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Correlations between Waist and Neck Circumferences and Obstructive Sleep Apnea Characteristics

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Abstract

PURPOSE—The body mass index (BMI), an estimate of body fat, provides a rather imprecise indication of risk for obstructive sleep apnea (OSA). We examined whether other measures, including waist and neck circumference, provide improved indicators of risk in treatment-naïve OSA subjects.

METHODS—We studied 59 OSA subjects [age, 48.8±10.0 years; BMI, 31.9±6.6 kg/m^2; apnea-hypopnea-index (AHI), 38.5±23.0 events/hour; sleep efficiency index (SEI, n=52), 78.6±14.4%; lowest oxygen saturation (SaO_2 nadir), 79.5±8.0%; systolic blood pressure (BP), 127.4±15.7 mmHg; diastolic BP, 80.1±9.1 mmHg; 43 male), and determined waist and neck circumferences (waist, 107.4±15.3 cm; neck, 41.8±4.7 cm), daytime sleepiness [Epworth sleepiness scale (ESS), 8.7±4.6], sleep quality [Pittsburgh sleep quality index (PSQI), 8.5±4.1], depression levels [Beck depression inventory II (BDI-II), 6.6±5.7), and anxiety levels [Beck anxiety inventory (BAI), 6.2±7.2]. We used partial correlation procedures (covariates, age and gender) to examine

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associations between BMI, waist, and neck circumferences vs. AHI, sleep, and neuropsychological variables.

RESULTS—BMI, waist, and neck circumferences were significantly correlated with SaO_2 nadir (BMI; r=−0.423, p=0.001; waist; r=−0.457, p<0.001; neck; r=−0.263, p=0.048), AHI (BMI; r=0.349, p=0.008; waist; r=0.459, p<0.001; neck; r=0.276, p=0.038), and systolic BP (BMI; r=0.354, p=0.007; waist; r=0.321, p=0.015; neck; r=0.388, p=0.003). SEI was significantly correlated with waist circumference (r=0.28, p=0.049), but higher with BMI (r=0.291, p=0.04).

CONCLUSIONS—No other significant waist or neck correlations emerged. This study suggests that waist and neck measures correlate better than BMI with select disease severity (SaO_2 nadir and AHI) in OSA subjects. The findings offer an easily-measured, ancillary means to assess OSA risk.

Keywords

Depression; Anxiety; Body mass index; Apnea hypopnea index; Systolic blood pressure

INTRODUCTION

Obstructive sleep apnea (OSA) is a chronic condition characterized by narrowing or obstruction of the upper airway causing intermittent airflow cessation with repeated diaphragmatic efforts to breathe during sleep, leading to successive, decreased arterial oxygen saturation followed by resaturation on arousal. In the United States, sleeping disorders are common, affecting about 10% of middle aged men and 3% women [1], with 80% of cases of at-least moderate OSA severity still undiagnosed [2]. Undiagnosed and untreated OSA results in severe ancillary medical outcomes, and is a massive economic burden; treating OSA patients after early diagnosis has been reported to save billions of dollars annually [3], and a better understanding and effective treatment of OSA can significantly improve society's health.

The need for improved indicators for OSA risk can be viewed in the context of cardiovascular changes in the condition. OSA triggers a marked surge in sympathetic nervous system activity and increases systolic and diastolic blood pressure (BP). High blood pressure is a leading cause of stroke, and a decrease in blood pressure can reduce cardiovascular complications. A 2-mmHg reduction in diastolic BP can result in a 17% decrease in hypertension prevalence, a 6% reduction in coronary heart disease risk, and a 15% decrease in stroke and transient ischemic attack [4]. Hypertensive OSA patients present with increased short-term blood pressure variability during sleep [5], with higher night-time systolic and 24-h diastolic blood pressure variations [6]. The progression of high blood pressure leads to neuropsychological issues, such as depression and anxiety [7, 8]. Depressive symptoms are often associated with excessive daytime sleepiness and poor quality of sleep [9], and both are prevalent in OSA condition. The major factors causing daytime sleep symptoms are intermittent nocturnal hypoxia, sleep fragmentation, and autonomic dysregulation [10]. In addition, OSA is associated with cognitive dysfunction majorly effecting working memory, short-term memory, and global cognitive functioning. The severity of sleep fragmentation induced by OSA have deleterious impact on attention/

vigilance, delayed long-term visual and verbal memory, visuospatial/constructional abilities, and executive function [11]. The range of neuropsychologic and physiologic deficits mandates easily-assessed indicators of OSA risk. Here, we measured blood pressure, daytime sleepiness and sleep quality, mood regulation, and cognitive functioning, which are commonly found deficient in the condition, and related those deficits to simple physical body measures.

