Clinical Guidelines of the Australasian Sleep Association (ASA)

The Australasian Sleep Association (ASA) is the peak scientific body in Australia and New Zealand representing clinicians, scientists and researchers in the broad area of sleep. The mission of the Australasian Sleep Association (ASA) is to lead and promote sleep health and sleep science in Australia and New Zealand and to facilitate the professional development of its members by providing education and training, fostering research and establishing clinical standards within the field. Within this context, over the past few years the clinical committee of the ASA has actively pursued a program of producing a series of clinical guidelines and position statements.

In this special supplement of Sleep Medicine, we highlight the first four position statements produced within this context. The first paper, Guidelines for Sleep Studies in Adults, was completed in mid-2014 and will be due for its first update in 2019. The document provides guidelines regarding the indications for all types of sleep studies in adults, as well as practical suggestions relating to the methods of performing and reporting sleep studies.

The second paper in this supplement is entitled, “Clinical Practice guidelines for Performing Sleep Studies in Children”. This guideline was completed in 2016 and is the first time the ASA has produced a document of this type relating specifically to paediatric sleep medicine. It provides best practice recommendations for investigating sleep disorders in children and conducting paediatric polysomnography, with particular emphasis on what is practical for an Australian context. This is particularly relevant given that access to paediatric polysomnography in Australia and New Zealand is far more restricted than it is for adults.

The third paper is an ASA position statement on the use of psychological and behavioural treatments in the management of insomnia in adults. Cognitive Behavioural Therapy for Insomnia (CBT-i) is a well proven yet underutilised intervention for insomnia—a condition with high prevalence and subsequent high community burden. The statement summarises the key features of and evidence of benefit for CBT-i and other behavioural techniques (such as mindfulness based therapy) and provides a useful framework upon which strategies to expand access to behavioural treatments can be based.

The final paper in this supplement is entitled, “The Management, Privacy and Medico-legal Issues of Electronic CPAP Data in Australia and New Zealand”. This paper includes a review of what CPAP data tracking systems are actually recording and telling clinicians and how this may impact on CPAP adherence. In this aspect it is complementary to the excellent position statement from the American Thoracic Society published in 2013 [1]. This current paper, however, expands on the topic by providing review and commentary on the privacy and legal implications of CPAP data monitoring systems. This is particularly relevant given the move over recent years towards cloud based data storage and automatic data uploads utilising modems installed into CPAP devices.

Position statements and clinical guidelines are neither absolute nor static documents. Their aim is to educate health care practitioners through evidence based review of a topic by experts in the field, and also provide practical guidance in areas where optimal evidence is not necessarily available. The field of sleep medicine and the technology it utilises is rapidly changing. Some aspects contained within any position statement or clinical guideline may over time become superseded by newly available evidence, or be influenced by evolving technology or models of care. Healthcare systems are also constantly adapting to changing funding and reimbursement models. As a consequence, these current guidelines need to be viewed alongside other literature on the topic, and viewed within the context in which they were written—the healthcare systems of Australia and New Zealand. Updates are planned for each statement five years after completion, until that time it is hoped they provide sleep medicine clinicians with useful information and subsequently help improve the sleep health of patients all over the world.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2017.03.016.

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Guidelines for sleep studies in adults – a position statement of the Australasian Sleep Association

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Executive summary

This a consensus statement by a committee of experienced sleep practitioners on the indications and performance of sleep studies in adults. The report draws significantly from several reviews of this type, which are referenced throughout the document [3–8,27,37,56] and randomised controlled trials. This guideline is designed to offer practical suggestions rather than act as an absolute standard. The guideline will require further modification as knowledge and technology continue to evolve. The committee was empanelled by the Australasian Sleep Association. Individual conflicts of interest were declared before the review began and are outlined in the Appendix. Individual conflict of interest statements were vetted by the ASA Board and were declared to all other committee members.

The report highlights the expanding and evolving nature of sleep investigations. It stresses the central role of the expert clinician in establishing the indications for sleep investigations and in the interpretation of sleep study results. A major concern regarding the performance of sleep studies is the lack of uniformity of definitions (e.g., definition of abnormal breathing events) between sleep-centres. This document seeks to improve standards within Australian and New Zealand by encouraging an evidenced-based approach to the performance of sleep testing, by promoting an internationally accepted and uniform set of definitions of sleep disordered breathing and by encouraging a high standard of laboratory quality control. This guideline provides indications for sleep studies and the methods for performing and reporting studies. The statement substantially revises and extends the 1994 and 2005 TSANZ/ASA [1,2] guideline on Sleep Studies.

The key changes are:

1. An extensively revised section on home-based and limited channel sleep studies. A clinical investigation flow chart is provided to inform readers of the options for diagnostic pathways for respiratory sleep disorders. The circumstances where the use of type 2, 3 & 4 sleep studies is not recommended has been incorporated into the document.

The committee notes:

a) for all types of sleep studies, the investigation is only one component of the diagnosis. Clinical history and examination are as important and are complimentary to the sleep study.

b) that type 2 studies have good diagnostic accuracy (to both "rule-in" and "rule-out" OSA) in selected patients and are an alternative to a type 1 study.

c) increasing evidence supporting the use of some home-based type 3 and 4 type sleep studies to "rule-in" (but not "rule-out") moderate to severe obstructive sleep apnoea. Such devices may therefore prove useful in populations where there is high prevalence of obstructive sleep apnoea or when combined with a validated sleep questionnaire(s) that enhance the pre-test probability of moderate to severe obstructive sleep apnoea. Additionally, research where type 3 & 4 studies have been used to rule in OSA have often been in carefully selected patient populations with minimal causes respiratory co-morbidities. The committee currently recommends that type 3 and 4 studies are used under the supervision of an accredited sleep physician who has a sound knowledge of the technical diagnostic capabilities and limitations of these devices plus access to type 1 and/or 2 studies.

d) that type 1 studies remain an important option in the diagnostic armamentarium for sleep disorders.

e) the use of a clinical tools appropriate for the patient population may help divide patients into high and low pre-test probability for moderate to severe OSA.

f) autotitrating positive airway pressure devices (APAP) in carefully selected populations are as effective as attended manual titration CPAP studies in determining optimal CPAP pressure.

g) there is no evidence to support “routine” in lab CPAP re-titration studies when the clinical response to CPAP treatment remains satisfactory.

2. Guidelines on the indications and performance of sleep studies in non-respiratory sleep disorders are included in the document. Specifically, the committee recommends that:

a) sleep studies are not required for the routine assessment of isolated insomnia, restless legs syndrome or uncomplicated parasomnias if one of these conditions are considered the likely primary abnormality. Such conditions are usually diagnosed with confidence following careful history and examination.

b) polysomnography be considered if there is a suspicion of overlapping disorders (e.g. co-existing sleep disordered breathing) or if, following careful clinical assessment, there is doubt about the diagnosis. It is recognised however that insomnia and OSA may co-exist in up to 30% of sleep clinic populations [109].

c) an expanded EEG and EMG montage plus continuous synchronised video recording is employed in cases of sleep movement or behaviour disorders that are violent or potentially dangerous, or where there is diagnostic uncertainty. These additional measurements can be helpful...
in distinguishing between a sleep related seizure disorder, REM behaviour disorder and NREM parasomnias.
d. in lab polysomnography be performed in all cases of suspected primary hypersonmia or narcolepsy to rule out co-existing disorders such as OSA that can contribute to daytime sleepiness.
e. the multiple sleep latency test (MSLT) is used as an aid in the diagnosis of patients suspected of narcolepsy or idiopathic hypersonmia.
f. daytime tests which assess the ability to resist sleep such as the maintenance of wakefulness test (MWT) may be helpful in the management of sleepy patients, particularly for medicolegal and occupational driving purposes, or where there is a discrepancy between PSG findings and symptoms.

Introduction

The document examines the indications and standards for sleep studies under the following headings:

- Respiratory Sleep Disorders
- Movement and Behavioural Sleep Disorders
- Non-respiratory Disorders of Excessive Daytime Sleepiness
- Insomnia and Other Disorders Characterised by Insufficient Sleep

1. Respiratory sleep disorders

The need for common definitions and diagnostic methodologies has become evident, for both clinical and research purposes, particularly in the measurement of respiratory sleep disorders.

Historically, an important problem with respiratory sleep studies has been the lack of agreement on recording methods and on definitions of abnormal respiratory events. In 1992, an evidenced-based guideline on the measurement and scoring of respiratory events was published by the AASM [16] and commonly known as the “Chicago” recommendations. In 2007, scoring rules were subject to a major revision by the AASM [17] and constituted a major advance but contained important omissions and ambiguities, noted by the ASTA/ASA commentary 2010 [120]. During this later period, among adults 30–70 years of age, approximately 13% of men and 6% of women have moderate to severe OSA using the same definitions for apnoea and hypopnoea. This increase in prevalence mandates the need to identify methods to reduce the cost and increase the availability of investigations for OSA [9,10].

Additionally, alternate models of care supported by high quality evidence have emerged in the Australian setting. These studies indicate that motivated and well trained health practitioners (sleep nurses [14] and general practitioners [15]) with appropriate sleep physician back-up and utilising type 4 sleep studies and APAP devices can deliver comparable care to traditional models (involving sleep physicians utilising type 1 studies) in carefully selected groups of patients with moderate to severe OSA.

1.1. Types of respiratory diagnostic sleep studies [5]

Respiratory sleep studies may be divided into two broad categories:
- Polysomnography (type 1 & 2)
- Limited channel sleep studies (type 3 & 4)

In turn these studies may be supervised as follows:
- Attended
- Unattended

Their duration may be:
- Full night
- Split-night
- Restricted duration

Type 1

Polysomnography study (PSG) requires the continuous recording of multiple physiological variables to measure sleep architecture and cardio-respiratory function during sleep. This type of study is the reference standard against which other respiratory monitoring are evaluated. There is a large body of evidence supporting type 1 sleep studies reliability and accuracy. There is some debate regarding whether type 1 sleep study represents the “gold standard” against which the other types of studies should be compared.

The signals routinely recorded include: two electroencephalogram (EEG) signals, bilateral electro-oculograms (EOGs), submental electromyography (EMG), electrocardiography (ECG), bilateral anterior tibial muscle activity, arterial O2 saturation, sound, respiratory thoraco-abdominal movements, airflow (nasal pressure and oronasal thermocouples) and body position. Other variables may be additionally recorded such as digital video, transcutaneous CO2, and oesophageal pressure (to assess respiratory effort). A Type 1 study refers to a laboratory based PSG.

PSG allows measurement of sleep stage and accurate quantification of respiratory events (against time spent asleep). REM sleep is frequently associated with exacerbation of the sleep related breathing abnormality and, in some cases sleep disordered breathing may be confined entirely to REM sleep. It distinguishes obstructive from central events, determines the effects of body position on sleep disordered breathing, allows the recognition of some alternative diagnoses (e.g. periodic limb movement disorder, parasomnias) and may suggest other sleep disorders (e.g. narcolepsy, chronic sleep restriction due to circadian rhythm disturbance). It provides information on sleep fragmentation and arousals which are likely important in the genesis of daytime symptoms arising from abnormal sleep-related respiratory events. Body and head position assessment can be clarified with synchronised video monitoring.

Type 2

A Type 2 study refers to a portable PSG device that is unattended by trained sleep laboratory staff. It records a minimum of seven channels including EEG, EOG, chin EMG, ECG or heart rate, airflow, respiratory effort and oxygen saturation. A higher success rate is generally obtained when type 2 studies are set up by experienced personnel in the home environment. This type of monitor allows...
for sleep staging and therefore calculation of an AHI. It is configured in a fashion that allows studies to be performed in the home.

The available literature indicates that type 2 studies can be used as part of a diagnostic pathway to rule in and rule out suspected sleep apnoea (Fig. 1). Type 2 studies set up in the home have been thoroughly evaluated by the Sleep Heart Health Research (SHHS) Group who performed over 7000 studies to a high technical standard [70] with reasonable agreement with repeated studies at home [71] and in the laboratory [72]. Its application in a general clinical sleep apnoea population has been confirmed [73]. A New Zealand group [73] reported a failure rate of 6.6%, which was similar to SHHS [70] and an earlier clinical study [74]. It is important to distinguish the method by which type 2 studies are implemented. There are three standard methods: 1. the patient comes to the laboratory and is “wired up and sent home; 2. a technician comes to the patient’s house and “wires the patient up”; and 3. the patient is provided with instructions and undertakes the “wire up” at home unsupervised. The failure rates of patients wired up in the lab and sent home is higher than home set up with supervision [80–81]. Nevertheless, this fact needs to be weighed against the practicalities of having staff available to safely go to

![Algorithm for Diagnosis of Suspected OSA](image)

**Fig. 1.** Algorithm for diagnosing suspected OSA. *There is variability in the definition of moderate to severe OSA. Readers should note in some articles it is listed as an AHI ≥15 [67] and in others an AHI ≥30 [76] based on the AASM Chicago criteria of 1999 [16]. A variety of clinical tools can be used to divide patients into high and low probability for moderate to severe OSA. **NB A repeat Type 2 study is unable to be billed via Medicare within 12 months of the original test.

![Graph of Oximeter Derivatives](image)

**Fig. 2.** This figure illustrates from an early in-lab study of patients with high probability of OSA indicating a significant correlation of simultaneous ODI with AHI (where desaturation ≥ 3% was part of the definition of hypopnoea) from Rauscher [87]. Acknowledgement: European Respiratory Journal Jun 1991, 4 (6) 655–659.

![Graph of Oximeter Derivatives](image)

Fig. 4. Receiver operating characteristics (ROCs) for a type 3 study derived-AHI cut-off points, compared to a type 1 study PSG-AHI at a threshold of >30 events/hour. Each point represents sensitivity plotted against specificity corresponding to different cut-off points of the type 3 study derived-AHI. Using a cut point of the type 3 study derived-AHI of 18, a sensitivity of 73% and a specificity of 80% is obtained. Lower cut-off points give better sensitivity but worse specificity. [86] Acknowledgment: European Respiratory Journal Aug 1997, 10 (8) 1720–1724.

patient’s homes. Campbell [73] found that signal loss is higher and there is a small under-estimate of AHI (approximately 10%) due to greater loss of respiratory effort signals in the home compared to laboratory environment. These differences did not alter treatment advice. A meta-analysis of laboratory versus portable sleep studies [84] concluded that they both provide similar diagnostic information, but type 2 studies may underestimate severity of AHI by around 10%. A screening process of the referral needs to be undertaken by the clinician approving the study to ensure the safety of staff visiting the patient’s home is adequate.

Patients who may be unsuitable for Type 2 diagnostic studies:

A. Patient related factors
1. Neuropsychological
   • Severe intellectual disability (this may also be an issue for type 1 studies)
   • Neuromuscular disease
   • Major communication difficulties
2. Severe physical disability with inadequate carer attendance
3. Home environment unsuitable – a number of factors need to be considered including noise level, partner/family interactions, distance from sleep lab and the safety of any attending staff
4. Discretionary
   • symptoms or results of former testing do not equate with clinical impression
   • patients seeking a second opinion where the original diagnosis is uncertain.
   • where “serious” medico-legal consequences may be relevant
B. Sleep disorder related factors
   • Parasomnia/seizure detection requiring infrared camera or extended EEG montage
   • Transcutaneous CO2 monitoring required
   • Where video confirmation regarding body positional/rotational aspects of sleep disordered breathing is essential

Type 3
Limited channel sleep studies (type 3 and 4) have a more restricted number of parameters measured, usually a combination of respiratory variables including arterial O2 saturation, respiratory effort and airflow. In general, sleep staging is omitted from limited sleep studies.

Type 3 studies have at least four variables monitored: oximetry plus respiratory effort (chest, abdominal, or both), airflow (nasal or oral by pressure or thermistor), head or body position, jaw movement, ECG, tonometry (a marker of autonomic control), actigraphy and sound (vibration detection or true sound recording). Thus, there is a variety of monitors that can be utilised to achieve a type 3 study. In order to review these more thoroughly, the SCOPER categorization system has been established which includes monitoring Sleep, Cardiovascular, Oximetry, Position, Effort and Respiratory parameters [85]. It is recommended that at a minimum, respiratory effort, airflow and oximetry are recorded [85]. Automation of some or all of the data analysis is generally feasible.

Tonometry is also available (marker of autonomic control and thereby sleep) as an adjunct to oximetry. Expensive disposable equipment is required (e.g. Watchpat). It may add a small amount to diagnostic accuracy of AHI, by reducing the denominator (“total recording time” to “total sleep time”).

At least 15 studies have compared type 3 studies with polysomnography results, either simultaneously or on consecutive nights. Most have low AHI thresholds (AHI>5) with correlation coefficients 0.58–0.95, sensitivities of 84–100% and specificities 59–100%. The key finding of one such study is illustrated in Fig. 4 [86]. A type 4 study incorporates only one or two monitors – for example oximetry, heart rate or airflow. Oximetry is the cornerstone signal of sleep apnoea monitoring. In comparison with all other signals it is the most accurate, quantifiable, reliable and informative signal. The development of the multivariate length oximetry and reduction in size has made oximetry a ubiquitous and accurate marker of hypoaxemia. The accuracy of oximetry is estimated to be ±2% between the arterial oxyhaemoglobin saturation range of 70–100% and can accurately track change. Technical factors such as adequate signal acquisition, averaging time and storage sampling frequency are crucial to the reliability of oximetry. Failure rates as low as 3% of patients studied have been reported [76].

A variety of oximetry derived parameters have been compared to PSG derived AHI with reasonable sensitivities and specificities (Figs. 2, 3). Oximetry with heart rate can be used to derive an oxygen desaturation index (ODI) which can be further delineated based upon a ≥3% or ≥4% desaturation (ODI3 or ODI4, respectively). Oximetry can diagnose moderate to severe OSA (AHI >15) with correlation coefficients 0.58–0.95, sensitivities of 84–100% and specificities 59–100%. The key finding of one such study is illustrated in Fig. 4 [86]. A type 4 study incorporates only one or two monitors – for example oximetry, heart rate or airflow. Oximetry is the cornerstone signal of sleep apnoea monitoring. In comparison with all other signals it is the most accurate, quantifiable, reliable and informative signal. The development of the multivariate length oximetry and reduction in size has made oximetry a ubiquitous and accurate marker of hypoaxemia. The accuracy of oximetry is estimated to be ±2% between the arterial oxyhaemoglobin saturation range of 70–100% and can accurately track change. Technical factors such as adequate signal acquisition, averaging time and storage sampling frequency are crucial to the reliability of oximetry. Failure rates as low as 3% of patients studied have been reported [76].

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Limitations of oximetry with heart rate are seen in cardiac, pulmonary and neurological patients where differentiation between obstructive and central sleep apnoea is especially important. Low baseline awake oxygen saturation will reduce specificity further. Supplemented oxygen therapy may negate the utility of signal interpretation. The reliance on only one or two signals means that redundancy of information during signal drop out may be very limited. Other limitations include lack of positional data and use of time in bed rather than actual sleep time in calculating the level of respiratory disturbance.

Questionnaires completed by the patient and bed partner regarding estimates of sleep time, body position, presence of snoring etc. may help compliment data recorded from type 4 studies.

It is essential that if type 3 and 4 studies are being used to rule in OSA, there are clearly defined pathways for: a) assessing the pre-test probability of a patient having moderate to severe OSA; b) patients with co-morbidities that could confound the results are excluded; c) inconclusive tests or results at odds with the clinical suspicion are referred for type 1 or 2 sleep studies and d) in an appropriately resourced clinical environment, where the patients unsuitable for type 3 and 4 studies have been excluded, a positive type 3 or 4 study for moderate to severe OSA in the setting of a high pre-test probability of OSA should result in the cessation of further investigation for sleep disordered breathing.

Oximetry’s variable specificity (41%–100%) and sensitivity (31%–98%) across a range of studies is due to differences in:

a. study populations (history, examination, questionnaires)
b. comparison groups: usually type 1 attended polysomnography. These studies have limitations
   1. threshold (AHI) for sleep apnoea [86].
   2. denominator for AHI (Total sleep time vs Total recording time).
   3. same vs consecutive night
c. device settings
   signal acquisition averaging time (e.g. 2 vs 0.1 s)
   storage sampling frequency (e.g. 1.0 to 0.1 Hz)
d. artefact detection
e. medical conditions e.g. Raynaud’s phenomenon & haemoglobinopathies

Patients who may be unsuitable for Type 3 and 4 Diagnostic studies include:

1. Populations with a low-pre-test probability of moderate to severe OSA
2. Patients reporting symptoms suggestive of a condition other than sleep disordered breathing which will require more extensive monitoring, e.g. parasomnia, narcolepsy, periodic limb movement disorder, nocturnal epilepsy etc.
3. Patients with any of the following (where nocturnal hypoventilation or central sleep apnoea is likely):
   a. Neuromuscular disease
   b. Severe COPD or restrictive lung disease
   c. Hypoxia and/or hypercapnia at rest, or requiring supplemental oxygen therapy
d. Morbid obesity and/or suspected obesity hypoventilation syndrome
   e. Significant cardiovascular disease, i.e. recent hospitalisation for acute MI, unstable angina, decompensated heart failure
   f. Chronic narcotic use
4. Inability to perform overnight oximetry in a non-monitored environment, e.g. active significant psychiatric disease

1.2. Choosing the type of diagnostic respiratory sleep study

In choosing which test or tests are to be used, physicians should have a clear understanding of: (a) the indications for testing; (b) the sensitivity and specificity of the test(s) to diagnose sleep disordered breathing; (c) the overall utility of the test taking into consideration the prevalence of sleep apnoea in their population; (d) the cost/benefit balance of the test in their particular clinical setting; (e) the technical limitations of the monitoring signals utilised in each particular study type; and (f) comorbidities which need to be considered in choosing the type of sleep study.

1.2.1. Screening tools to divide patients into high and low probability for moderate to severe OSA

A key recommendation of this guideline is Fig. 1 on page 8. Suitable patients with a high pre-test probability of moderate to severe OSA and no significant cardiorespiratory co-morbidities could be initially investigated with a Type 3 or 4 study. This approach would be particularly useful in high prevalence populations or where access to Type 1 or 2 studies is limited. This approach may also be helpful in triaging patients in services that predominantly perform Type 1 or 2 studies. Moderate to severe OSA was chosen because it is estimated that 83% or men and 92% of women with this severity of OSA have not been diagnosed [63]. Additionally, patients with moderate to severe OSA have the highest morbidity and also adhere and respond to treatment better than those with mild disease [64]. The definition of moderate to severe OSA is variable. Sometimes it is defined as an AHI ≥30 events/hour based on the 1999 AASM “Chicago” criteria [16], and on other occasions ≥15 events/hour on the same criteria. Screening tools can assist in determining which patients have a high pre-test probability of moderate to severe OSA. Screening tools that have high sensitivity and negative predictive value properties, maximise the utility of Type 3 and 4 sleep studies.

An ideal screening questionnaire should have three important characteristics [66]:

1) Feasibility: Patients and healthcare providers should find the questionnaire user friendly;
2) Accuracy: There should be a clear validation process that leads to high accuracy parameters;
3) Generalizability: Valid results should be realized when the questionnaire is used on different target populations, i.e., the questionnaire has been validated in different study populations.

There are at least four potential settings where use of a screening questionnaire could be beneficial:

- general practice
- sleep clinic
- pre-operative clinic
- occupational setting

There have been two recent reviews of screening questionnaires for OSA [78,79]. Three questionnaires demonstrate good to reasonable psychometric properties to support their validity. These are the Berlin Questionnaire [65], STOPBANG [67–69] and OSA50 [76].

Berlin Questionnaire

The Berlin questionnaire is an OSA questionnaire developed for and validated in primary care and categorises patients as either high or low risk for OSA based on self-reports of snoring, daytime sleepiness, hypertension and obesity [65]. The 11 questions were chosen by a panel of sleep physicians without prior evaluation as to their respective discriminatory values. Although published a decade ago, the Berlin questionnaire is not widely used, possibly
because of the time required for completion and scoring [76]. The predictive parameters of the Berlin questionnaire vary depending on to which patient population they are applied. The sensitivity was 86% in primary care patients [65], and 57–68% in sleep laboratory patients [107]. There are some important methodological limitations of the Berlin questionnaire. Patients were pre-screened prior to utilising the questionnaire and analysis of the PSG was not blinded to the result of the Berlin questionnaire.

**STOP-Bang Clinical Tool [66–69]**

This was developed and validated initially in pre-operative clinics in patients without previously diagnosed OSA. The design of this questionnaire was robust with factor analysis, reliability check and a pilot study used to determine the screening tool. The tool initially comprised of four yes/no questions covering the domains of Snoring, Tiredness, Observed apnoeas and blood Pressure (STOP). It was initially designed to detect any severity of obstructive sleep apnoea in a pre-operative cohort of patients. Following design of the questionnaire, it was then administered to 1875 patients all of whom were invited to undertake an overnight polysomnographic study. The sensitivities of the STOP score with the AHI based on AASM Chicago criteria [16] were analysed. The positive predictive values of STOP questionnaire for AHI ≥5 events/hour was further enhanced by the addition of the parameters of BMI greater than 35 kg/m², age >50, neck circumference >40 cm and male gender.

The STOP-Bang questionnaire (Table 1) is quick to administer and can divide patients into low and high risk for OSA. The predictive parameters are good for moderate to severe OSA, i.e. AHI ≥30 events/hour on Chicago criteria [16].

There has been a further study [67] which has refined the utility of the STOP-Bang screening tool for surgical patients. Scores of <3 make it unlikely patients will have an AHI >15 events/hour (Chicago criteria, reference 16). A score of 5–8 greatly increases the probability of an AHI >15 events/hour.

The STOP-Bang score has been assessed in sleep clinic populations [68,69]. The first study [68] looked at a variety of ways in which information from the STOP-Bang score could be used more discriminately to predict AHI in sleep clinic populations. This study examined 1426 patients in whom the STOP-Bang score was derived and all patients underwent a Type 1 sleep study. Rather than using a binary result (low and high risk for OSA) for the STOP-Bang score, the study examined the benefit of a cumulative score (0–8) for predicting severity of OSA. For ease of use, a linear model was recommended and the results are displayed in Fig. 5.

The second study [69], looked at the Sleep Heart Health Study cohort and identified the patients as having a high risk for moderate to severe OSA (RDI ≥15 events/hour [RDI defined in reference [70])] based on a STOP-Bang score of ≥3. The sensitivity utilising this test was 87%.

A published four-variable screening tool (reference [71]) was also applied to this population and it demonstrated good specificity (92.3%) in ruling out moderate to severe OSA. The four variable screening tool divides weight and blood pressure into a number of categories and also accounts for gender and snoring.

**OSA50**

The OSA50 [72] has recently been developed in an Australian general practice health care setting. The aim of this study was to develop and validate a simplified two-stage method for identifying moderate to severe OSA (AHI > 30 events/hour of sleep based on Chicago criteria [16]) in primary care consisting of an easy-to-administer screening questionnaire followed by a type 4 sleep study. The type 4 sleep study utilised was the ApneaLink.

In the development data set, four items were significantly predictive of moderate to severe OSA. See Table 2.

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Table 1
Fig. 5. Percent of patients with OSA defined by AHI categories based on cumulative STOP-Bang score (From reference [68]). Republished with permission of [Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine], from [The STOP-Bang equivalent model and prediction of severity of obstructive sleep apnoea: relation to polysomnographic measurements of the apnoea/hypopnea index. Farney RJ, Walker BS, Farney RM, Snow GL, Walker JM, J Clin Sleep Med 7: 459–65. 2011.]; permission conveyed through Copyright Clearance Center, Inc.
The area under the curve to detect moderate to severe OSA (AHI \( \geq 30 \)) using the OSA-50 questionnaire was 0.84 (95% CI 0.75 to 0.94, \( p < 0.001 \)) for the developmental data set. Using a cut-off score of \( \geq 5 \) out of 10, this questionnaire had a sensitivity of 100% (95% CI 86%–100%), NPV of 100% (95% CI 73%–100%), specificity of 29% (95% CI 17%–44%) and PPV of 48% (95% CI 35%–63%).

