Technology *Report*

A Review of Guidelines for Referral of Patients to Sleep Laboratories

Canadian Coordinating Office for Health Technology Assessment

A Review of Guidelines for Referral of Patients to Sleep Laboratories

David Hailey MSc PhD Grad RIC[1]
Khai Tran MSc PhD[2]
Robert Dales MD Diplomate ABSM MSc[3]
Shaila Mensinkai MA MLIS[2]
Lynda McGahan MSc[2]

May 2005

[1] Department of Public Health Sciences, University of Alberta, Edmonton AB
[2] Canadian Coordinating Office for Health Technology Assessment (CCOHTA), Ottawa ON
[3] Division of Respirology, Department of Medicine, University of Ottawa, Ottawa ON

Reviewers

These individuals kindly provided comments on this report.

External Reviewers

Charles F.P. George, MD FRCPC
Professor of Medicine
University of Western Ontario
London ON

A.C. Peter Powles, MBBS FRCPC
Chief of Medicine
St. Joseph's Health Centre
Toronto ON

Michael Littner, MD
Professor of Medicine
VA Greater Los Angeles Healthcare System
and David Geffen School of Medicine at UCLA
Los Angeles, California

CCOHTA Scientific Advisory Panel Reviewers

Gina Bravo, PhD
Professor,
Department of Community Health Sciences
Faculty of Medicine,
University of Sherbrooke
Sherbrooke QC

Ruth L. Collins-Nakai, MD MBA FRCPC FACC
Cardiologist
Edmonton AB

Authorship

Acknowledgements

Conflicts of Interest

No conflicts of interest were declared by any of the authors.

Technology Name

Examinations performed in sleep laboratories

Disease or Condition

Conditions include obstructive sleep apnea in adults and in children, other conditions that produce sleep disturbances, respiratory disorders, sudden infant death syndrome, insomnia, depression with insomnia, narcolepsy, parasomnias, restless legs syndrome and periodic limb movement disorder.

Technology Description

Patients who stay overnight in a sleep laboratory are monitored while asleep using polysomnography (PSG), which measures and records multiple physiological parameters. Investigation of excessive daytime sleepiness is undertaken in sleep laboratories using the multiple sleep latency test or the maintenance of wakefulness test.

The Issue

Sleep laboratories are specialized facilities and demand for their services is increasing. Health care providers need assurance that the examinations and services in sleep laboratories are clinically appropriate.

Assessment Objectives

Our aim was to identify recommendations for the investigation of individuals in sleep laboratories, as made in guidelines prepared by professional bodies; and to review the nature, quality and relevance of the evidence cited in support of these recommendations.

Methods

Using a literature search, we identified the guidelines that were prepared by professional bodies on the use of sleep laboratory investigations for sleep disorders. For each application, pertinent guideline recommendations for and against the use of sleep laboratory examinations were listed. The quality and relevance of evidence from primary studies cited in support of the guidelines were assessed.

Conclusions

- The reviewed guidelines contain detailed information for health professionals. Many recommendations are supported by studies on sleep laboratory applications.
- The evidence for some applications is of limited quality and the cited studies are not always directly relevant to the recommendations made. Several recommendations reflect consensus positions and no evidence is cited in support.
- Evidence of relatively good quality was provided for the use of sleep laboratory examinations in obstructive sleep apnea, though recommendations on this application differed.
- Evidence supporting recommendations on sleep laboratory testing in relation to sudden infant death syndrome, insomnia; and depression and insomnia is also of reasonable quality and relevance.
- Further good quality studies of many sleep laboratory applications are needed.

EXECUTIVE SUMMARY

The Issue

The demand for sleep laboratory services is increasing, with service rates varying among jurisdictions. Sleep laboratories are expensive, specialized facilities. Health care providers need assurance that the services performed in sleep laboratories are clinically appropriate.

Objective

Our objective was to review recommendations, made in guidelines prepared by professional bodies, for the investigation of individuals in sleep laboratories. The report identifies relevant recommendations and reviews the evidence cited in support.

Methods

Several electronic databases were searched for guidelines and associated reviews that appeared from 1992 onwards, on the use of sleep laboratory investigations for sleep disorders. The searches were not limited to the English language and were updated periodically. Guidelines and associated reviews were selected and recommendations related to the selection of patients for examination in sleep laboratories were identified. Publications cited in support of the recommendations were reviewed considering the type and design of the study; population; and quality and relevance of the evidence. For each sleep laboratory application, pertinent guideline recommendations were listed and the studies cited in support were reviewed. The quality and relevance of evidence in support of recommendations were rated on three-point scales.

Results

The 37 guidelines and associated reviews that were identified cover 18 applications of sleep laboratory investigation. Of the 81 recommendations identified, 46 are supported by evidence from primary studies. Another four are supported by an absence of available evidence. For the other 31, either no evidence is provided or there is support by consensus. The cited evidence from the primary studies was judged to be highly relevant to the recommendation in 18 cases, of some relevance in 22 and of little or no relevance in six. In the following summaries for prominent applications, judgements on evidence quality and relevance are given as good, fair or limited.

Obstructive sleep apnea (OSA) diagnosis: Full-night polysomnography (PSG) is recommended for most patients (evidence, fair; relevance, good). If there is a high probability of OSA, a cardiorespiratory sleep study may be an alternative to full-night PSG, provided repeat PSG is permitted (evidence, limited; relevance, fair). Other guidelines reach different conclusions: limited sleep studies are an adequate first-line method of diagnostic assessment for OSA. The mainstay in the assessment of suspected OSA is a careful history, including standardized questionnaires (evidence, fair; relevance, good).

Obstructive sleep apnea titration: A full night of PSG with continuous positive airway pressure (CPAP) titration is recommended (evidence, limited; relevance, good). Split-night study is an alternative if four criteria are met (evidence, limited; relevance, good). Auto-CPAP devices may be used during attended titration (evidence, good; relevance, good), but not for those with congestive heart failure (absence of evidence for that patient group).

Obstructive sleep apnea follow-up: Follow-up PSG is indicated after a good response to oral appliance treatment, surgical treatment or substantial weight change in patients on CPAP (evidence, limited; relevance, good). A contrary recommendation is that follow-up PSG is not routinely indicated in patients whose symptoms continue to be resolved (supported by consensus).

Other respiratory disorders: PSG is indicated for patients with chronic obstructive pulmonary disease and for those with neuromuscular disorders (evidence, limited; relevance, fair). PSG is not indicated to diagnose chronic lung disease (evidence, fair; relevance, limited).

Obstructive sleep apnea in children: Perform PSG for diagnosis if other techniques give negative results (evidence, fair; relevance, good). PSG is not indicated for obese children unless there is chronic snoring or disturbed sleep (evidence, limited; relevance, good).

Other conditions in children: PSG is indicated in several conditions (evidence, limited; relevance, limited), but not for the routine evaluation of infants with an uncomplicated apparent life-threatening event (supported by consensus).

Repeat PSG in children: Retest high risk children for OSA after surgery if snoring persists (evidence, limited; relevance, fair). PSG is not needed for children with mild to moderate OSA, who experience a complete resolution of symptoms (supported by consensus).

Sudden infant death syndrome: PSG is not indicated as a screening tool (evidence, good; relevance, good).

Treatment for snoring: PSG or a cardiorespiratory study is indicated before surgery (evidence, limited; relevance, good).

Insomnia: PSG is indicated when sleep-related breathing disorders or periodic limb movement disorder (PLMD) are suspected; or for precipitous arousals (evidence, fair; relevance, fair). PSG is not indicated for the routine evaluation of insomnia (supported by consensus) or for the evaluation of insomnia due to psychiatric disorders (evidence, good; relevance, fair).

Depression with insomnia: PSG and the multiple sleep latency test (MSLT) are not routinely indicated for diagnosis (evidence, good; relevance, good).

Narcolepsy: PSG and MSLT are routinely indicated in the evaluation of suspected narcolepsy (evidence, fair; relevance, fair).

Restless legs syndrome (RLS) and PLMD: PSG is indicated when the diagnosis of PLMD is considered (evidence, fair; relevance, fair). There are differing conclusions about RLS; PSG is not routinely indicated for diagnosis (evidence, fair; relevance, fair); and PSG is recommended for RLS in six situations (supported by consensus).

Parasomnias: PSG is indicated for seizure cases when the EEG is inconclusive (evidence, limited; relevance, limited). PSG is not indicated for uncomplicated cases (evidence, good; relevance, good).

Conclusion

The clinical guidelines that were reviewed contain detailed information for health professionals. Most recommendations are supported by studies on sleep laboratory applications. The level of evidence for many applications is of limited quality and some cited studies are not relevant to the recommendations made. Many recommendations reflect consensus positions and no evidence is cited in support. There is a need for more good quality studies of many sleep laboratory applications.

GLOSSARY

Attended study: A sleep study during which a technician is present to monitor recordings and assist the patient, if necessary.

Auto-titrating nasal continuous positive airway pressure (APAP): A version of CPAP in which the positive pressure applied to the patient's upper airways changes continuously during sleep.

Cardiorespiratory sleep study: A study with a minimum of the following four channels: respiratory effort, airflow, arterial oxygen saturation, electrocardiography or heart rate.

Central apnea: Apnea where inspiratory muscle activity fails after an exhalation.

Continuous positive airway pressure (CPAP): A treatment for obstructive sleep apnea syndrome in which continuous air pressure is applied to the upper airways while the patient is asleep and breathing spontaneously.

Efficacy: Performance of a technology under ideal conditions or conditions of best practice.

Effectiveness: Performance of a technology under "routine" conditions, for example, when it has become widely distributed in a health care system.

Laser-assisted uvulopalatoplasty: A technique used as a treatment for snoring.

Limited study: A sleep study in which only some of the parameters of sleep-related disorders are monitored. A limited PSG study evaluates only heart rate and rhythm, chest and abdominal movement, airflow through the mouth and nose and blood oxygen levels (see Cardiorespiratory sleep study). Limited sleep studies, which only record breathing efforts and blood oxygen levels, have been performed using portable devices (see Portable monitoring).

Mixed apnea: Apnea in which both central and obstructive apnea occur during the same episode.

Obstructive apnea: Apnea where inspiratory muscle activity is present without airflow.

Portable monitoring: Use of a portable device for monitoring sleep activity outside the sleep laboratory.

Sleep laboratory: A facility to investigate sleep disorders during sleep.

Split-night study: A sleep study in which diagnosis of OSA is undertaken during the first part of the night and is followed by titration to obtain effective CPAP settings for treatment.

Titration: Adjustment of CPAP settings during sleep to provide optimum values.

Wrist actigraphy: Measurement of wrist movement activity.

ABBREVIATIONS

AARC	American Association of Respiratory Cardiologists
AASM	American Academy of Sleep Medicine
AHI	apnea-hypopnea index
AI	apnea index
ALTE	apparent life threatening event
APAP	automatic positive airway pressure
APT	Association of Polysomnography Technologists
ASDA	American Sleep Disorders Association
COPD	chronic obstructive pulmonary disease
CPAP	continuous positive airway pressure
CPSO	College of Physicians and Surgeons of Ontario
CSS	cross-sectional study
DoA	disorders of arousal
ECG	electrocardiography
EDS	excessive daytime sleepiness
EEG	electroencephalography
EMG	electromyography
EOG	electroocculography
ESS	Epworth Sleepiness Scale
FEV1	forced expiratory volume in one second
LAUP	laser-assisted uvulopalatoplasty
MSLT	multiple sleep latency test
MWT	maintenance of wakefulness test
NIH	National Institutes of Health
NOD	nocturnal oxyhemoglobin desaturation
NREM	non-rapid eye movement
NRCT	non-randomized controlled trial
NSD	no statistically significant difference
NTRR	night-time respiratory recording
OSA	obstructive sleep apnea syndrome
OSAHS	obstructive sleep apnea/hypopnea syndrome
$PaCO_2$	arterial carbon dioxide pressure
PaO_2	arterial oxygen pressure
PET_{CO2}	end-tidal carbon dioxide tension
PLM	periodic limb movement
PLMD	periodic limb movement disorder
PM	portable monitor
PSG	polysomnography
RAI	respiratory arousal index
RBD	rapid eye movement behaviour disorder
RCT	randomized controlled trial
RCT-C	randomized crossover trial
RDI	respiratory disturbance index
REM	rapid eye movement

REML	REM latency
RLS	restless legs syndrome
SaO$_2$	arterial oxygen saturation
SAS	sleep apnea-hypopnea syndrome (see OSA)
SIDS	sudden infant death syndrome
SOREMP	sleep-onset rapid eye movement period
SpO$_2$	oxyhemoglobin saturation
SPT	sleep period time
SS	statistically significant
UARS	upper airway resistance syndrome
VC	vital capacity
VPSG	video polysomnography

TABLE OF CONTENTS

1   INTRODUCTION

1.1 Background

Sleep disorders are common complaints, particularly among middle-aged and elderly people. More than 80 sleep disorders are listed in the International Classification of Sleep Disorders. These include sleep-related breathing disorders, neuromuscular disorders with sleep-related symptoms, chronic lung disease, narcolepsy, parasomnias (arousal disorders), sleep-related epilepsy, restless legs syndrome, periodic limb movement disorder, depression with insomnia and circadian rhythm sleep disorders.[1] Those who suffer from significant sleep disorders are subject to medical risks and reduced quality of life resulting from daytime sleepiness and other symptoms.

An analysis of data from the Canadian General Social Survey indicated that 24% of the Canadian population older than 15 report suffering from insomnia.[2] A survey in the US of middle-aged adults found sleep-disordered breathing affects 9% of women and 24% of men when defined by abnormal sleep study criteria alone. Some 2% of women and 4% of men met the diagnostic criteria for the most common sleep disorder, obstructive sleep apnea syndrome (OSA), with an abnormal sleep study and daytime hypersomnolence.[3] Rates in Canada are likely similar.

OSA is a syndrome characterized by recurrent episodes of partial or complete upper airway obstruction during sleep. This manifests as a reduction in (hypopnea) or complete cessation (apnea) of airflow despite ongoing inspiratory efforts. Overnight monitoring demonstrates five or more obstructed breathing events per hour during sleep.[4] OSA is associated with excessive daytime sleepiness, cognitive and personality problems and hypertension.[5]

Accurate diagnosis and monitoring of sleep disorders are important in their management. For persons with severe symptoms, the approach to diagnosis and management of the sleep disorder may involve observation in a sleep laboratory. Sleep laboratory investigations may also be needed during the day to monitor wakefulness.

1.2 Technology Overview

A key component of sleep laboratories is the availability of physicians, technicians and nurses who are expert in the management of sleep disorders.

In many investigations, patients are required to stay overnight in the sleep laboratory where they are monitored using polysomnography (PSG). PSG simultaneously monitors and records multiple physiological parameters during sleep. A full range of PSG measurements includes electroencephalography (EEG), electroocculography (EOG), submental electromyography (EMG), electrocardiography (ECG), respiratory movement or respiratory effort, nasal or oral airflow, pulse oximetry and limb movement EMG. PSG must be performed in a hospital or in a sleep centre that is equipped with specialized machines and has a qualified technician in constant attendance.

An area of debate is the extent to which the full range of PSG measurements are required for the diagnosis and monitoring of specific sleep disorders. The use of fewer channels has been proposed and implemented by some sleep centres.

Portable systems that are intended to assess sleep apnea have been developed for use in settings outside the sleep laboratory. Type II devices measure both respiratory and sleep variables, Type III devices allow for the assessment of cardiorespiratory variables, but do not include EEG, EOG or chin EMG and Type IV devices monitor one or two cardiorespiratory parameters.

A commonly used treatment for moderate to severe OSA is nasal continuous positive airway pressure (CPAP), which involves the application of continuous pressure to the upper airways through a nose or face mask while the patient is asleep and breathing spontaneously. In addition to diagnosing OSA, sleep laboratories undertake adjustment (titration) of CPAP settings to provide optimum values for the patient. The adjustment of air pressure is made by a technician in response to PSG measurements. The technician also intervenes if the mask leaks or persistent hypoxemia occurs after airway patency is restored.

Auto-titrating CPAP devices allow the positive pressure level applied to the patient through the mask to continually change during sleep. APAP has the potential to permit unattended titration in sleep laboratories or in the patient's home.

Patients attending sleep laboratories often undergo one or more overnight PSG recordings, depending on the type of sleep disorder and the purpose of the investigation. Shorter investigations are also used, such as split-night studies, in which diagnosis and CPAP titration are performed on the same night.

The investigation of excessive daytime sleepiness (EDS) is typically undertaken using the multiple sleep latency test (MSLT). This measures the speed of falling asleep under conditions that favour sleep, in a series of 20-minute trials undertaken during the day. The maintenance of wakefulness test (MWT), which measures the ability to stay awake when desired, has also been used. This test has been less validated than the MSLT.

Sleep laboratory investigations are expensive because of the length of the examinations, the need for a qualified technician to be in attendance and the cost of equipment. For example, the cost per study for the diagnosis of OSA and CPAP titration in Alberta is approximately $1,500.[6]

1.2.1 Sleep laboratory services in Canada

In Canada, the number of sleep laboratories and sleep study rates vary among jurisdictions. There are also associated differences in waiting times for consultations and PSG examinations. According to a recent review by Flemons *et al.,*[7] annual numbers of sleep studies for OSA per 100,000 population varied from 28 in Newfoundland to 776 in Ontario, which accounted for 80.5% of all studies. The average for Canada was 370 studies per 100,000. According to Flemons *et al.,* comparative rates for other countries are UK 42.5, Belgium 177, Australia 282 and the US 427.

There has been increasing demand for sleep laboratory services. In Canada, the demand for services is suggested by the waiting times for sleep specialist consultations, which according to Flemons *et al.,* averages four to six months in eastern and western Canada.[7] In one US health maintenance organization (Group Health Cooperative of Puget Sound), the PSG rate increased from 91.7 to 205.3 per 100,000 persons enrolled between 1991 and 1994 − a 124% increase.[8] In Australia, the number of paid sleep study items in the Medicare Benefits Schedule increased from 3,025 to 28,894 between 1990 to 1991 and 1996 to 1997.[9]

Guidelines on the use of PSG and other approaches for investigating the most common sleep disorders have been published by professional organizations, mostly from the US. The College of Physicians and Surgeons of Ontario[16], as well as the Canadian Sleep Society and the Canadian Thoracic Society, developed Canadian guidelines that were published in 1996.[10]

2   THE ISSUE

Health care providers need assurance that the examinations and services in sleep laboratories are clinically appropriate, given that demand is increasing, rates of service vary among jurisdictions and sleep laboratories are expensive and specialized. There is a need to consider the recommendations that are included in the guidelines prepared by professional organizations and the evidence on which the recommendations are based.

3   OBJECTIVES

The objective of this report is to review the recommendations for the investigation of individuals in sleep laboratories made in guidelines prepared by professional bodies. The report is intended to help decision makers who are involved in the provision of sleep laboratory services and those involved in the management of patients with sleep disorders.

This objective is accomplished by addressing the following questions:
- What are the recommendations made in guidelines prepared by professional bodies related to the investigation of individuals in sleep laboratories?
- What is the evidence on which the recommendations are based?
- What is the quality of the evidence?
- What is the relevance of the evidence to the recommendations?

4   METHODS

A protocol for the review of guidelines was written a priori and was followed throughout the project.

4.1   Literature Search Strategy

Published guidelines and associated reviews on the use of sleep laboratory investigations for sleep disorders were obtained in October 2002 by cross-searching MEDLINE®, EMBASE®, BIOSIS Previews®, PASCAL and PsychINFO® databases on the DIALOG® search system from 1992 onwards. The search was updated in January 2004. Subject headings and keywords were used to search sleep disorders. A guideline filter was used to limit the retrieval to the guidelines identified. A language limit was not used. Parallel searches were performed and updated on PubMed, CINAHL and the Cochrane Library databases. PubMed, CINAHL and Cochrane searches were last updated in June 2004. Search details are given in Appendix 1.

An extensive search of appropriate web sites was also performed to retrieve clinical practice and evidence-based guidelines (Appendix 2).

Grey literature publications were retrieved by searching the web sites of health technology assessment and related agencies. Databases, such as those of the University of York NHS Centre for Reviews and Dissemination and LILACS, the Latin American and Caribbean Center on Health Sciences Information, were searched for additional information.

In addition, reference lists of retrieved guidelines were hand searched to examine the evidence on which the guidelines were based. Subject experts were consulted.

4.2   Selection Criteria and Method

4.2.1   Selection criteria

Guidelines and associated reviews prepared in support of their provisions were selected when they included advice and recommendations on the use of examinations in sleep laboratories for persons with sleep disorders. Guidelines that considered only the operation of equipment were excluded. Priority was given to the most recent version of updated guidelines.

Reference to previous guidelines was made, where appropriate, with regard to recommendations and previously cited literature.

4.2.2   Selection method

Selection of the guidelines produced by professional bodies and associated reviews, identified by the literature search, was undertaken independently by three reviewers (DH, LM and KT).

4.3   Data Extraction Strategy

From preliminary inspection, the applications of sleep laboratories were identified and grouped into categories. For each category covered in a guideline, recommendations related to the selection of patients for examination in a sleep laboratory were identified. Recommendations related to other aspects of managing sleep disorders were not considered.

For each recommendation, two reviewers (DH, KT) independently identified studies and other publications cited in the guideline as providing support.

If the recommendation referenced a review or a general descriptive article, the publication was retrieved and used as background information, but was not reviewed in detail. No attempt was made to retrieve and assess earlier information that was cited in these secondary sources.

Where the citation was linked to a primary study, the paper was retrieved and reviewed independently by DH and KT with the aid of a structured form (Appendix 3). Consideration was given to the type and design of study, study population, quality of evidence and relevance of evidence and conclusions to the recommendation made in the guideline.

Recommendations in one of the identified guidelines[11] were supported by a comprehensive systematic review.[12] For that guideline, summaries of evidence given in the review were cited, rather than re-appraising the studies it had considered.

For each recommendation in the other guidelines, an indication of the overall quality of the evidence provided by the studies cited in support was obtained by assigning ratings on the following basis:
A=Most or all of the cited evidence came from well conducted, prospective controlled studies.
B=The cited evidence was based on both controlled studies and case series; there might be minor shortcomings in the way that some studies were conducted.
C=The cited evidence was based only on case series or on case series plus controlled studies that had substantial limitations.
O=Either no evidence was provided or the recommendation was supported only by a consensus statement.
N=The recommendation was supported by an absence of evidence that was identified by the guideline.

For those recommendations where studies were cited in support, judgements were made on the relevance of the evidence, as follows:
A=The cited evidence was highly relevant to the recommendation.
B=The cited evidence had some relevance to the recommendation.
C=The cited evidence had little or no relevance to the recommendation.

These ratings of quality and relevance of the cited evidence were made independently by two of the authors (DH and KT) and any differences resolved by consensus.

4.4 Data Analysis Methods

In view of the range of conditions investigated by sleep laboratories and the diversity of supporting data, a series of qualitative reviews was prepared.

Retrieved information was synthesized in two ways. First, summaries were prepared for each application of the recommendations for and against the use of sleep laboratories, with details on the nature of supporting evidence and ratings of its quality and relevance.

In the second approach, a more extensive commentary was prepared for each sleep laboratory application, listing all pertinent recommendations from the guidelines and reviewing the studies that had been cited in support. For each application, commentaries were prepared on the nature of the supporting evidence and of its relevance to the guideline recommendations.

5   RESULTS

5.1 Guidelines Selected for Review

From the literature search, 37 publications met the selection criteria and were reviewed. These publications, comprising guidelines and accompanying reviews are listed in Appendix 4. Details of the guidelines and related documents excluded from the review are in Appendix 5.

From inspection of the guidelines, the 18 categories of potential sleep laboratory applications and their corresponding guidelines and summaries were identified (Table 1).

Table 1: Sleep laboratory applications

Application	Guideline References	Summary of Findings
Obstructive sleep apnea diagnosis	[10,13-22]	5.3.1, page 10
Obstructive sleep apnea titration	[16,18,23,24]	5.3.2, page 13
Obstructive sleep apnea follow-up	[23-25]	5.3.3, page 14
Respiratory disturbances complicated by cardiovascular factors	[26]	5.3.4, page 18
Other respiratory disorders	[19,23]	5.3.5, page 18
Obstructive sleep apnea in children	[27-29]	5.3.6, page 21
Other conditions in children that produce sleep disturbances	[28]	5.3.7, page 21
Repeat PSG in children	[19,27-29]	5.3.8, page 23
Idiopathic congenital hypoventilation syndrome	[30]	5.3.9, page 24
Sudden infant death syndrome (SIDS)	[19,31,32]	5.3.10, page 25
Treatment for snoring	[17,23,33]	5.3.11, page 25
Insomnia	[19,34]	5.3.12, page 27
Depression with insomnia	[23]	5.3.13, page 27
Narcolepsy	[19,23,35]	5.3.14, page 27
Restless legs syndrome or periodic limb movement disorder	[19,23,36-38]	5.3.15, page 29
Parasomnias and sleep-related epilepsy	[19,23,39,40]	5.3.16, page 32
Circadian rhythm disorders	[19,23,39]	5.3.17, page 32
Chronic fatigue syndrome	[41]	5.3.18, page 34

5.2 Summary of Recommendations and Supporting Evidence

In all, 81 recommendations in the reviewed guidelines were identified. Of the recommendations, 46 were supported by evidence from primary studies. Of these, six recommendations corresponded to category A, with most or all of the cited evidence from well conducted, prospective controlled studies. For 15 recommendations, cited evidence was based on both controlled studies and case series; 25 were supported only by results from case series or from case series plus controlled studies that had substantial limitations.

Four of the remaining recommendations were supported by an absence of available evidence identified by the guideline. For the other 31, either no evidence was provided or the guideline indicated that there was support by consensus.

The cited evidence from primary studies was judged to be highly relevant to the recommendation in 18 cases, of some relevance in 22 and of little or no relevance in six. A summary of indications and cited evidence for and against the use of sleep laboratory investigations for the applications considered is presented in Table 2, which does not refer to other guidelines that have recommendations consistent with those cited or to additional details of associated tests and descriptive comments.

5.3 Recommendations and Evidence for Specific Sleep Laboratory Applications

In this section, summaries of recommendations and supporting evidence cited in the reviewed guidelines are given for each of the sleep laboratory applications listed in Table 1. Each summary gives a description of the sleep disorder being considered, followed by a table listing the recommendations for use of sleep laboratory investigations and for any contra-indications or limitations for sleep laboratory use. Ratings are for the quality and relevance of the evidence cited in support of each recommendation (Section 4.3). For some applications, individual guidelines had different recommendations or emphases on indications for and against the use of sleep laboratory investigations. These differing conclusions are identified in the tables by an asterisk. For most applications, more complete reviews of the evidence cited in support of recommendations made in the guidelines, are provided in the appendices.

Table 2: Recommendations and evidence for sleep laboratory applications

Application	Indications for Sleep Laboratory Use	Limitations to Sleep Laboratory Use
Obstructive sleep apnea diagnosis	For most patients, full-night PSG recommended for diagnosis of sleep-related breathing disorders[23]* (evidence quality=B, relevance=A) Split-night studies and full-night studies are not equivalent; full-night PSG followed by full-night of CPAP titration is recommended[16] (evidence quality=B, relevance=A) If there is high probability of OSA, cardiorespiratory sleep study may be alternative to full-night PSG, provided repeat PSG is permitted[23] (evidence quality=C, relevance=B)	Limited sleep studies are adequate first-line method of diagnostic assessment for OSA[15]* (evidence quality=B, relevance=A) Mainstay in assessment of suspected OSA is a careful history, including standardized questionnaires[17]*(evidence quality=B, relevance=A)
Obstructive sleep apnea titration	Certain APAP devices may be used during attended titration[24] (evidence quality=A, relevance=A). Full night of PSG with CPAP titration is recommended[1] (evidence=C, relevance=A). Split-night study is an alternative if four criteria are met[1] (evidence=C, relevance=A)	Patients with congestive heart failure are not candidates for APAP; unattended APAP is not established[24] (evidence quality=N)
Obstructive sleep apnea follow-up	Follow-up PSG indicated after good response to oral appliance treatment,* surgical treatment, substantial weight change in patients on CPAP[27] (evidence quality=C, relevance=A)	Follow-up PSG not routinely indicated in patients whose symptoms continue to be resolved[23]* (evidence quality=O)
Respiratory disturbances complicated by cardiovascular factors	PSG may be indicated in patients with:[26] • COPD with awake PaO_2 >55 torr (evidence quality=C, relevance=B) • Restrictive ventilatory impairment secondary to chest wall and neuromuscular disturbances • Disturbances in respiratory control with awake $PaCO_2$ >45 torr cyclic arrythmias that appear to increase in frequency during sleep (evidence quality=O)	Risk-benefit ratios should be assessed if medically unstable inpatients are to be transferred from clinical setting to sleep laboratory for overnight PSG (evidence quality=O)
Other respiratory disorders	PSG indicated for patients with neuromuscular disorders to evaluate symptoms not adequately diagnosed and for patients with COPD[23] (evidence quality=C, relevance=B)	PSG not indicated to diagnose chronic lung disease or evaluate nocturnal hypoxemia[23] (evidence quality=B, relevance=C)

Application	Indications for Sleep Laboratory Use	Limitations to Sleep Laboratory Use
Obstructive sleep apnea in children	PSG is gold standard for OSA diagnosis; perform if other techniques give negative results[27] (evidence quality =B, relevance=A)	PSG not indicated for obese child unless there is chronic snoring or disturbed sleep[28] (evidence quality=C, relevance =A)
Other conditions in children that produce sleep disturbances	PSG indicated in neuromuscular disease, some patients with bronchopulmonary dysplasia, cystic fibrosis, asthma, ALTE[28] (evidence quality=C, relevance=C)	PSG not recommended for routine evaluation of infants with uncomplicated ALTE[28] (evidence quality=O)
Repeat PSG in children	Retest high risk children for OSA after surgery if snoring persists[27] (evidence quality=C, relevance=A)	Not needed for children with mild to moderate OSA with complete resolution of symptoms[28] (evidence quality=O)
Idiopathic congenital hypoventil-ation syndrome	Each infant should have detailed recording in pediatric respiratory physiology laboratory to evaluate spontaneous breathing during sleep and wakefulness[30] (evidence quality=O)	
Sudden infant death syndrome	PSG indicated in investigation of certain physical symptoms, e.g., ALTE, cyanotic attacks[32] (evidence quality=O)	PSG not indicated as screening tool for SIDS[32] (evidence quality=A, relevance=A)
Treatment for snoring	Pre-operative PSG or cardiorespiratory study[23] (evidence quality=C, relevance=A)	
Insomnia	PSG indicated when sleep-related breathing disorders or PLMD suspected; initial diagnosis uncertain; precipitous arousals[34] (evidence quality=B, relevance=B)	PSG not indicated for routine evaluation of insomnia (evidence quality=O)

Evaluation of insomnia due to psychiatric disorders[34] (evidence quality=A, relevance=B) |
| Depression with insomnia | PSG may be useful in identifying early response to antidepressants[39] (evidence quality=A, relevance=C) | PSG, MSLT not routinely indicated for diagnosis[23] (evidence quality=A, relevance=A) |
| Narcolepsy | PSG and MSLT routinely indicated in evaluation of suspected narcolepsy[35] (evidence quality=B, relevance=B) | Debatable whether PSG is required to make the diagnosis in every case[19] (evidence quality=O) |
| Restless legs syndrome or periodic limb movement disorder | PSG is recommended for RLS in six situations[36]* (evidence quality=O)

Diagnosis of RLS or PLMD is suggested by clinical examination, with full-PSG[19]* (evidence quality=O)

PSG is recommended when diagnosis of PLMD is considered[23,37] (evidence quality=B, relevance=B) | PSG not routinely indicated for diagnosis of RLS[23]* (evidence quality=B, relevance=B) |
| Parasomnias | Seizure cases when EEG inconclusive; atypical cases[23] (evidence quality=C, relevance=C) | PSG not indicated for uncomplicated cases[23] (evidence quality=A, relevance=A) |

Application	Indications for Sleep Laboratory Use	Limitations to Sleep Laboratory Use
Circadian rhythm disorders	PSG is useful when diagnosis is persistent circadian rhythm disorders, such as delayed phase syndrome[19] (evidence quality=O)	PSG not routinely indicated for diagnosis of circadian rhythm sleep disorders[23] (evidence quality=C, relevance=B)
Chronic fatigue syndrome	If specific alternative diagnoses (e.g., sleep apnea) suggested by clinical history or exam, further investigations may be warranted[41] (evidence quality=O)	

ALTE=apparent life threatening event; APAP=automatic positive airway pressure; COPD=chronic obstructive pulmonary disease; CPAP=continuous positive airway pressure; OSA=obstructive sleep apnea; EEG=electroencephalography; MSLT=multiple sleep latency test; PLMD=periodic limb movement disorder; PSG=polysomnography; RLS=restless legs syndrome; *recommendations where there appear to be different conclusions or emphases.

5.3.1 Obstructive sleep apnea diagnosis

Obstructive sleep apnea syndrome (OSA) is a sleep and breathing disorder characterized by recurrent episodes of partial or complete upper airway obstruction during sleep. This manifests as a reduction in (hypopnea) or complete cessation (apnea) of airflow, despite ongoing inspiratory efforts. Overnight monitoring demonstrates five or more obstructed breathing events per hour during sleep. OSA may be associated with excessive daytime sleepiness, cognitive and personality problems and high blood pressure. The diagnosis of OSA is commonly undertaken in a sleep laboratory using PSG.

This application is the most widely covered by the publications reviewed. Thirteen guidelines give recommendations on the diagnosis of OSA (Appendix 6). Detailed discussion is included in the 1997 ASDA guideline[23,39] and in the publication of the Scottish Intercollegiate Guidelines Network.[15]

Most recommendations in the guidelines on the diagnosis of OSA focus on the extent to which the use of full-night PSG is required to produce a confident diagnosis. Several less expensive approaches to OSA diagnosis have been proposed. These include the use of self-administered sleep questionnaires, overnight pulse oximetry and portable sleep apnea monitoring devices.

Three guidelines use the 1997 ASDA publication as their main source of evidence and include some additional material. The Connecticut Thoracic Society report[16] refers to two additional case series and the Swiss Respiratory Society[17] cites a non-randomized controlled trial (NRCT) on the use of sleep questionnaires. The Argentine Association of Respiratory Medicine[18] does not cite specific evidence in support of recommendations on PSG nor on the use of daytime studies, but refers to several publications related to lower level studies that may need to be considered in situations where more detailed investigations, such as those offered by a sleep laboratory, are unavailable.

Six guidelines from Canada,[10,19] Spain,[20] Finland[21] and the US[22,26] did not cite evidence specifically in support of their recommendations, other than referral to other guidelines or statements and should be regarded as secondary authorities. Another publication[42] includes a recommendation on sleep medicine consultation and PSG for snoring patients who have poor periodontal health or who are edentulous, but it does not cite appropriate specific evidence.

The cited evidence in support of full-night PSG is limited. Four cohort studies and seven case series are cited to provide evidence that a single night of PSG is usually sufficient to diagnose OSA. Five case series indicated that full-night PSG is desirable for OSA diagnosis as the number of apneas varies throughout the night. A further study suggested that PSG during the first two hours of sleep is a reliable basis for the diagnosis.

The two additional small studies cited by the Connecticut Thoracic Society report[16] provide further evidence against the use of split-night studies for the diagnosis of OSA.

The guideline from the Scottish Intercollegiate Guidelines Network[15] takes a different perspective from that of the ASDA. It recommends that limited sleep studies to assess respiratory events are an adequate first-line method of diagnostic assessment for OSA and that full PSG with EEG-based sleep staging is unnecessary to diagnose sleep apnea in most patients. PSG should be available in regional sleep centres for patients who have typical symptoms of excessive day time sleepiness, but no objective evidence of obstructive sleep apnea on limited testing.

The highest quality evidence cited is a NRCT, which included follow-up data for patients who had been prescribed CPAP after a diagnosis of OSA. Five other controlled trials and four case series are also considered. The studies indicate the potential for limited sleep studies use, leading to judgements on the trade-off between poorer diagnostic performance than full PSG and appropriate use of limited resources. The statement that full PSG is not necessary to diagnose sleep apnea in most patients is supported by limited evidence.

The findings of the controlled study, cited by the Swiss Respiratory Society guideline,[17] were relevant to the recommendation that standardized questionnaires such as the Epworth Sleepiness Scale (ESS) may be used to assess suspected OSA, particularly to identify severe OSA from primary snoring and central sleep apnea.

Table 3: Recommendations and supporting evidence for
obstructive sleep apnea diagnosis

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
For most patients, full-night PSG is recommended for diagnosis of sleep-related breathing disorders.[16,23]∗ Split-night studies and full-night studies are not equivalent. Full-night PSG followed by full night of CPAP titration is accepted gold standard and is recommended.[16]	Four cohort studies and seven case series indicating a single night of PSG is usually sufficient to diagnose OSA (evidence quality=B, relevance=A) One NRCT and five case series indicate that full-night PSG is desirable for OSA diagnosis as several apneas vary throughout night (evidence quality=B, relevance=A)	Limited sleep studies to assess respiratory events are adequate first-line method of diagnostic assessment for OSA.[15]∗ Mainstay in assessment of patients with suspected OSA is careful history, including standardized questionnaires.[17]∗ No consensus on use of split-night studies.[18]	6 NRCT and 5 case series indicating potential for limited studies to identify OSA, but not to rule it out (evidence quality=B, relevance=A) 1 NRCT, reference to other guidelines (evidence quality=B, relevance=A) No specific literature cited (evidence quality=O)
For patients in high-pretest-probability stratification group, cardiorespiratory sleep study may be acceptable alternative to full-night PSG, provided repeat testing with full-night PSG is permitted for symptomatic patients who have a negative cardio-respiratory sleep study.[23]	10 case series, 6 with use of multifactorial analysis; approaches using questionnaires to screen patients for OSA have potential to rule out further investigations in sleep laboratory, but significant proportions of persons with OSA would be incorrectly excluded from referral for PSG (evidence quality=C, relevance=B)	Simplified (level III) studies reserved for situations where there are long waiting lists, access to PSG is impossible or for follow-up or re-evaluation[18]	7 series with comparison of methods (evidence quality=C, relevance=B)
Some Type 3 portable monitors could be used in laboratory to decrease or increase probability that patient has AHI >15. Such use would require limitations.[43]	Studies considered in systematic review (evidence quality=B, relevance=A)	Type 2 portable monitors not recommended for clinical use to evaluate patients with OSA.[43] Type 3 portable monitors are not recommended for unattended use or to rule in and rule out OSA.[43]	Studies considered in systematic review (evidence quality=N) Studies considered in systematic review (evidence quality=B, relevance=A)

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	Evidence	Recommendations	Evidence
		Type 4 portable monitors not recommended for attended or unattended use to increase probability that patient has AHI >15.[43] Unattended portable recording for assessment of OSA is acceptable only in following situations: • for patients with severe clinical symptoms indicative of OSA, when initiation of treatment is urgent and PSG is unavailable • for patients unable to be studied in sleep laboratory • for follow-up studies after diagnosis with PSG and therapy initiation[13]	Studies considered in systematic review (evidence quality=B, relevance=A) 6 small case series, subjective assessment of some outcomes (evidence quality=C, relevance=B)

AHI=apnea-hypopnea index; OSA=obstructive sleep apnea; PSG=polysomnography.

5.3.2 Obstructive sleep apnea titration

Continuous positive airways pressure (CPAP) is an effective means of treating obstructive sleep apnea (OSA). An essential component of the use of CPAP is the establishment of pressure, which is applied to the patient's airways during treatment, to minimize episodes of apnea. This process is known as titration.

Guideline recommendations include several points on the extent to which sleep laboratory investigations are required for the titration process, rather than the use of home-based measurements, including those that use automatic airway pressure (APAP) devices. The guidelines also consider the use of split-night studies where initial diagnostic PSG is followed by CPAP titration during PSG on the same night, rather than diagnosis and titration being undertaken on separate nights (Appendix 7).

The most detailed information on this application is provided by the 1997 ASDA guideline[23,39] and the 2002 report by the American Academy of Sleep Medicine (AASM) on APAP devices.[24]

The ASDA document recommends a full night of PSG with CPAP titration for patients with a documented diagnosis of a sleep-related breathing disorder for which CPAP is warranted. A cardiorespiratory sleep study without an EEG recording is not recommended for titration. A split-night study is an alternative to one full night of diagnostic PSG followed by a second night of titration, if four criteria are met. Six case series and material considered under OSA diagnosis are cited in support.

The 2002 AASM report supports the use of certain APAP devices during attended titration. Cited evidence in support includes four randomized studies and six non-randomized comparisons. APAP is not recommended for split-night titration, for unattended titration or for patients with congestive heart failure or chronic obstructive pulmonary disease (COPD).

An earlier guideline from the American Thoracic Society [44] recommends that titration should include recordings of sleep, respiration and oxygenation, with various body positions, NREM and REM, to determine optimum pressure. Four small case series are cited in support. Two other US guidelines[16,22] also make recommendations, citing the ASDA and AASM guidelines as support.

The findings from small non-controlled studies suggest that certain monitors in sleep recordings, including upper airway pressure, sleep staging and body positions (supine versus lateral), are essential to obtain optimal CPAP titration. Two small case series support the need for the inclusion of an EEG recording in a cardiorespiratory sleep study used for titration.

Four papers on split-night studies, all moderate to large case series, support the recommendation that effective CPAP titration can be obtained from partial PSG if certain criteria are met. There were definitional or procedural limitations for two of these studies.

5.3.3 Obstructive sleep apnea follow-up

Use of sleep laboratory investigations may be indicated to follow-up patients who have previously been examined and prescribed CPAP for the management of their OSA symptoms.

The most detailed information on this application is provided by the ASDA 1997 and 1995 publications.[23,25,39] Reference to follow-up of OSA in a 2002 AASM publication[24] follows from these earlier guidelines (Appendix 8).

Three other guidelines make reference to the follow-up of OSA. The Swiss Respiratory Society[17] refers to the 1997 ASDA guideline[23] and cites no other evidence. Those from the Group Sommeil de la SPLF[45] and the American Thoracic Society[44] make statements that are not supported by cited evidence.

