THE EUROPEAN SOCIETY OF MEDICINE
Medical Research Archives, Volume 13 Issue 6

REVIEW ARTICLE

Sleep Goal Index (SGI) – New Outcome Measures, Beyond Apnea Hypopnea Index

Kenny P. Pang FRCSEd, FRCSI, (OTO) [1]; **Ewa Olszewska**, MD, PhD [2]; **Claudio Vicini**, MD [3]; **Charlotte E. Pang** [4]; **Brian Rotenberg**, MD, MPH, FRCSC [5]

[1] Consultant, Otolaryngology, Asia Sleep Centre, Paragon, Singapore
[2] Professor, ENT, Medical University of Bialystok, Poland
[3] Professor, GB Morgagni-L Pierantoni Hospital, University of Ferrara and Bologna in Forli, Italy
[4] Student, Nanyang Girls High, Singapore
[5] Consultant, Otolaryngology, Western University, London, Ontario, Canada

OPEN ACCESS

PUBLISHED
30 June 2025

CITATION
Pang, KP., Olszewska, E., et al., 2025. Sleep Goal Index (SGI) – New Outcome Measures, Beyond Apnea Hypopnea Index. Medical Research Archives, [online] 13(6). https://doi.org/10.18103/mra.v13i6.6601

COPYRIGHT
© 2025 European Society of Medicine. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI
https://doi.org/10.18103/mra.v13i6.6601

ISSN
2375-1924

ABSTRACT

Snoring affects not only the bed-partner's sleep quality, but may also cause the break down in the marriage and relationship. Sleepiness, poor concentration and irritability affects quality of life, work and productivity. Successful treatment of an OSA patient would imply reversing debilitating symptoms faced by the patient, including the metabolic and oxidative stress that accompanies the disease load/burden. There is widespread evidence showing significant discordance between AHI used to denote outcomes of therapy and real-world clinical outcomes such as QOL, patient perception of disease, cardiovascular measures, disease burden and/or survival. It is widely accepted that AHI can vary from night to night, from laboratory to laboratory, from various nasal thermistor to pressure transducers. Different definitions of hypopnea used in different laboratories and software affect AHI values. The reliance on AHI as the only outcome measure assessed in clinical research is not in line with many other aspects of medicine that are becoming patient-centered as opposed to test-centered. Outcome measures of OSA should be based on end-organs effects rather than only one highly variable parameter, AHI. Too much weightage has been given to this single parameter (AHI) that is well known for its variability. Patients are concerned and affected by "real tangible" issues like loud snoring, daytime sleepiness, uncontrolled hypertension, obesity, high glucose levels; these are the effects of OSA as a systemic disease affecting end-organs, manifesting as these patient related symptoms or complaints. An example of more comprehensive outcome parameters would include end-organ effects like the Blood Pressure, Gross Weight (BMI), Oxygen Time Spent below 90% (T90), and AHI; these were collectively introduced as the Sleep Goal Index. This review article will highlight the short-comings of the AHI and illustrate holistic outcome measures that better reflect the oxidative stress that affect the OSA patient.

Keywords: Sleep Apnea, Sleep Goal Index, Outcome Measures, AHI

Introduction

Obstructive sleep apnoea (OSA) is a common sleep disorder that is due to recurrent upper airway collapse and obstruction due to a narrow upper airway and resulting in hypoxic events during sleep that affects the patient's neuro-cardiovascular system, resulting in strokes, heart attacks, arrythmias, and even sudden cardiac death. Some studies estimate that 93% of females and 82% of males with moderate to severe OSA remain undiagnosed[1,2] Scientific studies have shown strong undeniable association between OSA and hypertension[3], cardiovascular diseases[4], and congestive heart failure[5]. Many sleep specialists and cardiologist believe in the causative relationship between OSA and cardiovascular risk[3,4,5].

Traditionally, OSA severity is defined by the number of stoppages in breathing per hour during the night; these stoppages are quantified as apneas and hypopneas. The number of apnoea and hypopnea events per hour (AHI) would indicate the severity of OSA, noting that less than 5 is normal, AHI 5-15 is mild, AHI 15-30 is moderate and more than 30 is classified as severe OSA. Moreover, the effectiveness of any treatment for OSA has been based on a specific arbitrarily proposed 50% reduction of the AHI after treatment (compared to pre-treatment AHI) and an AHI below 20 (known as the Sher's success criteria)[6].