Results from the ApneaLink were analysed against AHI derived from a simultaneous Type 2 sleep study with scoring of respiratory events based on the AASM Chicago criteria [16]. ROC curves for the ApneaLink 3% ODI and AIHI20-50 (based on nasal flow) against PSG in the development group were highly predictive of moderate to severe OSA with ROC AUC values of 0.96 (95% CI 0.91 to 1.00, \( p < 0.001 \)) and 0.95 (95% CI 0.89 to 1.0, <0.001), respectively. The 3% ODI was selected for use in the two-stage model because oximetry was technically more reliable than nasal airflow measurements with fewer failures (only 3% patients with failed oximetry).

The diagnostic characteristics of the two-stage model using a cut-off values of \( \geq 5/10 \) for the OSA50 questionnaire and \( \geq 16/\text{h} \) for the 3% ODI, revealed the model was capable of identifying moderate to severe OSA with a high sensitivity and specificity and had an overall diagnostic accuracy (sum of the true positive and true negative rate) of 91% in the development set.

The two step model was then assessed on a validation group of 78 participants. Using cut offs of OSA50 score \( \geq 5/10 \) and a 3%ODI \( \geq 16/\text{h} \) on an ApneaLink, the sensitivity and specificity were both \( >80\% \), the negative predictive value was 96% and the overall diagnostic accuracy (sum of true positive and true negative rate) was 83%. Although the positive predictive value was lower that anticipated in the validation group at 56%, review of the “false positives” indicated that the minimum PSG AHI was 18.9 events/hour and half the group reported excessive daytime sleepiness with Epworth Sleepiness Scale scores \( >12 \), suggesting that these patients would likely gain benefit from therapy taking into account their symptoms.

There were minimal exclusion criteria used in this study. Only pregnant women, patients with significant cognitive impairment, a poorly controlled psychiatric disorder or patients who had previously received treatment for OSA were excluded.

### Summary of OSA Clinical Tools

There are an increasing number of well validated questionnaires for OSA that have been utilised in general practice populations, peri-operative and sleep clinic settings. These may assist clinicians in determining who is likely to have moderate to severe OSA and who may be suitable to proceed directly to Type 3 or 4 sleep studies. Additionally, these tools may be helpful in determining the urgency to proceed to a Type 1 or 2 study where type 3 or 4 sleep studies are not readily available.

### 1.3. Indications for sleep studies for sleep disordered breathing

There are three broad indications in relation to sleep related breathing disorders:

#### 1.3.1. Diagnostic studies: to aid making a diagnosis and to classify severity of sleep disordered breathing (SDB)

#### 1.3.2. Intervention studies: to implement and titrate, or confirm effectiveness of a new treatment

#### 1.3.3. Follow-up studies: to track the progress of a patient

#### 1.3.1. Diagnostic studies

Diagnostic studies are performed to identify and quantify severity of SDB:

- **1.3.1.1.** Suspected obstructive sleep apnoea (OSA) syndromes
- **1.3.1.2.** Suspected central sleep apnoea (CSA) syndromes
- **1.3.1.3.** Suspected sleep hypoventilation syndromes (sleep-disordered breathing associated with disorders of respiratory muscles, chest wall or lung e.g. muscular dystrophy, kyphoscoliosis, chronic obstructive pulmonary disease, and SDB associated with neurologic disorders)
- **1.3.1.4.** Suspected SDB associated with recognized predisposing non-respiratory disorders (e.g. congestive heart failure, significant tachyarrhythmias, neurological disease, morbid obesity, acromegaly, hypothyroidism)
- **1.3.1.5.** Suspected SDB when upper airway surgery or bariatric surgery is being contemplated to treat snoring or SDB.

#### 1.3.1.1. Suspected obstructive sleep apnoea syndromes

High-risk patients in whom the question of OSA arises tend to fall into one of the groups:

- a) Patients with a history of habitual loud snoring and marked daytime sleepiness and in whom apnoeas have also been witnessed. There is a high probability these patients have OSA and a sleep study is recommended. Higher risk patients include those who are obese (BMI > 35 kg/m\(^2\)), increased neck circumference (>43 cm in men, >40 cm in women), those with tonsillar hypertrophy and/or retrognathia. Patients with co-morbidities of congestive heart failure, atrial fibrillation, treatment refractory hypertension, type 2 diabetes, stroke, nocturnal dysrhythmias, pulmonary hypertension, high-risk driving populations (such as commercial truck drivers), and those being evaluated for bariatric surgery also represent high risk. The OSA50 or STOP-Bang

<table>
<thead>
<tr>
<th>If yes, SCORE</th>
<th>If no, Scanner</th>
<th>3</th>
<th>2</th>
<th>1</th>
<th>Total Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity</td>
<td>Waist circumference* - Males &gt;102cm or Females &gt;88cm</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Snoring</td>
<td>Has your snoring ever bothered other people?</td>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apnoea</td>
<td>Has anyone noticed that you stop breathing during your sleep?</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>50</td>
<td>Are you aged 50 years or over?</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**TOTAL SCORE:**  

\( \text{Score} \) / 10 points

*Waist circumference to be measured at the level of the umbilicus.*

---

**Table 2**

Patients with respiratory and/or upper airway muscle weakness or chest wall deformity may develop sleep hypoventilation in advance of daytime respiratory or right heart failure [24,25]. Hence, laboratory-based sleep studies should be considered in this group if symptoms of disturbed sleep, nocturnal dyspnoea, snoring, morning headache, daytime sleepiness, orthopnoea or progressive weakness are present [24]. Studies should also be considered if signs of pulmonary hypertension or other cardiopulmonary dysfunction occur. Elevation of awake PaCO2 and base excess in such patients may also indicate a sleep-related respiratory disturbance.

Sleep disordered breathing may also occur in association with some neurological disorders such as Congenital Central Hypoventilation Syndrome, brainstem or high spinal cord lesions.

1.3.1.4. Sleep-disordered breathing in association with recognized predisposing non-respiratory disorders. A sleep study should be considered in patients with these disorders particularly if there is a history of excessive daytime sleepiness or deteriorating cardiopulmonary function not explainable on other grounds. In conditions where the prevalence of OSA is high, such as acromegaly (prevalence of more than 50% in unselected patients [26]), a sleep study should be a routine investigation.

1.3.1.5. Suspected sleep apnoea where upper airway surgery or bariatric surgery is being considered to treat snoring or SDB. A sleep study should be undertaken whenever upper airway surgery or bariatric surgery is being contemplated to treat snoring. The reasons for this are:

a. the results may alert the surgeon and anaesthetist to the presence of clinically unsuspected sleep apnoea. This might indicate the need for alternative or additional treatments, or for particular vigilance in the early post-operative period because of increased potential for upper airway obstruction and/or impaired ability to arouse due to residual anaesthetic and analgesic effects.

b. the cause of excessive sleepiness is investigated in patients in whom excessive daytime sleepiness is part of the rationale for surgery.

1.3.2. Intervention studies

Intervention studies are performed to implement and titrate, or confirm the effectiveness of a treatment. Such therapies include pharmaceutical agents, oxygen administration, oral appliances, nasal expiratory airway pressure devices, continuous positive airway pressure (CPAP), upper airway surgery, sleep posture modification devices and non-invasive ventilation (NIV). The effectiveness or otherwise of these treatments to reverse or alleviate sleep disordered breathing should be confirmed in patients with OSA.

The majority of treatment studies undertaken are for OSAHS. Additionally most treatment studies involve application of CPAP. This document will confine itself to the description of types of studies, goals of treatment. The CPAP titration study is only one component of the treatment of sleep disordered breathing. Education of the patient and long term evaluation of the patients’ symptoms, quality of life, adherence and side effects of the treatment being equally important. Patient education and motivation to use CPAP accounts for a greater variance in CPAP adherence than standard biometric or anthropometric markers of OSA severity.

1.3.1.2. Suspected central sleep apnoea syndrome. Patients in whom the question of central sleep apnoea arises tend to fall into one of three groups:

a) Idiopathic Central Sleep Apnoea is an uncommon cause of SDB. In patients with a history of recurrent witnessed apnoeaic events associated with sleep fragmentation, excessive daytime sleepiness, and/or insomnia, should proceed to a laboratory-based sleep study.

b) Cheyne-Stokes Breathing Syndrome is characterised by a cyclical fluctuation in breathing with periods of central apnoea alternating with periods of hyperpnoea in a gradually waxing and waning fashion. Cheyne-Stokes breathing is often observed in association with congestive heart failure in 30-40% of patients with an ejection fraction of <40% and neurological disease (usually cerebrovascular) [20,21]. Hypersomnolence may occur as a result of arousals seen during the hyperpnoeic phase of the breathing cycle. Significant hypercapnea may occur during the hypopnoeic phase. OSA is also common in patients with severe congestive heart failure [20,22] and may predispose to a decline in left ventricular function and quality of life [23]. Suspected cases should proceed to a laboratory-based sleep study.

c) High Altitude Sleep Apnoea (not relevant in Australia or New Zealand).

1.3.1.3. Suspected sleep hypoventilation syndromes. Sleep-disordered breathing in association with disorders of respiratory muscles, chest wall or lung (e.g. muscular dystrophy, kyphoscoliosis, obesity hypoventilation syndrome, chronic obstructive pulmonary disease (COPD)).

In patients with respiratory disorders in whom complications such as right heart failure, polycythaeama and hypercapnic respiratory failure appear disproportionately severe relative to the impairment of daytime respiratory function, the possibility of OSA or sleep hypoventilation should be considered, particularly if obese and/or known to snore habitually. A laboratory-based sleep study should be considered in the investigation of such patients.
1.3.2.1. Intervention studies of CPAP therapy for OSAHS. Options of CPAP titration for patients with OSA:

1. Manual pressure adjustment by a sleep technologist during attended laboratory polysomnography (PSG) (This is regarded as the standard of care [27])
2. Split night studies
3. Autotitrating device titrations (see APAP section)
4. Diurnal sleep studies
5. Empiric pressure trials (outside the scope of this document)
6. Pressure determined by a priori equations (outside the scope of this document)

Standard components of a titration study:

1. All patients undertaking a titration study for sleep disordered breathing must have a diagnosis established by an acceptable method (see diagnostic sleep study section above) prior to a treatment study.
2. There should be a written protocol for each sleep service based on evidence and the local experience and judgement of sleep technologists and clinicians to attain the best possible titration in any given patient [27].
3. All Potential PAP titration patients (including those patients prior to a diagnostic study where the clinical suspicion of OSA is high and a Split-Night Study is a possibility) should receive adequate PAP education, hands-on demonstration, careful mask fitting, and acclimatization prior to titration [27,28].

Two randomised controlled trials [29,30] have demonstrated the benefits of adopting psychological interventions prior to the CPAP titration study to enhance rate of CPAP uptake, time to CPAP uptake and adherence.

1.3.2.1.1. Manual pressure adjustment by a sleep technologist during attended laboratory polysomnography (PSG). Aims of titration for patients with OSA:

A successful titration is one in which there is an trade-off between increasing CPAP pressure to eliminate respiratory events and decreasing CPAP pressure to minimize emergence of CPAP pressure-related side effects [31].

A Consensus recommendation from the AASM [27] asserted that the optimum pressure determined on titration study should reflect control of the patient’s obstructive breathing by a low RDI (preferably <5 events per hour) at the selected pressure, a minimum sea level SpO2 above 90% of the pressure, and with a leak within acceptable parameters at the pressure.

This guideline [27] went on to define a grading system for optimal, good, adequate and unacceptable titration based on consensus agreement of the PAP Titration Task Force and a system proposed by Hirshkowitz and Sharafkhaneh [32].

The grading system for manually titrated attended full or split sleep studies proposed the following:

An optimal titration reduces the RDI <5 per hour for at least 15-min duration and should include supine REM sleep at the selected pressure that is not continually interrupted by spontaneous arousals or awakenings. It should be noted that it may not be possible to achieve supine REM on the final treatment pressure for many patients.

A good titration reduces the overnight RDI to ≤10 per hour or by 50% if the baseline RDI is <15 per hour, and should include supine REM sleep that is not continually interrupted by spontaneous arousals or awakenings at the selected pressure.

An adequate titration is one that does not reduce the overnight RDI ≤10 per hour but does reduce the RDI by 75% from baseline (especially in severe OSA patients), or one in which the titration grading criteria for optimal or good are met with the exception that supine REM sleep did not occur at the selected pressure.

An unacceptable titration is one that does not meet any one of the above grades.

It is recommended that CPAP implementation in complex cases (e.g. patients with overlapping cardiorespiratory dysfunction or central sleep apnoea) be achieved by attended polysomnography.

1.3.2.1.2. Split night sleep studies. The AASM practice parameters for PSG [8] indicate split night sleep studies are reasonable when the following conditions are met:

a) an AHI of at least 40 events/hour is documented during a minimum of 2 h of diagnostic PSG or an AHI of 20–40 events/hour associated with pronounced obstructive features (e.g., if there are also repetitive long obstructions and major desaturations). However, at AHI values below 40 events/hour, determination of CPAP pressure requirements, based on split-night studies, may be less accurate than in full-night calibrations.

b) CPAP titration is carried out for more than 3 h (because respiratory events can worsen as the night progresses).

c) PSG documents that CPAP eliminates or nearly eliminates the respiratory events during REM and NREM sleep, including REM sleep with the patient in the supine position.

It assumed the patient will have had the same preparatory education and acclimatization as for a full night titration study (see standard components of a titration study above). Studies that have compared adequacy of prescribed pressure, CPAP adherence, and patient acceptance have found no significant differences for adult patients undergoing full-night vs. split-night CPAP titration studies with the possible exception that pressures determined from split-night studies may be lower for patients with mild-to-moderate OSA who may not manifest the maximal severity of their condition during the limited titration portion of the night [27].

A repeat Positive Airways Pressure titration study should be considered if the initial titration does not achieve the (b) or (c) criteria above. Alternatively, a trial of an autotitrating positive airways pressure device may be considered in selected patients.

1.3.2.1.3. Autotitrating positive airway pressure (APAP) sleep studies. The following section covers the many factors the clinician needs to consider about autotitrating CPAP. In each section, the evidence is summarised and recommendations are made. For many patients with uncomplicated OSA, APAP titration represents an acceptable way of determining an optimal long-term CPAP pressure.

It is difficult to make definitive statements about the use of autotitrating CPAP at home to determine the ideal pressure for the treatment of OSA or how it compares with titrating CPAP during a sleep study in a sleep laboratory as clinical studies would need to be performed with each model of autotitrating CPAP that become available. The ideal study would be a randomized controlled trial comparing manual titration in a sleep laboratory with the specific model of autotitrating CPAP assessing clinically relevant outcomes such as sleepiness, quality of life, cardiovascular disease, car crashes and mortality over a period of time.

In addition, it is not clear how best to determine the ideal CPAP level during polysomnography and in particular which parameters should be assessed when making decisions about altering pressure levels. It would be expected that when the level of CPAP chosen should eliminate obstructive apnoeas, hypopnoeas and snoring, but it is not clear how much emphasis should be placed on eliminating flattening of the inspiratory loops of pressure and flow
signals, eliminating arousals related to respiratory events, eliminating central apnoeas and hypopnoeas and what is the optimal oxygen saturation to achieve. There is a proposed system for adequate titration stated above [32] for in lab titration studies.

It has been shown in bench studies and patients that flattening of the flow contour from a CPAP system is associated with raised upper airway resistance and flow limitation [33]. It has also been shown in patients that the rounding of the CPAP inspiratory flow contour correlates better with low oesophageal pressures compared with elimination of apnoeas, hypopnoeas and arousals [34]. It is not clear though, whether eliminating flattening of the inspiratory loops either with manual titration or autotitrating CPAP produces greater beneficial clinical outcomes than only eliminating snoring, hypopnoeas and apnoeas. It is possible that raising the pressure too high may induce central apnoeas and hypopnoeas and that it may worsen clinical outcomes by reducing patient adherence to treatment and/or disturbing sleep itself.

The AASM has guidelines for manual titration of CPAP and recommends the elimination of apnoeas, hypopnoeas, snoring and respiratory effort related arousals (RERAs), although this is based on consensus rather than evidence [8,27].

Thus, it is not clear if manual titration of CPAP is the best method against which to compare home titration. Some advantages of manual titration during a laboratory sleep study are the ability to immediately deal with mask problems, recognize central or complex sleep apnoea and sleep related hypoventilation, to ensure that the patient has some sleep when supine, and to know if REM sleep occurred [35–37].

Home titration may have the advantage that the patient is sleeping in his/her more natural environment and so sleep during titration may better reflect his/her sleep when on long term treatment with CPAP. It is easier to have the titration over multiple nights so that other factors such as the use of alcohol and body position can also be included [38].

Even though there are not studies comparing both methods with long term clinical outcomes, there are studies in which the CPAP levels obtained with the two methods are compared. The published evidence is limited by the comparison of a specific device with a specific algorithm to manual titration. Due to proprietary commercial information, the details of the algorithms that are used in these machines to alter the level of CPAP and to identify hypopnoeas and apnoeas and produce an AH1 are not generally available to clinicians. Also, manufacturers are often updating their devices and releasing new models so even when there are published evaluations of pumps these evaluations do not necessarily apply to pumps being used currently [39]. Thus, choosing devices relies on clinical studies directly assessing autotitrating CPAP with standard CPAP, and assessing patients’ clinical responses to treatment with autotitrating CPAP. The latter approach can be difficult in individual patients in routine clinical care because of the placebo effect [39].

Suitable patients for autotitrating CPAP

If home autotitration is to be used to determine a suitable level of CPAP, care needs to be given in identifying patients who may or may not be suitable for this approach. Most studies using home autotitration have performed it in patients with moderate to severe OSA, with wide ranges of age, AH1 and symptoms with a few key exclusions.

Patients tend to be excluded if there is concern about the possibility of: having significant hypoxaemia during sleep unrelated to OSA, having central sleep apnoea, having other significant sleep disorders or having other medical disorders that make home titration difficult. One of the largest and more recent studies, the HomePAP study [40], had the following exclusion criteria for home autotitration:

- COPD (with FEV1/FVC < 70% & FEV1 < 50% predicted)
- Regular use of supplemental oxygen
- Waking oxygen level less than 92%
- Awake hypercapnia or hypoventilation syndrome
- Heart failure
- Chronic narcotic use
- Neuromuscular/chest wall disease
- Alcohol abuse
- Significant other sleep disorders
- Uncontrolled psychological or psychiatric disorder

Recommendation:

- Home autotitration of CPAP is suitable for a wide range of patients.
- Common sense is required to make sure that the patient is able to use the pump and mask at home.
- Home CPAP autotitration is not suitable for those who may have central sleep apnoea and those who may have significant hypoxaemia during sleep due to conditions other than OSA, significant other sleep disorders or other medical conditions that limit the patient’s ability to use the therapy at home during the titration phase.

Pressure level

Different autotitrating CPAP pumps have been designed to detect and respond to different parameters. Some devices simply adjust for apnoeas and hypopnoeas, others use these plus snoring and/or evidence of airflow limitation such as flattening of the inspiratory flow curve. Some have used detection of vibrations or the forced oscillation technique (FOT) [41,42].

Bench studies using a breath waveform stimulator to detect flattened inspiratory flow, and a patient simulator model to replicate snoring, obstructive apnoeas and central apnoeas, flow limitation and mouth leaks have shown that different autotitrating CPAP pumps vary widely in their ability to detect and respond to respiratory events, snoring and flattened inspiratory flow [42,43].

The recommended effective level of pressure from autotitration can be determined in different ways. It may be the maximum pressure that was achieved during the duration of the recording, or more commonly, it is the 95th or 90th percentile pressure. For example, the 95th percentile pressure is the pressure below which the pump delivers for 95% of the recorded time. In other words, the pressure only went above this level for 5% of the time. Llobres [44] directly compared the 90th and 95th percentiles and found the mean pressures to be similar, 10.7 ± 2.7 cm H2O and 11.5 ± 2.9 cm H2O, respectively. What is relevant for clinicians is the comparison between an effective, clinically relevant pressure determined by manual titration and one derived from autotitrating CPAP [44,45].

Rapid increases or significant fluctuations in CPAP without significant mask leak may indicate an inappropriate response of the CPAP pump to sleep/wake transitions. In these cases, titrating the CPAP level manually during polysomnography in a sleep laboratory may be necessary [46].

In summary, the effective pressure derived from autotitrating CPAP is similar to that obtained from manual titration of CPAP in a sleep laboratory. However, it should be recognized that this can vary from person to person and is dependent on the characteristics of the particular model of autotitrating CPAP and on the criteria that are used to adjust CPAP levels during manual titration and the skill and experience of the person applying them.
Recommendations:
- The effective pressure derived from autotitrating CPAP should be that which is recommended by the manufacturers. This is usually the 95th or 90th percentile pressure.
- It cannot be assumed that data from one model of autotitrating CPAP can be applied to another model.
- Rapid increases or significant fluctuations in CPAP without significant mask leak may indicate an inappropriate response of the CPAP pump to sleep–wake transitions. In these cases titrating the CPAP level manually during polysomnography in a sleep laboratory may be necessary.

The number of nights needed to determine an effective pressure

Recommendation:
- For many patients one night of autotitrating CPAP is enough to determine the effective pressure, but not in all patients. Some will need several nights, so a minimum of three nights is recommended [46,47].

Outcomes:

Patient outcomes are more important than precise pressure level when comparing with different modes of CPAP initiation, such as manual laboratory titration versus home autotitrating CPAP.

Polysomnographic outcomes

Overall, studies have shown that autotitrating CPAP at home is at least as effective as CPAP with manual titration in reducing the AHI and improving sleep architecture [48–50].

Patient outcomes

West [52] compared six months of Autoset (Resmed™) with six months of fixed CPAP after one week of Autoset (to determine the 95th percentile pressure) and six months of fixed CPAP with the level determined by a formula. There was no difference over the six months between the three groups in sleepiness (ESS and ESS-M), quality of life and blood pressure.

McArdle [51] found no difference in sleepiness, quality of life or blood pressure over four weeks when CPAP was titrated with manual laboratory titration aiming to eliminate apnoeas, hypopnoeas, snoring and flow limitation and reduce arousals, with one night of the ResMed Autoset at home to determine the 95th percentile pressure and one night of Autoset in the sleep laboratory.

Recommendations:
- Using fixed pressure CPAP when the effective pressure has been determined by autotitrating CPAP at home is at least as effective as CPAP which has been set with manual titration, in improving sleepiness and quality of life.
- There is no consistent patient preference for autotitrating CPAP or fixed CPAP.

Factors that may affect CPAP adherence

Recommendation:
- Using fixed pressure CPAP, when the effective pressure has been determined by autotitrating CPAP at home, is associated with similar adherence with CPAP as occurs when CPAP has been set with manual titration [51,52].

Conclusion

Using autotitrating CPAP at home for at least three nights is an option for determining the effective level of CPAP for patients planning to use fixed level CPAP in the long term. It is not possible to recommend either home titration of CPAP with an autotitrating pump or laboratory polysomnography to titrate CPAP over the other.

Home CPAP autotitration is not suitable for those who may have any of the following:
- central sleep apnoea,
- significant hypopnoea during sleep (due to conditions other than OSA),
- significant accompanying sleep disorders in addition to OSA or
- co-morbidities that limit home titration.

The recommended fixed pressure for long term use is the 95th or 90th percentile pressure achieved by the autotitrating pump as per the manufacturers’ instructions. It should be recognized that even after several nights or weeks of autotitration there can still be some doubt about the amount and type of sleep that the patient had during this period and how valid the recommended pressure might be. Thus, the patient should be reviewed soon after using CPAP with a fixed pressure level based on the level obtained from the autotitrating device. The clinical response should be assessed especially if there is persisting sleepiness or snoring. If there are any concerns about the clinical response the patient may need to have a sleep study using CPAP in a sleep laboratory.

In addition, the clinician should discuss the available options with each patient and take into consideration each patient’s preferences. Manufacturers are often updating their devices and releasing new models, so even when there are published evaluations of pumps these evaluations do not necessarily apply to pumps being currently marketed.

1.3.2.14. Diurnal sleep studies. Diurnal and nocturnal titration result in comparable therapeutic pressures, equivalent resolution of sleep disordered breathing and improvement in subjective sleepiness after 1–12 weeks of treatment, particularly for patients with severe OSA [27].

1.3.2.2. Sleep studies to assess efficacy of oral appliance therapy for OSA. Follow-up sleep testing is not indicated for patients with primary snoring [54].

Current guidelines [54] suggest all patients with OSA should undergo a follow up type 1 or type 3 sleep study. This is because the rate of treatment success is not predictable with oral appliances. Additionally, some patients experience an increase in AHI with oral appliance treatment.

This strategy may be questioned with a recent publication demonstrating in the subgroup of patients with mild and moderate OSA, a mean residual AHI less than ten events/hour was achieved [117]. In this study, only one type of specific oral appliance was utilised and results may not be applicable to the range of oral appliances available. The accompanying editorial [118] suggests for patients with mild to moderate OSA where symptoms are controlled no further testing is probably required.

Follow up however is especially important in patients with an elevated ODI 4%, severe OSA or patients with excessive daytime sleepiness.

1.3.2.3. Intervention studies for nasal expiratory airway pressure devices for OSA. Given that this technology has only recently been introduced into clinical practice, it is recommended that all patients treated with this device for OSA undergo sleep studies to assess its efficacy. An exception to this may be simple snoring where there has been reported good symptomatic benefit.
1.3.2.4. Sleep studies to assess the efficacy of behavioural strategies. The major behavioural treatment options include weight loss and positional therapy.

After substantial weight loss (i.e. 10% or more of body weight), a sleep study is routinely indicated to ascertain whether PAP therapy is still needed or whether adjustments in PAP level are necessary [8]. Autotitrating CPAP study may be another suitable way to reassess the effect of weight changes on pressure levels.

Because not all patients normalize AHI when non-supine, correction of OSA by positional therapy, should be documented with PSG before initiating this form of treatment as a primary therapy [56].

1.3.2.5. Sleep studies to assess efficacy following upper airway surgery for OSA patients. Given the heterogeneity of response to this type of therapy [57], it is recommended that polysomnography be performed to assess outcome.

1.3.2.6. Intervention studies of oxygen therapy. A sleep study is not necessary solely for the purposes of establishing a patient with COPD on home O2 treatment [2]. This decision is usually made on the basis of wakeful PaO2 [58]. There is no evidence that isolated nocturnal desaturation causes progressive pulmonary hypertension [59] and one study has shown no significant treatment effect on survival or pulmonary haemodynamics from nocturnal oxygen therapy in such patients [60].

With respect to OSA, oxygen supplementation is not recommended as a primary treatment [61]. If supplemental oxygen is used as an adjunct to other primary therapies to treat hypoxaemia, follow-up must include documentation of resolution of the hypoxaemia [54].

1.3.2.7. Intervention studies of NIV therapy for sleep disorders of breathing

1.3.2.7.1. Chronic alveolar hypoventilation (CAH) syndromes. The Noninvasive Positive Pressure Ventilation (NPPV) Titration Task Force of the American Academy of Sleep Medicine recommends the following [62]:

“NPPV titration with polysomnography (PSG) is the recommended method to determine an effective level of nocturnal ventilatory support in patients with CAH. In circumstances in which NPPV treatment is initiated and adjusted empirically in the outpatient setting based on clinical judgement, a PSG should be utilised if possible to confirm that the final NPPV settings are effective or to make adjustments as necessary.”

1.3.2.7.2. Disorders requiring servo-adaptive ventilators. Given the relative inexperience with this form of therapy, limited long term outcome data and the fact that adjustments in end expiratory pressure settings to stabilise the upper airway may be required, the committee recommends that a Type 1 study is indicated in the titration and assessment of sleep indices with this form of therapy. More studies are required to judge whether simpler forms of monitoring are sufficient to predict long term success with this therapy.

1.3.3. Follow-up studies

Where treatment for a sleep-related breathing disorder has been successfully instituted, it is important to ensure its long-term efficacy. Objective assessments of long term treatment adherence and breathing stability (e.g. microprocessor-based CPAP compliance, AHI and leak meters) and validated measures of effectiveness (e.g. Epworth Sleepiness Scale score, general or disease-specific quality of life measurements) are highly desirable.