Obesity is a major OSA risk factor, with a 40–45% OSA prevalence worldwide [12–18]. A standard measure for screening obesity is the body-mass-index (BMI), which is correlated with OSA disease severity. However, the measure only partially reflects obesity, as it measures excess body weight, but not body fat. Adipose tissue distribution of predominantly central fat deposition around the neck, trunk, and abdominal viscera is not accounted by BMI, and regional distribution of body fat measured by waist and neck circumference is a better determinant of OSA severity than generalized obesity [19–21].

While OSA is linked to daytime sleepiness, sleep disturbances, deteriorated cognition, and mood deficits, there is a paucity of knowledge as to which anthropological features strongly correlate with OSA symptoms and severity. Thus, our aim was to examine whether BMI or waist and neck measures correlate strongly with disease severity, sleep, cognitive, and neuropsychological scores in newly-diagnosed, treatment-naive OSA subjects. Although many epidemiological studies have shown relationships between OSA and anthropometric indices [22–25], reported the cut-off BMI values, neck circumference, and waist circumference in patient with OSA [26, 27], based on our knowledge, this is the first study exploring associations of BMI, neck, and waist measures with elaborated OSA characteristics, including apnea-hypopnea index (AHI), sleep efficiency index, arousal index, lowest oxygen saturation rate (SaO_2 nadir), baseline SaO_2 levels, ΔSaO_2, heart rate, systolic and diastolic blood pressure, daytime sleepiness, sleep quality, OSA risk levels, depression and anxiety symptoms, and cognitive impairments.

MATERIALS AND METHODS

Subjects

We studied 59 newly-diagnosed, treatment-naive OSA subjects. Demographic, physiologic, neuropsychologic, sleep, and cognitive data are summarized in Table 1. All OSA subjects showed moderate (AHI>15 and <30) to severe (AHI>30) severity through overnight polysomnography (PSG), and were recruited from the Sleep Disorder Laboratory at the University of California Los Angeles (UCLA) Medical Center. Self-reported questionnaires were filled by each subjects with information regarding history of diabetes, thyroid status, and alcohol intake. OSA subjects were not on any medications which impacted cardiovascular or mood regulation, such as β-blockers, α-agonists, angiotensin-converting enzyme inhibitors, vasodilators, or serotonin reuptake inhibitors. OSA subjects with a history of stroke, heart failure, diagnosed brain condition, metallic implants, or body weight more than 160 kg (scanner limitation) were excluded from the study. All subjects provided informed written consent prior to the study, and the protocol was approved by the Institutional Review Board of the UCLA.

Overnight Polysomnography

All OSA subjects underwent overnight sleep studies, consisting of at-least a 7 hour monitoring period of electroencephalogram (central and occipital), electromyogram, electrocardiogram (lead II), right and left extra-ocular eye movement, thoracic and abdominal wall movement, air flow, O_2 saturation, end-tidal CO_2 levels, snore volume, bilateral leg movement, and sleep position. All acquired PSG data were digitized and evaluated by a board-certified sleep physician at the UCLA Medical Center. The ratio of the total number of apnea and hypopneas to the total sleep time in hours were calculated to obtain AHI scores. Subjects with AHI values between 5–14 events/hour, 15–30 events/hour, and >30 events/hour were categorized as mild, moderate, and severe OSA, respectively. SaO_2 nadir and baseline SaO_2 levels were obtained from the sleep study, and ΔSaO_2 values were calculated by subtracting SaO_2 nadir from SaO_2 baseline. The sleep efficiency index (SEI) was measured by obtaining percentage of total time in bed actually spent in sleeping, and the arousal index (AI) was calculated as the total number of arousals scored per hour of sleep.

BMI, Waist, and Neck Circumference Measurement

We measured waist circumference in all OSA subjects by placing a measuring tape at the midpoint between the costal margin and iliac crest in the mid-axillary line, with subjects in the standing position, and the measures were obtained at the end of normal expiration. Neck circumference was also measured in the standing upright position; subjects were asked to look straight ahead, with shoulders down, but not hunched. The measuring tape was placed midway around the neck, between the mid-cervical spine (C3) and mid anterior neck, below the level of thyroid gland, however avoiding the shoulder/neck muscles to obtain precise values. Weight (kg) and height (m) were measured after removing the shoes, and BMI (kg/m^2) was computed by dividing the weight with the square of height.