Quantifying the number of stoppages in breathing might help to differentiate the OSA severity however, it still does not add any better understanding of its effects on the damage to the end-organs. Intuitively, it would make more clinical sense to measure the apneic effects on the end-organs and perhaps utilize these as outcome measures as effectiveness of any OSA therapy. Pang et al had introduced a comprehensive holistic outcome measure known as the Sleep Goal, that has gained great interest and much acceptance[7].

We review the medical literature on the shortcomings of the AHI and illustrate the need for more holistic outcome measures while illustrating the reasons for selecting these specific end-organ parameters.

Methodology

We performed a systematic literature search using Medline, Google Scholar, Cochrane Library and Evidence Based Reviews databases for relevant published studies that included OSA and blood pressure, OSA and its CPAP effects, OSA and obesity, OSA and BMI, upper airway surgery and blood pressure, oxygen duration below 90% and OSA, AHI and its reliability, AHI definitions and OSA severity, sleep apnea outcome measures, Sleep Goal Criteria and sleep apnea results. All included studies had to be published in English. All articles were evaluated for relevance, thoroughly studied and made reference to. This review article will first illustrate the inconsistencies and the inaccuracies of the AHI, describe the associations of OSA with its respective end-organ effects/damage; and finally introduce and explain the benefits of the more holistic outcome measure Sleep Goal Index.

Discussion

Most sleep specialists agree that there has been an over-reliance on this single AHI parameter. Moreover, there is consensus that there is a huge discordance between AHI used to denote outcomes/success of therapy and real-world clinical outcomes like, quality of life (QoL), patient perception of disease, daytime tiredness, snoring, cardiovascular measures (e.g., blood pressure, oxygen saturation), and/or survival. There are some inherent issues with the AHI that needs to be discussed.

THE LIABLE OR RELIABLE AHI

1. The overnight in-hospital polysomnogram has its limitations. For example, it is resource intensive, it requires the need of recording beds which is high cost with long waiting lists and intense labour requirements. Moreover, sleep specialists recognize the likelihood of a low sleep efficiency due to the first night-effect, coupled with the patient anxiety and the restriction of movements from the abundance of monitoring wires.
2. Night-to-night variability would also affect the AHI either pre or post-treatment. Chediak et al[8] reported that 32% of their patients had a difference of AHI ≥ 10 in two consecutive nights of PSG. Le Bon et al[9] also studied 2 consecutive nights of polysomnogram (PSG) in 243 OSA patients and showed that 2 nights had better sensitivity and specificity for actual AHI determination (however, may not be economically sound). Levendowski et al[10] reported a fairly weak correlation ($r = 0.44$) between overall AHI from the two PSG studies conducted approximately 40 days. However, Stepnowsky et al[11] did show in 1091 patients that the night-to-night Pearson correlation coefficients ranged between 0.88 and 0.90 for each pair of nights.
3. Another strong confounder would be the use of home-based sleep studies versus the in-laboratory sleep tests (level 1 polysomnogram), many articles have shown that the in-lab PSG scores higher AHI values than the home-based sleep studies[12,13]. Additionally, some authors report pre- and post-treatment sleep tests on OSA patients who had sleep tests performed at different sleep laboratories or using different types of home-based monitors; these are potential confounders that would skew the actual treatment results.
4. The fourth and major confounder, and perhaps the most common and widely debated factor, is the use

© 2025 European Society of Medicine

of different hypopnea definitions in different laboratory systems, and in addition, the use of different monitoring equipment (for example, the use of nasal thermistors versus the use of nasal airflow pressure sensors, different oximeters might affect the results too)[14]. Some studies have reported that the depth of desaturation varies by equipment manufacturer, and the criteria based on a 4% desaturation may not be the same at different sleep laboratories[14]. The definition of an apnea is fixed, it refers to a pause in respiration for more than 10 seconds and is seen in both central sleep apnea (CSA) and obstructive sleep apnea (OSA). Hypopnea is usually defined as reduction in ventilation of at least 50% that results in a decrease in arterial saturation of 4% or more. Some sleep centers define hypopnea as clinically significant when there is a 30% (or greater) reduction in nasal airflow lasting for 10 seconds or longer with an associated 4% (or greater) oxygen desaturation and/or if this results in an arousal or a fragmentation of sleep[15]. Many studies had also demonstrated that AHI can differ up to 30% if the definition of oxygen desaturation within the hypopnea definition, differed by only 1% (i.e., a desaturation of 3% or greater versus a desaturation of 4% or greater)[15].