Table 4: Recommendations and supporting evidence for
obstructive sleep apnea titration

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
Certain APAP devices may be used during attended titration to identify, by PSG, single pressure for use with standard CPAP for treatment of OSA[24]	1 RCT, 3 RCT-crossover and 6 non-randomized comparisons, indicating similar results from titration with APAP and CPAP (evidence quality=A, relevance=A)	Patients with congestive heart failure, COPD or nocturnal arterial oxyhemoglobin desaturation due to conditions other than OSA are not candidates for APAP titration. APAP devices are not recommended for split-night titration.[24] Use of unattended APAP to determine CPAP pressures or for self-adjusting APAP treatment in CPAP-naïve patients is not established.[24]	Persons with such conditions have so far been excluded from APAP trials (evidence quality=N) Absence of studies to validate such applications (evidence quality=N)
A full night of PSG with CPAP titration is recommended for patients with documented diagnosis of sleep-related breathing disorder for which CPAP is warranted.[23] PSG with CPAP titration is appropriate for patients with AI ≥20 per hour or AHI ≥30 per hour; AHI ≥10 per hour in patients with EDS; RAI ≥10 per hour in patients with EDS.[23]	6 case series and material presented previously on OSA diagnosis (evidence quality=C, relevance=A)		
A cardiorespiratory sleep study without EEG is not recommended.[23]	2 case series indicating association of EEG changes with UARS (evidence quality=C, relevance=A)		

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
Split-night study is alternative to 1 full night of diagnostic PSG followed by second night of titration if 4 criteria are met:[23] a) AHI≥40 documented during minimum 2 hours of diagnostic PSG or AHI of 20 to 40 based on clinical judgment b) CPAP titration performed for >3 hours c) PSG documents that CPAP eliminates or nearly eliminates respiratory events during REM and NREM sleep, including REM sleep with patient in supine position d) Second full-night PSG for CPAP titration is performed if diagnosis of sleep-related breathing disorder is confirmed, but criteria b and c are not met	3 case series comparing treatment pressures obtained using partial and full-night PSG; small series indicating respiratory events can worsen as night progresses (evidence quality=C, relevance=A)	Split-night studies and full-night studies are not equivalent. Full-night diagnostic studies provide more complete assessment of sleep-disordered breathing and are more likely to identify possibility and relevance of second diagnosis.[16] No consensus on use of split-night studies.[18]	4 series that showed clinical features of sleep apnea varied during night-time sleep (evidence quality=C, relevance=A) No specific citations; studies showing variation in apnea during night are reasons given for lack of consensus (evidence quality=O)

AHI=apnea hypopnea index; AI=apnea index; APAP=automatic positive airway pressure; COPD=chronic obstructive pulmonary disease; CPAP=continuous positive airway pressure; EDS=excessive daytime sleepiness; EEG=electroencephalogram; NREM= non-rapid eye movement; OSA=obstructive sleep apnea; PSG=polysomnography; RAI=respiratory arousal index; REM=rapid eye movement; UARS=upper airways resistance syndrome.

The 1997 ASDA guideline[23] recommends follow-up PSG or a cardiorespiratory study is routinely indicated for the assessment of treatment results in six situations. Such a study is not routinely indicated in patients whose symptoms continue to be resolved with CPAP treatment. A MSLT is also not routinely indicated for most patients with sleep-related breathing disorders. The 1995 ASDA publication[25] states that follow-up PSG is not indicated for those with primary snoring or mild OSA, unless symptoms worsen. Patients with moderate to severe OSA should undergo PSG or another objective measure of respiration with the oral appliance in place, after final adjustments for a fit.

Only limited evidence, from studies of poor quality and of partial relevance, is cited in support. Recommendations made in the 1997 ASDA guideline regarding patients who have a good clinical response seem to be contradictory. One states that a follow-up study is routinely indicated for the assessment of treatment results after a good clinical response to oral appliance treatment to ensure a therapeutic benefit.

A further recommendation is that follow-up PSG or cardiorespiratory study is not routinely indicated in patients whose symptoms continue to resolve with CPAP treatment.

Table 5: Recommendations and supporting evidence for
obstructive sleep apnea follow-up

Indications for Sleep Laboratory Use		Limitations for Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
Follow-up PSG or cardiorespiratory study is indicated for assessment of treatment results:[23] a) after good clinical response to oral appliance treatment* b) after surgical treatment of patients with moderate to severe OSA c) after surgical treatment of patients with sleep apnea or lack of response to CPAP d) after substantial weight loss has occurred in patients on CPAP for treatment of sleep-related breathing disorders e) after substantial weight gain has occurred in patients previously treated with CPAP successfully f) when clinical response is insufficient or when symptoms return despite good initial response to treatment with CPAP	Previous guidelines are cited to support recommendations a to c (evidence quality=O) Recommendations d and e: 4 case series indicating weight variation may be linked to sleep disorders in some individuals and major changes in weight may imply current treatment with CPAP no longer effective (evidence quality=C, relevance=C) No direct evidence cited to support recommendation f (evidence quality=O)	Follow-up PSG or cardiorespiratory study not routinely indicated in patients whose symptoms continue to be resolved with CPAP treatment.* MSLT not routinely indicated for most patients with sleep-related breathing disorders.	No evidence cited (evidence quality=O) Reference made to information presented elsewhere in guideline and to earlier guideline (evidence quality=O) .
Patients being treated with fixed CPAP on the basis of APAP titration or being treated with APAP must be monitored to determine treatment effectiveness and safety.[24]	Based on committee consensus and recommendations given in previous guideline[23] (evidence quality=O)		
Patients with moderate to severe OSA should undergo PSG or other objective measure of respiration with oral appliance in place after final adjustments for fit.[25]	Cited review paper which gives data on CPAP use from earlier studies (evidence quality=O)	Follow-up PSG not indicated for those with primary snoring or mild OSA unless symptoms worsen[25]	No evidence cited (evidence quality=O)

APAP=automatic positive airway pressure; CPAP=continuous positive airway pressure; OSA=obstructive sleep apnea;
PSG=polysomnography.

5.3.4 Respiratory disturbances complicated by cardiorespiratory factors

COPD is a disorder that is characterized by reduced maximal expiratory flow and slow forced emptying of the lungs; features that do not change markedly in several months. This limitation in airflow is only minimally reversible with bronchodilators. A sleep laboratory application may be considered for patients with COPD or other respiratory disturbances, who also have certain complications and for those with apparent sleep-related cardiac conditions.

This application was considered in a guideline from the AARC-APT[26] (Appendix 9). Recommendations support the possible use of PSG in patients with COPD and other respiratory conditions, subject to certain criteria. A retrospective cross-sectional study is cited in support and indicates the significance of nocturnal oxyhemoglobin desaturation[46] as a risk factor, with the implication that this condition should be identified using sleep studies. Three narrative reviews and two earlier guidelines provide background information.

5.3.5 Other respiratory disorders

OSA and upper airway resistance syndrome (UARS) are most commonly evaluated in sleep laboratories. Other respiratory disorders known to cause sleep disturbance include COPD, neuromuscular diseases, other chronic lung diseases and asthma.

This category of disorders is considered by the 1997 ASDA guideline[23,39] (Appendix 10). It states that PSG is routinely indicated for patients with neuromuscular disorders and sleep-related symptoms that are inadequately diagnosed by sleep history, assessment of sleep hygiene and review of sleep diaries. PSG is not indicated to diagnose chronic lung disease and nocturnal hypoxemia in patients with lung disease. These are usually adequately evaluated by oximetry.

Five NRCTs and 10 case series are cited selectively in support of PSG use. Most of the cited studies had small patient numbers. Most of the studies on neuromuscular diseases are relevant to the recommendations, though it is unclear from the cited evidence that PSG is routinely indicated for this heterogeneous group of disorders when other approaches such as sleep history are insufficient.

Most of the papers dealing with the investigation of persons with lung disease also described studies (eight NRCT, eight case series) with small populations.

Table 6: Recommendations and supporting evidence for
respiratory disturbances complicated by cardiorespiratory factors

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
PSG may be indicated in patients with the following:[26] • COPD with awake PaO_2 >55 torr, but illness is complicated by pulmonary hypertension, right heart failure, polycythemia or EDS • Restrictive ventilatory impairment secondary to chest wall and neuromuscular disturbances whose illness is complicated by chronic hypoventilation, polycythemia, pulmonary hypertension, disturbed sleep, morning headaches or daytime sleepiness and fatigue • Disturbances in respiratory control with awake $PaCO_2$ >45 torr or whose illness is complicated by factors listed above • Cyclic arrythmias, abnormalities of atrioventricular ectopy that seem to increase in frequency during sleep	Retrospective CSS indicating the significance of nocturnal oxyhemoglobin desaturation as a risk factor, with the implication that this condition should be identified using sleep studies (evidence quality=C, relevance=B) 3 narrative reviews, 2 earlier guidelines (evidence quality=O)	Risk-benefit ratios should be assessed if medically unstable inpatients are to be transferred from clinical setting to sleep laboratory for overnight PSG	No evidence cited (evidence quality=O)

CSS=cross-sectional study; COPD=chronic obstructive pulmonary disease; EDS=excessive daytime sleepiness; $PaCO_2$=arterial carbon dioxide pressure; PaO_2=arterial oxygen pressure; PSG=polysomnography.

Table 7: Recommendations and supporting evidence for
other respiratory disorders

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	Evidence	Recommendations	Evidence
PSG routinely indicated for patients with neuromuscular disorders and sleep-related symptoms to evaluate symptoms inadequately diagnosed by sleep history, assessment of sleep hygiene and review of sleep diaries[23]	5 NRCT and 10 case series considering diagnosis and monitoring of sleep-related variables in myasthenia gravis, post-polio syndrome, cystic fibrosis, neuromuscular disease, kyphoscoliosis, amyotrophic lateral sclerosis, Duchenne muscular dystrophy, myotonic dystrophy; several studies focused on physiological investigations (evidence quality=C, relevance=B)	PSG not indicated to diagnose chronic lung disease[23]	2 NRCT and 2 case series; 3 studies showed drop in oxygen saturation during sleep, 1 indicated OSA is uncommon in this group (evidence quality=B, relevance=B)
If [COPD] patient's symptoms suggest diagnosis of OSA or periodic limb movement disorder, indications for PSG are the same as for those disorders in patients without chronic lung disease[23]	Reference to other guidelines (evidence quality=O)	Nocturnal hypoxemia in patients with chronic obstructive, restrictive or reactive lung disease is usually adequately evaluated by oximetry and does not require PSG[23]	Six NRCT and six case series, mostly small studies, suggesting support for use of oximetry to assist in diagnosis and monitoring of COPD (evidence quality=B, relevance=C)
		Sleep disturbance in patients with asthma should be attributed to inadequately controlled asthma unless more specific indications for other sleep-related breathing disorders exist[23]	OSA uncommon in several small case series of asthmatics; transient hypoxemia usually modest (evidence quality=C, relevance=B)
Full-PSG with respiratory monitoring including pCO_2 by transcutaneous capnometry warranted in patient with unexplained persisting or early morning hypercapnia[19]	Consensus (evidence quality=O)		

NRCT=non-randomized controlled trial; COPD=chronic obstructive pulmonary disease; OSA=obstructive sleep apnea; PSG=polysomnography.

5.3.6 Obstructive sleep apnea in children

OSA in childhood is a disorder of breathing during sleep, characterized by prolonged partial upper airway obstruction or intermittent complete obstruction. Complications may include growth abnormalities, neurologic disorders and cor pulmonale. Recommendations relate to otherwise healthy children older than one year with OSA secondary to adenotonsillar hypertrophy and/or obesity.

This application is covered by guidelines from the American Academy of Pediatrics (AAP) and[27,29] the American Thoracic Society.[28] It is also mentioned in the CPSO guideline[19] (Appendix 11).

The AAP guideline supports overnight PSG as the gold standard for diagnosis of OSA in children having adenotonsillectomy, habitual snoring, behaviour disturbance, hyperactivity and excessive daytime sleepiness. Studies cited in support include two NRCTs, two cross-sectional studies and seven case series.

The American Thoracic Society document recommends PSG for adenotonsillectomy postoperative monitoring, some cases of laryngomalacia and evaluation of children with sickle cell disease and OSA. Six small case series are cited.

The studies cited by the AAP provide indications of appropriate PSG criteria in children, which differ from those for adults. It is noted that PSG for children has not been well standardized in its performance or its interpretation. Several studies show the limitations of abbreviated methods in providing an adequate diagnosis of OSA. Most studies had a limited number of participants, often heterogeneous in age. Therefore, the findings are prone to selection bias and have weak statistical significance.

Most of the cited references in the American Thoracic Society guideline covered background information on a particular disorder, but were not related to indications for PSG in diagnosis or evaluation of OSA in children.

5.3.7 Other conditions in children that produce sleep disturbances

Several conditions of sleep disturbance in children are covered in an American Thoracic Society guideline[28] (Appendix 12). The possible use of PSG in children with bronchopulmonary dysplasia, cystic fibrosis, asthma, neuromuscular disease or alveolar hypoventilation syndrome was examined.[28]

No specific evidence was cited in support of the recommendations. Background material covering physiological or clinical issues that relate to sleep disturbances was provided for each condition.

Table 8: Recommendations and supporting evidence for
obstructive sleep apnea in children

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	Evidence	Recommendations	Evidence
Overnight PSG is gold standard for diagnosis of OSA in children having adenotonsillectomy, habitual snoring, behaviour disturbance, hyperactivity, and excessive daytime sleepiness.[27,29] Other techniques, such as videotaping, nocturnal pulse oximetry and daytime nap studies may be useful to discriminate between primary snoring and OSA if PSG results are positive. Because of high false-negative rates, PSG should be performed if other diagnostic techniques give negative results.[27,29]	2 NRCT, 2 CSS and 7 case series in which PSG findings were compared to those from alternative methods in patients referred with sleep disordered breathing (evidence quality=B, relevance=A)		
PSG is recommended in:[28] • adenotonsillectomy postoperative monitoring • children with laryngomalacia whose symptoms are worse asleep than awake or who have failure to thrive or cor pulmonale • evaluation of child with sickle cell disease and OSA or frequent veno-occlusive crises during sleep	2 case series in which patients were monitored after surgery (evidence quality=C, relevance=B) Small case series, description of PSG in monitoring pre- and post-surgery (evidence quality=C, relevance=B) 3 case series (evidence quality=C, relevance=C)	PSG not indicated for obese children unless there is unexplained awake hypercapnia, chronic snoring, increased work of breathing during sleep, disturbed sleep, daytime hypersomnolence, polycythemia or cor pulmonale[28]	Case series, PSG showed 37% were mildly abnormal; no correlation between weight, age or gender and any measurement on PSG Case series in which sleep questionnaire, weight and presence of adenotonsillar tissue were predictive of OSA (up to 81%) (evidence quality=C, relevance=A)

CSS=cross-sectional study; NRCT=non-randomized controlled trials; OSA=obstructive sleep apnea; PSG=polysomnography.

Table 9: Recommendations and supporting evidence for
other conditions in children that produce sleep disturbances

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	Evidence	Recommendations	Evidence
PSG with CO_2 monitoring indicated in children with neuromuscular disease or alveolar hypoventilation syndrome with complications	No specific evidence cited[28] (evidence quality=O)		
Bronchopulmonary dysplasia: PSG may be indicated to detect upper airway obstruction; can be used with pH monitoring to document relationship between reflux and respiratory events			
Cystic fibrosis: patients receiving supplemental oxygen may require PSG to rule out OSA; consider PSG for assessing potential adverse effects of supplemental oxygen during sleep in patients with advanced lung disease who are hypercapnic when awake			
Asthma: Consider PSG with pH monitoring if concerned about presence of gastroesophageal reflux during sleep as a trigger for nocturnal symptoms; PSG may help in defining frequency and type of apnea, cardiac, blood gas and sleep alterations in certain infants with apnea or ALTE[28]		PSG not recommended for routine evaluation of infants with uncomplicated ALTE.[28]	No evidence cited (evidence quality=O)

ALTE=apparent life threatening event; CO_2=carbon dioxide; OSA=obstructive sleep apnea; PSG=polysomnography

5.3.8 Follow-up PSG in children

The use of sleep laboratory investigations may be indicated for the follow-up of children who had surgery or other interventions to manage OSA symptoms.

This application is covered by guidelines from the American Academy of Pediatrics,[27,29] the American Thoracic Society[28] and the CPSO[19] (Appendix 13).

Table 10: Recommendations and supporting evidence for
follow-up PSG in children

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
Children who have adenotonsillectomy should have clinical re-evaluation postoperatively; high risk patients should undergo objective testing (be retested for OSA if snoring persists and possibly if pre-operative AHI is high)[27]	Prospective study of 30 children who had surgery, 26 with follow-up PSG (evidence quality=C, relevance=B)		
Repeat PSG is recommended for children previously diagnosed with OSA who exhibit persistent snoring or other symptoms of sleep-disordered breathing. If weight loss is primary therapy, PSG should be repeated to determine if weight loss program has delayed severity of OSA. For a child under one year or with severe OSA, follow-up PSG should be considered. In children with neuromuscular disease, reassessment with PSG should be scheduled at least annually. PSG is indicated periodically in children with alveolar hypoventilation syndrome and should occur at least annually.[28]	Recommendations are based on consensus, no supporting evidence cited (evidence quality=O)	Children with mild to moderate OSA who have complete resolution of snoring and disturbed sleep patterns do not need a follow-up PSG[28]	No evidence is cited (evidence quality=O)

AHI=apnea hypopnea index; OSA=obstructive sleep apnea; PSG=polysomnography.

5.3.9 Idiopathic congenital hypoventilation syndrome

Idiopathic congenital hypoventilation syndrome is a rare condition characterized by generally adequate ventilation while the patient is awake, but by alveolar hypoventilation with typically normal respiratory rates and shallow breathing during sleep.

The American Thoracic Society has recommended that each infant with this condition should have a detailed recording in a pediatric respiratory physiology laboratory.[30] No literature was cited in support.

Table 11: Recommendations and supporting evidence for
idiopathic congenital hypoventilation syndrome

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	Evidence	Recommendations	Evidence
Each infant should have detailed recording in pediatric respiratory physiology laboratory to evaluate spontaneous breathing during sleep and wakefulness[30]	No evidence cited (evidence quality=O)		

5.3.10 Sudden infant death syndrome (SIDS)

SIDS is the sudden death of an infant under one year of age, which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene and review of clinical history. Association with respiratory disorders during sleep has been suggested for some SIDS cases.

Two guidelines[31,32] recommended against the use of PSG as a screening tool for SIDS, but they supported its use in the investigation of certain physical symptoms where infants appear to be at risk. The CPSO document[19] indicated that there is no test or parameter available with sufficient sensitivity to make a formal sleep study of clinical use in children with an ALTE, based on findings from a retrospective study of potential markers of patterns of breathing and heart rate variability of 10 SIDS cases and 100 age-matched controls[47] (Appendix 14).

5.3.11 Treatment for snoring

Laser-assisted uvulopalatoplasty (LAUP) is a technique used as a treatment for snoring. Sleep studies are used to identify sleep-related breathing disorders, including OSA, before surgery. LAUP is not recommended for the treatment of OSA.

Three guidelines provide advice on this application[17,23,33,39] (Appendix 15). The 2001 AASM follows the 1997 ASDA recommendations in supporting the use of PSG or cardiorespiratory study for those who are candidates for laser-assisted uvulopalatoplasty as a treatment for snoring. Recommendations are based on two relevant case series and earlier standards of practice documents. The Swiss Respiratory Society's 2001 publication makes similar recommendations, but does not cite evidence in support.

Table 12: Recommendations and supporting evidence for SIDS

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
PSG is indicated in investigation of certain physical symptoms, ALTE, cyanotic attacks, neonates with clinically evident increased oxygen requirement, unclear drop in saturation, suspected disorders of autonomic respiratory control and suspected OSA[32]	Not supported by specific references in guideline, but are consistent with recommendations made under sections of other guidelines considered in this report (evidence quality=O)	PSG not indicated as screening tool for SIDS[31,32]	7 prospective studies on large cohorts of infants who had recordings of respiratory and cardiac function; either NSD or overlapping values for apneas for SIDS cases and controls (evidence quality=A, relevance=A)
Only if there is supportive evidence (arterial blood gases, abnormal EEG, evidence of neurologic disease) is sleep study indicated to evaluate a significant sleep-related respiratory disorder[19]	Consensus (evidence quality=O)	Sleep studies do not provide additional information in children with ALTE in whom rigorous clinical evaluation has failed to reveal underlying cause or abnormality[19]	(Evidence quality=B, relevance=A)

ALTE=apparent life threatening event; OSA=obstructive sleep apnea; NSD=no significant difference; PSG=polysomnography; SIDS=sudden infant death syndrome.

Table 13: Recommendations and supporting
evidence for treatment of snoring

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
A surgical candidate for LAUP, as treatment for snoring, should undergo pre-operative clinical evaluation and PSG or cardiorespiratory study to determine if the candidate has a sleep-related breathing disorder, including sleep apnea[17,23,33]	Guideline on use of portable recording in assessment of PSG; 2 case series indicating unreliability of clinical examination in diagnosis of OSA (evidence quality=C, relevance=A)		

LAUP=laser-assisted uvulopalatoplasty; OSA=obstructive sleep apnea; PSG=polysomnography.

5.3.12 Insomnia

Difficulty in initiating or maintaining sleep is common. Potential sleep laboratory involvement is related to chronic insomnia, where symptoms have persisted for many weeks and may be associated with other sleep-related disorders.

An AASM guideline,[34] which updates a 1995 ASDA publication, considers this application. The CPSO document[19] also discusses the application, drawing mainly on the 1995 ASDA work (Appendix 16).

The use of PSG is recommended by the AASM when sleep-related breathing disorders or PLMD is suspected, when initial diagnosis is uncertain, treatment fails or precipitous arousals occur. PSG is not indicated for routine evaluation of insomnia, including that which is associated with psychiatric disorders, fibromyalgia or chronic fatigue syndrome.

The level of support provided by the cited studies varies. Some studies are of limited quality, but the bigger issue is the relevance of the findings to the recommendations. Several studies indicate areas of unreliability in the potential application of PSG, such as its use in the evaluation of persons with psychiatric disorders.

5.3.13 Depression with insomnia

Depression with insomnia is a complaint of difficulty with sleep associated with a psychiatric diagnosis of depression, either unipolar or the depressive phase of a bipolar illness. This application is considered by the 1997 ASDA guideline[23,39] (Appendix 16). Recommendations indicate neither PSG nor MSLT is routinely indicated in establishing the diagnosis of depression, but PSG may be useful in identifying an early response to antidepressant medication.

Cited evidence consists of one meta-analysis on sleep and psychiatric disorders and 13 primary studies, some of which seem to be not directly relevant to the recommendations. None of the studies were concerned with the diagnosis of depression. The meta-analysis indicated reduction in REML was not a specific marker for depression and the findings in three small studies show sleep characteristics are not specifically associated with psychiatric status. The use of PSG in identifying early response to antidepressants is not supported by the two studies cited.

5.3.14 Narcolepsy

Narcolepsy is a condition characterized by irresistible sleepiness and untimely occurrences of partial REM sleep, such as cataplexy or sleep paralysis. Assessment of the severity and cause of the condition is important before proceeding to treatment with medication.

The AASM[35] considered narcolepsy in a guideline that follows material in the 1997 ASDA publication.[23,39] Both recommend the use of PSG and MSLT to evaluate suspected narcolepsy, with repeat testing where necessary, to investigate new symptoms. The 1996 CPSO guideline[19] also referenced narcolepsy (Appendix 18).

Both prospective and retrospective observational studies have established the usefulness of MSLT in improving the certainty of diagnosis of narcolepsy and of PSG in identifying or excluding other sleep disturbance conditions.

Table 14: Recommendations and supporting evidence for insomnia

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
PSG is indicated when sleep-related breathing disorders or PLMD is suspected	6 case series indicating diagnostic gains through PSG for patients with OSA or PLMD; 2 large NRCTs measuring prevalence of OSA and nocturnal myoclonus in insomniacs (evidence quality=B, relevance=B)	PSG not indicated for routine evaluation of transient or chronic insomnia; PSG not indicated for routine evaluation of insomnia due to psychiatric disorders	Based on committee consensus (evidence quality=O) 10 NRCTs indicate PSG is unreliable for such assessment (evidence quality=A, relevance=B)
PSG is indicated when initial diagnosis is uncertain, treatment fails (behavioural or pharmacologic) or precipitous arousals occur with violent or injurious behaviour[34]	7 case series and 1 chart review; level of support for recommendations provided by cited studies varies considerably (evidence quality=C, relevance=B)	PSG not clinically useful in differentiating insomnia associated with dementia from other forms of insomnia, including insomnia associated with depression	2 NRCTs, 2 case series and committee consensus (evidence quality=B, relevance=B)
		PSG not useful in establishing diagnosis of insomnia associated with fibromyalgia or chronic fatigue syndrome, because alpha-delta sleep pattern described in fibromyalgia syndrome is non-specific finding[34]	3 small case series and committee consensus (evidence quality=C, relevance=B)

NRCT=non-randomized controlled trial; OSA=obstructive sleep apnea; PLMD=periodic limb movement disorder; PSG=polysomnography.

Table 15: Recommendations and supporting evidence for
depression with insomnia

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
PSG may be useful in identifying early response to antidepressant medication[39]	1 RCT and 1 NRCT on PSG measures and the use of tricyclic antidepressants; neither provide convincing support for this use of PSG (evidence quality=A, relevance=C)	Neither PSG nor MSLT is routinely indicated in establishing diagnosis of depression[23]	1 meta-analysis and 3 NRCTs indicating neither PSG nor MSLT is useful in diagnosis of depression (evidence quality=A, relevance=A); 8 other studies indicate sleep characteristics not specifically associated with psychiatric status (evidence quality=B, relevance=B)

MSLT=multiple sleep latency test; NRCT=non-randomized controlled trial; PSG=polysomnography; RCT=randomized controlled trial.

5.3.15 Restless legs syndrome and periodic limb movement disorder

Restless legs syndrome (RLS) is a disorder characterized by disagreeable leg sensations that usually occur before sleep onset and cause an almost irresistible urge to move the legs. Periodic limb movement disorder (PLMD) is characterized by periodic episodes of repetitive and highly stereotyped limb movements that occur during sleep.

A German Sleep Society[36] guideline considers adult patients with probable or definite RLS. The AASM 1999 guideline,[37,38] the ASDA 1997 publication[23,39] and the CPSO guideline[19] cover RLS and PLMD (Appendix 19).

The AASM[37,38] and ASDA publications[39] recommend that PSG is not routinely indicated in the management of RLS, but that it is indicated in the diagnosis of PLMD. The German Sleep Society[36] guideline recommends the use of PSG in adult patients with probable or definite RLS in six situations.

The US guideline recommendations on RLS are supported by a small NRCT and three small case series. The cited material provides reasonable indications that the diagnosis of RLS can be adequately undertaken outside the sleep laboratory and that PSG may underestimate or miss RLS in some patients. No evidence is cited to support recommendations on the wider use of PSG given in the other guideline.

Recommendations on PLMD are supported by studies of generally modest quality and power (five comparing clinical assessment with PSG; one series with clinical interviews and no comparison with PSG; and two studies that identified additional symptoms detected by PSG in some patients).

Table 16: Recommendations and supporting evidence for narcolepsy

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
PSG and an MSLT performed on the day after PSG evaluation are routinely indicated in evaluation of suspected narcolepsy; repeat testing is necessary when initial results are negative or ambiguous and when clinical history strongly indicates diagnosis of narcolepsy[23,35]	2 NRCT and 7 case series; diagnosis of narcolepsy may be complex, as typical clinical diagnostic features will not always be present (evidence quality=B, relevance=B)	Debatable whether PSG is required to make diagnosis in every case[19]	Review article, consensus (evidence quality=O)
PSG re-evaluation should be considered if symptoms of sleepiness increase significantly or if specific symptoms develop that suggest new or increased sleep abnormalities[35]	Committee consensus (evidence quality=O)		
Follow-up sleep studies indicated to evaluate objective reponse of daytime sleepiness to treatment and if symptoms deteriorate or where there is suspicion another sleep disorder may co-exist; MWT is helpful in evaluating response to treatment of narcolepsy and related conditions[19]	Consensus (evidence quality=O)		

MSLT=multiple sleep latency test; MWT=maintenance of wakefulness test; NRCT=non-randomized controlled trial; PSG=polysomnography.

Table 17: Recommendations and supporting evidence for
restless legs syndrome and periodic limb movement disorder

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
Diagnosis of RLS or PLMD suggested by clinical examination by full PSG; 2 nights of monitoring may be required to avoid false-negative findings[19] *	Consensus position (evidence quality=O)	PSG not routinely indicated to diagnose or treat RLS[23]* Diagnostic criteria are based on patient's history or bed partner observations for RLS[37]*	2 consensus statements from many sleep disorders centres (evidence quality=O); 1 small NRCT and 3 case series indicating PSG findings may confirm clinical diagnosis, but may underestimate or miss RLS in some patients (evidence quality=B, relevance=B)
PSG recommended in adults with probable or definite RLS when:[36] • symptoms atypical or affected by other disorders • ongoing severe insomnia or lack of drug efficacy • daily sleepiness as leading symptom; patient not impaired by RLS symptoms • severe RLS treated daily with drugs • additional sleep-related respiratory disorder, continuing RLS symptoms under pharmacotherapy • expert's report needed for judicial purposes	No evidence is cited. (evidence quality=O)		
Diagnostic criteria for PLMD based on patient's history combined with PSG.[37]	2 NRCT and 6 case series comparing clinical assessment with PSG (evidence quality=B, relevance=B)		

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	Evidence	Recommendations	Evidence
PSG indicated when diagnosis of PLMD considered because of complaints of repetitive limb movements during sleep and frequent awakenings, fragmented sleep, difficulty main-taining sleep or EDS[23]			

EDS=excessive day time sleepiness; NRCT=non-randomized controlled trial; PLMD=periodic limb movement disorder; PSG=polysomnography; RLS=restless legs syndrome.

5.3.16 Parasomnias and sleep-related epilepsy

Parasomnias are undesirable physiological phenomena that occur predominantly during sleep and may be associated with sleep disorder diagnoses such as arousal disorders, sleepwalking, sleep terrors and REM sleep behaviour disorder (RBD). Sleep-related epilepsy and sleep-related psychiatric disorders are considered in the differential diagnosis.[39]

The main source of information for this application is the 1997 ASDA guideline.[23,39] The CPSO guideline[19] includes suggested indications for full PSG and refererence is also made to PSG in a publication from the International League Against Epilepsy[40] (Appendix 20).

The findings of most studies cited in the ASDA publication provided some indication that PSG can assist in the diagnosis of parasomnias. PSG diagnostic criteria for each of these sleep disorders appear to be established and distinct. Drug treatment of parasomnias often give favourable results and PSG confirmation of the diagnosis is needed before treatment.

The quality of the cited evidence is limited. There are five small non-randomized comparative studies, not all with well matched controls and 22 case series, most with few patients.

5.3.17 Circadian rhythm disorders

Circadian rhythm disorders include time zone change (jet lag) disorder, shift-work disorder, irregular sleep-wake patterns, delayed sleep-phase syndrome, advanced sleep-phase syndrome and non-24-hour sleep-wake disorder.

The 1997 ASDA guideline[23,39] recommended that PSG not be routinely indicated for the diagnosis of circadian rhythm sleep disorders. The three studies cited in support did not relate to the use of sleep laboratories for routine diagnosis and did not demonstrate a value for PSG in recognizing specific circadian rhythm sleep disorders or in directing treatment. The CPSO guideline states that PSG is useful when the diagnosis is persistent circadian rhythm disorders, such as delayed phase syndrome.[19] This reflects a consensus position (Appendix 21).

Table 18: Recommendations and supporting evidence for
parasomnias and sleep-related epilepsy

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	**Evidence**	**Recommendations**	**Evidence**
PSG, including video recording and additional EEG channels, routinely indicated to assist with diagnosis of paroxysmal arousals or other sleep disruptions thought to be seizure-related when clinical evaluation and EEG are inconclusive	3 case series on diagnosis of parasomnias with sleep-related injuries (evidence quality=C, relevance=B)	PSG not routinely indicated: • in cases of typical, uncomplicated and non-injurious parasomnias when diagnosis is clearly delineated. • for patients with epilepsy who have no specific complaints consistent with sleep disorder	2 reviews and 5 studies cited as "very consistent descriptive literature" (evidence quality=A, relevance=A); absence of direct evidence; limitations of 1 clinical series noted; consideration of other diagnostic options for epilepsy (evidence quality=N)
PSG indicated when evaluating patients with sleep behaviours suggestive of unusual or atypical parasomnias	3 small NRCT and 10 mostly small case series on investigation of sleepwalking (evidence quality=C, relevance=C)		
PSG may be indicated in situations with forensic considerations; PSG may be indicated when presumed parasomnia does not respond to conventional therapy[23]	2 small NRCT and 6 case series on investigation of rapid eye movement behaviour disorder (evidence quality=C, relevance=B); 3 case series on investigation of other parasomnias, some associated with epilepsy (evidence quality=C, relevance=C)		.
Sleep recordings can increase accuracy of epilepsy diagnosis when EEG fails to show epileptiform activity and level of clinical suspicion justifies investigation; PSG should always be used for all-night recordings to record epileptic nocturnal activity[40]	No supporting evidence cited (evidence quality=O)		

EEG=electroencephalography; NRCT=non-randomized controlled trial; PSG=polysomnography.

Table 19: Recommendations and supporting evidence for
circadian rhythm disorders

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	Evidence	Recommendations	Evidence
PSG useful when diagnosis is persistent circadian rhythm disorders, such as delayed phase syndrome[19]	Earlier guideline from ASDA[48,49] and consensus (evidence quality=O)	PSG not routinely indicated for diagnosis of circadian rhythm sleep disorders[23]	3 small studies, NRCT, CSS and case series with consideration of testing procedure's diagnostic value (evidence quality=C, relevance=B)

CSS=cross-sectional study; NRCT=non-randomized controlled trial; PSG=polysomnography.

5.3.18 Chronic fatigue syndrome

Chronic fatigue syndrome is a recognizable pattern of fatigue-related symptoms. If fatigue is prolonged beyond six months, is disabling and is accompanied by other characteristic constitutional and neuropsychiatric symptoms, then chronic fatigue syndrome should be considered.

A Royal Australasian College of Physicians[41] publication mentions that if specific alternative diagnoses (e.g., sleep apnea or multiple sclerosis) are suggested by clinical history or examination, further investigations may be warranted. No evidence is cited in relation to sleep apnea.

As noted under Section 5.3.12, the CPSO document[19] concludes that PSG anomalies provide objective physiologic evidence for the non-restorative sleep symptoms of fibromyalgia and chronic fatigue syndrome. These anomalies, however, may not be specific in patients with these conditions.

Table 20: Recommendations and supporting evidence for
chronic fatigue syndrome

Indications for Sleep Laboratory Use		Limitations to Sleep Laboratory Use	
Recommendations	Evidence	Recommendations	Evidence
Only 7 types of laboratory tests are recommended for routine evaluation of chronic fatigue syndrome; if specific alternative diagnoses (e.g., sleep apnea) are suggested by clinical history or examination, further investigations may be warranted.[41]	No evidence cited in relation to sleep apnea (evidence quality=0)		

6   DISCUSSION

This report about guidelines that provide advice on the referral of patients to sleep laboratories is focused on their recommendations and on the evidence cited in support of the recommendations.

The guidelines and reviews contain much valuable information for those who are responsible for the clinical management of persons with sleep-related disorders. The scope and detail of these publications vary. Some of the more detailed guidelines and reviews, such as those issued by the American Academy of Sleep Medicine and the American Academy of Pediatrics, include details of systematic approaches to the identification and review of pertinent literature during their preparation. The 1997 American Sleep Disorders Association[23,39] guideline and review cover several sleep disorders and have been used as reference sources by other publications. The less detailed guidelines tend to reflect consensus positions or summaries in secondary sources, rather than basing the recommendations on primary studies.

6.1   Evidence in Support of Guideline Recommendations

The level of evidence cited in support of recommendations on many sleep laboratory applications appears to be limited, as indicated in the quality of evidence ratings given in Section 5.3. In all, 81 recommendations included in the reviewed guidelines are mentioned. Of the 81 recommendations, 46 were supported by evidence from primary studies. Of these, six corresponded to category A, with most or all of the cited evidence from well conducted, prospective controlled studies. For 15 recommendations, cited evidence was based on both controlled studies and case series and 25 recommendations were supported only by results from case series or from case series plus controlled studies that had substantial limitations.
Four of the remaining recommendations were supported by an absence of available evidence identified by the guideline. For the other 31, either no evidence was provided or the guideline indicated that there was support by consensus.

Judgements on the relevance of the cited evidence from primary studies suggested that it was highly relevant to the recommendation in 18 cases, of some relevance in 22 and of little or no relevance in six.

6.2   Context of the Guidelines and Supporting Evidence

The publications from professional bodies that have been reviewed are clinical guidelines. The recommendations made may reflect best clinical practice and draw on broader background material related to the conditions they address. However, they often have weak support from the evidence cited.

Much of the cited evidence is of limited quality and some of the recommendations on indications are contradictory. Studies cited in the guidelines in support of recommendations are mostly small and often non-comparative. They rarely relate to the routine use of sleep laboratories.

Sometimes, approaches that are used, for example multi variate analysis, would not be used in routine sleep laboratory operation. In some cases, the cited studies may be providing some evidence of the associations between sleep characteristics and a disorder, but they do not address the practical clinical use of such information.

In most cases, guidelines are not specific about the proportions of patients with particular conditions who will require sleep laboratory studies. Some selection rules have been developed, but may require further validation or may not be applicable in a routine situation. Also, as clinical guidelines, appropriate discretion is allowed for the individual professional's clinical judgement. In some areas, approaches and technologies are still developing and in almost all areas, good quality studies are needed to strengthen and clarify the evidence on efficacy, effectiveness and safety.

The information in the guidelines provides guidance for policy makers and others who are concerned about the planning of sleep laboratory facilities, because details are provided about the recommended approaches to the management of sleep-related disorders. They leave, however, many questions unanswered. Of the publications reviewed, only the Scottish guideline[15] gave any consideration of how recommendations on sleep studies might relate to the practicalities of the health system in terms of available sleep laboratories and appropriately trained personnel. Recommendations likely to be associated with additional resource use by the health service are listed with details about sleep centres and the management of OSA in Scotland.

Appropriate application of the principles outlined in many of the guidelines will help to achieve efficient and effective use of sleep laboratory resources by ensuring the use of clinically appropriate investigations for those who require them. As indicated by Flemons *et al.*[7] in their discussion of Canadian services for the diagnosis and treatment of suspected sleep apnea, the potential requirements for PSG investigations of OSA, if guideline recommendations are followed, are not matched by available resources.

The Canadian guidelines considered in this review appeared in 1996. An update to the guideline published by the College of Physicians and Surgeons of Ontario is in preparation. This report may prove helpful as a source of information for the development of further Canadian guidelines.

6.3   Scope and Limitations of the Review

The first objective for this report was to identify the recommendations on the investigation of individuals in sleep laboratories made in guidelines prepared by professional bodies. The second was to review the evidence cited by the guidelines in support of the recommendations.

Pertinent guidelines were identified through a detailed literature search. Recommendations related to the use of sleep laboratories were listed and attention drawn to instances where there were differences in emphasis between guidelines.

The review of the evidence in support of the recommendations was subject to several limitations. Only those studies cited by the guidelines in support of their recommendations were reviewed. A systematic review of all pertinent literature was not undertaken. In view of the range of sleep

disorders covered by the guidelines, the diversity of supporting publications and data they cite, a series of qualitative reviews was prepared. Details related to the quality of the cited studies and of their relevance to recommendations are provided, but no attempt has been made to quantify these attributes for individual studies. However, ratings of the overall quality and relevance of evidence cited in support of each recommendation have been given.

The information provided gives an indication of the extent of evidence available with respect to the sleep laboratory applications considered.   In some cases, additional information will have emerged from studies that were published since the guidelines were completed. No attempt has been made to review such literature for this report.

7   CONCLUSIONS

The publications on the use of sleep laboratory investigations prepared by professional organizations are clinical guidelines. They contain detailed information for health professionals who are involved in the management of sleep disorders. The recommendations made in the guidelines are generally supported by the results from studies in the literature. The level of evidence for many applications, however, is of limited quality and some cited studies are not directly relevant to the recommendations made. Many recommendations reflect consensus positions and no evidence is cited. There is a need for additional studies of many sleep laboratory applications.

The appropriate application of principles outlined in many of the guidelines will help to achieve the effective use of sleep laboratory resources. The resolution of clinical uncertainties and resource dilemmas will require additional studies and further assessments that are beyond the scope of this report.

8 REFERENCES

1. American Sleep Disorders Association. *The international classification of sleep disorders: diagnostic and coding manual*. Rochester, MD: American Sleep Disorders Association; 1990.

2. Sutton DA, Moldofsky H, Badley EM. Insomnia and health problems in Canadians. *Sleep* 2001;24(6):665-70.

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

4. American Academy of Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep* 1999;22(5):667-89.

5. National Commission on Sleep Disorders Research. *Wake up America: a national sleep alert*. Vol 2. Bethesda (MD): The Commission; 1995.

6. Hailey D, Jacobs P, Mayers I, Mensinkai S. *Auto-titrating nasal continuous positive airway pressure systems in the management of obstructive sleep apnea* [Technology report no 39]. Ottawa: Canadian Coordinating Office for Health Technology Assessment; 2003.

7. Flemons WW, Douglas NJ, Kuna ST, Rodenstein DO, Wheatley J. Access to diagnosis and treatment of patients with suspected sleep apnea. *Am J Respir Crit Care Med* 2004;169(6):668-72.

8. Pack AI, Gurubhagavatula I. Economic implications of the diagnosis of obstructive sleep apnea. *Ann Intern Med* 1999;130(6):533-4.

9. Southcott AM. Sleep studies. *Aust Prescr* 1998;21(2):40-3.

10. George CF, Standards Committees, Canadian Sleep Society and Canadian Thoracic Society. Standards for polysomnography in Canada. *CMAJ* 1996;155(12):1673-8.

11. Chesson AL, Berry RB, Pack A. Practice parameters for the use of portable monitoring devices in the investigation of suspected obstructive sleep apnea in adults. *Sleep* 2003;26(7):907-13.