Routine follow-up sleep studies are not necessary in patients who have experienced a reversal of symptoms and are stable. However, weight change (greater than 10% in either direction) or the recurrence of snoring or daytime sleepiness on CPAP or oral appliances may indicate the need for repeat diagnostic and/or therapeutic studies.

Persistence of daytime sleepiness despite optimal use of CPAP is common [111]. In the quoted study, 32% of patients with an initially elevated Epworth Sleepiness Scale score had a persistently elevated score three months later despite objective CPAP adherence of >5 h/night. Additional causes of sleepiness such as depression, diabetes mellitus, obesity per se, medications and sleep restriction need to be considered.

2. Movement and behavioural disorders of sleep

2.1. Parasomnias

These conditions are undesirable physical events or experiences that occur during entry into sleep, within sleep or during arousals from sleep. Such conditions include: disorders of arousal from NREM sleep (confusional arousals, sleepwalking and sleep terrors, sleep-talking), REM Sleep Behaviour Disorder, nightmares and bruxism.

A clinical evaluation of the parasomnia with emphasis on age of onset, time of event relative to sleep onset, frequency, regularity and duration of event is often sufficient to diagnose common, uncomplicated, non-injurious parasomnias without the need for polysomnography [8].

2.2. Seizure disorders

Epilepsy is a chronic condition characterised by the occurrence of paroxysmal electrical discharges in the brain and manifested by changes in consciousness, motor control or sensory function. The term ‘sleep related seizure disorder’ encompasses conditions with recurrent seizures during sleep.

Indications for in lab polysomnography with parasomnias and sleep related seizure disorders: [8]

Polysomnography with an extended bilateral montage and video monitoring is recommended to assist with the diagnosis of parasomnias or other sleep disorders that are thought to be seizure-related when the initial clinical evaluation and results of standard EEG are inconclusive.

Polysomnography, with additional EEG derivations and video recording, is indicated in evaluating sleep related behaviours that are violent or otherwise potentially injurious to the patient or others.

Polysomnography is indicated when evaluating patients with sleep behaviours suggestive of parasomnias that are unusual or atypical because of the patient’s age at onset; the time, duration, or frequency of occurrence of the behaviour; or the specifics of the particular motor patterns in question (e.g., stereotypic, repetitive, or focal).

Polysomnography may be indicated when the presumed parasomnia or sleep related seizure disorder does not respond to conventional therapy.

Technical Factors related to sleep studies [8]

In digital EEG recordings, the sampling rate must be adequate to identify brief paroxysmal discharges.

The minimum channels required for the diagnosis of parasomnia or sleep-related seizure disorder include sleep-scoring channels (EEG, EOG, chin EMG); EEG using an expanded bilateral montage; and EMG for body movements (anterior tibialis or
extensor digitorum). Synchronised audiovisual recording and documented technologist observations during the period of study are also essential.

Interpretation of polysomnography with video and extended EEG montage requires skills in both sleep medicine and seizure recognition. It is essential that a polysomnographer, sleep physician or neurologist experienced in seizure recognition be assigned to these studies. Where none is available, appropriate consultation is sought or the patient referred to a centre with the appropriate expertise.

2.3. Restless legs syndrome and periodic limb movements

Restless legs syndrome (RLS) is a sensorimotor disorder characterised by a complaint of irresistible urge to move the legs. Periodic Limb Movement Disorder (PLMD) is characterised by periodic episodes of repetitive, highly stereotyped periodic leg movements and accompanied by clinical sleep disturbance that cannot be accounted for by another primary sleep disturbance. Periodic limb movements during sleep often accompany RLS. Although PLMD can exist independent of RLS, it is estimated that 80.2% of individuals with RLS have evidence of PLMS on PSG [96].

Polysomnography is not routinely indicated to diagnose or treat restless legs syndrome, except where uncertainty exists in the diagnosis.

The evaluation should include a clinical history and physical examination. Special emphasis on complaints of leg discomfort, the occurrence of leg or body jerks, restless sleep, and reports of insomnia or excessive daytime sleepiness should be sought. The clinical history should include bed partner observation, if possible. Physical examination should focus on excluding a peripheral neuropathy that can mimic RLS. Serum ferritin, complete blood count, urinalysis, and biochemistry testing should be undertaken to look for secondary causes of RLS (e.g., iron deficiency anaemia, uraemia).

The validated NIH criteria can be used to establish the diagnosis of RLS [97]. The validated RLS rating scale [98] can be used to establish severity of patients’ symptoms; this scale may be useful in directing treatment as well.

Indications for polysomnography [8]:

1. If uncertainty exists about the diagnosis of restless legs syndrome.
2. Polysomnography is indicated when a diagnosis of periodic limb movement disorder is considered, without concomitant RLS.
3. If a concomitant sleep disorder, e.g., OSA is suspected.

Technical considerations:

The minimum channels required for the evaluation of periodic limb movements and related arousals include EEG, EOG, chin EMG, and left and right anterior tibialis surface EMG. Respiratory effort, airflow, and oximetry should be used simultaneously if sleep apnoea or upper-airway resistance syndrome is suspected to allow a distinction to be made between inherent periodic limb movements and those limb movements associated with respiratory events.

Intra-individual and a night-to-night variability exists in patients with periodic limb movement disorder, and a single study might not be adequate to establish this diagnosis.

3. Non respiratory disorders of excessive daytime sleepiness

3.1. Diagnosis of narcolepsy

The diagnosis of narcolepsy may be made confidently by history alone only when daytime hypersomnolence and classical cataplexy symptoms are present. The latter symptom is highly specific for this disease. However, sleep studies (PSG and multiple sleep latency test (MSLT)) are an invaluable adjunct to diagnosis particularly in cases in which a history of cataplexy is absent or equivocal. Additionally because this condition is lifelong and its diagnosis may have significant implications for driving and vocational choices, and medications to treat this disorder may carry significant risk, objective testing is highly desirable. PSG is used primarily to exclude other causes of excessive daytime sleepiness (e.g., OSA), and is also traditionally employed as part of the MSLT protocol to confirm that the patient had sufficient sleep the night prior to the MSLT. The MSLT findings of a mean sleep latency of less than 8 min and two or more sleep onset REM periods, in the absence of a history of chronic sleep restriction or acute sleep deprivation, or absence of another sleep disorder is consistent with a diagnosis of narcolepsy. The combination of a mean sleep latency of less than 5 min and two or more REM onset sleeps on MSLT is reasonably sensitive and specific for narcolepsy but still cannot be relied on alone for the diagnosis [99]. Following careful history taking and sleep testing (PSG and MSLT), there remains diagnostic uncertainty, CSF hypocretin levels might be considered [100]. In atypical cases, brain MRI may be useful to rule out structural lesions mimicking the condition.

3.2. Diagnosis of primary hypersomnia and other disorders leading to hypersomnolence

The diagnosis of primary hypersomnia is one of exclusion. PSG is therefore required to rule out common sleep disorders such as those that can lead to hypersomnolence. An assessment of sleep–wake schedules using a sleep diary with or without actigraphy over a 2–3 week period can also be helpful to exclude chronic sleep restriction. MSLT should be performed to objectively confirm the presence of hypersomnolence. Other rare disorders such as Prader–Willi Syndrome, Myotonic Dystrophy and Kleine–Levin Syndrome are associated with pathological sleepiness (and in the instance of the first two disorders, may be associated with sleep onset REMs) and may thus enter the differential diagnosis of idiopathic hypersomnia or narcolepsy. However, these conditions are usually readily identified by careful clinical history and examination, supplemented by genetic testing in some instances.

Depression

It is also important to recognise that depression is frequently associated with reports of daytime fatigue and sleepiness. Patient reported sleepiness is better predicted by the presence of depression than by AHI in both general and sleep clinic populations [114–116]. Clinical review of the patient before and after OSA diagnosis and treatment should therefore include an assessment for an underlying depressive disorder.

3.3. Quantification and verification of excessive daytime sleepiness for management purposes

Objective tests of daytime sleepiness such as MSLT and the maintenance of wakefulness test (MWT) are not recommended routinely for the management of patients with sleep disorders. Response to treatment can usually be judged clinically and with the assistance of validated questionnaires such as the Epworth Sleepiness Scale. However, where there is reason to suspect this type of assessment is unreliable (e.g. over reporting or under reporting of symptoms by patients), and it is important to have
a clear idea of the level of daytime impairment for management (e.g. driver licensing or medicolegal purposes), tests such as the MSLT and MWT may be useful. The MWT has a stronger a priori rationale as a test of daytime alertness, and may therefore be more relevant than the MSLT to assess occupational and driving safety. There can be considerable discrepancy between MSLT and MWT findings in the same subject [101] suggesting that the two tests provide different information about sleepiness or sleep propensity.

3.3.1. Multiple sleep latency test
The MSLT provides an objective measure of the ability or tendency to fall asleep. Four to five evenly spaced 20-min daytime nap opportunities with the patient lying in a quiet darkened room are provided and the time to sleep and REM sleep onset (if any) is quantified from EEG/EOG and EMG recordings. The patient is instructed to attempt to fall asleep and the mean sleep latency result is taken to be indicative of sleep propensity. The reader is directed to a recent American Academy of Sleep Medicine publication for a detailed description of the performance of the MSLT, reporting of results and test interpretation [7]. Measurement of respiratory parameters is not generally indicated in an MSLT. Sleep diaries or actigraphy prior to MSLT may be helpful in interpretation. The urine drug screen is usually performed in the morning but its timing and circumstances may be altered by the clinician.

Where an MSLT is conducted on a patient known to have a respiratory sleep disorder and using treatment, the test should be conducted with the patient using treatment, for example using CPAP. If this is not done there is a risk of prolonging sleep latency through the occurrence of respiratory events at sleep onset.

3.3.2. Maintenance of wakefulness test
The MWT provides an objective measure of the ability to stay awake for a defined time. This test consists of four evenly spaced 40-min test periods in the daytime during which the patient is asked to resist sleep while sitting comfortably in an armchair in a darkened room. Patients are not allowed to take extraordinary measures to stay awake (such as slapping, drinking or singing) during each 40 min trial. The patient must not engage in any activities prior to or during these test periods that may increase arousal levels. EEG/EOG and EMG are measured and the latency to sleep onset (if any) is quantified. The test has a stronger a priori rationale as a measure of a patient's daytime vigilance or ability to resist sleep than the MSLT. The reader is directed to the most recent American Academy of Sleep Medicine publication for a detailed description of the performance of the MWT, reporting of results and test interpretation [7]. Measurement of respiratory parameters is not generally indicated in a MWT. There are generally fewer normative data for the MWT than the MSLT but some normative data for the Australian population are available [102]. Drug screening may be indicated to ensure that sleepiness/wakefulness on the MWT is not influenced by substances other than medically prescribed drugs. The urine drug screen is usually performed in the morning but its timing and circumstances may be altered by the clinician.

3.3.3. Osler test
The Osler test [103] is essentially the same as the MWT with the exception that sleep onset is determined from psychomotor performance rather than EEG/EOG and EMG (i.e. absence of button pressing response to a light presented at frequent regular intervals). Mean sleep latency using this test agrees closely with MWT results [104] obtained in the same patients with sleep disorders. There is less experience in Australia and New Zealand with this test than MSLT and MWT, and fewer published reports of normal results. It has the potential advantage that it can be performed in centres that do not have a sleep laboratory. It may allow some cost savings when performed in a sleep laboratory because of reduced requirements for technician EEG real time observation and subsequent scoring.

4. Insomnia and disorders of insufficient sleep

4.1. Indications for sleep study
This is no evidence to support the routine use of PSG in the assessment of patients with insomnia [105] or circadian sleep disorder [106]. However, if there is a history suggestive of sleep disordered breathing, PLMD or complex parasomnias that might be contributing to prolonged sleep latency or disrupted sleep patterns, polysomnography should be considered. However it should be recalled that approximately 30% of patients have concomitant OSA and insomnia [109].

4.2. Sleep diary and actigraphy
Systematic reviews of subjective estimates of sleep and bed times made over days or weeks by patients who present with complaints of insomnia can be invaluable in assessing the nature of their insomnia (e.g. distinguishing delayed or advanced phase insomnia from psychophysiological insomnia) and can be useful in following response to treatment interventions. Sleep and bed times assessed objectively with 24-h actigraphy measurements may be used also in special circumstances to corroborate the subjective sleep reports or point to a possible problem of sleep misperception. Actigraphy usually includes estimates of light and dark exposure across the day night period.

5. Measurement techniques for sleep studies

5.1. Preparation and instructions to patients prior to study
The preparation and education of the patient prior to the study enhances the quality of data obtained. Results of studies performed while the patient is acutely unwell, such as early during an inpatient admission may be obscure the true nature of sleep disordered breathing. For example, studies performed while the patient is in respiratory failure may provide information for acute management but should not be used as a basis to establish the patient on long term treatment. Similarly, patients with COPD are likely to demonstrate more sleep hypoxaemia during an acute exacerbation of their disease.

The use of alcohol, sedatives and hypnotics immediately prior to the study may exaggerate an underlying problem with obstructive sleep apnoea, nevertheless patients chronically using such medications may experience rebound insomnia on the night of the sleep study if they are withheld. It is the responsibility of the clinician ordering the test to decide whether it should be performed following the withdrawal of aggravating drugs or after appropriate treatment of underlying disease. The decision is likely to hinge on the particular question to be answered. In some instances, studies under both circumstances may be desirable. At the very least, the physician reporting the test should be fully aware of the condition of the patient at the time of sleep study and interpret the result accordingly.

Patients should be instructed to follow normal activities of daily living prior to presentation for the study. Unless the study is being performed for a special purpose, patients should maintain their regular sleep habits prior to the study.
5.2. Polysomnography

5.2.1. General
Respiratory sleep studies should employ, whenever possible, non-invasive methods for evaluating sleep, respiratory and cardiac function. A complete and permanent record of the study should be made and a written report issued (see Laboratory Report).

5.2.2. Signal recording
Detailed guidelines for standardised signal recording methodology can be found in the AASM Manual for the Scoring of Sleep and Associated Events 2012 [119]. The ASTA/ASA commentary 2010 [18] provides interesting background reading on the earlier version of the AASM manual, but should not be used as the current methodology guideline. The ASA recommends that sleep laboratories adopt the recommended technical specifications (for adults) for signal recording (Visual, Cardiac and Respiratory) as published in the AASM 2012 Manual [119].

5.2.3. Other measurements
Other measurements may be incorporated into the sleep study to investigate specific disorders, such as oesophageal pH monitoring for gastro-oesophageal reflux. These should be considered as adjuncts to those outlined above.

Other protocols may be required for patients presenting to sleep clinics that use variables referred to in this section. Examples are the Multiple Sleep Latency Test and Maintenance of Wakefulness Test, which are used to objectively assess the degree of daytime sleepiness (see Section 3).

5.2.4. Calibration of measurements

Physical calibration

Calibration of quantitative instruments against appropriate standard reference values, where the absolute value is important, must occur at regularly scheduled intervals. Calibration of the electrical output signal of a quantitative instrument will also be necessary when, as is usually the case for PSG, the signal is recorded by a digital PSG recording system. For continuous signals (e.g. SpO2, PtcCO2, CPAP pressure, sound level, EEG), a minimum of two points must be performed that span the expected range of the measurement. In the case of level signals (such as body position and room light) calibration of each level must be performed.

The frequency of calibration depends on the stability of the transducer and the likelihood that an intrinsic or extrinsic factor could cause an error in the value reported. If the signal is critical to the interpretation of the study, for example SpO2 or CPAP pressure, it should be checked prior to each study.

Accurate determination of sleep stage requires measurement of the amplitude of the EEG signals and hence the gain of EEG amplifiers should also be calibrated. In most systems the gain is stable over a long period of time and monthly calibration of these amplifiers is adequate. The accuracy of high and low pass filter settings of all high-frequency amplifiers should also be checked.

Technical and digital specifications for PSG recording are stated in the AASM scoring manual [119]. The ASA recommends adoption of the technical and digital specifications for routine PSG recordings (including impedances, digital resolution, sampling rates and filter settings) as published in the AASM 2012 Manual [119].

Biological checks:

Biological checks are an important adjunct to physical calibration methods and apply to signals and phenomena for which there is no primary reference. Such checks include –

- Eyes closed/eyes open EEG.
- Eye movements (vertical, horizontal, blinks)
- Submental muscle activation.
- Respiration (nasal/oral flow, abdominal/thoracic movement)
- Limb movements.
- Biological checks must be performed at the beginning of each PSG.

Physical calibration checks and all biological checks should be appropriately labelled and permanently recorded along with the PSG to provide confirmation of signal accuracy and integrity.

5.2.5. Quality control
Information regarding quality management systems is contained within the current ASA/NATA document [108].

5.2.6. Data storage

The PSG record should be complete (allowing full disclosure of the raw data) and a copy retained. An appropriate data backup and recovery regime should be in place to guard against data loss.

5.3. Scoring and reporting

Detailed guidelines for standardised event definitions and scoring methodology can be found in companion documents AASM 2007 [17], AS/ASA commentary 2010 [18]. Further guidelines have been published by the AASM in 2012 [113,119].

The ASA recommends adoption of the adult scoring rules for sleep stage arousals, cardiac events, PLMS and respiratory events as published in the AASM 2012 Manual [119]. Definitions of Respiratory Events including apnoeas, hypopnoeas, respiratory effort-related arousals (RERA), hypoxia and Cheyne-Stokes breathing must be adopted without modification [119]. Hypopnoeas must be scored using the AASM recommended definition. Scoring of hypopnoeas as obstructive or central is optional [119].

General guidance regarding the parameters to be reported for Polysomnography has been published in the AASM 2012 Manual [119].

5.3.1. Severity criteria for OSA [13,54]

5.3.1.1. The apnoea – hypopnoea index (AHI)

The AHI has been used in clinical trials and epidemiological studies to classify delineate patients as having OSA and to classify the severity of OSA.

The Chicago criteria [16] recommend the following classification of OSA severity:

- Normal: AHI <5 events per hour of sleep.
- Mild OSA: AHI 5 < 15 events per hour of sleep.
- Moderate OSA: AHI 15 < 30 events per hour of sleep.
- Severe OSA: AHI >30 events per hour of sleep.

The use of an event frequency of 5 per hour as a minimum value to diagnose OSA was based on epidemiological data that suggest it may be associated with measurable health effects such as sleepiness, motor vehicle accidents and hypertension [89,90]. The latter risk appears substantial at 30 events per sleep hour. In addition, intervention studies suggest treatment of subjects with between five and 15 events per hour of sleep relieves sleepiness and may improve neurocognitive function [91—93].

This AHI grading needs to be used with caution in describing severity of sleep disordered breathing in present day laboratories. The research studies underpinning the Chicago recommendations on OSA severity used oronasal thermistors and respiratory inductance plethysmography to score respiratory events and in general, most definitions of hypopnoea incorporated a 4% arterial oxygen
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neither sensitive nor specifically correlated with oxygen desaturation but not an arousal. However, most clinical laboratories now use nasal pressure, a more sensitive index of airflow, to detect sleep disordered breathing events. Many laboratories have adopted the definition of hypopnoea which includes arousals and desaturation but not an arousal. However, most clinical laboratories now use nasal pressure, a more sensitive index of airflow, to detect sleep disordered breathing events. Many laboratories have adopted the definition of hypopnoea which includes arousals and does not mandate a 4% fall in arterial oxygen saturation but accepts a 3% fall in arterial oxygen saturation. Thus, studies will now generally be scored with higher AHI values than would have been scored in the original studies defining syndrome severity, but lower than those scored using Chicago criteria. Clinicians using the above criteria should adjust the AHI cut-offs based on the best available evidence (preferably data obtained from their own laboratory comparing previous and present day recording and scoring methods) [112].

5.3.1.2. Daytime sleepiness. An assessment of sleepiness severity should be undertaken in all patients using the Epworth Sleepiness Scale (ESS) [94], or another validated questionnaire to assess daytime sleepiness. It should be undertaken in all patients using the Epworth Sleepiness Scale (ESS) [94], or another validated questionnaire to assess daytime sleepiness. The Respiratory Disturbance Index (RDI) is defined as the AHI plus the RERA Index, and is optional for inclusion in reports of sleep studies [119]. There are no data which have validated the use of the RDI in describing the severity of sleep disordered breathing, and this Index should be used with caution as described in the preceding paragraph.

5.3.1.3. Sleep hypoxaemia. Severe oxygen desaturation has been classified as an oxygen saturation <85% for more than 2% of the total PSG sleep time, while minimal or no desaturation has been classified as no PSG sleep saturation less than 90%. Severe oxygen desaturation as defined by total sleep time with oxygen saturation of <85% is weakly associated with a reduction in some neurocognitive performance measures [95].

An oxygen saturation of <85% for more than 5% of sleep time (breathing air) has been suggested [16] as another threshold value defining severe sleep-related hypoventilation.

The nadir oxygen saturation during sleep has not been demonstrated to be an independent predictor of any short or long term cardiovascular or neurocognitive outcome, and is not considered to be a useful severity index.

5.3.1.4. Further considerations. The above are useful guidelines for grading the severity of sleep disordered breathing and its cardinal manifestation, daytime sleepiness. However, the clinician, in assessing the likely importance of disease and potential benefits of treatment in an individual patient, must be alert to specific aspects of the patient’s sleep study and other pertinent clinical findings. An assessment of the severity of sleep disordered breathing in clinical practice should include:

i) Careful evaluation of the PSG pattern of sleep disordered breathing. In general the higher the AHI the more severe the sleep apnoea, but important sleep-disordered breathing may be present with low AHI values. For example, RERAs are not included in the AHI, yet may be contributing to adverse clinical outcomes. Sleep disordered breathing events may be confined to the supine position and/or REM sleep. If supine sleep and/or REM sleep are under represented on the study night the computed AHI will underestimate the severity of the underlying OSA. The reader should note that supine sleep can potentially be over-represented on the PSG night. If obstructive events are unusually long, desaturation may be severe but AHI low. The level of oxygen desaturation is worthy of independent consideration particularly in the context of co-existing respiratory or cardiovascular disease and obesity. Thus, the reporting physician should carefully review sleep study findings including raw data, and a qualitative interpretation should be provided along with AHI and other data.

ii) The patient’s clinical circumstances. Marked daytime sleepiness with a low AHI may indicate that the severity of sleep disordered breathing has been underestimated by the sleep study, but equally, alternate or additional causes for the sleepiness may be present, such as poor sleep hygiene, chronic sleep restriction, sedating drugs, depression or periodic limb movements of sleep. The impact of the sleepiness should also be considered. For example, sleepiness in a long distance bus or truck driver will be of particular importance. There is emerging literature that suggests that moderate-severe OSA with elevated 4% Oxygen Desaturation Index is an independent risk factor for cardiovascular disease. Thus, the presence of co-existing cardiovascular risk factors or disease in a patient may influence the clinician to advise treatment at a lower AHI severity cut off than might normally be the case [54].

5.3.2. The laboratory report for type 1 and 2 sleep studies
A written report should be issued at the completion of all sleep studies detailing:

a) the variables measured.

b) sleep staging, including total sleep time, sleep efficiency, wake after sleep onset, sleep latency, stage R sleep latency, percentage of time in the various sleep stages, and frequency of arousals.

c) frequency and type of abnormal respiratory events (e.g. central or obstructive).

d) relationships of disordered breathing to posture sleep stage or treatment intervention when relevant.

e) oxygen saturation, described in quantitative terms using either a continuous saturation versus time plot or by using discrete intervals (e.g. sleep time spent within various ranges of saturation). The lowest saturation recorded during abnormal respiratory events should be noted.

f) transcutaneous PCO2 (PtcCO2) trends, where measured.

g) any disturbance of cardiac rate or rhythm, and its relationship to abnormal respiratory events, if measured. Include mean heart rate as sleep.

h) the frequency of periodic limb movements and any associated sleep fragmentation.

i) medications (including sedatives) and alcohol that may have influenced the results.

j) technical comments

k) scoring definitions used, and supporting references

l) the physician’s interpretation/conclusions should provide a summary of the relevant normal and abnormal findings from a review of the raw study data together with the above summary data, including comments on sleep staging, respiratory scoring, cardiac abnormalities, any abnormal behaviours or movements, and effectiveness of any applied therapy. The conclusion should provide a clear diagnosis and severity rating for diagnostic studies, and only make relevant recommendations regarding therapy from intervention studies.

The committee recommends a minimum of 12 min per study is required for the sleep physician to assimilate the clinical data, review the raw sleep study data, take into consideration technicians’
The respiratory event index (REI) is defined in the context of OOC testing devices as:

\[ \text{REI} = \frac{[\text{apneas} + \text{hypopneas}]}{\text{Total sleep or recording time (h)}} \]

Fig. 6. Reference [85]. Republished with permission of [journal of clinical sleep medicine: JCMS: official publication of the American Academy of Sleep Medicine], from [Obstructive sleep apnea devices for out-of-center (OOC) testing: technology evaluation. Collop NA et al. 7(5):531–548. 2011]; permission conveyed through Copyright Clearance Center, Inc.

comments and observations and prepare a report. For many studies a longer length of time will be required.

5.3.3. The laboratory report for type 3 and 4 sleep studies

Type 3 & 4 study reports should include the following:

1. Type of device used
2. Technical adequacy of test
3. Date of testing
4. Duration of test recording
5. Respiratory Event Index (REI) and total number of respiratory events. The criteria used for apneas & hypopneas should be defined (Fig. 6).
6. A summary of oxygen desaturations during recording period
   • which may be ODI
   • % of time below certain thresholds
   • Mean \( O_2 \) saturation
   • minimum and/or maximum \( O_2 \) saturations
7. Heart rate during the recording period
8. Interpretation (based upon test results and clinical information), including at a minimum whether the test results support a diagnosis of obstructive sleep apnea or not
9. Signature of interpreting sleep physician

The reader is directed to recent systematic reviews [83,85] for guidance on those specific devices that have been demonstrated to have diagnostic utility and those that have not. These reviews and the source references provide information on the respiratory parameters measured in each device. A companion paper provides guidance on how to assess the quality of studies on limited channel devices. In principle, statements concerning transducers and instrumentation made relevant to type 1 and 2 studies also apply to type 3 and 4 studies.

6. Sleep laboratory facilities and personnel requirements

Appropriate standards for sleep laboratory facilities and personnel are detailed elsewhere, in the “Accreditation of Sleep Disorders Services” document published jointly by the ASA and NATA [108]. These standards address requirements regarding: organization and administration; staffing and direction; policies and procedures; staff development, teaching and research; facilities; provision for emergencies; quality assurance; meetings; and the policies and procedures manual.

Conflict of interest

None declared.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2017.03.019.

Appendix

The following conflicts or potential conflicts of interest were declared by members of the committee.

1. Current or recent (last three years) involvement with company or companies with a financial interest in devices or methods for performing sleep studies
   a. Direct financial interest (Nil for JD, AN, JW, DM, CW, PR, CC-C, MN)
   b. Employee, or engaged in a consulting capacity (including medical advisory boards, expert testimony) (Nil for JD, AN, JW, DM, CW, PR, C-C, MN)
   c. Substantial research support (Nil for JD, AN, JW, DM, CW, PR, CC-C, MN)
   d. Sponsored attendance at national or international meetings (Nil for JD, AN, JW, DM, CW, PR, C-C, MN)

2. Direct or indirect financial benefit has been received from performing or reporting type I & 2 sleep studies (Yes — JD, AN, JW, DM, CW, C-C, MN, PR).

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Australasian Sleep Association clinical practice guidelines for performing sleep studies in children

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1. Executive summary

This clinical practice guideline, intended for use by paediatric respiratory and sleep practitioners, provides best practice recommendations for conducting paediatric sleep studies in Australia and New Zealand; it is applicable for children up to the age of 18 years. In particular this guideline provides recommendations in relation to the investigation of sleep-related respiratory disorders; non-respiratory causes of excessive daytime sleepiness; sleep-related movement disorders and the occurrence of abnormal or unusual behaviours during sleep. Clinical indications for diagnostic, interventional or follow-up sleep studies including the multiple sleep latency test (MSLT) are outlined. The recommendations specified in this document represent the consensus view of a committee of experienced paediatric sleep practitioners selected by the Australasian Sleep Association (ASA) and will require regular revision and modification as sleep medicine and technology continues to evolve.

