OSA amongst obese adults.

18 • Schwartz AR, Patil SP, Laffan AM, *et al.* Obesity and obstructive sleep apnea: pathogenic mechanisms and therapeutic approaches. Proc Am Thorac Soc 2008; 5:185–192.

This review provides a concise yet well written summary of the risk factors for OSA amongst adults and proposed mechanisms linking obesity with OSA.

19 Crummy F, Piper AJ, Naughton MT. Obesity and the lung: 2. Obesity and sleep-disordered breathing. Thorax 2008; 63:738–746.

20 Rodenstein DO, Dooms G, Thomas Y, *et al.* Pharyngeal shape and dimensions in healthy subjects, snorers, and patients with obstructive sleep apnoea. Thorax 1990; 45:722–727.

21 Malhotra A, White DP. Obstructive sleep apnoea. Lancet 2002; 360:237–245.

22 Brander PE, Mortimore IL, Douglas NJ. Effect of obesity and erect/supine posture on lateral cephalometry: relationship to sleep-disordered breathing. Eur Respir J 1999; 13:398–402.

23 Hoffstein V, Zamel N, Phillipson EA. Lung volume dependence of pharyngeal cross-sectional area in patients with obstructive sleep apnea. Am Rev Respir Dis 1984; 130:175–178.

24 • Martinez-Rivera C, Abad J, Fiz JA, *et al.* Usefulness of truncal obesity indices as predictive factors for obstructive sleep apnea syndrome. Obesity 2008; 16:113–118.

This study amongst a relatively large cohort provides a detailed comparison of the predictive value of various measures of obesity for OSA amongst adults. Results suggest measures of truncal obesity may be useful for service prioritization.

25 Onat A, Hergenc G, Uyarel H, *et al.* Obstructive sleep apnea syndrome is associated with metabolic syndrome rather than insulin resistance. Sleep Breath 2007; 11:23–30.

26 Hora F, Napolis LM, Daltro C, *et al.* Clinical, anthropometric and upper airway anatomic characteristics of obese patients with obstructive sleep apnea syndrome. Respiration 2007; 74:517–524.

27 Cuccia AM, Campisi G, Cannavale R, Colella G. Obesity and craniofacial variables in subjects with obstructive sleep apnea syndrome: comparisons of cephalometric values. Head Face Med 2007; 3:41.

28 Nashi N, Kang S, Barkdull GC, *et al.* Lingual fat at autopsy. Laryngoscope 2007; 117:1467–1473.

29 Tsuiki S, Isono S, Ishikawa T, *et al.* Anatomical balance of the upper airway and obstructive sleep apnea. Anesthesiology 2008; 108:1009–1015.

30 Ancoli-Israel S, Klauber MR, Stepnowsky C, *et al.* Sleep-disordered breathing in African-American elderly. Am J Respir Crit Care Med 1995; 152 (6 Pt 1): 1946–1949.

31 Redline S, Tishler PV, Hans MG, *et al.* Racial differences in sleep-disordered breathing in African-Americans and Caucasians. Am J Respir Crit Care Med 1997; 155:186–192.

32 Young T, Shahar E, Nieto FJ, *et al.* Predictors of sleep-disordered breathing in community-dwelling adults: the Sleep Heart Health Study. Arch Internal Med 2002; 162:893–900.

33 Li KK, Kushida C, Powell NB, *et al.* Obstructive sleep apnea syndrome: a comparison between Far-East Asian and white men. Laryngoscope 2000; 110 (10 Pt 1):1689–1693.

34 Ohdaira F, Nakamura K, Nakayama H, *et al.* Demographic characteristics of 3,659 Japanese patients with obstructive sleep apnea-hypopnea syndrome diagnosed by full polysomnography: associations with apnea-hypopnea index. Sleep Breath 2007; 11:93–101.

35 Kamil MA, Teng CL, Hassan SA. Snoring and breathing pauses during sleep in the Malaysian population. Respirology 2007; 12:375–380.

36 Kapsimalis F, Kryger MH. Gender and obstructive sleep apnea syndrome, part 2: mechanisms. Sleep 2002; 25:499–506.

37 Kapsimalis F, Kryger MH. Gender and obstructive sleep apnea syndrome, part 1: clinical features. Sleep 2002; 25:412–419.

38 • Anttalainen U, Saaresranta T, Kalleinen N, *et al.* Gender differences in age and BMI distributions in partial upper airway obstruction during sleep. Respir Physiol Neurobiol 2007; 159:219–226.
This is the first study of partial obstruction in association with obesity. Results show associations were stronger amongst men but not significant for either sex.