Therefore, it would be prudent to note that there is huge test-retest variability from the different available sleep tests in different medical centers, and that this is a major confounding factor in assessing treatment outcomes, purely by using AHI alone. Guidelines developed to standardize the scoring of sleep and detection of apneic-related events (i.e., accreditation by the American Academy of Sleep Medicine) appears ineffective in controlling the inherent variability of OSA when measured by PSG alone. The suggestion of multi-night PSG studies is both difficult for patients and very expensive. Sleep specialists using solely the AHI from the PSG for assessing treatment outcomes should factor in the increased variability, with perhaps high level of inaccuracy and discordance with the actual patient benefit as a whole.

From the patient's perspective, this parameter AHI is not intuitively informative to the patient; no patient would seek a consultation with a sleep specialist complaining that "my AHI is high". Patients are more concerned and affected by "real tangible" issues like loud snoring, daytime sleepiness, mood changes, irritability, memory loss, brain fog, uncontrolled hypertension, obesity and high glucose levels; these are the effects of OSA as a systemic disease affecting end-organs, manifesting as real patient related symptoms or complaints. Hence, it would make better clinical-sense to assess and utilize these end-organ effects of OSA in order to assess outcomes of intervention.

END-ORGAN EFFECTS AS OUTCOME MEASURES

It is important to highlight the strong association/link between OSA with blood pressure, gross weight/BMI, quality of life, hypoxemia, neuro-cognitive and the cardio-vascular effects supporting the utilization of parameters that reflect true effects of the apneas and hypopneas that the OSA patients experience.

OSA AND BLOOD PRESSURE (BP) - BLOOD PRESSURE REDUCES WITH EFFECTIVE TREATMENT

There are blood pressure (BP) surges in OSA patients at night that can be observed in both the systemic and pulmonary circulation[16] (unlike normal people where there is a nocturnal 10-20% dip); there are also documented cyclical variations of the heart rate (i.e., sinus tachycardia/bradycardia[17]), extreme cases of arrhythmias have also been documented that might lead to sudden cardiac death[18]. Hypoxic effects of OSA on the cardiovascular (CV) system manifest as daytime arterial hypertension, and documented increased risk of stroke, heart failure, and myocardial infarction[19]. Studies have shown that OSA patients have a higher incidence of hypertension, as high as 1.5 to 2.7 times[20,21,22], and treatment of these OSA patients with CPAP have consistently and reliably showed a decrease in blood pressure[23,24]. A randomized controlled trial (RCT) conducted by Weaver *et al* [25] showed that in 239 patients randomized to CPAP and placebo, after eight weeks of CPAP treatment; CPAP significantly improved the FOSQ scores of patients with OSA and there was a significant reduction in diastolic BP values from baseline by -1.93 mmHg (95% CI, −3.8 to 0.0; P=0.048) between the two groups.

In the Multicenter OSA Interventional Cardiovascular (MOSAIC) trial, 391 patients with OSA were randomized to 6 months of auto-adjusting CPAP therapy versus standard care. Patients on CPAP treatment significantly improved subjective daytime sleepiness (adjusted treatment effect on ESS −2.0; 95% CI, −2.6 to −1.4; P<0.0001). However, this positive treatment effect on symptoms was not accompanied by a reduction in blood pressure[26]. The findings of the MOSAIC study were confirmed by a meta-analysis published by Bratton *et al* [27] in which the individual data of 1,206 patients from four RCTs were evaluated. Although CPAP treatment reduced OSA severity and sleepiness, overall, it did not to have a beneficial effect on BP, except in those patients who used CPAP for >4 h/night; suggesting that a minimum of 4 hours use per night is needed[27].

Lozano *et al* [28] had 64 patients randomized to receive CPAP versus conventional medical treatment alone. After a 3-month CPAP usage period, patients who used CPAP >5.8 hours showed a greater reduction than patients treated with standard medication in daytime diastolic BP −6.12 mmHg (95% CI, −1.45 to −10.82; P=0.004), 24-

h diastolic BP –6.98 mmHg (95% CI, −1.86 to −12.1; P=0.009) and 24-h systolic BP −9.71 mmHg (95% CI, −0.20 to −19.22; P=0.046) [28, 29]. These scientific studies illustrate that OSA affects blood pressure and effective therapy for OSA would result in a decrease in the blood pressure of the patient.