Standards and indications for polysomnography (PSG) in children were first published by the American Thoracic Society [1] and more recently by the American Academy of Sleep Medicine [2]. These documents outline evidence-based recommendations regarding technical aspects of sleep study recording and clinical situations where polysomnography is or is not indicated. These publications were considered by the panel when formulating recommendations in the current document and where necessary modifications have been made for Australasian practice. Where available, randomised controlled trials have been cited to substantiate recommendations.

This clinical practice guideline is not intended as the only source of guidance in the assessment of sleep disorders in children. In particular it is noted that for all types of sleep studies the laboratory investigation is only one component of diagnosis. Clinical history and examination are as important and complementary to the sleep study. Treatment decisions should not be based on a test result alone. However, as is the case for any diagnostic test, it is crucial that the necessity and urgency of the test, and the subsequent results, are evaluated by a clinician with the necessary expertise and knowledge to interpret the findings accurately.

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Summary of Recommendations

This clinical practice guideline makes the following major recommendations in relation to undertaking sleep studies in children: Obstructive Sleep Apnoea

- PSG is indicated when clinical assessment suggests the diagnosis of obstructive sleep apnoea syndrome (OSA).
- Abbreviated testing is indicated in selected cases as a clinical adjunct tool for the assessment of children with suspected obstructive sleep apnoea (OSA) following paediatric sleep specialist review.
- PSG is indicated if clinical assessment suggests OSA and abbreviated testing is inconclusive for the diagnosis of obstructive sleep apnoea.
- PSG is indicated when the severity of OSA is in doubt or if there is significant risk of post-surgical complications (e.g., idiopathic thrombocytopenic purpura).
- PSG is indicated when clinical assessment suggests the presence of residual OSA following AT or other treatment modalities.
- PSG is indicated following AT in children where there is the presence of ongoing symptoms or other clinical features suggestive of persistent OSA, particularly in those with: moderate to severe OSA pre-operatively, obesity, craniofacial abnormalities that predispose to the development of upper airway obstruction, neurologic disorders (e.g., neuromuscular disorders, spina bifida, Chiari malformation, meningomyelocele) and high risk congenital conditions (e.g., Down Syndrome, Prader–Willi Syndrome). See Appendix 1.

Central Sleep Apnoea and Non-Obstructive Hypoventilation

- PSG is indicated if clinical evaluation or abbreviated testing suggests the presence of central apnoea that is: (i) unexpected for development/age (ii) of prolonged duration or (iii) associated with significant oxygen desaturation or frequent bradycardia, pallor change, sleep disturbance or other daytime sequelae.
- PSG is indicated in cases of apnoea of infancy that are unresponsive to treatment or unusually severe or persistent or if there is diagnostic doubt over the aetiology of events (i.e. obstructive versus central).
- PSG is indicated when clinical assessment suggests the diagnosis of Congenital Central Hypoventilation Syndrome (CCHS).
- PSG or abbreviated testing is indicated when clinical assessment or other investigations (e.g., pulmonary function tests) suggests the presence of non-obstructive hypoventilation due to neuromuscular disease (NMD), obesity, kyphoscoliosis or chest wall deformity.
- PSG or abbreviated testing is indicated pre-operatively in at-risk children with neuromuscular disorders undergoing major orthopaedic, thoracic or upper airway surgery to screen for undetected SDB which may be exacerbated by sedation or analgesics.

Polysomnography for Children Receiving Respiratory Support

- PSG is indicated for children receiving positive airway pressure support (CPAP or BPAP) to titrate the level of pressure support required and to periodically re-evaluate the appropriateness of settings, including synchrony between machine and patient.
For Personal Use Only

Therapy

Hypersomnia and Narcolepsy

Parasomnias and Epilepsy

Children with underlying chronic lung disease

Prader-Willi Syndrome (PWS) and Growth Hormone (GH) Therapy

Apparent Life Threatening Event (ALTE)

PSG is not indicated for infants as part of a routine evaluation for an uncomplicated ALTE.

PSG is indicated for infants who have experienced an ALTE and where there is clinical suspicion of underlying sleep disordered breathing or if there is ongoing or unexplained oxygen desaturation.

Prader-Willi Syndrome (PWS) and Growth Hormone (GH) Therapy

PSG is indicated for children with PWS prior to and six to ten weeks following the commencement of GH treatment and thereafter as clinically indicated.

Children with underlying chronic lung disease

PSG is indicated for children with chronic lung disease (e.g., asthma, cystic fibrosis, bronchopulmonary dysplasia) or unexplained pulmonary hypertension if there is clinical suspicion of sleep-disordered breathing.

Movement Disorders

PSG is not routinely indicated to diagnose restless legs syndrome (RLS), with the diagnosis based on clinical features.

PSG is indicated for children suspected of having periodic limb movement disorder (PLMD).

Parasomnias and Epilepsy

PSG is not indicated if clinical assessment is consistent with a typical childhood parasomnia.

PSG is indicated if a parasomnia is atypical or if excessive daytime sleepiness or impaired daytime functioning is present that cannot be explained by other causes.

Hypersomnia and Narcolepsy

The MSLT preceded by overnight PSG is indicated in children as part of the evaluation for suspected narcolepsy and excessive daytime sleepiness.

2. Introduction and background

The knowledge and practice of paediatric sleep medicine has expanded markedly over the past few decades. As a result there has been a concomitant increase in demand for the clinical investigation of sleep disorders, particularly with respect to overnight polysomnography (PSG). Polysomnography (also known as a Type 1 study) is the reference standard employed to diagnose and evaluate a range of sleep disorders and is defined as the simultaneous measurement and recording of multiple physiological and behavioural variables during sleep. Overnight PSG is generally conducted in a dedicated sleep laboratory or sleep centre, supervised by trained staff. During PSG physiological signals related to sleep state, ventilation, cardiac function, respiratory effort and body and leg movements are usually measured. Historically overnight PSG has been utilised in children largely to evaluate suspected sleep-disordered breathing. However, increasing recognition and understanding of the non-respiratory sleep disorders has seen the utilisation of PSG widen within the practice of paediatric sleep medicine.

This clinical practice guideline provides best practice recommendations for the respiratory and non-respiratory indications for conducting paediatric sleep studies in Australia and New Zealand and is applicable to children up to the age of 18 years. These recommendations represent the consensus view of a committee of experienced paediatric sleep practitioners selected by the Australian Sleep Association (ASA). Individual conflicts of interest were declared before the review began and were vetted by the ASA Board and were declared to all other committee members.

The report highlights the expanding and evolving nature of sleep investigations. It stresses the central role of the expert clinician in establishing the indications for sleep investigations and in the interpretation of sleep study results. This document seeks to improve standards within Australian and New Zealand by encouraging an evidenced-based approach to the performance of sleep testing, by promoting an accepted and uniform set of definitions of sleep disordered breathing and by encouraging a high standard of laboratory quality control.

The document considers the clinical indications for paediatric sleep studies under the following topics:

- Types of sleep studies.
- Respiratory indications for sleep studies.
- Non-respiratory indications for sleep studies.
- Sleep laboratory facilities and personnel requirements.
- Methodological and technical considerations.

Detailed guidelines regarding the technical specifications for recording sleep and the scoring of sleep and related events are documented elsewhere [3,4].

2.1. Indications for a sleep study

There are three broad indications for performing a sleep study:

- Diagnostic: to assist in the diagnosis and evaluation of the severity of a sleep disorder.
- Assessment of an intervention: to initiate, confirm the adequacy of, or titrate a treatment.
Follow-up/surveillance: to evaluate the persistence, re-emergence or worsening of a sleep disorder or to evaluate the impact of a treatment over time as the child grows and develops.

Diagnostic studies
Diagnostic studies can be used to:

- Identify sleep disordered breathing.
- Investigate sleep-related causes of excessive daytime sleepiness (EDS) such as narcolepsy or frequent arousals (e.g., periodic limb movements).
- Identify and investigate causes of abnormal sleep/wake patterns or sleep fragmentation.
- Delineate the aetiology of episodic, unusual or paroxysmal phenomena occurring during sleep (e.g., parasomnias versus seizures).

Intervention studies
Intervention sleep studies are undertaken to initiate and/or titrate treatment for a sleep-related breathing disorder, such as continuous positive airway pressure or non-invasive ventilation. This enables the modality of treatment to be titrated directly in response to abnormal respiratory events observed during sleep so that the therapy provides optimal support for that patient.

Follow-up studies
Where treatment for a sleep disorder has been instituted, it may be important to confirm the effectiveness of that treatment. Assessment may also be required if symptoms persist or reappear following treatment or if symptoms worsen in cases previously assessed as having primary snoring or mild obstructive sleep apnoea (e.g., if there is significant weight gain). For children receiving positive airway pressure support (e.g., CPAP, BPAP) routine follow up sleep studies are indicated to ensure that the efficacy of treatment is maintained with growth and development or to evaluate if therapy is still needed or can be withdrawn. If some cases follow up sleep studies may also be required to ensure that pharmacotherapy is effective or does not elicit or worsen a sleep disorder (e.g., growth hormone therapy in Prader–Willi Syndrome). Follow up PSG may also be indicated in some medical conditions where there is an increased risk of sleep-disordered breathing but where individuals may be asymptomatic (e.g., central sleep apnoea in achondroplasia).

Persistence of daytime sleepiness, despite treatment, may require additional or repeat testing to confirm satisfactory adherence to therapy, to establish objectively the level of ongoing daytime sleepiness and/or to rule out alternative causes of daytime sleepiness. The maintenance of wakefulness test (MWT) may be undertaken to evaluate the efficacy of treatment in overcoming daytime sleepiness.

Other Sleep-related Tests

Multiple sleep latency test (MSLT)
As an adjunct to overnight PSG, a MSLT may be conducted to objectively evaluate an individual’s level of daytime sleepiness and sleep architecture. The MSLT consists of 4 or 5 twenty minute nap opportunities given at 2 h intervals on the day following an overnight PSG. The basic parameters measured are (i) latency (time taken) to fall asleep (if sleep occurs), which is averaged over the naps to give a mean sleep onset latency and (ii) latency to REM sleep (if observed). No widely accepted norms for children currently exist. Adult norms for “pathologic” levels of sleepiness are generally applied to adolescents and older children but are not relevant or utilised for children <5 years of age. Normal mean sleep latency in a school age child is 16–18 min [5]. Pathologic levels of sleepiness are generally associated with narcolepsy but may also occur as a result of significant chronic sleep deprivation, circadian misalignment or a number of other medical conditions (e.g., epilepsy, Kleine–Levin Syndrome) or due to certain medications/drugs (and hence a drug screen is undertaken as part of the protocol). A short mean sleep onset latency (<8 min) in addition to either (i) REM sleep occurring in 2 or more naps (sleep-onset REM sleep or SOREMs) during a MSLT or (ii) the presence of SOREM on the overnight PSG and in one nap during MSLT is highly suggestive of narcolepsy [6,111].

Maintenance of wakefulness test (MWT) – As the MWT is rarely performed in children refer to the adult document “Guidelines for sleep studies in adults – a position statement of the Australasian Sleep Association” [7].

2.2. Types of sleep studies: levels of investigation

It is evident that the burden of disease associated with sleep disorders in children is great, given their high prevalence and significant associated morbidities [8]. This is especially the case with OSA, which affects 1–5% of children [9]. There is pressure on the specialized facilities throughout Australia and New Zealand to meet growing demands for diagnosis and management, with the result that the waiting list for sleep studies in many centres is very long. This high demand for investigation plus the relatively costly and labour intensive nature of attended PSG (type 1 study) has led to the development of a number of simplified or “abbreviated” sleep studies. These abbreviated studies (types 2–4) are classified according to the number of channels of physiological data that is recorded (classification is detailed below).

As objective testing is preferable to clinical evaluation alone [9–13], some centres may choose to carry out abbreviated studies particularly to prioritise the waiting list for PSG and/or adenosonillectomy (as the first line treatment for uncomplicated OSA in most children) [14]. Given the potential for continuing adverse consequences of undiagnosed and untreated OSA (including neurocognitive, behavioural and cardiovascular impairment [9]), a pragmatic approach to diagnosis that expedites treatment for symptomatic children is appropriate.

In choosing which test or tests are to be used physicians should have a clear understanding of:

(a) The question to be answered by undertaking the test (i.e., indications for testing);
(b) The sensitivity and specificity of the test(s) to diagnose a particular sleep disorder;
(c) The overall utility of the test, taking into consideration the prevalence of a given sleep disorder in the paediatric population;
(d) The cost/benefit balance of the test in a given clinical setting(s);
(e) The technical limitations of the monitoring signals utilised in each particular study type;
(f) Comorbidities and other factors which may affect the reliability and interpretation of the result.

Ultimately the type of sleep study performed will be defined by local facilities, experience and expertise. If unattended and/or abbreviated sleep studies are all that is available it is essential that paediatricians reviewing the study report remain objective and realistic about the limitations of such tests. For sleep studies of any type, the raw data must be available for review by the sleep specialist reporting the study, to enable evaluation of the study quality and to ensure the technical adequacy and accuracy of scoring and interpretation of results.

Sleep studies may be divided into two broad categories: (1) a Type 1 or “comprehensive” study (attended polysomnography)
which is considered the reference standard against which other diagnostic methods are evaluated, or (2) home-based or limited channel sleep studies (Type 2–4) where fewer physiological variables are recorded (see below for more detail). Limited channel studies are usually undertaken using specific portable equipment and are often undertaken in the home or on the hospital ward. Sleep studies may be supervised (continuous attendance by medical, scientific/technical or nursing staff specifically trained in conducting sleep studies) or unsupervised (staff are absent during the recording period). The study duration may be:

- **A full night**: a study conducted over the entire normal sleep period, beginning at the usual bedtime and usually lasting more than 6 h.
- **A full night but split into two parts**: a diagnostic component (commonly characterised by identification of moderate or severe OSA) followed by a therapeutic intervention component (most commonly CPAP titration).
- **Of limited or restricted duration**: a study where the planned length of study is less than 6 h. Such studies would include “nap” studies conducted during the hours of daylight e.g. to investigate sleep disordered breathing in neonates.

### Type 1 Sleep Studies

A Type 1 sleep study (PSG) is considered the reference standard against which other diagnostic methods are evaluated [9,12,13,15]. A type 1 study refers to polysomnography carried out throughout the night in a sleep laboratory in the presence of trained technical staff and under the supervision of a qualified sleep specialist. It involves the continuous recording of multiple physiological variables so that detailed measures of sleep and breathing can be made. Recommended signals include: electroencephalogram (EEG), bilateral electro-oculogram (EOG), submental electromyography (EMG), electrocardiography (ECG), leg EMG, arterial oxygen saturation, respiratory thoraco-abdominal movements, nasal pressure and oronasal airflow, sound, a measure of carbon dioxide (transcutaneous or end tidal), body position and digital video recording. The reader is referred to the AASM [4] and ASTA/ASA guidelines [3] for details of the variables that should be measured during PSG and the technical recording specifications.

PSG enables measurement of sleep architecture (the amount and distribution of the various stages of sleep), quantification of sleep disturbance (arousals and awakenings) and accurate quantification of abnormal respiratory events against time spent asleep. Rapid eye movement (REM) sleep is frequently associated with exacerbation of a sleep-related breathing abnormality and, in some cases, sleep apnoea/hypopnoea may be confined entirely to REM sleep. Thus the quantification of different sleep stages during the night may be important. PSG distinguishes obstructive from central breathing abnormalities, determines the effects of body position on sleep disordered breathing and enables the diagnosis of alternative sleep disorders (e.g. periodic limb movement disorder, parasomnias). Polysomnography also provides information on sleep fragmentation and arousals which are likely important in the genesis of daytime symptoms.

Overall there is a large body of evidence supporting the reliability and accuracy of type 1 sleep studies for detecting the presence of OSA, and the test-retest reliability or consistency of results across multiple nights of PSG recordings has been demonstrated in several studies [9,10,13,16–18]. While it is recognised that some children may not sleep as well on the first night they are in a sleep laboratory because of anxiety, an unfamiliar environment and the attached sensors, research evaluating this so-called “first night effect” suggests few are misclassified on the basis of a single night PSG [17–21].

### Type 2 Sleep Studies

A Type 2 study refers to an abbreviated PSG that is unattended by trained sleep laboratory staff. It is usually undertaken at home but may also be carried out on a hospital ward. A higher success rate in terms of good quality signals may be achieved when the study is set up by experienced personnel rather than by parents or untrained health care professionals [19,22]. A type 2 or ambulatory study must record a minimum of seven channels including EEG, EOG, chin EMG, ECG or heart rate, airflow, respiratory effort and oxygen saturation. The most important distinction from level 3 and 4 studies is that sleep itself is quantified as well as respiratory variables. Audio/video monitoring improves the reliability of unattended studies [23].

This type of testing allows for sleep staging and therefore calculation of total sleep time and respiratory indices (e.g. apnoea–hypopnoea index, AHI). One of the major limitations of unattended studies is the potential for data loss overnight, particularly airflow signals (thermistor or nasal pressure) which are crucial for determination of respiratory events. However, this potential concern regarding data loss has not been borne out by recent studies showing high success rates in children [22]. However, studies to date have included uncomplicated normal mainly school-age children and excluded children with complex medical problems, neurodevelopment delay and significant co-morbidities (e.g., in Marcus et al., only 6 children were developmentally abnormal). Further research is required to compare the feasibility and accuracy of Type 2 with Type 1 studies in children with medical conditions who form a significant proportion of children referred to specialist sleep centres for evaluation of sleep disorders.

### Type 3 Sleep Studies

Type 3 or limited channel sleep studies, sometimes called cardiorespiratory sleep studies, have a more restricted number of parameters that are measured. Usually type 3 studies have at least four variables that are monitored (depending on the type of monitor used): oximetry plus a selection of respiratory effort (chest, abdominal, or both), airflow (nasal or oral by pressure or thermistor), head or body position, ECG, tonometry (a marker of autonomic control), actigraphy (vibration detection) and/or sound/video recording. These studies may be attended or unattended and can be performed in the sleep laboratory, in hospital or at home.

The diagnostic accuracy of type 3 studies is potentially limited by the need to estimate total sleep time using data other than EEG. Total recording time is often used as the denominator, resulting in an underestimate of the severity of sleep disordered breathing. Also sleep disordered breathing leading to arousal and secondary sleep disruption rather than oxygen desaturation may be missed by limited channel studies (type 3 and 4) where sleep and arousals are not quantified. Video recording may reduce this as body movement arousals can be scored visually [24].

While level 3 studies are of relatively low cost, there is limited evidence to date of their efficacy. They have a variable failure rate due to data loss, particularly when the setup is performed by caregivers rather than trained staff. Rosen et al. [25] using a level III device at home on children aged 8–11 years, demonstrated 94% were technically adequate and, when compared to a subset undergoing laboratory PSG, showed good sensitivity and specificity. However, although this cohort study reported ‘excellent agreement’, no actual data was reported. Zucconi et al. [26] evaluated a level III device in the hospital ward (children 3–6 year, N = 12) compared with laboratory PSG on an alternative night. This showed good sensitivity (89%) for increasing obstruction but poor specificity. Poels et al. [27] used an in-home recording device set up by the carer in 2–7 year old children (n = 24) prior to adenotonsillectomy and due to a high failure rate (25–30%) concluded
level 3 studies were of limited use. Conversely, Jacob et al. [24] reported that limited channel home testing plus video was an adequate (83%) and practical option in the evaluation of routine OSA in children (n = 21 2–12 years) with adenotonsillar hypertrophy (Positive Predictive Value (PPV) > 70% and Negative Predictive Value (NPV) > 90%). This latter study involved specialist staff setting up the unit at home. A recent study by Tan et al. found that clinical decisions at the extremes of sleep-disordered breathing (normal/primary snoring with obstructive apnoea—hypopnoea index (OAHI) < 1, or severe OSA with OAHI > 10/h) were unaffected by the use of type 3 studies, whereas under-estimation of events on a type 3 study may have significant impacts on treatment decision in the middle range of OAHI (1–10/h) [28].

Type 3 studies may be useful in cases where a child is too unwell to have a formal study undertaken in a sleep laboratory, or as a relatively simple way of determining the efficacy of treatment in the early stages of initiation of non-invasive respiratory support. However, overall the literature suggests that level 3 sleep studies typically have a high positive predictive value for the presence of OSA but a significant false negative rate (low negative predictive value) i.e., rule in but do not rule out OSA.

**Type 4 Sleep Studies**

A Type 4 study is one that incorporates only one or two measured parameters – for example oxygen saturation, heart rate, transcutaneous carbon dioxide or airflow. Type 4 studies are usually unattended and conducted in the patient’s home or on a hospital ward. Oximetry is an accurate, quantifiable, reliable and informative signal [29]. The development of the multi-wave length oximetry and reduction in size has made oximetry a ubiquitous and accurate marker of hypoxemia. As for Type 3 studies, level 4 studies reliably rule in but do not rule out OSA. Focussing on dips in oxygenation will miss children with OSA with significant sleep disruption but not desaturation (high positive predictive value but low negative predictive value). As with all tests, it is critical to understand the indications for pulse oximetry, what the instrument measures and reports, its limitations and how to interpret its results. Technical factors such as adequate signal acquisition, averaging time and storage sampling frequency are crucial to the reliability of oximetry data. Careful choices of equipment and software are needed for services undertaking oximetry for diagnostic purposes, and results should be interpreted by trained practitioners. Errors in interpretation may lead to inappropriate treatment decisions being made.

Limitations of type 4 studies are seen in cardiac, pulmonary and neurological patients where differentiation between obstructive and central sleep apnoea is especially important, as this is not possible using type 4 studies (the pattern of desaturation may be identical). Low baseline oxygen saturation will reduce specificity further, while supplemental oxygen therapy may negate the utility of signal interpretation. Other limitations include difficulty in identifying artefact, lack of positional data and the use of time in bed rather than actual sleep time in calculating the level of respiratory disturbance.

Despite these limitations, the appearance of the trend and the analysis of desaturation events measured on overnight oximetry may be helpful as a quick, inexpensive alternative to a PSG for the diagnosis of uncomplicated OSA [14]. In the context of a sleep laboratory with a high (60%) pre-test probability of OSA in referred patients, a positive oximetry trend graph (defined as 3 or more clusters of desaturation events and at least 3 desaturations <90%) had a positive predictive values of 97% and could therefore reliably predict the need for adenotonsillectomy in uncomplicated patients without the need for further testing [29]. On the other hand a patient with a negative oximetry had a post-test probability of OSA of 47% and therefore required further testing to exclude or confirm the diagnosis of OSA. Children with frequent oxygen desaturation events <80% are at increased risk of requiring major airway intervention such as re-intubation in the post-operative period [30–32], a finding that facilitates planning of surgical intervention and perioperative care [33]. Recently Horwood et al. [14] demonstrated that oximetry studies evaluated with the McGill Oximetry Score expedite diagnosis and treatment of children with adenotonsillar hypertrophy referred for suspected OSA and Pavone et al. [33] demonstrated good reproducibility of oximetry testing.

In the hospital environment Type 4 studies particularly oximetry and transcutaneous carbon dioxide may be useful for the acute assessment of children and young people with neuromuscular disease at risk of development of hypoventilation or as an easy method of checking adequacy of non-invasive respiratory support. For other medically fragile children where hypoventilation during sleep may be a component of their respiratory status (e.g. achondroplasia, spina bifida, severe obesity etc) assessment of the child’s gas exchange may be helpful in prioritising further evaluation or planning for definitive PSG at a future date.

**Summary**

In summary it is important to highlight that no research has been conducted on the validity of home monitoring (level 2, 3 or 4 studies) for the range of children typically referred for PSG for suspected OSA. Most research studies utilising limited channel recording devices have excluded very young children and those with significant co-morbidities. It is therefore recommended that limited channel diagnostic devices be used in developmentally normal children who are >2 years of age with a high clinical suspicion of OSA following specialist review. A comprehensive clinical history and assessment is essential to help exclude conditions other than sleep disordered breathing e.g. PLMD. While research has not identified safety concerns for home unattended studies, they still require particular attention to safety (e.g., entanglement). Technically reliable information depends on adequate parental instruction, and when possible monitors should be setup by trained staff. Despite the availability of limited channel sleep studies, access to type 1 PSG studies remains vital for the comprehensive and definitive assessment of sleep disordered breathing in children, particularly those with complex health needs. However, irrespective of which type of test is utilised, it is crucial that the results are evaluated and reviewed by a clinician with the necessary expertise and knowledge to interpret the findings accurately and in the context of the clinical history and the developmental stage of the child (i.e., an accredited paediatric sleep physician).

3. Respiratory indications for sleep studies in children

**3.1. Obstructive sleep apnoea**

**Background**

Sleep-related upper airway obstruction is a common disorder of childhood that is considered to represent a spectrum of severity ranging from primary snoring at the mild end to obstructive sleep apnoea at the severe end. OSA is defined as the presence of periods of partial upper airway obstruction and/or intermittent complete upper airway obstruction resulting in abnormalities in ventilation (hypoxia and/or hypercapnia) and/or sleep disruption [1]. Primary snoring is characterised by habitual snoring without ventilatory abnormalities and relatively preserved sleep architecture. OSA is estimated to affect between 1 and 5% of children [9]. It occurs across all age groups but peaks in the preschool years due to adenotonsillar enlargement at a time when the upper airway is still
Concentration or neurobehavioral de
in children includes snoring, restless sleep, hyperhidrosis and poor
obstructive apnoea often observed. The clinical presentation of OSA
during PSG varies widely with prolonged periods of partial upper
children. The pattern of upper airway obstruction seen in children
diatric OSA are different to that observed in adults [34] and
mended for these clinical populations. The neurocognitive, behav-
increased risk of developing OSA (see Appendix I), with more
laryngomalacia (e.g. no history of stridor) [41]. Thus children with a
obstruction has also been reported in atypical presentations of
chest wall and neurological disorders including myelomeningocele
and cardiovascular sequelae of OSA are well documented [9].
neurocognitive, behav-
careful screening and a lower threshold for evaluation recom-
associated with disturbed sleep, with a low threshold for evaluation with
PSG if symptoms of OSA are present [38,39].
Other pre-disposing risk factors for OSA include upper airway
craniofacial anomalies (particularly those associated with mid-face
hypoplasia or micrognathia), neuromuscular disease (including
hypotonia and hypertonia), structural abnormalities of the
wall and neurological disorders including myelomeningocele
and Chiari malformation [40]. A high prevalence of upper airway
abnormalities has also been reported in atypical presentations of
laryngomalacia (e.g. no history of stridor) [41]. Thus children with a
range of craniofacial, genetic or neurological disorders are at
increased risk of developing OSA (see Appendix I) and more
careful screening and a lower threshold for evaluation is recom-
for these clinical populations. The neurocognitive, behav-
and cardiovascular sequelae of OSA are well documented [9].
The presentation, pathophysiology and clinical findings of paed-
OSA are different to those observed in adults [34] and therefore
different diagnostic criteria are applied to infants and
children. The pattern of upper airway obstruction seen in children
during PSG varies widely with prolonged periods of partial upper
airway obstruction (obstructive hypoventilation) rather than frank
obstructive apnoea often observed. The clinical presentation of OSA
in children includes snoring, restless sleep, hyperhidrosis and poor
concentration or neurobehavioral deficits. The prevalence of
enuresis and parasomnias is increased in children with OSA [42].
While snoring is the cardinal symptom of OSA in older children,
infants may have little or no snoring [1]. Furthermore habitual
snoring occurs in 7–15% of children [43] but clinical history is
unable to distinguish primary snoring from OSA [1].

While adenotonsillectomy (AT) is commonly the first line of
treatment for children with OSA, other treatment modalities may
include anti-inflammatory therapies, positive airway pressure
support, nasopharyngeal airway, mandibular distraction, rapid
maxillary expansion or mandibular advancement splint.