39 Santiago-Recuerda A, Gomez-Terreros FJ, Caballero P, *et al.* Relationship between the upper airway and obstructive sleep apnea-hypopnea syndrome in morbidly obese women. Obes Surg 2007; 17:689–697.

40 Sampol G, Munoz X, Sagales MT, *et al.* Long-term efficacy of dietary weight loss in sleep apnoea/hypopnoea syndrome. Eur Respir J 1998; 12:1156–1159.

41 Collard P, Pieters T, Aubert G. Compliance with nasal CPAP in obstructive sleep apnea patients. Sleep Med Rev 1997; 1:33–44.

42 • Trenell MI, Ward JA, Yee BJ, *et al.* Influence of constant positive airway pressure therapy on lipid storage, muscle metabolism and insulin action in obese patients with severe obstructive sleep apnoea syndrome. Diabetes Obes Metab 2007; 9:679–687.
This study demonstrates that regular CPAP use is required for associated reductions in weight and metabolic disturbance.

43 • Banerjee D, Yee BJ, Piper AJ, *et al.* Obesity hypoventilation syndrome: hypoxemia during continuous positive airway pressure. Chest 2007; 131:1678–1684.
Regular CPAP use may relieve OSA amongst obese adults but may not resolve hypoventilation, reducing the potential therapeutic benefit.

44 Fritscher LG, Mottin CC, Canani S, Chatkin JM. Obesity and obstructive sleep apnea-hypopnea syndrome: the impact of Bariatric surgery. Obes Surg 2007; 17:95–99.

45 Sugerman HJ, Fairman RP, Sood RK, *et al.* Long-term effects of gastric surgery for treating respiratory insufficiency of obesity. Am J Clin Nutr 1992; 55 (2 Suppl):597S–601S.

46 Peluso L, Vanek VW. Efficacy of gastric bypass in the treatment of obesity-related comorbidities. Nutr Clin Pract 2007; 22:22–28.

47 Fritscher LG, Canani S, Mottin CC, *et al.* Bariatric surgery in the treatment of obstructive sleep apnea in morbidly obese patients. Respiration 2007; 74:647–652.

48 • Haines KL, Nelson LG, Gonzalez R, *et al.* Objective evidence that Bariatric surgery improves obesity-related obstructive sleep apnea. Surgery 2007; 141:354–358.
This large study demonstrates that Bariatric surgery not only reduces OSA severity amongst obese adults, but also minimizes CPAP requirement.

49 Varela JE, Hinojosa MW, Nguyen NT. Resolution of obstructive sleep apnea after laparoscopic gastric bypass. Obes Surg 2007; 17:1279–1282.

50 Lettieri CJ, Eliasson AH, Greenburg DL. Persistence of obstructive sleep apnea after surgical weight loss. J Clin Sleep Med 2008; 4:333–338.

51 Peppard PE, Young T. Exercise and sleep-disordered breathing: an association independent of body habitus. Sleep 2004; 27:480–484.

52 • Quan SF, O'Connor GT, Quan JS, *et al.* Association of physical activity with sleep-disordered breathing. Sleep Breath 2007; 11:149–157.
This study demonstrates regular exercise reduces the risk for OSA, particularly amongst those most at risk – obese men.

53 Przybylowski T, Bielicki P, Kumor M, *et al.* Exercise capacity in patients with obstructive sleep apnea syndrome. J Physiol Pharmacol 2007; 58 (Suppl 5 (Pt 2)):563–574.

54 • Smith SS, Doyle G, Pascoe T, *et al.* Intention to exercise in patients with obstructive sleep apnea. J Clin Sleep Med 2007; 3:689–694.
The potential benefits of regular exercise to obese OSA patients are hindered by reduced motivation to exercise. This study highlights the potential need for behavioural, cognitive or both approaches to maximize treatment efficacy.

55 Bidad K, Anari S, Aghamohamadi A, *et al.* Prevalence and correlates of snoring in adolescents. Iran J Allergy Asthma Immunol 2006; 5:127–132.

56 O'Brien LM, Sitha S, Baur LA, Waters KA. Obesity increases the risk for persisting obstructive sleep apnea after treatment in children. Int J Pediatr Otorhinolaryngol 2006; 70:1555–1560.

57 •• Amin R, Anthony L, Somers V, *et al.* Growth velocity predicts recurrence of sleep-disordered breathing 1 year after adenotonsillectomy. AmJ Respir Crit Care Med 2008; 177:654–659.
This is a well conducted study, which identifies gain velocity in BMI, obesity and ethnicity as primary factors influencing treatment efficacy for OSA in children. These findings provide clinicians with important means of assessing postoperative risk and follow-up requirements.