12. Flemons WW, Littner MR, Rowley JA, Gay P, Anderson WM, Hudgel DW, et al. Home diagnosis of sleep apnea: a systematic review of the literature. An evidence review cosponsored by the American Academy of Sleep Medicine, the American College of Chest Physicians, and the American Thoracic Society. *Chest* 2003;124(4):1543-79.

13. Standards of Practice Committee of the American Sleep Disorders Association. Practice parameters for the use of portable recording in the assessment of obstructive sleep apnea. *Sleep* 1994;17(4):372-7.

14. Ferber R, Millman R, Coppola M, Fleetham J, Murray CF, Iber C, et al. Portable recording in the assessment of obstructive sleep apnea. *Sleep* 1994;17(4):378-92.

15. Scottish Intercollegiate Guidelines Network. *Management of obstructive sleep apnoea/hypopnoea syndrome in adults: a national clinical guideline*. Edinburgh: The Network; 2003. Clinical guideline no 73. Available: http://www.sign.ac.uk/pdf/sign73.pdf (accessed 2004 Aug 13).

16. Connecticut Thoracic Society, Connecticut Neurological Society. Obstructive sleep apnea, polysomnography, and split-night studies: consensus statement of the Connecticut Thoracic Society and the Connecticut Neurological Society. *Conn Med* 2000;64(8):465-8.

17. Schoch OD, Bloch KE, Bischof E, Brändli O, Fitting JW, Frey JG, et al. Care of patients with obstructive sleep apnea syndrome in Switzerland. *Oto Rhino Laryngol Nova* 2001;11(3-4):178-85.

18. Sala H, Nigro C, Rabec C, Guardia AS, Smurra M. Consenso Argentino de trastornos respiratorios vinculados al sueño [Argentine consensus on sleep-related breathing disorders]. *Medicina* 2001;61(3):351-63.

19. Task Force on Clinical Practice Parameters and Facility Standards for Sleep Medicine. *Clinical practice parameters and facility standards for sleep medicine*. 1st ed. Toronto: College of Physicians and Surgeons of Ontario; 1996.

20. Barbé F, Amilibia J, Capote F, Durán J, Mangado NG, Jiménez A, et al. Diagnóstico del síndrome de apneas obstructivas durante el sueño. Informe de Consenso del Área de Insuficiencia Respiratoria y Trastornos del Sueño [Diagnosis of obstructive sleep apnea syndrome. Consensus report from the Respiratory Insufficiency and Sleep Disorders Group]. *Arch Bronconeumol* 1995;31(9):460-2.

21. Laitinen LA, Anttalainen U, Pietinalho A, Hämäläinen P, Koskela K, Expert Advisory Group. Sleep apnoea: Finnish National guidelines for prevention and treatment 2002-2012. *Respir Med* 2003;97(4):337-65.

22. Loube DI, Gay PC, Strohl KP, Pack AI, White DP, Collop NA. Indications for positive airway pressure treatment of adult obstructive sleep apnea patients: a consensus statement. *Chest* 1999;115(3):863-6. Available: http://www.chestjournal.org/cgi/reprint/115/3/863.pdf.

23. Indications for Polysomnography Task Force, Standards of Practice Committee, American Sleep Disorders Association. Practice parameters for the indications for polysomnography and related procedures. *Sleep* 1997;20(6):406-22.

24. Littner M, Hirshkowitz M, Davila D, Anderson WM, Kushida CA, Woodson BT, et al. Practice parameters for the use of auto-titrating continuous positive airway pressure devices for titrating pressures and treating adult patients with obstructive sleep apnea syndrome. An American Academy of Sleep Medicine report. *Sleep* 2002;25(2):143-7.

25. American Sleep Disorders Association. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances. *Sleep* 1995;18(6):511-3.

26. American Association of Respiratory Care, Association of Polysomnography Technologists. AARC-APT clinical practice guideline. Polysomnography. *Respir Care* 1995;40(12):1336-43.

27. Schechter MS, Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome. Technical report: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2002;109(4):e69. Available: http://www.pediatrics.org/cgi/reprint/109/4/e69.pdf.

28. American Thoracic Society. Standards and indications for cardiopulmonary sleep studies in children. *Am J Respir Crit Care Med* 1996;153(2):866-78.

29. Marcus CL, Chapman D, Ward SD, McColley SA, Herrerias CT, Stillwell PC, et al. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2002;109(4):704-12.

30. American Thoracic Society. Idiopathic congenital central hypoventilation syndrome: diagnosis and management. *Am J Respir Crit Care Med* 1999;160(1):368-73.

31. Kerbl R. SIDS und Polygraphie [SIDS and polygraphic recordings]. *Wien Klin Wochenschr* 2000;112(5):204-8.

32. Ipsiroglu OS, Kerbl R, Urschitz M, Kurz R. Österreichisches SIDS-Konsensus-Gespräch anässlich der Wiener SIDS-Präventionskampagne "Sicheres Schlafen" [4th Austrian SIDS-Consensus Meeting and Viennese SIDS prevention campaign "Safe Sleep"]. *Wien Klin Wochenschr* 2000;112(5):187-92.

33. Littner M, Kushida CA, Hartse K, Anderson WM, Davila D, Johnson SF, et al. Practice parameters for the use of laser-assisted uvulopalatoplasty: an update for 2000. *Sleep* 2001;24(5):603-19.

34. Littner M, Hirshkowitz M, Kramer M, Kapen S, Anderson WM, Bailey D, et al. Practice parameters for using polysomnography to evaluate insomnia: an update. *Sleep* 2003;26(6):754-60.

35. Littner M, Johnson SF, McCall WV, Anderson WM, Davila D, Hartse SK, et al. Practice parameters for the treatment of narcolepsy: an update for 2000. *Sleep* 2001;24(4):451-66.

36. Hornyak M, Kotterba S, Trenkwalder C, Behmenburg C, Benes H, Clarenbach P, et al. Consensus statement from the German Sleep Society: indications for performing polysomnography in the diagnosis and treatment of restless legs syndrome. *Sleep Med* 2002;3(5):457-8.

37. Chesson AL, Wise M, Davila D, Johnson S, Littner M, Anderson WM, et al. Practice parameters for the treatment of restless legs syndrome and periodic limb movement disorder . An American Academy of Sleep Medicine Report. Standards of Practice Committee of the American Academy of Sleep Medicine. *Sleep* 1999;22(7):961-8.

38. Hening W, Allen R, Earley C, Kushida C, Picchietti D, Silber M. The treatment of restless legs syndrome and periodic limb movement disorder. An American Academy of Sleep Medicine Review. *Sleep* 1999;22(7):970-99.

39. Chesson AL, Ferber RA, Fry JM, Grigg-Damberger M, Hartse KM, Hurwitz TD, et al. The indications for polysomnography and related procedures. *Sleep* 1997;20(6):423-87.

40. Flink R, Pedersen B, Guekht AB, Malmgren K, Michelucci R, Neville B, et al. Guidelines for the use of EEG methodology in the diagnosis of epilepsy. International League Against Epilepsy: commission report. Commission on European Affairs: Subcommission on European Guidelines. *Acta Neurol Scand* 2002;106(1):1-7.

41. Royal Australasian College of Physicians. Chronic fatigue syndrome. Clinical practice guidelines--2002. *Med J Aust* 2002;176 Suppl:S23-S56. Available: http://www.mja.com.au/public/guides/cfs/cfs2.pdf.

42. Raphaelson M, Hakim TS. Diagnosing sleep apnea in dental patients. *Dent Clin North Am* 2001;45(4):797-816.

43. ATS/ACCP/AASM Taskforce Steering Committee. Executive summary on the systematic review and practice parameters for portable monitoring in the investigation of suspected sleep apnea in adults. *Am J Respir Crit Care Med* 2004;169(10):1160-3.

44. American Thoracic Society. Indications and standards for use of nasal continuous positive airway pressure (CPAP) in sleep apnea syndromes. American Thoracic Society. Official statement adopted March 1994. *Am J Respir Crit Care Med* 1994;150(6 Pt 1):1738-45.

45. Valensi PL. Surveillance des patients traités par pression positive continue : recommandation groupe sommeil de la SPLF. *Rev Mal Respir* 2000;17(3):709-11.

46. Fletcher EC, Donner CF, Midgren B, Zielinski J, Levi-Valensi P, Braghiroli A, et al. Survival in COPD patients with a daytime PaO2 >60 mm Hg with and without nocturnal oxyhemoglobin desaturation. *Chest* 1992;101(3):649-55.

47. Gordon D, Southall DP, Kelly DH, Wilson A, Akselrod S, Richards J, et al. Analysis of heart rate and respiratory patterns in sudden infant death syndrome victims and control infants. *Pediatr Res* 1986;20(7):680-4.

48. Thorpy M, Chesson A, Kader G, Millman R, Potolicchio S, Reite M, et al. Practice parameters for the use of polysomnography in the evaluation of insomnia. *Sleep* 1995;18(1):55-7.

49. Reite M, Buysse D, Reynolds C, Mendelson W. The use of polysomnography in the evaluation of insomnia. *Sleep* 1995;18(1):58-70.

50. Chesson A, Hartse K, Anderson WM, Davila D, Johnson S, Littner M, et al. Practice parameters for the evaluation of chronic insomnia . An American Academy of Sleep Medicine report. Standards of Practice Committee of the American Academy of Sleep Medicine. *Sleep* 2002;23(2):237-41.

51. Sateia MJ, Doghramji K, Hauri PJ, Morin CM. Evaluation of chronic insomnia. An American Academy of Sleep Medicine review. *Sleep* 2000;23(2):243-308.

52. Standards of Practice Committee of the American Sleep Disorders Association. Practice parameters for the use of polysomnography in the evaluation of insomnia. *Sleep* 1995;18(1):55-7.

53. Littner MR, Kushida C, Wise M, Davila DG, Morgenthaler T, Lee-Chiong T, et al. Practice parameters for clinical use of the multiple sleep latency test and the maintenance of wakefulness test. *Sleep* 2005;28(1):113-21.

54. Arand D, Bonnet M, Hurwitz T, Mitler M, Rosa R, Sangal B. The clinical use of the MSLT and MWT. *Sleep* 2005;28(1):123-44.

55. Villa MP, Brunetti L, Bruni O, Cirignotta F, Cozza P, Donzelli G, et al. [Guidelines for the diagnosis of childhood obstructive sleep apnea syndrome]. *Minerva Pediatr* 2004;56(3):239-53.

56. Littner MR, Kushida C, Anderson WM, Bailey D, Berry RB, Hirshkowitz M, et al. Practice parameters for the dopaminergic treatment of restless legs syndrome and periodic limb movement disorder. *Sleep* 2004;27(3):557-9.

57. Hening WA, Allen RP, Earley CJ, Picchietti DL, Silber MH. An update on the dopaminergic treatment of restless legs syndrome and periodic limb movement disorder. *Sleep* 2004;27(3):560-83.

58. Allen RP, Picchietti D, Hening WA, Trenkwalder C, Walters AS, Montplaisir J. Restless legs syndrome: diagnostic criteria, special considerations, and epidemiology. A report from the restless legs syndrome diagnosis and epidemiology workshop at the National Institutes of Health. *Sleep Med* 2003;4(2):101-19.

59. Rombaux P, Bertrand B, Boudewyns A, Deron P, Goffart Y, Hassid S, et al. Standard ENT clinical evaluation of the sleep-disordered breathing patient; a consensus report. *Acta Otorhinolaryngol Belg* 2002;56(2):127-37.

60. Chesson AL, Littner M, Davila D, Anderson WM, Grigg-Damberger M, Hartse K, et al. Practice parameters for the use of light therapy in the treatment of sleep disorders. *Sleep* 1999;22(5):641-60.

61. American Sleep Disorders Association. Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. *Sleep* 1995;18(4):285-7.

62. Sadeh A, Hauri PJ, Kripke DF, Lavie P. The role of actigraphy in the evaluation of sleep disorders. *Sleep* 1995;18(4):288-302.

63. American Electroencephalographic Society. Guideline fifteen: guidelines for polygraphic assessment of sleep-related disorders (polysomnography). *J Clin Neurophysiol* 1994;11(1):116-24.

64. American Electroencephalographic Society. Guideline twelve: guidelines for long-term monitoring for epilepsy. *J Clin Neurophysiol* 1994;11(1):88-110.

65. American Electroencephalographic Society. American Electroencephalographic Society guidelines for polygraphic assessment of sleep-related disorders (polysomnography). *J Clin Neurophysiol* 1992;9(1):88-96.

66. Borbély AA, Åkerstedt T, Benoit O, Holsboer F, Oswald I, Committee on Hypnotics and Sleep Physiology, European Sleep Research Society. Hypnotics and sleep physiology: a consensus report. *Eur Arch Psychiatry Clin Neurosci* 1991;241(1):13-21.

67. Mendelson WB. Use of the sleep laboratory in suspected sleep apnea syndrome: Is one night enough? *Cleve Clin J Med* 1994;61(4):299-303.

68. Meyer TJ, Eveloff SE, Kline LR, Millman RP. One negative polysomnogram does not exclude obstructive sleep apnea. *Chest* 1993;103(3):756-60.

69. Iber C, O'Brien C, Schluter J, Davies S, Leatherman J, Mahowald M. Single night studies in obstructive sleep apnea. *Sleep* 1991;14(5):383-5.

70. Yamashiro Y, Kryger MH. CPAP titration for sleep apnea using a split-night protocol. *Chest* 1995;107(1):62-6. Available: http://www.chestjournal.org/cgi/reprint/107/1/62.pdf (accessed 2004 Mar 30).

71. Sanders MH, Kern NB, Costantino JP, Stiller RA, Studnicki K, Coates J, et al. Adequacy of prescribing positive airway pressure therapy by mask for sleep apnea on the basis of a partial-night trial. *Am Rev Respir Dis* 1993;147(5):1169-74.

72. Scharf SM, Garshick E, Brown R, Tishler PV, Tosteson T, McCarley R. Screening for subclinical sleep-disordered breathing. *Sleep* 1990;13(4):344-53.

73. Charbonneau M, Marin JM, Olha A, Kimoff RJ, Levy RD, Cosio MG. Changes in obstructive sleep apnea characteristics through the night. *Chest* 1994;106(6):1695-701.

74. Sanders MH, Black J, Costantino JP, Kern N, Studnicki K, Coates J. Diagnosis of sleep-disordered breathing by half-night polysomnography. *Am Rev Respir Dis* 1991;144(6):1256-61.

75. Dealberto MJ, Ferber C, Garma L, Lemoine P, Alpérovitch A. Factors related to sleep apnea syndrome in sleep clinic patients. *Chest* 1994;105(6):1753-8.

76. Haraldsson PO, Carenfelt C, Knutsson E, Persson HE, Rinder J. Preliminary report: validity of symptom analysis and daytime polysomnography in diagnosis of sleep apnea. *Sleep* 1992;15(3):261-3.

77. Douglass AB, Bornstein R, Nino-Murcia G, Keenan S, Miles L, Zarcone VP, et al. The Sleep Disorders Questionnaire. I: Creation and multivariate structure of SDQ. *Sleep* 1994;17(2):160-7.

78. Deegan PC, McNicholas WT. Predictive value of clinical features for the obstructive sleep apnoea syndrome. *Eur Respir J* 1996;9(1):117-24.

79. Rauscher H, Popp W, Zwick H. Model for investigating snorers with suspected sleep apnoea. *Thorax* 1993;48(3):275-9.

80. Crocker BD, Olson LG, Saunders NA, Hensley MJ, McKeon JL, Allen KM, et al. Estimation of the probability of disturbed breathing during sleep before a sleep study. *Am Rev Respir Dis* 1990;142(1):14-8.

81. Viner S, Szalai JP, Hoffstein V. Are history and physical examination a good screening test for sleep apnea? *Ann Intern Med* 1991;115(5):356-9.

82. Flemons WW, Whitelaw WA, Brant R, Remmers JE. Likelihood ratios for a sleep apnea clinical prediction rule. *Am J Respir Crit Care Med* 1994;150(5 Pt 1):1279-85.

83. Gyulay S, Olson LG, Hensley MJ, King MT, Allen KM, Saunders NA. A comparison of clinical assessment and home oximetry in the diagnosis of obstructive sleep apnea. *Am Rev Respir Dis* 1993;147(1):50-3.

84. Man GC, Kang BV. Validation of a portable sleep apnea monitoring device. *Chest* 1995;108(2):388-93. Available: http://www.chestjournal.org/cgi/reprint/108/2/388.pdf (accessed 2004 Mar 30).

85. Emsellem HA, Corson WA, Rappaport BA, Hackett S, Smith LG, Hausfeld JN. Verification of sleep apnea using a portable sleep apnea screening device. *South Med J* 1990;83(7):748-52.

86. Orr WC, Eiken T, Pegram V, Jones R, Rundell OH. A laboratory validation study of a portable system for remote recording of sleep-related respiratory disorders. *Chest* 1994;105(1):160-2.

87. Redline S, Tosteson T, Boucher MA, Millman RP. Measurement of sleep-related breathing disturbances in epidemiologic studies. Assessment of the validity and reproducibility of a portable monitoring device. *Chest* 1991;100(5):1281-6.

88. Gyulay S, Gould D, Sawyer B, Pond D, Mant A, Saunders N. Evaluation of a microprocessor-based portable home monitoring system to measure breathing during sleep. *Sleep* 1987;10(2):130-42.

89. White DP, Gibb TJ, Wall JM, Westbrook PR. Assessment of accuracy and analysis time of a novel device to monitor sleep and breathing in the home. *Sleep* 1995;18(2):115-26.

90. Douglas NJ, Thomas S, Jan MA. Clinical value of polysomnography. *Lancet* 1992;339(8789):347-50.

91. Montserrat JM, Alarcón A, Lloberes P, Ballester E, Fornas C, Rodriguez-Roisin R. Adequacy of prescribing nasal continuous positive airway pressure therapy for the sleep apnoea/hypopnoea syndrome on the basis of night time respiratory recording variables. *Thorax* 1995;50(9):969-71.

92. Bradley PA, Mortimore IL, Douglas NJ. Comparison of polysomnography with ResCare Autoset in the diagnosis of the sleep apnoea/hypopnoea syndrome. *Thorax* 1995;50(11):1201-3.

93. Stradling JR, Davies RJ. Is it necessary to record sleep? *Sleep* 1996;19(10 Suppl):S251-S254.

94. Whittle AT, Finch SP, Mortimore IL, MacKay TW, Douglas NJ. Use of home sleep studies for diagnosis of the sleep apnoea/hypopnoea syndrome. *Thorax* 1997;52(12):1068-73. Available: http://thorax.bmjjournals.com/cgi/reprint/52/12/1068 (accessed 2004 Aug 23).

95. Rees K, Wraith PK, Berthon-Jones M, Douglas NJ. Detection of apnoeas, hypopnoeas and arousals by the AutoSet in the sleep apnoea/hypopnoea syndrome. *Eur Respir J* 1998;12(4):764-9.

96. Bennett LS, Langford BA, Stradling JR, Davies RJ. Sleep fragmentation indices as predictors of daytime sleepiness and nCPAP response in obstructive sleep apnea. *Am J Respir Crit Care Med* 1998;158(3):778-86. Available: http://ajrccm.atsjournals.org/cgi/reprint/158/3/778 (accessed 2004 Aug 23).

97. Engleman HM, McDonald JP, Graham D, Lello GE, Kingshott RN, Coleman EL, et al. Randomized crossover trial of two treatments for sleep apnea/hypopnea syndrome: continuous positive airway pressure and mandibular repositioning splint. *Am J Respir Crit Care Med* 2002;166(6):855-9. Available: http://ajrccm.atsjournals.org/cgi/reprint/166/6/855 (accessed 2004 Aug 23).

98. Carrasco O, Montserrat JM, Lloberes P, Ascasco C, Ballester E, Fornas C, et al. Visual and different automatic scoring profiles of respiratory variables in the diagnosis of sleep apnoea-hypopnoea syndrome. *Eur Respir J* 1996;9(1):125-30.

99. García Díaz EM, Capote Gil F, Cano Gómez S, Sánchez Armengol A, Carmona Bernal C, Soto Campos JG. Poligrafía respiratoria en el diagnóstico del síndrome de apneas obstructivas durante el sueño [Respiratory polygraphy in the diagnosis of obstructive sleep apnea syndrome]. *Arch Bronconeumol* 1997;33(2):69-73.

100. Lloberes P, Montserrat JM, Ascaso A, Parra O, Granados A, Alonso P, et al. Comparison of partially attended night time respiratory recordings and full polysomnography in patients with suspected sleep apnoea/hypopnoea syndrome. *Thorax* 1996;51(10):1043-7.

101. Ross SD, Allen IE, Harrison KJ, Kvasz M, Connelly J, Sheinhait IA. *Systematic review of the literature regarding the diagnosis of sleep apnea*. Rockville (MD): Agency for Health Care Policy and Research; 1999. AHCPR publication no 99-E002. Available: http://www.ncbi.nlm.nih.gov/books/bv.fcgi?call=bv.View..ShowSection&rid=hstat1.chapter.2 (accessed 2004 Aug 23).

102. Krieger J, Sforza E, Petiau C, Weiss T. Simplified diagnostic procedure for obstructive sleep apnoea syndrome: lower subsequent compliance with CPAP. *Eur Respir J* 1998;12(4):776-9.

103. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;14(6):540-5.

104. Shelton KE, Woodson H, Gay S, Suratt PM. Pharyngeal fat in obstructive sleep apnea. *Am Rev Respir Dis* 1993;148(2):462-6.

105. Pouliot Z, Peters M, Neufeld H, Kryger MH. Using self-reported questionnaire data to prioritize OSA patients for polysomnography. *Sleep* 1997;20(3):232-6.

106. Jamieson AO. Split-night studies: A new standard? Forcing the examination of outcome. *Sleep* 1991;14(5):381-2.

107. Fanfulla F, Patruno V, Bruschi C, Rampulla C. Obstructive sleep apnoea syndrome: Is the "half-night polysomnography" an adequate method for evaluating sleep profile and respiratory events? *Eur Respir J* 1997;10(8):1725-9.

108. Findley LJ, Wilhoit SC, Suratt PM. Apnea duration and hypoxemia during REM sleep in patients with obstructive sleep apnea. *Chest* 1985;87(4):432-6.

109. Salmi T, Telakivi T, Partinen M. Evaluation of automatic analysis of SCSB, airflow and oxygen saturation signals in patients with sleep related apneas. *Chest* 1989;96(2):255-61.

110. Stoohs R, Guilleminault C. MESAM 4: an ambulatory device for the detection of patients at risk for obstructive sleep apnea syndrome (OSAS). *Chest* 1992;101(5):1221-7.

111. Lévy P, Pépin JL, Deschaux-Blanc C, Paramelle B, Brambilla C. Accuracy of oximetry for detection of respiratory disturbances in sleep apnea syndrome. *Chest* 1996;109(2):395-9. Available: http://www.chestjournal.org/cgi/reprint/109/2/395 (accessed 2004 May 11).

112. Issa FG, Morrison D, Hadjuk E, Iyer A, Feroah T, Remmers JE. Digital monitoring of sleep-disordered breathing using snoring sound and arterial oxygen saturation. *Am Rev Respir Dis* 1993;148(4 Pt 1):1023-9.

113. Svanborg E, Larsson H, Carlsson-Nordlander B, Pirskanen R. A limited diagnostic investigation for obstructive sleep apnea syndrome. Oximetry and static charge sensitive bed. *Chest* 1990;98(6):1341-5.

114.   Cooper BG, Veale D, Griffiths CJ, Gibson GJ. Value of nocturnal oxygen saturation as a screening test for sleep apnoea. *Thorax* 1991;46(8):586-8.

115.   Williams AJ, Yu G, Santiago S, Stein M. Screening for sleep apnea using pulse oximetry and a clinical score. *Chest* 1991;100(3):631-5.

116.   Series F, Marc I, Cormier Y, La Forge J. Utility of nocturnal home oximetry for case finding in patients with suspected sleep apnea hypopnea syndrome. *Ann Intern Med* 1993;119(6):449-53.

117.   Farney RJ, Walker LE, Jensen RL, Walker JM. Ear oximetry to detect apnea and differentiate rapid eye movement (REM) and non-REM (NREM) sleep. Screening for the sleep apnea syndrome. *Chest* 1986;89(4):533-9.

118.   Martin SE, Wraith PK, Deary IJ, Douglas NJ. The effect of nonvisible sleep fragmentation on daytime function. *Am J Respir Crit Care Med* 1997;155(5):1596-601.

119.   Cohen-Mansfield J, Waldhorn R, Werner P, Billig N. Validation of sleep observations in a nursing home. *Sleep* 1990;13(6):512-25.

120.   Lord S, Sawyer B, Pond D, O'Connell D, Eyland A, Mant A, et al. Interrater reliability of computer-assisted scoring of breathing during sleep. *Sleep* 1989;12(6):550-8.

121.   Jacobs D, Ancoli-Israel S, Parker L, Kripke DF. Twenty-four-hour sleep-wake patterns in a nursing home population. *Psychol Aging* 1989;4(3):352-6.

122.   Rasche H, Köster R, Schultz-Coulon HJ. Differentialdiagnose der Rhonchopathie mit Hilfe des MESAM-Systems [Differential diagnosis of rhonchopathy using the MESAM system]. *HNO* 1991;39(12):451-9.

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

124.   Ancoli-Israel S, Klauber MR, Kripke DF, Parker L, Cobarrubias M. Sleep apnea in female patients in a nursing home. Increased risk of mortality. *Chest* 1989;96(5):1054-8.

125.   Lord S, Sawyer B, O'Connell D, King M, Pond D, Eyland A, et al. Night-to-night variability of disturbed breathing during sleep in an elderly community sample. *Sleep* 1991;14(3):252-8.

126.   Richards K. Techniques for measurement of sleep in critical care. *Focus Crit Care* 1987;14(4):34-40.

127.   Ancoli-Israel S. Ambulatory cassette recording of sleep apnea. In: Ebersole JS, editor. *Ambulatory EEG monitoring*. New York: Raven Press; 2004. p.299-315.

128.   Rowley JA, Aboussouan LS, Badr MS. The use of clinical prediction formulas in the evaluation of obstructive sleep apnea. *Sleep* 2000;23(7):929-38.

129.   Bloch KE, Schoch OD, Zhang JN, Russi EW. German version of the Epworth Sleepiness Scale. *Respiration* 1999;66(5):440-7.

130.   Guilleminault C. Clinical features and evaluation of obstructive sleep apnea. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 2nd ed. Philadelphia: WB Saunders; 1994. p.667-77.

131.   Kryger MH. Management of obstructive sleep apnea: overview. In: Kryger MH, Roth T, Dement WC, editors. *Principles and practice of sleep medicine*. 2nd. Philadelphia: WB Saunders; 1994. p.736-47.

132. Guilleminault C, Stoohs R, Clerk A, Cetel M, Maistros P. A cause of excessive daytime sleepiness. The upper airway resistance syndrome. *Chest* 1993;104(3):781-7.

133. Guilleminault C, Stoohs R, Duncan S. Snoring (I). Daytime sleepiness in regular heavy snorers. *Chest* 1991;99(1):40-8.

134. Guilleminault C, Stoohs R, Kim YD, Chervin R, Black J, Clerk A. Upper airway sleep-disordered breathing in women. *Ann Intern Med* 1995;122(7):493-501.

135. Smith PL, Wise RA, Gold AR, Schwartz AR, Permutt S. Upper airway pressure-flow relationships in obstructive sleep apnea. *J Appl Physiol* 1988;64(2):789-95.

136. Issa FG, Sullivan CE. The immediate effects of nasal continuous positive airway pressure treatment on sleep pattern in patients with obstructive sleep apnea syndrome. *Electroencephalogr Clin Neurophysiol* 1986;63(1):10-7.

137. Liistro G, Stanescu DC, Veriter C, Rodenstein DO, Aubert-Tulkens G. Pattern of snoring in obstructive sleep apnea patients and in heavy snorers. *Sleep* 1991;14(6):517-25.

138. Issa FG, Sullivan CE. Upper airway closing pressures in obstructive sleep apnea. *J Appl Physiol* 1984;57(2):520-7.

139. American Thoracic Society, Medical Section of the American Lung Association. Indications and standards for cardiopulmonary sleep studies. *Am Rev Respir Dis* 1989;139(2):559-68.

140. Sanders MH, Stiller RA. Positive airway pressure in the treatment of sleep-related breathing disorders. In: Chokroverty S, editor. *Sleep disorders medicine: basic science, technical considerations, and clinical aspects*. Boston: Butterworth-Heinemann; 1994. p.455-72.

141. Schmidt-Nowara W, Lowe A, Wiegand L, Cartwright R, Perez-Guerra F, Menn S. Oral appliances for the treatment of snoring and obstructive sleep apnea: a review. An American Sleep Disorders Association Review. *Sleep* 1995;18(6):501-10.

142. Standards of Practice Committee of the American Sleep Disorders Association. Practice parameters for the treatment of obstructive sleep apnea in adults: the efficacy of surgical modifications of the upper airway. An American Sleep Disorders Association Report. *Sleep* 1996;19(2):152-5.

143. Noseda A, Kempenaers C, Kerkhofs M, Houben JJ, Linkowski P. Sleep apnea after 1 year domiciliary nasal-continuous positive airway pressure and attempted weight reduction. Potential for weaning from continuous positive airway pressure. *Chest* 1996;109(1):138-43. Available: http://www.chestjournal.org/cgi/reprint/109/1/138 (accessed 2004 Jun 28).

144. Peiser J, Lavie P, Ovnat A, Charuzi I. Sleep apnea syndrome in the morbidly obese as an indication for weight reduction surgery. *Ann Surg* 1984;199(1):112-5.

145. Charuzi I, Lavie P, Peiser J, Peled R. Bariatric surgery in morbidly obese sleep-apnea patients: short- and long-term follow-up. *Am J Clin Nutr* 1992;55(2 Suppl):594S-6S.

146. Pillar G, Peled R, Lavie P. Recurrence of sleep apnea without concomitant weight increase 7.5 years after weight reduction surgery. *Chest* 1994;106(6):1702-4.

147. Thorpy MJ, for the Standards of Practice Committee of the American Sleep Disorders Association. The clinical use of the Multiple Sleep Latency Test. *Sleep* 1992;15(3):268-76.

148. Sforza E, Krieger J, Weitzenblum E, Apprill M, Lampert E, Ratamaharo J. Long-term effects of treatment with nasal continuous positive airway pressure on daytime lung function and pulmonary hemodynamics in patients with obstructive sleep apnea. *Am Rev Respir Dis* 1990;141(4 Pt 1):866-70.

149. Krieger J, Grucker D, Sforza E, Chambron J, Kurtz D. Left ventricular ejection fraction in obstructive sleep apnea. Effects of long-term treatment with nasal continuous positive airway pressure. *Chest* 1991;100(4):917-21.

150. Kribbs NB, Pack AI, Kline LR, Smith PL, Schwartz AR, Schubert NM, et al. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am Rev Respir Dis* 1993;147(4):887-95.

151. Schwartz AR, Gold AR, Schubert N, Stryzak A, Wise RA, Permutt S, et al. Effect of weight loss on upper airway collapsibility in obstructive sleep apnea. *Am Rev Respir Dis* 1991;144(3 Pt 1):494-8.

152. Strohl KP, Cherniack NS, Gothe B. Physiologic basis of therapy for sleep apnea. *Am Rev Respir Dis* 1986;134(4):791-802.

153. Strohl KP, Chester CS. Polysomnography for breathing disorders in sleep. In: Nochomovitz ML, Cherniack NS, editors. *Noninvasive respiratory monitoring*. New York: Churchill Livingstone; 1986. p.153-66.

154. Sullivan CE, Issa FG, Bruderer J, McCaley V, Bye PT, Grunstein R, et al. Treatment of cardiorespiratory disturbances during sleep. *Interdisciplinary Top Gerontol* 1987;22:47-67.

155. Quera-Salva MA, Guilleminault C, Chevret S, Troche G, Fromageot C, Crowe McCann C, et al. Breathing disorders during sleep in myasthenia gravis. *Ann Neurol* 1992;31(1):86-92.

156. Steljes DG, Kryger MH, Kirk BW, Millar TW. Sleep in postpolio syndrome. *Chest* 1990;98(1):133-40.

157. Muller NL, Francis PW, Gurwitz D, Levison H, Bryan AC. Mechanism of hemoglobin desaturation during rapid-eye-movement sleep in normal subjects and in patients with cystic fibrosis. *Am Rev Respir Dis* 1980;121(3):463-9.

158. Gay PC, Westbrook PR, Daube JR, Litchy WJ, Windebank AJ, Iverson R. Effects of alterations in pulmonary function and sleep variables on survival in patients with amyotrophic lateral sclerosis. *Mayo Clin Proc* 1991;66(7):686-94.

159. Guilleminault C, Kurland G, Winkle R, Miles LE. Severe kyphoscoliosis, breathing, and sleep: the "Quasimodo" syndrome during sleep. *Chest* 1981;79(6):626-30.

160. Bye PT, Ellis ER, Issa FG, Donnelly PM, Sullivan CE. Respiratory failure and sleep in neuromuscular disease. *Thorax* 1990;45(4):241-7.

161. Khan Y, Heckmatt JZ, Dubowitz V. Sleep studies and supportive ventilatory treatment in patients with congenital muscle disorders. *Arch Dis Child* 1996;74(3):195-200.

162. Hill NS. Noninvasive ventilation. Does it work, for whom, and how? *Am Rev Respir Dis* 1993;147(4):1050-5.

163. McNicholas WT, Coffey M, Fitzgerald MX. Ventilation and gas exchange during sleep in patients with interstitial lung disease. *Thorax* 1986;41(10):777-82.

164. Perez-Padilla R, West P, Lertzman M, Kryger MH. Breathing during sleep in patients with interstitial lung disease. *Am Rev Respir Dis* 1985;132(2):224-9.

165. Bye PT, Issa F, Berthon-Jones M, Sullivan CE. Studies of oxygenation during sleep in patients with interstitial lung disease. *Am Rev Respir Dis* 1984;129(1):27-32.

166. Carroll N, Bain RJ, Smith PE, Saltissi S, Edwards RH, Calverley PM. Domiciliary investigation of sleep-related hypoxaemia in Duchenne muscular dystrophy. *Eur Respir J* 1991;4(4):434-40.

167. Smith PE, Edwards RH, Calverley PM. Ventilation and breathing pattern during sleep in Duchenne muscular dystrophy. *Chest* 1989;96(6):1346-51.

168. van der Meché FG, Bogaard JM, van der Sluys JC, Schimsheimer RJ, Ververs CC, Busch HF. Daytime sleep in myotonic dystrophy is not caused by sleep apnoea. *J Neurol Neurosurg Psychiatry* 1994;57(5):626-8.

169. Cirignotta F, Mondini S, Zucconi M, Barrot-Cortes E, Sturani C, Schiavina M, et al. Sleep-related breathing impairment in myotonic dystrophy. *J Neurol* 1987;235(2):80-5.

170. Calverley PM, Brezinova V, Douglas NJ, Catterall JR, Flenley DC. The effect of oxygenation on sleep quality in chronic bronchitis and emphysema. *Am Rev Respir Dis* 1982;126(2):206-10.

171. Catterall JR, Douglas NJ, Calverley PM, Shapiro CM, Brezinova V, Brash HM, et al. Transient hypoxemia during sleep in chronic obstructive pulmonary disease is not a sleep apnea syndrome. *Am Rev Respir Dis* 1983;128(1):24-9.

172. Chaouat A, Weitzenblum E, Krieger J, Ifoundza T, Oswald M, Kessler R. Association of chronic obstructive pulmonary disease and sleep apnea syndrome. *Am J Respir Crit Care Med* 1995;151(1):82-6.

173. Coccagna G, Lugaresi E. Arterial blood gases and pulmonary and systemic arterial pressure during sleep in chronic obstructive pulmonary disease. *Sleep* 1978;1(2):117-24.

174. Cormick W, Olson LG, Hensley MJ, Saunders NA. Nocturnal hypoxaemia and quality of sleep in patients with chronic obstructive lung disease. *Thorax* 1986;41(11):846-54.

175. Douglas NJ, Calverley PM, Leggett RJ, Brash HM, Flenley DC, Brezinova V. Transient hypoxæmia during sleep in chronic bronchitis and emphysema. *Lancet* 1979;1(8106):1-4.

176. Fleetham J, West P, Mezon B, Conway W, Roth T, Kryger M. Sleep, arousals, and oxygen desaturation in chronic obstructive pulmonary disease. The effect of oxygen therapy. *Am Rev Respir Dis* 1982;126(3):429-33.

177. Fletcher EC, Gray BA, Levin DC. Nonapneic mechanisms of arterial oxygen desaturation during rapid-eye-movement sleep. *J Appl Physiol* 1983;54(3):632-9.

178. Schulte FJ, Albani M, Schnizer H, Bentele K. Neuronal control of neonatal respiration - sleep apnea and the sudden infant death syndrome. *Neuropediatrics* 1982;13 Suppl:3-14.

179. Guilleminault C, Cummiskey J, Motta J. Chronic obstructive airflow disease and sleep studies. *Am Rev Respir Dis* 1980;122(3):397-406.

180. Littner MR, McGinty DJ, Arand DL. Determinants of oxygen desaturation in the course of ventilation during sleep in chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1980;122(6):849-57.

181. Wynne JW, Block AJ, Hemenway J, Hunt LA, Flick MR. Disordered breathing and oxygen desaturation during sleep in patients with chronic obstructive lung disease (COLD). *Am J Med* 1979;66(4):573-9.

182. Fletcher EC, Miller J, Divine GW, Fletcher JG, Miller T. Nocturnal oxyhemoglobin desaturation in COPD patients with arterial oxygen tensions above 60 mm Hg. *Chest* 1987;92(4):604-8.

183. American Thoracic Society. Cardiorespiratory sleep studies in children. Establishment of normative data and polysomnographic predictors of morbidity. *Am J Respir Crit Care Med* 1999;160(4):1381-7. Available: http://ajrccm.atsjournals.org/cgi/reprint/160/4/1381 (accessed 2004 May 5).

184. Marcus CL, Omlin KJ, Basinki DJ, Bailey SL, Rachal AB, Von Pechmann WS, et al. Normal polysomnographic values for children and adolescents. *Am Rev Respir Dis* 1992;146(5 Pt 1):1235-9.

185. Rosen CL, D'Andrea L, Haddad GG. Adult criteria for obstructive sleep apnea do not identify children with serious obstruction. *Am Rev Respir Dis* 1992;146(5 Pt 1):1231-4.

186. Rosen CL. Clinical features of obstructive sleep apnea hypoventilation syndrome in otherwise healthy children. *Pediatr Pulmonol* 1999;27(6):403-9.

187. Marcus CL, Greene MG, Carroll JL. Blood pressure in children with obstructive sleep apnea. *Am J Respir Crit Care Med* 1998;157(4 Pt 1):1098-103. Available: http://ajrccm.atsjournals.org/cgi/reprint/157/4/1098 (accessed 2004 May 5).

188. Carroll JL, McColley SA, Marcus CL, Curtis S, Loughlin GM. Inability of clinical history to distinguish primary snoring from obstructive sleep apnea syndrome in children. *Chest* 1995;108(3):610-8. Available: http://www.chestjournal.org/cgi/reprint/108/3/610 (accessed 2004 May 5).

189. Brouillette RT, Morielli A, Leimanis A, Waters KA, Luciano R, Ducharme FM. Nocturnal pulse oximetry as an abbreviated testing modality for pediatric obstructive sleep apnea. *Pediatrics* 2000;105(2):405-12.

190. Sivan Y, Kornecki A, Schonfeld T. Screening obstructive sleep apnoea syndrome by home videotape recording in children. *Eur Respir J* 1996;9(10):2127-31.

191. Goldstein NA, Sculerati N, Walsleben JA, Bhatia N, Friedman DM, Rapoport DM. Clinical diagnosis of pediatric obstructive sleep apnea validated by polysomnography. *Otolaryngol Head Neck Surg* 1994;111(5):611-7.

192. Lamm C, Mandeli J, Kattan M. Evaluation of home audiotapes as an abbreviated test for obstructive sleep apnea syndrome (OSAS) in children. *Pediatr Pulmonol* 1999;27(4):267-72.

193. Marcus CL, Keens TG, Ward SL. Comparison of nap and overnight polysomnography in children. *Pediatr Pulmonol* 1992;13(1):16-21.

194. Saeed MM, Keens TG, Stabile MW, Bolokowicz J, Davidson Ward SL. Should children with suspected obstructive sleep apnea syndrome and normal nap sleep studies have overnight sleep studies? *Chest* 2000;118(2):360-5. Available: http://www.chestjournal.org/cgi/reprint/118/2/360 (accessed 2004 May 5).

195. Carroll JL, Loughlin GM. Diagnostic criteria for obstructive sleep apnea syndrome in children. *Pediatr Pulmonol* 1992;14(2):71-4.

196. McColley SA, April MM, Carroll JL, Naclerio RM, Loughlin GM. Respiratory compromise after adenotonsillectomy in children with obstructive sleep apnea. *Arch Otolaryngol Head Neck Surg* 1992;118(9):940-3.

197. Rosen GM, Muckle RP, Mahowald MW, Goding GS, Ullevig C. Postoperative respiratory compromise in children with obstructive sleep apnea syndrome: Can it be anticipated? *Pediatrics* 1994;93(5):784-8.

198. Silvestri JM, Weese-Mayer DE, Bass MT, Kenny AS, Hauptman SA, Pearsall SM. Polysomnography in obese children with a history of sleep-associated breathing disorders. *Pediatr Pulmonol* 1993;16(2):124-9.

199. Mallory GB, Fiser DH, Jackson R. Sleep-associated breathing disorders in morbidly obese children and adolescents. *J Pediatr* 1989;115(6):892-7.

200. Marcus CL, Crockett DM, Ward SL. Evaluation of epiglottoplasty as treatment for severe laryngomalacia. *J Pediatr* 1990;117(5):706-10.

201. Maddern BR, Reed HT, Ohene-Frempong K, Beckerman RC. Obstructive sleep apnea syndrome in sickle cell disease. *Ann Otol Rhinol Laryngol* 1989;98(3):174-8.