Although AT is effective in treating OSA in many children, some
individuals and clinical populations are at increased risk of residual
OSA after surgery. Estimates of residual OSA following AT vary
(13–29%) in low risk groups but as high as 73% in high risk groups [9];
with increased risk of residual obstruction related to a range of fac-
tors including pre-existing co-morbidities and moderate to severe
pre-operative OSA [44–46]. Incomplete resolution of OSA is more
common in children with craniofacial abnormalities that obstruct the
upper airway [47], and neurologic disorders such as myelome-
ingocele [48] and Down syndrome [49]. Residual OSA is also more
common in obese children [46,50,51]. In a large randomised
controlled trial of AT for OSA, residual OSA was detected in 33% of
obese subjects [51]. Recurrence of OSA one year following AT has also
been demonstrated [44]. Given the high prevalence of residual OSA
clinicians should evaluate children postoperatively for symptoms of
OSA and parents advised of the potential recurrence of OSA.

**Consensus recommendations**

- PSG is indicated when clinical assessment suggests the diag-
nosis of obstructive sleep apnoea (OSA).
- Abbreviated testing is indicated in selected cases as a clinical
adjunct tool for the assessment of children with suspected
obstructive sleep apnoea (OSA) following paediatric sleep
specialist review.
- PSG is indicated if clinical assessment suggests OSA and abbre-
viated testing is inconclusive for the diagnosis of obstructive
sleep apnoea.
- PSG is indicated when the severity of OSA is in doubt or if there
is significant risk of post-surgical complications (e.g., idiopathic
thrombocytopenic purpura).
- PSG is indicated when clinical assessment suggests the presence
of residual OSA following AT or other treatment modalities.
- PSG is indicated following AT in children where there is the
presence of ongoing symptoms or other clinical features sug-
gestive of persistent OSA, particularly in those with: moderate
to severe OSA pre-operatively, obesity, craniofacial abnor-
malities that pre-dispose to the development of upper airway
obstruction, neurologic disorders (e.g., neuromuscular disor-
ders, spina bifida, Chiari malformation, meningomyelocele) and
high risk congenital conditions (e.g., Down Syndrome, Pra-

### 3.2. Central sleep apnoea and non-obstructive hypoventilation

**Background**

Central sleep apnoea is characterised by absent respiratory
effort occurring in a cyclical or intermittent pattern arising from a
deficiency in central respiratory control. Non-obstructive hypo-
ventilation is characterised by reduced pulmonary ventilation
arising from diminished central respiratory drive, neuromuscular
anomalies or restrictive lung disorders. The aetiology of these
abnormal respiratory patterns may be congenital or acquired
[52,53] and can result in significant hypercapnia and/or hypoxia.
Clinical indicators of non-obstructive sleep disordered breathing
include witnessed apnoea, nocturnal dyspnoea, restless sleep or
frequent arousals, hyperhidrosis, morning headaches, waking
unrefreshed or tired, elevated awake PaCO2 or base excess, changes
in behaviour or neurocognition or daytime tiredness. However
some patients may be asymptomatic, particularly in the early
stages of non-obstructive hypventilation.

**Central sleep apnoea**

Discrete respiratory pauses are a normal feature of sleep in all
age groups and are typically observed in REM sleep or following
sighs and body movements. These events are generally of brief
duration (<20 s) and do not usually result in significant blood gas
exchange abnormalities or sleep disruption [54]. Clinically relevant
central apnoeas are those of prolonged duration (>20 s) or if
associated with oxygen desaturation, cyanosis, cortical arousal or
bradycardia. Brief central apnoeas are common in term infants in the first few months of life (particularly in REM sleep), decreasing in frequency with postnatal development due to maturation of the respiratory system. Central apnoea can also be seen in some individuals at sleep onset due to instability of ventilatory control occurring at wake/sleep transitions, but these events typically resolve once sleep deepens.

Children with OSA sometimes display an increased frequency or severity of central apnoea [55] which can resolve following AT [56]. Children at risk of cervico-medullary or brain stem compression (e.g., Chiari malformation, meningomyelecele, spina bifida, achondroplasia) are at increased risk of developing central apnoea [40,57]. An increased frequency and/or severity of central apnoea has also been reported in children with a range of neurological disorders (e.g., Joubert syndrome) and those with neuro-developmental disorders such as Down syndrome [58] and Prader–Willi syndrome [59], due to impaired ventilatory control.

Periodic breathing

Periodic breathing (PB) is defined as a series of three or more central pauses lasting >3 s separated by no more than 20 s of normal breathing usually presenting in a crescendo/decrescendo pattern [4]. Periodic breathing is very common in premature infants reflecting the immaturity of the respiratory system. Periodic breathing can also be seen in term infants, peaking between 2 and 4 weeks of age, where up to 2–5% of total sleep time may be spent in this breathing pattern. Periodic breathing can be seen in both REM and NREM sleep but the pattern appears to differ between these two sleep states [54]. Elevated levels of PB in infancy or PB observed in older children is considered abnormal [54]. Periodic breathing is sometimes seen in children at sleep onset due to instability of ventilatory control occurring at the wake/sleep transition and this typically resolves once sleep deepens.

Apnoea of prematurity

Due to the immaturity of the respiratory control centre, central apnoea and PB are very common in premature infants, with the prevalence inversely related to birth weight and gestational age [60]. Other potential structural abnormalities of the respiratory system related to pre-term birth include narrow Airways, chest wall distortion, diaphragmatic fatigue, poor neuromuscular control and reduced functional residual capacity (FRC) which can all be exacerbated during sleep, particularly active sleep [61]. Upper airway obstruction is therefore also a frequent feature of SDB in preterm infants, with mixed apnoea the most common type of respiratory event observed [62]. Rapid and profound oxygen desaturation may occur with respiratory events of relatively short duration while significant bradycardia and pallor may also be associated with abnormal breathing. Given that apnoea of prematurity (central sleep apnoea or periodic breathing) is virtually ubiquitous in very premature infants, PSG would not be indicated in those cases [1] and the apnoea can be managed and monitored clinically. There are selected cases of post-term infants however where other medical disorders have been ruled out and PSG may be indicated to confirm a diagnosis of idiopathic apnoea of infancy [2], define the extent of gas exchange abnormality and rule out hypoventilation [1]. In this context PSG may be useful to determine the need for treatment and/or home monitoring.

Congenital central hypoventilation syndrome

Congenital Central Hypoventilation Syndrome (CCHS) is a rare disorder of ventilatory control associated with autonomic nervous system (ANS) dysregulation [63]. The clinical presentation and severity of CCHS is variable but in general it is characterised by sleep-related respiratory insufficiency and a diminished ventilatory response to hypercapnia and hypoxia. A mutation in the PHOX2B gene which is involved in ANS development has been identified as the cause. While ventilation is usually most abnormal in NREM sleep (or quiet sleep for infants), REM sleep and wake ventilatory abnormalities can also occur [64]. Most children with CCHS typically present at birth or in infancy with severe central hypoventilation requiring ventilation, but a milder phenotype can also present later in older children or adults.

Non-obstructive hypoventilation

In children, non-obstructive hypoventilation is commonly seen in neuromuscular disorders (e.g., spinal muscular atrophy, Duchenne muscular dystrophy, myotonic dystrophy), in restrictive lung disorders (e.g., kyphoscoliosis, chest wall deformities) and sometimes in individuals with morbid obesity. In addition to hypoxia (which may be intermittent or sustained) the development of carbon dioxide retention may cause blunting of central chemoreceptor function further exacerbating sleep-disordered breathing [65,66]. Upper airway obstruction may also co-occur due to diminished muscle activity, abnormal neurological control or mechanical breathing disadvantages [67].

While the development and pattern of sleep-disordered breathing is highly variable in different neuromuscular diseases (NMD), respiratory muscle weakness and scoliosis are two major contributing factors to the development of non-obstructive hypoventilation [68]. It has been estimated that up to 62% of children with NMD have some degree of sleep-disordered breathing [69]. In children with progressive NMD, non-obstructive hypoventilation may develop gradually but daytime symptoms and clinical assessment such as pulmonary function testing frequently fail to identify the advent of non-obstructive hypoventilation until daytime respiratory abnormalities are present indicating advanced disease progression [70,71].

There is some evidence to suggest that commencement of non-invasive ventilation in the early stages of sleep-related hypoventilation slows disease progression and reduces hospital admissions [72]. Therefore it is recommended that children with progressive NMD have regular clinical assessment to review disease progression and symptoms of SDB which may need evaluation with PSG to determine if non-invasive ventilation should be considered [73,74]. In particular, the risk of sleep-related hypoventilation is increased if scoliosis or diaphragmatic weakness are present, and after the loss of ambulation in Duchenne muscular dystrophy [68,75]. Additionally, as non-obstructive hypoventilation may not be clinically evident in children with NMD, pre-surgery PSG should be considered if major surgical procedures are planned (e.g., scoliosis surgery) due to the risk of adverse responses to the administration of anaesthetics or sedatives. A forced vital capacity (FVC) below 1 L and/or 40% predicted [76] or the presence of daytime hypercapnia are also clinical indicators for conducting PSG.

Relatively few studies in children have examined the effect of restrictive lung disease on sleep-related respiratory patterns. The relatively small thorax of children with achondroplasia is believed to put them at increased risk of developing sleep-related hypoxia due to restrictive lung disease, although not all studies report this finding [77]. While restrictive lung disease has been reported in some children with achondroplasia [78,79], it appears to be less prevalent than OSA in this population. Adults with kyphoscoliosis are known to have breathing difficulties during sleep and the same finding may be expected in children but studies are lacking [34].
Non-obstructive hypoventilation due to morbid obesity (obesity hypoventilation syndrome) is typically characterised by a combination of symptoms including increased carbon dioxide retention during sleep, sleep-related hypoxia not caused by upper airway obstruction and daytime somnolence not explained by other causes. Some individuals may also show awake hypercarbia or other adverse cardiovascular abnormalities such as pulmonary hypertension [80]. The pathophysiology of obesity hypoventilation syndrome (OHS) is complex and not fully understood. The increased mass loading on the pulmonary system reduces chest wall compliance and FRC leading to increased work of breathing and reduced oxygen reserve. Blunted ventilatory drive has been demonstrated in adults with OHS [81] and though data are lacking for children, similar findings may apply.

**Consensus recommendations**

- PSG is indicated if clinical evaluation or abbreviated testing suggests the presence of central apnoea that is: (i) unexpected for development/age (ii) of prolonged duration or (iii) associated with significant oxygen desaturation or frequent bradycardia, pallor change, sleep disturbance or other daytime sequelae.
- PSG is indicated in cases of apnoea of infancy that are unresponsive to treatment or unusually severe or persistent or if there is diagnostic doubt over the aetiology of events (i.e. obstructive versus central).
- PSG is indicated when clinical assessment suggests the diagnosis of Congenital Central Hypoventilation Syndrome (CCHS).
- PSG or abbreviated testing is indicated when clinical assessment or other investigations (e.g. pulmonary function tests) suggest the presence of non-obstructive hypoventilation due to NMD, obesity, kyphoscoliosis or chest wall deformity.
- PSG or abbreviated study is indicated pre-operatively in at-risk children with neuromuscular disorders undergoing major orthopaedic, thoracic or upper airway surgery to screen for undetected SDB which may be exacerbated by sedation or analgesics.

### 3.3. Polysomnography for children receiving respiratory support

**Background**

The treatment of sleep-disordered breathing by positive airway pressure (PAP) support has increased significantly in the paediatric population in recent years, particularly with the advent of suitable mask interfaces for infants and children. Continuous positive airway pressure (CPAP) and bi-level positive airway pressure (BPAP) are used to treat a range of sleep-related breathing abnormalities including upper airway obstruction and non-obstructive hypoventilation. An initial sleep study is generally performed on PAP to optimise treatment as pressure levels can be manually titrated directly in response to abnormal respiratory events observed during sleep. Subsequent repeat sleep studies are important in optimising long term management, particularly to determine if pressure requirements have changed as a result of the child’s growth and development, if symptoms reappear while on PAP or if additional or alternate treatment has been instituted [82]. The frequency of follow up should be based on clinical symptoms, the child’s growth rate, clinical stability or disease progression or other factors that may precipitate a worsening of sleep disordered breathing (e.g., significant weight gain). However, children receiving PAP should have at least an annual PSG, with young children and those with a rapidly progressive disorder often requiring studies more frequently (e.g. 4–6 monthly). Clinical or physiological improvement has been demonstrated in children with SDB and neuromuscular disorders when BPAP was titrated during PSG [82–84].

Regular review of interface fit and suitability is also important. Following reports in the literature, clinicians should be mindful of the potential development of mid-face hypoplasia in children receiving PAP. Regular assessment and techniques to try and minimise the development of mid-face hypoplasia (e.g. mask rotation, nasal cushions) are recommended with a low threshold for referral to a craniofacial or orthodontic specialist. Furthermore, it is important to note that PSG is only one component in the treatment of sleep disordered breathing, with education of the patient and family and long term evaluation of the patient’s symptoms, quality of life, adherence and side effects of the treatment being equally important. Patient education and motivation to use CPAP account for a greater variance in PAP adherence than standard biometric or anthropometric markers of OSA severity [85].

Other methods for treating SDB include tracheostomy, mechanical ventilation and humidified high-flow nasal cannula therapy. Polysomnography is particularly useful for assessing children using these treatments as hyperventilation may worsen during sleep and awake ventilator settings may therefore be inadequate during sleep. There are no published papers for this indication. There are some studies that have demonstrated the clinical usefulness of PSG using a downsized or capped tracheostomy tube as part of the evaluation of readiness for decannulation in children with long-term tracheostomy [86,87]. Some children treated with tracheostomy for sleep-related breathing disorders may benefit from polysomnography as part of the evaluation prior to decannulation but this is not universally accepted. These children should however be followed clinically after decannulation to assess for recurrence of symptoms of sleep related breathing disorders.

**Consensus recommendations**

- PSG is indicated for children receiving positive airway pressure support (CPAP or BPAP) to titrate the level of pressure support required and to periodically re-evaluate the appropriateness of settings, including synchrony between machine and patient.
- PSG or abbreviated testing may be beneficial in children treated with invasive or negative pressure ventilation to adjust ventilator settings.
- PSG is indicated in children with symptoms of sleep-disordered breathing following tracheostomy decannulation. PSG may be clinically useful in some children (e.g., achondroplasia, Pierre Robin Sequence) with tracheostomy capped or downsized as part of the evaluation prior to decannulation.
- PSG is indicated in children with tracheostomy for upper airway obstruction where the co-occurrence of another breathing disorder (e.g., central sleep apnoea) is suspected.
- PSG is not routinely indicated for the titration of supplementary oxygen in children with chronic lung disease except in patients who are known, suspected or considered at risk of developing hypoventilation, central apnoea or hypercapnia with supplemental oxygen.

### 3.4. Other respiratory indications for sleep studies

#### 3.4.1. Apparent life threatening event (ALTE)

The most widely used definition of an ALTE is an episode that is “frightening to the observer and that is characterized by some combination of apnoea (central or occasionally obstructive), color change (usually cyanotic, but occasionally erythematous or plethoric), marked change in muscle tone (usually marked limpness), choking, or gagging” [88]. The majority of ALTEs occur in children under the age...
of one year, with premature infants at increased risk [89]. It is recommended that the investigation and management of ALTE is predicated on the perceived severity of the presentation and the clinical history/physical examination of the child [54,90]. Guidelines for initial assessment and follow-up have been published [90]. Approximately 20% of ALTEs for which an explanation is found are believed to arise from respiratory causes including obstruction and central apnoea [54,90]. In general overnight polysomnography is only indicated in selected infants where there is suspicion of sleep-disordered breathing (including periodic breathing and obstructive sleep apnoea), evidence of oxygen desaturation or the presentation of repeated ALTEs of unknown aetiology [54].

Several studies [91–93] have shown subtle, nonspecific PSG abnormalities in some infants who had experienced an ALTE. The PSG findings were not predictive of recurrence of ALTE. Studies of infants who eventually succumbed to sudden infant death syndrome (SIDS) [94–96] demonstrated PSG abnormalities that were neither sufficiently distinctive nor predictive to support routine use of PSG for children at risk for SIDS. If, however, obstructive sleep apnoea is clinically suspected [97], or if bradycardia is demonstrated on cardiac monitoring in the absence of central apnoea, or if an underlying disorder of breathing control (e.g., CCHS) is suspected, consideration of a PSG should be discussed with a paediatric sleep centre. Thus PSG is not indicated as part of the routine evaluation of an ALTE unless the clinical indicators specified above are observed.

Consensus recommendations

- PSG is not indicated for infants as part of a routine evaluation for an uncomplicated ALTE.
- PSG is indicated for infants who have experienced an ALTE and where there is clinical suspicion of underlying sleep disordered breathing or if there is ongoing or unexplained oxygen desaturation.

3.4.2. Prader–Willi syndrome and growth hormone therapy

Sleep and respiratory abnormalities are common in Prader–Willi Syndrome (PWS) and include central and obstructive sleep apnoea, impaired ventilatory control, excessive daytime sleepiness and altered REM sleep distribution [98,99]. Sleep-disordered breathing including OSA is frequently found in children with PWS in the absence of symptoms [100,101]. Children diagnosed with PWS are frequently prescribed human growth hormone (GH), which has been shown to have a multitude of beneficial effects including improved linear growth and body composition as well as improvements in cognitive and developmental trajectories [102]. However there have been reports of sudden death in PWS children having symptoms suggestive of OSA who were receiving GH supplementation. The mechanism underpinning the association between sudden death and OSA in children with PWS has not been completely elucidated, but an increase in the volume of lymphoid tissue in the upper airway following GH treatment is a contributing factor. In response to this increased risk of OSA and potential sudden death, screening for sleep-disordered breathing prior to commencement of GH in children with PWS has been instituted in many countries. Treatment of OSA is usually recommended prior to commencement of GH therapy. Similarly, PSG is recommended soon after GH treatment has commenced (usually 6–10 weeks), particularly in younger children [102,103], given evidence for the development of severe OSA in some children in this time period, prompting withdrawal of GH while OSA was treated [104]. In addition, for the duration of GH treatment, children with PWS should be regularly monitored for symptoms of OSA and PSG repeated in the event of the development of new or worsening symptoms, as OSA can develop later in the course of treatment with GH [105].

Consensus recommendations

- PSG is indicated in children with PWS prior to and six to ten weeks following the commencement of GH treatment and thereafter as clinically indicated.

3.4.3. Sleep-disordered breathing in children with underlying chronic lung disease

Children with underlying lung disease (e.g., cystic fibrosis, bronchopulmonary dysplasia, asthma) may be at increased risk of developing SDB. These patients have reduced functional reserve and may become hypoxic during sleep, particularly in REM sleep when accessory respiratory muscle hypotonia occurs [34]. Other factors that may exacerbate sleep disordered breathing in children with chronic lung disease include increased bronchoconstriction during sleep, reduced mucociliary clearing or cough and increased arousals or sleep fragmentation [99].

Consensus recommendations

- PSG is indicated for children with chronic lung disease (e.g., asthma, cystic fibrosis, bronchopulmonary dysplasia) or unexplained pulmonary hypertension if there is clinical suspicion of sleep-disordered breathing.

4. Non-respiratory indications for sleep studies in children

4.1. Movement disorders

Background

Sleep-related movement disorders are characterised by involuntary, often stereotypical movements that disturb the initiation or maintenance of sleep. These disorders include restless legs syndrome (RLS), periodic limb movement disorder (PLMD), sleep-related leg cramps, bruxism and rhythmic movement disorder [106].

Restless legs syndrome (RLS) is a neurological sensorimotor disorder characterised by an irresistible urge to move the legs often accompanied by uncomfortable or painful sensations of the lower extremities. These symptoms are generally worse in the evening, prior to sleep onset or when inactive and are relieved by moving the legs [106]. Periodic Limb Movement Disorder (PLMD) is characterised by periodic episodes of repetitive, highly stereotyped periodic leg movements during sleep accompanied by a complaint of sleep disturbance or daytime fatigue/tiredness that cannot be accounted for by another primary sleep disturbance. Periodic limb movements during sleep often co-occur with RLS and can be exacerbated by certain medications. The symptoms of RLS and PLMD can be difficult for younger children to articulate [107] and are sometimes incorrectly categorised as “growing pains.”

Clinical evaluation should include a detailed history and physical examination. Special emphasis on complaints of leg discomfort, the occurrence of leg or body jerks, restless sleep, and reports of insomnia or excessive daytime sleepiness should be sought. Physical examination should focus on excluding a peripheral neuropathy that can mimic RLS. Serum ferritin, complete blood count, urinalysis, and biochemistry testing should be undertaken to look for secondary causes of RLS (e.g., iron deficiency anaemia, uraemia). Generally RLS is diagnosed from clinical history and therefore polysomnography is not routinely indicated except where
uncertainty exists in the diagnosis, in which case the presence of PLMs might be collaborative evidence [108]. Polysomnography is required to diagnose PLMD particularly as the individual is usually unaware of the muscle movements and as parental report is often unreliable with a low positive predictive value. A periodic limb movement index of more than 5 per hour is considered abnormal in children [109]. As night-to-night variability in periodic limb movements has been documented in some children a single study might not be adequate to establish the diagnosis or severity of PLMD [110].

Consensus recommendations

- PSG is not routinely indicated to diagnose restless legs syndrome (RLS), with the diagnosis based on clinical features.
- PSG is indicated for children suspected of having periodic limb movement disorder (PLMD).

4.2. Parasomnias and epilepsy

Background

Parasomnias are common in childhood and are defined as “undesirable physical events or experiences that occur during entry into sleep, within sleep or during arousals from sleep” [111]. Depending on their presentation, parasomnias may be a normal feature of sleep (primary parasomnias) or may point to other underlying medical conditions or pathology (secondary parasomnias) [112]. There are three broad categories: parasomnias associated with NREM sleep (e.g., sleep walking, sleep terrors and confusional arousals); parasomnias occurring in REM sleep (e.g., REM-sleep behaviour disorder, isolated sleep paralysis [111]); and other parasomnias (e.g., sleep-related enuresis, catathrenia, sleep-related eating disorder) [113]. Parasomnias can present as simple or complex motor behaviours, abnormal sleep-related movements or vocalisations, and are associated with varying degrees of consciousness or perception, autonomic activation and emotional expression.

The unusual or abnormal behaviours seen in some parasomnias can mimic sleep-related seizures and vice versa, as some of the motor and behavioural manifestations observed are common to both disorders [114]. In particular nocturnal frontal lobe epilepsy can present in a similar fashion to REM sleep parasomnias, although clinical features are helpful in differentiating the two [115]. Co-existing sleep disorders (e.g., OSA) may worsen seizures occurring during sleep.

A careful clinical evaluation of the suspected parasomnia with emphasis on age of onset, time of event relative to sleep onset, frequency, regularity and duration of event, and a detailed account of the event itself, is often sufficient to diagnose common, uncomplicated, non-injurious parasomnias without the need for polysomnography. However, polysomnography may be indicated if clinical evaluation is inconclusive (e.g., REM-sleep behaviour disorder or seizure activity), if excessive daytime sleepiness or impaired daytime functioning is present (not explained by other causes) or if the presence of another sleep disorder (e.g., OSA, PLMD) is suspected, as these may be worsen parasomnias. For example the prevalence of sleep walking and sleep terrors has been found to be elevated in pre-pubertal children with OSA and restless legs syndrome [42]. Utilising PSG to investigate unusual or atypical events occurring during sleep should include the recording of EMG, good quality audio-video and if available, an expanded bilateral EEG montage. Liaison with a paediatric neurologist may be judicious in cases where doubt remains about the diagnosis or in the advent of unusual EEG findings which are not be routinely encountered in the sleep laboratory. PSG (with an extended bilateral EEG montage if available) may be helpful for the diagnosis of seizures occurring during sleep but is generally not the first line investigation.

Consensus recommendations

- PSG is not indicated if clinical assessment is consistent with a typical childhood parasomnia.
- PSG is indicated if a parasomnia is atypical or if excessive daytime sleepiness or impaired daytime functioning is present that cannot be explained by other causes.

4.3. Hypersomnia and narcolepsy — polysomnography, multiple sleep latency test (MSLT)

Background

Excessive sleepiness in a child may present as sleeping longer than peers, age inappropriate napping or a change in sleeping pattern to sleeping greater amounts. However due to a general lack of knowledge in the primary care setting with respect to age appropriate sleep patterns and developmental changes, sleepiness in children is often under-recognised or misinterpreted [116]. For example the diagnosis of narcolepsy in children is often delayed by up to 10 years despite debilitating daytime sleepiness often being one of the first symptoms [117]. While a common cause of excessive sleepiness is insufficient or poor quality sleep (for a variety of reasons including intrinsic sleep disorders such as OSA), hypersomnias of central origin are not typically characterised by insufficient overnight sleep or by misaligned circadian rhythms [106]. The third edition of the International Classification of Sleep Disorders (ICSD-3) recognises eight distinct types of hypersomnia of central origin, several of which may be encountered in paediatric practice [111].

Narcolepsy

Narcolepsy is a debilitating lifelong neurological disorder characterised by hypersomnolence and the pathologic manifestation of REM-sleep like phenomena. The clinical features of narcolepsy include chronic excessive daytime sleepiness, disturbed sleep, cataplexy, sleep paralysis and hypnagogic or hypnopompic hallucinations. Not all features may be present in all patients, and excessive daytime sleepiness is often the only feature in children. The diagnosis of narcolepsy in children can be challenging due to the highly variable presentation of this disorder, the inability of younger children to describe their symptoms and the sometimes atypical presentation of some features such as cataplexy [118]. Misinterpretation of symptoms and misdiagnosis are not uncommon. Objective diagnosis of narcolepsy is achieved by performing a multiple sleep latency test (MSLT) where the propensity to fall asleep across the day and the presence of sleep-onset REM periods during naps are examined.

While the diagnosis of narcolepsy may be reasonably certain based on a clinical history of daytime hypersomnolence and cataplexy (which is highly specific to this condition), a MSLT preceded by PSG is still recommended for children in order to obtain objective evaluation. Furthermore, although infrequent, attempts to secure stimulant medication or exaggerated parental report of symptomology may elicit a false or misleading clinical history. Additionally because this condition is life long and its diagnosis may have significant implications for driving and vocational choices, and medications to treat this disorder may carry significant risk, objective testing is highly desirable.
 Overnight PSG immediately prior to the MSLT is used primarily to exclude other causes of excessive daytime sleepiness (e.g. OSA), and to confirm that the patient had sufficient sleep the night prior to the MSLT. Repeat MSLT or performing a maintenance of wakefulness test (MWT) may be indicated to evaluate the effectiveness of treatment or to comply with driver licensing authority requirements. Additional details for performing MSLT or MWT are provided later. Of note, the most recent International Classification of Sleep Disorders (ICSD-3) has revised the diagnostic criteria for narcolepsy [111].

Kleine–Levin Syndrome

Kleine–Levin Syndrome is a rare condition sometimes seen in adolescents (male predominance) characterised by recurrent or periodic hypersomnia interspersed with normal periods of alertness and behaviour. The hypersonomolent periods may also be accompanied by cognitive and mood changes, hyperphagia and hypersexuality. Overnight PSG findings during the symptomatic period include decreased slow wave and REM sleep, shortened REM latency and reduced sleep efficiency [119]. Multiple sleep latency testing shows reduced sleep latency but no sleep-onset REM periods as seen in narcolepsy [114]. Of note, reduced CSF hypocretin levels have been demonstrated in one out of 14 year old subject while symptomatic that normalised when not in the hypersonomolent phase [120].

Idiopathic Hypersomnia

The diagnosis of idiopathic hypersomnia is one of exclusion. Overnight PSG is therefore required to rule out other common sleep disorders such as OSA that can cause hypersonomolence. An assessment of sleep-wake schedules using a sleep diary with or without actigraphy over a 2–3 week period can also be helpful to exclude chronic sleep restriction. MSLT should be performed to objectively confirm the presence of hypersonomolence. Other rare medical disorders (e.g. Prader–Willi Syndrome and Bardet–Biedl Syndrome) are associated with pathological sleepiness (and may also be associated with sleep-onset REM periods) and may thus be part of the differential diagnosis of idiopathic hypersomnia or narcolepsy. However, these conditions are usually readily identified by careful clinical history and examination, supplemented by genetic testing in some instances.

