Many studies have attempted to identify the possible abnormalities that predispose individuals to OSAS; however, a definitive conclusion has not been possible due to the multifactorial nature of the disease. More specifically, obesity, mandibular growth deficiency, retrognathia, and marked flexion of the cranial base are predisposing factors commonly cited in the literature. An increase in the volume of the tongue and of the lateral pharyngeal walls can also increase the probability of developing the disease. In addition, transverse maxillary deficiency has also been indicated as a predisposing factor.

INCIDENCE/ PUBLIC HEALTH IMPACT: Obstructive sleep apnea (OSA), the most common type of sleep-disordered breathing, poses a significant medical challenge to industrialized societies because it is common (estimate prevalence at least 2% for adult women and 4% for adult men[1,2] ; The National Commission on Sleep Disorders Research estimated that minimal SDB (RDI >5) affects 7-18 million people in the United States and that relatively severe cases (RDI >15) affect 1.8-4 million people. The prevalence increases with age. Sleep Disordered Breathing remains undiagnosed in approximately 92% of affected women and 80% of affected men. Similar data have been found in an epidemiologic study from Pennsylvania.[3, 4] More recent research indicates a prevalence of 4% for women and 9% for men. Data from the Wisconsin Cohort Study indicate that the prevalence of OSA in people aged 30-60 years is 9-24% for men and 4-9% for women.) OSA carries cardiovascular and safety risks, and compromises quality of life.[5] OSA is associated with hypertension,[6-8] diabetes[9] and cardiovascular disease,[10] thereby making it a significant public health concern. Longitudinal observational cohort studies indicate that severe OSA significantly increases the risk of cardiovascular events[10], including stroke and death.[11,12] These associated medical conditions are in part due to the chronic intermittent hypoxia (CIH) and sleep deprivation occurring during sleep-disordered breathing. The primary molecular domains likely affected are sympathetic activity, oxidative stress and inflammation[13].OSA is also associated with a diminished quality of life[14-16] and an increased rate of motor vehicle accidents.[17] Deficits in neurocognitive functioning occur, including diminished vigilance, executive functioning and motor coordination.[18] OSA is considered a chronic disease that requires lifelong management.[19]

THERAPY: Positive airway pressure (PAP) is the accepted first-line therapy for patients with OSA, and is presumed to be highly efficacious, virtually eliminating OSA.[20] PAP works via airway expansion through pressurization of the upper airway. Significant improvements in objective and subjective sleepiness, quality of life, neurocognitive function, and levels of inflammatory biomarkers[21-27] have been demonstrated following the use of PAP.[21] Furthermore, effective treatment of severe OSA by PAP reduces the risk of cardiovascular events.[11] The major limitation to the clinical effectiveness of PAP however, is non-adherence or inability to tolerate PAP therapy. When PAP adherence is defined as 4 hours or greater of nightly use, 29-83% of patients with OSA have been reported to be non-adherent to treatment.[28]

As adherence to PAP therapy is reported to be low, the need for alternative treatments is apparent. Currently, it is unclear what alternative therapy should be used to treat patients with moderate to severe OSA who are inadequately treated by PAP. Surgical procedures which affect airway expansion have been shown to provide benefit in selected patients, but no predictive method for producing a favorable surgical outcome with any given procedure is currently available.[29] Pharmaceutical and neuro-stimulatory approaches have been the subject

of research, but no therapies in these categories are, as yet, generally available. Mandibular repositioning appliances (MRAs), which hold the mandible anteriorly and inferiorly during sleep, have been shown to open the passive pharynx in a dose-dependent fashion.[30] Even though they convey therapeutic efficacy in approximately 50% of unselected patients,[31,32] are preferred by most patients,[31] and are recommended as a CPAP alternative for mild-to-moderate OSA,[33] MRAs are not as commonly used to treat OSA. The reason for this is probably multifactorial, but a lack of reliable selection methods would appear to play an important role. Oral appliance therapy has been shown to be superior to CPAP regarding treatment success in patients with mild to moderate OSAS in the short term. [34] MAS are now widely used for the treatment of OSA both as a primary therapy and as an alternative for patients with severe OSA who are unwilling or unable to tolerate CPAP.5 MAS are a simple, reversible, quiet, and cost effective therapy for selected patients with OSA[33]. Accurate selection of patients for surgical or nonsurgical therapy (based upon the ability of a given therapy to affect a critical degree of airway expansion) may have clinical utility in managing patients with OSA.