Heart Rate, Systolic, and Diastolic Blood Pressure

Heart rate (HR), systolic and diastolic blood pressure values were collected in the seated position, with the left arm flexed at the level of heart. These measures were collected using an automated device, with the cuff wrapped one inch above the antecubital fossa on each subject, and data were recorded.

Sleep Quality, Daytime Sleepiness, and OSA Risk Assessment

Two self-administered questionnaires were used to investigate sleep quality and daytime sleepiness in all OSA subjects. Sleep quality was assessed using the Pittsburgh sleep quality index (PSQI) [28], and daytime sleepiness was evaluated with the Epworth sleepiness scale (ESS) [29]. A score >5 and >9 were considered abnormal for PSQI and ESS, respectively. The Berlin questionnaire was administered to evaluate the risk of having OSA, which incorporates questions about snoring (category 1), daytime somnolence (category 2), and BP (category 3). Positive scores in two or more categories were considered to be high risk; otherwise, subjects were scored as low risk [30].

Examination of Mood and Anxiety

Depressive and anxiety symptoms were evaluated using the Beck Depression Inventory (BDI-II) [31] and Beck Anxiety Inventory (BAI) [32], respectively. These questionnaires are self-administered, with 21 questions in each inventory, and scores for each question varying from 0–3, with each total score ranging from 0–63 depending on severity of symptoms. Subjects with values >9 for BDI-II or BAI were considered to have depressive or anxiety symptoms, respectively.

Cognitive Assessment

Trail-making tests (TMT) A and B were used to evaluate cognitive function, including executive function, visual search, mental flexibility, scanning, and speed of processing. TMT A consists of 25 circled numbers spread randomly on a page that must be connected sequentially, and TMT B is similar to TMT A, except the numbers are alternated with letters [33]. The scores are based on total time taken for finishing the task (lower score indicates better performance), where values >31s for TMT A, and >63s, for TMT B are considered as abnormal [34]. The Montreal Cognitive assessment (MoCA) test was also used to examine cognitive domains, including attention and concentration, executive functions, memory, language, visuo-constructional skills, conceptual thinking, calculations, and orientation in OSA subjects. A global MoCA score ≥26 was considered normal [35].

Statistical Analyses

The Statistical Package for the Social Sciences (SPSS, v24.0, Armonk, NY, USA) was used for assessment of demographic, biophysical, physiological, mood, sleep, and cognitive variables. Partial correlation (covariates, age and gender) procedures were used to evaluate the relationships between BMI and waist and neck circumference vs AHI, sleep, mood, cognitive, and physiologic variables. Neck and waist circumferences and BMI values were compared between low- and high-risk OSA groups determined from Berlin scores using analysis of covariance (ANCOVA; covariates, age and gender).

RESULTS

Among 59 OSA subjects enrolled in this study, one male and one female subject was diagnosed with Type-2 diabetes and one female was in pre-diabetic condition. Of 52 OSA subjects, two female and one male subjects reported with history of thyroid disorders. Two male and one female subject reported themselves as heavy drinker/alcoholic, and 19 subjects identified as social drinker with number of drinks varying from 2–3 times per week to per month.

Relationship Between Biophysical Measures and Heart Rate and Blood Pressure

Systolic BP showed significant correlations with waist, neck, and BMI measures, and neck circumference showed the highest correlation coefficient with optimal statistical significance level among all three measures. Diastolic BP and resting heart rate correlations did not reach significant levels with any anatomical measures (Table 2).

Relationship Between Biophysical and Overnight Sleep Measures

All three measures, including BMI, neck, and waist measures, showed significant correlations with AHI, SaO_2 nadir, and ΔSaO_2, but waist circumference showed the strongest correlation coefficient with the optimal statistical significance level. SEI showed significant correlations with waist circumference and BMI, and BMI had higher correlation coefficients at the same statistical significance levels as waist circumference (Table 3).