Other Causes of Hypersonomia

Other causes or medical conditions associated with central hypersonomia include neurological disorders, head trauma or neurosurgery, medications, substance abuse, psychiatric disorders and menstrual-related hypersonomia [106].

Multiple Sleep Latency Test (MSLT)

A MSLT must be performed immediately following in-laboratory PSG. The overnight PSG is conducted to exclude other potential causes of daytime sleepiness (such as OSA or PLMD) and needs to demonstrate that an adequate amount of sleep has occurred (minimum 6 h) prior to the MSLT. However the presence of these disorders does not rule out the existence of narcolepsy, as narcolepsy can co-exist with other sleep disorders. If excessive daytime sleepiness or other clinical features of narcolepsy from causes other than narcolepsy such as hypersonomia associated with a medical or psychiatric condition, recurrent hypersonomia, and other hypersonomias.

Consensus recommendations

- The MSLT preceded by overnight PSG is indicated in children as part of the evaluation for suspected narcolepsy and excessive daytime sleepiness.

5. Sleep disorders service procedures, facilities and personnel requirements

The following sections outline technical and practical recommendations for performing attended paediatric polysomnography (type 1 sleep study) in order to facilitate uniformity in practice and to set the minimum standards of practice for the delivery of high quality sleep medicine services. While minimum standards for sleep disorders services have been published jointly by the ASA and the National Association of Testing Authorities (NATA) (2012) the information contained in the following sections highlights or
expands on some of the recommendations or considers items not specifically contained in the ASA/NATA accreditation document.

5.1. Policies and procedures

A paediatric sleep disorders service should have in place (or adhere to existing broader organisational) policies and procedures with respect to:

- Standardised methodology for conducting PSG and related procedures (including positive airway pressure (PAP) and supplementary oxygen titration protocols).
- Administrative processes (e.g., handling and management of referrals).
- Maintenance of patient confidentiality.
- Equipment testing and safety.
- Data and file management/security (for both paper and digital records/reports).
- Staff training, supervision and ongoing education/development including regular staff appraisal.
- Quality assurance and improvement including proficiency testing, auditing processes and document control.
- Identification of non-conformities to established practice or procedures and subsequent corrective action and resolution.
- Emergency procedures.
- Patient safety.
- Consumer feedback and complaint resolution.
- Occupational health and safety including manual handling.
- Infection prevention and control including cleaning of equipment, electrodes and sensors and other items used during PSG and related procedures.

All policies and procedures should be documented and must be readily accessible by staff in the form of a manual or in electronic format. Policies and procedures should reflect current knowledge and best practice for sleep medicine service provision and should therefore be reviewed at appropriate intervals. Processes should be in place whereby any changes or updates to policies and procedures are effectively communicated to relevant staff.

5.2. Referral for polysomnography and related procedures

All requests for PSG and related procedures should be evaluated prior to testing for appropriateness of investigation by a paediatric sleep physician. Prior to a diagnostic PSG each patient should be clinically evaluated by a paediatric sleep physician. Polysomnography and related procedures should only be conducted once the sleep laboratory has received an appropriately authorised test request completed by a qualified paediatric sleep physician or a medical officer under his or her supervision. At a minimum the referral should contain the following:

- Patient identifying details.
- Reason for referral.
- Relevant clinical history including current medications.
- If applicable, PAP settings or level of supplementary oxygen the patient is receiving.
- Type of testing to be undertaken (e.g., diagnostic PSG, titration PSG, MSLT) and if applicable, any specific instructions with respect to the conduct of the test procedure, particularly if there are any variations or additional requirements from normal testing protocols.
- Any special instructions or considerations in relation to testing (e.g., special staffing requirements).
- The time frame or urgency in which testing is to occur.

- Any pertinent patient information that may be helpful to overnight staff (e.g., physical limitations, developmental delay, anxiety or behavioural issues).

5.3. Sleep facility personnel requirements

Paediatric sleep studies should be performed by staff that are skilled in dealing with children of varying ages and developmental stages, and who may present with medical or behavioural co-morbidities. Depending on the nature of the sleep disorders service, staff performing paediatric sleep studies may comprise nurses, technical officers/scientists or clinical physiologists. Paediatric PSG requires a higher staffing ratio compared to adult studies with a ratio of no less than one nurse/sleep technologist to two patients required overnight. However in some cases a higher staffing level may be required (e.g. for tracheostomy patients). All staff must be trained in performing paediatric cardiopulmonary resuscitation covering the age range of patients that are seen within the service, with regular updates. In addition, staff should be instructed in the relevant laboratory legislation relating to child protection. A criminal history check prior to working with children is generally required by most state authorities. As some clinical groups (e.g., infants or children with neuromuscular disorders) are at increased risk for complications following respiratory infection, staff should be encouraged to keep up to date with relevant immunisations.

5.4. Sleep recording rooms and environmental conditions

A paediatric sleep facility should engender an environment that is non-threatening and child friendly. Patient recording rooms must be designated body-protected electrical areas. Bedrooms must be child safe with appropriate bedding for age (e.g., cots) that meets up-to-date industry standards. Staff must be trained in the safe operation of cots and beds. Bedroom and bathroom amenities need to accommodate wheelchair bound patients and patient lifters need to be available where needed to transfer the patient to and from the bed. Staff must be adequately trained in the safe operation of patient lifters (annual refresher training is recommended if use is infrequent).

Space and amenities for the parent or caregiver to sleep in the same room as the child during the test procedure should be available. This will enable the parent or caregiver to reassure and comfort the child if needed as well as provide routine night time care (e.g., toileting or feeding). Co-sleeping of a parent/caregiver and child in the same bed during a PSG is not ideal as it may (i) not be safe or practical in the laboratory setting, (ii) cause difficulties with staff accessing sensors, (iii) cause sleep disturbance to the child, (iv) result in signal artefact if the parent/caregiver lies on or makes contact with sensors, and (v) the parent may obscure the child from view of the video camera - all of which may impact on PSG interpretation. However, many children co-sleep with parents and may have difficulty sleeping if this is not permitted. The decision to permit co-sleeping should therefore be made at the local level depending on safety concerns, site specific policies and practicalities. Any indications of unsafe sleeping practices that may be happening in the home should be brought to the attention of the referring physician so that parents can be appropriately counselled. Alternative strategies include allowing the parent or caregiver to lie in the bed with the child until the child is asleep or moving the parent bed next to the child’s bed. In some cases children with neurodevelopmental disorders may have an assistance dog which should be permitted to be stay with the child throughout testing.

Appropriate storage and heating facilities for milk or infant formula must be available with facilitation of overnight feeds for
infants/young children during PSG recording. Provisions must be made for the appropriate storage of patient medications. If stored in a central location, medication and milk/infant formula must be labelled with appropriate patient identifiers.

Bedrooms should have effective soundproofing, lighting and temperature control. The latter is particularly important for premature infants and neonates as they have poor thermal control. If a premature infant or neonate becomes too cool, motor activity increases resulting in restless/fragmented sleep. Furthermore, both low and high ambient temperatures have been shown to alter arousal thresholds as well as apnoea frequency and duration in infants [126,127]. High ambient temperatures may also degrade the quality of electrophysiological signals during PSG in the form of sweat artefact so it is important to be able to cool recording rooms if needed.

Provisions need to be made for medical emergencies including an emergency call system in patient areas and a complete range of age-appropriate resuscitation equipment as well as oxygen and suction located at the bedside. The patient’s name, age and current body weight should be readily available in the event that medical assistance in an emergency. A management plan should be in place for children with epilepsy in the event that a seizure occurs. Staff should consult with the parent or caregiver as to the typical presentation of the patient’s seizures and any known triggers. A paediatric sleep/respiratory physician should be on call during sleep studies should advice be needed during the study. Clinical criteria for contacting the on-call paediatric sleep/respiratory physician during the study should be in place in the event of severely abnormal PSG findings or other concerns. Similarly a mechanism must be in place that alerts day staff (scoring analyst and/or the referring physician) that a child has had an abnormal PSG where urgent intervention or treatment may be required. Consideration should be given for a mock medical emergency exercise to be undertaken on an annual basis to ensure staff are aware of the correct emergency protocols and to ensure that emergency procedures are up to date and effective. This is particularly important if a sleep disorders service is situated in an isolated location or where relatively few staff may be present in the immediate vicinity outside of normal business hours.

6. Methodological and technical considerations for performing paediatric polysomnography

The AASM manual for the scoring of sleep and associated events (2007) together with associated documentation [128–131] provides guidelines for PSG recording and interpretation (including paediatric PSG) which have been valuable in standardising practice, particularly in North America. In 2011, the Australasian Sleep Technologists Association and the Australasian Sleep Society produced consensus guidelines for recording and scoring sleep studies in children in Australia and New Zealand. In 2012 the AASM guidelines were updated [132] and currently version 2.2 is the most recent published scoring manual produced by the AASM [4]. Read together, the AASM and ASTA/ASA documents provide practice parameters for recording and scoring of sleep studies in children, including technical requirements, recording specifications, scoring of sleep and respiratory events. The reader is referred to these documents as the information contained therein will not be repeated here.

6.1. Polysomnography – patient preparation

Preparation of patients prior to polysomnography

Polysomnography is a complex physiological test that is especially demanding of young children and parents and the methods and facilities used for conducting adult sleep studies may not meet the needs of children. Successful testing depends on a child-friendly environment and staff experienced in caring for children in health care settings. Particular challenges for the paediatric sleep technologist will be dealing with children who are scared, too young to understand what is happening, developmentally delayed, have an aversion to tactile or somatosensory stimuli or who have undergone numerous or recent painful hospital procedures. Furthermore many children being evaluated for a sleep disorder will have neurobehavioural or medical co-morbidities which can be challenging to manage. Lastly, parents may also be very anxious about their child having a sleep study, particularly with respect to the application of electrodes and sensors.

Preparation and education of the child and family prior to the study enhances the quality of data obtained as it reduces anxiety and improves the likelihood of a successful study. Ideally this should be done at the time of the clinic visit including an explanation of why the child needs the test and what will be involved. In addition, families should be given printed information or photographs to take away which explain in simple terms what will happen during testing and any preparations they need to make. Provision for visiting the sleep laboratory or trying on some of the sensors beforehand may also be helpful in reducing anxiety in some children. Desensitisation activities prior to testing for children with tactile or other aversions (e.g., nasal cannula) may also be beneficial. A parent or adult care-giver should be present with the child throughout the test and age-appropriate distractions available to assist staff in setting up the monitoring leads. Ideally the adult staying overnight should not snore although this is not always possible or practical. Encouraging children to bring their own pillows, wear their favourite pyjamas and bring a favourite toy can make the experience less alienating. Ideally the timing of the test should be appropriately geared to the bedtimes of young children. Where appropriate an interpreter may be helpful in explaining the test preparation and procedure to families from non-English speaking backgrounds.

Illness and Immunisation

In general polysomnography should not be performed while a patient is acutely unwell, as illness (e.g., fever, vomiting) may obscure PSG findings or the acute illness may exacerbate the true nature of sleep disordered breathing or other sleep problems. Furthermore certain respiratory symptoms (e.g., a runny or blocked nose) may create difficulties in obtaining good airflow signals during PSG leading to uncertainties in test interpretation and diagnosis. It is also advisable not to perform PSG on the same day as immunisations as immunisation may result in a fever and restless sleep.

Medications

Certain medications may alter normal sleep and respiratory physiology. For example:

- Hypnotics, sedatives and opioid based analgesics may worsen or induce sleep-disordered breathing and may alter sleep architecture and arousal thresholds.
- Antidepressant, antipsychotic and some antiepileptic medications may alter sleep architecture, arousal thresholds and features of the sleep EEG (e.g., spindles) and muscle tone.

Other medications that may be commonly encountered in the paediatric setting and which may influence PSG findings include caffeine, melatonin, nasal steroids, antihistamines, methylphenidate.
or dexamphetamine and clonidine. Administration of sedating agents for the sole purpose of facilitating sleep during a PSG is not recommended. It is the responsibility of the sleep clinician ordering the test to decide whether the PSG should be performed on any medication that may impact respiratory or sleep physiology or whether the PSG should be performed following the withdrawal of confounding drugs. Considerations should include:

- Whether the medication is being used in the acute setting (e.g., to treat intercurrent illness) or in the chronic/long term setting thereby representing the patient’s usual circumstances.
- Contraindications to medication withdrawal.
- The clinical indication for the PSG.

In some circumstances a study both on and off medication may be desirable. Withdrawal of medication prior to a PSG needs to be undertaken with appropriate medical supervision, and a suitable washout period should be instituted to minimise rebound effects. For MSLT studies, medications that could affect the results of the MSLT should be discontinued two weeks prior to the study (if not medically contraindicated). The paediatric sleep physician reporting the test should be fully aware of the condition of the patient at the time of the study including medication usage and interpret the result accordingly.

Activities on the day of Polysomnography

Patients should be instructed to follow normal activities of daily living prior to presentation for their PSG. Unless the study is being performed for a special purpose, patients should maintain their regular sleep habits prior to the study. It is advisable to ask patients to refrain from consuming caffeinated or energy drinks on the day of the PSG.

Preparation of patients during polysomnography

Preparing a paediatric patient for a sleep study requires a flexible and adaptable approach depending on the age or developmental level of the child and their medical history. While individual sleep facilities may differ with respect to patient preparation, at the start of PSG the following issues should be anticipated:

- Site specific protocols for obtaining informed consent should be followed.
- Sufficient information about each patient’s clinical history should be made available to staff performing the PSG in the event that the on-call physician needs to be contacted or if a medical emergency should arise.
- The sleep technologist performing the test should be provided with the necessary patient and test information to maximise the quality and integrity of the PSG. Reviewing the child’s last PSG, particularly with respect to how the child coped with the procedure, may be helpful in anticipating what might happen. Any potential medical emergencies or concerning events (e.g., breath holding spells) should be made known to staff before starting the study.
- If a considerable amount of time has elapsed between when the patient was referred by the paediatric sleep physician and the test date, then the sleep technologist may need to consult with the referring or on call sleep physician if there are concerns about changes in the patient’s medical history or status.
- The patient should be assessed for any potential contraindications to PSG methodology. For example, care needs to be taken in cleaning electrode sites in children with haemophilia and EEG electrodes should not be applied on suture lines in children who have undergone cranial or neurosurgery.
- Prior to starting the PSG setup it is important to ascertain if the patient has any allergies to any of the tapes/adhesives, pastes or cleaning products used during the procedure. If there is uncertainty about a particular product apply a small amount on the underside of the patient’s forearm and monitor for any signs of skin reaction. Patients should be screened for any allergy to products containing latex with consideration given to using only latex-free gloves in the unit.
- Attaching the necessary electrodes and sensors can be a challenging process for both the technologist and the child. Where possible, engaging the parent or caregiver during the PSG setup will help reassure an anxious or scared child. Having the child sit on a parent’s lap or allowing the child to place a few electrodes on the parent or a doll/teddy can be helpful in eliciting cooperation. Useful distractions include stickers, interactive books, toys or games (e.g., hand held water games), colouring in or watching a non-stimulating video. Strategies for making PSG more child friendly and dealing with children with neurodevelopmental disorders have been published [133–135].

Repeat Polysomnography

Repeat PSG may be indicated in children who are receiving continuous positive airway pressure (CPAP) therapy or non-invasive ventilation (NIV) to assess whether therapy is still required or to check that current levels of therapy are appropriate. Growth and/or treatment (e.g., surgery, weight loss) may render CPAP/NIV unnecessary or may require changes to therapy settings. Alternatively a repeat “diagnostic” PSG may be required to assess the necessity of respiratory support in children who are non-compliant with therapy or who are having significant side effects. There is evidence in adults that a CPAP washout effect, i.e. CPAP therapy may have a residual beneficial effect on the severity of sleep–disordered breathing (temporarily less severe) for several days after withdrawal from treatment [136] but this has not been specifically studied in children. It has been recommended that CPAP be discontinued several days prior to PSG if considering discontinuation of therapy [137], however this should only be undertaken at the clinician’s discretion. Some children may need to be progressively weaned from NIV therapy. The provision of an oximeter for the duration of the NIV withdrawal period may be a judicious safety precaution in certain cases.

6.2. Polysomnography—test procedure

Equipment Calibrations

The integrity of the high frequency physiological signals collected during PSG (EEG, EOG, EMG, ECG) is highly dependent on the amplifiers, gains and filters of the PSG recording system. It is therefore recommended that two types of machine calibrations are performed to check the accuracy of the high-frequency amplifiers and the high and low pass filters [138–140].

(i) An all-channel calibration where all the channels are set to the same gain and filter settings and a signal of known properties (usually a negative 50 μV DC signal) is sent through all the channels simultaneously. This tests the integrity of the amplifier as each channel should respond the same way with respect to signal polarity, amplitude and decay time constant.

(ii) A montage calibration where the gain and filter specifications used during the PSG recording are set for each channel as is appropriate for the physiological parameter being recorded. Once again a signal of known properties is sent through all the channels simultaneously and each channel is
checked to confirm the signal response is appropriate (if not gain or filter settings may not be set or working correctly). Recording channels with similar settings (e.g., EEG and EMG) should display an identical output.

Regular calibration of any ancillary monitoring equipment (e.g., DC output devices such as pulse oximeters or transcutaneous carbon dioxide [TcCO2] monitors) is essential to ensure accurate signal acquisition and processing by the PSG system. For continuous signals (e.g., pulse oximetry [SpO2], TcCO2, PAP pressure, sound level) a minimum of 3 points (if possible) should be calibrated that span the expected range of the measurement (as two points will always give a straight line!). In the case of step calibrations (such as body position and room light) a calibration of each level/type should be performed. All physical calibrations should be appropriately labelled and permanently recorded.

The frequency of equipment and signal calibrations depends on the stability of the transducer and the likelihood that an intrinsic or extrinsic factor could cause an error in the value reported. If the signal is critical to the interpretation of the study, for example SpO2 or PAP pressure, it should be calibrated prior to each study. If loss of signal is critical to the interpretation of the study, for example SpO2, extrinsic factor could cause an error in the value reported. If the loss of signal would still allow interpretation and the transducer is or PAP pressure, it should be calibrated prior to each study. If loss of signal is critical to the interpretation of the study, for example SpO2, extrinsic factor could cause an error in the value reported. If the loss of signal would still allow interpretation and the transducer is stable, less frequent calibration is acceptable. In most systems the gain is stable over a long period of time and monthly calibration of these amplifiers is adequate.

**Sensor and electrode application**

Minimum technical and recording specifications for paediatric PSG including sensor and electrode placement are detailed in:

- The AASM Manual for the Scoring of Sleep and Associated Events [4].
- The ASTA/ASA addendum to AASM Guidelines for recording and scoring of paediatric sleep [3].

The attachment of sensors and electrodes should be standardised to ensure consistent and accurate results for PSG interpretation. Variations from normal methodology should be clearly documented on the study log with an explanation of the reason for the variation. Any such variations should be discussed in the reporting paediatric sleep physician. It is beyond the scope of this document to discuss the precise methods for electrode and sensor application and this information should be detailed in a protocols and procedures manual. However there are a number of considerations that are of particular relevance in the paediatric population:

- The skin of children (especially infants) is much thinner than in adults and care needs to be taken with cleaning electrode/sensor sites and removing adhesive tapes. Similarly, care must be taken when using sensors that produce heat – e.g., the oximeter and TcCO2 probe. These sensors should be repositioned during the recording period to minimise the likelihood of patients receiving skin damage.
- EEG electrodes should not be applied over the fontanelle of infants.
- It is not uncommon for infants or children to have allergies or sensitivities to tapes and adhesives. Hypoallergenic products should be used if this is the case.
- Children with neurodevelopmental disorders often have sensory sensitivities particularly tactile or somatosensory aversions which can make applying electrodes and sensors challenging. This should be made known to staff prior to the PSG visit as it may necessitate a higher staffing level at the start of the night. Engaging the parent/caregiver during the PSG process will be important as they best know their child. Desensitisation prior to the PSG may need to be considered if the sensitivities are severe.
- Despite the best efforts of staff there may be occasions when children are intolerant of the sensors and electrodes used during PSG. Guidelines should therefore be in place for these situations indicating the minimum sensors or electrodes that are needed to obtain clinically useful information.
- An extended bilateral EEG montage with continuous video monitoring should be considered for patients if the following are present or clinically suspected: seizures, parasomnias, unexplained hypersomnolence that does not appear to be classic narcolepsy, unusual sleep-related body movements or sleep-related paroxysmal events/ arousals.
- Some children with refractory epilepsy may present with a vagal nerve stimulator (VNS) in situ. This should be noted on the study log and/or PSG report as VNS activation is known to cause reductions in airflow. The resultant flow limitation may be severe enough to warrant treatment with CPAP [141]. Activation of the VNS can usually be seen as artefact on the submental EMG which can be correlated with airflow reductions in airflow. Alternatively, an additional surface electrode can be applied close to the site of the VNS electrode in the neck to detection activation [135].
- Due to the higher risk of entanglement with electrode and sensor cables in infants and children must be under continuous visual surveillance while setting up and conducting PSG. Further, some parasomnias such as sleep walking which are more common in children and injuries may result if children abruptly try to get out of bed. Most modern PSG systems now come with synchronised infra-red recording and cameras with zoom-in capability are invaluable for scoring sleep in infants where behavioural observations such as eye opening or closure are important adjuncts to the EEG in determining wake or sleep.

**Electrode Impedance Verification**

Once all sensors and electrodes are attached the sleep technologist should visually check all signals to verify they are displaying correctly and free of artefact. An impedance check should be conducted (and recorded) for all the AC recording channels (EEG, EOG, EMG, ECG) including the reference and ground channels. Ideally impedances for individual electrodes should be below 5 KΩ. It is also important that impedances are as equal as possible between paired electrodes otherwise common mode rejection (elimination of electrical interference) will not be optimised. However it is not always possible to obtain low impedance levels in paediatric studies for a variety of reasons including patient tolerance and compliance. While striving to obtain low impedances is strongly encouraged, there will be situations where a compromise may need to be made in order to be able to collect some information rather than none at all. Discretion should be used in situations where the signal quality looks reasonable and can be interpreted but the entire PSG might be at risk is further attempts to obtain lower impedances are made. An impedance check should be performed again near the end of the PSG recording to confirm signal integrity or on any occasion during the study if it appears that signal quality has diminished or there is evidence of artefact in the signal.

**Patient Bio-calibrations**

Patient bio-calibrations are important for validating signal integrity and responsiveness and serve as a baseline scoring reference for each subject. Bio-calibrations should be performed at the beginning of each PSG once impedance checks have been done and corrective action taken if needed. Examples of commonly
performed bio-calibrations are shown below. Each manoeuvre should be conducted for an appropriate length of time to verify signal response. For younger or developmentally delayed children, abridged bio-calibrations may be possible for the simpler instructions. For infants or toddlers signal responsiveness and sensitivity may be assessed by viewing the patient spontaneously performing various actions and confirming that the signals are responding appropriately. Bio-calibrations should be recorded as part of the PSG in the event they are needed during PSG scoring for signal referencing or corroboration.

<table>
<thead>
<tr>
<th>Recording Parameter Verified</th>
<th>Patient Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEG (alpha reactivity)</td>
<td>Eyes open, staring straight ahead</td>
</tr>
<tr>
<td>EOG</td>
<td>Eyes closed, keeping head still</td>
</tr>
<tr>
<td>EOG</td>
<td>Move eyes left/right, keeping head still</td>
</tr>
<tr>
<td>EOG</td>
<td>Move eyes up/down, keeping head still</td>
</tr>
<tr>
<td>Submental EMG</td>
<td>Smile</td>
</tr>
<tr>
<td>Respiratory bands</td>
<td>Hold breath</td>
</tr>
<tr>
<td>Nasal pressure</td>
<td>Deep/big inspiration</td>
</tr>
<tr>
<td>Thermocouple/thermistor</td>
<td>Breathe through nose only</td>
</tr>
<tr>
<td>Diaphragmatic/intercostal EMG</td>
<td>Breathe through mouth only</td>
</tr>
<tr>
<td>Nasal pressure</td>
<td>Deep/big breath in</td>
</tr>
<tr>
<td>Anterior tibialis EMG</td>
<td>Flex left leg</td>
</tr>
<tr>
<td>Microphone/sound</td>
<td>Snoring sound</td>
</tr>
</tbody>
</table>

**PSG Documentation**

Adequate documentation by the sleep technologist during the PSG is crucial for interpreting the results. Even though the PSG system may have audio-visual recording capacity not everything may have been clearly captured during the recording process. For example, if snoring is coming through the audio-channel, is it that the system may have audio-visual recording capacity not everything is not crucial for interpreting the results. Even though the PSG signal referencing or corroboration. Part of the PSG in the event they are needed during PSG scoring for signal referencing or corroboration.

Additional documentation that should be chronicled or recorded with each PSG (or related test) includes: the referral information; the date and time of the test; the identity of all staff performing various aspects of the testing; the equipment used in testing including the name and version of software used for PSG acquisition and scoring.

**Study Duration and Split Night Studies**

A PSG recording should be at least 8 h in duration and should ideally collect at least 6 h of sleeping data (depending on age). A PSG during which less than 6 h of sleep was observed or that contained less than 6 h of technically sound data should be interpreted with caution particularly if REM sleep was not observed.

A split night PSG may be appropriate in patients (generally infants) presenting with uncomplicated cyclical central apnoea/periodic breathing in order to institute and titrate supplementary oxygen therapy. Protocols should be in place for this clinical scenario.

**6.3. Polysomnography analysis and reporting**

Standards for scoring and analysing paediatric sleep studies are documented in:

- The AASM Manual for the scoring of sleep and associated events [4].
- The ASTA/ASA addendum to AASM Guidelines for recording and scoring of paediatric sleep [3].

All studies should be reported by a qualified paediatric sleep physician who reviews the raw/scored data. The patient’s relevant clinical data must be disclosed to the reporting physician to aid in interpretation of the test result. At a minimum this should include the age and associated medical condition(s) of the child and medications taken at the time of the sleep study. In addition the reporting physician should review or be aware of the technical observations made by the overnight and scoring technologist. All
studies should be reported in light of this clinical and technical information.

Severity Criteria for Sleep Disordered Breathing

The obstructive apnoea—hypopnoea index (OAHI) has been used in clinical trials and epidemiological studies to classify patients as either having OSA or being normal, as well as to classify the severity of OSA.

The following classification of OSA severity is commonly used in clinical practice:

- Normal: OAHI <1 events per hour of sleep
- Mild OSA: OAHI 1 – ≤5 events per hour of sleep
- Moderate OSA: OAHI 5 – ≤10 events per hour of sleep
- Severe OSA: OAHI >10 events per hour of sleep

A classification for the severity of central and non-obstructive sleep disordered breathing has not been established.

Acknowledgements

Thanks to Angela Campbell and Bruce Williamson for reviewing the “Methodological and Technical Considerations for Performing Paediatric Polysomnography” section of this document. Thank you also to Dr Nick Antic whose encouragement and commitment for developing standards of practice in sleep medicine in Australasia are particularly recognised.

Conflict of interest

The following conflicts or potential conflicts of interest were declared by members of the committee:

All authors have no conflicts of interests.

1. Current or recent (last 3 years) involvement with a company or companies with a financial interest in devices or methods for performing sleep studies:
   a. Direct financial interest
   b. Employee, or engaged in a consulting capacity (including medical advisory boards, expert testimony)
   c. Substantial research support
   d. Sponsored attendance at national or international meetings

2. Financial benefit received (personally, spouse or dependents, or department) from performing or reporting sleep studies
   a. Direct benefit received
   b. Departmental benefit received

Individual COI statements are available from the secretariats of the Australasian Sleep Association and the Thoracic Society of Australia and New Zealand.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2017.03.020.