While a large variety of surgical procedures for treatment of OSA have been reported, several short term observational studies indicate that (outside of tracheostomy) maxillomandibular advancement surgery (MMA) may the most clinically effective surgical therapy for patients with moderate to severe OSA who are not effectively treated by PAP therapy.[35-40,29] MMA consists of surgical facial advancement by concomitant maxillary and mandibular osteotomies to enlarge the caliber of the posterior upper airway space. Although the level of evidence is low, substantial and consistent short term reductions in AHI have been observed following MMA.[29] Furthermore, the short term reductions in AHI following MMA may be maintained on a long term basis studies[24, 26] Preliminary short term reports indicate that MMA may result in improvement in subjective sleepiness,[41,42] health-related quality of life[43] and blood pressure.[35]

Hypoglossal nerve stimulation has been more recently proposed as a method for affecting airway expansion. This technique- which has been reported amid several prominent publications- involves implantation of a battery-powered sensor and stimulatory electrical leads to the hypoglossal nerve at the base of the tongue. Patients who underwent this procedure were highly selected based upon subjective exam criteria. Risks associated with this technique include the potential for permanent hypoglossal (motor) nerve damage- leading to the potential for dysphagia and swallowing problems, as well as pneumothorax and failure to cure OSA. At the time of generation of this writing, the majority of publications show some improvement in OSA (based upon AHI), although frank cures have been rarely seen. Importantly, however, understand that the overwhelming majority of published scientific articles (which are short-term in nature) regarding this procedure have been funded by commercial interests- hence, commercial bias cannot be excluded.

Literature Cited

1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. Apr 29 1993;328(17):1230-5. [Medline].

2. Centers for Medicare and Medicaid Services. Decision Memo for Continuous Positive Airway Pressure (CPAP) Therapy for Obstructive Sleep Apnea (OSA) (CAG-00093R2). Available from: US Department of Health and Human Services. Accessed March 2010. Available at http://bit.ly/hEQDIk.

3. Schwab RJ, Pasirstein M, Pierson R, Mackley A, Hachadoorian R, Arens R, et al. Identification of upper airway anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. *Am J Respir Crit Care Med*. Sep 1 2003;168(5):522-30. [Medline].

4. Goetting, C , Downey III R. *Sick, symptomatic and undiagnosed*. San Antonio, TX: 2010. Paper to be presented at the Annual Meeting of the Association of Professional Sleep Society.