OSA AND BMI / GROSS WEIGHT

It is well accepted that obese patients have a higher incidence of OSA, however, patients with OSA may not be obese[31]. The anatomy of the upper airway is essentially a balance between the contents (soft tissues) and its container/box (skeletal framework). Some OSA patients have a "local" problem (i.e., a localized problem in their upper airway, like huge tonsils, thick redundant palate, long uvula, huge lingual tonsils) versus some patients who have a "global" problem (i.e., generalized obesity). There have been papers illustrating the use of clinical prediction models as prediction of OSA and its severity, these all include the BMI[32,33]. Many studies demonstrate that a BMI greater than 40 is also a predictor of poorer surgical outcomes, and that obesity is significantly related to and associated with fat deposition in the posterior tongue, cervical fat area and parapharyngeal area[34]. Ahn et al had also showed a strong correlation between tongue volume (on MRI) and BMI[35]; Kim et al showed that the tongue fat percentage was higher in OSA patients compared to normal (matched BMI) subjects (42% versus 24%) (36). Parapharyngeal fat have also been shown to be enlarged in apneics and to contribute to airway narrowing[37]. Therefore, it is important for the sleep specialist to appreciate that a reduction in BMI would not only reduce the overall oxidative metabolic stress but also, inadvertently also increase the upper airway space in totality.

OSA AND EPWORTH SLEEPINESS SCALE

The Epworth Sleepiness Scale (ESS) was first described in 1991, from the Epworth Hospital in Melbourne, Australia; it was used not only for OSA, but other sleep disorders as well[38]. It is essentially a self-reported questionnaire that evaluates the tendency to fall asleep in eight various daily situations. The ESS score ranges from 0 to 24, and a score equal to or greater than 10 indicates excessive daytime sleepiness[38]. The ESS has been extensively utilized, investigated and validated by numerous articles; it suffices to highlight that the ESS is sensitive for sleepiness but not specific for any particular sleep disorder.

OSA AND QUALITY OF LIFE

Quality of life (QoL) is considered one of the most fundamental patient-reported outcomes in healthcare. It is practical and reflective of the patient's true subjective reported symptoms. The improvement in QoL should be used to determine whether any intervention is useful, beneficial or should be standard of care. For most OSA patients, a reduction in their QoL is often reflected by symptoms such as excessive daytime sleepiness, fatigue, poor sleep quality, irritability, poor concentration, low work productivity, reduced libido, loss of interest and loud snoring that affects their bed partner. Hence, any treatment deemed successful should result in an improvement in the QoL for these patients.

In patients with OSA, the domains of physical functioning, general health, and vitality appear to be the most severely impacted[39]. Studies have showed that the impaired quality of life ranges from 41% to 88% depending on QoL domain assessed and the assessment tool[40,41]. There are various generic quality of life instruments such as the Medical Outcomes Study, SF-36 or SF-12, and /or the Pittsburgh Sleep Quality Index (PSQI)[42]. Some validated OSA-specific instruments may be more sensitive and appropriate for patients with OSA [43]. Instruments such as the Calgary Sleep Apnea Quality of Life Inventory (SAQLI) or Functional Outcomes of Sleep Questionnaire (FOSQ) have also been shown to be useful. There have been a number of studies investigating the impact of treatment of OSA on QoL[43]. In some randomized controlled trials, QoL improves after adequate treatment of OSA with continuous positive airway pressure[44, 45], oral appliances[46], and upper airway surgery[47]. Other acceptable generic validated QoL instruments include, but are not limited to, the following: Medical Outcomes Study SF-36[48], Medical Outcomes Study SF-12 [49], Nottingham Health Profile [50], EuroQoL [51], EQ-5D [52], FOSQ [53], SAQLI [54].

SLEEP-GOAL AS A HOLISTIC SUCCESS CRITERIA

To the patient, the AHI is a nebulous concept, while other clinical outcomes measures are more relevant, including subjective sleepiness, irritability, poor focus, brain fog, forgetfulness, snoring level and level of performance (all of these are felt by the patient). The main objective in OSA treatment is to prevent long-term deleterious effects of the disease (e.g., high blood pressure, metabolic oxidative stress, strokes and other cardiovascular morbidity), yet paradoxically, such parameters/ assessments are notably not utilized and relatively invisible to both medical and surgical studies evaluating treatment outcomes. Moreover, when AHI is utilized, sleep specialists may have trivialized its short-comings and accepted that the archaic criteria of 50% reduction in AHI and an AHI less than 20, is gospel truth. This criterion, based on historical literature was arbitrarily developed, and did not study/research or stratify any patients nor did they have any clinical data, parameters or assessments.