Appendix I

Children at risk of sleep-disordered breathing should be routinely assessed for clinical indicators of sleep problems with a low threshold for further investigation.

Specific medical conditions associated with an increased risk for the development of sleep-disordered breathing

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Down Syndrome</td>
</tr>
<tr>
<td>Prader—Willi Syndrome</td>
</tr>
<tr>
<td>Achondroplasia</td>
</tr>
<tr>
<td>Neuroromuscular disorders (e.g., Duchenne Muscular Dystrophy, Myotonic Dystrophy, Spinal Muscular Atrophy)</td>
</tr>
<tr>
<td>Craniofacial syndromes affecting the upper airway (e.g., Pfeiffer, Treacher-Collins, Crouzon &amp; Aper's Syndrome; Pierre Robin sequence)</td>
</tr>
<tr>
<td>Genetic disorders affecting respiratory control (e.g., Congenital Central Hypoventilation Syndrome, Rett Syndrome, Joubert Syndrome)</td>
</tr>
<tr>
<td>Metabolic Storage disorders (e.g., Hurler's Syndrome)</td>
</tr>
<tr>
<td>Cerebral palsy</td>
</tr>
<tr>
<td>Leigh's Disease</td>
</tr>
<tr>
<td>Chiari malformation</td>
</tr>
<tr>
<td>Spina bifida</td>
</tr>
</tbody>
</table>

Risk factors which pre-dispose the development of sleep-disordered breathing

<table>
<thead>
<tr>
<th>Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenotonsillar hypertrophy</td>
</tr>
<tr>
<td>Retrognathia</td>
</tr>
<tr>
<td>Mid-face hypoplasia</td>
</tr>
<tr>
<td>Macroglossia</td>
</tr>
<tr>
<td>Structural abnormalities of the upper airway (e.g., laryngomalacia, cleft palate, congenital oral anomalies, vocal cord palsy)</td>
</tr>
<tr>
<td>Hypotonia</td>
</tr>
<tr>
<td>Chronic lung disease</td>
</tr>
<tr>
<td>Premature birth</td>
</tr>
<tr>
<td>Chest wall deformity</td>
</tr>
<tr>
<td>Scoliosis</td>
</tr>
<tr>
<td>Epilepsy</td>
</tr>
<tr>
<td>Face and neck burns</td>
</tr>
<tr>
<td>Moderate to severe obesity</td>
</tr>
<tr>
<td>Family history of sleep disordered breathing</td>
</tr>
<tr>
<td>Disadvantaged socioeconomic status</td>
</tr>
<tr>
<td>Ethnicity</td>
</tr>
</tbody>
</table>

References


Australasian Sleep Association position statement regarding the use of psychological/behavioral treatments in the management of insomnia in adults

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Keywords:
Insomnia
Cognitive Behaviour Therapy-Insomnia
Treatment effectiveness
Treatment guidelines

ABSTRACT

Insomnia disorder is a high prevalence condition with a high disease burden, which, left untreated, can increase risk of poorer health outcomes. Due to Insomnia’s tendency towards having a chronic course, long-term treatment approaches are needed to reduce the impact of Insomnia over time. After reviewing the available literature, The Australasian Sleep Association (ASA) recommends Cognitive Behavior Therapy for Insomnia (CBT-I) as a first line treatment in the management of Insomnia. The ASA notes that in addition to CBT-I, there is emerging evidence for the use of Mindfulness Based Therapy for Insomnia when used in combination with behavioural techniques (MBT-I). CBT-I should be used whenever possible, and treatments should be limited to the lowest necessary dose and shortest necessary duration. CBT-I, whilst the most effective long-term treatment, does not work for everybody across all circumstances, so there will be circumstances in which other treatments are required (e.g., pharmacotherapy). Improving access to CBT-I is an important issue which will involve raising awareness of the effectiveness of CBT-I, increasing the number of trained practitioners, and the development of effective low-intensity treatments that can be offered in the first instance.

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1. Background

1.1. Insomnia disorder definition

Insomnia disorder as defined in the Diagnostic and Statistical Manual fifth Edition (DSM-V, [2]) as longstanding (more than 3 months) subjective difficulty initiating asleep, maintaining sleep, or waking too early, accompanied by distress about the experience of daytime fatigue and its impact on day-time functioning. The difficulty occurs at least three nights per week, despite adequate opportunity for sleep. Adjustment Insomnia/Acute Insomnia is less than 1 month in duration.

1.2. Insomnia disorder subtypes

- The DSM-V outlines five insomnia subtypes: sleep initiation insomnia; sleep maintenance insomnia, early morning awakening, a combination of these three core symptoms, or non-restorative sleep.
- The International Classification of Sleep Disorders-3 (ICSD-3, [1]) outlines four chronic insomnia subtypes: psychophysiological insomnia (insomnia that occurs due to a learned response of increased arousal whilst attempting to sleep), idiopathic insomnia (lifelong insomnia), paradoxical insomnia (sleep state misperception), and inadequate sleep hygiene (insomnia due to poor sleep habits).
- According the DSM-V and ICSD-3, to receive a diagnosis of insomnia disorder, the insomnia should be clinically significant on its own even though it may occur at the same time as another physical or mental condition.
- The primary and secondary insomnia distinction has been removed in the DSM-V and ICSD-3, in order to emphasise the mutually exacerbating nature of chronic insomnia with other mental and physical conditions. ‘Insomnia disorder’ is now recognized as a condition requiring independent clinical attention, regardless of other medical problems that may be present.

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Although the primary and secondary insomnia distinction is no longer used, the most common diagnostic classification systems, they are defined here as these terms appear widely in the Insomnia literature.

Primary insomnia: Insomnia that is not directly attributable to a medical, psychiatric, or environmental cause. Primary insomnia according to the ICSD may be psychophysiological insomnia (objectively verifiable impaired sleep), idiopathic insomnia (childhood onset), or paradoxical insomnia (sleep state misperception).

Secondary insomnia: Insomnia that is result of other causes such as (medical condition, medication or substance, mental disorder, inadequate sleep hygiene).

1.3. Prevalence and course

Insomnia is the most prevalent of all sleep disorders in the general population [31]. In terms of prevalence of Insomnia symptoms, population-based studies across various countries suggest that approximately 30% of adults report one or more of the symptoms of insomnia: difficulty initiating sleep, difficulty maintaining sleep, waking up too early, and in some cases, non-restorative or poor quality of sleep. Indeed, acute Insomnia prevalence rates are higher, at approximately 30% [3]. Prevalence of individuals meeting full criteria for insomnia disorder is estimated at 10% among the general population with increased rates in increasing age, female gender, and presence of medical or psychiatric illness. A recent Australian study estimated the prevalence to be 7% [14]. Importantly, chronic insomnia is a persistent, long-term disorder. For example, research has suggested that three quarters of individuals reporting insomnia at baseline still report having insomnia 1 year later and almost half reported having insomnia at three consecutive annual assessment points [26].

1.4. Burden

Sleep is an important component of health and has a large negative impact on quality of life, affecting how well people think, work and interact with others (e.g., Ref. [17]). Individuals with insomnia experience fatigue, mood disturbance, and distress [25], have greater absenteeism rates, 60% higher healthcare costs as compared to the general population (e.g., Refs [36,39]) and an increased risk of accidents [7]. There is also an increased risk for depression, anxiety, suicide, and substance use, with insomnia being an independent risk factor. The economic burden in Australia is estimated to be $5.1 billion per year [14].

2. The use of cognitive behavioral treatments in insomnia disorder

The main treatment goals in insomnia are: (1) to reduce nocturnal hyperarousal, (2) to improve sleep quality and quantity, (3) to reduce insomnia related daytime impairments, (4) reduce the distress and anxiety associated with poor sleep. Psychological/behavioral treatment approaches assume that physiological and cognitive hyperarousal (tension and worry) contribute to the evolution and maintenance of poor sleep. Poor sleep results in unhelpful sleep habits (e.g., irregular sleep routine, excessive caffeine use, long daytime naps) that maintain insomnia over time. Cognitive Behavior Therapy for Insomnia (CBT-I) is a multicomponent treatment that consists of an educational component, cognitive interventions (correcting unhelpful beliefs, reducing worry, reducing cognitive hyperarousal) and behavioural interventions (sleep restriction, stimulus control) which work to reduce arousal and behaviours/routines that interfere with sleep.

Meta-analyses and systematic reviews support the efficacy of CBT-I interventions for insomnia in both younger and older adults [5,18,40,43]. Dismantling studies suggest that both Cognitive-maximises both acute and long term effects [15]. Research investigating the combination of CBT-I and pharmacotherapy suggests this is effective as long as the pharmacotherapy is short-term and adverse effects of medications are assessed [24,26,28]. As far as stand-alone treatments are concerned, CBT-I has the best efficacy [43]. The acute effects of CBT-I are comparable with or superior to those of hypnotic medications and are maintained for up to 3 years of follow-up [23]. There is emerging evidence that mindfulness-based treatment for insomnia (MBT-I) is also efficacious [11,33,34].

Outcome studies support the use of both individual and group CBT-I treatments [18,27,30]. There is also evidence for the effectiveness of telephone, printed self help, and online cognitive-behavioural therapy (for reviews see Refs. [16,41,42,46]). One randomised controlled trial of individual face-to-face versus online CBT-I exists and this suggests a superiority of face-to-face treatment [20]. There is evidence to suggest that brief, weekly telephone support [15] or personalised motivational feedback emails [21] may enhance outcomes for online treatments. Telephone, printed self help, and internet-based options (with personalised support where possible) are recommended by the ASA as part of a stepped-care approach, or in places where face-to-face treatments are unavailable or too costly, or when there is a low level of complexity in the insomnia disorder [4,9,16,44].

A proportion of Insomnia patients have circadian rhythm abnormalities that can result in sleep onset insomnia (delayed sleep phase) or early waking insomnia (advanced sleep phase). Careful timing of exposure to bright light in the morning or evening, respectively, may be a behavioural intervention that is useful in such cases [19,22].

2.1. Recommendation that CBT-I is a first line treatment

The ASA recommends treatments with either Level I or Level II evidence. The Australian National Health and Medical Research Council [29] and the Oxford Centre for Evidence Based Medicine [35] use the following classification system when assessing evidence for specific treatments.

**Level I**
- Evidence obtained from a systematic review of all relevant randomised controlled trials (meta-analyses).

**Level II**
- Evidence obtained from at least one properly designed randomised controlled trial.

The ASA recommends CBT-I as a first line treatment of insomnia disorder as it has extensive level I evidence. It is universally accepted as the best treatment modality for insomnia disorder, in the long term.

2.2. CBT-I description

The table below provides a summary of the core components of CBT-I (Table 1).
CBT-I — cognitive behavioral therapy for insomnia.

Note: Although patients with chronic insomnia should adhere to rules of good sleep hygiene, there is insufficient evidence to indicate that sleep hygiene alone is effective in the treatment of chronic insomnia. It should be used in combination with other therapies. It is considered a part of CBT-I, so it is recommended alongside the other components of CBT-I (see table).

Source: http://annals.org/data/Journals/AIM/934264/7tt1_Table_1_Components_of_CBT-i.jpeg.

**Table 1**

<table>
<thead>
<tr>
<th>Component</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive therapy</td>
<td>Aims to identify, challenge, and replace dysfunctional beliefs and attitudes about sleep and insomnia. Such misconceptions may include unrealistic expectations of sleep, fear of missing out on sleep, and overestimation of the consequences of poor sleep.</td>
</tr>
<tr>
<td>Stimulus control</td>
<td>Behavioral instructions aimed at strengthening the association between bed and sleep and preventing conditioning of the patient to associate bed with other stimulating activities. Such instructions include avoiding nonsleep activities in the bedroom; going to bed only when sleepy; and leaving the bedroom when unable to sleep for 15–20 min, returning to bed only when sleepy.</td>
</tr>
<tr>
<td>Sleep restriction</td>
<td>Behavioral instruction to limit time in bed to match perceived sleep duration in order to increase sleep drive and further reduce time awake in bed. Time allowed in bed is initially restricted to the average time perceived as sleep per night and then adjusted to ensure sleep efficiency remains &gt;85%.</td>
</tr>
<tr>
<td>Sleep hygiene</td>
<td>General recommendations relating to environmental factors, physiologic factors, behavior, and habits that promote sound sleep. Specific instruction s include advice on control of the bedroom environment, including avoiding visual access to a clock; regular sleep scheduling and avoiding long daytime naps; and limiting alcohol, caffeine, and nicotine intake, especially before bed.</td>
</tr>
<tr>
<td>Relaxation</td>
<td>Any relaxation technique that the patient finds effective can be used to limit cognitive arousal and reduce muscular tension to facilitate sleep. Specific techniques that may be used include meditation, mindfulness, progressive muscle relaxation, guided imagery, and breathing techniques.</td>
</tr>
</tbody>
</table>

2.3. Comorbid insomnia

Insomnia disorder is frequently comorbid with other physical and mental disorders. Traditional treatment for insomnia with comorbid conditions has focused on treating the comorbid condition with the expectation that the insomnia will resolve. Recent studies, however, suggest this approach is not the most appropriate. Instead, treating both conditions simultaneously may improve the outcomes for each [38]. There is extensive research demonstrating the effectiveness of CBT-I in the context of comorbid conditions (see Ref. [12]).

Assessment and consideration of comorbid conditions in treatment planning is important.

2.4. Mindfulness Based Therapy for Insomnia (MBT-I)

Ong and colleagues [32–34] developed an adaptation of Mindfulness Based Stress Reduction (MBSR) tailored specifically to insomnia that is called Mindfulness-Based Therapy for Insomnia (MBT-I). MBT-I is a program that integrates behavioural techniques for insomnia in addition to Mindfulness meditation techniques. Mindfulness is a very practical intervention and helps individuals to learn to recognize and manage unpleasant thoughts and feelings including those associated with poor sleep. There is emerging evidence for the effectiveness of Mindfulness as a stand-alone treatment and in combination with behavioural techniques (e.g., Refs. [34,45]). MBT-I has level II evidence, in so far as research is emerging on this relatively new treatment (for a review see Ref. [11]). At this point in time CBT-I remains the gold standard of treatment.

2.5. Combination therapy

There is a role for combining CBT-I with hypnotic medication, particularly when commencing treatment. An insight in to how treatments might be combined comes from work by Morin et al. [24,26,28], where 160 patients were randomised to CBT-I or CBT-I plus the non-benzodiazepine, zolpidem 10 mg, for 6 weeks. The group who used zolpidem together with CBT-I for 6 weeks, then stopped zolpidem, did better at 6 months than those able to continue to use zolpidem beyond that 6-week point, or who had CBT-I alone. Further work needs to be done in this area to clarify the sequencing of these treatments, which have different mechanisms of action and time courses of onset and offset of effect, and may well be complementary.

3. Issues of access to psychological treatment

Despite its high prevalence and burden, insomnia often goes unrecognised and untreated. Many individuals with insomnia don't seek professional help, but rather use self-help remedies of limited benefit [25]. This may be due to fatigue and reduced motivation, perceived lack of treatment success, and the perception that insomnia is a benign condition that can be managed without professional input. Given the potential seriousness of the condition, efforts should be made to educate patients regarding treatment options. Of those Insomnia sufferers that do seek professional help, treatment is usually limited to pharmacotherapy [6,13]. Data from many developed countries show that hypnotics are the most commonly used insomnia treatments for people seeking help for insomnia in primary care. Australian data from 2987 general practice treatment episodes of sleep disorders showed 81% of patients reporting a new problem of insomnia were prescribed a medication [6]. Rates of referral for advice or counselling were much lower than for other disorders and only 0.8% of patients were referred for specialist care compared to an average across all general practice presentations of 8.3%.

Access to CBT-I is an important issue and can be a barrier to people being referred. However, established patterns of referral and practice can take time to change. Training health professionals in the delivery of CBT-I is an imperative to increase accessibility. However, using a stepped care approach, with lower intensity methods of delivering care as the starting point may help to reduce access problems [9]. There are not yet adequate studies incorporating a stepped care approach and so it is not clear whether failure at an entry level self-help CBT-I (e.g., online), reduces the success of subsequent therapist-led CBT-I.

3.1. Patients and their physicians need more choices for treating insomnia

The National Sleep Foundation recognizes that patients and their healthcare providers should have access to a wide variety of treatment choices. Patients should be empowered to ask for and receive the best possible help for their insomnia from their healthcare provider. GPs and physicians should receive education regarding evidence-based treatment options for insomnia.
4. Treatment principles

4.1. Codes of practice

Clinicians practicing in the field of behavioural sleep medicine should follow best practice procedures including following evidence-based behavioural and psychological interventions, and adhering to relevant codes of conduct.

4.2. Appropriate and recognised training and experience

A range of clinicians such as psychologists, nurses, sleep physicians, and GPs may work in settings where it is helpful to offer CBT-I. Insomnia usually does not resolve with general psychotherapy [37] and so clinicians should have specific training in the theory and delivery of CBT-I. Competency in CBT-I also requires the clinician to be versed in the science of sleep; content that is not typically well covered (if covered at all) in most mental health graduate training programs. Training may include attendance at professional CBT-I workshops, online CBT-I courses (e.g. through the Australian Psychological Society/ASA). Experience may include practice of CBT-I under supervision of an experienced clinician. Ability to recognize common comorbid mental (e.g., anxiety & depression) and physical (e.g., OSA, COPD, GERD, Cardiopulmonary disease, chronic pain) disorders is also important in order to facilitate appropriate onward referrals for treatment of these other conditions.

4.3. Thorough clinical assessment

A range of clinicians such as psychologists, nurses, sleep physicians, and GPs may work in settings where it is helpful to assess for insomnia disorder or symptoms. Insomnia is primarily diagnosed by clinical evaluation through a thorough sleep history, current 7–14 days sleep diary, and detailed medical, substance, and psychiatric history (including suicide/self harm risk assessments, particularly for patients presenting with depression). For effective delivery of psychological treatments, the sleep history is an important part of the clinical assessment and should cover the history of and current details regarding the sleep complaints, pre-sleep conditions, sleepwake patterns, other sleep-related symptoms, lifestyle factors, and daytime consequences. The history helps to establish the type and evolution of insomnia, perpetuating factors, and identification of comorbid medical, substance, and/or psychiatric conditions. If another sleep disorder is suspected (e.g., OSA, PLMs) a sleep physician should be involved and a polysomnographic sleep study conducted. If available, an objective measure of sleep (such as a home based, limited channel PSG or wrist actigraphy over a 1–2 week period) may be appropriate. This can identify patients who may sleep less than 5 h per night (for whom there are adverse outcomes, e.g., Ref [10]) and can also identify the extent of any sleep state misperception.

4.4. ‘Dose’ of CBT-I

CBT-I is most commonly administered in 4–8 individual or group therapy sessions at weekly or bi-weekly intervals (e.g., Refs. [8,13,27]). Insomnia comorbid with other psychiatric conditions and/or sleep disorders potentially may require more sessions with equal emphasis placed on interventions for each disorder. Specially trained mental health professionals are the appropriate professionals to deliver any psychiatric care.

4.5. Combined therapy (CBT-I plus medication)

Combined therapy (CBT-I plus medication) should be directed by (1) symptom pattern; (2) treatment goals; (3) past treatment responses; (4) patient preference; (5) cost; (6) availability of other treatments; (7) comorbid conditions; (8) contraindications; (9) concurrent medication interactions; and (10) side effects.

4.6. Self-help resources

There is evidence that CBT-I self help resources such as books and online programs can achieve effective results. Particularly when access to face-to-face CBT-I is limited, CBT-I via self-help should be considered.

4.7. Stepped care models (e.g., Ref. [9])

For straightforward insomnia experienced by adults with good motivation, and literacy skills, self-help/internet-based CBT-I may be a highly accessible, cost effective first-line treatment. For patients with need for greater support (e.g., comorbid mental and/or physical disorders), face-to-face CBT-I where the treatment can be tailored to the individual may be preferable.

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Conflict of interest

None declared.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2017.03.017.

References

Abstract

Study objective: Continuous Positive Airway Pressure (CPAP) is considered to be the gold standard treatment for obstructive sleep apnoea (OSA). CPAP monitoring systems allow tracking of patient CPAP adherence and treatment efficacy, by measuring residual sleep-disordered breathing, hours of CPAP use, and mask leak etc. The American Thoracic Society (ATS) published a position paper in 2013 highlighting issues of interpreting CPAP data such as a lack of consistency between CPAP manufacturers data algorithms, legal implications of CPAP data and implications for CPAP adherence. This paper extends on this work by investigating these issues in an Australasian context.

Method: A review of current literature on CPAP monitoring systems, privacy and security of CPAP data for major Australasian CPAP providers, and CPAP adherence was undertaken. A legal review was also commissioned for issues related to privacy and security of CPAP data.

Results: CPAP manufacturers’ utilize different algorithms for respiratory event detection and clinicians need to be aware the implications for interpreting CPAP data. Australasian CPAP manufacturers have created security/privacy policies with the intent to follow relevant legislation to protect patients’ CPAP data, however they do need to be constantly reviewed and updated to avoid data breaches and changes to agreements. No guarantees can be provided by the Australasian Sleep Association on CPAP manufacturers’ compliance with these policies and there is the potential for some degree of liability for physicians and CPAP providers associated with CPAP data. Lastly, providing patients with feedback on their CPAP usage and OSA management appears to have positive influence CPAP adherence.

Conclusions: CPAP data provides many opportunities to increase OSA patient care and to help patients self-manage this chronic condition. However, issues relating to lack of standardization of CPAP parameters, privacy, security, and legal implications will need to be managed in this changing technologic and clinical environment.

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Introduction

Obstructive sleep apnoea (OSA) is a very common sleep disorder characterized by repeated closure or partial closure of the upper airway during sleep. Untreated OSA is associated with symptoms of tiredness and excessive daytime sleepiness as well as several negative health outcomes, including hypertension and vascular disease, impaired cognition and mood, an increased risk of motor vehicle accidents and increased mortality. Continuous positive airway pressure (CPAP) is considered to be the gold standard treatment for OSA and evidence suggests that CPAP treatment reduces cardiovascular mortality and morbidity [12], daytime sleepiness [3], and the risk of motor vehicle accidents [4] in patients with OSA. Despite the advantages of CPAP treatment, adherence to CPAP is often suboptimal with approximately 50% of patients being non-adherent in the long term [5]. CPAP manufacturers have made advancements in recent years by implementing internal and
remote monitoring systems to assess CPAP adherence and efficacy. These CPAP monitoring systems are able to record CPAP efficacy (residual sleep-disordered breathing), hours of CPAP use, and mask leak for example. However, whilst many of these measures seem intuitively useful, there are no published standards on how to use the data from monitoring systems, legal implications for physicians, CPAP providers or patients using these systems, nor have there been many studies to assess if monitoring patients' CPAP data improves adherence. The American Thoracic Society published a position paper on the issue in 2013 [6] to facilitate greater understanding by providers on how to interpret CPAP tracking system data, and to stimulate further research into CPAP monitoring systems' impact on patient adherence and how they affect OSA outcomes. Whilst this paper has done well to highlight the emerging issues and lack of research on CPAP monitoring, it has not provided guidelines on how sleep physicians should use this data, how frequently they should monitor it, or what the legal responsibilities are for the sleep physician or CPAP supplier if a patient is not optimally treated or adherent to treatment. This current position paper extends on the ATS's paper for use in Australia and New Zealand. It will discuss the current evidence for the use of CPAP monitoring data, especially in regards to privacy and medico-legal issues and the benefits of incorporating it into clinical practice, in order to develop Australia and New Zealand clinical practice standards on how best to manage CPAP data.

**CPAP monitoring systems overview**

CPAP monitoring systems measure a range parameters including hours of CPAP use (length of time the device is turned on and a measurable breathing signal is detected, irrespective of ramp pressure setting or size of mask leak), residual sleep disordered breathing (event detection) and mask leak [6]. They can also record patient pressure settings, and the type of Positive Airway Pressure (PAP) the patient is receiving. The information is recorded on a computerized Secure Digital (SD) card or USB storage device, which the patient brings to their sleep physician or CPAP provider, or can also be uploaded through wireless technology to a secure PAP website. The information can then be reviewed by the PAP therapist or sleep physician to identify any issues with PAP (or lack thereof) and help guide how well the patient's OSA is managed.

When reviewing CPAP monitoring system data, providers need to be aware that PAP devices all use different manufacturer-specific propriety algorithms to calculate OSA parameters such as Apnoea Hypopnea Index (AHI), leak and other measures [7]. There are several limitations to this lack of standardization between PAP device algorithms, including the lack of standard definition for parameters, which has implications for interpretation of the data, the ability to compare CPAP data between different manufacturer devices and the aim of standardizing treatment management [6]. For example, a ResMed device (S9 model) scores an apnoea where the 2 s moving average root mean square ventilation, as measured by an internal flow sensor, falls below 25% of long term ventilation for 10 s [6]. This compares to Fisher and Paykel Healthcare's Infosmart™ Web software, which identifies flow patterns to score respiratory events and defines apnoea as an 80% reduction in flow relative to baseline as determined by previous breaths (see Table 1 below for Event Detection Algorithms differences between manufacturers [6]). Mask leak is also calculated differently between the different manufacturers’ CPAP machines as outlined in Table 2. These discrepancies between manufacturers’ parameter algorithms highlight some difficulties with interpretation of non-standardized CPAP data. They also present a challenge for incorporating CPAP data into patients’ electronic medical record unless they use the PAP providers CPAP Management Software. Taken together, these issues with discrepancies in CPAP algorithms indicate that work should be undertaken to standardize these measures to facilitate interpretation and clinical understanding of PAP data and its management.

In addition to differences in PAP parameter algorithms, the Apnoea Hypopnea Index (AHI) reported by CPAP tracking systems is not the same parameter as AHI reported by polysomnography (PSG) [7]. AHI as reported by PAP devices provides averaged data for the residual AHI and residual Apnoea Index (AI) whilst a person is using CPAP, not necessarily total sleep time as in Polysomnography (PSG). PAP devices calculate AHI based only on reduction of airflow as measured by an internal flow sensor, in contrast to PSG, which uses respiratory flow patterns derived from nasal pressure and thermistor, EEG arousal, abdominal and thoracic movement and arterial oxygen desaturation. Hence, PAP reported AHI or AI is not a true measure of PSG derived AHI, which is not always understood in clinical practice or by patients. For example, Berry et al. [8] compared respiratory events as calculated by PSG or a PAP device (ResMed Auto M Series, Philips). They found that when comparing 148 studies, AHI and AI correlated well, but not Hypopnea index (HI). They also found that the PAP data tended to overestimate AHI when PSG derived AHI was low, but underestimate the AHI when PSG derived was high. The authors concluded that there was relatively good agreement between the two measures for apnoea detection, but not as good for detecting hypopneas. This finding has been replicated [9,10], suggesting that PAP devices lack of oximetry, respiratory effort etc. make it difficult for these devices to accurately identify more subtle respiratory events and lead to some discrepancies between PSG outcomes. Based on these findings, the ATS position from a clinical perspective is that PAP derived AHI can be used when AHI is low (<10 events per hour) or high (>20 events per hour), but when residual AHI is intermediate, the PAP derived AHI and AI are difficult to interpret and should be examined clinically, taking into account the individual patient's context [6,8]. Therefore any patient with a residual AHI >10 events per hour should be clinically reviewed to identify any issues with PAP fit etc. (mask leak, incorrect pressure) or additional disorders of sleep fragmentation, such as periodic limb movements, which can cause irregularities in depth of breathing, classified as hypopneas by many proprietary algorithms. Patients who appear well treated but complain of residual OSA symptomatology, such as excessive daytime sleepiness, should also be reviewed, as CPAP data is not a substitute for clinical expertise.