5. Flemons WW**.** Clinical practice. Obstructive sleep apnea. *N Engl J Me*d 2002;347:498-504.

6. Nieto FJ, Young TB, Lind BK, Shahar E, Sanet JM, Redline S, D'Agostino RB, Newman AB, Lebowitz MD, Pickering TG. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community based study. *JAMA* 2000; 283:1829-1836.
7. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. *New Engl J Med* 2000; 342:1378-1384.
8. Young T, Peppard P, Palta M, Hla KM, Finn L, Morgan B, Skatrud J. Population-based study of sleep disordered breathing as a risk factor for hypertension. *Arch Intern Med* 1997; 157:1745-1752.
9. Punjabi NM, Shahar E, Redline S, Gottileb DJ, Givelber R, Resnick HE. Sleep-Disordered Breathing, Glucose Intolerance, and Insulin Resistance. *Am J Epidemiol* 2004;160:521–530.
10. Shahar E, Whitney CW, Redline S, Lee ET, Newman AB, Nieto FJ, O'Connor GT, Boland LL, Schwartz JE, Samet JM. Sleep-disordered breathing and cardiovascular disease. Cross sectional results of the sleep heart health study. *Amer J Respir Crit Care Med* 2001; 163:19-25.
11. Marin JM, Carrizo SJ, Vicente E, Agusti AGN. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. The Lancet; 365(9464): 1046-1053.
12. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. N Engl J Med 2005; 353: 2034-41.
13. Arnardottir ES; Mackiewicz M; Gislason T; Teff KL; Pack AI. Molecular signatures of obstructive sleep apnea in adults: A review and perspective. SLEEP 2009;32(4):447-470. *(review of biomarkers).*
14. Moyer CA, Sonnad SS, Garetz SL, Helman JL, Chervin RD. Quality of life in obstructive sleep apnea: a systematic review of the literature. *Sleep Med* 2001; 2:477-491.
15. Reimer MA, Flemons WW. Quality of life in sleep disorders. *Sleep Medicine Reviews* 2003; 7:335-349.
16. Moore P, Bardwell WA, Ancoli-Israel S, Dimsdale JE. Association between polysomnographic sleep measures and health related quality of life in obstructive sleep apnea. *J Sleep Res* 2001; 10:303-308.
17. George CFP, Smiley A. Sleep apnea and automobile crashes. *Sleep* 1999; 22:790-795.
18. Beebe DW, Groesz L, Wells C, Nichols A, McGee K. The neuropsychological effects of obstructive sleep apnea: a meta-analysis of norm-referenced and case-controlled data. *Sleep* 2003; 26(3): 298-307.

19. Epstein LJ, Kristo D, Strollo PJ, Friedman N, Malhotra A, Patil SP, Ramar K, Rogers R, Schwab RJ, Weaver EM, Weinstein MD. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea. J Clin Sleep Med 2009; 5(3): 263-276.
20. Giles TL, Lasserson TJ, Smith BH, White J, Wright J, Cates C. Continuous positive airways pressure for obstructive sleep apnoea in adults. *Cochrane Database Syst Rev* 2006; 3:CD001106.
21. Arnardottir ES; Mackiewicz M; Gislason T; Teff KL; Pack AI. Molecular signatures of obstructive sleep apnea in adults: A review and perspective. SLEEP 2009;32(4):447-470. *(review of biomarkers).*
22. Liu H, Liu J, Xiong S, Shen G, Zhang Z, Xu Y. The change of interleukin-6 and tumor necrosis factor in patients with obstructive sleep apnea syndrome. J Tongji Med Univ 2000;20:200-2.
23. Shamsuzzaman AS, Winnicki M, Lanfranchi P, et al. Elevated C-reactive protein in patients with obstructive sleep apnea. Circulation 2002;105:2462-4.
24. Yokoe T, Minoguchi K, Matsuo H, et al. Elevated levels of C-reactive protein and interleukin-6 in patients with obstructive sleep apnea syndrome are decreased by nasal continuous positive airway pressure. Circulation 2003;107:1129-34.
25. Minoguchi K, Tazaki T, Yokoe T, et al. Elevated production of tumor necrosis factor-alpha by monocytes in patients with obstructive sleep apnea syndrome. Chest 2004;126:1473-9.
26. Bravo Mde L, Serpero LD, Barcelo A, Barbe F, Agusti A, Gozal D. Inflammatory proteins in patients with obstructive sleep apnea with and without daytime sleepiness. Sleep Breath 2007;11:177-85.
27. Baessler A, Nadeem R, Harvey M, Madbouly E, Younus A, Sajid H, Naseem J, Asif A, Bawaadam H. Treatment for sleep apnea by continuous positive airway pressure improves levels of inflammatory markers – a meta-analysis. Journal of Inflammation 2013; 10:13.
28. Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy. *Proc Am Thorac Soc* 2008; 5: 173-178.
29. Caples SM; Rowley JA; et al: Surgical Modifications of the Upper Airway for obstructive sleep apnea in adults: a systemic review and meta-analysis. SLEEP 2010; 33(10): 1396-1407.
30. Isono S, Tanaka A, Sho Y, Konno A, Nishino T. Advancement of the mandible improves velopharyngeal airway patency. J Appl Physiol 1995;79:2132-8.
31. Ferguson KA, Cartwright R, Rogers R, et al. Oral Appliances for snoring and obstructive sleep apnea: a review. Sleep 2006;29:244-62.
32. Clark GT, Blumenfeld I, Yoffe N, Peled E, Lavie P. A crossover study comparing the efficacy of continuous positive airway pressure with anterior mandibular positioning devices on patients with obstructive sleep apnea. Chest 1996;109:1477-83.
33. Kushida CA, Morgenthaler TI, Littner MR, et al. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances: An update for 2005. Sleep 2006;29:240-3.
34. Hoekema A, Stegenga B, van der Aa JG, Meinesz AF, van der Hoeven JH, Wijkstra PJ. Nap-titration: an effective alternative for continuous positive airway pressure titration. Resp Med 2006;100:705-13.
35. Prinsell JR. Maxillomandibular advancement surgery in a site specific treatment approach for obstructive sleep apnea in 50 consecutive patients. *Chest* 1999; 116: 1519-1529.
36. Riley RW, Powell NB, Li, KK, Troell RJ, Guilleminault C. Surgery and obstructive sleep apnea: long-term clinical outcomes. *Otolaryngol Head Neck Surg* 2000; 122:415-21.