202. Samuels MP, Stebbens VA, Davies SC, Picton-Jones E, Southall DP. Sleep related upper airway obstruction and hypoxaemia in sickle cell disease. *Arch Dis Child* 1992;67(7):925-9.

203. Craft JA, Alessandrini E, Kenney LB, Klein B, Bray G, Luban NL, et al. Comparison of oxygenation measurements in pediatric patients during sickle cell crises. *J Pediatr* 1994;124(1):93-5.

204. Suen JS, Arnold JE, Brooks LJ. Adenotonsillectomy for treatment of obstructive sleep apnea in children. *Arch Otolaryngol Head Neck Surg* 1995;121(5):525-30.

205. Guilleminault C, Ariagno R, Korobkin R, Nagel L, Baldwin R, Coons S, et al. Mixed and obstructive sleep apnea and near miss for sudden infant death syndrome: 2. Comparison of near miss and normal control infants by age. *Pediatrics* 1979;64(6):882-91.

206. Southall DP, Richards JM, Rhoden KJ, Alexander JR, Shinebourne EA, Arrowsmith WA, et al. Prolonged apnea and cardiac arrhythmias in infants discharged from neonatal intensive care units: failure to predict an increased risk for sudden infant death syndrome. *Pediatrics* 1982;70(6):844-51.

207. Identification of infants destined to die unexpectedly during infancy: evaluation of predictive importance of prolonged apnoea and disorders of cardiac rhythm or conduction: first report of a multicentred prospective study into the sudden infant death syndrome. *Br Med J (Clin Res Ed)* 1983;286(6371):1092-6.

208. Schechtman VL, Harper RM, Wilson AJ, Southall DP. Sleep apnea in infants who succumb to the sudden infant death syndrome. *Pediatrics* 1991;87(6):841-6.

209. Southall DP, Richards JM, Stebbens V, Wilson AJ, Taylor V, Alexander JR. Cardiorespiratory function in 16 full-term infants with sudden infant death syndrome. *Pediatrics* 1986;78(5):787-96.

210. Waggener TB, Southall DP, Scott LA. Analysis of breathing patterns in a prospective population of term infants does not predict susceptibility to sudden infant death syndrome. *Pediatr Res* 1990;27(2):113-7.

211. Kahn A, Blum D, Rebuffat E, Sottiaux M, Levitt J, Bochner A, et al. Polysomnographic studies of infants who subsequently died of sudden infant death syndrome. *Pediatrics* 1988;82(5):721-7.

212. Monod N, Plouin P, Sternberg B, Peirano P, Pajot N, Flores R, et al. Are polygraphic and cardiopneumographic respiratory patterns useful tools for predicting the risk for sudden infant death syndrome? A 10-year study. *Biol Neonate* 1986;50(3):147-53.

213. Bentele KH, Albani M. Are there tests predictive for prolonged apnoea and SIDS? A review of epidemiological and functional studies. *Acta Paediatr Scand Suppl* 1988;342:1-21.

214. Hunt CE, Brouillette RT, Hanson D. Apnea-onset definition significantly affects pneumogram results. *Sleep* 1988;11(3):286-90.

215. Khosh M, Keidar A, Zammit GK, Krespi YP. Self-reported symptoms and polysomnographic findings in laser-assisted uvulopalatoplasty candidates [abstract]. *Otolaryngol Head Neck Surg* 1994;43.

216. Coleman RM, Roffwarg HP, Kennedy SJ, Guilleminault C, Cinque J, Cohn MA, et al. Sleep-wake disorders based on a polysomnographic diagnosis. A national cooperative study. *JAMA* 1982;247(7):997-1003.

217. Lichstein KL, Riedel BW, Lester KW, Aguillard RN. Occult sleep apnea in a recruited sample of older adults with insomnia. *J Consult Clin Psychol* 1999;67(3):405-10.

218. Roehrs T, Conway W, Wittig R, Zorick F, Sicklesteel J, Roth T. Sleep-wake complaints in patients with sleep-related respiratory disturbances. *Am Rev Respir Dis* 1985;132(3):520-3.

219. Zorick FJ, Roth T, Hartze KM, Piccione PM, Stepanski EJ. Evaluation and diagnosis of persistent insomnia. *Am J Psychiatry* 1981;138(6):769-73.

220. Edinger JD, Hoelscher TJ, Webb MD, Marsh GR, Radtke RA, Erwin CW. Polysomnographic assessment of DIMS: empirical evaluation of its diagnostic value. *Sleep* 1989;12(4):315-22.

221. Vgontzas AN, Kales A, Bixler EO, Manfredi RL, Vela-Bueno A. Usefulness of polysomnographic studies in the differential diagnosis of insomnia. *Int J Neurosci* 1995;82(1-2):47-60.

222. Reynolds CF, Coble PA, Black RS, Holzer B, Carroll R, Kupfer DJ. Sleep disturbances in a series of elderly patients: polysomnographic findings. *J Am Geriatr Soc* 1980;28(4):164-70.

223. Kales A, Bixler EO, Soldatos CR, Vela-Bueno A, Caldwell AB, Cadieux RJ. Biopsychobehavioral correlates of insomnia, part 1: role of sleep apnea and nocturnal myoclonus. *Psychosomatics* 1982;23(6):589-600.

224. Jacobs EA, Reynolds CF, Kupfer DJ, Lovin PA, Ehrenpreis AB. The role of polysomnography in the differential diagnosis of chronic insomnia. *Am J Psychiatry* 1988;145(3):346-9.

225. Hauri PJ, Friedman M, Ravaris CL. Sleep in patients with spontaneous panic attacks. *Sleep* 1989;12(4):323-37.

226. Mendelson WB, James SP, Garnett D, Sack DA, Rosenthal NE. A psychophysiological study of insomnia. *Psychiatry Res* 1986;19(4):267-84.

227. Vitiello MV, Bokan JA, Kukull WA, Muniz RL, Smallwood RG, Prinz PN. Rapid eye movement sleep measures of Alzheimer's-type dementia patients and optimally healthy aged individuals. *Biol Psychiatry* 1984;19(5):721-34.

228. Prinz PN, Peskind ER, Vitaliano PP, Raskind MA, Eisdorfer C, Zemcuznikov N, et al. Changes in the sleep and waking EEGs of nondemented and demented elderly subjects. *J Am Geriatr Soc* 1982;30(2):86-93.

229. Prinz PN, Vitaliano PP, Vitiello MV, Bokan J, Raskind M, Peskind E, et al. Sleep, EEG and mental function changes in senile dementia of the Alzheimer's type. *Neurobiol Aging* 1982;3(4):361-70.

230. Reynolds CF, Kupfer DJ, Hoch CC, Houck PR, Stack JA, Berman SR, et al. Sleep deprivation as a probe in the elderly. *Arch Gen Psychiatry* 1987;44(11):982-90.

231. Vitiello MV, Bliwise DL, Prinz PN. Sleep in Alzheimer's disease and the sundown syndrome. *Neurology* 1992;42(7 Suppl 6):83-93.

232. EEG arousals: scoring rules and examples: a preliminary report from the Sleep Disorders Atlas Task Force of the American Sleep Disorders Association. *Sleep* 1992;15(2):173-84.

233. Gillin JC, Duncan W, Pettigrew KD, Frankel BL, Snyder F. Successful separation of depressed, normal, and insomniac subjects by EEG sleep data. *Arch Gen Psychiatry* 1979;36(1):85-90.

234. Haynes SN, Follingstad DR, McGowan WT. Insomnia: sleep patterns and anxiety level. *J Psychosom Res* 1974;18(2):69-74.

235. Merica H, Blois R, Bovier P, Gaillard JM. New variables for defining sleep continuity. *Physiol Behav* 1993;54(5):825-31.

236. Thase ME, Kupfer DJ, Fasiczka AJ, Buysse DJ, Simons AD, Frank E. Identifying an abnormal electroencephalographic sleep profile to characterize major depressive disorder. *Biol Psychiatry* 1997;41(9):964-73.

237. Arriaga F, Paiva T, Matos-Pires A, Cavaglia F, Lara E, Bastos L. The sleep of non-depressed patients with panic disorder: a comparison with normal controls. *Acta Psychiatr Scand* 1996;93(3):191-4.

238. Hohagen F, Lis S, Krieger S, Winkelmann G, Riemann D, Fritsch-Montero R, et al. Sleep EEG of patients with obsessive-compulsive disorder. *Eur Arch Psychiatry Clin Neurosci* 1994;243(5):273-8.

239. Hurwitz TD, Mahowald MW, Kuskowski M, Engdahl BE. Polysomnographic sleep is not clinically impaired in Vietnam combat veterans with chronic posttraumatic stress disorder. *Biol Psychiatry* 1998;44(10):1066-73.

240. Lauer CJ, Krieg JC, Garcia-Borreguero D, Özdaglar A, Holsboer F. Panic disorder and major depression: a comparative electroencephalographic sleep study. *Psychiatry Res* 1992;44(1):41-54.

241. Pecknold JC, Luthe L. Sleep studies and neurochemical correlates in panic disorder and agoraphobia. *Prog Neuropsychopharmacol Biol Psychiatry* 1990;14(5):753-8.

242. Stein MB, Enns MW, Kryger MH. Sleep in nondepressed patients with panic disorder: II. Polysomnographic assessment of sleep architecture and sleep continuity. *J Affect Disord* 1993;28(1):1-6.

243. Allen SR, Seiler WO, Stähelin HB, Spiegel R. Seventy-two hour polygraphic and behavioral recordings of wakefulness and sleep in a hospital geriatric unit: comparison between demented and nondemented patients. *Sleep* 1987;10(2):143-59.

244. Evans BM, Bartlett JR. Prediction of outcome in severe head injury based on recognition of sleep related activity in the polygraphic electroencephalogram. *J Neurol Neurosurg Psychiatry* 1995;59(1):17-25.

245. Manu P, Lane TJ, Matthews DA, Castriotta RJ, Watson RK, Abeles M. Alpha-delta sleep in patients with a chief complaint of chronic fatigue. *South Med J* 1994;87(4):465-70.

246. Mahowald MW, Mahowald ML, Bundlie SR, Ytterberg SR. Sleep fragmentation in rheumatoid arthritis. *Arthritis Rheum* 1989;32(8):974-83.

247. Moldofsky H, Scarisbrick P, England R, Smythe H. Musculoskeletal symptoms and non-REM sleep disturbance in patients with "fibrositis syndrome" and healthy subjects. *Psychosom Med* 1975;37(4):341-51.

248. Hauri P, Hawkins DR. Alpha-delta sleep. *Electroencephalogr Clin Neurophysiol* 1973;34(3):233-7.

249. Benca RM, Obermeyer WH, Thisted RA, Gillin JC. Sleep and psychiatric disorders. A meta-analysis. *Arch Gen Psychiatry* 1992;49(8):651-68.

250. Benson KL, King R, Gordon D, Silva JA, Zarcone VP. Sleep patterns in borderline personality disorder. *J Affect Disord* 1990;18(4):267-73.

251. Kupfer DJ, Ehlers CL, Frank E, Grochocinski VJ, McEachran AB, Buhari A. Persistent effects of antidepressants: EEG sleep studies in depressed patients during maintenance treatment. *Biol Psychiatry* 1994;35(10):781-93.

252. Lauer CJ, Riemann D, Wiegand M, Berger M. From early to late adulthood. Changes in EEG sleep of depressed patients and healthy volunteers. *Biol Psychiatry* 1991;29(10):979-93.

253. Lauer CJ, Schreiber W, Holsboer F, Krieg JC. In quest of identifying vulnerability markers for psychiatric disorders by all-night polysomnography. *Arch Gen Psychiatry* 1995;52(2):145-53.

254. Nofzinger EA, Thase ME, Reynolds CF, Himmelhoch JM, Mallinger A, Houck P, et al. Hypersomnia in bipolar depression: a comparison with narcolepsy using the multiple sleep latency test. *Am J Psychiatry* 1991;148(9):1177-81.

255. Reynolds CF, Hoch CC, Buysse DJ, Houck PR, Schlernitzauer M, Pasternak RE, et al. Sleep after spousal bereavement: a study of recovery from stress. *Biol Psychiatry* 1993;34(11):791-7.

256. Vitiello MV, Prinz PN, Avery DH, Williams DE, Ries RK, Bokan JA, et al. Sleep is undisturbed in elderly, depressed individuals who have not sought health care. *Biol Psychiatry* 1990;27(4):431-40.

257. Ware JC, Brown FW, Moorad PJ, Pittard JT, Cobert B. Effects on sleep: a double-blind study comparing trimipramine to imipramine in depressed insomniac patients. *Sleep* 1989;12(6):537-49.

258. Cartwright RD, Wood E. Adjustment disorders of sleep: the sleep effects of a major stressful event and its resolution. *Psychiatry Res* 1991;39(3):199-209.

259. Rosenthal LD, Merlotti L, Young DK, Zorick FJ, Wittig RM, Roehrs TA, et al. Subjective and polysomnographic characteristics of patients diagnosed with narcolepsy. *Gen Hosp Psychiatry* 1990;12(3):191-7.

260. Aldrich MS. The clinical spectrum of narcolepsy and idiopathic hypersomnia. *Neurology* 1996;46(2):393-401.

261. Mitler MM, Van den Hoed J, Carskadon MA, Richardson G, Park R, Guilleminault C, et al. REM sleep episodes during the Multiple Sleep Latency Test in narcoleptic patients. *Electroencephalogr Clin Neurophysiol* 1979;46(4):479-81.

262. Richardson GS, Carskadon MA, Flagg W, Van den Hoed J, Dement WC, Mitler MM. Excessive daytime sleepiness in man: multiple sleep latency measurement in narcoleptic and control subjects. *Electroencephalogr Clin Neurophysiol* 1978;45(5):621-7.

263. Zorick F, Roehrs T, Koshorek G, Sicklesteel J, Hartse K, Wittig R, et al. Patterns of sleepiness in various disorders of excessive daytime somnolence. *Sleep* 1982;5 Suppl 2:S165-S174.

264. Amira SA, Johnson TS, Logowitz NB. Diagnosis of narcolepsy using the multiple sleep latency test: analysis of current laboratory criteria. *Sleep* 1985;8(4):325-31.

265. Moscovitch A, Partinen M, Guilleminault C. The positive diagnosis of narcolepsy and narcolepsy's borderland. *Neurology* 1993;43(1):55-60.

266. Van den Hoed J, Kraemer H, Guilleminault C, Zarcone VP, Miles LE, Dement WC, et al. Disorders of excessive daytime somnolence: polygraphic and clinical data for 100 patients. *Sleep* 1981;4(1):23-37.

267. Roehrs T, Zorick F, Sicklesteel J, Wittig R, Roth T. Excessive daytime sleepiness associated with insufficient sleep. *Sleep* 1983;6(4):319-25.

268. Dahl RE, Holttum J, Trubnick L. A clinical picture of child and adolescent narcolepsy. *J Am Acad Child Adolesc Psychiatry* 1994;33(6):834-41.

269. Rosenthal L, Folkerts M, Roehrs T, Zorick F, Roth T. Sleepiness and sleep onset REM periods in the absence of clinical symptomatology. *Biol Psychiatry* 1994;36(5):341-3.

270. Broughton R, Dunham W, Newman J, Lutley K, Duschesne P, Rivers M. Ambulatory 24 hour sleep-wake monitoring in narcolepsy-cataplexy compared to matched controls. *Electroencephalogr Clin Neurophysiol* 1988;70(6):473-81.

271. Mitler MM, Hajdukovic R, Erman MK. Treatment of narcolepsy with methamphetamine. *Sleep* 1993;16(4):306-17.

272. Uchiyama M, Mayer G, Meier-Ewert K. Differential effects of extended sleep in narcoleptic patients. *Electroencephalogr Clin Neurophysiol* 1994;91(3):212-8.

273. Intrinsic sleep disorders. In: *The international classification of sleep disorders, revised : diagnostic and coding manual*. Rochester, MN: American Sleep Disorders Association; 2001. p.27-71.

274. Walters AS, International Restless Legs Syndrome Study Group. Toward a better definition of the restless legs syndrome. *Mov Disord* 1995;10(5):634-42.

275. Montplaisir J, Boucher S, Poirier G, Lavigne G, Lapierre O, Lespérance P. Clinical, polysomnographic, and genetic characteristics of restless legs syndrome: a study of 133 patients diagnosed with new standard criteria. *Mov Disord* 1997;12(1):61-5.

276. Wetter TC, Pollmächer T. Restless legs and periodic leg movements in sleep syndromes. *J Neurol* 1997;244(4 Suppl 1):S37-S45.

277. Montplaisir J, Boucher S, Nicolas A, Lesperance P, Gosselin A, Rompré P, et al. Immobilization tests and periodic leg movements in sleep for the diagnosis of restless leg syndrome. *Mov Disord* 1998;13(2):324-9.

278. Coccagna G, Lugaresi E. Restless legs syndrome and nocturnal myoclonus. *Int J Neurol* 1981;15(1-2):77-87.

279. Pelletier G, Lorrain D, Montplaisir J. Sensory and motor components of the restless legs syndrome. *Neurology* 1992;42(9):1663-6.

280. Walters AS, Hening WA, Chokroverty S. Review and videotape recognition of idiopathic restless legs syndrome. *Mov Disord* 1991;6(2):105-10.

281. Walters AS, Hening WA, Kavey N, Chokroverty S, Gidro-Frank S. A double-blind randomized crossover trial of bromocriptine and placebo in restless legs syndrome. *Ann Neurol* 1988;24(3):455-8.

282. Walters A, Hening W, Côté L, Fahn S. Dominantly inherited restless legs with myoclonus and periodic movements of sleep: a syndrome related to the endogenous opiates? *Adv Neurol* 1986;43:309-19.

283. Bixler EO, Kales A, Vela-Bueno A, Jacoby JA, Scarone S, Soldatos CR. Nocturnal myoclonus and nocturnal myoclonic activity in the normal population. *Res Commun Chem Pathol Pharmacol* 1982;36(1):129-40.

284. Coleman RM, Pollak CP, Weitzman ED. Periodic movements in sleep (nocturnal myoclonus): relation to sleep disorders. *Ann Neurol* 1980;8(4):416-21.

285. Mendelson WB. Are periodic leg movements associated with clinical sleep disturbance? *Sleep* 1996;19(3):219-23.

286. Dickel MJ, Mosko SS. Morbidity cut-offs for sleep apnea and periodic leg movements in predicting subjective complaints in seniors. *Sleep* 1990;13(2):155-66.

287. Edinger JD, McCall WV, Marsh GR, Radtke RA, Erwin CW, Lininger A. Periodic limb movement variability in older DIMS patients across consecutive nights of home monitoring. *Sleep* 1992;15(2):156-61.

288. Mosko SS, Dickel MJ, Paul T, LaTour T, Dhillon S, Ghanim A, et al. Sleep apnea and sleep-related periodic leg movements in community resident seniors. *J Am Geriatr Soc* 1988;36(6):502-8.

289. Wittig R, Zorick F, Piccione P, Sicklesteel J, Roth T. Narcolepsy and disturbed nocturnal sleep. *Clin Electroencephalogr* 1983;14(3):130-4.

290. Bliwise D, Petta D, Seidel W, Dement W. Periodic leg movements during sleep in the elderly. *Arch Gerontol Geriatr* 1985;4(3):273-81.

291. Edinger JD, Marsh GR, McCall WV, Erwin CW, Lininger AW. Sleep variability across consecutive nights of home monitoring in older mixed DIMS patients. *Sleep* 1991;14(1):13-7.

292. Buysse DJ, Reynolds CF, Kupfer DJ, Thorpy MJ, Bixler E, Manfredi R, et al. Clinical diagnoses in 216 insomnia patients using the International Classification of Sleep Disorders (ICSD), DSM-IV and ICD-10 categories: a report from the APA/NIMH DSM-IV Field Trial. *Sleep* 1994;17(7):630-7.

293. Allen RP, Earley CJ. Augmentation of the restless legs syndrome with carbidopa/levodopa. *Sleep* 1996;19(3):205-13.

294. Yamashiro Y, Kryger MH. Acute effect of nasal CPAP on periodic limb movements associated with breathing disorders during sleep. *Sleep* 1994;17(2):172-5.

295. Schenck CH, Milner DM, Hurwitz TD, Bundlie SR, Mahowald MW. A polysomnographic and clinical report on sleep-related injury in 100 adult patients. *Am J Psychiatry* 1989;146(9):1166-73.

296. Aldrich MS, Jahnke B. Diagnostic value of video-EEG polysomnography. *Neurology* 1991;41(7):1060-6.

297. Guilleminault C, Moscovitch A, Leger D. Forensic sleep medicine: nocturnal wandering and violence. *Sleep* 1995;18(9):740-8.

298. Broughton RJ. Sleep disorders: Disorders of arousal? Enuresis, somnambulism, and nightmares occur in confusional states of arousal, not in "dreaming sleep". *Science* 1968;159(819):1070-8.

299. Jacobson A, Kales A, Lehmann D, Zweizig J. Somnambulism: all-night electroencephalographic studies. *Science* 1965;148:975-7.

300. Kales A, Jacobson A, Paulson MJ, Kales JD, Walter RD. Somnambulism: psychophysiological correlates. I. All-night EEG studies. *Arch Gen Psychiatry* 1966;14(6):586-94.

301. Fisher C, Kahn E, Edwards A, Davis DM. A psychophysiological study of nightmares and night terrors. I. Physiological aspects of the stage 4 night terror. *J Nerv Ment Dis* 1973;157(2):75-98.

302. Fisher C, Kahn E, Edwards A, Davis DM. A psychophysiological study of nightmares and night terrors. The suppression of stage 4 night terrors with diazepam. *Arch Gen Psychiatry* 1973;28(2):252-9.

303. Vela A, Dobladez B, Rubio ME, Ramos M, Suengas A, Bujan M, et al. Action of bromazepam on sleep of children with night terrors. I. Sleep organization and heart rate. *Pharmatherapeutica* 1982;3(4):247-58.

304. Halász P, Ujszászi J, Gádoros J. Are microarousals preceded by electroencephalographic slow wave synchronization precursors of confusional awakenings? *Sleep* 1985;8(3):231-8.

305. Crisp AH, Matthews BM, Oakey M, Crutchfield M. Sleepwalking, night terrors, and consciousness. *BMJ* 1990;300(6721):360-2.

306. Kavey NB, Whyte J, Resor SR, Gidro-Frank S. Somnambulism in adults. *Neurology* 1990;40(5):749-52.

307. Popoviciu L, Delast-Popoviciu D, Delast-Popoviciu R, Bagathai I, Bicher G, Buksa C, et al. Parasomnias (non-epileptic nocturnal episodic manifestations) in patients with magnesium deficiency. *Rom J Neurol Psychiatry* 1990;28(1):19-24.

308. Montagna P, Sforza E, Tinuper P, Cirignotta F, Lugaresi E. Paroxysmal arousals during sleep. *Neurology* 1990;40(7):1063-6.

309. Blatt I, Peled R, Gadoth N, Lavie P. The value of sleep recording in evaluating somnambulism in young adults. *Electroencephalogr Clin Neurophysiol* 1991;78(6):407-12.

310. Llorente MD, Currier MB, Norman SE, Mellman TA. Night terrors in adults: phenomenology and relationship to psychopathology. *J Clin Psychiatry* 1992;53(11):392-4.

311. Moldofsky H, Gilbert R, Lue FA, MacLean AW. Sleep-related violence. *Sleep* 1995;18(9):731-9.

312. Schenck CH, Bundlie SR, Ettinger MG, Mahowald MW. Chronic behavioral disorders of human REM sleep: a new category of parasomnia. *Sleep* 1986;9(2):293-308.

313. Sforza E, Zucconi M, Petronelli R, Lugaresi E, Cirignotta F. REM sleep behavioral disorders. *Eur Neurol* 1988;28(5):295-300.

314. Culebras A, Moore JT. Magnetic resonance findings in REM sleep behavior disorder. *Neurology* 1989;39(11):1519-23.

315. Schenck CH, Mahowald MW. Polysomnographic, neurologic, psychiatric, and clinical outcome report on 70 consecutive cases with REM sleep behavior disorder (RBD): sustained clonazepam efficacy in 89.5% of 57 treated patients. *Cleve Clin J Med* 1990;57 Suppl:S9-S23.

316. Tachibana N, Sugita Y, Terashima K, Teshima Y, Shimizu T, Hishikawa Y. Polysomnographic characteristics of healthy elderly subjects with somnambulism-like behaviors. *Biol Psychiatry* 1991;30(1):4-14.

317. Schenck CH, Mahowald MW. Injurious sleep behavior disorders (parasomnias) affecting patients on intensive care units. *Intensive Care Med* 1991;17(4):219-24.

318. Lapierre O, Montplaisir J. Polysomnographic features of REM sleep behavior disorder: development of a scoring method. *Neurology* 1992;42(7):1371-4.

319. Schenck CH, Hurwitz TD, Mahowald MW. Symposium: normal and abnormal REM sleep regulation: REM sleep behaviour disorder: an update on a series of 96 patients and a review of the world literature. *J Sleep Res* 1993;2(4):224-31.

320. Pedley TA, Guilleminault C. Episodic nocturnal wanderings responsive to anticonvulsant drug therapy. *Ann Neurol* 1977;2(1):30-5.

321. Maselli RA, Rosenberg RS, Spire JP. Episodic nocturnal wanderings in non-epileptic young patients. *Sleep* 1988;11(2):156-61.

322. Plazzi G, Tinuper P, Montagna P, Provini F, Lugaresi E. Epileptic nocturnal wanderings. *Sleep* 1995;18(9):749-56.

323. Nousiainen U, Suomalainen T, Mervaala E. Clinical benefits of scalp EEG studies in intractable seizure disorders. *Acta Neurol Scand* 1992;85(3):181-6.

324. Sasaki M, Kurosaki Y, Mori A, Endo S. Patterns of sleep-wakefulness before and after transmeridian flight in commercial airline pilots. *Aviat Space Environ Med* 1986;57(12 Pt 2):B29-B42.

325. Alvarez B, Dahlitz MJ, Vignau J, Parkes JD. The delayed sleep phase syndrome: clinical and investigative findings in 14 subjects. *J Neurol Neurosurg Psychiatry* 1992;55(8):665-70.

326. Rotenberg VS. The competition between SWS and REM sleep as index of maladaption to shift work. *Homeostasis* 1991;33:235-8.

APPENDIX 1: Literature Search Strategies

Guide to Search Syntax (DIALOG®, CINAHL)

!	Explode the search term (i.e., retrieve the search concept plus all narrower terms (DIALOG®)
EXP	Explode the search term (CINAHL)
?	Truncation symbol, single character. Retrieve plural and variant endings.
(w)	Proximity operator. Words must be adjacent.
()	Proximity operator. Words must be adjacent.
(n)	Proximity operator. Words must be near each other in any order.
ab	Search in article abstract.
de	Descriptor i.e., subject heading (a controlled, thesaurus term in DIALOG®)
mjx	Major subject heading (CINAHL)
mnx	Minor subject heading (CINAHL)
ti	Search in titles
pt	Publication type (CINAHL)

DATABASES	LIMITS	SUBJECT HEADINGS/KEYWORDS
DIALOG® MEDLINE® BIOSIS Previews® EMBASE® PASCAL PsycINFO®	1992- Human	(sleep disorders! OR sleep apnea syndromes!)/de [MEDLINE®] OR (sleep disorder OR sleep apnea OR sleep apnea syndrome OR sleep-disordered breathing OR obstructive sleep apnea OR obstructive sleep apnea syndrome OR snoring OR hypersomnia OR sleep deprivation OR bruxism OR insomnia OR restless legs syndrome)/de [BIOSIS®] OR (sleep disorder! OR snoring! OR restless legs syndrome! OR sleep deprivation OR sleep-disordered breathing OR bruxism!)/de [EMBASE®] OR (sleep disorders! OR bruxism OR restless legs syndrome! OR narcolepsy!)/de [PsycINFO®] OR

polysomnography(L)standards/de

OR

(sleep()apnea OR sleep()apnoea OR snoring OR OSA OR OSAS OR SAHS OR apneic()episode? OR hypopneic()episode? OR restless()leg?()syndrome? OR bruxism OR narcolep? OR periodic()limb()movement()disorder? OR somnabulism OR sleepwalking OR sleep()walking OR insomnia OR sleep()terror? OR upper()airway()resistance()syndrome? OR sleep()related()epileps? OR parasomnias OR circadian()rhythm(1N)disorder? OR nocturnal()myoclonus OR REM()behavio?r()disorder? OR sleep()problem? OR day time()sleepiness OR excessive()sleepiness)/ti,ab

OR

(sleep(2N)centre? OR sleep(2N)center? OR sleep(2N)lab? OR centre(2N)sleep()stud? OR sleep(2N)clinic? OR sleep(2N)institute? OR sleep(2N)research?)/ti,ab

AND

(guidelines! OR health planning guidelines OR standards OR consensus development conferences!) /de [MEDLINE®]

OR

(guidelines OR standards OR health planning guidelines OR practice guidelines OR consensus development conferences OR professional standards)/de [BIOSIS®]

OR

(guidelines! OR treatment outcome OR planning guideline)/de [EMBASE®]
OR

(guidelines! OR professional standards!)/de [PsycINFO®]
OR

59

		(DT=Guideline OR DT=Practice Guideline OR DT=Consensus Development Conference OR DT= Consensus Development Conference, NIH) [MEDLINE®] OR (guideline? OR recommend? OR consensus OR standard? OR parameter? OR position()paper? OR position()statement? OR treatment()outcome?)/ti,ab *Performed 15 Oct 2002* *Total hits = 408 records* *Update performed 09 Jan 2004* *Total hits = 239 records*
PubMed Cochrane Library	1992 - present	Same MeSH headings and keywords as the origingal DIALOG® MEDLINE search. Searches performed 10 October 2002; last update June 2004
CINAHL Direct	1992 – present Human	EXP sleep-disorders.mjx.mnx. OR polysomnography.mjx.mnx OR ***AND*** Same keywords as DIALOG® search ***AND*** practice guidelines.mjx.mnx. OR Practice guidelines.pt. OR nursing interventions.pt OR *Same keywords as DIALOG® search will be used.* *Performed 30 Nov 2002* *Total hits = 57 records* *Update performed 14 January 2004* *Total hits = 6 records*
Search of HTA, near-HTA web sites for published, in-progress and planned reports		e.g., INAHTA, NZHTA; AHRQ; University of York NHS Centre for Reviews and Dissemination – CRD databases, etc.

APPENDIX 2: Web Sites of Professional Associations Checked

Professional Association	URL
American Academy of Family Physicians	http://www.aafp.org/
American Academy of Pediatrics	http://www.aap.org/
American Academy of Sleep Medicine	http://www.aasmnet.org
American Association of Respiratory Care	http://www.aarc.org/
American College of Chest Physicians	http://www.chestnet.org/
American Sleep Apnea Association	http://www.sleepapnea.org/
American Thoracic Society	http://www.thoracic.org/
The Association of Polysomnographic Technologists	http://www.aptweb.org/
Australasian Sleep Association	http://www.sleepaus.on.net/
Austrian Sleep Research Association	http://www.schlafmedizin.at/schlaf/english/inhalt/allgemeines.html
Belgian Association for the Study of Sleep	http://www.belsleep.org/_home.htm
CMA Infobase. Canadian Medical Association	http://mdm.ca/cpgsnew/cpgs/index.asp
Canadian Paediatric Society	http://www.cps.ca/
Canadian Sleep Society	http://www.css.to/
Canadian Task Force on Preventive Health Care	http://www.ctfphc.org/
European Respiratory Society	http://www.ersnet.org/ers/
European Sleep Research Society	http://www.esrs.org/
European Society of Sleep Technologists	http://www.esst.org/
Federation of Latin American Sleep Societies	http://www.flass.icb.usp.br/index2.html
Finnish Sleep Research Society	http://www.sus.fi/index-en.html?
French Society of Sleep Research	http://sommeil.univ-lyon1.fr/SFRS/
German Sleep Society	http://www.uni-marburg.de/sleep/dgsm/welcome_.html
Institute for Clinical Systems Improvement	http://www.icsi.org/index.asp
National Center on Sleep Disorders Research - NIH	http://rover.nhlbi.nih.gov/about/ncsdr/
National Guideline Clearinghouse (USA)	http://www.guideline.gov
National Sleep Foundation	http://www.sleepfoundation.org/
New Zealand Guidelines Group	http://www.nzgg.org.nz/index.cfm
Ontario Guidelines Advisory Committee (GAC)	http://gacguidelines.ca/
Ontario Program for Optimal Therapeutics	http://www.opot.org/
Restless Legs Syndrome Foundation	http://www.rls.org/
Scandinavian Sleep Research Society	http://www.ssrs.org/
Scottish Intercollegiate Guidelines Network (SIGN)	http://www.show.scot.nhs.uk/sign/index.html
Sleep Research Society	http://www.sleepresearchsociety.org/site/
Swiss Society of Sleep Research	http://www.swiss-sleep.ch/home.html
The Sleep Apnoea Trust (United Kingdom)	http://www.sleepmatters.org/
World Federation of Sleep Research Societies	http://www.wfsrs.org/homepage.html

APPENDIX 3: Primary Study Review Form

Cited Studies		
Sleep Laboratory Application		
Guideline		
Recommendation		
Study (*give reference*)		
Review Study	Yes:	No: (*give reasons why not*)
Study Objective		
Study Population and Setting		
Study Design and Approach		
Study Quality (*consider briefly*)		
Study Findings		
Relevance of Findings to Guideline Recommendation		
Comments		

APPENDIX 4: Selected Guidelines and Associated Reviews

Title	Organization	Year
Management of obstructive sleep apnoea/ hypopnoea in adults. A national clinical guideline[15]	Scottish Intercollegiate Guidelines Network	2003
Practice parameters for using polysomnography to evaluate insomnia: an update[34]	American Academy of Sleep Medicine	2003
Practice parameters for the use of portable monitoring devices in the investigation of suspected sleep apnea in adults[11,43]	American Thoracic Society/ American College of Chest Physicians and American Academy of Sleep Medicine	2003
Sleep apnoea : Finnish national guidelines for prevention and treatment[21]	Finnish Lung Health Association	2003
Indications for performing polysomnography in the diagnosis and treatment of restless legs syndrome[36]	German Sleep Society	2002
Parasomnias and sleep-related epilepsy[40]	International League Against Epilepsy	2002
Practice parameters for the use of auto-titrating continuous positive airway pressure devices for titrating pressures and treating adult patients with obstructive sleep apnea syndrome[24]	American Academy of Sleep Medicine	2002
Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome[29]	American Academy of Pediatrics	2002
Technical report: diagnosis and management of childhood obstructive sleep apnea syndrome[27]	American Academy of Pediatrics.	2002
Chronic fatigue syndrome. Clinical practice guidelines[41]	Royal Australasian College of Physicians	2002
Practice parameters for the treatment of narcolepsy: an update for 2000[35]	American Academy of Sleep Medicine	2001
Practice parameters for the use of laser-assisted uvulopalatoplasty: an update for 2000[33]	American Academy of Sleep Medicine	2001
Care of patients with obstructive sleep apnea syndrome in Switzerland[17]	Swiss Respiratory Society	2001
Argentine consensus on sleep-related breathing disorders[18]	Argentine Association of Respiratory Medicine	2001
Diagnosing sleep apnea in dental patients[42]	Raphaelson M & Hakim TS	2001
Surveillance of patients treated with continuous positive pressure[45]	Groupe Sommeil de la SPLF (France)	2000
Obstructive sleep apnea, polysomnography and split-night studies: consensus statement[16]	Connecticut Thoracic Society and Connecticut Neurological Society	2000
Practice parameters for the evaluation of chronic insomnia[50]	American Academy of Sleep Medicine	2000
Evaluation of chronic insomnia[51]	American Academy of Sleep Medicine	2000
SIDS und polygraphie[31] (SIDS and polygraphic recordings)	German Association for Sleep Medicine	2000

Title	Organization	Year
Osterreichisches SIDS – consensus – gespräch anlässlich der Wiener SIDS – präventionskampagne 'Sicheres Schlafen'[32] (4th Austrian SIDS – Consensus Meeting and Viennese SIDS prevention campaign "Safe Sleep")	Austrian SIDS consensus meeting	2000
Idiopathic congenital central hypoventilation syndrome. Diagnosis and management[30]	American Thoracic Society	1999
Practice parameters in the treatment of restless legs syndrome and periodic limb movement disorder[37]	American Academy of Sleep Medicine	1999
The treatment of restless legs syndrome and periodic limb movement disorder[38]	American Academy of Sleep Medicine	1999
Indications for positive airway pressure treatment of adult obstructive sleep apnea patients[22]	Loube DI et al. [consensus statement from six institutions]	1999
Practice parameters for the indications for polysomnography and related procedures[23]	American Sleep Disorders Association	1997
The indications for polysomnography and related procedures[39]	American Sleep Disorders Association	1997
Clinical practice parameters and facility standards for sleep medicine[19]	College of Physicians and Surgeons of Ontario	1996
Standards and indications for cardiopulmonary sleep studies in children[28]	American Thoracic Society	1996
Standards for polysomnography in Canada[10]	Canadian Sleep Society/Canadian Thoracic Society	1996
Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances[25]	American Sleep Disorders Association	1995
Practice parameters for the use of polysomnography in the evaluation of insomnia[52]	American Sleep Disorders Association	1995
Diagnosis of obstructive sleep apnea syndrome[20]	Respiratory Insufficiency and Sleep Disorders Group (Spain)	1995
AARC-APT clinical practice guideline Polysomnography[26]	American Association of Respiratory Cardiologists – Association of Polysomnography Technologists	1995
Indications and standards for use of nasal continuous positive airway pressure (CPAP) in sleep apnea syndromes[44]	American Thoracic Society	1994
Practice parameters for the use of portable recording in the assessment of obstructive sleep apnea[13]	American Sleep Disorders Association	1994
Portable recording in the assessment of obstructive sleep apnea[14]	American Sleep Disorders Association	1994

Two publications from the AASM on the use of the MSLT and MWT[53,54] were expected to be published in 2004, but were unavailable for this review. A recent Italian publication on diagnosis of OSA in children [55] was noted during revision of the report, but was excluded in the review.

APPENDIX 5: Guideline and Publications Excluded from Review

Organization or Authors	Publication		Description
Littner M, Kushida CA, Anderson WM, Bailey D, Berry RB, Hirshkowitz M, *et al.*[56]	Practice parameters for the dopaminergic treatment of Restless Legs Syndrome and Periodic Limb Movement Disorder	Sleep 2004; 27(3):557-559	Treatment with dopinergic agonists
Hening WA, Allen RP, Earley CJ, Picchietti DL, Silber MH; Restless Legs Syndrome Task Force of the Standards of Practice Committee of the American Academy of Sleep Medicine[57]	An update on the dopaminergic treatment of restless legs syndrome and periodic limb movement disorder	Sleep 2004; 27(3):560-83	Treatment with dopinergic agonists
Allen RP, Picchietti D, Hening WA, Trenkwalder C, Walters AS, Montplaisir J[58]	Restless legs syndrome: diagnostic criteria, special considerations and epidemiology	Sleep Medicine 2003; 4:101-119	Report from workshop
Rombaux Ph, Bertrand B, Boudewyns A, Deron Ph, Goffart Y, Hassid S, *et al.*[59]	Standard ENT clinical evaluation of the sleep-disordered breathing patient: a consensus report	Acta oto-rhino-laryngologica belg 2002; 56: 127-137	Consensus report and statement on clinical ENT examination
AASM[4]	Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research	Sleep 1999; 22: 667-689	Definitional and technical material
AASM[60]	Practice parameters for the use of light therapy in the treatment of sleep disorders	Sleep 1999; 22(5): 641-660	Treatment approach
ASDA[61]	Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders	Sleep 1995; 18(4): 285-287	Details of adjunctive technique
ASDA[62]	The role of actigraphy in the evaluation of sleep disorders	Sleep 1995; 18(4): 288-302	Details of adjunctive technique
American Electroencephalographic Society[63]	Guideline 15: guidelines for polygraphic assessment of sleep-related disorders (polysomnography)	J Clin Neurophysiol 1994;11:116-124	Technical aspects of PSG

Organization or Authors	Publication		Description
American Electroencephalographic Society[64]	Guideline twelve: guidelines for long-term monitoring for epilepsy	J Clin Neurophysiol 1994; 11:88-110	Unrelated to sleep laboratories
American Electroencephalographic Society[65]	American Electroencephalographic Society guidelines for polygraphic assessment of sleep-related disorders (polysomnography)	J Clin Neurophysiol 1992; 9:88-96	Technical guidelines on measurement
European Sleep Research Society, Committee on hypnotics and sleep physiology[66]	Hypnotics and sleep physiology: a consensus report	Eur Arch Psychiatry Clin Neurosci 1991; 241:13-21	Effects of hypnotics on EEG sleep parameters

APPENDIX 6: Identified Guidelines and Recommendations for Obstructive Sleep Apnea Diagnosis

1. ASDA, 1997[23,39]

Recommendations
- PSG is routinely indicated for the diagnosis of sleep-related breathing disorders.
- For most patients, full-night PSG is recommended for the diagnosis of sleep-related breathing disorders.
- For patients in the high-pretest-probability stratification group, a cardiorespiratory sleep study may be an acceptable alternative to full-night PSG, provided that repeat testing with full-night PSG is permitted for symptomatic patients who have a negative cardiorespiratory sleep study. (By using a cardiorespiratory sleep study to test only those patients who are in the high-pretest-probability group, the clinician will reduce the probability of false-negative studies so that the need for PSG is lessened)

Evidence
The ASDA Review[39] summarizes the characteristics of sleep apnea syndrome, including central sleep apnea and OSA. Areas covered include symptoms and associated risk factors, snoring and observed sleep apneas, excessive daytime sleepiness (EDS), obesity, insomnia and electrocardiography (ECG) changes. Cited literature on these sections was not reviewed for this report, nor was literature on the treatment of OSA considered.

The ASDA review cites 11 studies (four cohort and seven case series) that indicate one night of PSG is usually sufficient to diagnose OSA. For example, when 50 suspected OSA patients in the study of Mendelson et al.[67] were subjected to two consecutive nights of PSG, 46 patients were diagnosed with OSA on the first night and 49 on the second, for apnea-hypopnea index (AHI) ≥ 5; and 42 on the first night and 46 on the second night, for AHI≥ 10. The authors concluded that one night of PSG should generally suffice. A second positive recording might be expected in a small group of patients who have a negative first study.