Let us consider a very common example, patient A with a pre-operative AHI of 95 whom, after surgery, has a post-operative PSG result of AHI 21; this patient would likely

© 2025 European Society of Medicine

experience significant measurable symptomatic and clinical improvement with a huge decrease in disease burden (in terms of obesity, hypertension, cardiovascular effects and oxidative stress) even though this is not defined as a successful numerical surgical outcome by the old Sher AHI criteria; consider another patient B with baseline pre-operative AHI of 35, and who had reduced the AHI post-operatively to under 14, this is considered a successful numerical AHI outcome even though the likelihood of clinical cardiovascular or QoL impact may be minimal, compared to patient A. Intuitively, one would agree that patient A benefitted significantly more than patient B, but yet patient A was labeled as a "surgical failure". Moreover, not forgetting, that in much of the reported literature, the pre- and post-surgery sleep test may have been done in different sleep laboratories, using different definitions of hypopnea and different sleep diagnostic devices, all of which can confound the result. Hence, utilizing AHI as just one single variable with which to gauge success of therapy, either medical or surgical, is flawed and allows it to hold too much weight in the field of sleep medicine.

Pang et al (2020) had proposed the SLEEP-GOAL outcome parameters previously with good acceptance amongst sleep specialists [7]. The SLEEP-GOAL success criteria covers more holistic, comprehensive and inclusive parameters that reflect patient complaints compared to AHI alone. Four years on, Pang et al (2025) had done further validation on 618 OSA patients, and presented a more compendious version of the SLEEP-GOAL outcome parameters known as the Sleep-Goal Index (SGI) comprising of the four main major parameters: Blood Pressure, Gross Weight (BMI), Oxygen Time Spent below 90% (T90), and AHI [55].

SLEEP-GOAL OUTCOME MEASURES

Sleep-Goal Index

The SLEEP-GOAL[7] outcome measures published in 2020, relates closely with the end-organ effects/parameters of the OSA patient. It reflects the cardiovascular and neuro-cognitive effects of the OSA disease process, oxidative stress and the OSA disease load. Based on the medical evidence on these parameters, Pang et al[7] had assigned SLEE as minor criteria and PGOAL as the major criteria. Successful improvement post treatment is denoted by: -

S = **Snoring** – VAS reduction by 50%

L =sleep **Latency** – increase by 50% time latency

E = **ESS** – a reduction of 50% and < 10

E = **Execution** time – an improvement by 50%

P = blood **Pressure** – reduction of either SBP or DBP by 7mmHg or both by 5mmHg

G = **Gross** weight / BMI – reduction of GW by 8% or drop in BMI by 2 points

O = **Oxygenation** (time spent < 90%) – improvement by 50%

A = **AHI** – reduction by 50%

L = **Life quality** (QOL) score – improvement by 50%

Sleep Goal Index (SGI)

The Sleep-Goal Index (SGI) [55] is the more condensed and concise use of the main major criteria of the SLEEP-GOAL parameters, it consists of blood pressure, gross weight/BMI, time spent oxygen saturation <90%, and AHI.

P = blood **Pressure** – reduction of either SBP or DBP by 7mmHg or both by 5mmHg (A)

G = **Gross** weight / BMI – reduction of GW by 8% or drop in BMI by 2 points (B)

O = **Oxygenation** (time spent < 90%) – improvement by 50% (C)

A = **AHI** – reduction by 50% (D)

The authors had compared SGI with Sher's criteria using the McNemar's test. Based on these 4 parameters, it was noted that fulfilling any 2 out of 4 SGI parameters would be just as sensitive as the Sher's criteria, with additionally being more holistic and representative of the patients' end-organ effects and oxidative stress. The authors showed that the overall success rates of the four SGI pairs that fulfilled SGI criteria as "success" were as follows – A and B – 41.8%, A and C – 56.2%, A and D – 55.8%, B and C – 54.5%, B and D – 56.7%, C and D – 60.9% (see labelled A, B, C, D above) (55). The authors noted that all the two combinations were very similar and close to Sher's criteria of a success rate of 55.7%, indicating that any of these 2 out of the 4 SGI parameters combined, would be as sensitive as and as stringent as Sher's criteria [55].