37. Waite PD, Wooten V, Lachner J, Guyette RF. Maxillomandibular advancement surgery in 23 patients with obstructive sleep apnea syndrome. *J Oral Maxillofac Surg* 1989;47:1256-1261.
38. Conradt R, Hochban W, Brandenburg U, Heitman J, Peter JH. Long term follow up after surgical treatment of obstructive sleep apnea by maxillomandibular advancement. *European Resp J* 1997; 10: 123-128.
39. Bettega G, Pepin J, Veale D, Deschaux C, Raphael B, Levy P. Obstructive sleep apnea syndrome. Fifty-one consecutive patients treated by maxillofacial surgery. *Amer J Respir Crit Care Med* 2000; 162: 641-649.
40. Holty J-E C, Guilleminault C. Maxillomandibular advancement for the treatment of obstructive sleep apnea: A systematic review and meta-analysis. *Sleep Medicine Reviews* 2010; 14: 287–297.
41. Dattilo DJ, Drooger SA. Outcome assessment of patients undergoing maxillofacial procedures for the treatment of sleep apnea: comparison of subjective and objective results. J Oral Maxillofac Surg 2004;62(2):164-8.
42. Goodday R, Bourque S. Subjective outcomes of maxillomandibular advancement surgery for treatment of obstructive sleep apnea syndrome. J Oral Maxillofac Surg 2012;70(2):417-20.
43. Marklund M, Persson M, Franklin KA. Treatment success with a mandibular advancement device is related to supine-dependent sleep apnea. Chest 2000;17:1065-72.
44. Cartwright RD. Predicting response to the tongue retaining device for sleep apnea syndrome. Arch Otolaryngol 1985;111:385-8.
45. Mehta A, Qian J, Petocz P, Darendeliler MA, Cistulli PA. A randomized, controlled study of a mandibular advancement splint for obstructive sleep apnea. Am J Respir Crit Care Med 2001;163:1457-61.
46. Liu Y, Lowe AA, Fleetham JA, Park YC. Cephalometric and physiologic predictors of the efficacy of an adjustable oral appliance for treating obstructive sleep apnea. Am J Orthod Dentofacial Orthop 2001;120:639-47.
47. Otsuka R, Almeida FR, Lowe AA, Ryan F. A comparison of responders and non-responders to oral appliance therapy for the treatment of obstructive sleep apnea. Am J Orthod Dentofacial Orthop 2006;129:222-9.
48. Chan AS, Lee RW, Srinivasan VK, Darendeliler MA, Grunstein RR, Cistulli PA. Nasopharyngoscopic evaluation of oral appliance therapy for obstructive sleep apnoea. Eur Respir J 2010;35:836-42.
49. Tsuiki S, Kobayashi M, Namba K, Oka Y, et al. Optimal positive airway pressure predicts oral appliance response to sleep apnea. Eur Respir J 2010;35:1098-105.
50. Boot H, Poublon RM, Van Wegen R, et al. Uvulopalatopharyngoplasty for the obstructive sleep apnoea syndrome: value of polysomnography, Mueller manoeuvre and cephalometry in predicting surgical outcome. Clin Otolaryngol Allied Sci 1997;22: 504–510.
51. Liu SA, Li HY, Tsai WC, Chang KM. Associated factors to predict outcomes of uvulopharyngopalatoplasty plus genioglossal advancement for obstructive sleep apnea. Laryngoscope 2005;115:2046–2050.
52. Kezirian EJ, White DP, Malhotra A, Ma W, McCulloch CE, Goldberg AN. Inter-rater reliability of drug-induced sleep endoscopy. Arch Otolaryngol Head Neck Surg 2010;136: 393–397.
53. Yoshida K, Fukatsu H, Ando Y, Ishigaki T, Okada T. Evaluation of sleep apnea syndrome with low-field magnetic resonance fluoroscopy. Eur Radiol 1999;9:1197–1202.