Six of 11 patients prospectively studied by Meyer et al.[68] who were suspected of having OSA, had a negative PSG result on the first night, but a positive second study with a significant rise of AHI from 3.1 ± 1.0 to 19.8 ± 4.7. Thus, a negative first-night study is insufficient to exclude OSA in patients with one or more clinical markers of the disease.

Split-night studies: In the ASDA review,[39] six references were cited in support of the second recommendation. Three of these evaluated split-night PSG in the determination of effective continous positive airway pressures (CPAP) for OSA patients.[69-71] The other three compared PSG findings for the entire night with those for a portion of the night.

Scharf et al.[72] studied sleep patterns in 40 hypertensive men who were recruited from a general medical clinic. The subjects underwent overnight PSG, in which the AHI and percent of the time during which arterial oxygen saturation (SaO$_2$) was <90% (T90) were evaluated. The first 90 minutes of the overnight study was evaluated separately. Scoring for the first 90 minutes had a low sensitivity for correctly diagnosing OSA (42%) and a specificity of 100%.

A late afternoon nap study was conducted for subjects with an AHI ≥10 with the intention of assessing the efficacy of short studies performed in the afternoon. The sensitivity of these studies was found to be low (60%) and they failed to detect a number of subjects with increased AHI.

While evaluating the severity of OSA in 66 patients with an AHI of 40 to 125 events per hour, Charbonneau et al.,[73] showed that as the night progressed, the mean apnea duration significantly increased from 27.2 s to 34.6 s, mainly from increases during non-rapid eye movement (NREM) sleep. The proportion of time spent in apnea also increased because of increases in mean apnea duration and the proportion of REM sleep. Thus, for a selected population with severe OSA, diagnosis should require full-night PSG. This was a retrospective analysis of PSG records.

However, in a population with a wider range of severity of sleep-disordered breathing, including inappropriate sleepiness, snoring, putatively observed apnea or a combination of these signs and symptoms, Sanders et al.[74] found that partial PSG was as accurate as full-night PSG for the diagnosis of sleep-disordered breathing. Analyses were undertaken of the polysomnograms from 48 of 50 consecutive patients who were referred for evaluation of sleep-disordered breathing. Breathing and oxygenation data collected during the first half of the PSG examination (PSG - ½) were correlated with those collected during full-PSG. At a cut-off frequency of 10 events per hour, the sensitivity, specificity and predictive values of all parameters were high (around 90%) in identifying patients with sleep-disordered breathing during PSG - ½. Similar results were attained with a cut-off frequency of five per hour. The findings appear contradictory to the recommendation.

Because of the time consuming nature and the high cost of full-night PSG, alternative approaches for the diagnosis of OSA have been suggested. These have included the use of sleep questionnaires,[75-77] statistical models,[78-82] home oximetry,[83] portable monitoring devices,[84-89] limited PSG recording[90] and cardiorespiratory recording for CPAP titration.[91]

Use of sleep questionnaires: Dealberto et al.[75] assessed the use of a self-administered sleep questionnaire as a tool for OSA diagnosis in 129 patients with different subcategorized sleep disorders. It was found that snoring and breathing arrest during sleep could be used to predict AHI≥10, taking into account age, gender and body mass index (BMI). Among subjective sleep questionnaire items, however, only daytime sleepiness was related to drops of transcutaneous oxygen tension and this item was not related to AHI≥10. Only limited details on the questionnaire and its use are presented.

Scharf et al.[72] also evaluated the use of self-administered questionnaires and short sleep studies in screening for sleep-disordered breathing. Each subject completed a self-administered questionnaire, which included items related to the frequency of occurrence of disordered sleep. Using a linear regression approach, it was found that no features of the symptom questionnaire strongly predicted AHI and only self-reported snoring strongly predicted T90 (the percentage of sleep time during which the saturation was <90%). The results of the study showed that the self-administered symptom questionnaire was not useful for predicting sleep-disordered breathing in a small cohort unselected for sleep-related symptoms.

A Swedish study in which the use of a self report questionnaire and day time PSG was compared to the nocturnal PSG of 42 habitual snorers gave positive predictive values of about 60% and 20% to 30% false-positives.[76]

Douglas et al.[77] describe the development of a sleep disorders questionnaire and diagnostic scales, using an earlier instrument from Stanford University and data from patients with different catagories of sleep disorder. Responses to the questionnaire were obtained from 519 persons, 435 of whom were clinical sleep disorder patients. It was shown that the sleep apnea scale discriminated between the patient and control groups (sensitivity 81%, specificity 85%). The authors comment on the limitations of the study, including population size for some groups and characteristics of the control group. Also, the questionnaire was too long to be used as a general-population epidemiological screening tool and future work would be based on five to 10 items selected from the full questionnaire, which would be studied for the purpose of general-population screening. The authors caution that the questionnaire scales are not a replacement for a clinical assessment by a trained sleep clinician, plus PSG.

Statistical models: Five other studies used multifactorial analysis developed from clinical data and questionnaire responses to predict disturbance of breathing during sleep in populations of 100 to 410 patients.[78-82] The predictors for OSA included male gender, habitual snoring, neck circumference, hypertension, sleep supine, waking with heartburn and dozing while driving.[78,82]

The approach used by Crocker et al.[80] correctly classified 33 of 36 patients with AHI>15 (sensitivity 92%) and 35 of 69 with AHI≤15 (specificity 51%). Poorer performance was obtained during a subsequent study of 98 patients referred for snoring or daytime sleepiness (sensitivity 79%, specificity 50%).[83]

Deegan and McNicholas[78] reported results for 250 consecutive patients who had clinical assessment, PSG and completed a self-assessment questionnaire. No single factor was a useful predictor of OSA. Using an AHI threshold of 15, about a third of patients could have been correctly classified as having OSA or not, on the basis of clinical features and oximetry data. Other patients would still have required full-PSG to provide a confident diagnosis. Rauscher et al.[79] used a regression model based on questionnaire responses from habitual snorers and patients with OSA, plus overnight pulse oximetry in a study of 116 patients referred for investigation of heavy snoring. All patients with an AHI of <10 could be identified from the regression model, plus oximetry data and the authors suggest that snorers with negative results from oximetry do not need PSG.

Flemons et al.[82] developed a clinical prediction rule based on the examination of 180 patients referred to a sleep disorder centre. Clinical variables associated with the AHI were based on anthropomorphic measures and responses to a questionnaire. Clinical scores of <5 or >15 were associated with a high probability of an absence or presence of an AHI of 10 or 20. The ASDA review notes that the high post-test probability reported in this paper was lower for AHI >20 than for AHI >10. Viner et al.[81] compared predictive models based on subjective clinical impressions or clinical and anthropomorphic features with PSG findings in 410 patients referred for suspected OSA. For patients with a low predicted probability of sleep apnea, the model based on clinical data correctly excludes about 30% of patients without OSA, which could result in a reduction in those sent for PSG.

The ASDA review notes the possibility that low probability patients had upper airways resistance syndrome (UARS) was not addressed in the studies.

Home and portable device studies: Gyulay et al.[83] compared clinical assessment to home oximetry in the diagnosis of OSA in 14 patients, who were referred for assessment of snoring or daytime sleepiness. They showed that analysis of oximetry data by counting desaturations alone gave sensitivity not superior to that of clinical assessment. Clinical assessment identified patients with AHI ≥15 with a sensitivity of 79% and specificity of 50%. The likelihood of AHI ≥15 using the regression equation of Crocker et al.[80] (68% sensitivity and 55% specificity) was not superior to the global clinical impression of the physicians.

Five out of six studies assessing the use of portable sleep apnea monitoring devices in a laboratory have shown that data from the devices and those of PSG were highly correlated (correlation coefficients for apnea index (AI) or AHI >0.90).[84-87,89]

Sensitivity and specificity varied from study to study. High sensitivity and specificity (95% and 96%) were found by Emsellem et al.[85] in 67 patients referred with a tentative diagnosis of OSA. They suggested that each patient identified as having sleep apnea, using a portable system, should be completely studied in a sleep laboratory. They also noted that a negative result with a portable device will exclude only sleep apnea and further assessment would be needed for other types of sleep-related complaints.

Orr et al. found a sensitivity of 93% and specificity of 89% for a respiratory disturbance index (RDI) value of 15.[86] Man and Kang[84] reported lower sensitivity (85.7%), but a specificity of 94.7% at a cut-off value of AHI ≥15 and suggested that the monitoring device would be useful in identifying patients without significant sleep apnea. White et al.[89] reported high sensitivity, but only moderate specificity (90.7% and 70.4%) at an AHI threshold of 10. There was poor correlation (r=0.33) between the two systems when the portable monitor was used at home.[89] Redline et al.[87] considered the study findings in the context of use in epidemiologic studies of general populations.

In a small study of 14 subjects with sleep apnea,[88] the correlation coefficient for RDI was 0.70 with a significant difference in findings (p<0.01) between the portable monitor and PSG. Sensitivity in detecting apneic episodes was 78% using the manufacturer's criteria in 12 subjects.

The portable systems are designed for the detection of sleep apnea only, particularly in high probability groups and are not intend to replace PSG in the diagnosis of a wide spectrum of sleep disorders. A negative result with a portable screening device only rules out sleep apnea. Thus, a full PSG to investigate other causes of sleepiness should still be considered for patients with a negative portable monitor study result who have significant symptoms of excessive daytime sleepiness.

Cardiorespiratory recording: Douglas et al.[90] prospectively examined the value of electrophysiological and respiratory monitoring in 200 consecutive adults who had been referred to a sleep laboratory and who were considered to need further study. PSG showed that 15 patients had OSA and 11 had PLMD. OS-A could be accurately diagnosed by recording only the

breathing pattern (AHI) and time in bed. Recording sleep electrophysiologically was of no diagnostic value. Of those with OSA, 66% could be diagnosed with oximetry alone, but many of the patients not diagnosed by oximetry had moderately severe OSA and benefited from treatment.

Relevance to recommendations

Two of three small observational studies and one retrospective study indicated that full-night PSG is desirable for OSA diagnosis. One of these, however, reported partial-night findings for only the first 90 minutes of sleep. The third study had contrary findings and suggested PSG during the first two hours of sleep is a reliable basis for diagnosis.

Approaches using questionnaires to screen patients for OSA appear to have some potential to rule out further investigations in a sleep laboratory, for those with a low probability of OSA, though the robustness and applicability in routine practice would need consideration. From the data reported for the cited studies, even with the more elaborate approaches, significant proportions of persons with OSA would be incorrectly excluded from referral for PSG.

Several studies provided indications of the potential usefulness of portable monitors, though the predictive value of these varied and their scope of application should be kept in perspective.

2. Scottish Intercollegiate Guidelines Network, 2003[15]

Recommendations
- Limited sleep studies to assess respiratory events are an adequate first-line method of diagnostic assessment for OSAHS.
- Individual sleep centres should examine the balance of benefits associated with using a specific sleep study against their resources, geographical catchment, equipment available and the diagnostic algorithm used.
- Full PSG with EEG-based sleep staging is unnecessary to diagnose sleep apnea in most patients. It should be available in regional sleep centres for patients who have typical symptoms of excessive daytime sleepiness, but no objective evidence of obstructive sleep apnea on limited testing.
- Oximetry studies cannot exclude OSAHS. Studies using oximetry alone may have a role in the initial assessment of OSAHS, but their limitations must be fully appreciated before using them to make diagnostic and therapeutic decisions.
- The technology used to make the diagnosis is less important than the level of experience and training available to interpret the result.

(The first statement is presented in the guideline as a recommendation and the others as good practice points, which are recommended best practice based on the clinical experience of the guideline development group).

Evidence

The guideline cited the 1997 ASDA review,[39] Bradley *et al.*[92] and a narrative review[93] to support the observations that although PSG is accepted in North America as the gold standard for the diagnosis of sleep apnea, it has never been independently validated. Observational studies indicate

that PSG may be useful in the diagnosis of sleep apnea, although there is night-to-night variation in reproducibility; and different centres use different thresholds in the diagnosis of OSA.

The Douglas et al.[90] study is used to support the statement that the clinical value of performing PSGs on all patients with daytime sleepiness has been questioned. No reference is made to the diagnosis of PLMD in that study or the proportions of patients diagnosed by PSG alone.

Limited sleep studies, using a reduced combination of the range of variables in full PSG, are introduced in the guideline with reference to three studies.[92,94,95]

Whittle et al.[94] compared outcomes for 149 patients with suspected OSA who had a home study as an initial investigation with 75 individuals who were investigated with PSG. The home study included airflow recording, ECG, measurement of chest wall movement and pulse oximetry.

On the basis of findings from a previous validation study, home studies showing >30 apneas plus hypopneas per hour were regarded as diagnostic of OSA and the subjects proceeded to treatment (usually CPAP). Those with values of <30 apneas plus hypopneas per hour who did not have daytime sleepiness were not investigated further. The remaining subjects, including those whose home study was unsuccessful, were then given full PSG.

Of the 149 subjects, 29% were diagnosed with OSA and were offered treatment. Twenty-seven (18%) had an unsuccessful home study and proceeded to PSG. Of the 80 with <30 apneas plus hypopneas per hour, 18 had low Epworth Sleepiness Scale (ESS) scores or refused further investigations. Of the 58 individuals who proceeded to PSG, 18 with severe OSA on PSG (>30) had home studies showing <30 and four of those <15. The authors indicate that their results suggest home studies alone cannot exclude a diagnosis of OSA in those with daytime sleepiness.

Under the local financial and administrative arrangements, use of the home study protocol resulted in faster diagnosis for the patients and lower costs for the provider than an approach based on PSG.

The two earlier studies by the same group[92,95] were case series in which PSG recordings were made in parallel with a limited sleep system (Autoset). All apneas were scored by both systems, but 41% more hypopneas on PSG, which were clinically significant.

The guideline refers to the Bennett et al. study,[96] which measured the improvement in symptoms after CPAP and used a regression analysis to identify the best predictors of improvement. PSG was said not to have been the best predictor, though it appears investigators reported only those variables in PSG [body movement and electroencephalographic (EEG) sleep depth] that contributed to the model. The guideline also refers to a study by Engleman et al.[97] in suggesting PSG derivatives such as AHI or EEG arousals were no better at predicting improvement than the simpler indices. That study, however, appears to have involved only home monitoring in a comparison of treatments.

Three studies are cited in which there was comparison of hospital-based partial channel PSG and full PSG.[98-100] Two of these are from the same group[98,100] and compare partially attended night-time time respiratory recording (NTRR) and full PSG in patients with suspected OSA using a randomized crossover (RCT-C) design (76 and 36 individuals). Sensitivity and specificity of NTRR versus PSG were 82% and 90%. Mean values for AHI using NTRR were lower than those obtained with full PSG (22.7 ± 2.4 versus 32.2 ± 3.0 events per hour), because of the under-recognition of hypopneas.

The third study[99] compared respiratory polygraphy with previously obtained PSG findings in 101 patients. The criteria for diagnosis of OSA with polygraphy were that AHI and the desaturation index were both ≥ 10. When data for both indices are considered, overall sensitivity was 90% and specificity 85%.

The guideline draws attention to the possible decrease in diagnostic certainty as a disadvantage of limited home studies, which do not allow assessment of sleep presence, quality or duration, or the diagnosis of conditions other than OSA. The guideline cites the systematic review published by AHCPR[101] in noting that reported sensitivity and specificity ranges are 32% to 100% compared to full PSG and are dependent on the equipment and definitions of events used.

The guideline cites contrasting results on the issue of whether patients diagnosed with OSA in limited sleep studies have poorer CPAP use afterwards. A study from France by Krieger et al.[102] in which there was follow-up for two years after the prescription of CPAP, found patients who were diagnosed with an ambulatory procedure had higher drop-out rates than those in two groups diagnosed with PSG (21.7% versus 10.0% and 6.25%; $p<0.05$). They also had lower rates of use of their CPAP. In the Scottish study by Whittle et al.,[94] however, there was no difference between the home study diagnosis group and controls in the proportions offered CPAP, continuing to use CPAP or in nightly use of CPAP (follow-up four months to 20 months after diagnosis).

The statement on oximetry is supported by reference to the study by Douglas et al.[90], the AHCPR review [101] and other publications that report high false-negative rates and diverging conclusions on the usefulness of this method.

The guideline states that sleep questionnaires may be useful in the initial assessment of OSA, but not for diagnosis. The statement is supported by three studies[103-105] showing a mean sensitivity and specificity of 42% and 68% respectively, when comparing questionnaire sampling to full PSG.

The guideline draws attention to the importance of well trained staff at sleep centres, but no evidence is cited in support.

Relevance to recommendations

The guideline recommends use of limited sleep studies as a first-line approach to the diagnosis of OSA. It is unclear whether home-based or laboratory-based studies are preferred, but the information provided in the cited studies from Scotland might suggest the former. The strongest evidence comes from a controlled trial, which included the useful feature of long-term follow-up data for patients who had been prescribed CPAP after a diagnosis of OSA. Five other controlled trials and four case series are also considered, some of which relate to earlier studies by the same group. Overall, the studies indicate the potential for use of limited sleep studies, leading to judgements on the trade-off between poorer diagnostic performance than PSG and appropriate use of limited resources. The statement that full PSG is unnecessary to diagnose sleep apnea in most patients seems to be poorly supported, given the need for follow-up PSG in a large proportion of those who receive limited sleep studies.

3. Connecticut Thoracic Society, 2000[16]

Recommendations
- Split-night studies and full-night studies are not equivalent. Full-night diagnostic studies provide a more complete assessment of sleep-disordered breathing and are more likely to benefit from the possibility and relevance of a second diagnosis.
- Full-night PSG followed by a full-night of CPAP titration is the accepted gold standard and is the recommended method of evaluation and treatment of sleep-disordered breathing.

Evidence

The recommendations are supported by reference to the 1997 ASDA publications,[23,39] a short commentary[106] and six studies.[69-71,73,107,108]

Four studies showed clinical features of sleep apnea varied during night-time sleep.[71,73,107,108] That by Charbonneau et al.[73] has already been considered on page 68 in review of the evidence cited in the 1997 ASDA publication. A small study by Findley et al.[108] also found significant differences in apnea duration and the frequency of arterial oxyhemoglobin saturation (SaO_2) during REM and NREM sleep of 12 OSA patients (AHI >5) and 12 normal subjects. Apneas were longer during REM sleep than NREM sleep for all patients.

Fanfulla et al.[107] studied 29 consecutive OSA patients and evaluated the first part (PSG_1) and second part (PSG_2) of a standard PSG examination for the ability to diagnose OSA. PSG_1 had low sensitivity value (66%). It was representative of full PSG and similar to PSG_2 only in those patients with REM phase sleep in the first part of the night. They concluded that split-night studies were inappropriate for evaluating sleep-disordered breathing in OSA patients when REM phase sleep does not occur in the first part of the night.

Studies with larger populations have concluded that split-night studies may be appropriate for a specific population of OSA patients.[69-71] The commentary by Jamieson[106] suggests that split-night studies done with the dual objective of diagnosis and therapy represent a compromise in the standard of care and often fall short of accomplishing either goal.

Relevance to recommendations
The additional information cited refers to two small studies that provide further evidence against the use of split-night studies for the diagnosis of OSA.

American Thoracic Society/American College of Chest Physicians/AASM, 2003, 2004[11,43]
Recommendations
- Type 2 portable monitors (comprehensive portable PSG, both attended and unattended setting) are not recommended for clinical use to evaluate patients with sleep apnea.
- Type 3 portable monitors (modified portable sleep apnea testing in attended setting) appear capable of being used to decrease or increase the probability that the patient has an AHI >15.
- Type 3 portable monitors (modified portable sleep apnea testing in unattended setting) are not recommended for use to decrease the probability that the patient has an AHI <15 or to increase the probability that the patient has an AHI >15.
- Type 4 portable monitors (continuous single or dual bioparameter recording in attended setting) with oximetry and at least one other airflow parameter are not recommended for routine use to increase, decrease or both increase and decrease the probability that a patient has an AHI >15.
- Type 4 portable monitors (continuous single or dual bioparameter recording in unattended setting) with oximetry and at least one other airflow parameter are not recommended for use in the diagnosis of sleep apnea or confirming that a patient has an AHI >15 or AHI <15.

Evidence
The recommendations from the practice parameters for portable monitoring devices (PMs) are based on the findings of a comprehensive systematic review by Flemons *et al.*[12] The following details are taken from summaries given in that publication.

The review covered studies published in English of patients ≥18 years with any diagnosis of OSA, with a PM used for diagnosis and PSG or other acceptable objective test used for the diagnosis of sleep apnea. After completion of the study, each analysis group was ≥10 subjects. In assessment of the studies, evidence levels used were defined as:

- level I: blinded comparison, consecutive patients, reference standard performed on all patients
- level II: blinded comparison, non-consecutive patients, reference standard performed on all patients
- level III: blinded comparison, consecutive patients, reference standard not performed on all patients
- level IV: reference standard was not applied blindly or independently.

In addition, seven other aspects of the study's methods were scored and a quality rating (from a to d) was assigned based on the number of indicators for which the study met the criteria.

A total of 51 studies were selected for review and a non-quantitative synthesis performed.

Type 2 PMs: There were four studies describing five groups of patients published about Type 2 monitors (comprehensive PSG). Three were rated as having level IV evidence and one study was rated as having level II evidence. The summary to the practice parameters document[43]

comments that Type 2 PMs did not have adequate available data to recommend their use based on the small number of published studies, absence of sensitivity or specificity data and the low level of evidence.

Type 3 PMs: There were 12 published studies, describing 14 groups of patients. Of the nine undertaken in sleep laboratories, three were level II, five level III and one level IV. Of the assessments of use at home, two were level II and two level IV.

Twelve studies reported 13 comparisons between PM and PSG. Eight of nine attended monitor studies were of higher evidence level and quality rating (three studies had level I evidence and a quality rating of a) and all had a low likelihood ratio (< 0.2). Seven of the eight studies had a low percentage of false-negative results. In contrast, two of the four unattended studies had a higher level of evidence and higher quality rating (both had level II evidence and a quality rating of a or b). Both studies had low likelihood ratios, but a relatively high percentage of false-negative results.

In a concluding synopsis, the review states that Type 3 monitors have utility to both reduce and to increase the probability that a patient may have sleep apnea in the attended setting. The utility in the unattended setting is not as well-established.

The summary to the practice parameters document[43] comments that Type 3 PMs may be acceptable in an attended in-laboratory setting, both to rule in and rule out OSA. Such use would require several limitations. These include application to a sleep clinic population without significant comorbidities, tendency for Type 3 devices to underestimate PSG-defined AHI, a need for patients with a negative or non-diagnostic Type 3 study to undergo a definitive evaluation to determine the cause of symptoms, a need for a subsequent PSG if CPAP titration is required and consideration that Type 3 PMs are not recommended for split-night studies. Use in an unattended setting is not recommended.

Type 4 PMs: There were 35 studies using Type 4 monitors on 38 populations. Of 29 reports of patients studied in the sleep laboratory, 17 had level I or II evidence and 12 were level IV. Four of nine studies in the home setting gave level I or II evidence and five were level IV. Synopses of findings of the review were as follows:
- Oximetry alone can reduce and can increase the probability of sleep apnea both in an attended and unattended setting. In the latter situation, however, the results should be considered preliminary. The addition of a second signal showed results similar to those using oximetry alone and similar conclusions can be drawn. Nasal pressure may be useful in an attended setting, but no conclusions can be made about its use in an unattended setting. (Based on results of seven comparisons that combined oximetry with at least one other parameter, five in an attended setting; 19 comparisons evaluating the use of oximetry alone as a reference, 13 of which were attended; and seven studies of nasal pressure).
- Oximetry alone can increase the probability of sleep apnea both in an attended and unattended setting. In the latter situation, however, the utility appears to be less compared with the attended setting. The addition of a second signal showed results similar to those using oximetry alone. The evidence is lacking, however, to suggest this type of signal combination can be used in an unattended setting. The results for nasal pressure in the

attended setting should be considered preliminary, but suggest the utility of this approach is still in question. No conclusions can be made about its use in an unattended setting (based on the results of six comparisons using oximetry and a second parameter, eight studies of oximetry alone in the attended setting and three studies on nasal pressure, all attended).

- The utility of using oximetry alone to both increase and reduce the probability of sleep apnea is not well established in the attended or unattended setting, with or without an additional channel. Results should be considered preliminary. The utility of nasal pressure as a signal for increasing and decreasing the probability of sleep apnea has not been established in the attended or unattended setting [based on results of four studies with oximetry and at least one other sensor, all in the attended setting, 10 studies using oximetry alone (nine attended, one unattended home study) and two studies of nasal pressure, both in the attended setting].

Relevance to recommendations

The guideline gives a perspective on the place of PMs in sleep studies, based on an analysis of recent literature. Judgements on the applicability of PMs follow available data on their sensitivity and specificity and on the reliability or in some cases, the absence of evidence. The recommendations indicate an emerging role for Type 3 PMs in the attended sleep laboratory setting, subject to several limitations. Use of Type 2 and Type 4 monitors, and of all types of PMs in unattended settings, is not supported.

4. Argentine Association of Respiratory Medicine[18]

Recommendations

- PSG is the reference method for the diagnosis of respiratory sleep disorders.
- Daytime studies are indicated for patients with inverted rest or wakefulness times or if night-time studies are impossible. A negative daytime study in a patient with clinical suspicion will require a night-time study.
- Simplified (level III) studies are reserved for situations where there are long waiting lists, access to PSG is impossible or for follow-up of previously diagnosed patients to evaluate response to treatment or for re-evaluation if symptoms recur.
- Pulse oximetry is not recommended.
- There is no consensus on the use of split-night studies.

Evidence

No references are cited in regard to the recommendations on PSG and on the use of daytime studies.

Of seven studies cited regarding OSA diagnosis using portable devices (Type III), five[84,85,87-89] were included in the ASDA guideline, 1997.[23,39] Salmi *et al.*[109] assessed sleep-related apneas using an automated device to record respiratory and body movements, oxygen saturation (SaO_2) and airflow in 55 patients with clinically suspected OSA. Data from automatic analysis were compared with those obtained during simultaneous daytime PSG during naps. The automated device had high sensitivity in that the periodic breathing pattern was found in all patients with apnea index (AI) >5 and had high sensitivity in detecting periodic movement. The AI was comparable between manual and automated analysis. In patients with AI >5, the mean SaO_2 was 92.4±2.2% lower than in patients without apneas. The duration of apneas was slightly shorter

than in manual analysis, but was sufficient for screening purposes. The difference between the manual and automated methods was clinically significant in three patients. The findings of the study may have limited validity as full PSG was not used as a control.

A digital recording device (MESAM 4) was used in the Stoohs et al.[110] study to screen 56 patients for OSA by monitoring oxygen saturation, heart rate, snoring and body position. The patients were referred with complaints of disrupted sleep or EDS, including narcolepsy, isolated daytime sleepiness, OSA and PLMD. All patients were subjected simultaneously to nocturnal PSG and MESAM 4 recordings. PSG identified 26 patients with OSA, while MESAM 4 identified 25 subjects with OSA using the oxygen algorithm; all had a respiratory disturbance index (RDI) $\geq$10 with PSG. The sensitivity and specificity for SaO_2 measured by MESAM 4 as compared with PSG were 97% and 92% respectively. Heart rate and snoring were less accurate. The authors that suggest the device can be helpful to clinicians and epidemiologists as a low-cost screening device for subjects with OSA and habitual snoring.

The guideline also considers the use of Type IV studies (continuous recording of one or two variables) where more comprehensive diagnostic approaches are unavailable. The guideline cites a publication by Levy et al.[111] in stating that the sensitivity and specificity of night-time pulse oximetry in the diagnosis of OSA varied in different studies from 40% to 100% and from 39% to 100% respectively. The main reason for this wide variation has been the method used in analyzing the pulsed oxygen saturation (SpO_2) level. Studies with low sensitivity and high specificity used a decrease of >3%[83,111-115] as an abnormality criterion, while studies with high sensitivity and low specificity did not take the magnitude of the decrease into consideration.[83,111,114,116,117] The guideline indicates that the use of pulse oximetry should be reserved for situations where a more appropriate technology is unavailable. Operator expertise is also important.

No specific citations are made in respect to recommendations on split-night studies; studies showing variation in apnea during the night are the reasons given for the lack of consensus. The guideline cites a study by Martin et al.[118] indicating that the observation of >80 apneas-hypopneas during the first two hours of diagnostic PSG is an indication to proceed with a split-night study.

Relevance to recommendations
Much of the evidence cited in this guideline relates to lower level studies that may need to be considered in situations where more detailed investigations are unavailable. The cited studies were of limited quality because of a lack of appropriate controls or small populations. The potential variability of different portable device models makes it difficult to reach firm conclusions.

5. ASDA, 1994[13]

Recommendations

Standard PSG is the accepted test for the diagnosis and assessment of OSA. Unattended portable recording for assessment of OSA is acceptable only:

- for patients with severe clinical symptoms indicative of OSA when the initiation of treatment is urgent and PSG is unavailable
- for patients unable to be studied in a sleep laboratory
- for follow-up studies after diagnosis with PSG and therapy initiation.

Evidence

The Cohen-Mansfield *et al.*[119] study was intended to validate sleep observations as tools in the assessment of sleep in the nursing home. Night-shift staff were trained to perform sleep observations on 20 elderly nursing-home residents. The observational sleep assessment instrument (OSAI) documented the occurrence of sleep, disruptions in sleep, breathing, snoring, myoclonic movements and body restlessness. The results of the OSAI were compared with those of portable sleep monitors (four-channel sleep respiratory monitor and wrist activity monitor). It was found that detection of sleep patterns using the OSAI method yields high sensitivity and specificity. Although it was concluded that the OSAI is reliable and valid for examining sleep and sleep pathology in this elderly population, the study population was small and there was a lack of inter-rater assessment. The external validity of this instrument remains to be tested on a larger population.

In a similar fashion, Lord *et al.*[120] examined the inter-rater reliability of a portable monitoring system, scoring records of breathing during sleep of 26 elderly subjects. The raters were a medical student, a nurse practitioner and a family physician. The unweighted kappa statistic was used to measure agreement among raters. Agreement was found to be better for variables describing breathing (kappa 0.71 to 0.87) than for those describing sleep (kappa 0.34 to 0.57). Complete agreement among the three raters occurred in 17 cases.

Wrist actigraphy was used to record sleep patterns in 19 elderly nursing-home residents during a 24-hour period.[121] On average, the data showed subjects woke up three or four times per hour and some woke up as many as 11 to 12 times per hour. Men awoke more often than women between 12:00 a.m. and 1:00 a.m. Sleep fragmentation might be associated with compensation for lost sleep, increased total time in bed, wakening of social constraints and deterioration of the circadian sleep-wake rhythm. The findings were limited to a small and distinct population and had limited relevance to the recommendation, as the portable system was not used to diagnose or assess OSA.

The Rasche *et al.*[122] study evaluated an alternative system to PSG (the MESAM-system) in the diagnosis of rhonchopathy in 94 patients with a history of snoring. OSA was diagnosed in 19 patients and confirmed in 10 of those individuals who were re-examined in a sleep laboratory.

Two studies used PMs to determine the prevalence of sleep-disordered breathing in the elderly population, to examine whether sleep apnea is a predictor of mortality and to determine if gender has any effect on survival rate.[123,124] Using the PM with a questionnaire, Ancoli-Israel *et al.*[123]

found that 24% of 427 elderly volunteers had AI ≥5. Women showed significantly less sleep-disordered breathing than men (20% of women versus 28% of men). For hypopnea among 384 people, 81%, 62% and 44% had RDI ≥5, RDI ≥10 and RDI ≥20 respectively. A previous study from the same group of investigators showed that 43% of 233 nursing-home residents had AI ≥5 and 70% had RDI ≥5.

Lord *et al.*[125] concluded that portable monitoring systems could be used to monitor and classify sleep breathing disorders in the elderly population, although night-to-night variability of certain subjects may result in diagnostic misclassification. Thirty retirees having a variety of health problems, but none having clinical features to suggest OSA, underwent two pairs of consecutive night studies, separated by four to six months. Night-to-night agreement appeared better for measures of RDI and oxygenation than for estimates of sleep quality. Twenty-eight subjects had at least two nights of satisfactory recordings. The variation in RDI in some subjects had little overall effect on their classification into normal and abnormal groups, using a cut-off RDI value of 15. The study, however, is prone to selection and detection bias for its small and distinct population of elderly subjects with mild disturbance of breathing during sleep.

Reference is also made to reviews by Richards[126] on the advantages and disadvantages of sleep measurement techniques in critical care and by Ancoli-Israel[127] on the advantages and disadvantages of the ambulatory cassette recording of sleep apnea.

Relevance to recommendations
The cited studies provide support for the use of portable systems to assess sleep-related breathing disorders in specific cases such as in critical care and care of elderly patients. The studies have limitations and may be prone to selection and detection bias because of small and specific study populations and subjective assessments.

6.   Raphaelson and Hakim, 2001[42]

Recommendation
* Snoring patients with poor periodontal health or who are edentulous should be referred for sleep medicine evaluation. If there is moderate or severe subjective sleepiness, consider referral for sleep medicine consultation and PSG.

Evidence
The Raphaelson and Hakim[42] review covered mostly the background of clinical features of OSA, PSG, split-night studies, oximetry and portable studies. One study was cited to support its recommendations.[128] Four published prediction formulas were evaluated on 370 patients with sleep-disordered breathing who were referred to a sleep laboratory for PSG. PSG analysis showed that of 370 patients, 248 (67%) had an AHI≥10 and 180 (49%) had an AHI≥20. Analysis of the models for predicting the presence of OSA indicated that for AHI≥10, sensitivity ranged from 76% to 96% and specificity 13% to 54%. For AHI≥20, sensitivity ranged from 33% to 39% and specificity 87% to 93%. All models performed better for men. It was concluded that the clinical prediction models tested are insufficiently accurate to discriminate between patients with or without OSA, but could be useful in prioritizing patients for split-night PSG.

Relevance to recommendations
There was no appropriate cited evidence related to the use of PSG in snoring patients with poor periodontal health. The cited references were either background information or generally applied to OSA patients.

7. Swiss Respiratory Society, 2001[17]

Recommendations
- PSG is indicated to evaluate patients with a recurrence of symptoms during treatment with CPAP, oral appliances or after upper airway surgery.
- PSG is not indicated for routine follow-up if patients report the sustained optimal treatment effects of CPAP.
- The mainstay in the assessment of patients with suspected OSA is a careful history, including standardized questionnaires.

Evidence
The main support for the recommendations of this guideline is the ASDA's[23,39] 1997 publications.

The Bloch *et al.*[129] study was cited for the use of questionnaires to assess suspected OSA patients. The Epworth sleepiness scale (ESS), a questionnaire for the assessment of subjective daytime sleepiness, was translated and validated for use in German-speaking countries. Patients with sleep disorders (n=174) and normal subjects (n=159) received the ESS by mail and brought the filled-out questionnaire to their first appointment. A complete medical and sleep-related history was taken and a physical examination was performed. If the suspicion of a sleep-related breathing disorder was high, a level IV study was performed. If the level IV study was non-diagnostic, a full PSG was performed. If the likelihood of a sleep-related breathing disorder was low or another sleep disorder suspected, then a PSG was performed first.

The results showed that the mean ESS score of normal subjects (5.7±3.0) was significantly lower than that of the patients (13.0±5.1). Among patients, there was no significant correlation between gender or age and the ESS score. In patients with primary snoring and sleep apnea, the ESS score correlated significantly, but weakly, with both the RDI (r=0.26) and the percentage of time spent at an oxygen saturation <90% (r=0.35). Patients with severe OSA had significantly higher ESS scores than patients with primary snoring or with central sleep apnea.

Relevance to recommendations
The findings of this controlled study were relevant to the recommendation that standardized questionnaires such as the ESS can be used to assess suspected OSA, particularly to identify severe OSA from primary snoring and central sleep apnea.

Other guidelines
The following guideline documents did not cite evidence in support of their recommendations, other than the referral to other guidelines or statements.

8. Canadian Sleep Society and Canadian Thoracic Society, 1996[10]

Recommendations
- Indications [for PSG] can be considered under four headings of disorders, which may occur in isolation or in combination: insomnia; excessive sleepiness; behavioural or arousal disorders (parasomnias); and circadian sleep disorders.
- A patient with symptoms related to disrupted sleep or waking can be referred for PSG by any physician.
- The diagnosis of OSA and the prescription of nasal CPAP may be adequately performed during an afternoon sleep study. However, an afternoon study should be performed only in cases of obvious severe apnea or if the waiting list is excessively long. For all other cases, overnight PSG is required.

9. College of Physicians and Surgeons of Ontario (CPSO), 1996[19]

Recommendation
Full overnight sleep study is the standard for those individuals in whom OSA is expected, particularly if CPAP or surgical therapy is considered.

10. Sociedad Española de Neumología y Cirugía Torácica, 1995[20]
[Respiratory Insufficiency and Sleep Disorders Group (Spain)]

Recommendations
- Definitive diagnosis of OSA should be made in a setting where full PSG can be undertaken.
- In centres without such facilities, OSA diagnosis should be regarded as provisional, pending confirmation by a reference centre.
- Diagnosis of OSA by oximetry is not recommended.

11. Loube *et al.* 1999[22]

Recommendations
- PSG is indicated for the diagnosis of possible OSA; a six-hour minimum duration is preferred for a full-night study and at least a two-hour duration for a split-night study.
- Standard diagnostic PSG may be performed in a health care facility or in the patient's home if a trained technologist is in attendance. Unattended PSG is not validated.
- Patients with a respiratory disturbance index of >40 events per hour during the first two hours of a diagnostic PSG receive a split-night study.
- Limited channel PSG may be indicated for patients with high pre-test probability of OSA, based on validated screening algorithms.

12. Finnish national guidelines for prevention and treatment 2002 to 2012[21]

Recommendations
If a patient has typical indications of sleep apnea from basic evaluations, the diagnosis is primarily made on the basis of sleep registration. Limited sleep registration versus extensive

sleep registration indication depends on symptoms and findings (extensive would correspond to full-PSG):

- highly probable sleep apnea on the basis of symptoms and findings (limited)
- snoring patients lacking other symptoms suggesting sleep apnea (limited)
- probable sleep apnea on the basis of symptoms and findings (limited)
- possibly other than sleep apnea, atypical symptoms and findings (extensive)
- results from extensive sleep registration are unclear or no treatment results are reached despite good treatment compliance (extensive).

For patients under two years of age, extensive sleep registration is always recommended.

13. AARC–APT, 1995[26]

Recommendations
PSG may be indicated for patients:

- with excessive daytime sleepiness or insomnia
- with snoring associated with observed apneas or excessive daytime sleepiness
- with other symptoms of sleep-disordered breathing as described in the International Classification of Sleep Disorders.

Other recommendations from this guideline are considered in section 5.3.4.

APPENDIX 7: Identified Guidelines and Recommendations for Obstructive Sleep Apnea Titration

Guideline

1. AASM, 2002[24]

Recommendations
- Patients with congestive heart failure, COPD or nocturnal arterial oxyhemoglobin desaturation due to conditions other than OSA are not candidates for APAP titration.
- Patients who do not snore should not be titrated with an APAP device that relies on vibration or sound in the device's algorithm.
- APAP devices are not recommended for split-night titration.
- Certain APAP devices may be used during attended titration to identify, by PSG, one pressure for use with standard CPAP for the treatment of OSA.
- The use of unattended APAP to either initially determine pressures for fixed CPAP or for self-adjusting APAP treatment in CPAP-naïve patients is not established.
- Treatment of OSA or the use of APAP must be based on a prior diagnosis of OSA.

Evidence
This guideline and a supporting review on the use of APAP for titration and treatment were considered in a 2003 assessment by CCOHTA.[6] Conclusions in the CCOHTA assessment were consistent with points made in the guideline. There is some evidence, primarily from small studies [one RCT, three randomized crossover trials (RCT-C) and six non-randomized comparisons], supporting the efficacy of APAP in attended titration in a sleep laboratory. Its use in unattended titration is not established. The CCOHTA report noted that in most studies, patients suffering from cardiac, pulmonary and other medical conditions were excluded. There are potential safety issues with the use of APAP for such individuals if the technology is used in particular settings, without prompt access to technical support. A separate review of literature cited to support the recommendations in the AASM guideline was not undertaken for this report.

2. ASDA, 1997[23,39]

Recommendations
- PSG is indicated for CPAP titration in patients with sleep-related breathing disorders.
- A full night of PSG with CPAP titration is recommended for patients with a documented diagnosis of sleep-related breathing disorder for whom CPAP is warranted.
- PSG with CPAP titration is appropriate for patients with AI >20 per hour or AHI >30 per hour, regardless of symptoms; AHI >10 hour in patients with excessive daytime sleepiness; a respiratory arousal index of >10 per hour in those with excessive daytime sleepiness.
- A cardiorespiratory sleep study without EEG recording is not recommended for CPAP titration. CPAP titration should include the ability to perform sleep staging and to identify and treat arousals.

For CPAP titration, a split-night study (initial diagnostic PSG followed by titration during PSG on the same night) is an alternative to one full night of diagnostic PSG followed by a second night of titration if four criteria are met:

a) an AHI of ≥40, documented during a minimum of two hours of diagnostic PSG; or AHI of 20 to 40 based on clinical judgment
b) CPAP titration is carried out for >3 hours (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
d) a second full-night PSG for CPAP titration is performed if the diagnosis of a sleep-related breathing disorder is confirmed, but criteria b and c are not met.

Evidence

Use of PSG for titration: The position taken in the guideline on PSG and titration follows in part from discussion and material on the diagnosis of OSA given in the accompanying review.[39]

The overview in the review from ASDA[39] includes reference to a book chapter on clinical features and evaluation of obstructive sleep apnea.[130] The chapter is a narrative review with 49 references, bringing together studies and commentaries from the mid-1960s to 1990. Clinical symptoms in OSA and clinical evaluations of patients with OSA including cardiovascular changes are presented. PSG is regarded as an objective evaluation of OSA syndrome and standard PSG recordings are detailed.