We are comparing SGI to the old Sher's criteria, demonstrating that because SGI has 4 parameters instead of only one very unreliable parameter AHI (as discussed above), SGI would intuitively be more holistic and superior to the AHI as a stand-alone parameter.

Conclusion

The SGI parameters are easy to measure, consistent and reproducible. The SGI is realistic and holistic for OSA patients undergoing treatment for OSA. The patient's treatment outcome could already be measurable by 2 out of the 4, SGI criteria. We would like to propose the use of Sleep Goal Index as a treatment outcome measure as it is holistic, comprehensive, easily measured and better patient-appreciated as a treatment outcome measure.

Disclosure: This review article was not funded. There are no financial disclosures and no conflict of interest for all the above authors

Conflict of Interest: All the authors declare that he/she has no conflict of interest.

© 2025 European Society of Medicine

References

1. Young T, Evans L, Finn L, Palta M. Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle aged men and women. Sleep 1997; 20(9): 705-6.
2. Nieto JF, Young TB, Lind BK, Shahar E, Samet JM, Redline S, et al. Association of SDB, sleep apnea and hypertension in a large community-based study. JAMA 2000; 283(14): 1829-36.
3. Shakar E, Whitney CW, Redline S, Lee ET, Newman AB, Nieto FJ, et al. Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study. Am J Respir Critical Care Med 2001; 163(1): 19-25.
4. Bradley TD. Right and Left ventricular functional impairment and sleep apnea. Clin Chest Med. 1992; 13(3): 459-79.
5. Sher AE, Schechtman KB, Piccirillo JF. The efficacy of surgical modifications of the upper airway in adults with obstructive sleep apnea syndrome. Sleep 1996; 19(2): 156-77.
6. Baldwin CM, Griffith KA, Nieto FJ, O'Connor GT, Walsleben JA, Redline S. The association of sleep-disordered breathing and sleep symptoms with quality of life in the Sleep Heart Health Study. Sleep 2001; 24(1): 96-105
7. Pang KP, Baptista PM, Olszewska E, Braverman I, Carrasco-Llatas M, Kishore S, Chandra S, Yang HC, Chan YH, Pang KA, Pang EB, Rotenberg B. SLEEP-GOAL: A multicenter success criteria outcome study on 302 obstructive sleep apnoea (OSA) patients. Med J Malaysia. 2020 Mar;75(2):117-123.
8. Chediak AD, Acevedo-Crespo JC, Seiden DJ, Kim HH, Kiel MH. Nightly variability in the indices of sleep-disordered breathing in men being evaluated for impotence with consecutive night polysomnograms. Sleep. 1996;19:589–592
9. Le Bon O, Hoffmann G, Tecco J, Staner L, Noseda A, Pelc I, Linkowski P. Mild to moderate sleep respiratory events: one negative night may not be enough. Chest. 2000; 118:353–359.
10. Levendowski D, Zack N, Rao SM. Assessment of the test-retest reliability of laboratory polysomnography. Sleep Breath. 2008.
11. Stepnowsky CJ Jr[1], Orr WC, Davidson TM. Nightly variability of sleep-disordered breathing measured over 3 nights. Otolaryngol Head Neck Surg. 2004 Dec;131(6):837-43.
12. Pordzik J, Seifen C, Ludwig K, Ruckes C, Huppertz T, Matthias C, Gouveris H. Waiting for in-Lab Polysomnography May Unnecessarily Prolong Treatment Start in Patients with Moderate or Severe OSA at Home Sleep Apnea Testing. Nat Sci Sleep. 2024 Dec 5;16:1881-1889
13. Teixeira RCP, Cahali MB. In-Laboratory Polysomnography Worsens Obstructive Sleep Apnea by Changing Body Position Compared to Home Testing. Sensors (Basel). 2024 Apr 27;24(9):2803
14. Zafar S, Ayappa I, Norman R. Choice of oximeter affects apnea-hyponea index sleep staging reliability. Sleep. 2004;27:1171–1179.
15. Pevernagie DA, Gnidovec-Strazisar B, Grote L, Heinzer R, McNicholas WT, Penzel T, Randerath W, Schiza S, Verbraecken J, Arnardottir ES. On the rise and fall of the apnea-hypopnea index: A historical review and critical appraisal. J Sleep Res. 2020 Aug;29(4):e13066
16. Guilleminault C., Connolly S., Winkle R. Cyclical variation of the heart rate in sleep apnoea syndrome. Mechanisms, and usefulness of 24 h electrocardiography as a screening technique. *The Lancet*. 1984;1(8369):126–131
17. Rossi V. A., Stradling J. R., Kohler M. Effects of obstructive sleep apnoea on heart rhythm. *European Respiratory Journal*. 2013;41(6):1439–1451
18. Gami A. S., Olson E. J., Shen W. K., et al. Obstructive sleep apnea and the risk of sudden cardiac death: a longitudinal study of 10,701 adults. *Journal of the American College of Cardiology*. 2013;62(7):610–616.
19. Marin J. M., Carrizo S. J., Vicente E., Agusti A. G. N. 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*. 2005;365(9464): 1046–1053
20. Calhoun DA, Jones D, Textor S, Goff DC, Murphy TP, Toto RD, et al. Resistant hypertension: diagnosis, evaluation, and treatment: a scientific statement from the American Heart Association Professional Education Committee of the Council for High Blood Pressure Research. Circulation. 2008;117:e510–e526
21. Muiesan ML, Salvetti M, Rizzoni D, Paini A, Agabiti-Rosei C, Aggiusti C, et al. Resistant hypertension and target organ damage. Hypertens Res. 2013;36:485–491
22. Pedrosa RP, Drager LF, Gonzaga CC, Sousa MG, de Paula LK, Amaro AC, et al. Obstructive sleep apnea: the most common secondary cause of hypertension associated with resistant hypertension. Hypertension. 2011;58:811–817.
23. Milleron O, Pillière R, Foucher A, et al. Benefits of obstructive sleep apnoea treatment in coronary artery disease: a long-term follow-up study. Eur Heart J 2004;25:728-34
24. Marin JM, Carrizo SJ, Vicente E, et al. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. Lancet 2005;365:1046-53.
25. Weaver TE, Mancini C, Maislin G, et al. Continuous positive airway pressure treatment of sleepy patients