Associations Between Morphological Measures and Cognitive Scores

Cognitive scores obtained from the MoCA (waist, r = 0.03, p = 0.85; neck, r = −0.13, p = 0.32; BMI, r = 0.02, p = 0.91) and all MoCA subdomains, including visuospatial executive function (waist, r = −0.12, p = 0.38, neck, r = −0.16, p = 0.24, BMI, r = −0.15, p= 0.28), naming (waist, r = 0.30, p = 0.03, neck, r = 0.20, p = 0.14, BMI, r = 0.22, p = 0.10), attention (waist, r = −0.08, p = 0.58, neck, r = −0.26, p = 0.06, BMI, r = −0.01, p = 0.95), language (waist, r = −0.06, p = 0.65, neck, r = −0.23, p = 0.09, BMI, r = 0.05, p = 0.74), abstraction (waist, r = 0.12, p = 0.38, neck, r = −0.07, p = 0.62, BMI, r = 0.04, p = 0.75), delayed recall (waist, r = 0.11, p = 0.41, neck, r = 0.10, p = 0.48, BMI, r = 0.04, p = 0.76), and orientation (waist, r = 0.09, p = 0.51, neck, r = 0.03, p = 0.81, BMI, r = 0.10, p = 0.48), and TMT A (waist, r = 0.15, p = 0.27; neck, r = 0.19, p = 0.15; BMI, r = 0.17, p = 0.21) and TMT B (waist, r = −0.03, p = 0.80; neck, r = 0.08, p = 0.55; BMI, r = −0.13, p = 0.35) did not show statistically significant correlations with any of the morphological measures.

Associations Between Morphological Measures and Mood and Sleep Symptoms

Mood and sleep scores showed non-significant correlations with all three morphological measures (Table 4).

Assessment of Low- and High-Risk OSA with Morphological Measures

Waist and neck circumferences and BMI did not differ significantly in low- and high-risk OSA group, as evaluated using Berlin questionnaire [low-risk (n=5)], high-risk (n=28), mean ± SE, p, waist, 98.4±6.1, 106.1±2.5, 0.25; neck, 40.3±1.5, 41.3±0.6, 0.57; BMI, 28.2±2.3, 31.6±0.9, 0.19).

DISCUSSION

Neck and waist circumferences showed high correlations between multiple OSA neuropsychological and physiological measures, with stronger correlations in some of these measures as compared to BMI. Neck circumference showed the strongest correlation with systolic BP among all three measures, with optimal statistical significance levels. Waist circumference showed the strongest correlations with AHI, SaO_2 nadir, and ΔSaO_2 levels, as compared to neck circumference and BMI. BMI showed stronger correlations only with SEI, as compared to neck and waist circumferences.

Biophysical Measures and Blood Pressure

The OSA condition eventually leads to hypertension, with transient elevations of systolic and diastolic BP with each obstructive event, and sustained elevations continuing during

wakefulness. The chronic nature of hypertension likely derives from over-activity of the sympathetic nervous system, a consequence of damage to neural structures regulating sympathetic tone, likely induced by oxidative stress and inflammation [36]. Our study reveals a positive correlation of systolic BP with neck and waist circumferences and BMI. Neck circumference emerged with the strongest correlation coefficients and optimal significance level among the three measures with systolic BP, suggesting that neck circumference should be preferred over BMI for predicting BP in OSA.

Morphological and Overnight Sleep Measures

AHI, an indicator of sleep apnea severity, is closely associated with body weight [37]. A 10% increase in weight can lead to a 32% increase in AHI, and a 10% decrease in weight can result in a 26% decrease in AHI scores [38]. Neck and waist circumferences are newly-identified clinical features that may be associated with AHI. Several recent studies showed correlations of AHI with BMI, neck, and waist circumference, which is consistent with our study [22, 23, 39, 40]. However, there are mixed reports on better correlates, with few studies showing neck circumference as better correlated with AHI [23, 24, 39, 40], and others showing waist circumference as a better correlate [22, 41], findings which are consistent with our study. In addition, there are a few studies where BMI values are better correlates of AHI than waist and neck circumferences [42, 43]. In the midst of different anthropologic measures, our findings suggest that waist circumference to be better correlated with OSA symptoms and severity among three measures i.e. waist and neck circumference, and BMI.

SEI, which reflects the time spent sleeping in bed, is correlated positively with waist circumference and BMI. SEI has several sleep stage components, including N1, N2, N3, and REM sleep. The positive correlations of SEI with BMI and waist circumference provide an indication towards longer sleep duration than normal, and have implications for obesity, diabetes, hypertension, and cardiovascular diseases [44].

The lowest saturations (SaO_2 nadir) levels accompanying apnea during sleep are correlated with all three measures in our study. Waist circumference correlated strongly among all measures with high significance levels, suggesting that subjects with higher waist circumference achieve lowest oxygen saturation levels, and result in more hypoxia. The delta change in SaO_2 in our study showed significant correlations with all three measures, with waist circumference possessing the strongest correlation coefficient with higher levels of significance among all three measures.