The clinical features of OSA are presented in a second cited book chapter.[131] A rationale for why PSG must be done if OSA is suspected clinically is included. PSG is used to confirm the diagnosis, to ascertain the severity of the physiological disturbances, which will then act as a guide to further therapy and to evaluate the response to nasal CPAP. Monitoring should include all sleep stages. Ideally, PSG should include an entire night to indicate the patient's usual sleep and a second night to evaluate the response to CPAP therapy. Patients with upper airway resistance syndrome may require the measurement of pleural pressure.

Cardiorespiratory sleep studies: The ASDA guideline mentions that residual hypopneas and upper airways resistance syndrome (UARS) with arousals may require additional titration to determine optimal therapeutic pressures and that these additional adjustments require EEG recording.

In a prospective study of 41 subjects with moderate to severe OSA, who had CPAP titration performed both in a sleep laboratory using full PSG and in the respiratory ward using night-time respiratory recordings only, it was found that night-time respiratory recording allowed a reasonable choice of CPAP pressure levels to abolish all the night-time respiratory disturbances.[91] Respiratory equipment was used, which continuously recorded and displayed arterial oxygen saturation (SaO_2), airflow, chest and abdominal motion and body position. UARS, however, was not addressed in this study.

The requirement for electroencephalography (EEG) in cardiorespiratory sleep study before CPAP titration is supported by two studies[132,133] showing the number of transient alpha EEG arousals, usually >10 arousals per hour of sleep, is one of the main characteristics in the identification of UARS. These patients are middle-aged, relatively slim, snorers and nonapneic. Also, EEG may provide an indication of disturbed sleep with corresponding sleep fragmentation that is not easily recognizable by visual scoring.

Guilleminault et al.[132] found that 15 of 48 patients who had reported excessive daytime sleepiness had frequent (≥10 per hour) transient alpha EEG arousals. The mean transient alpha EEG arousal was 31.3±12.4 arousals per hour of sleep. The arousals were directly related to an abnormal increase in respiratory efforts during sleep. Therapeutic trials using nasal CPAP showed no change in rapid eye movement (REM) sleep, a significant increase in the percentaage of stages 3 and 4 NREM sleep, elimination of daytime sleepiness (mean sleep latency changed from 5.1±1.0 min to 13.5±2.1 min) and a decrease of mean alpha EEG arousal index to 8±2 per hour of sleep.

A small study by the same group[133] investigated the relationship between short EEG arousals during nocturnal sleep, the esophageal pressure (P_{es}) nadir and airflow decrease in 15 men who were heavy snorers. Monitoring indicated some individuals in this group of snorers may present significant increases in P_{es} nadir, with an abrupt decrease in the flow, leading to EEG arousals. CPAP treatment decreased the mean respiratory disturbance index (RDI) (2.66±1.45 versus 0.53±0.51), eliminated short EEG arousal index (16.38±11.87 versus 0.91±0.88) improved MSLT scores (10.77±2.47 versus 14.59±1.20) and lowered SaO_2 (90.7±2.46 versus 93.8±0.86). Thus, the score of transient alpha EEG arousals and monitoring of esophageal pressure (indicator of respiratory effort) would help detect this particular breathing disorder. A further study by this group that found 38 of 334 women (11%) had UARS.[134]

Split-night studies: Three studies are cited in the accompanying review[39] in support of the recommendation on split-night studies.

Iber et al.[69] found that effective CPAP could be documented on single-night studies in 320 (78%) of 412 OSA patients with an apnea index (AI) ≥20 who underwent trials between 1984 and 1989. Baseline PSG was performed in the first part of the night before beginning titration, which included time with the patient in REM sleep in the supine position. For those in whom the single-night studies were successful, there was a 99% reduction in frequency of obstructive events and improvement in the lowest oxygen saturation to 94±5%. For 10% of this series of patients, there was inadequate sleep time to permit titration and 12% did not tolerate CPAP on the first night.

Sanders et al.[71] noted the definitions of effective CPAP and effective pressure used by Iber et al. would have permitted persistent mild to moderate sleep-disordered breathing and no confirmatory data were presented in that study on the adequacy of CPAP levels and of patient acceptance on subsequent nights.

Sanders et al. examined 50 consecutive patients diagnosed with non-body position-dependent OSA. Abbreviated diagnostic PSG was followed by CPAP titration. The patients then returned to the sleep laboratory on a subsequent night for titration for a full night. It was found that of 50

patients, 31 were satisfactorily treated with CPAP during partial and full PSG without a change in the interface. However, 45% of these patients required an alteration in pressure. Average CPAP pressure while receiving full PSG was significantly higher than during partial PSG, though the authors note the clinical significance of the difference is arguable. All AI-related variables were lower after full-night PSG than after partial PSG, though the differences were not SS. Fifteen patients changed the interface and eight patients changed modality, seven from CPAP to bilevel positive airway pressure (BiPAP), during the trial. The ASDA[39] review noted that there were some errors in measurement of outcomes in this study.

A similar study design was used by Yamashiro and Kryger.[70] With 107 patients newly diagnosed with OSA, a split-night protocol was found to be sufficient to determine effective CPAP. For patients with an AHI<20 (n=69), CPAP pressure was SS lower using the split-night protocol. For patients with higher apnea-hypopnea index (AHI) values (n=38), the pressure following split-night study was also lower than that with full-night titration, but the difference was not SS.

The Charbonneau et al.[73] study showed that respiratory events can worsen as the night progresses, providing support for the recommendation that CPAP titration should be carried out for >3 hours in a split-night study.

3. Connecticut Thoracic Society, 2000[16]

Recommendations
- PSG is routinely indicated for the diagnosis of sleep-related breathing disorders. Split-night studies and full-night studies are not equivalent. Full-night PSG followed by a full night of CPAP titration is the accepted gold standard and recommended for most patients.

Evidence
The two studies on split-night protocols that were cited[70,71] have been discussed in the review of the ASDA 1997 guideline.

This guideline reaches more conservative conclusions than those of the earlier ASDA document, in part from consideration of some of the same evidence.

4. American Thoracic Society, 1994[44]

Recommendations
- Titration of CPAP should include recordings of sleep, respiration and oxygenation. The nasal pressure should be raised to a level that eliminates apneas, hypopneas, desaturation and sleep fragmentation. Various body positions and NREM and REM sleep need to be recorded to determine optimum pressure. If oxyhemoglobin desaturation persists, supplemental oxygen or ventilatory assistance can be administered through a nose mask.

Evidence
Four prospective studies[135-138] were cited in support of the recommendation. In the Smith et al.[135] study, the pressure relationships were examined in six men with moderate or severe OSA who had significant hypersomnolence, loud snoring and demonstrated OSA in all-night PSG recording.

These patients underwent all-night PSG recordings followed by separate sleep studies to examine the pressure-flow relationship of the upper airway during non-rapid eye movement (NREM) sleep. Pressure-flow relationships were monitored under two conditions: with a tight-fitting nasal mask and with a face mask after completion of the nasal CPAP flow curves. It was proposed that a major defect in patients with OSA is an elevation of critical pressure surrounding the upper airway rather than changes in the resistive properties of the upper airway. Thus, thorough recordings of sleep studies should be undertaken to determine the upper airway pressure-flow relationships in OSA.

Issa and Sullivan[138] used the upper airway closing pressures to examine the influence of sleep state, posture and level of airway pressure on the stability of the upper airway in patients with OSA. Eighteen patients with moderate to severe OSA, selected on the basis of all-night PSG, underwent a five-day in-hospital treatment program with nasal CPAP, followed by long-term home treatment. It was found that the upper airway closing pressure was lowered (i.e., airway was more collapsible) in stage I-II NREM and REM sleep, compared with that during stage III-IV NREM. In all three sleep stages, the upper airway closing pressure was lower in the supine position than in the lateral position. In stage I-II NREM and REM sleep, the elevation of nasal CPAP did not influence the upper airway closing pressure. In stage III-IV NREM sleep, however, the upper airway became progressively more stable as CPAP was elevated. Low levels of CPAP abolished OSA in all patients.

In a later study by the same group,[136] nasal CPAP applied to 12 patients with moderate to severe OSA, abolished OSA symptoms in all individuals, who were studied in a sleep laboratory for four consecutive nights. Stage I-II NREM sleep was significantly decreased and stage III-IV NREM sleep was increased on the first treatment night. The REM sleep and REM density were also increased. Sleep stages were still significantly different to the control on the third treatment night.

Liistro et al.[137] measured respiratory characteristics during sleep in five nonapneic, heavy snorers and in five OSA patients. The data showed that the pattern of snoring, hysteresis and temporal relationship between supraglottic pressure and flow rate were different in nonapneic and OSA patients. There appears to be limited relevance to the recommendation.

Different measurement techniques of cardiopulmonary sleep studies were described in a guideline prepared by the American Thoracic Society.[139] The guideline had limited discussion on CPAP titration, although it suggested that in patients with severe and unambiguous OSA, initiation of treatment with nasal CPAP may be incorporated into the diagnostic study night.

5. Loube DL et al. 1999[22]

Recommendations
- CPAP titration on night subsequent to diagnostic PSG. Specific parameters to be monitored include EEG, EOG, EMG, oronasal airflow, chest wall effort, body position, snore microphone, ECG, and oxyhemoglobin saturation.
- Some APAP systems are effective in determining the optimal CPAP setting for most OSA patients.

Evidence
In this consensus statement, the indications for CPAP titration, including minimum parameters to be monitored and analyzed, were mostly based on material in the publications prepared by ASDA[23] and AASM[24]. An additional cited reference was a Sanders and Stiller[140] book chapter reviewing the application of positive airway pressure in the treatment of sleep-related breathing disorders. Its emphasis was on the authors' results and experiences with their patients.

Relevance to recommendations
On the basis of earlier studies on the diagnosis and treatment of OSA, first-night PSG diagnosis followed by a second night for CPAP titration using PSG, remains the gold standard.

The findings from small non-controlled studies suggest that monitors in sleep recordings, including upper airway pressure, sleep staging and body positions (supine versus lateral), are essential to obtain optimal CPAP titration. These studies, however, might be prone to selection and detection bias.

Two small case series support the need for the inclusion of EEG recording in cardiorespiratory sleep study used for titration.

Four papers on split-night studies, all on moderate to large case series, support the recommendation that effective CPAP titration can be obtained from partial PSG, if certain criteria are met. Definitional or procedural limitations were noted for two of these studies.

The evidence from several small studies using randomized crossover designs supports a role for APAP in titration in a sleep laboratory. The absence of studies in CPAP-naïve patients, the need for attendant interventions in some patients and the exclusion of certain categories of patients from the studies were reasons for not supporting the use of APAP for unattended titration.

APPENDIX 8: Identified Guidelines and Recommendations for Obstructive Sleep Apnea Follow-up

1. ASDA, 1997[23,39]

Recommendations

Follow-up PSG or a cardiorespiratory study is routinely indicated for the assessment of treatment results:

1. after a good clinical response to oral appliance treatment in patients with moderate-to-severe OSA, to ensure therapeutic benefit
2. after the surgical treatment of patients with moderate to severe OSA, to ensure satisfactory response
3. after the surgical treatment of patients with sleep apnea whose symptoms return despite a good initial response; a lack of response to CPAP may require a repeat PSG evaluation
4. after substantial weight loss has occurred in patients on CPAP for the treatment of sleep-related breathing disorders, to ascertain whether CPAP is still needed at the previously titrated pressure
5. after substantial weight gain has occurred in patients previously treated with CPAP successfully, who are again symptomatic despite the continued use of CPAP, to ascertain whether pressure adjustments are needed
6. when clinical response is insufficient or when symptoms return despite a good initial response to CPAP treatment.

Follow-up PSG or cardiorespiratory study is not routinely indicated in patients whose symptoms continue to be resolved with CPAP treatment.

A MSLT is not routinely indicated for most patients with sleep-related breathing disorders.

Evidence

Cited evidence for recommendation 1 is the 1995 ASDA guideline,[25] which in turn refers to a Schmidt-Nowara *et al.* review.[141] The review concluded that snoring and OSA are consistently improved in most patients who used oral appliances. Limited follow-up data indicated that oral discomfort caused by oral appliances is a common, but tolerable side effect. Dental and mandibular complications appear to be uncommon. The long-term risk of these complications and other adverse effects on breathing are poorly defined. Also, data on long-term compliance were limited and all were based on patients' reports, which may overestimate actual use. The 1995 ASDA guideline specifies follow-up for persons with moderate to severe OSA in terms of office visits to the referring clinician and the dentist. There is no cited reference to the use of PSG.

Recommendations 2 and 3 are supported by a 1996 ASDA guideline,[142] which states, "Once the surgical site has adequately healed, patients with pre-operatively symptomatic or moderate-to-severe sleep apnea should undergo a follow-up evaluation to assess the presence of residual disease. This evaluation includes an objective measure of the presence and severity of obstructive sleep apnea and of the sleep disruption."

Recommendations 4 and 5 follow from observations in the review supporting the guideline[39] that efficacy of weight reduction as a treatment for OSA in obese patients is variable. Four studies[143-146] reported the long-term follow-up of patients after weight reduction. In the first, Noseda *et al.*[143] followed-up 39 individuals after one year of CPAP use and attempted weight loss through counselling (n=36) or gastroplasty. There was a significant improvement in breathing during sleep and in sleep fragmentation, with a correlation between a drop in apnea hypopnea index (AHI) and the reduction in body mass index (BMI). Four patients were successfully weaned from CPAP, three of whom had a substantial decrease in weight.

The other three studies followed morbidly obese individuals who had undergone weight reduction surgery. In two studies,[144,145] in the short term, weight loss after surgery was associated with a decrease in AHI. The regression analysis indicated that 19% of variance in the AHI was accounted for by body weight loss, pre-operative AHI and time between surgery and the time study. Longer-term follow-up at about seven years in six individuals suggested that subsequent weight gain might be associated with increased AHI. Pillar *et al.*[146] found a poor correlation between BMI and AHI in 14 individuals followed to 7.5 years and concluded that morbid obesity was not the only causative factor of OSA for these patients.

The recommendations appear to follow from the perception that weight variation may be linked to sleep disorders in some individuals and that changes in weight may imply current treatment with CPAP is no longer effective. There is no direct evidence supporting either recommendation, nor is any evidence cited specifically in support of recommendation 6.

No evidence is cited in support of the recommendation that follow-up PSG or cardiorespiratory study is not routinely indicated in patients whose symptoms continue to be resolved with CPAP treatment. This recommendation appears to be contrary to what is said in recommendation 1.

The recommendation regarding the use of the MSLT is not supported by a reference to the literature. Reference is made to information presented elsewhere in the guideline and to a 1992 ASDA guideline[147] on the use of this test in relation to obtaining an objective measurement of daytime sleepiness where this is required.

2. AASM, 2002[24]

Recommendation
- Patients being treated with fixed CPAP on the basis of APAP titration or being treated with APAP must be followed to determine treatment effectiveness and safety.

Evidence
The guideline states that this recommendation is based on committee consensus and previous recommendations given in the 1997 guideline.[23]

Methods for following patients may include questionnaires for sleepiness and continued snoring, follow-up PSG or cardiorespiratory studies, assessment of physical conditions such as an increase in weight, and capturing information stored on the automatic positive airway pressure (APAP) or CPAP devices.

3. ASDA, 1995[25]

Recommendations
- Follow-up PSG is not indicated for those with primary snoring or mild OSA unless symptoms worsen.
- Patients with moderate to severe OSA should undergo PSG or another objective measure of respiration with oral appliance in place after final adjustments for fit.

Evidence
No evidence is cited in support of the first recommendation.

The Schmidt-Nowara *et al.*[141] review was cited to support the second recommendation. All reports reviewed showed improvement in the average AHI with oral appliances. The degree of improvement varied, however, as some patients did not improve or became worse. Up to 40% of patients with an initial AHI>20 remained above that level with treatment. In addition, no available treatment for OSA provides the ideal combination of a high rate of success and patients' acceptance without complications. These observations might form the basis for the guideline to recommend that follow-up PSG be given to patients with moderate to severe OSA during treatment, to ensure satisfactory therapeutic benefit.

4. Swiss Respiratory Society, 2001[17]

Recommendations
- PSG is indicated to evaluate patients with recurrence of symptoms during treatment with CPAP, oral appliances or after upper airway surgery.
- PSG or respiratory polygraphy is not indicated for routine follow-up if patients report sustained optimal treatment effects of CPAP.

Evidence
Reference is made to the 1997 ASDA guideline; no other evidence is cited.

5. Group Sommeil de la SPLF, 2000[45]

Recommendation
- The use of polygraphy or PSG for verifying the efficacy of treatment pressure should be reserved for particular clinical situations.

Evidence
No supporting evidence is cited in the document.

6. American Thoracic Society, 1994[44]

Recommendation
- A lack of clinical response to CPAP may require a repeat CPAP evaluation by PSG to verify proper equipment function and to exclude alternative concomitant sleep disorders.

Evidence
No evidence is cited for this recommendation. Recommendations regarding clinical follow-up in this consensus statement are supported by reference to studies on the longer term effects of CPAP, pattern of CPAP use and effect of weight loss.[148-151]

7. CPSO, 1996[19]

Recommendations
- Overall, most patients require a single-night diagnostic study and after treatment intervention, a single-night follow-up study.
- Follow-up PSG may be required if sleep-disordered breathing, periodic limb movement or parasomnia disorder are identified at the time of the initial study and treatment is undertaken. A follow-up may be indicated if there is a significant change in the patient's original symptoms.
- Follow-up PSG and MSLT are generally required to evaluate the objective response of idiopathic hypersomnia to therapy. Follow-up studies may be needed if symptoms change.
- Follow-up studies such as overnight PSG and MSLT or MWT are indicated to evaluate the objective response of the daytime sleepiness (narcolepsy) to treatment or if symptoms deteriorate. They are also indicated when there is suspicion that another sleep disorder such as PLMD or OSA may coexist and cause daytime fatigue and hypersomnolence.
- A repeat sleep study may be indicated to objectively evaluate the response of PLMD to treatment particularly in a patient in whom symptoms persist or in whom symptoms deteriorate.
- There are no indications for follow-up sleep studies to evaluate the response of snoring to treatment. A follow-up sleep study may be indicated in patients with simple snoring if their clinical condition changes (e.g., weight gain, onset of day time sleepiness)
- Follow-up PSG is not indicated for patients with either primary snoring or mild OSA, unless symptoms worsen or do not resolve.

Evidence
The recommendations are based on consensus.

Relevance to recommendations
Several recommendations may reflect the expected standards of prudent clinical practice. However, only limited evidence from studies of generally poor quality and that are of partial relevance, are cited in support. Recommendations made in the 1997 ASDA guideline regarding the follow-up of patients who have a good clinical response appear to be contradictory.

APPENDIX 9: Identified Guidelines and Recommendations for Regulatory Disturbances Complicated by Cardiorespiratory Factors

1. AARC-APT, 1995[26]

Recommendations

PSG may be indicated in patients with:

- COPD whose awake PaO_2 is >55 torr, but whose illness is complicated by pulmonary hypertension, right heart failure, polycythemia or excessive daytime sleepiness
- restrictive ventilatory impairment, secondary to chest wall and neuromuscular disturbances, whose illness is complicated by chronic hypoventilation, polycythemia, pulmonary hypertension, disturbed sleep, morning headaches or daytime sleepiness and fatigue
- disturbances in respiratory control with awake $PaCO_2$>45 torr or whose illness is complicated by pulmonary hypertension, polycythemia, disturbed sleep, morning headaches or daytime sleepiness and fatigue
- nocturnal cyclic bradyarrhythmia or tachyarrythmias; nocturnal abnormalities of atrioventricular ectopy that appear to increase in frequency during sleep.

Risk-benefit ratios should be assessed if medically unstable inpatients are to be transferred from the clinical setting to a sleep laboratory for overnight PSG.

Evidence

Publications cited in support of the recommendations in this guideline included the 1989 American Thoracic Society consensus statement,[139] the 1994 ASDA standards of practice,[14] three reviews[152-154] and a retrospective study.[46]

Strohl and Chester[153] reviewed the techniques of PSG available to monitor cardiopulmonary variables in adult human subjects during sleep. The review also discussed the classification of breathing disorders in sleep, based on clinical history and PSG data.

A Strohl et al.[152] review considered the clinical presentation and causes of sleep apnea. The treatments for sleep apnea that were evaluated included tracheostomy, positive pressure, nocturnal oxygen therapy, surgical procedures on the upper airway, medical treatment to increase upper airway size and medical devices to treat central apnea.

Sullivan et al.[154] reviewed the treatment of cardiorespiratory disturbances during sleep and the pathophysiologic mechanisms of sleep apnea, with reference to the authors' studies. The review includes anecdotal comments on the occurrence of nocturnal oxyhemoglobin desaturation (NOD) in patients with COPD and the use of nocturnal oxygen therapy.

The retrospective study[46] compared survival in COPD patients with or without NOD. Data on 169 individuals from five centres were used. Actuarial survival (Kaplan-Meier) was significantly longer in patients who did not have NOD (five-year survival 69% versus 52%). In patients with

NOD, there was a trend towards shorter survival in those who did not receive nocturnal oxygen treatment, but the difference was not SS. Data selection by the five centres for submission is a potential source of bias.

Relevance to recommendations
The retrospective study gives an indication of the significance of NOD as a risk factor, with the implication that this condition should be identified using sleep studies. The reviews essentially provide background information, including suggestions on some practicalities of PSG testing.

APPENDIX 10: Identified Guidelines and Recommendations for Other Respiratory Disorders

1. ASDA, 1997[23,39]

Recommendations
- PSG is routinely indicated for patients with neuromuscular disorders and sleep-related symptoms to evaluate symptoms inadequately diagnosed by sleep history, assessment of sleep hygiene and review of sleep diaries.
- PSG is not indicated to diagnose chronic lung disease.
- Nocturnal hypoxemia in patients with chronic obstructive, restrictive or reactive lung disease is usually adequately evaluated by oximetry and does not require PSG.
- If a patient's symptoms suggest a diagnosis of OSA or PLMD, then indications for PSG are the same as for those disorders in patients without chronic lung disease.
- Sleep disturbance in patients with asthma should be attributed to inadequately controlled asthma, unless more specific indications for other sleep-related breathing disorders exist.

Evidence
Myasthenia gravis: Quera-Salva et al.[155] assessed the presence of breathing disorders during sleep and determined whether a correlation existed between daytime pulmonary function and any sleep-related breathing disorder in 20 patients with myasthenia gravis. All patients underwent daytime pulmonary function tests and PSG recordings during sleep and had evidence of daytime diaphragmatic weakness. In older patients, moderately increased BMI, abnormal total lung capacity and abnormal blood gas concentrations were indicators of oxygen desaturation of <90% during sleep. These clear indicators were not found in all patients who had sleep-disordered breathing. Of the 20 patients, 11 had sleep disordered breathing [respiratory disturbance index (RDI) >5]; one of these had OSA, the remainder had central apneas-hypopneas.

Post-polio syndrome: Steljes et al.[156] determined whether sleep studies are helpful in detecting patients with remote poliomyelitis whose respiratory insufficiency was undetected or undertreated. Of 13 patients, five (group 1) patients had respiratory assistance from rocking beds at night; and eight (group 2) had no respiratory assistance. PSG was performed on all patients. Significant oxygen desaturations occurred in all patients in group 1; nasal continuous positive airway pressure (CPAP) was ineffective in this group, but four of the five showed dramatic improvement in sleep quality and gas exchange when placed on a mechanical ventilator. Five of eight patients in group 2 had sleep apnea and hypopnea with poor sleep quality. CPAP was helpful in three of the patients and nasal mask ventilation in one. The authors suggest that sleep studies should be performed on post-polio patients who have excessive daytime sleepiness and respiratory complaints.

Cystic fibrosis: Muller et al.[157] determined when the largest decreases in SaO_2 occurred in a group of non-obese, normal young adults and in patients with cystic fibrosis. The study consisted of 20 patients with cystic fibrosis and five controls. The largest decrease in oxygen saturation occurred during REM sleep in normal subjects (mean difference 2±0.31%) and patients (mean difference 7.4±1.3%). During the transition from NREM to REM, there was a decrease in tonic

intercostal and diaphragmatic tonic muscle activity. In general, there was less tonic activity throughout REM sleep than NREM sleep. There appears to be no direct implication from these findings for the use of sleep laboratory investigations. (Cystic fibrosis is also considered in section 5.3.7.)

Amyotrophic lateral sclerosis (ALS): Gay et al.[158] determined the relationship of pulmonary funcion test abnormalities with quality of sleep and survival in 21 patients with ALS. Thirteen patients survived until the end of the 18-month study. Breathing abnormalities and nocturnal hypoventilation occurred in all patients. Maximal inspiratory pressure was 86% sensitive for predicting the presence of minimal nocturnal oxygen saturation of ≤80% and 100% sensitive for predicting 18-month survival. Sleep was less altered in patients with ALS than was expected. These investigators put in place a screening process for patients with ALS using a sleep questionnaire, spirometry, determination of maximal respiratory pressure and outpatient overnight oximetry. Those patients with a substantial reduction in oxygen saturation should undergo a determination of arterial blood gases, after which PSG may be performed.

Kyphoscoliosis: Guilleminault et al.[159] conducted sleep studies on five patients with severe kyphoscoliosis, four of whom had a history of daytime sleepiness or disrupted sleep. The patients underwent all-night PSG and monitoring of respiration and oxygen saturation. In all subjects, the lowest oxygen saturation occurred during REM sleep, which was associated with an increase in the mean duration of apneic events. There may be limited significance in the results of this small series for sleep laboratory use.

Neuromuscular disease: There were two studies on neuromuscular disease, one on adult patients[160] and one on children.[161] A commentary[162] was also cited discussing the use of non-invasive ventilation in patients with muscular disorders.

Bye et al.[160] examined the arterial oxygenation and the breathing pattern during sleep in 20 patients with neuromuscular disorders. Such patients may maintain oxygenation during NREM sleep, but develop oxygen desaturation during REM sleep. Eleven of the 13 patients with vital capacity <55% predicted and eight of the 11 with a maximum inspiratory mouth pressure <30% predicted had hypercapnia (arterial carbon dioxide pressure $PaCO_2$ of 6 kPa). There was a direct relation between minimum arterial oxygen saturation (SaO_2) during REM sleep and vital capacity and daytime arterial oxygen pressure (PaO_2), $PaCO_2$ and percentage fall in vital capacity, from the erect to supine position. Diaphragm weakness was a likely determinant of the fall in SaO_2 during REM sleep when intercostal and accessory muscle activity was inhibited. The authors recommend serial measurements of vital capacity and daytime blood gas tensions for the initial evaluation of patients with neuromuscular disorders and respiratory muscle weakness. As there are limitations in the predictive value of these methods, a sleep study with the measurement of SaO_2 and transcutaneous carbon dioxide tension is necessary to assess the extent of nocturnal respiratory failure.

Khan et al.[161] used PSG to confirm nocturnal hypoxemia due to hypoventilation in eight ambulant children with congenital muscle disorders. PSG showed that the presence of nocturnal hypoxemia with SaO_2 was on average <90% for 49% of sleep and 80% for 19% of sleep accompanied by severe hypoventilation. Sleep was well preserved despite profound hypoxemia.

Nasal ventilation was effective in correcting sleep hypoxemia. (Neuromuscular disease in children is also discussed in section 5.3.7.)

Interstitial lung disease: McNicholas et al.[163] determined whether oxygen desaturation during sleep is a common feature in seven non-snoring patients with severe interstitial lung disease and whether supplemental nocturnal oxygen treatment is needed. All patients had episodes of oxygen desaturation (mean awake SaO_2 was 92.9±0.3% versus mean SaO_2 during sleep 83.2±2.1%). These episodes were transient, however, and mean SaO_2 showed a slight fall between wakefulness and sleep (NREM, 91.5% and REM, 90.4%). The study suggests that nocturnal oxygen treatment need not be considered in patients with interstitial lung disease, unless the level of oxygenation while they are awake indicates the need for such treatment.

Perez-Padilla et al.[164] compared sleep study results for 11 patients with interstitial lung disease and those of 11 controls. They found that SaO_2 was lower in patients with interstitial lung disease during wakefulness and sleep. The mean SaO_2 dropped in REM sleep in the patients, but not in the control subjects. Minimum SaO_2 was also lower in patients (80.0±4.0%) than in the controls (89.5±1.4%). The maximal oxygen desaturation during sleep was inversely correlated with awake SaO_2. The incidence of apnea and hypopnea in patients was low (AHI of 1.3±0.45 versus 2.9±0.82 in control subjects).

Bye et al.[165] showed that 13 patients with interstitial lung disease often developed falls in SaO_2 during REM sleep. The occurrence of hypoxemia in sleep was not confined to patients with predominant airways obstruction, but also occurred in patients with interstitial lung disease who had restrictive ventilatory impairment. The authors suggested that a sleep study should be considered as part of the overall assessment in managing patients with severe interstitial lung disease, even if awake blood gas tensions are normal. Nocturnal oxygen therapy might be a valuable form of treatment in such patients.

Duchenne muscular dystrophy: Two small studies on Duchenne muscular dystrophy were cited. One investigated overnight oxygenation in 10 patients using home oximetry in comparison with PSG for four individuals.[166] The other[167] study examined overnight PSG in six patients using a crossover design with patients breathing air or oxygen on consecutive nights. Both studies found that hypoventilation and associated oxygen desaturation occurred in REM sleep in these patients. In the study by Smith et al.,[167] however, none of the patients desaturated while breathing oxygen, suggesting an approach to alleviating episodic nocturnal hypoventilation.

Carroll et al.[166] suggested that home oximetry is an adequate method comparable to PSG to investigate sleep-related hypoxemia in Duchenne muscular dystrophy. The small number of subjects in this study suggests the need for further work.

Myotonic dystrophy: The results of both studies cited for myotonic dystrophy[168,169] showed that daytime sleepiness in myotonic dystrophy is usually caused by the dysfunction of central sleep regulation (central apneas) and not by disturbed nocturnal breathing. The sleep pattern showed a normal ratio between REM and NREM sleep. From these studies, the indication of PSG for these patients is inconclusive.

COPD: Twelve studies on COPD were cited.[170-181] It appears that EEG recording in association with oximetry is sufficient to monitor patients with COPD, as the falls of SaO_2 were more common during periods of REM sleep, which is a vulnerable period in subjects with COPD for prolonged nonapneic oxygen desaturation. Although short apneic episodes might occur in a few instances during REM sleep, they did not appear to cause a reduction in SaO_2. A group of OSA patients with associated COPD had been identified through SaO_2 that was lower than that for the remainder, while PSG data (i.e., AI, AHI and time spent in apnea) could not differentiate.[172] The reported efficacy of oxygen therapy given to COPD patients differed between studies. Details of the studies appear in Table 1.

Table 1: Studies on COPD

Studies	Population	Findings	Comments
Calverly[170]	20 patients with chronic bronchitis and emphysema (13 of whom had low arterial PO_2 and elevated PCO_2 "blue and bloated," 7 of whom had relatively normal PO_2 and PCO_2 "pink and puffing"); 9 control subjects	Both groups of patients differed significantly from controls in that they had lower stable level of oxygen saturation during sleep, transient hypoxemic episodes and a greater fall in SaO_2 during these episodes; transient falls in SaO_2 were more common during periods of REM sleep than during NREM sleep in all subjects; when breathing oxygen, all patients showed an improvement in level of nocturnal oxygen saturation and sleep pattern	Same patient group as Catterall *et al.*[171]
Catterall[171]	20 patients with severe chronic bronchitis and emphysema (13 of whom had hypoxemia and hypercapnia, 7 normal arterial gas tension); 20 control subjects	NSD between groups in total duration of irregular breathing or number of apneic episodes; hypoxic episodes (SaO_2 falls of >10%) occurred more frequently during sleep in all subjects of hypoxemic and hypercapnic group	
Chaouat[172]	265 patients (22 female, 243 male, mean age 54±10 years) with OSA, having AHI >20 events/h	30 of 265 patients (11%) identified as having COPD (obstructive spirographic pattern defined by FEV_1/VC ratio≤60%); PaO_2 was lower, $PaCO_2$ was higher and pulmonary arterial pressure was higher in the overlap group; hypoxemia (PaO_2 ≤65 mm Hg) and hypercapnia ($PaCO_2$ ≥45 mm Hg) were observed in 17 and 8 of 30 overlap patients respectively; nocturnal SaO_2	Direct relevance to routine sleep laboratory investigations unclear.

Studies	Population	Findings	Comments
		was lower in the overlap patients. >10% of patients with OSA have associated COPD; risk of respiratory insufficiency and cor pulmonale higher in these patients	
Coccagna[173]	12 male patients with COPD; 8 controls	Sleep parameters of both patient and control groups were similar. In patients with COPD, alveolar hypoventilation worsened progressively through stages of slow-wave sleep and was aggravated during REM sleep. The same phenomenon was seen for pulmonary arterial pressure. Maximum increase of $PaCO_2$ value and maximum decrease in PaO_2 value during sleep were greater on COPD than in control group.	Establishing physiological association, direct relevance to routine sleep laboratory investigations unclear
Cormick[174]	50 patients with chronic bronchitis or emphysema; 40 control patients (30 men, 10 women, mean age 63 ± 8 years) without symptomatic lung disease interviewed during attendance at clinic; sleep studies on subgroup of 16 COPD patients	COPD patients reported more difficulty in getting to sleep, staying asleep and more daytime sleepiness than controls; sleep studies of 16 patients showed 6 had hypercapnia ($PaCO_2$ >45 mm Hg) and 7 had hypoxia (PaO_2 <60 mm Hg) while awake. 11 of 16 patients spent 4% to 98% of sleeping time with SaO_2 >4% less the baseline level; desaturation was worse during REM sleep than during NREM sleep; patients with lower SaO_2 and higher $PaCO_2$ when awake spent sleeping time at lower level of arterial oxygenation and had greater arterial hypoxemia during episodes of disordered breathing	Direct relevance to routine sleep laboratory investigations unclear
Douglas[175]	12 patients with chronic bronchitis and emphysema and 4 healthy subjects as controls	Transient hypoxemia (desaturation >10%), occurred in all 10 "blue and bloated" patients; hypoxemic period, usually associated with reduction in chest movement and gas flow, occurred during REM sleep	Identification of hypoxemic episodes in REM sleep

Studies	Population	Findings	Comments
Fleetham[176]	24 patients (19 males, 6 females, mean age 65.1±1.8 years) with COPD (FEV_1/FVC ratio after inhaled bronchodilators <70%); control group taken from different study; PSG given to all subjects.	All patients desaturated during sleep (SaO_2 during sleep drops >5% below awake SaO_2); lowest mean SaO_2 (maximum oxygen desaturation) was during REM sleep; apneic episodes uncommon among patients; oxygen therapy had no apparent affect on sleep quality; relief of hypoxemia with supplemental oxygen had no affect on arousal frequency	Data suggested associated phenomenon such as hypercapnia causes arousals
Fletcher[177]	7 non-obese male subjects (53 to 68 years) with COPD documented by spirometry and clinical history	Alveolar hypoventilation and gas exchange abnormalities contributed to observed hypoxemia; mean minute ventilation decreased during episodes of REM sleep, because of decreased tidal volume with reduction in inspiratory pressure; short apneas occurred in a few instances during REM sleep, but did not appear to cause reductions in SaO_2 of >4%; arterial oxygen desaturation occurred during REM sleep	Investigation of non-apneic mechanisms for desaturation during REM sleep; direct relevance to routine sleep laboratory investigations unclear
Fletcher[182]	152 patients from medical chest clinic with diagnosis of COPD using nocturnal PSG to detect oxyhemoglobin desaturation; 17 subjects disqualified because of discovery of sleep apnea or inability to sleep in laboratory	37 (27%) of 135 subjects showed nocturnal oxyhemoglobin desaturation; anthropomorphic, pulmonary function and historic factors failed to separate desaturators from non-desaturators; desaturators had significantly higher $PaCO_2$ and lower PaO_2 levels than non-desaturators.; continuous oxyhemoglobin monitoring during sleep remains the only reliable tool for detecting nocturnal desaturation	PSG with EEG monitoring needed to rule out desaturation; authors note clinical relevance of data to treatment of COPD patients is unknown
Guilleminault[179]	26 patients (21 men, 5 women; age range 27 to 76 years) with COPD referred to the sleep disorders clinic to rule out obstructive component during sleep; COPD	Most extreme O_2 desaturations in all COPD patients with sleep apnea syndrome were observed with OSA during REM sleep; REM periods during which lowest O_2 saturations were recorded contained recurrent	Limited direct relevance to COPD cases without OSA

Studies	Population	Findings	Comments
	population was apneic	long apneic events with decrease in O_2 saturation; patients' history or daytime studies are not reliable tools	
Littner[180]	9 male patients with COPD, but without apnea, recruited from chest clinic patient population; 5 healthy control subjects.	In 6 of 9 patients, acute arterial oxygen desaturation of $\geq 11\%$ occurred during REM sleep	Unclear relevance to routine sleep laboratory use
Wynne[181]	7 patients with COPD.	Disordered breathing caused 42% of episodes of desaturation, all of which were <1 minute; all episodes of desaturation lasting >5 minutes occurred in REM sleep and were not caused by disordered breathing; mean maximal desaturation was 22%	No direct relevance to routine sleep laboratory use

AHI=apnea hypopnea index; COPD=chronic obstructive pulmonary disease; FEV_1=forced expiratory volume in one second; NREM=non-rapid eye movement; NSD=no significant difference; OSA=obstructive sleep apnea; $PaCO_2$=arterial carbon dioxide pressure; PaO_2=arterial oxygen pressure; PSG=polysomnography; REM=rapid eye movement; SaO_2=arterial oxygen saturation; VC=vital capacity.

Relevance to recommendations
The ASDA review[39] states that a large amount of literature has described sleep disturbances and sleep-related breathing disorders in patients with neuromuscular diseases; and that examples in it are cited selectively. Most of the cited studies had small patient numbers. Several focused on physiological investigations and their immediate relevance to the routine use of sleep laboratory investigations was not always clear. Despite these limitations, most of the studies on neuromuscular diseases are relevant to the recommendations, though it is unclear from the cited evidence that PSG is routinely indicated for all in this heterogeneous group of disorders when other approaches such as sleep history are insufficient.

Most of the papers dealing with the investigation of persons with COPD also describe studies with small populations. There is support for the use of oximetry to assist in diagnosis and monitoring of COPD. The ASDA review[39] noted that oximetry is not a reliable tool to distinguish OSA from COPD and PSG is required to accurately distinguish upper airway disorders from other types of respiratory affects on sleep.

The ASDA review commented that OSA has not been a feature in several case series of asthmatics that have been studied for other reasons and also transient hypoxemia is usually modest compared to that which occurs in persons with COPD.

APPENDIX 11: Identified Guidelines and Recommendations for Obstructive Sleep Apnea in Children

1. American Academy of Pediatrics, 2002[27,29]
Recommendations
- Overnight PSG is the gold standard for the diagnosis of OSA in children (1<age<18) having adenotonsillectomy, habitual snoring, behaviour disturbance, hyperactivity and excessive daytime sleepiness. PSG is the only method that quantifies ventilatory and sleep abnormalities and is recommended as the diagnostic test of choice.
- Other diagnostic techniques, such as videotaping, nocturnal pulse oximetry and daytime nap studies may be useful in discriminating between primary snoring and OSA if the results of PSG are positive. Because of the high rate of false-negative results of other diagnostic techniques, PSG should be performed.

Evidence
There are marked differences between OSA in children and OSA in adults. Adult criteria for OSA cannot be used to identify children with OSA. Key features pertaining to childhood OSA considered in a workshop, convened by the American Thoracic Society,[183] are summarized here.
- The clinical manifestations of OSA in children can include snoring, laboured breathing during sleep, nocturnal enuresis, hyperactivity and attention deficit.
- Childhood OSA can result in pulmonary hypertension, systemic hypertension, growth failure, excessive daytime sleepiness, behavioural problems and impaired academic performance.
- On the basis of normative data, an AI=1 is often chosen as a cut-off for normality.
- Childhood OSA is often associated with pulmonary hypertension, resulting from nocturnal hypoxemia, hypercarbia and respiratory acidosis. This can lead to cor pulmonale and congestive heart failure. Children with OSA may develop marked sinus arrhythmia and bradycardia, but other types of arrhythmias seem to be rare.
- Hypertension is less common in children than in adults and therefore would be less expected in children with OSA.
- Failure to thrive (growth less than the fifth percentile for age) is a complication of childhood OSA.
- Sleep fragmentation and hypoxemia would affect the neuropsychological and cognitive performance of children, as in adults.
- Daytime sleepiness occurs less frequently in children with OSA than in adults with OSA.
- Empirical evidence showed the deleterious consequences of OSA on neurocognitive function and behaviour.
- Tonsillectomy and adenoidectomy are the standard first-line treatment for childhood OSA. However, children with OSA are at greater postoperative risk for respiratory compromise than children undergoing tonsillectomy and adenotonsillectomy for other indications.
- Children with severe OSA (RDI>19), with contributing factors to OSA, such as obesity and cerebral palsy, may be incompletely cured by surgery.
- More studies are required to determine which clinical and polysomnographic parameters are predictors of OSA and to establish definitive normative data on breathing during sleep.

PSG values for children: Marcus et al.[184] established normal PSG values for children and adolescents, which are different from those of adults. Overnight PSG was given to 50 healthy children and adolescents (28 males, 22 females, mean age 9.7±4.6 years, range 1.1 to 17.4 years) who were recruited from families of hospital employees. A standard questionnaire was given to the parents of each child. In this study population, the mean AI was 0.1±0.5 (range 0.0 to 3.1) events per hour of total sleep time. No child had obstructive apneas >10 seconds in duration; 30% of children had central apneas ≥10 seconds in duration and one child had a central apnea associated with SaO_2 <90%. OSA >10 seconds in duration. Maximum end-tidal carbon dioxide (PET_{CO2}) was 46±4 (range 38 to 53) mm Hg, and hypoventilation (PET_{CO2} >45 mm Hg) was 7.0±19% (range 0.0% to to 91%) of total sleep time. The arterial oxygen saturation (SaO_2) nadir was 96±2% (range 89 to 98). These authors recommended that the following values for PSG measures should be considered abnormal for the pediatric age group: AI >1; central apneas associated with desaturation <90%; peak PET_{CO2} >53 mm Hg or PET_{CO2} >45 mm Hg for >60% of total sleep time; and SaO_2 values <92%.