54. Horner RL, Mohiaddin RH, Lowell DG, et al. Sites and sizes of fat deposits around the pharynx in obese patients with obstructive sleep apnoea and weight matched controls. Eur Respir J 1989;2: 613–622.
55. 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.
56. Shelton KE, Woodson H, Gay SB, Suratt PM. Adipose tissue deposition in sleep apnea. Sleep 1993;16(Suppl):S103; discussion S103–S105.
57. Schwab RJ, Gupta KB, Gefter WB, Metzger LJ, Hoffman EA, Pack AI. Upper airway and soft tissue anatomy in normal subjects and patients with sleep-disordered breathing. Significance of the lateral pharyngeal walls. Am J Respir Crit Care Med 1995;152(Pt 1): 1673–1689.
58. Schwab RJ. Properties of tissues surrounding the upper airway. Sleep 1996;19 (Suppl): S170–S174.
59. Schwab RJ, Pack AI, Gupta KB, et al. Upper airway and soft tissue structural changes induced by CPAP in normal subjects. Am J Respir Crit Care Med 1996;154(Pt 1):1106–1116.
60. Ikeda K, Ogura M, Oshima T, et al. Quantitative assessment of the pharyngeal airway by dynamic magnetic resonance imaging in obstructive sleep apnea syndrome. Ann Otol Rhinol Laryngol 2001;110:183–189.
61. Moon IJ, Han DH, Kim JW, et al. Sleep magnetic resonance imaging as a new diagnostic method in obstructive sleep apnea syndrome. Laryngoscope 2010;120:2546–2554.
62. Suto Y, Matsuda E, Inoue Y, Suzuki T, Ohta Y. Sleep apnea syndrome: comparison of MR imaging of the oropharynx with physiologic indexes. Radiology 1996;201:393–398.
63. Donnelly LF, Surdulescu V, Chini BA, Casper KA, Poe SA, Amin RS. Upper airway motion depicted at cine MR imaging performed during sleep: comparison between young Patients with and those without obstructive sleep apnea. Radiology 2003;227: 239–245.
64. Shin L, Holbrook A., et al: Improved Sleep MRI at 3 Tesla in Patients With Obstructive Sleep Apnea (Technical Note). Journal of Magnetic Resonance Imaging 2013: 000:000-000: 1-6.
65. Okada T, Fukatsu H, Ishigaki T, Yasuma F, Kayukawa Y. Ultralow- field magnetic resonance imaging in upper airways obstruction in sleep apnea syndrome. Psychiatry Clin Neurosci 1996;50: 285–289.
66. Dort LC, Hadjuk E, Remmers JE. Mandibular advancement and obstructive sleep apnoea: a method for determining effective mandibular protrusion. Eur Respir J 2006;27:1003-9.
67. Tsai WH, Vazquez JC, Oshima T, et al. Remotely controlled mandibular positioner predicts efficacy of oral appliances in sleep apnea. Am J Respir Crit Care Med 2004;170:366-70.
68. Vazquez JC, Tsai WH, Flemons WW, et al. Automated analysis of digital oximetry in the diagnosis of obstructive sleep apnoea. Thorax 2000;55:302-7.
69. Petelle B, Vincent G, Gagnadoux F, et al. One-night mandibular advancement titration for obstructive sleep apnea syndrome. Am J Respir Crit Care Med 2002;165:1150-3.
70. Remmers J; Charkhandeh S; Grosse J; Topor Z; Brant R; Santosham P; Bruehlmann S. Remotely controlled mandibular protrusion during sleep predicts therapeutic success with oral appliances in patients with obstructive sleep apnea. *SLEEP* 2013;36(10):1517-1525.