Polysomnograms, considered the gold-standard measure for OSA diagnosis, have their own limitations. Polysomnography is time consuming, labor intensive and expensive, and requires an overnight stay in a sleep laboratory [37]. Finding an anthropologic measurement which shows good correlations with OSA severity, sleep measures, and neuropsychological state would be a useful contribution. Although these measurements cannot replace a polysomnogram, they may be useful in initial screening of OSA.

Further studies may reveal differences between male and female adipose distributions which might affect OSA characteristics. BMI, in combination with upper extremity adipose

distributions were more deterministic of OSA in men; whereas, for women, BMI was the only significant predictor [45]. Distinguishing these attributes can help further studies better understand the processes underlying the greater risk for males in a number of OSA symptoms [45]. Tailoring the diagnostic approach for OSA patients according to gender or perhaps even race may improve accuracy.

CONCLUSIONS

The data suggest that waist and neck circumferences show improved correlations with select disease severity (SaO_2 nadir and AHI) in OSA patients than BMI, and provide impetus to alter the standard measurements used to assess OSA patients. These findings indicate that waist and neck circumferences are strongly associated with disease severity. The morphologic features may provide useful indicators of risk for symptoms of OSA, and help define patients who are most likely to benefit from polysomnography evaluation.

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ABBREVIATIONS

AHI	Apnea-hypopnea index
AI	Arousal index
BAI	Beck anxiety inventory
BDI-II	Beck depression inventory (II)
BMI	Body mass index
BP	Blood pressure
ESS	Epworth sleepiness scale
HR	Heart rate
MoCA	Montreal cognitive assessment
OSA	Obstructive sleep apnea
PSG	Polysomnography
PSQI	Pittsburgh sleep quality index
SaO_2 nadir	Lowest oxygen saturation rate
ΔSaO_2	SaO_2 baseline - SaO_2 nadir
SEI	Sleep efficiency index

TMT	Trail making tests

References

1. Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Increased prevalence of sleep-disordered breathing in adults. Am J Epidemiol. 2013; 177(9):1006–14. [PubMed: 23589584]
2. Lee W, Nagubadi S, Kryger MH, Mokhlesi B. Epidemiology of Obstructive Sleep Apnea: a Population-based Perspective. Expert Rev Respir Med. 2008; 2(3):349–64. [PubMed: 19690624]
3. Knauert M, Naik S, Gillespie MB, Kryger M. Clinical consequences and economic costs of untreated obstructive sleep apnea syndrome. World J Otorhinolaryngol Head Neck Surg. 2015; 1(1): 17–27. [PubMed: 29204536]
4. Cook NR, Cohen J, Hebert PR, Taylor JO, Hennekens CH. Implications of small reductions in diastolic blood pressure for primary prevention. Arch Intern Med. 1995; 155(7):701–9. [PubMed: 7695458]
5. Sasaki N, Ozono R, Edahiro Y, Okita T, Teramen K, Kisaka T, Fujiwara S, Kihara Y. Short-term blood pressure variability in hypertensive patients with obstructive sleep apnea syndrome. Sleep Biol Rhythms. 2014
6. Shi J, Piao J, Liu B, Pan Y, Gong Y, Deng X, Sun W, Lu S, Li Y. Obstructive sleep apnea increases systolic and diastolic blood pressure variability in hypertensive patients. Blood Press Monit. 2017; 22(4):208–12. [PubMed: 28394772]
7. Nasilowska-Barud A, Kowalik M. Characteristics of depressive changes and anxiety in patients with essential hypertension. Ann Univ Mariae Curie Sklodowska Med. 2004; 59(1):428–33. [PubMed: 16146025]
8. Dumitrescu A, Dumitrescu DM, Lepadatu D, Molfea VA, Pandele GI. Clinical characteristics of depressive disorders in hypertensive patients. Rev Med Chir Soc Med Nat Iasi. 2009; 113(2):386–90. [PubMed: 21495342]
9. Regestein Q, Natarajan V, Pavlova M, Kawasaki S, Gleason R, Koff E. Sleep debt and depression in female college students. Psychiatry Res. 2010; 176(1):34–9. [PubMed: 20079935]
10. Slater G, Steier J. Excessive daytime sleepiness in sleep disorders. J Thorac Dis. 2012; 4(6):608–16. [PubMed: 23205286]
11. Bucks RS, Olaithe M, Eastwood P. Neurocognitive function in obstructive sleep apnoea: a meta-review. Respirology. 2013; 18(1):61–70. [PubMed: 22913604]
12. Young T, Peppard PE, Taheri S. Excess weight and sleep-disordered breathing. J Appl Physiol (1985). 2005; 99(4):1592–9. [PubMed: 16160020]
13. Rajala R, Partinen M, Sane T, Pelkonen R, Huikuri K, Seppalainen AM. Obstructive sleep apnoea syndrome in morbidly obese patients. J Intern Med. 1991; 230(2):125–9. [PubMed: 1865163]
14. Richman RM, Elliott LM, Burns CM, Bearpark HM, Steinbeck KS, Caterson ID. The prevalence of obstructive sleep apnoea in an obese female population. Int J Obes Relat Metab Disord. 1994; 18(3):173–7. [PubMed: 8186815]
15. Vgontzas AN, Tan TL, Bixler EO, Martin LF, Shubert D, Kales A. Sleep apnea and sleep disruption in obese patients. Arch Intern Med. 1994; 154(15):1705–11. [PubMed: 8042887]
16. Laaban JP, Cassuto D, Orvoen-Frija E, Iliou MC, Mundler O, Leger D, Oppert JM. Cardiorespiratory consequences of sleep apnoea syndrome in patients with massive obesity. Eur Respir J. 1998; 11(1):20–7. [PubMed: 9543265]
17. Van Boxem TJ, De Groot GH. Prevalence and severity of sleep disordered breathing in a group of morbidly obese patients. Neth J Med. 1999; 54(5):202–6. [PubMed: 10365562]
18. Punjabi NM, Sorkin JD, Katzel LI, Goldberg AP, Schwartz AR, Smith PL. Sleep-disordered breathing and insulin resistance in middle-aged and overweight men. Am J Respir Crit Care Med. 2002; 165(5):677–82. [PubMed: 11874813]
19. Vgontzas AN, Papanicolaou DA, Bixler EO, Hopper K, Lotsikas A, Lin HM, Kales A, Chrousos GP. Sleep apnea and daytime sleepiness and fatigue: relation to visceral obesity, insulin resistance, and hypercytokinemia. J Clin Endocrinol Metab. 2000; 85(3):1151–8. [PubMed: 10720054]