Due to the different derivation of AHI from PSG versus CPAP data, the ATS position statement [8] recommends that more concise terminology is used for PAP derived AHI. The authors from this ATS position paper recommend that the terminology be changed to residual AHIFlow to highlight this AHI is only based on reduction of airflow. In addition, they recommend that studies need to be conducted to determine what is a clinically meaningful AHIFlow based on outcome data, as it is unknown how this measure can be reliably interpreted to identify how well a patient's OSA is actually managed. There is also a lack of research on clinical outcomes of PAP derived AHI and improvements in OSA symptomatology such as cardiovascular, cognitive and daytime functional outcomes. The ASA supports this recommendation and encourages CPAP manufacturers to integrate this new terminology into their PAP devices and also for further research to be conducted on how AHIFlow is associated with clinical outcomes. Manufacturers are also encouraged to collaborate to work towards providing a standardized methodology and criteria for identifying and characterizing flow limited events.
Table 1
Event detection algorithms.

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>Apnoea event detection</th>
<th>Hypopnea event detection</th>
</tr>
</thead>
<tbody>
<tr>
<td>ResMed Unit (90 Model)</td>
<td>Apnoea is defined when the 2-s moving average root mean square ventilation (based on a pneumotachograph) falls below 25% of the long-term ventilation for 10-s.</td>
<td>Hypopnea is defined when all the following conditions are met: 1. The 12-s moving average root mean square ventilation falls below 50% of the long-term ventilation 2. The hypopnea is not immediately followed by an apnoea 3. The hypopnea contains one or more partially obstructed breaths.</td>
</tr>
<tr>
<td>Philips unit (System One model)</td>
<td>Apnoea is detected after a moving window of 3–4 min is established and flow decreases by more than 80% for at least 10 s.</td>
<td>Hypopnea is detected when moving window of 3–4 min is established and flow decreases by 40–80% for at least 10 s.</td>
</tr>
<tr>
<td>DeVilbiss Healthcare IntelliPAP unit (SmartCode remote data retrieval system)</td>
<td>A reduction in a flow signal of &gt;90% of the baseline for 10 s.</td>
<td>A reduction in a flow signal of &gt;50% of the baseline flow for 10 s.</td>
</tr>
<tr>
<td>Fisher &amp; Paykel Healthcare</td>
<td>&gt;80% reduction in flow relative to a baseline determined from previous breaths.</td>
<td>&gt;40% reduction in flow relative to a baseline determined from previous breaths.</td>
</tr>
<tr>
<td>Infosmart™ Web software</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


Table 2
Continuous passive airway pressure leak measurements.

<table>
<thead>
<tr>
<th>CPAP Manufacturer</th>
<th>How leak is measured</th>
<th>Large leak threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>Philips</td>
<td>Intentional leak subtracted from total flow</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Leak condition where the leak level exceeds a present</td>
<td></td>
</tr>
<tr>
<td></td>
<td>“flow vs. pressure” curve</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(the averaged leak through all mask exhalation ports at</td>
<td></td>
</tr>
<tr>
<td></td>
<td>various pressure)</td>
<td></td>
</tr>
<tr>
<td>ResMed</td>
<td>Unintentional leak (device flow-intentional leak) + mouth leak.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>95th percentile leak</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(&lt;24 L/min with nasal resistance and &lt;36 L/min with full face interface)</td>
<td></td>
</tr>
<tr>
<td>Fisher &amp; Paykel Healthcare</td>
<td>Total leak, including mask and exhaust flow from mask</td>
<td></td>
</tr>
<tr>
<td></td>
<td>A leak value of &gt;60 L/min</td>
<td></td>
</tr>
<tr>
<td>DeVilbiss Healthcare</td>
<td>Records high leak flow time</td>
<td></td>
</tr>
<tr>
<td></td>
<td>A leak value of &gt;95 L/min</td>
<td></td>
</tr>
<tr>
<td>IntelliPAP</td>
<td>the leak was above 95 L/min</td>
<td></td>
</tr>
</tbody>
</table>


Summary of ASA position on CPAP tracking systems data

- Providers and clinicians should be aware of the different PAP manufacturers’ algorithms for event detection and how they impact CPAP data.
- Steps should be put in place to standardize the algorithms across PAP manufacturers.
- PAP derived AHI is not the same measure as PSG derived AHI and PAP manufacturers should adopt a new name for this PAP parameter as AHIFlow.
- From a clinical perspective PAP derived AHI (AHIFlow) is supplementary to a comprehensive clinical assessment. PAP derived AHI could be considered clinically useful if it is either very low (AHI < 10) or high (AHI > 20), however control of OSA needs to be reviewed if there is a discrepancy between symptoms and PAP data. For example, when PAP derived AHI is >10 or if patients continue to report OSA symptomatology despite low PAP derived AHI — as it is not known exactly how AHIFlow correlates with clinical outcomes.
- Research needs to be considered to investigate the correlation between AHIFlow and clinical OSA outcomes.

Privacy and security policies for CPAP data

The convenience and technological advancement that CPAP data provides, also present privacy and security concerns for all who access, retain and use the data collected in relation to patients. PAP data is either stored on a portable memory card/stick and can be uploaded to a CPAP data management program (or electronic medical record at a patient’s CPAP review appointment), or it can be transmitted at various intervals via a wireless modem in the PAP machine to the PAP provider’s data management program or host website. The location of the stored data and the corresponding legislation that protects the data, along with how the data is protected during transmission have become important issues that need to be managed carefully. This section will highlight these security and privacy concerns for the CPAP data.

PAP companies operating in Australia and New Zealand, such as Philips, ResMed and Fisher & Paykel Healthcare, all have different approaches to PAP data management and depending on where they store their ‘cloud’ data, have to comply with different legislation. All of these companies operating in Australia need to comply with the 1988 Australian Privacy Act, but if the CPAP data is stored in the USA, for example, there are additional legislative issues with which the company must comply. Whilst it is common for people to be concerned with Australian data being hosted overseas, healthcare data in the USA has been especially protected under the Health Insurance Portability and Accountability Act (HIPAA) of 1996. As part of HIPAA, the Privacy Rule (effective April 24, 2003) and Security Rule (effective April 21, 2005) were implemented. The Privacy Rule established national standards to protect individuals’ medical records and other personal health information. The Security Rule specifies a series of administrative, technical, and physical security procedures to be used to assure the confidentiality, integrity, and availability of electronic health information.

This legislation benefits Australian CPAP companies hosting data in the USA in several ways, firstly by using the specific guidance related to the recommended security controls environment. The term “control environment” describes the defined actions and protections that a company should exercise when processing personal and sensitive data. By comparison an “ad-hoc environment” would not have defined controls that are followed, and important protective actions could be skipped.

The US government provided detailed guidance to companies to comply with the HIPAA Security Rule in 2008 in the form of the...
Quality, security and access

An entity who holds the private information of others needs to take all reasonable steps to ensure that the information held is accurate and up to date as much as is possible. They need to ensure that all reasonable steps are taken to protect the information from misuse, interference or loss. Further reasonable steps need to be taken to ensure there is no unauthorized access, modification or disclosure of the private health or medical information of patients or others. Importantly, the rights largely reside with the patient. Access to the information about a person must in general terms be provided when they ask for it. There are exceptions to this principle, although they arise only in limited circumstances.

Summary of ASA position on Australia’s three major PAP providers’ privacy policies

- Philips, ResMed and Fisher & Paykel Healthcare have created policies with the intent to follow relevant legislation to protect patients PAP data (Privacy Act 1988 in Australia, HIPAA, and New Zealand Privacy Policy) and have measures in place to protect patient’s data. However, these do need to be constantly reviewed and updated to avoid data breaches and changes to agreements. We cannot ensure that Philips, ResMed and Fisher & Paykel Healthcare comply with the law in relation to the data they collect we do double-check what they do and whether it complies with the law or is appropriately up to date.

Cloud storage

CPAP data in general terms is capable of storage on a cloud-based platform. A feature of cloud-based platforms is that the information is stored geographically where the information is stored. The person or entity seeking to store the information needs to ensure as reasonably as is possible that the cloud storage provider does not breach the privacy principles. A theme in the privacy principles is ensuring, from the perspective of an Australian entity, that the same or similar sets of principles with regard to the treatment of patient’s private information applies where the information is stored. Whilst this provides significant logistical and practical challenges, it is an important feature of compliance.
Table 3
Comparison of privacy polices between the three major PAP providers operating in Australia and New Zealand.

<table>
<thead>
<tr>
<th>Name of CPAP Data Management Program</th>
<th>Philips</th>
<th>ResMed</th>
<th>Fisher and Paykel Healthcare (FPH)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location of stored cloud data</td>
<td>USA</td>
<td>USA</td>
<td>May be outside of Australia and New Zealand.</td>
</tr>
<tr>
<td>Privacy Legislation to comply with</td>
<td>• 1988 Privacy Act in Australia</td>
<td>• 1988 Privacy Act in Australia</td>
<td>• 1988 Privacy Act of Australia</td>
</tr>
<tr>
<td></td>
<td>• Applicable state legislation (for public health entities)</td>
<td>• Applicable state legislation (for public health entities)</td>
<td>• Applicable state legislation (for public health entities)</td>
</tr>
<tr>
<td></td>
<td>• HIPAA Privacy and Security Rules in USA</td>
<td>• HIPAA Privacy and Security Rules in USA</td>
<td>• HIPAA Privacy and Security Rules in USA</td>
</tr>
<tr>
<td></td>
<td>• MyAir Australia is covered by the privacy policy for users anywhere</td>
<td>• MyAir Australia is covered by the privacy policy for users anywhere</td>
<td>• 1993 Privacy Act of New Zealand</td>
</tr>
<tr>
<td></td>
<td>from North, South and Central America, Canada, Europe and Brazil, and</td>
<td>from North, South and Central America, Canada, Europe and Brazil, and</td>
<td></td>
</tr>
<tr>
<td></td>
<td>as such is governed by laws of New South Wales, Australia.</td>
<td>as such is governed by laws of New South Wales, Australia.</td>
<td></td>
</tr>
<tr>
<td>Data Export Service Security</td>
<td>• CPAP report is encrypted during transmission</td>
<td>• Personal data is encrypted during transmission also at rest</td>
<td>All data is encrypted in transit, and stored data is</td>
</tr>
<tr>
<td></td>
<td>• Secure transfer protocol and accessed with username and password</td>
<td>• Employers now have an annual assessment of security and privacy</td>
<td>encrypted at rest</td>
</tr>
<tr>
<td></td>
<td>• Independent security audit yearly</td>
<td>• controls which are based on Privacy Law from Australia and other</td>
<td>Staff are trained on their obligations when handling data</td>
</tr>
<tr>
<td></td>
<td>• Access to data center strictly controlled</td>
<td>• jurisdictions</td>
<td>to comply with relevant legislation</td>
</tr>
<tr>
<td></td>
<td>• Data is encrypted at rest</td>
<td>• InView is the most audited and controlled system in ResMed at</td>
<td>Only a small restricted/controlled team have access to</td>
</tr>
<tr>
<td></td>
<td>• Routine vulnerability scans performed</td>
<td>present Quarterly and ad-hoc vulnerability scans completed using Nexpose</td>
<td>data, and only</td>
</tr>
<tr>
<td></td>
<td>• Data retained for 7 years (minimum)</td>
<td>• The source code is now assessed for security and vulnerabilities</td>
<td>as required to maintain the system</td>
</tr>
<tr>
<td></td>
<td>• Philips employees undergo a background check and have security</td>
<td>• using a mixture of automated and specialist reviews</td>
<td></td>
</tr>
<tr>
<td></td>
<td>in place for departing employees</td>
<td>• Only a small restricted/controlled team have access to</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Modems remain the property of Philips at all times</td>
<td>• Health IT Board (NHITB)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Privacy in the Assessments and Security Risk Assessments of</td>
<td>• ResMed security and privacy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>application and application enhancements</td>
<td>and compliance with Privacy Principles APP-8 and APP-11.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Privacy (Corporate Binding Rules) and Security Policies</td>
<td>• MyAirIT is the most audited and controlled system in ResMed at</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Privacy and Security training</td>
<td>present Quarterly and ad-hoc vulnerability scans completed using Nexpose</td>
<td></td>
</tr>
<tr>
<td>Who owns the data?</td>
<td>• Personal data for consumers in Australia is owned by the data subject</td>
<td>• Personal data for consumers in Australia is owned by the data subject</td>
<td>Ownership of data remains with the originator</td>
</tr>
<tr>
<td>Disclosure to third parties</td>
<td>No, with exceptions</td>
<td>No, with exceptions</td>
<td>No, with exceptions:</td>
</tr>
<tr>
<td></td>
<td>• Agreement allows for disclosure of de-identified data to Philips or</td>
<td>• Only shared to the extent reasonable necessary to perform their</td>
<td>Government authorities or regulatory bodies, where FPH are</td>
</tr>
<tr>
<td></td>
<td>3rd parties</td>
<td>functions and they will not be authorized to use it for any other</td>
<td>under legal obligation to do so</td>
</tr>
<tr>
<td></td>
<td>• Unless third parties need access to perform duties in the context of</td>
<td>function, unless user has consented to such disclosure</td>
<td>Third parties engaged by FPH to provide services in</td>
</tr>
<tr>
<td></td>
<td>providing the application to you</td>
<td>• May also share personal information in the event that the business of</td>
<td>connection with the uses of personal</td>
</tr>
<tr>
<td></td>
<td>• Or required to by law enforcement authorities</td>
<td>ResMed is transferred to another entity, by way of sale, merger etc.</td>
<td>information about client (e.g. provide CPAP information to healthcare provider, physician etc.)</td>
</tr>
<tr>
<td></td>
<td>• Agreement allows for Philips to change the conditions of the</td>
<td>• May be accessed by law enforcement authorities or courts if required</td>
<td>As otherwise required by Law</td>
</tr>
<tr>
<td></td>
<td>contract without prior notice to customers</td>
<td>to by law</td>
<td></td>
</tr>
<tr>
<td>Consent process</td>
<td>• For DreamMapper, consent is gained by creating account and agreeing</td>
<td>• By using the MyAir website and providing consent to the terms and</td>
<td>Use of website signifies agreement to privacy policy</td>
</tr>
<tr>
<td></td>
<td>to the privacy notice</td>
<td>conditions during the registration process</td>
<td>Healthcare providers are responsible for obtaining</td>
</tr>
<tr>
<td></td>
<td>• If patient wants to delete data they need to contact Health Care</td>
<td>• ResMed does honor the ‘right to be forgotten’ and patients can have</td>
<td>obtaining consent from patients before providing their</td>
</tr>
<tr>
<td></td>
<td>Provider. If patient does not want to use DreamMapper anymore, they</td>
<td>personal data completely removed if requested.</td>
<td>personal information to InfoSmart Web</td>
</tr>
<tr>
<td></td>
<td>can delete the DreamMapper App and any DreamMapper data will be</td>
<td>• May be accessed by law enforcement authorities or courts if required</td>
<td></td>
</tr>
<tr>
<td></td>
<td>deleted from mobile device</td>
<td>to by law</td>
<td></td>
</tr>
</tbody>
</table>

For DreamMapper, consent is gained by creating account and agreeing to the privacy notice. If patient wants to delete data they need to contact Health Care Provider. If patient does not want to use DreamMapper anymore, they can delete the DreamMapper App and any DreamMapper data will be deleted from mobile device.
proceedings associated with CPAP data. What is the expectation of sleep physicians and CPAP providers to monitor CPAP data long term, after the acute treatment phase has passed? There are currently no guidelines in place to manage this and due to the complexity of the issue it does require legal counsel to explore and put in place appropriate guidelines and standards for practitioners in the field to follow, to avoid the prospect of legal ramifications. There is no way to remove all prospect of liability. However, good risk management and prudent insurance practices will be essential for a practice to ensure that if a claim is made maximum protection both under the law and commercially is provided to a practice or for a practice to ensure that if a claim is made maximum protection both under the law and commercially is provided to a practice or treating professional.

Also, there could be financial legal issues involved with access to CPAP data. In the USA, Centers for Medicare and Medicaid Services (CMS) regulate rebates for CPAP machines. Patients can lose access to reimbursement for their PAP device if they are non-adherent according to the CMS guidelines (PAP usage of greater than or equal to 4 h per night on 70% of nights during a consecutive thirty day period anytime during the first 3 months of initial usage). Employers in the USA are also able to access PAP data on employees in professions such as commercial driving, and employees can be fired if they do not adequately adhere to this definition of CPAP adherence. These guidelines are not followed in Australia, but similar policies could potentially be brought in by insurance companies and employers, despite lack of evidence for this definition of adherence and associated clinical outcomes. The evidence for this will be expanded on in the next section, but it does highlight some possible legal implications for patients that have not been explored in Australia.

Summary of ASA position on legal implications of CPAP data

- There is the potential for some degree of liability for Physicians and CPAP providers for accidents that occur if patients are on CPAP. Currently, there are no guidelines for the frequency of review of CPAP data management programs now that this information can be wirelessly updated daily. The ASA presently endorses that CPAP data should be reviewed either by the sleep clinic or CPAP provider at the regular time intervals as reported in the ASA “Best Practice Guidelines for CPAP Therapy” [11] (at 7, 30 and 60 days, then at 12 months, and yearly after that due to the chronic nature of OSA). This might be the de facto standard; until a legal review has been conducted to what extent providers should be monitoring CPAP data outside of regular review times. However, as documented earlier in this position statement, CPAP data should not be assessed in isolation. Interpretation of CPAP data needs to be performed within the context of an overall clinical review of the patient. The ASA recommends that clinicians review is of primary importance in the assessment of OSA therapy and that CPAP data is supplementary to this comprehensive clinical assessment. Once the patient is stabilized on therapy, the clinical review may be undertaken either by the sleep physician/specialist, or another medical practitioner (e.g. primary care physician) with input from the CPAP provider, with referral back to the sleep specialist as is medically necessary.

Australian insurance companies and employers should have an interest in customer/employee adherence to PAP, however using CPAP data according the CMS definition does not have adequate research to support its definition and how it translates to clinical outcomes.

CPAP data and impact on CPAP adherence

The implementation of CPAP tracking systems provides a unique opportunity to monitor and support patients with their successful use of CPAP. Sleep physicians and CPAP therapists can now ‘check in’ on how adherent patients are when patients bring their data card in to their review CPAP appointment, or via wireless technology. Whilst this new technology provides opportunities to track patients between review appointments (via wireless transmission of PAP data) no guidelines have been published on how sleep physicians/providers should use patient data. In particular, there are no guidelines on how often patients should be monitored remotely and have PAP data checked; or if a patient is well ‘controlled’, whether they need to be monitored over time. Also, with Physicians and Providers having access to PAP data it brings up the issue of what is the duty of care to the patient to review this information. These issues will be explored throughout this section.

Overall adherence to PAP is poor, with only 50% of patients adhering to therapy long term [5]. Whilst studies investigating adherence to PAP with the help of CPAP data are limited, several recent studies are showing promising results for increasing adherence rates [12]. Fox et al. [13] utilized PAP data to conduct a telehealth intervention by responding quickly to any difficulties such as high mask leak, high residual AHI etc. and phoned patients to rectify the problem. Patients in the telemedicine/PAP data arm were found to have greater mean PAP adherence than the standard care patients after 3 months (191 min compared to 105 min). A similar study investigated adherence with wireless telemonitoring

<table>
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<tr>
<td>- Philips recognize and take seriously their responsibility to protect the personal and sensitive data you entrust to Philips from loss, misuse or unauthorized access, and use a variety of security controls, technologies and organizational procedures to help protect personal and sensitive data.</td>
<td>- ResMed will take all reasonable measures to protect personal data and will comply with the applicable data protection law.</td>
<td>- Will take all reasonable and appropriate steps to protect secure information but FPH will not be liable in anyway for a breach of security or unintended loss or disclosure of information due to the website being linked to the internet.</td>
<td></td>
</tr>
<tr>
<td>- Philips specifies absolutely no liability of any responsibility within the fullest extent of the law.</td>
<td>- Despite these security measures, there is no guarantee of absolute security with respect to information sent through the Internet. If ResMed is aware of a personal data breach, patient will be contacted in a timely fashion.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- ResMed performs Mandatory Data Breach Notification for all jurisdictions, regardless of whether that country has enacted legislation on this matter.</td>
<td></td>
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</table>
of CPAP data (AutoSet Spirit Flow Generator Unit: ResMed) in newly diagnosed OSA patients [14]. Stepnowsky et al. [14] found after 2 months, participants who were able to view his/her CPAP usage via an interactive website had higher usage of PAP of 4.1 h per night, compared with participants on usual care of 2.8 h PAP usage per night. Lastly, Kuna et al. [15] recently published a study indicating CPAP adherence is significantly improved when patients have Web access to information about their use of PAP treatment. Interestingly, they found that the addition of a financial incentive for patients to use PAP in the first week of treatment did not significantly improve PAP usage further.

Philips also released a White Paper on their ‘in house’ PAP data trial for patients, using a patient engagement tool called DreamMapper (formerly SleepMapper) [16]. DreamMapper not only allows patients to access their CPAP data via an app, it also uses psychological theories of behavior change to assist patients to engage with PAP treatment. DreamMapper interfaces with Philips EncoreAnywhere software that is managed by the CPAP provider or sleep physician, and provides patients with access to their adherence and therapy data, as well as an education tool. In this study, researchers investigated adherence by comparing patients in the EncoreAnywhere database who were using DreamMapper to a similar group of patients who did not use the DreamMapper App. When comparing 15,242 patient records, they found that patients in the DreamMapper group (n = 7641) had a higher adherence rate of 78% when compared to the Standard Care Group (n = 7801) adherence rate of 56% (p < 0.0001). Average use of PAP in the DreamMapper group was 4.5 ± 2.3 h, compared to Standard care usage of 3.1 ± 2.6 h. In addition, DreamMapper also appeared to assist “struggling users” (those who were using CPAP less than 2 h on average per night in the first two weeks) more so than did Standard Care, as 33% of DreamMapper users who were classed as “Struggling Users” went on to meet the CMS adherence guideline (4 h per night, 70% of nights) compared to only 11% of Standard Care users. A similar White Paper was recently published by Philips with Australian data and also suggests that the use of the DreamMapper App improves adherence to CPAP [17]. Whilst Philips sponsored these studies, it does suggest that providing patients with CPAP data via an app can benefit patient adherence to CPAP. Even in patients who are classed as “struggling users”. Taken together, these studies suggest that when patients have the opportunity to access information about their PAP usage, their adherence to treatment is positively impacted.

Whilst increasing CPAP adherence is important, the level of CPAP adherence to provide clinically significant benefit remains debatable. According to the Centers for Medicare and Medicaid Services (CMS) in the U.S., the definition of PAP adherence is PAP usage of greater than or equal to 4 h per night on 70% of nights during a consecutive thirty-day period anytime during the first 3 months of initial usage. In the USA, patients can lose access to reimbursement for their PAP device if they are not adherent according to the CMS guidelines.

Despite this well used CMS definition of adherence, research supporting this definition is limited. Marin et al. [2] investigated cardiovascular events in severe untreated OSA patients, followed for over 10 years. Results suggest that treatment with PAP for 4 h or more per night reduced the raised cardiovascular risk seen in untreated patients. However, as part of the study protocol, if the usage of CPAP was less than 4 h per night upon review, CPAP treatment was stopped after a period of time, so it is unclear if less than 4 h of CPAP treatment was at all beneficial for cardiovascular risk. Another study by Campos-Rodriguez et al. [17] suggested that cardiovascular morbidity and mortality rates vary according to CPAP use. Whilst patients who use CPAP for >6 h per night had the highest survival rate, even patients using CPAP between 1 and 6 h had survival odds comparative to people who used CPAP for more than 6 h per night after approximately 48 months. This suggests that the higher PAP usage per night the better, however cardiovascular risk can still be improved significantly when patients use PAP for at least 1 h per night.

Other studies suggest a dose–response relationship between PAP usage and outcomes. For example, Weaver et al. [18] found that the longer PAP was used per night, the greater the improvement in both subjective and objective measures of sleepiness. However, this relationship flattened at 7 h PAP usage per night, and only 30% of patients treated with PAP had normal MSLTs after treatment, indicating that some treated patients were still sleepy. Zimmerman et al. [19] reported similar dose–response relationships when it came to verbal memory assessments, and Antic et al. [20] found that even when patients were maximally compliant with CPAP treatment as tracked by CPAP data, patients did not improve on all neurobehavioral measures such as Functional Outcomes of Sleep Questionnaire (FOSQ) and Epworth Sleepiness Scale (ESS). However, the authors did find that sleepiness scores on the ESS had greater improvement with increased usage of CPAP.

Taken together, there is strong evidence that patients with OSA who adhere to CPAP treatment experience benefit across the board, in terms of cardiovascular health, neurobehavioral and cognitive functions. There does appear to be a dose–response relationship between these improvements and CPAP usage, however the optimal level of CPAP treatment has not been conclusively proven and it would appear that some patients using PAP for only a few hours per night can still experience benefit. Patients that are optimally treated may also still report as “sleepy” and may still have an abnormal MSLT, however, increasing adherence to PAP provides patients with the best chance of optimally managing the symptoms of OSA. Initial research into utilizing PAP data to increase adherence to treatment is promising and should be researched further and incorporated into clinical practice.

Summary of ASA position on CPAP data use for improving adherence

- Providing patients with feedback on their CPAP usage and OSA management appears to have positive influences on adherence to CPAP, although this does require further evidence.
- Patients should be encouraged to engage in CPAP data management programs or applications, such as DreamMapper (Philips) or MyAir (ResMed) to improve their usage of CPAP and overcome any obstacles such as mask leak.
- CPAP data should be checked by the CPAP provider in accordance with the current ASA Position on Guidelines for CPAP provision, i.e. at approximately 7, 30, 60 days and then 12 months post treatment initiation. Patients should then be reviewed every 12 months after that to check on ongoing adherence, given that long term adherence rate is approximately 50% and OSA treatment is generally required long term. Reports are to be provided or made available to the Sleep Physician/Sleep Clinic (in accordance with privacy guidelines) to provide a summary of this information including average usage or night-to-night usage, residual PAP derived AHI and Mask Leak, to facilitate Physician follow up, particularly if the patient is not optimally treated (not wearing mask for time spent asleep, large mask leak, side effects, etc.). This is especially important at the commencement of treatment. It is acknowledged that involvement of the patient’s primary care provider (and potentially CPAP provider) in the follow up clinical assessment of patients stabilized on CPAP therapy is often necessary – as long as there is a mechanism for referral back to the treating specialist in the case of clinical problems.
● Patients should be encouraged to adhere to PAP treatment at all times when asleep due to the dose–response relationship for most outcomes. However, physicians and PAP providers should still be supporting patients whose adherence is <60%, as some research suggests that even using PAP > 1 h per night can provide benefit to some patients.

● CPAP data should be used to help stratify access to review for those that need it most, such as those with low adherence, AHI >10, mask leaks, residual OSA symptomatology.

Summary

CPAP data has many benefits including the ability to provide objective data on patients’ CPAP adherence and residual sleep disordered breathing, a way to engage patients in increasing their CPAP usage and allowing CPAP providers and physicians to intervene early if patients are having difficulties/not adhering to treatment by monitoring through wireless technology. However, this ability to monitor and record CPAP data also raises concerns with privacy issues for patient data, along with possible legal implications for physicians, PAP providers and patients in the event of accidents, if patients are not adherent or properly managed according to CPAP data. With any information that is stored on the Internet, there are always threats to privacy as no information can ever be completely safe and secure. There is also always a possibility that records you hold about patients can be the subject of subpoena in legal proceedings. In this context the laws of Privacy will often be waived. This is particularly so where the person whose information is held is involved in the case. This provides the Australia and New Zealand sleep community with opportunities to do further research on CPAP data parameters and how they correlate with clinical outcomes, improving CPAP adherence with CPAP data, and also to conduct a proactive legal review on implications of CPAP data for physicians, PAP providers and patients. There are also significant opportunities to provide better management of OSA for patients by being able to identify early barriers to adherence and to help them by monitoring data externally and providing quick feedback. In summary, CPAP data provides many opportunities for OSA patient care and to help patients self-manage their chronic condition. In addition, there are issues that relate to privacy, security, legal implications and the lack of standardization of PAP parameters and clinical implications that will need to be managed in this changing technologic and clinical environment.

Acknowledgements

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Conflict of interest

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The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: doi:10.1016/j.sleep.2017.03.018.

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