Epidemiology of OSA

SDB is common in the United States. The National Commission on Sleep Disorders Research estimated that minimal SDB (RDI >5) affects 7-18 million people in the United States and that relatively severe cases (RDI >15) affect 1.8-4 million people. The prevalence increases with age. SDB remains undiagnosed in approximately 92% of affected women and 80% of affected men.

OSA is increasingly prevalent, in both adults and children, in modern society. The estimated prevalence has been 2% for women and 4% for men.[4,5] Similar data have been found in an epidemiologic study from Pennsylvania.[2,6] More recent research indicates a prevalence of 4% for women and 9% for men. Data from the Wisconsin Cohort Study indicate that the prevalence of OSA in people aged 30-60 years is 9-24% for men and 4-9% for women.

The prevalence in children is less certain, but many centers are seeing increasing numbers of adolescent patients, who are often obese and present similarly to many of their adult counterparts, with the important exception that they may be sleepy and/or hyperactive. A 2007 study has suggested that approximately 6% of adolescents have weekly SDB.[7]

International statistics

The prevalence of OSA in non-American populations has only been studied in men and has been found to be as low as 0.3% (England) and as high as 20-25% (Israel and Australia). The prevalence of OSA in Australian men is estimated to be 3%.

Age distribution for OSA

Aging is an important consideration of risk for OSA. OSA prevalence increases 2-3 times in older persons (>65 y) compared with individuals aged 30-64 years,[8,9] with an estimated rate as high as 65% in a community sample of people older than 65 years.[10]

After age 65 years, no further relative disparity is noted in the incidence of OSA. One explanation for this plateau is the relative increase in mortality in persons older than 65 years; however, data to support this contention, as attractive as it appears, are insufficient. Scant data are available to help clinicians determine if clinical management should differ between the age cohorts.

Sex distribution for OSA

The male-to-female ratio in community-based studies is 2-3:1.[4,19] Androgenic patterns of body fat distribution (deposition in the trunk, including the neck area) predispose men to OSA. In general, sex hormones may affect neurologic control of UA-dilating muscles and ventilation.

In population studies that have examined the incidence of OSA, women were not only less likely than men to have OSA but also less likely to be diagnosed early in the disease process. Survival rates are lower for women than for men, after an OSA diagnosis has been established by PSG, presumably due to the delayed OSA diagnosis.