20. Millman RP, Carlisle CC, Mcgarvey ST, Eveloff SE, Levinson PD. Body fat distribution and sleep apnea severity in women. Chest. 1995; 107(2):362–6. [PubMed: 7842762]
21. Dancey DR, Hanly PJ, Soong C, Lee B, Shepard J Jr, Hoffstein V. Gender differences in sleep apnea: the role of neck circumference. Chest. 2003; 123(5):1544–50. [PubMed: 12740272]
22. Kang HH, Kang JY, Ha JH, Lee J, Kim SK, Moon HS, Lee SH. The associations between anthropometric indices and obstructive sleep apnea in a Korean population. PLoS One. 2014; 9(12):e114463. [PubMed: 25474257]
23. Ahbab S, Ataoglu HE, Tuna M, Karasulu L, Cetin F, Temiz LU, Yenigun M. Neck circumference, metabolic syndrome and obstructive sleep apnea syndrome; evaluation of possible linkage. Med Sci Monit. 2013; 19:111–7. [PubMed: 23403781]
24. Onat A, Hergenc G, Yuksel H, Can G, Ayhan E, Kaya Z, Dursunoglu D. Neck circumference as a measure of central obesity: associations with metabolic syndrome and obstructive sleep apnea syndrome beyond waist circumference. Clin Nutr. 2009; 28(1):46–51. [PubMed: 19010573]
25. Ho AW, Moul DE, Krishna J. Neck Circumference-Height Ratio as a Predictor of Sleep Related Breathing Disorder in Children and Adults. J Clin Sleep Med. 2016; 12(3):311–7. [PubMed: 26518700]
26. Davidson TM, Patel MR. Waist circumference and sleep disordered breathing. Laryngoscope. 2008; 118(2):339–47. [PubMed: 18091340]
27. Soylu AC, Levent E, Sariman N, Yurtlu S, Alparslan S, Saygi A. Obstructive sleep apnea syndrome and anthropometric obesity indexes. Sleep Breath. 2012; 16(4):1151–8. [PubMed: 22139137]
28. Carpenter JS, Andrykowski MA. Psychometric evaluation of the Pittsburgh Sleep Quality Index. J Psychosom Res. 1998; 45(1):5–13. [PubMed: 9720850]
29. Johns MW. Reliability and factor analysis of the Epworth Sleepiness Scale. Sleep. 1992; 15(4): 376–81. [PubMed: 1519015]
30. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. Ann Intern Med. 1999; 131(7):485–91. [PubMed: 10507956]
31. Beck AT, Steer RA, Ball R, Ranieri W. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. J Pers Assess. 1996; 67(3):588–97. [PubMed: 8991972]
32. Beck AT, Epstein N, Brown G, Steer RA. An inventory for measuring clinical anxiety: psychometric properties. J Consult Clin Psychol. 1988; 56(6):893–7. [PubMed: 3204199]
33. Moses JA Jr. Test review-Comprehensive Trail Making Test (CTMT). Arch Clin Neuropsychol. 2004; 19(5):703–8. [PubMed: 15330000]
34. Tombaugh TN. Trail Making Test A and B: normative data stratified by age and education. Arch Clin Neuropsychol. 2004; 19(2):203–14. [PubMed: 15010086]
35. Nasreddine ZS, Phillips NA, Bedirian V, Charbonneau S, Whitehead V, Collin I, Cummings JL, Chertkow H. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. J Am Geriatr Soc. 2005; 53(4):695–9. [PubMed: 15817019]
36. Dopp JM, Reichmuth KJ, Morgan BJ. Obstructive sleep apnea and hypertension: mechanisms, evaluation, and management. Curr Hypertens Rep. 2007; 9(6):529–34. [PubMed: 18367017]
37. Punjabi NM. The epidemiology of adult obstructive sleep apnea. Proc Am Thorac Soc. 2008; 5(2): 136–43. [PubMed: 18250205]
38. Peppard PE, Young T, Palta M, Dempsey J, Skatrud J. Longitudinal study of moderate weight change and sleep-disordered breathing. JAMA. 2000; 284(23):3015–21. [PubMed: 11122588]
39. Cizza G, De Jonge L, Piaggi P, Mattingly M, Zhao X, Lucassen E, Rother KI, Sumner AE, Csako G, Study NSE. Neck circumference is a predictor of metabolic syndrome and obstructive sleep apnea in short-sleeping obese men and women. Metab Syndr Relat Disord. 2014; 12(4):231–41. [PubMed: 24571423]
40. de Borges PT, Filho ES, Araujo TM, Neto JM, Borges NE, Neto BM, Campelo V, Paschoal JR, Li LM. Correlation of cephalometric and anthropometric measures with obstructive sleep apnea severity. Int Arch Otorhinolaryngol. 2013; 17(3):321–8. [PubMed: 25992029]
41. Kim JH, Koo YC, Cho HJ, Kang JW. Relationship between various anthropometric measures and apnea-hypopnea index in Korean men. Auris Nasus Larynx. 2017