© 2025 European Society of Medicine

with milder obstructive sleep apnea: results of the CPAP Apnea Trial North American Program (CATNAP) randomized clinical trial. Am J Respir Crit Care Med 2012;186:677-83

26. Craig SE, Kohler M, Nicoll D, et al. Continuous positive airway pressure improves sleepiness but not calculated vascular risk in patients with minimally symptomatic obstructive sleep apnoea: the MOSAIC randomised controlled trial. Thorax 2012;67:1090-6
27. Bratton DJ, Stradling JR, Barbé F, et al. Effect of CPAP on blood pressure in patients with minimally symptomatic obstructive sleep apnoea: a meta-analysis using individual patient data from four randomised controlled trials. Thorax 2014;69:1128-35.
28. Lozano L, Tovar JL, Sampol G, et al. Continuous positive airway pressure treatment in sleep apnea patients with resistant hypertension: a randomized, controlled trial. J Hypertens 2010;28:2161-8
29. Martínez-García MA, Capote F, Campos-Rodríguez F, et al. Effect of CPAP on blood pressure in patients with obstructive sleep apnea and resistant hypertension: the HIPARCO randomized clinical trial. JAMA 2013;310:2407-15.
30. Pang KP, Woodson BT. The Pang-Woodson Protocol. Chapter 4. Advanced Surgical Techniques in Snoring and OSA. Chief Editor: Pang KP, Co-Editors: Rotenberg BR, Woodson BT. 1st Edition. Plural Publishing.
31. Thong JF, Pang KP. Clinical parameters in obstructive sleep apnea: are there any correlations? OHNS 2008 Dec;37(6):894-900.
32. Pang KP, Terris DJ, Podolsky R. Severity of obstructive sleep apnea: correlation with clinical examination and patient perception. Otolaryngol Head Neck Surg. 2006 Oct;135(4):555-60.
33. Friedman M, Ibrahim H, Bass L. Clinical staging for sleep-disordered breathing. Otolaryngol Head Neck Surg 2002;127(1):13–21.
34. Nashi N, Kang S, Barkdull GC, Lucas J, Davidson TM. Lingual fat at autopsy. The Laryngoscope. 2007;117(8):1467–73.
35. Ahn SH, Kim J, Min HJ, Chung HJ, Hong JM, Lee JG, Kim CH, Cho HJ. Tongue Volume Influences Lowest Oxygen Saturation but Not Apnea-Hypopnea Index in Obstructive Sleep Apnea. PLoS One. 2015 Aug 17;10(8)
36. Kim AM, Keenan BT, Jackson N, Chan EL, Staley B, Poptani H, Torigian DA, Pack AI, Schwab RJ. Tongue fat and its relationship to obstructive sleep apnea. Sleep. 2014 Oct 1;37(10):1639-48.
37. Shelton KE, Woodson H, Gay S, Suratt PM. Pharyngeal fat in obstructive sleep apnea. Am Rev Respir Dis. 1993;148:462–6.
38. Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. Sleep. 1991;14(6):540–545
39. Baldwin CM, Griffith KA, Nieto FJ, O'Connor GT, Walsleben JA, Redline S. The association of sleep-disordered breathing and sleep symptoms with quality of life in the Sleep Heart Health Study. Sleep. 2001;24:96–105
40. D'Ambrosio C, Bowman T, Mohsenin V. Quality of life in patients with obstructive sleep apnea: effect of nasal continuous positive airway pressure--a prospective study. Chest. 1999;115:123–9