Rosen et al.[185] studied 20 children with suspected OSA (10 males; 10 females; average age five years, range eight months to 16 years) who had loud snoring and laboured breathing and cyclic oscillations of oxyhemoglobin desaturation during sleep. Episodes of complete obstructive apnea were generally absent [mean apnea index (AI) 1.9±3.2 events per hour]. Three of 20 (15%) children had AI ≥5. Baseline awake SaO_2 was 98% and during sleep, the value was 66±13%. Of the 194 episodes of severe desaturation (defined as a decrease in SaO_2 >15% for ≥30 seconds), 17 (9%) occurred in association with obstructive apnea events. PET_{CO2} values as high as 58±6 mm Hg were recorded in children whose waking values were 35 mm Hg to 45 mm Hg. It was concluded that adult criteria for OSA will fail to identify most children with sleep-related upper airway obstruction. Criteria based on quantitative SaO_2, PET_{CO2} values and possible paradoxical respiratory efforts may be better indices of the presence and severity of the upper airway obstruction in children.

Rosen[186] evaluated 326 children (5.8 years, range one to 12 years old) referred because of snoring and difficult breathing. OSA was diagnosed in 59% of the children, using PSG criteria for children (abnormal values PSG AI ≥1; 4% desaturation episodes of >1.5 events per hour; or SpO_2 nadir <92%; hypoventilation as indicated by end tidal carbon dioxide tension ($EtCO_2$) values ≥50 mm Hg for >9% of total sleep time or peak $EtCO_2$ values ≥55 mm Hg). Diagnoses for other children in this cohort were primary snoring (25% of 326 individuals), no snoring (10%) or upper airway resistance syndrome (UARS) (6%).

Primary snoring and OSA: Marcus et al.[187] compared blood pressures in 41 children with OSA (mean age five years) and 26 primary snorers (mean age eight years). The diagnostic category for each child was established by PSG. It was found that children with OSA had significantly higher diastolic blood pressure during wakefulness and sleep, compared with those with primary snoring. There were NSD in the systolic blood pressure indices during either sleep or wakefulness between the two groups.

A retrospective study of 83 children with snoring and/or sleep-disordered breathing, who were referred for PSG, evaluated the ability of a clinical OSA score and other questions about sleep, breathing and daytime symptoms to distinguish primary snoring from OSA.[188] Based on PSG

results, 48 patients were classified as having primary snoring and 35 as OSA. Peak end-tidal CO_2 (49±3.2 versus 55±8.2 mm Hg); lowest arterial oxygen saturation measured by pulse oximetry (95±1.9 versus 82±14%); and AHI (0.27±0.3 versus 8.4±6 events per hour) indicated that the diagnosis of primary snoring versus OSA was reasonable. As compared with PSG, the OSA score misclassified about one of four patients. It was concluded that primary snoring in children cannot be reliably distinguished from OSA by clinical history alone.

Reliability of abbreviated studies: In a large cross-sectional study, Brouillette et al.[189] assessed data for each of 349 patients (six months to 18 years of age) who were referred to a pediatric sleep laboratory for possible OSA. A comparison was made of information from a parental questionnaire, PSG results and a simultaneously obtained pulse oximetry trend and event graphics. Of 349 patients, 210 (60%) had OSA as defined by PSG (AHI ≥1). Oximetry trend graphs were classified as positive for OSA in 93 patients and negative or inconclusive in 256. Of the 93 oximetry results read as positive, PSG confirmed OSA in 90 patients. A positive oximetry result might be a good predictor of an abnormal PSG, but a negative result cannot be used to rule out OSA (sensitivity 42.9%, specificity 97.8%, positive predictive value 97%). A limitation with this study was that 89 PSGs were done in a sleep laboratory and the remainder at home.

All three studies[190-192] assessing the utility of home audio or video clinical history and physical examination in the diagnosis of childhood OSA, on a limited number of participants, concluded that these abbreviated methods can be suggestive of OSA, but PSG remains the gold standard.

In a group of children (two to six years old) with suspected OSA, PSG and video test results corresponded for 49 of 58 individuals (84%). PSG detected 36 abnormals, while video tests detected 41 abnormals.[190] Comparing with PSG, the overall interpretation of the video test yielded a sensitivity of 94% and a specificity of 68%. All patients with video test scores ≤5 had normal PSG and patients with scores ≥11 had abnormal PSG. The authors suggested that patients having test scores from six to 10 require PSG. It was concluded that 30 minutes of home video recording during sleep is a reliable screening method for OSA in children. A limitation of the study was the small number of individuals in each test-score group.

Lamm et al.[192] evaluated whether home audiotape could accurately distinguish children with OSA from those who had primary snoring in a broader age range population (mean age 5.7, range 1.8 to 18). Fifteen patients in a primary snoring group (AHI<5) and 14 in an OSA group (AHI ≥5) were assessed using a sleep questionnaire, 15-minute home audio-video tape and overnight PSG. There were NSDs between the two groups for physical characteristics or questionnaire responses. The sensitivity of audiotape as a predictor of OSA was 43% and the specificity was 80%. It was concluded that home audiotape can be suggestive of OSA, but is unable to reliably distinguish primary snoring from OSA.

The findings in the Goldstein et al.[191] study, which examined the accuracy of clinical diagnosis (clinical history, physical examination and video recording) of OSA in 30 children who had habitual snoring (1 to 14 years), showed that clinical diagnosis was sensitive (92.3%), but not specific (29.4%) as compared with PSG. The AAP review[27] draws attention to several

qualifications regarding this study, including use of a more restrictive PSG criterion for diagnosing OSA (AHI >15) , possibility of spectrum bias and undocumented reproducibility of the tape evaluation.

Nap PSG was found to be of limited reliability in the diagnosis of childhood OSA as compared with nocturnal PSG. Marcus et al.[193] compared one hour of daytime PSG to overnight PSG in 40 children suspected of sleep-disordered breathing. Significantly more children demonstrated OSA during overnight studies than during nap studies (38 versus 28). The duration of the longest apnea was also longer during overnight PSG. There were no false-positive results in nap studies. It was concluded that nap PSG may be effective for screening purposes, but overnight PSG should be performed if nap PSG is inconclusive.

Saeed et al.[194] found that in a retrospective chart review of 143 children (mean age 5.6) referred for the evaluation of OSA, secondary to isolated adenotonsillar hypertrophy, 59% of nap studies were mildly abnormal, while 66% of the overnight studies were abnormal. No individual nap study parameter had good sensitivity at predicting abnormal overnight PSG, but most had good specificity. It was concluded that nap study parameters are not sensitive in predicting abnormal overnight findings.

2. American Thoracic Society, 1996[28]

Recommendations
- PSG is recommended:
    - to differentiate benign or primary snoring from pathologic snoring (OSAS)
    - to evaluate the child with disturbed sleep patterns, excessive daytime sleepiness, cor pulmonale, failure to thrive or polycythemia unexplained by other factors or conditions, especially if the child also snores
    - if the physician is uncertain whether clinical observation of obstructed breathing is sufficient to warrant surgery or if a child needs intensive postoperative monitoring after adenotonsillectomy or other pharangeal surgery
    - in children with laryngomalacia whose symptoms are worse asleep than awake or who have failure to thrive or cor pulmonale
    - during evaluation of the child with sickle cell disease having either OSA or frequent veno-occlusive crises during sleep
    - for CPAP titration and re-evaluation of the settings

PSG is not indicated for the obese child unless there is unexplained awake hypercapnia, chronic snoring, increased work of breathing during sleep, disturbed sleep, daytime hypersomnolence, polycythemia or cor pulmonale. If excess daytime sleepiness is found not to be due to OSA, based on the results of PSG; or if excessive daytime sleepiness persists after treatment, an MSLT can be used to quantitate and determine if excess daytime sleepiness is secondary to narcolepsy.

Evidence
PSG criteria are discussed in the Rosen et al.[185] study and in an editorial comment on the problems and limitations of the OSA criteria in children.[195] Both articles note that the adult criteria for OSA are not applicable to children.

Attention should be paid to OSA children who underwent surgical treatment of the adenoids and/or the tonsils. The McColley et al.[196] study determined the frequency of postoperative respiratory compromise in 69 patients (age <18 years) who had PSG-documented OSA and were observed postoperatively in the pediatric intensive care unit. The findings showed that 16 (23%) of these patients had severe respiratory compromise, defined as intermittent or continuous oxygen saturation of ≤70% or hypercapnia, requiring intervention.

Postoperative respiratory complications after tonsillectomy or adenoidectomy in children with OSA were also described by Rosen et al.[197] This was a retrospective study, which paid attention to factors contributing to OSA, postoperative respiratory complications and intervention strategies. The study dealt with complications in 37 children (age ≤15) with OSA, proven by PSG, who underwent adenotonsillectomy. It was found that 10 of the 37 children had postoperative respiratory compromise. They had more severe apnea on their postoperative PSG (mean RDI, 67±44 versus 32±19). Nasal CPAP and bilevel CPAP were used successfully to manage the pre-operative or postoperative upper airway obstruction in five of these 10 children.

Silvestri et al.[198] evaluated 32 obese children who were referred for snoring and difficulty breathing during sleep, using a sleep history questionnaire, airway radiographs, ECG and PSG. PSG abnormalities could be predicted with up to 81% reliability using the percent ideal body weight, presence of adenotonsillar tissue and presence of ≥5 symptoms from the questionnaire.

In a retrospective study, Mallory et al.[199] determined the incidence of OSA in 41 obese patients of the age range of three to 20 years, mean age 10.4 years, who were referred from a pediatric obesity clinic for PSG and showed histories of abnormal breathing during sleep. Sleep questionnaires were given to parents and pulmonary function tests to 17 of the patients who were old enough to perform maximal respiratory manoeuvers. The pulmonary function tests showed that 18% (three of 17) with a restrictive defect and 47% (eight of 17) with obstructive changes. PSG results showed that 37% (15 of 41) of the patients were mildly abnormal due to apnea, hypopnea, excessive arousals or abnormalities in gas exchange. There was no correlation between weight, age or gender and any measurement on the PSG. The authors suggested that children with morbid obesity are at risk for sleep-associated breathing disorders and that their PSG abnormalities are usually mild. The results of this study may underestimate OSA in obese children because of the limited number and wide range of age of participants. The population in this study did not have obesity-hypoventilation syndrome, with hypercapnia throughout the PSG. The findings are inconclusive as to whether PSG is indicated for obese children.

Marcus et al.[200] evaluated the effectiveness of epiglottoplasty in the treatment of six infants with severe laryngomalacia, monitored by daytime nap PSG. Before epiglottoplasty, all patients had abnormal PSG. They all had obstructive apneas; four had hypoxemia and four had hypoventilation. Follow-up PSG showed that all patients improved after the operation.

Of three references for sickle cell disease, one evaluated the use of PSG in the diagnosis and follow-up of patients who underwent adenotonsillectomy.[201] The other two used pulse oximetry to study the incidence of OSA and hypoxia in sickle cell disease[202] and to evaluate the efficacy of pulse oximetry in estimating O_2 saturation in children during sickle cell crises.[203] The findings of the latter two studies have limited relevance for the recommendations.

The Maddern *et al.*[201] study evaluated OSA in 21 children and adolescents (mean age 8.75 years, range two to 21 years) with sickle cell disease. Clinical history and physical examination were taken and pre-and post-PSG were given to all subjects who underwent adenotonsillectomy. Thirteen patients had adenotonsillectomy and all had improvement in subjective complaints (snoring, sleep disturbance, restlessness, school attendance and performance) and objective sleep study parameters. Increased end-tidal CO_2 during sleep was a significant predictor of disease, while O_2 saturation monitoring was shown to be unreliable. The study suggested that PSG is valuable in diagnosis and in the identification of severely affected patients before and after adenotonsillectomy, because surgery in sickle cell disease patients carries a substantial risk.The conclusion should be taken with caution, however, because of the limited population and the lack of appropriate control (i.e., sickle cell patients without OSA) in this study.

3.   CPSO, 1996[19]

The CPSO guideline takes a position consistent with that of the AAP. PSG is indicated for any child in whom OSA is clinically suspected, particularly if CPAP or surgery is being considered. PSG is not required in every child with uncomplicated snoring. Two reviews are cited in support.

Relevance to recommendations
The cited studies provide indications of appropriate PSG criteria in children, which differ from those for adults. Further context is provided by the AAP review[27] in its discussion of PSG for children. PSG in this application has been poorly standardized in its performance and its interpretation. For example, an AI value=1 as a cut-off for normality is statistically significant, but it is unknown what level is clinically significant. Also, the test-retest reliability of overnight PSG has never been evaluated in children.

Several studies show the limitations of abbreviated methods in providing an adequate diagnosis of OSA. Abbreviated diagnostic techniques often yielded a high proportion of false-negatives. Claims in some of these reports that abbreviated methods would be useful in a screening role need to be qualified by the post-hoc nature of the analyses.

Most studies had a limited number of participants who often were heterogeneous in age (ranging from few months to 18 years). The findings are therefore prone to selection bias and have weak statistical significance.

Most of the cited references in the American Thoracic Society guideline covered background information on a particular disorder, but were unrelated to the indications for PSG in the diagnosis or evaluation of OSA in children. There was no evidence cited regarding PSG in CPAP titration or in the investigation of excessive daytime sleepiness (EDS). A guideline was cited in support of the use of MSLT to quantitate EDS and determine if it is secondary to narcolepsy.

APPENDIX 12: Identified Guidelines and Recommendations for Other Conditions in Children that Produce Sleep Disturbances

1. American Thoracic Society, 1996[28]

Recommendations
- Bronchopulmonary dysplasia: PSG may be indicated to detect upper airway obstruction during sleep. PSG can be used with esophageal pH monitoring to document the temporal relationship between gastroesophageal reflux and respiratory events.
- Cystic fibrosis: patients receiving supplemental oxygen may require PSG to rule out OSA if there is a history of snoring, desaturation episodes during sleep, cor pulmonale, polycythemia or disturbed sleep. PSG should also be considered for assessing the potential adverse effects of supplemental oxygen during sleep in patients with advanced lung disease who are hypercapnic when awake.
- Asthma: PSG with esophageal pH monitoring should be considered if there is concern about the presence of gastroesophageal reflux during sleep as a trigger for nocturnal symptoms.
- Neuromuscular disease: PSG with CO_2 monitoring is indicated in patients with sleep-related respiratory disturbances associated with neurological diseases.
- Alveolar hypoventilation syndrome: PSG with CO_2 assessment is indicated in children with hypoventilation syndrome. PSG is recommended for any patients with clinically stable hypoventilation syndrome who develop cor pulmonale, polycythemia, morning headache, deterioration in mental status or altered growth patterns.
- Respiratory problems in newborn infants: PSG is not recommended for the routine evaluation of infants with uncomplicated ALTE.
- PSG may be helpful in defining frequency and type of apnea and the extent of cardiac, blood gas and sleep alterations in certain infants with apnea or ALTE.

Evidence
No specific evidence was cited in support of these recommendations. For each condition, the guideline provides a background covering physiological or clinical issues that relate to sleep disturbances.

APPENDIX 13: Identified Guidelines and Recommendations for Follow-up PSG in Children

1. American Academy of Pediatrics, 2002[27,29]

Recommendations
- Children who have adenotonsillectomy should have clinical re-evaluation postoperatively; high risk patients should undergo objective testing, be retested for OSA if snoring persisted and possibly if pre-operative AHI is high.

Evidence
Suen et al.[204] reported a prospective study of 69 children aged one to 14 who were referred for evaluation of suspected OSA. Based on PSG results, 35 children (51%) had a respiratory disturbance index (RDI) >5 and 30 of them underwent adenotonsillectomy. Of the 30 who had surgery, 26 had follow-up PSG. All had a lower RDI after surgery, though four individuals still had RDI >5. A pre-operative RDI≤19.1 was the major predictor of a postoperative RDI ≤5. History and physical examination alone were not sufficient to assess the severity of OSA or the likelihood of an adequate response to surgical treatment.

These authors recommend postoperative PSG for all patients whose initial RDI is >19 and also for those with persistent snoring after surgery to ensure normalization of respiratory function.

2. American Thoracic Society, 1996[28]

Recommendations
- Repeat PSG is recommended for children previously diagnosed with OSA who exhibit persistent snoring or other symptoms of sleep-disordered breathing. If possible, the study should be deferred until at least four weeks post-surgery.
- When weight loss is the primary therapy for OSA, PSG should be repeated to determine if the weight loss program has delayed the severity of the OSA.
- Children with mild to moderate OSA, who have complete resolution of snoring and disturbed sleep patterns, do not need a follow-up PSG.
- PSG is recommended if a child needs intensive postoperative monitoring after adenotonsillectomy or other pharangeal surgery.
- For a child under one year or a child with severe OSA, follow-up PSG should be considered. The child should have a routine clinical follow-up for early detection of a recurrence.
- In children with neuromuscular disease, periodic reassessment with PSG should be scheduled at least annually.
- PSG is indicated periodically in children with alveolar hypoventilation syndrome. The frequency of a follow-up study will vary depending on the clinical stability of the child, but should occur at least annually. PSG should be used to determine the effects of any pharmacologic trials.

Evidence
The recommendations are based on consensus. The only cited reference specific to these is the McColley *et al.*[196] study in relation to the recommendation for repeat PSG in OSA children who have adenotonsillectomy.

3. CPSO, 1996[19]

Recommendations
- Children with OSA must have follow-up PSG (regardless of surgical therapy) to assess the response to treatment and to ensure that the AHI is reduced to the normal range.
- Most children require only clinical follow-up to manage simple snoring. If the child's clinical condition deteriorates, (e.g., increased daytime sleepiness, secondary enuresis, observed obstructive events, failure to thrive), then follow-up sleep studies are indicated.

Evidence
This was a consensus position.

Relevance to recommendations
There was no evidence cited for repeat PSG in children with indicated disorders, other than for OSA at post-adenotonsillectomy.

APPENDIX 14: Identified Guidelines and Recommendations for Sudden Infant Death Syndrome

1. **German Association for Sleep Medicine, Pediatric Workgroup; Austrian SIDS consensus meeting**[31,32]

Recommendations
- PSG is not indicated as a screening tool for SIDS.
- PSG is indicated in the investigation of certain physical symptoms, e.g., ALTE, cyanotic attacks, neonates with clinically evident increased oxygen requirement, unclear drop in saturation, suspected disorders of autonomic respiratory control and suspected OSA.

Evidence

Early papers cited by the guidelines included a PSG comparison of 29 near-miss SIDS cases with 30 normal controls[205] and two case reports included at the end of a review.[178] In the first of these, there were a few differences between the groups, though between three weeks and four and a half months, the number of mixed and obstructive apneas of >3 seconds during total sleep time was greater for the near-miss group. In the two case reports, a death from SIDS and a near miss were preceded by apneic episodes.

A series of studies from the Cardiothoracic Institute in London and other centres[206-210] established that measurement of apnea and cardiac disorders in infants was unable to predict increased risk for SIDS. Most of this work involved prospective studies on large cohorts of infants who had recordings of respiratory and cardiac function. Recordings for those individuals who subsequently suffered SIDS were compared with those for matched controls. Results are summarized in Table 1.

A Belgian study,[211] using a similar approach, reviewed recordings for 11 SIDS cases and 22 controls, from a cohort of 2,000. In this series, central apneas were longer in all sleep states for SIDS victims compared with controls, but none exceeded 14 seconds. Obstructive and mixed apneas were seen in eight of 11 SIDS infants compared to three of 22 controls, which were SS more frequent and lasted longer. The authors, however, did not consider such associations to be of value for population screening, noting an absence of markers in some future SIDS victims and an overlap in values between the SIDS and control groups.

Monod *et al.*[212] studied 1,000 infants during a 10-year period with PSG recordings for SIDS siblings, near-miss SIDS and controls. They found NSD in breathing characteristics between the groups. Three infants diagnosed as SIDS at autopsy had no significant abnormalities in their recordings.

Table 1: Studies of apnea and cardiac disorders in infants and risk for SIDS

Studies	Population	Findings
Southall[206]	1,157 pre-term after discharge from NICU; n=40 with prolonged apnea or extreme bradycardia, 40 controls; 5 SIDS, (6 other deaths)	No SIDS victim had apnea >20 seconds or abnormal bradycardia or arrhythmia
Southall[207]	6,194 full-term, 2,337 pre-term, recordings in first week and at home after 6 weeks; 211 controls; 29 SIDS (10 other unexpected deaths)	No recordings for SIDS victims showed prolonged apnea or abnormal cardiac rhythm or conduction
Southall[209]	Recordings of 16 SIDS victims compared with those of 127 controls	No differences between groups in breathing patterns, heart rate or respiratory rate could be demonstrated; these measurements unable to identify majority of full-term infants at risk for SIDS (authors noted that highly skewed distribution of periodic breathing patterns and apneic pauses in normals means a large number of controls needed to define normal range – a deficiency in many published reports)
Waggener[210]	9,856 infants (from Southall *et al.* studies); recordings from 10 SIDS victims and 10 matched controls	NSD between groups in breathing patterns (small numbers)
Schechtman[208]	6,914 (from Southall); 22 recordings of 16 SIDS victims compared with 66 recordings from surviving infants; included quiet sleep and REM sleep	In second month of life (7 SIDS, 21 controls), SIDS infants showed fewer respiratory pauses than other infants, mostly associated with fewer short respiratory pauses. NSD between the groups during first month of life (authors note only central and mixed apnea detected, obstructive apnea might follow different pattern)

NICU=neonatal intensive care unit; REM=rapid eye movement; SIDS=sudden infant death syndrome.

A review by Bentele and Albani[213] brings together research in this area up to the mid-1980s. They noted different methods have been applied to detect functional abnormalities of cardiorespiratory control during sleep with the aim of obtaining more specific and sensitive predictors of subsequent severe apnea and SIDS. They found that the results of numerous studies were at variance and often controversial. None of the tests improved the ability to predict the risk for SIDS. One reason for this may be a lack of standardization of methods with study groups and conditions for testing infants.

The issue of standardization is also addressed by Hunt *et al.*,[214] who found that there were significant differences between results from pneumograms, for which different definitions for apnea onset and apnea termination had been used. Normative standards for comparison should use the same scoring definition.

The recommendations on indications for PSG are not supported by specific references in the guidelines.

Relevance to recommendations
The cited studies indicate that PSG and other monitoring methods cannot provide a useful measure of risk for SIDS.

Recommendations on the use of PSG in selected infants to investigate symptoms, such as suspected OSA, are consistent with recommendations made under sections of other guidelines considered in this report.

APPENDIX 15: Identified Guidelines and Recommendations for Treatment of Snoring

Recommendations

AASM, 2001[33]
- Surgical candidates for LAUP as a treatment for snoring should undergo a pre-operative clinical evaluation and a PSG or cardiorespiratory study to determine if the candidate has a sleep-related breathing disorder, including sleep apnea.

ASDA, 1997[23,39]
- A pre-operative clinical evaluation that includes PSG or a cardiorespiratory sleep study is routinely indicated to evaluate for the presence of obstructive sleep apnea in patients before they undergo LAUP.

Swiss Respiratory Society, 2001[17]
- Pre-operative PSG or respiratory polygraphy is indicated before surgical interventions for the treatment of snoring and OSA.

Evidence

LAUP is a technique used as a treatment for snoring. The principal references cited to support the recommendations are guidelines.[13,14] One of these[13] deals with portable recording in the assessment of OSA and includes details on the scope and application, including use in cardiorespiratory studies.

The 2001 AASM document makes reference to the Viner *et al.*[81] study, which documented the unreliability of clinical examination in diagnosing OSA. Also, an abstract cited by the AASM[215] reported that of 73 patients seeking LAUP treatment, 69 (95%) had OSA by PSG, even though 41% presented with a complaint of snoring.

The AASM guideline[33] states that LAUP is not recommended for the treatment of sleep-related breathing disorders, including OSA. The Swiss guideline also refers to surgery for OSA, but does not cite any specific evidence.

APPENDIX 16: Identified Guidelines and Recommendations for Insomnia

1. AASM, 2003[34]

Recommendations

1. PSG is indicated when sleep-related breathing disorders or PLMD are suspected.
2. PSG is indicated when initial diagnosis is uncertain, treatment fails (behavioural or pharmacologic) or precipitous arousals occur with violent or injurious behaviour.
3. PSG is not indicated for the routine evaluation of transient or chronic insomnia.
4. PSG is not indicated for the routine evaluation of insomnia due to psychiatric disorders.
5. PSG is not clinically useful in differentiating insomnia associated with dementia from other forms of insomnia, including insomnia associated with depression.
6. PSG is not useful in establishing the diagnosis of insomnia associated with fibromyalgia or chronic fatigue syndrome, because the alpha-delta sleep pattern described in fibromyalgia syndrome is a non-specific finding.

Evidence

This guideline was prepared as an update to a document published in 1995.[49] The six recommendations given in the guideline that relate to PSG in the evaluation of insomnia are supported by 27 cited studies and by committee consensus.[34] Some of the studies were considered in preparing the 1995 guideline.

Recommendation 1: Of eight references cited in relation to the first recommendation, five showed support for the use of PSG in the evaluation of elderly patients with persistent insomnia associated with sleep apnea and PLM.[216-220] Three appeared to have more limited relevance.[221-223]

In a large prospective study, Coleman *et al.*[216] used PSG data for 4,689 subjects from 11 clinics to classify nine types of insomnia. Other categories were insomnia associated with psychophysiological factors (15.3%), substance dependence (12.4%) and RLS-PLMD (12.2%). The authors suggested that complaints from patients with persistent insomnia (>4 weeks) or sleep apnea and PLM, particularly in the elderly, should be investigated with PSG before sleep medication is prescribed.

In one study,[217] PSG testing of 80 adult volunteers (age ≥59 years) with insomnia of mean duration eight years, but with no clinical diagnosis of OSA was conducted. It was found that 29% to 43% of the subjects, depending on the AHI used, had undiagnosed sleep apnea. As the rate of occult sleep apnea increases with age and clinical diagnosis cannot detect this condition in a large proportion of older adults presenting with insomnia, the authors suggested that the use of PSG in research on older adults with insomnia is needed to preclude substantial representation of occult sleep apnea.

Roehrs *et al.*[218] evaluated the relationship of sleep-wake complaints and sleep-related respiratory disturbances. They found that patients with insomnia had fewer and shorter apneas with little hypoxemic effect, while patients with excessive sleepiness had more and longer primarily obstructive apneas that produced significant hypoxemia. There was potentially selection bias as

the study population consisted of 16 patients with insomnia (14 women and two men) and 65 patients with excessive daytime sleepiness (63 men and two women). The authors considered that it was important to differentiate insomnia with a respiratory disturbance from other insomnia conditions and suggested that PSG be used to evaluate patients with complaints of insomnia that persist for several months.

The Zorick *et al.*[219] and Edinger *et al.*[220] studies found that patient diagnoses associated with insomnia are heterogeneous and can be subcategorized by PSG.

Zorick *et al.*[219] assigned 84 patients with chronic insomnia to 10 diagnostic categories on the basis of medical, psychiatric and PSG examinations. Twenty normal subjects, who were paid volunteers, acted as controls. Patients in the psychiatric disorder, RLS and respiratory impairment categories had sleep patterns that were SS different to those of normals. Those in the diagnostic categories of nocturnal myoclonus, subjective complaint, atypical PSG features and circadian rhythm disturbance showed NSD from controls. The potential practical significance of such differences is unclear. There were small numbers in each diagnostic group and there was substantial variance for sleep parameter values.

Edinger *et al.*[220] performed medical, psychiatric, behavioural and PSG evaluations on 100 outpatients presenting with chronic insomnia. PSG gave important diagnostic information in 65% of the sample (34% primary sleep disorder diagnosis, 15% secondary diagnosis, 16% rule out sleep disorders). The predictive performance of the clinical examination for periodic movements of sleep and sleep apnea, was significantly poorer than the evaluation that included PSG. Patients >40 years had a SS higher rate of positive PSG findings than younger patients. The authors suggest that PSG be used routinely with older insomniacs and with younger patients who have failed initial treatment. Such conclusions appear contrary to the recommendation.

Two large prospective controlled studies by the same group estimated the prevalence of sleep apnea and nocturnal myoclonus in patients with insomnia and normal controls. They showed that sleep apnea and nocturnal myoclonus were not common causes of insomnia.

Kales *et al.*[223] found that the prevalence of these conditions were similar in 200 insomniac patients and 100 normal controls. Similar findings were reported by Vgontzas *et al.*[221] for 375 insomniacs and 150 normal controls. Eight patients and two controls presented with ≥30 apneic events per night and of these, only one met the criteria for recommending treatment. Also, small proportions of insomniacs and controls (11.5% and 8.5%) displayed nocturnal myoclonus.

The Reynolds *et al.*[222] study, where sleep patterns were assessed in 27 elderly patients, is of little relevance to the recommendation. Of 19 chronic insomniacs, two-thirds had depression or psychophysiologic disturbance. Six of seven patients with excessive sleepiness had a primary sleep disorder such as sleep apnea or narcolepsy-cataplexy. The study dealt with a highly selected population and was prone to selection bias and incorrect subcategorization.

Recommendation 2: This recommendation is the same as that included in the previous guideline.[49] It is based on eight publications cited in the previous document.

Two of the cited references appear to be particularly relevant to the use of sleep laboratories.

Jacobs *et al.*[224] examined the accuracy of the differential diagnosis of 123 patients with chronic insomnia with or without PSG. This was a retrospective study based on chart review with independent scoring by two raters. In this series of patients, who had been evaluated during a period of five years, 63% had been diagnosed before PSG as having present or past mental illness. In 49% of the cases, the sleep laboratory findings resulted in substantial modification of the initial face to face diagnosis through providing information not previously suspected from clinical evaluation as important to the patient's chronic insomnia. The authors suggested that PSG is indicated for patients with insomnia who have failed to respond to "routine" clinical intervention.

Hauri *et al.*[225] evaluated the presence of sleep abnormalities in 24 drug-free panic patients (there were 14 drop-outs from 38 selected patients for various reasons). Each panic patient was matched by gender and age with a normal sleeper. Because of the limited and select population, this study was prone to selection bias. PSG findings for panic patients were comparable to those for normal sleepers, though movement time (amount of large body movement during sleep) was increased in panic patients. The authors comment that the SS of the findings on movement time is marginal. Panic patients spent about three times as much time in large body movements as normal sleepers in all stages except delta sleep. Eight panic attacks arising out of sleep were recorded. The findings seemed to support the recommendation for PSG diagnosis of a subgroup of patients suffering from panic attacks.

A previous ASDA review cites an overview that indicates spontaneous movement disorders can occur during sleep and may have specific PSG findings that are useful in diagnosis.[226] Three studies cited indicate Alzheimer's disease is associated with a loss of rapid eye movement (REM) sleep and slow-wave sleep that is proportional to the degree of cognitive impairment.[227-229] In one study,[230] changes in sleep architecture were sufficient to distinguish elderly patients with dementia from those with depression with 80% accuracy, though diagnosis of dementia for such patients is established clinically.[227] One group considered sleep impairment to be "a rather non-specific concomitant of each dementing condition and [it] does not easily differentiate the various causes of dementia."[231] These publications appear to provide background to the recommendation, rather than direct support.

Recommendation 3: The recommendation on the lack of usefulness in the routine evaluation of transient or chronic insomnia is the same as noted in the 2000 AASM publication[50] on practice parameters for the evaluation of chronic insomnia and in a 1992 ASDA report[232] on scoring rules and examples of EEG arousals. The recommendation is based on committee consensus.

The AASM review published in association with the 2000 practice parameters[51] noted that there has been controversy about the diagnostic use of PSG in insomnia and cites specific recommendations on this topic provided in earlier AASM-ASDA guidelines.[22,39,49]

Recommendation 4: Ten references are cited in support of this recommendation, though not all of them appear to be of direct relevance.[233-242]

Gillin et al.[233] assessed the all-night EEG sleep patterns of 41 normal subjects, 56 patients with depression and 18 insomniacs. The study showed that the EEG data of the three groups were distinct and could distinguish between patients with primary depression, primary insomnia and normal controls, using multivariate analysis. However, only 77% of the insomniacs were correctly classified using this approach. Though shortened REML was associated with insomnia, seven of the 18 insomniacs had sleep latency within one standard deviation of the normal mean.

Haynes et al.[234] compared the sleep patterns, anxiety level and muscle tension of insomniacs and non-insomniacs. The study population was college students: 76 insomniacs and 208 non-insomniacs in study I; and 101 individuals in study II, which was undertaken to replicate the findings of the first study (number of insomniacs not reported). Assessment was based on the use of a sleep behaviour questionnaire. The self-reported sleep pattern results indicated that insomniacs had a general disruption of sleep patterns, high manifest anxiety and high muscle tension, but did not differ from non-insomniacs in the frequency of sleep-incompatible behaviours. The study does not seem to be relevant for the recommendation regarding the use of PSG.

The Merica et al.[235] study, which is irrelevant for the recommendation, investigated the use of proposed new variables for defining sleep continuity. The new variables were able to differentiate between patients suffering from depression, dysthymia and insomnia.

Thase et al.[236] performed a series of analyses to identify an abnormal EEG sleep profile used to characterize major depressive disorders. Sleep studies were undertaken on 44 depressed inpatients, 181 depressed outpatients and 44 healthy controls. An index score based on reduced REML, increased REM density and decreased sleep efficiency was able to discriminate between the groups. However, 55% of the depressed outpatients had "normal" sleep profiles.

Five similar studies compared the EEG sleep patterns of panic patients or depressed patients with those of normal controls.[237,238,240-242] In the first three, the studied population sizes were small, though the studies were controlled with matching age and gender. The study findings are summarized here.

- Although patients with panic disorder had lower sleep efficiency and a higher percent of wake time based on total sleep period, REM sleep characteristics were identical to those of controls (n=14 in each group).[237]
- Patients with panic disorder had normal sleep with a modest reduction in total sleep time and delta sleep; no impairment in sleep maintenance and continuity was found (n=16 in each group).[242]
- Sleep efficiency was SS lower and wake % SPT higher among patients with obsessive-compulsive disorder, compared to controls (n=22 in each group). Sleep architecture did not differ between the groups.[238] No positive correlation was found between sleep variables and rating inventories for obsession-compulsions, depression and anxiety.
- When compared to 12 controls, patients with panic disorder (n=22) and those with major depression (n=12) had shortened REML, which is a feature of depression-like sleep. However, PSG findings showed more differences than similarities between these disorders, suggesting that REML is a common final pathway of different alterations in sleep regulation. The architecture of the first NREM period in the panic disorder patients was similar to that of control subjects.[240]

Pecknold and Luthe[241] found that there was no correlation for sleep patterns of a group of 44 panic patients with reported parameters of sleep architecture in endogenous depression as reported in the literature. A comparison group of 11 patients with generalized anxiety disorder and insomnia was included in the sleep studies. Compared to the literature control values, these individuals had more stage 3 and less stage 4 sleep and increased total sleep time

Utilizing conventional PSG, Hurwitz et al.[239] evaluated the sleep of 18 Vietnam combat veterans with post-traumatic stress disorder and 10 healthy non-combat-exposed Vietnam era veterans. Although many symptoms of disturbed sleep and daytime sleepiness were reported by post-traumatic stress disorder subjects, no significant differences for any PSG variable between the two groups appeared on two successive nights.

Recommendation 5: The recommendation is based on two recent studies, work cited in the previous practice parameter considered under recommendation 2 above[227-230] and committee consensus.

Allen et al.[243] evaluated the sleep patterns of 30 hospitalized patients with dementia and of 14 non-demented patients used as controls. Both groups had a mean age of >80 years. Continuous PSG recordings were undertaken during 72 hours. Although patients with dementia had less total sleep time than control subjects, no differences were noted between dementia subgroups. There were no differences between controls and demented patients in terms of NREM-REM cycle and no association between the severity of the clinical condition and any of the sleep parameters in demented patients.

The Evans et al.[244] study focused on the prediction of outcome in severe head injury patients and appears to be of limited relevance to the recommendation. The sleep activity of 138 of 154 unselected patients with head injury, who were admitted to a neurosurgical unit, was determined. Sixteen seizure patients were excluded. It was found that the continuing presence of EEG-EEG polygraph activity, similar to that for normal sleep, correlated well with the severity of brain damage after head injury and could be used to predict outcome.

Recommendation 6: This recommendation is the same as in the previous guideline and is based on three cited studies,[245-247] a description of alpha-delta sleep[248] and committee consensus. Although the studies seem to support the notion that the alpha-delta sleep pattern described in fibromyalgia syndrome is a non-specific finding, the results should be interpreted with caution because of the lack of appropriate controls and the small sample sizes that may be insufficient to permit subclassification.

Manu et al.[245] assessed the presence of alpha wave intrusions during NREM (alpha-delta sleep) and its relationship to fibromyalgia, major depression and chronic fatigue syndrome in 30 consecutive patients who had experienced persistent tiredness for >6 months and had not been hospitalized during the three months before evaluation. The study found that NREM sleep was not significantly correlated with fibromyalgia, chronic fatigue syndrome, major depression or primary sleep disorders, but was significantly more common among patients who had chronic fatigue without major depression.

Mahowald *et al.*[246] investigated the sleep patterns in 16 patients with chronic, active rheumatoid arthritis and found that although none had normal sleep physiology (two of 16 had sleep apnea, all 16 had frequent PLM and frequent arousal), there was no evidence of any type of sleep deprivation. The alpha-delta sleep pattern was present in 13 of 16 and hyper-somnolence in seven of 16. None of the patients accurately recognized the degree of their sleep disruption.

Moldofsky *et al.*[247] found that in separate small studies, the alpha-delta sleep patterns were present in a fibrositis syndrome group (seven out of 10 patients) and in six paid volunteers who did not have this condition.

2. CPSO, 1996[19]
The CPSO document concludes that PSG anomalies provide objective physiologic evidence for the non-restorative sleep symptoms of fibromyalgia and chronic fatigue syndrome. These sleep physiological anomalies, however, may not be specific for these syndromes.

Relevance to recommendations
The level of support for the recommendations provided by the cited studies varies. Many of the papers seem to provide essentially background information and the link between this and the recommendation, informed also by committee consensus, is not always clear. Some of the studies are of limited quality, but the bigger issue for this series of recommendations on sleep laboratory applications is the relevance of the findings. Several studies indicate areas of unreliability in the potential application of PSG, such as use in the evaluation of persons with psychiatric disorders. Some difficulties in this field are well summarized by Reite *et al.* in the earlier review from ASDA.[49] "Therefore, published evidence suggests that patients with different types of chronic insomnia might be distinguishable on the basis of EEG sleep findings. However, closer consideration of these findings reveals further questions and problems. For instance, several studies use multivariate statistical techniques to distinguish patient groups. Such techniques cannot be generalised readily to other laboratories and other populations. On the other hand, most studies report group differences, but do not indicate any criterion values for evaluating the sensitivity and specificity of particular parameters. One reason is that group differences tend to be small in magnitude and inconsistent from one study to the next. Furthermore, most studies have defined different groups on clinical grounds, and even if group differences can be demonstrated polysomnographically, it is not clear whether this information helps in elucidating pathophysiology or treatment course."

PSG remains a valuable tool to diagnose insomnia associated with sleep-related breathing disorders such as sleep apnea and PLMD.

APPENDIX 17: Identified Guidelines and Recommendations for Depression with Insomnia

1. ASDA, 1997[23,39]

Recommendations
- Neither a PSG nor a MSLT is routinely indicated in establishing the diagnosis of depression.
- PSG may be useful in identifying early response to antidepressant medication.

Evidence
Cited evidence consists of one meta-analysis on sleep and psychiatric disorders[249] and 13 primary studies.[220,223,224,240,250-258] Not all of these reports appear to be directly relevant to the recommendations.

The meta-analysis by Benca *et al.*[249] reviewed 177 studies with data from 7,151 patients. Reduction in REML, which had been suggested as a specific marker for depression, was found in persons with affective disorders, but also occurred in other categories. Also, significant differences in REML from controls could not be demonstrated in some affective disorder subgroups. The meta-analysis appeared to be of reasonable quality. The authors draw attention to limitations in the literature they reviewed and to a number of assumptions made in undertaking their analysis.

Benson *et al.*[250] studied the sleep patterns of 18 male veterans who met DSM-III criteria for borderline personality disorder, who were further grouped into those with or without an affective disorder (eight and 10 patients respectively). The data were compared with those of 15 normal controls. All-night PSG recordings showed that the normal controls had better quality of sleep (as judged by measurements of sleep continuity, stage 1 and stage 4 sleep) than the two borderline groups, which could not be differentiated using any measure of sleep continuity or staging. No group differences were found for REM sleep variables. The authors concluded that REML does not distinguish between those in this patient group with or without a history of affective disorder, nor does it discriminate between those with borderline personality disorder and normal subjects. For all the borderline patients, REML was not clearly related to self-rated or clinician-related depression. The small size of the study is a limitation.

Vitiello *et al.*[256] found that there was no difference in all types of sleep measures between a group of 24 with geriatric-onset major depressive disorder who had a minimum history of seeking treatment for depression and 24 gender and age-matched controls. They suggested that elderly individuals who have a diagnosed major depression, but who have not sought health care, do not necessarily manifest sleep disturbances thought to be characteristic of major depression. The authors suggest that the degree of severity of major depressive disorders and the heterogeneous nature of this condition as possible factors that might account for the differences between their results and those from earlier studies.

Nofzinger *et al.*[254] compared PSG and MSLT findings for 25 hypersomniacs with bipolar depression with those for 23 non-depressed narcoleptic patients. No abnormalities were noted for the bipolar depression group in the results from the MSLT. REM sleep was absent during

daytime naps in the depressed patients. The depressed patients seemed to have adequate nocturnal sleep duration and quality.

These studies suggested that neither PSG nor MSLT is useful in the diagnosis of depression.

Three studies on the use of PSG in evaluation of chronic insomnia[220,223,224] are also cited in the guideline. Their findings have limited relevance to the recommendation regarding diagnosis of depression. Two studies[223,224] showed that among the insomniac patients, a group could be identified as having psychopathological factors.

Four studies[240,252,253,255] compared the EEG sleep studies of patients with major depression of different etiology to those of normal controls. From these studies, two characteristics seem to differentiate the two groups; the increased REM density and the reduced REML in patients with major depression.