42. Franco I, Reis R, Ferreira D, Xara S, Ferreira W, Bettencourt N, Antunes A. The impact of neck and abdominal fat accumulation on the pathogenesis of obstructive sleep apnea. Rev Port Pneumol (2006). 2016; 22(4):240–2. [PubMed: 27013321]

43. Wu WT, Tsai SS, Shih TS, Lin MH, Chou TC, Ting H, Wu TN, Liou SH. The Association between Obstructive Sleep Apnea and Metabolic Markers and Lipid Profiles. PLoS One. 2015; 10(6):e0130279. [PubMed: 26115005]

44. Buxton OM, Marcelli E. Short and long sleep are positively associated with obesity, diabetes, hypertension, and cardiovascular disease among adults in the United States. Soc Sci Med. 2010; 71(5):1027–36. [PubMed: 20621406]

45. Wysocki J, Charuta A, Kowalcze K, Ptaszynska-Sarosiek I. Anthropometric and physiologic assessment in sleep apnoea patients regarding body fat distribution. Folia Morphol (Warsz). 2016; 75(3):393–99. [PubMed: 26711651]

Brief Summary

a. **Current Knowledge/Study Rationale:** Body mass index (BMI) was initially considered an indicator of risk for obstructive sleep apnea (OSA); however, recent studies have shown better correlation of neck and waist measures with OSA disease severity than BMI. Among all these, none of the studies explored in-depth relationships of BMI, neck & waist measures with OSA sleep severity, bio-physiologic measures, mood, and cognition status in the condition.

b. **Study Impact:** The findings suggest that waist and neck circumferences are strongly associated with OSA disease severity. The morphologic features may provide useful indicators of risk for OSA symptoms, and may help define patients who are most likely to benefit from polysomnography evaluation.

Table 1

Demographics, biophysical, physiologic, neuropsychologic, sleep, and cognitive variables from OSA subjects.