Three large epidemiologic studies have demonstrated that the prevalence of OSA in women appears to increase after menopause.[20-22] In these studies, women on hormone replacement therapy (HRT) had a prevalence similar to that of premenopausal women. Postmenopausal women are 3 times more likely to have moderate-to-severe OSA compared with premenopausal women. Women who are on HRT are half as likely to have OSA compared with postmenopausal women who are not on HRT.[23]

Premenopausal women with OSAHS tend to be more obese than men with the same severity of disease. Thin women with symptoms of OSAHS appear to have an increased frequency of craniofacial abnormalities.

Evidence indicates that women underreport the symptoms of loud snoring and witnessed apneas, leading to underreferral to sleep centers. This may explain the marked male predominance (male-to-female ratio of approximately 8:1) in sleep center–based studies. Additionally, women have lower AHIs than men, even after correcting for other demographic factors such as BMI and neck circumference.[24-26]

Prevalence of OSA by race or ethnicity

African American individuals appear to be more predisposed to SDB than white persons. This increased predisposition varies according to age. The odds ratio is greater than 3 in children younger than 13 years and is 1.88 in persons younger than 25 years. In elderly African Americans, the risk is increased 2-fold. Examination of craniofacial morphology found that brachycephaly is associated with an increased AHI in whites but not in African Americans.[3]

Chinese patients with OSA have a more crowded upper airway and relative retrognathia compared with their white counterparts, with statistical controls for BMI and neck circumference.[27] Asians are known to have a shorter cranial base and a more acute cranial base flexure, increasing OSA risk, with BMI and neck circumference being roughly equal. Therefore, interestingly, obesity plays a more prominent role in OSA predisposition in whites than in Chinese persons. This may serve to underscore the role that craniofacial factors have in Chinese patients.

Other populations that may be at increased risk include Mexican Americans and Pacific Islanders.

References:

1. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. *Chest*. Jul 2007;132(1):325-37. [Medline]. [Full Text].

2. Schwab RJ, Pasirstein M, Pierson R, Mackley A, Hachadoorian R, Arens R, et al. Identification of upper airway anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. *Am J Respir Crit Care Med*. Sep 1 2003;168(5):522-30. [Medline].
3. Cakirer B, Hans MG, Graham G, Aylor J, Tishler PV, Redline S. The relationship between craniofacial morphology and obstructive sleep apnea in whites and in African-Americans. *Am J Respir Crit Care Med*. Mar 2001;163(4):947-50. [Medline].
4. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med*. Apr 29 1993;328(17):1230-5. [Medline].

5. Centers for Medicare and Medicaid Services. Decision Memo for Continuous Positive Airway Pressure (CPAP) Therapy for Obstructive Sleep Apnea (OSA) (CAG-00093R2). Available from: US Department of Health and Human Services. Accessed March 2010. Available at http://bit.ly/hEQDIk.

6. Goetting, C , Downey III R. *Sick, symptomatic and undiagnosed*. San Antonio, TX: 2010. Paper to be presented at the Annual Meeting of the Association of Professional Sleep Society.

7. Johnson EO, Roth T. An epidemiologic study of sleep-disordered breathing symptoms among adolescents. *Sleep*. Sep 1 2006;29(9):1135-42. [Medline].

8. Bixler EO, Vgontzas AN, Ten Have T, Tyson K, Kales A. Effects of age on sleep apnea in men: I. Prevalence and severity. *Am J Respir Crit Care Med*. Jan 1998;157(1):144-8. [Medline].

9. Young T, Shahar E, Nieto FJ, Redline S, Newman AB, Gottlieb DJ, et al. Predictors of sleep-disordered breathing in community-dwelling adults: the Sleep Heart Health Study. *Arch Intern Med*. Apr 22 2002;162(8):893-900. [Medline].

10. Ancoli-Israel S, Kripke DF, Klauber MR, Mason WJ, Fell R, Kaplan O. Sleep-disordered breathing in community-dwelling elderly. *Sleep*. Dec 1991;14(6):486-95. [Medline]. [Full Text].