Lauer *et al.*[252] suggested that REM density is more likely a biological marker for major depression than REML, because the latter showed NSD between patients and controls until they were older than 34 years. Their study, however, was based on a comparison of 76 patients with depression and 51 healthy controls, so the specificity of REM density as a marker for depression in relation to other psychiatric disorders was not addressed.

A small PSG study comparing the sleep patterns of 22 persons with panic disorder with those of 12 persons with major depression and 12 controls found that both groups of patients had shortened REML.[240] The authors suggested that shortened REML is "merely a common final pathway of different alterations in sleep regulation." REM density patterns were normal in those with panic disorder, but increased in those with depression.

In another study,[253] this research team found that a group of 54 healthy subjects with close relatives suffering from depression or other psychiatric disorders, had reduced slow-wave sleep and increasing REM density in the first sleep cycle. As a psychiatric disorder would develop in a minority of these persons, however, a more individualized approach was needed in looking for biological markers. Follow-up was undertaken on 18 individuals who had shown a sleep pattern resembling those of depressed patients. This study seems to be of no practical significance for sleep laboratory referral and the use of PSG characteristics to help identify persons at risk for depression was not established.

Another study cited[255] examined sleep patterns in bereaved persons who were not suffering from depression and did not appear relevant to the diagnosis of depression.

Two studies are cited in the supporting review[39] in respect to whether PSG is useful in identifying an early response to antidepressant medication. Kupfer *et al.*[251] used EEG sleep studies rather than full PSG, to monitor the effect of imipramine during the maintenance treatment of 27 patients, with a median follow-up of 34 months. REM sleep parameters including REML were affected immediately after the start of treatment and remained unchanged throughout follow-up. It is concluded that sustained clinical improvement is accompanied by persistent sleep alterations. The authors noted that observed changes may not be specific for

depressed patients, as they may represent alterations related to chronic imipramine. This study showed an association in a non-controlled case series with treatment by a specific antidepressant.

Ware *et al.*[257] examined the sleep of depressed insomniac patients, who were treated with either trimipramine or imipramine, in a double-blind randomized group design (n=14 and 16), during a 30-day period. Despite the fact that imipramine and trimipramine appeared equal in their antidepressant efficacy, there were pronounced differences in PSG parameters. Most PSG variables indicated a normalization of sleep for the trimipramine condition, with the elimination of objective evidence of sleep disturbance, whereas the imipramine condition showed no improvement from the baseline data and a worsening for some variables, such as the percent of time awake and percent of deep sleep. Depression improved similarly in each group of patients. The authors concluded that antidepressants may vary in their effects on sleep, even if they have similar affects on depression. Also REM sleep suppression does not necessarily accompany clinical improvement in depression.

Relevance to recommendations
The findings in some of the cited references support the recommendation of using routine PSG or MSLT in the diagnosis of depression, in that sleep characteristics are not specifically associated with psychiatric status. None of the studies were concerned with the diagnosis of depression.

The use of PSG in identifying an early response to antidepressants is not supported by the cited evidence. Nor is it clear how sleep studies would be clinically useful in this application.

APPENDIX 18: Identified Guidelines and Recommendations for Narcolepsy

1. AASM, 2001[35]

Recommendations

- For patients suspected of having narcolepsy, an all-night PSG is done primarily to ascertain the presence of concurrent sleep disorders. It is followed immediately by a MSLT to help confirm the diagnosis.
- PSG re-evaluation should be considered if the symptoms of sleepiness increase or if specific symptoms develop that suggest new or increased sleep abnormalities, as might occur in disorders such as sleep apnea or PLMD.

2. ASDA, 1997[23,39]

Recommendations

- PSG and a MSLT performed on the day after PSG evaluation are routinely indicated in the evaluation of suspected narcolepsy.
- Initial PSG and MSLT occasionally fail to identify narcolepsy. Repeat testing is necessary when the initial results are negative or ambiguous and the clinical history strongly indicates a diagnosis of narcolepsy.

Evidence

The 2001 AASM publication[35] cites two earlier guidelines (ASDA 1992[147] and ASDA 1997[39]) in support of the first recommendation. The second recommendation is indicated as being the same as in the previous guideline[39] and is based on committee consensus.

Characteristics of narcolepsy and its diagnosis: The most pervasive and sometimes the only symptom of narcolepsy initially is EDS. Narcolepsy, however, is one of many causes of hypersomnolence and the presence and severity of other disorders causing this cannot always be reliably evaluated by the clinical history or physical examination.[39] As treatment for narcolepsy requires long-term medication, assessment of the severity and cause of the condition is important before proceeding to treatment.

The diagnostic features of narcolepsy include a history of hypersomnolence, cataplexy, sleep paralysis, hallucinations, short sleep latencies, early onset of REM sleep shown by PSG and short sleep latency and presence of REM sleep on two or more daytime naps shown by MSLT. However, all of these findings are only present 50% of the time.[39] The ASDA review cites the Rosenthal *et al.*[259] study, which retrospectively compared PSG and MSLT data of those with narcolepsy to follow-up questionnaires reported by these patients. Of the 119 patients (50%) who returned questionnaires, only 48% had the tetrad of narcolepsy symptoms.

In a large retrospective study, Aldrich[260] reviewed the clinical findings, questionnaire results and sleep studies in 3,618 patients with narcolepsy, idiopathic hypersomnia, mild sleep apnea and EDS not otherwise specified. Of the 67 individuals diagnosed with narcolepsy, 42% did not have a history of cataplexy.

Reliability of the MSLT: The ASDA review cites two studies to support its statement that a finding on MSLT of a mean sleep latency of <5 minutes and >2 sleep onset REM periods (SOREMPs) is considered diagnostic for narcolepsy when clinical history is indicative of this and sleep fragmentation due to other sleep disorders is absent on all-night PSG. In the first of these studies,[261] 40 patients with narcolepsy, who had respiratory abnormalities and nocturnal myoclonus ruled out by PSG all had >2 SOREMPs in a five-nap protocol on the MSLT. Fourteen control subjects, who were reported in a companion article,[262] had no REM sleep episodes on the MSLT. That article described two small studies that compared all-night PSG and MSLT data of pre-selected narcoleptic and control subjects (totals 27 and 14 respectively). Although those with narcolepsy fell asleep more readily, had less total REM sleep, more wake time after sleep onset and more body movements than controls, they did not differ from the controls in terms of latency to nocturnal REM sleep. No reliable parameters of nocturnal sleep presented that could be used to unambiguously diagnose narcolepsy.

The MSLT, on the other hand, differentiated those with narcolepsy from controls (mean sleep latency 2.0 to 3.2 minutes for narcoleptics versus 7.0 to 12.1 minutes for controls). This small study established the basis for a diagnostic approach.

In the second study[263] cited in the ASDA review, 161 patients referred because of excessive sleepiness were evaluated with PSG and MSLT. In the MSLT, the 50 patients with narcolepsy had a higher proportion of naps with REM sleep than other diagnostic groups and SS shorter latency to REM.

Amira *et al.*[264] examined retrospectively the MSLT data for 144 of a consecutive series of 157 patients presenting with EDS or other symptoms suggesting narcolepsy. All but one of 52 patients who exhibited ≥2 SOREMPs on the MSLT had clinically apparent narcolepsy. There was one person with narcolepsy among the 80 individuals who had no SOREMPs and nine from the 12 with one SOREMP. From these data, the criterion for diagnosis of narcolepsy of ≥2 SOREMPs had a sensitivity of 84% and a specificity of 99%. The criterion of a sleep latency of <5 minutes had a sensitivity of 57% and specificity of 94% (35 of 40 individuals had narcolepsy).

Moscovitch *et al.*[265] suggested that a history of cataplexy was a stronger discriminant of narcolepsy than the presence of ≥2 SOREMPs on MSLT. Their data indicated that MSLT with the ≥2 SOREMP criterion identified 72% of the patient population as having narcolepsy whereas a history of cataplexy identified only 65%. As indicated in the ASDA review,[39] ≥2 SOREMPs were more likely in patients with narcolepsy with cataplexy than in those without. Their findings were based on the retrospective evaluation of the records of 306 subjects with ESD unrelated to obstructive sleep apnea or other known syndromes.

In a series of 100 patients with EDS described by van den Hoed *et al.*,[266] 41 of 46 individuals with narcolepsy (89%) had a history of cataplexy and 39 had >2 SOREMs on the MSLT.

Nocturnal PSG and subsequent MSLT can be used to differentially diagnose patients with insufficient sleep from patients with narcolepsy. Roehrs *et al.*[267] retrospectively compared data from sleep questionnaires, nocturnal PSG and MSLT of 59 consecutive, unselected patients

receiving an insufficient sleep diagnosis and 66 selected patients with narcolepsy. Narcolepsy patients having disturbed nocturnal sleep were excluded. The study showed that as compared with narcolepsy, patients with insufficient sleep had atypically high sleep efficiency at night, prolonged sleep time, elevated percentage of stage 3 to 4 and REM sleep, displayed moderate sleepiness and no SOREMPs on MSLT. Results from the study suggested that PSG and MSLT could distinguish between the two patient groups.

Narcolepsy in childhood and early adolescence is often misdiagnosed or undiagnosed, although it can be indicated by nocturnal PSG followed by MSLT. Dahl *et al.*[268] presented 16 consecutive cases of children and adolescents having proven narcolepsy with onset of symptoms by age 13 years or younger. Only one of the 16 had the classical clinical tetrad of symptoms. Four patients were initially misdiagnosed with psychiatric disorder before the detection of narcolepsy. The study suggests that irritability, mood disturbances, non-specific personality changes, social and academic impairment and obesity should raise the suspicion of narcolepsy. PSG studies are required to confirm the diagnosis.

Rosenthal *et al.*[269] presented three cases of subjects with MSLT as evidence of narcolepsy, but no overt clinical symptoms. The data for these subjects displayed ≥ 2 or more SOREMPs and short mean sleep latency (<5 minutes).

Three studies lack direct relevance to the recommendations. Broughton *et al.*[270] monitored 24-hour sleep-wake patterns in 10 persons with narcolepsy-cataplexy and 10 controls by ambulant EEG polygraphy. They suggested that ambulatory monitoring was useful in evaluating the nature of the sleep disturbance under "real life" conditions and in analyzing response to treatment, but it remained uncertain whether the results obtained had diagnostic specificity. Mitler *et al.*[271] used PSG and MSLT to assess the treatment of narcolepsy using methamphetamine. Uchiyama *et al.*[272] investigated the effects of extended night sleep on subsequent daytime sleep propensity.

Idiopathic hypersomnia: The ASDA review[39] states indications for the evaluation of idiopathic hypersomnia, a disorder of presumed central nervous system cause, are the same as for narcolepsy.

3. CPSO, 1996[19]

Recommendations
- Supportive evidence may be provided by MSLT or PSG.
- Follow-up sleep studies such as PSG, MLST or MWT are indicated to evaluate the objective response of daytime sleepiness to treatment and if symptoms deteriorate or where there is suspicion another sleep disorder may co-exist and cause day time fatigue and sleepiness.
- It is debatable whether PSG is required to make the diagnosis in every case.

Evidence
For narcolepsy diagnosis, the guideline cites a review article and a case series to support its position on whether PSG is needed in every case. Reference to the use of MWT in evaluating response to treatment is supported by a narrative review. Otherwise, the recommendations reflect a consensus.

Relevance to recommendations
Several observational studies of fair quality have indicated that the diagnosis of narcolepsy may be complex, as typical clinical diagnostic features will not always be present. Both prospective and retrospective observational studies have established the usefulness of MSLT in improving the certainty of diagnosis of narcolepsy and of PSG in identifying or ruling out other sleep disturbance conditions.

APPENDIX 19: Identified Guidelines and Recommendations for Restless Legs Syndrome (RLS) and Periodic Limb Movement Disorder (PLMD)

1. AASM, 1999[37,38]

Recommendations

- Diagnostic criteria are available and are based on a patient's history and/or bed partner's observations for RLS; and the patient's history combined with PSG for PLMD.

2. ASDA, 1997[23,39]

Recommendations

- PSG is not routinely indicated to diagnose or treat RLS.
- PSG is indicated when a diagnosis of PLMD is considered because of complaints by the patient or an observer of repetitive limb movements during sleep and frequent awakenings, fragmented sleep, difficulty maintaining sleep or excessive daytime sleepiness.

3. German Sleep Society, 2002[36]

Recommendations

PSG is recommended in adult patients with probable or definite RLS in the following situations:

- based on the patient's history and clinical symptoms, RLS is probable, but symptoms may appear atypical or are affected by other disorders
- ongoing severe insomnia and/or lack of efficacy in patients with typical RLS symptoms treated with sufficient dosages of dopaminergic drugs
- the patient complains about daily sleepiness as leading symptoms; RLS symptoms are present, but patient does not feel impaired by them
- the patient (<30 years old) suffers from severe RLS (severity scale >25) and should be treated daily with dopaminergic drugs or opioid medication
- the patient is diagnosed with RLS and additional sleep-related respiratory disorder and complains about continuing symptoms of RLS under pharmacotherapy
- an expert's report is needed for judicial purposes.

4. CPSO, 1996[19]

Recommendations

- The diagnosis of RLS or PLMD is suggested by clinical examination. It is confirmed by full PSG. Because of night-to-night variability, two nights of monitoring may be required to avoid false-negative findings. Unattended monitoring of limb movements with bilateral sensors or EMGs may be a cost-effective method when clinical history suggests the disorder.

Restless legs syndrome: The AASM review[38] cites lists of minimal criteria for the diagnosis of RLS proposed through consensus between many sleep disorders centres.[273,274] Both lists refer to symptoms that can be considered through clinical history and observation outside the sleep laboratory.

The review makes the following points regarding the possible use of PSG for an assessment of RLS. Not all patients with RLS have demonstrated a periodic limb movement index (PLMI) >5 on PSG, so not all can be evaluated with PSG.[274] The most thorough PSG study published at the time that the review was prepared had shown 80% of a series of 133 RLS patients had a PLMI>5. For a group of 49 patients who had PSG for two consecutive nights, 82% had a PLMI>5 on each night and this proportion rose to 88% if the worst night for each patient was used.[275] According to a review article,[276] not all patients with PLMs have RLS symptoms.

The review also cites a study by Montplaisir *et al.*[277] in stating the correlation between RLS symptoms and PSG findings has been inadequately studied. That study assessed the use of immobilization tests with EMG recordings, made either while the patient is sitting on the bed with legs outstretched with instructions not to move (SIT test) or with the legs immobilized on a stretcher (FIT test). Sixteen patients with RLS and 16 age-matched controls were studied in a sleep laboratory for two consecutive nights with the SIT and FIT tests being administered before PSG in a crossover design. There were no differences between patients and control subjects in terms of sleep latency, total sleep time and the percent of sleep stages; though patients woke more frequently and had lower sleep efficiency. The SIT test was found to discriminate between patients and controls better than the FIT test or the PLMI, but the authors noted that further validation would be needed. The AASM review makes the same point and notes that the test is limited by the need to perform it in a laboratory.

The 1997 ASDA review[39] cites three publications as evidence for RLS. The Coccagna and Lugaresi[278] paper is primarily a review, but reference is made to polygraphic recordings made on the authors' patients, 32 of whom had RLS and eight nocturnal myoclonus. For the RLS cases, the motor phenomena recorded by polygraphy confirmed the patients' descriptions of their symptoms. When compared with 21 recordings for a population of young normal adults, those for RLS patients showed reduced total sleep time and sleep efficiency, prolonged sleep latencies, increased stage 1 sleep, decreased stage 3 and 4 sleep and increased awakenings.

The Pelletier *et al.*[279] paper described the use of the FIT test in 10 patients with RLS, all of whom also had PSG. The PLMI index (PLMs/total sleep time) was normal in one patient, low in five and provided an underestimation of the severity of RLS.

The Walters *et al.*[280] study included PSG and EMG measurements on 11 RLS patients, all of whom had PLMs and showed significant increases in awakenings and decreases in sleep efficiency compared with historical values for age and gender.

Some studies cited in the ASDA review provided background relating to the condition and were not directly relevant to the recommendations. Coleman *et al.* included the numbers of PSG investigations involving sleep-related myoclonus and RLS as part of a survey of patients' records from sleep-wake disorders clinics.[216] Reports by Walters *et al.*[281,282] described a trial of

dopamine agonist bromocriptine in RLS patients and case studies on two individuals whose symptoms were abolished by opiates.

One study[275] was cited to suggest that repeated PSGs seem to improve the specificity of PLM in sleep recordings. A correlation between clinical symptoms and PSG findings was seen and most RLS patients had PLM (index >5) in sleep. Multiple PSG appeared to increase the sensitivity of PLM in sleep. The authors comment that although RLS is often associated with PLMs, PLM in sleep is not mandatory for the diagnosis of RLS.

In the six situations listed in the German Sleep Society consensus statement, no evidence is cited in support of the recommendations on the use of PSG for adult patients with RLS. The conclusions of the CPSO guideline seem to be based on consensus.

Periodic limb movement disorder: In PLMD, the limb movements occur during sleep and are often associated with arousals or awakenings. PSG has been included in its diagnosis in part to determine the number or frequency of PLMs and to distinguish it from other sleep disorders. Minimal diagnostic criteria given by International Classification of Sleep Disorders[273] do not include a reference to PSG.

The ASDA review[39] lists 18 publications as providing evidence for PLMs during sleep. Some of these, such as a report and earlier papers by Ancoli-Israel *et al.*[123] Bixler *et al.*[283] Coleman *et al.*[284] and Mendelson[285] are concerned with the prevalence and impact of PLMs, particularly in older persons. In several studies, persons with PLMD reported being less satisfied with their sleep and had PSG evidence of sleep disturbances.

Dickel and Mosko[286] studied 100 community residents ≥60 years of age who underwent three consecutive nights of PSG and also completed sleep questionnaires, sleep logs and sleep interviews. Based on the five per hour cut-off, 58% had PLM, but the frequency of subjective sleep-wake and mood disturbance was low across the methods of assessment. Higher cut-offs also proved to be weak or ineffective in predicting subjective sleep-wake and mood disturbance. In a small series (n=15) studied by Edinger *et al.*,[287] using PSG variability in either movement indices or movement-related arousal indices seemed to have little effect on PLMD severity classification or clinical treatment decisions derived from the blind examinations of PSG.

In a group of 46 community resident seniors studied by Mosko *et al.*,[288] subjects with PLMs had evidence of objective sleep disturbance, but only one quarter of them admitted to subjective sleep complaints or daytime sleepiness. The severity of PLM failed to predict sleep-wake complaints and vice versa. Wittig *et al.*[289] found, in a series of 57 individuals with narcolepsy, that those with PLM had SS more awakenings, stage 1 sleep, wake after sleep onset and disturbed sleep architecture than those who did not. There was NSD in MSLT results.

Several studies provide support for the use of PSG in the diagnosis of PLMD through documenting the poor performance of alternative methods. Douglass *et al.*[77] developed a sleep disorder questionnaire to accomplish multiple goals, one of which was to estimate the chance of a waiting-list patient having a sleep disorder diagnosable by PSG. Their study included 519 individuals, 435 of whom were clinical sleep-disorder patients. The controlled trial included five

groups: persons with sleep apnea, narcolepsy, psychiatric conditions or PLMD and normal controls. The PLMD patients scored lowest on the PLM scale in the questionnaire, which also showed the poorest sensitivity and specificity. PLMD was poorly identified by the questionnaire. The mean age of the control group (25 to 28 years) was lower than that of the patients (e.g., 50 years in sleep apnea), so that there was a population bias.

A retrospective study, based on a chart review of 123 patients, assessed the accuracy of differential diagnosis of chronic insomnia with or without sleep laboratory findings. Sleep laboratory studies modified the clinical findings in 49% of the patients, including 10 of the 15 persons with a final diagnosis of nocturnal myoclonus.[224]

Bliwise et al.[290] measured PLMs in a group of 63 elderly patients and related these to renal function and symptoms of RLS and disturbed sleep. Few individuals with >40 PLMs had symptoms characteristic of RLS (13 of 23). Individuals with high and low numbers of PLMs could not be distinguished by difficulties in staying asleep, daytime sleepiness or fatigue.

Edinger et al.[291] studied 20 patients who had complaints of insomnia. Using ambulatory PSG recording during three consecutive nights, they found that 11 were suffering from PLMs. Ten of the 20 patients experienced four or more first-night effects and there was intrasubject variability for sleep parameters, suggesting the need for >1 night of PSG tests.

Also cited in the ASDA review is a study by Buysse et al. that focused on the classification of clinical diagnoses in 216 patients with insomnia.[292] On the basis of interviews by sleep specialists, before PSG, there was one primary diagnosis of PLMD, but 56 instances where PLMD was given as a secondary or rule-out diagnosis.

Allan and Early[293] found NSD between the number of PLMs of 30 patients with RLS (given as 40 in the text ASDA text[39]) and 16 patients with PLMD, though those with RLS had a significantly higher percentage of PLMs associated with arousals. This study is cited as an example of the response of PLMD to pharmacologic treatment, though in this case, augmentation of RLS with carbidopa-levodopa presented an adverse effect. Finally, a paper by Yamashiro and Kryger[294] reported on the use of CPAP in 15 patients with sleep-disordered breathing and PLMs. The PLMI decreased significantly in this group after CPAP treatment (baseline, 17.8 ± 10.1; CPAP, 9.2 ± 5.7; $p < 0.05$).

Relevance to recommendations
The cited material provides reasonable indications that the diagnosis of RLS can be adequately undertaken outside the sleep laboratory. There is also evidence, from case series, of the unreliability of sleep studies for diagnosing RLS. No evidence is available to support recommendations on the wider use of PSG given in one guideline.

There are indications of the diagnostic increment provided by PSG in the investigation of PLMD, though from studies of generally modest quality and power.

APPENDIX 20: Identified Guidelines and Recommendations for Parasomnias and Sleep-related Epilepsy

1. International League against Epilepsy, 2002[40]

Recommendations
- Sleep recordings can increase the accuracy of epilepsy diagnoses when standard EEG fails to show any epileptiform activity and the level of clinical suspicion justifies this investigation.
- PSG should always be used for all-night recordings to record epileptic nocturnal activity and to differentiate this activity from non-epileptic events.

Evidence
No supporting evidence is cited in the document.

2. ASDA, 1997[23,39]

Recommendations
- PSG, including video recording and additional EEG channels in an extended bilateral montage, is routinely indicated to assist with the diagnosis of paroxysmal arousals or other sleep disruptions thought to be seizure-related, when the initial clinical evaluation and results of a standard EEG are inconclusive.
- PSG is indicated when evaluating patients with sleep behaviours suggestive of parasomnias that are unusual or atypical.
- PSG may be indicated in situations with forensic considerations, (e.g., if onset follows trauma or if the events themselves have been associated with personal injury).
- PSG may be indicated when the presumed parasomnias or sleep-related epilepsy does not respond to conventional therapy.
- PSG is not routinely indicated in cases of typical, uncomplicated and non-injurious parasomnias when the diagnosis is clearly delineated or for patients with epilepsy who have no specific complaints consistent with a sleep disorder.

Evidence
Twenty-seven references cited to support the use of PSG, including electroencephalography (EEG), equipped with or without video recording, to diagnose and monitor parasomnias and sleep-related epilepsy are summarized below. In addition, the ASDA publication cites two reviews and five primary studies to support the diagnosis of uncomplicated parasomnias on the basis of historic clinical features and a retrospective study commenting on the limited direct evidence supporting diagnosis of sleep-related epilepsy by PSG. These publications have not been reviewed in detail for this report.

Sleep-related injury: The review of three studies on PSG diagnosis of parasomnias and sleep-related injury[295-297] is summarized in Table 1.

Table 1: Studies on sleep-related injury

Study	Population	Approach	Findings	Comments
Schenck[295]	100 consecutive adults (seen during 6 years) complaining of repeated nocturnal injury to themselves or their bed partners	PSG and clinical evaluation using questionnaire, psychiatric interviews before PSG, MSLT for patients with suspected EDS	PSG identified 5 diagnostic categories in 91 patients: night terrors and sleepwalking (n=54), REM sleep behaviour disorders (n=36), dissociative disorders (n=7), nocturnal seizures (n=2) and sleep apnea (n=1)	PSG studies established diagnosis in 65 patients and strongly supported clinical diagnosis in 26
Aldrich[296]	122 patients (65 children, 57 adults) with suspected parasomnias	Video-EEG polysomnography (VPSG) for 1 (n=106) or 2 (n=16) nights	VPSG provided 43 (35%) studies leading to definite diagnosis, 37 (30%) studies supporting specific diagnosis and 42 (34%) inconclusive studies, VPSG was useful diagnostically in 28 of 36 patients who had known epilepsy	VPSG, with capability for EEG analysis at 30 mm/s, can provide useful diagnostic information in up to 2/3 of patients with variety of unexplained nocturnal movements or behaviours
Guilleminault[297]	41 subjects with complaint of nocturnal wandering; 29 (19 men) classified as "violent group" and 12 (5 men) as non-violent group	PSG, wake and sleep EEGs, ambulatory EEG in home environment; clinical interviews, psychiatric evaluations	PSG was useful in making presumptive diagnosis of sleep disordered breathing (4/41), REM behaviour disorder (3/41), somniloquy (11/41), limb movements (4/41) and abrupt sitting up in bed (4/41) occurred in stages 3 to 4 NREM sleep; several subjects had normal PSG monitoring; no full parasomnia events in the laboratory	Study suggests nocturnal PSG alone not helpful in defining nocturnal wandering; repeated ambulatory monitoring, in home environment if possible, is helpful diagnostic tool

EDS=excessive day time sleepiness; EEG=electroencephalography; MSLT=multiple sleep latency test; NREM=non-rapid eye movement; PSG=polysomnography; REM=rapid eye movement; VPSG=video polysomnography.

Somnambulism: Somnambulism (sleepwalking) and sleep night terrors are defined as "disorder of arousal, not of sleep, arising out of slow-wave sleep (stages 3 and 4), associated with body movement, intense autonomic activation, mental confusion and disorientation, autonomic behaviour, relative non-reactivity to external stimuli, poor response to efforts to provoke behavioural wakefulness, retrograde amnesia for many intercurrent events and fragmentary or no recall of apparent dreams."[298]

Table 2 summarizes 12 studies on sleepwalking cited by the ASDA review.[39]

Table 2: Studies on somnambulism (sleepwalking)

Studies	Population	Approach	Findings	Comments
Jacobson[299]	9 subjects who had most frequent sleep walking, selected from 40 purported sleepwalkers	Prospective study using continuous EEG recording	Sleepwalking incidents occurred during slow-wave sleep and were unrelated temporally to REM periods; typically, incidents began during stages 3 or 4 of sleep.	Indicated assumed association between dreaming and sleepwalking is questionable
Kales[300]	4 sleepwalkers (age 9 to 11 years) and 4 normal children (age 7 to 11 years)	All-night EEG recordings	All sleepwalking incidents occurred in slow-wave sleep (NREM), typically stages 3 and 4, and not during REM sleep; abundant occurrence of paroxysmal, high-voltage bursts of delta-frequencies during slow-wave sleepwalkers, but not in normal controls	Overlap population from Jacobson;[299] small number of participants
Fisher[301]	11 subjects suffering from stage 4 night terrors.	EEG, EKG, respiration, eye movements observed for periods varying from 4 to 19 nights, for total of 101 nights	11 subjects had 12 spontaneous stage 3 to 4 night terrors during 101 nights (range 1 to 38); stage 4 arousals designated as night terrors are preceded by longer periods of stage 4 than arousals of lesser intensity; about 2/3 of stage 4 and night terrors occur in first NREM period	Considered physiological aspects of night terrors; about 2/3 night terrors arose in first NREM period; little direct relevance to recommendations on sleep laboratory use

Studies	Population	Approach	Findings	Comments
Fisher[302]	Case series of 6 patients suffering from night terrors, age 27 to 34 years	EEG, EKG, respiration, eye movements observed	Diazepam treatment decreased stage 4 more rapidly and steeply than decline in incidence of night terrors	Small series; concerned with suppression of night terrors using pharmaceuticals; little relevance for recommendations on sleep laboratories
Vela[303]	Study on 6 children suffering from night terrors	PSG during several nights while being treated with bromazepam	PSG recordings showed slow-wave sleep (stages 3 and 4) reduced on third night of drug's administration and was maintained after drug was discontinued; other sleep parameters not significantly affected	Effect of drug therapy on night terror episodes inconclusive because of small number of participants and short duration of study; no relevance to recommenda-tions on sleep laboratory use
Halasz[304]	9 persons (age 15 to 35 years, mean age 23.4 years) suffering from sleepwalking or night terrors and 8 age- and gender-matched healthy subjects as controls	PSG during first night of sleep in sleep laboratory	Number of microarousals significantly larger in patient group than in controls, owing to elevation of the number of micro-arousals preceded by EEG slow-wave synchronization (MAS) in NREM sleep (stages 3 to 4)	No DoA events recorded by PSG; high frequency of MAS a potential marker for confusional awakenings
Crisp[305]	12 patients referred consecutively to a clinic; 6 diagnosed with sleepwalking and 6 with night terrors	10 of 12 patients subjected to psychological measurement through questionnaires and experien-tial index; also underwent all-night PSG	Of 5 sleepwalkers studied, 4 showed shift from stage 4 sleep to arousal; 3 of 5 patients with night terrors showed a shift from stage 4 to arousal	Use of PSG in diagnosis is secondary; findings are therefore of limited relevance
Whyte[306]	10 adults with complaints of sleep walking	All subjects completed sleep questionnaires and underwent	Sleepwalking observed almost exclusively in deep NREM sleep; 41 events (86%) initiated from stage 4 sleep, 2	Sleepwalking not confined to first third of night, as reported by others; small number of

Studies	Population	Approach	Findings	Comments
		PSG studies	from stage 3 (5%) and 4 from stage 2 (9%); no unusual activity seen with REM	participants
Popoviciu[307]	27 patients with parasomnias (night terrors, nocturnal motor automatisms, nocturnal verbal automatisms and bruxisms) associated with magnesium deficiency	PSG and video during one night	EEG abnormalities occurring in slow-wave sleep (especially in the stages 1b, 2 and 3) with disappearance in REM sleep	Selected cases of patients with magnesium deficiency; control values for PSG from earlier studies, little relevance to recommendations on sleep laboratories
Montagna[308]	6 patients complaining of troublesome awakenings during night resulting in poor sleep and daytime tiredness	PSG	PSG showed paroxysmal short-lasting arousals slow-wave (NREM) sleep, associated with complex movements and autonomic activation; no tonic discharges shown on ictal and interictal EEG, except 1 patient had tonic-clonic seizure during sleep	Authors suggested these paroxysmal arousals resemble other NREM parasomnias such as pavor nocturnus
Blatt[309]	24 sleep walkers, mean age 19.5 years and 12 controls who had normal sleep, mean age 26.1 years	All-night PSG; those analyzing records blinded to patients' classification	As compared with controls, sleepwalkers had more epochs containing hypersynchronous delta wave (HSD) (59.6±60.1 versus 1.7±3.2), higher proportion of HSD to total time spent in stage 3 to 4 (24.9±21.1% versus 1.1±2.0%), more stage 3 to 4 sleep interruptions (8.4±5.7 versus 3.7±1.7) and larger proportion of sleep time in stage 3 to 4 (30.6±11.7% versus 22.6±6.8%).	Sensitivity and specificity of PSG recordings remain to be investigated; suggested as easy to use variables to characterize adult sleepwalking

Studies	Population	Approach	Findings	Comments
Llorente[310]	11 patients from medically affiliated sleep disorders program who were identified with night terrors	Retrospective chart review and structured clinical interview; 10 patients had PSG evaluation, 6 agreed to interview	PSG documented EEG arousals in 10 patients, 9 of these from slow-wave (NREM) sleep; none met criteria for panic disorders and night terror events typically dissociated from psychiatric disorders	Events that occur in NREM sleep can be detected by PSG; small number of participants; limited generalizability
Moldofsky[311]	64 consecutive patients (63 adults) referred for investigation of sleepwalking or sleep terrors; categorized according to clinical history into three groups: serious violence during sleep to other, property or self; harmful, but not destructive behaviour; non-violent behaviour	PSG for 1 night and medical-psychiatric examination	During PSG, 20 patients showed sudden behavioural arousal from stage 2 or slow-wave (stages 3 and 4) sleep. 2 patients showed arousal with vocalizations from REM sleep, but both also showed arousals from stage 2 or 4 NREM sleep on the same night	Being male and having <2% stage 4 sleep in overnight PSG yielded 89% sensitivity, 80% specificity for identifying individuals who were violent to others

DoA=disorders of arousal; EEG=electroencephalography; EKG=electrocardiogram; HSD=hysynchronous delta wave; MAS= microarousals preceeded by EEG slow-wave synchronization; NREM=non-rapid eye movement; PSG=polysomnography; REM= rapid eye movement.

Rapid eye movement behaviour disorder: The minimum criteria for rapid eye movement behaviour disorder (RBD) are elevated submental electromyography (EMG) tone and/or excessive phasic submental and/or limb EMG twitching; abnormal REM sleep behaviours during PSG (prominent limbs or truncal jerking; complex, vigorous or violent behaviours) or a history of injurious or disruptive sleep behaviours; and absence of EEG epileptiform activity during REM sleep. The findings of eight cited studies are summarized in Table 3.

Table 3: Studies on rapid eye movement behaviour disorder

Studies	Population	Approach	Findings	Comments
Schenck[312]	Case series of 4 men and 1 woman having aggressive behaviour during sleep	Overnight PSG	PSG did not detect seizures, but did show REM sleep pathology with variable loss of chin atonia, extraordinary increased limb-twitch activity and increased REM ocular activity and density	Authors suggested these REM sleep behavioural disorders constitute another category of parasomnias
Sforza[313]	6 patients with characteristic episodes of behavioural disorders during REM sleep	VPSG	PSG data showed a decrease in first REM latency, an absence of stage 4 of NREM, altered phasic motor activity and behavioural episodes during REM sleep, even with normal chin muscle atonia, which appears to be a special case of RBD	PSG diagnostic in all cases
Culebras[314]	6 patients with characteristics of RBD, referred during 2 years.	Overnight PSG, plus head MRI	PSG recordings revealed no muscle atonia and intermittent surges of muscle tone during segments of REM sleep in all patients. Aperiodic clonic activity of limb muscles and motor restlessness was prevalent during REM stages in all patients although periodic (leg myoclonus) and aperiodic muscle activity (twitches, jerks) were also observed in NREM sleep	PSG indicated EMG elevations in all patients
Schenck[315]	Retrospective study, 70 consecutive patients previously diagnosed with RBD during a 6.5 year period	PSG, sleep-wake interview, MSLT (in some cases), neurologic evaluation,	Both sleep architecture and customary cycling among sleep stages were generally well preserved; REM sleep percentage was elevated and REM sleep latency was reduced in 43% and	All PSGs demonstrated EMG elevation during REM sleep; EMG activity during NREM sleep; authors suggested

139

Studies	Population	Approach	Findings	Comments
		psychiatric evaluation	66% of the patients, respectively; REM atonia was lost in RBD patients; slow-wave (stage 3/4) sleep percentage was elevated in most patients	PSG confirmation of diagnosis RBD is required before treatment
Tachibana[316]	7 healthy elderly people, complaining of nocturnal sleepwalking behaviours; 14 controls.	PSG	PSG data revealed presence of "stage 1-REM with tonic EMG activity" (equivalent to REM sleep without muscle atonia), sometimes accompanied with abnormal behaviour in first group; NSD of sleep architecture and parameters between two groups, except presence of higher REM density in group of elders complaining of nocturnal sleepwalking-like behaviours	Small number of participants.
Schenck[317]	Retrospective study of 20 patients with complaints of injurious, sleep-related behaviours who were admitted to ICU while their parasomnias were active	PSG	PSG studies diagnosed 17 of 20 patients (85%) with REM sleep behaviour disorder (vigorous dream enactment during REM sleep and lack of evidence of seizure activity on EEG) and 3 of 20 patients (15%) with night terrors or sleepwalking (complex behaviour or abrupt arousals arising from NREM sleep)	Selected and specific population.

Studies	Population	Approach	Findings	Comments
Lapierre[318]	5 adult RBD patients and 5 normal controls	PSG; recorded tonic and phasic EMG events	PSG recordings showed tonic EMG activity occurred during >94% of total REM time in RBD patients while normal controls had no tonic EMG activity during REM sleep; REM density in RBD patients was similar to controls; sleep variables not statistically different between RBD patients and normal controls; clonazepam treatment reduced stage 3 and 4 of NREM sleep, increased REM sleep, reduced REM density and phasic submental EMG density	Controlled prospective study; small number of participants; quantitative scoring of EMG activity
Schenck[319]	96 patients diagnosed with RBD during 9 year period	Review of PSG and other findings, comparison with reported data from other centres	80% prevalence of elevated percentage of stage 3/4 (slow-wave) sleep; 61.4% periodic and 37.5% aperiodic limb movements during NREM sleep	Review updating number of RBD cases diagnosed at the sleep disorders center of authors

EMG=electromyography; ICU=intensive care unit; MRI=magnetic resonance imaging; MSLT=multiple sleep latency test; NREM=non-rapid eye movement; NSD=no significant difference; PSG=polysomnography; RBD=rapid eye movement behaviour disorder; REM=rapid eye movement; VPSG=video polysomnography.

Other parasomnias: There were three studies on other parasomnias, some associated with epilepsy, which are summarized in Table 4.

Video-EEG telemetry: The ASDA review also refers to the use of video-EEG telemetry, which gives similar EEG coverage and video capability to VPSG, without being able to record sleep staging, respiratory movements or limb movements. One study is referred to as an example in Table 5.

Table 4: Studies on other parasomnias

Studies	Population	Approach	Findings	Comments
Pedley[320]	6 patients between 17 and 32 years with unusual sleepwalking episodes characterized by screaming or unintelligible vocalizations	>1 night polygraphic monitoring plus separate EEG investigation	PSG revealed all patients had normal waking-sleep cycles, but 4 patients had epileptiform abnormalities on EEG; treatment with phenytoin or carbamazepine eliminated abnormal nocturnal behaviours	Small number of participants
Maselli[321]	12 patients (age 19 to 29 years) with episodic nocturnal wanderings characterized by frequent attacks of screaming, ambulation and complex automatisms during sleep; none had history of seizures, 3 had history of parasomnias and 4 had family members with history of parasomnias	PSG and daytime EEG	Potentially epileptiform activity in 4 patients; attacks conclusively epileptic in origin and were unaccompanied by ictal EEG patterns; anticonvulsant medication reduced or eliminated attacks in treated patients	Nocturnal distribution and frequency of attacks variable in this group and were not diagnostic
Plazzi[322]	4 patients complaining of stereotyped paroxysmal ambulation and other complex motor activities during sleep	Video-PSG	Video-PSG monitoring demonstrated ictal epileptic discharges at onset of sleepwalking episodes. All patients displayed minor motor episodes resembling short nocturnal paroxysmal dystonia attacks and paroxysmal arousals; clinical aspects represented the same epileptic syndrome with variable presentations	Small number of participants

EEG=electroencephalography; PSG=polysomnography.

Table 5: Study on video-EEG telemetry

Study	Population	Approach	Findings	Comments
Nousianen[323]	64 patients with intractable seizure disorders	Routine EEG, ambulatory EEG and intensive video monitoring	When reduction or withdrawal of antiepileptic drug used to activate seizures, ictal ambulatory EEG or intensive video monitoring recordings identified 89% (57 out of 64 patients); intensive EEG-video recordings most successful when clinical events frequent or inducible; combined routine and ambulatory EEG findings also successful in diagnosis of epilepsy and seizure classification	Intensive EEG video recordings or combination of routine and ambulatory EEG recordings suggested to be useful in diagnosis of epilepsy and seizure classification

3. CPSO, 1996[19]

The CPSO document lists suggested indications for full PSG in parasomnias as rule out sleep-related seizures, potentially violent or injurious behaviours, EDS, disruption of other household members and a history which suggests sleep apnea, to confirm the presence of REM sleep behavioural disorder and features suggestive of Munchausen's by proxy. Five review articles are cited in support.

Relevance to recommendations

The findings of most cited studies provided an indication that PSG, including video-EEG recordings, assisted in the diagnosis of parasomnias, including sleepwalking or night terrors, RBD and sleep-related epilepsy. PSG diagnostic criteria for each sleep disorder appear to be established and distinct. Drug treatment of parasomnias often yielded favourable results and PSG confirmation of the diagnosis is needed before treatment.

The quality of the cited evidence is limited. There are five small non-randomized comparative studies, not all with well matched controls and 22 case series, most with few patients.

APPENDIX 21: Identified Guidelines and Recommendations Circadian Rhythm Disorders

1. ASDA, 1997[23,39]

Recommendation
- PSG is not routinely indicated for the diagnosis of circadian rhythm sleep disorders.

2. CPSO, 1996 [19]

Recommendation
- PSG is useful when the diagnosis is persistent circadian rhythm disorders, such as delayed phase syndrome. However, sleep diaries and actigraphy may be more economic in the assessment of chronic circadian rhythm disturbances.

3. Canadian Sleep Society/Canadian Thoracic Society, 1996[10]

Recommendation
- Indications [for PSG] can be considered under four broad headings… disorders of the timing of the main sleep period (circadian sleep disorders).

Evidence
The ASDA review cites three studies on circadian rhythm sleep disorders where there was consideration of the diagnostic value of the testing procedure. All studies had small sample sizes and inherent population biases.[324-326] None related to the use of sleep laboratories for routine diagnosis. The ASDA states the studies that used PSG to evaluate circadian rhythm sleep disorders found it useful in identifying sleep-structure changes, but none demonstrated a value for PSG in recognizing specific circadian rhythm sleep disorders or in directing treatment.

The CPSO document refers to a recommendation in the 1995 ASDA guideline on the use of PSG in the evaluation of insomnia,[48] which follows a review by Reite *et al.*[49] Reite *et al.* suggested that PSG could be helpful in cases where a delayed sleep phase syndrome occurs with another sleep disorder. No direct evidence is cited. The CPSO conclusions on the use of sleep diaries and actigraphy are based on consensus.

No evidence is cited by the other Canadian guideline.

Relevance to recommendations
There is an absence of evidence to support the use of PSG in the management of circadian rhythm disorders.