Variables	OSA subject (n=59) (mean ± SD)
Age (years)	48.8 ± 10.0
Gender (M/F)	43/16
BMI (kg/m^2)	31.9 ± 6.6
Waist Circumference (cm)	107.3 ± 15.3
Neck Circumference (cm)	41.7 ± 4.6
Heart rate (beats/min)	73.6 ± 12.4
Systolic BP (mmHg)	127.4 ± 15.7
Diastolic BP (mmHg)	80.1 ± 9.1
AHI (events/h)	38.5 ± 23
SEI (%)	78.9 ± 14.5 (n=53)
AI (arousal/h)	35.2 ± 20.2 (n=43)
SaO_2 nadir (%)	79.5 ± 8.0
SaO_2 Baseline (%)	94.6 ± 2.0
Δ SaO_2 (%)	15.1 ± 7.4
PSQI	8.5 ± 4.1
ESS	8.7 ± 4.6
BDI-II	6.6 ± 5.7
BAI	6.2 ± 7.2
TMT A (sec)	26.0 ± 9.3
TMT B (sec)	62.5 ± 27.2
MoCA score	
Global	26.0 ± 3.5
Visuospatial	4.1 ± 1.0
Naming	2.9 ± 0.3
Attention	5.3 ± 1.1
Language	2.4 ± 0.9
Abstraction	1.9 ± 0.5
Delayed Recall	3.4 ± 1.5
Orientation	5.9 ± 0.2

OSA, obstructive sleep apnea; SD, standard deviation; BMI, body mass index; BP, blood pressure; AHI, apnea hypopnea index; SEI, sleep efficiency index; AI, arousal index; SaO_2 nadir, lowest oxygen saturation during sleep; SaO_2 baseline, average oxygen saturation during awake; ΔSaO_2, difference of oxygen saturation between SaO_2 nadir and baseline; PSQI, Pittsburgh sleep quality index; ESS, Epworth sleepiness scale; BDI-II, Beck depression inventory II; BAI, Beck anxiety inventory; TMT A, Trail-making test A; TMT B, Trail-making test B; MoCA, Montreal Cognitive Assessment.

Table 2

Correlations between physiological measures and heart rate and blood pressure in OSA subjects.

Variables	HR (r, p)	Systolic BP (r, p)	Diastolic BP (r, p)
Waist	0.07, 0.59	0.32, 0.02	0.15, 0.26
Neck	0.22, 0.11	0.40, 0.002	0.22, 0.11
BMI	0.20, 0.15	0.35, 0.007	0.21, 0.12

HR, heart rate; BP, blood pressure; r, correlation coefficient; p, significant level

Table 3

Correlations between biophysical and overnight PSG data in OSA subjects.

Vars	AHI (r, p)	SEI (n=53) (r, p)	AI (n=43) (r, p)	SaO_2 nadir (r, p)	SaO_2 Baseline (r, p)	Δ SaO_2 (r, p)
Waist	0.45, <0.001	0.29, 0.04	−0.15, 0.35	−0.46, <0.001	−0.24, 0.07	0.43, 0.001
Neck	0.30, 0.03	0.14, 0.32	−0.06, 0.70	−0.28, 0.03	−0.02, 0.86	0.30, 0.02
BMI	0.35, 0.008	0.30, 0.04	−0.04, 0.82	−0.42, 0.001	−0.19, 0.15	0.40, 0.002

Vars, variables; AHI, apnea hypopnea index; SEI, sleep efficiency index; AI, arousal index; SaO_2 nadir, lowest oxygen saturation during sleep; SaO_2 baseline, average oxygen saturation during awake; ΔSaO_2, difference of oxygen saturation between SaO_2 nadir and baseline; r, correlation coefficient; p, significant level.

Table 4

Associations between biophysical measures and mood and sleep symptoms in OSA subjects.

Variables	PSQI (r, p)	ESS (r, p)	BDI-II (r, p)	BAI (r, p)
Waist	0.22, 0.11	0.23, 0.09	−0.08, 0.55	0.23, 0.08
Neck	0.16, 0.23	0.25, 0.07	0.05, 0.70	0.23, 0.09
BMI	0.16, 0.25	0.17, 0.21	−0.09, 0.50	0.12, 0.38

PSQI, Pittsburgh sleep quality index; ESS, Epworth sleepiness scale; BDI-II, Beck Depression Inventory II; BAI, Beck Anxiety Inventory; r, correlation coefficient; p, significant level.