11. Redline S, Kump K, Tishler PV, Browner I, Ferrette V. Gender differences in sleep disordered breathing in a community-based sample. *Am J Respir Crit Care Med*. Mar 1994;149(3 Pt 1):722-6. [Medline].

12. Hla KM, Young TB, Bidwell T, Palta M, Skatrud JB, Dempsey J. Sleep apnea and hypertension. A population-based study. *Ann Intern Med*. Mar 1 1994;120(5):382-8. [Medline].

13. Shahar E, Redline S, Young T, Boland LL, Baldwin CM, Nieto FJ, et al. Hormone replacement therapy and sleep-disordered breathing. *Am J Respir Crit Care Med*. May 1 2003;167(9):1186-92. [Medline].

14. Young T, Finn L, Austin D, Peterson A. Menopausal status and sleep-disordered breathing in the Wisconsin Sleep Cohort Study. *Am J Respir Crit Care Med*. May 1 2003;167(9):1181-5. [Medline].

15. Young T, Skatrud J, Peppard PE. Risk factors for obstructive sleep apnea in adults. *JAMA*. Apr 28 2004;291(16):2013-6. [Medline].

16. O'Connor C, Thornley KS, Hanly PJ. Gender differences in the polysomnographic features of obstructive sleep apnea. *Am J Respir Crit Care Med*. May 2000;161(5):1465-72. [Medline].

17. Ware JC, McBrayer RH, Scott JA. Influence of sex and age on duration and frequency of sleep apnea events. *Sleep*. Mar 15 2000;23(2):165-70. [Medline].

18. Dancey DR, Hanly PJ, Soong C, Lee B, Shepard J Jr, Hoffstein V. Gender differences in sleep apnea: the role of neck circumference. *Chest*. May 2003;123(5):1544-50. [Medline].

19. Davies RJ, Ali NJ, Stradling JR. Neck circumference and other clinical features in the diagnosis of the obstructive sleep apnoea syndrome. *Thorax*. Feb 1992;47(2):101-5. [Medline]. [Full Text].

20. Hla KM, Young TB, Bidwell T, Palta M, Skatrud JB, Dempsey J. Sleep apnea and hypertension. A population-based study. *Ann Intern Med*. Mar 1 1994;120(5):382-8. [Medline].

21. Shahar E, Redline S, Young T, Boland LL, Baldwin CM, Nieto FJ, et al. Hormone replacement therapy and sleep-disordered breathing. *Am J Respir Crit Care Med*. May 1 2003;167(9):1186-92. [Medline].

22. Young T, Finn L, Austin D, Peterson A. Menopausal status and sleep-disordered breathing in the Wisconsin Sleep Cohort Study. *Am J Respir Crit Care Med*. May 1 2003;167(9):1181-5. [Medline].

23. Young T, Skatrud J, Peppard PE. Risk factors for obstructive sleep apnea in adults. *JAMA*. Apr 28 2004;291(16):2013-6. [Medline].

24. O'Connor C, Thornley KS, Hanly PJ. Gender differences in the polysomnographic features of obstructive sleep apnea. *Am J Respir Crit Care Med*. May 2000;161(5):1465-72. [Medline].

25. Ware JC, McBrayer RH, Scott JA. Influence of sex and age on duration and frequency of sleep apnea events. *Sleep*. Mar 15 2000;23(2):165-70. [Medline].

26. Dancey DR, Hanly PJ, Soong C, Lee B, Shepard J Jr, Hoffstein V. Gender differences in sleep apnea: the role of neck circumference. *Chest*. May 2003;123(5):1544-50. [Medline].

27. Patil SP, Schneider H, Schwartz AR, Smith PL. Adult obstructive sleep apnea: pathophysiology and diagnosis. *Chest*. Jul 2007;132(1):325-37. [Medline]. [Full Text].