58 Mitchell RB, Kelly J. Outcome of adenotonsillectomy for obstructive sleep apnea in obese and normal-weight children. Otolaryngol Head Neck Surg 2007; 137:43–48.

59 Shine NP, Lannigan FJ, Coates HL, Wilson A. Adenotonsillectomy for obstructive sleep apnea in obese children: effects on respiratory parameters and clinical outcome. Arch Otolaryngol Head Neck Surg 2006; 132:1123–1127.

60 Tauman R, Gulliver TE, Krishna J, *et al.* Persistence of obstructive sleep apnea syndrome in children after adenotonsillectomy. J Pediatr 2006; 149:803–808.

61 Xu Z, Jiaqing A, Yuchuan L, Shen K. A case-control study of obstructive sleep apnea-hypopnea syndrome in obese and nonobese Chinese children. Chest 2008; 133:684–689.

62 Guimaraes CV, Kalra M, Donnelly LF, *et al.* The frequency of lingual tonsil enlargement in obese children. AJR Am J Roentgenol 2008; 190:973–975.

63 Redline S, Tishler PV, Schluchter M, *et al.* Risk factors for sleep-disordered breathing in children. Associations with obesity, race, and respiratory problems. Am J Respir Crit Care Med 1999; 159 (5 Pt 1):1527–1532.

64 • Rudnick EF, Walsh JS, Hampton MC, Mitchell RB. Prevalence and ethnicity of sleep-disordered breathing and obesity in children. Otolaryngol Head Neck Surg 2007; 137:878–882.
This study directly assesses ethnic risk for OSA amongst children, and confirms that African–American children are at increased risk.

65 Wing YK, Hui SH, Pak WM, *et al.* A controlled study of sleep related disordered breathing in obese children. Arch Dis Child 2003; 88:1043–1047.

66 Lam YY, Chan EY, Ng DK, *et al.* The correlation among obesity, apnea-hypopnea index, and tonsil size in children. Chest 2006; 130:1751–1756.

67 de la Eva RC, Baur LA, Donaghue KC, Waters KA. Metabolic correlates with obstructive sleep apnea in obese subjects. J Pediatr 2002; 140:654–659.

68 Kohler M, Lushington K, Couper R, *et al.* Obesity and risk of sleep related upper airway obstruction in Caucasian children. J Clin Sleep Med 2008; 4:129–136.

69 Goodwin JL, Kaemingk KL, Fregosi RF, *et al.* Clinical outcomes associated with sleep-disordered breathing in Caucasian and Hispanic children: the Tucson Children's Assessment of Sleep Apnea study (TuCASA). Sleep 2003; 26:587–591.

70 McKenzie SA, Bhattacharya A, Sureshkumar R, *et al.* Which obese children should have a sleep study? Respir Med (in press).

71 Marcus CL, Fernandes Do Prado LB, Lutz J, *et al.* Developmental changes in upper airway dynamics. J Appl Physiol 2004; 97:98–108.

72 Arens R, Marcus CL. Pathophysiology of upper airway obstruction: a developmental perspective. Sleep 2004; 27:997–1019.

73 Bandla P, Huang J, Karamessinis L, *et al.* Puberty and upper airway dynamics during sleep. Sleep 2008; 31:534–541.

74 • Kaditis AG, Alexopoulos EI, Hatzi F, *et al.* Adiposity in relation to age as predictor of severity of sleep apnea in children with snoring. Sleep Breath 2008; 12:25–31.
This study directly assesses the change in association between obesity and OSA by age amongst children and adolescents. The results suggest that obesity increases the risk for OSA amongst older children and adolescents only.

75 Apostolidou MT, Alexopoulos EI, Chaidas K, *et al.* Obesity and persisting sleep apnea after adenotonsillectomy in Greek children. Chest 2008 [Epub ahead of print].

76 Davis CL, Tkacz J, Gregoski M, *et al.* Aerobic exercise and snoring in overweight children: a randomized controlled trial. Obesity 2006; 14:1985–1991.

77 • Kalra M, Mannaa M, Fitz K, *et al.* Effect of surgical weight loss on sleep architecture in adolescents with severe obesity. Obes Surg 2008; 18:675–679.
Severity of OSA and body mass were significantly improved at around 1 year following surgery amongst obese adolescents. This result, combined with the adult literature, provides evidence that weight-reduction surgery amongst children may also be an effective means of treating OSA in severe cases.

78 Riley DJ, Santiago TV, Edelman NH. Complications of obesity-hypoventilation syndrome in childhood. Am J DisChild 1976; 130:671–674.