41. Jenkinson C, Stradling J, Petersen S. Comparison of three measures of quality-of-life outcome in the evaluation of continuous positive airways pressure therapy for sleep apnoea. J Sleep Res. 1997;6:199–204.
42. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res. 1989 May;28(2):193-213.
43. Jing J, Huang T, Cui W, Shen H. Effect on quality of life of continuous positive airway pressure in patients with obstructive sleep apnea syndrome: a meta-analysis. Lung. 2008;186:131–44
44. Weaver TE, Mancini C, Maislin G, et al. Continuous positive airway pressure treatment of sleepy patients with milder obstructive sleep apnea: results of the CPAP Apnea Trial North American Program (CATNAP) randomized clinical trial. Am J Respir Crit Care Med. 2012;186:677–83. *This article illustrates the results of CPAP on sleepiness.*
45. Kuna ST, Gurubhagavatula I, Maislin G, et al. Noninferiority of functional outcome in ambulatory management of obstructive sleep apnea. Am J Respir Crit Care Med. 2011;183:1238–44.
46. Phillips CL, Cistulli PA, Grunstein RR. Health outcomes of continuous positive airway pressure versus oral appliance treatment for obstructive sleep apnea: a randomized controlled trial. Am J Respir Crit Care Med. 2013;187:8. *Elegant study showing the comparisons.*
47. Woodson BT, Steward DL, Weaver EM, Javaheri S. A randomized trial of temperature-controlled radiofrequency, continuous positive airway pressure, and placebo for obstructive sleep apnea syndrome. Otolaryngol Head Neck Surg. 2003;128:848–61
48. Ware J, Kosinski M, Keller S. SF-36 physical and mental health summary scales: a user's manual. Health Assessment Lab. 1994
49. Ware JE, Jr., Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. Med Care. 1992;30:473–83.
50. Hunt SM, McKenna SP, McEwen J, Backett EM, Williams J, Papp E. A quantitative approach to perceived health status: a validation study. J Epidemiol Community Health. 1980;34:281–6.
51. EuroQol Group. EuroQol--a new facility for the measurement of health-related quality of life. Health Policy. 1990;16:199–208

© 2025 European Society of Medicine

52. Rabin R, de Charro F. EQ-5D: a measure of health status from the EuroQol Group. Ann Med. 2001;33:337–43
53. Weaver TE, Laizner AM, Evans LK, et al. An instrument to measure functional status outcomes for disorders of excessive sleepiness. Sleep. 1997;20:835–43
54. Flemons WW, Reimer MA. Measurement properties of the calgary sleep apnea quality of life index. Am J Respir Crit Care Med. 2002;165:159–64
55. Pang KP, Olszewska E, Braverman I, Yang CH, Alkan U, Chan YH, Vicinic C, Cammarato G, Bovolenta E, Cheng RCT, Unadkat S, Siow JK, Shocat I, Bahgat A, Carrasco-Llatas M, Kishore S, Chandra S, Chan YH, Baptista P, Casale M, Pang SB, Lim JW, Montevecchi F, Pang E, Pang CE, Moffa A, Rotenberg B. Sleep Goal Index: A new success criteria in 618 OSA patients. MJM. 2025. *Submitted*

© 2025 European Society of Medicine