American Academy of Sleep Medicine

Guideline Update

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The AASM published a clinical practice guideline to make important updates to the recommendations for the diagnosis of obstructive sleep apnea in the 2005 Practice Parameters for the Indications for Polysomnography and Related Procedures. These updates are based on a comprehensive review of the evidence on the diagnostic testing approaches for adults with obstructive sleep apnea with polysomnography. The clinical practice guideline is an essential update to the recommendations found in this practice parameter document:

Kapur VK, Auckley DH, Chowdhuri S, Kuhlmann DC, Mehra R, Ramar K, Harrod CG. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an American Academy of Sleep Medicine clinical practice guideline. J Clin Sleep Med. 2017;13(3):479–504.

Practice Parameters for the Indications for Polysomnography and Related Procedures: An Update for 2005

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Summary: These practice parameters are an update of the previously-published recommendations regarding the indications for polysomnography and related procedures in the diagnosis of sleep disorders. Diagnostic categories include the following: sleep related breathing disorders, other respiratory disorders, narcolepsy, parasomnias, sleep related seizure disorders, restless legs syndrome, periodic limb movement sleep disorder, depression with insomnia, and circadian rhythm sleep disorders. Polysomnography is routinely indicated for the diagnosis of sleep related breathing disorders; for continuous positive airway pressure (CPAP) titration in patients with sleep related breathing disorders; for the assessment of treatment results in some cases; with a multiple sleep latency test in the evaluation of suspected narcolepsy; in evaluating sleep related behaviors that are violent or otherwise potentially injurious to the patient or others; and in certain atypical or unusual parasomnias. Polysomnography may be indicated in patients with neuromuscular disorders and sleep related symptoms; to assist in the diagnosis of paroxysmal arousals or other sleep disruptions thought to be seizure related; in a presumed parasomnia or sleep related seizure disorder that does not respond to conventional therapy; or when there is a strong clinical suspicion of periodic limb movement sleep disorder. Polysomnography is not routinely indicated to diagnose chronic lung disease; in cases of typical, uncomplicated, and noninjurious parasomnias when the diagnosis is clearly delineated; for patients with seizures who have no specific complaints consistent with a sleep disorder; to diagnose or treat restless legs syndrome; for the diagnosis of circadian rhythm sleep disorders; or to establish a diagnosis of depression.

Key Words: Practice parameters; Practice guidelines; Standards of practice; Polysomnography; Sleep related breathing disorders; Sleep disorders; Narcolepsy; Parasomnias; Restless legs syndrome; Periodic limb movement sleep disorder; Insomnia; Circadian rhythm sleep disorders.

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1.0 INTRODUCTION

IN 1997, THE AMERICAN ACADEMY OF SLEEP MEDICINE (AASM, FORMERLY THE AMERICAN SLEEP DISORDERS ASSOCIATION [ASDA]) published practice parameters for polysomnography (PSG) and related procedures.[1] The conditions addressed included sleep related breathing disorders, other respiratory disorders, narcolepsy, parasomnias and sleep related seizure disorders, restless legs syndrome and periodic limb movement sleep disorder, depression with insomnia, and circadian rhythm sleep disorders. Since that time awareness of sleep disorders has grown. For example, in 1990 there were 110,000 office visits for sleep apnea. By 1998, this had risen to 1.3 million per year. The diagnosis of some sleep disorders requires objective documentation with PSG.

Prior to 1997, the AASM had not published recommendations on most of the individual conditions that were addressed in the original practice parameters for PSG. Since then, there have been several AASM practice parameter publications that overlap with the focus of the recommendations of this paper. When such overlap occurs, this update will reference the relevant recommendations. The purpose of this update is to reissue, modify and, if necessary, replace recommendations for indications for PSG and related procedures based on the scientific literature published since 1997. The numbers in brackets refer to the sections in the review paper[2] that accompanied the original practice parameter paper.

2.0 METHODS

The Standards of Practice Committee of the AASM reviewed the indications for polysomnography in the diagnosis of commonly encountered sleep disorders. On the basis of the review, the

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Standards of Practice Committee (SPC) developed the recommendations included in this paper. These recommendations mainly pertain to adults, since the indications for PSG in the diagnosis of sleep disorders in pediatric patients may be different. Nevertheless, the recommendations for some sleep disorders, such as parasomnias and sleep related seizure disorders, are applicable to adult, adolescent, and pediatric patients. In most cases, the recommendations are based on evidence from studies published in peer-reviewed journals. However, where scientific data are absent, insufficient, or inconclusive, recommendations are based upon task force consensus. The Board of Directors of the AASM approved these recommendations.

All members of the SPC and the Board of Directors completed detailed conflict-of-interest statements. The participants in this process may be directors or members of sleep disorders centers and recognize that they participate in sleep-center based studies. However, many additionally have substantial experience with the use of ambulatory equipment for sleep studies. Otherwise, conflicts-of-interest with regard to the actions of the SPC and the Board were not felt to be present. These practice parameters define principles of practice that should meet the needs of most adult patients in most situations. These guidelines should not be considered inclusive of all proper methods of care or exclusive of other methods of care reasonably directed to obtaining the same results. The ultimate judgment regarding the propriety of any specific care must be made by the clinician in light of the individual circumstances presented by the patient and the availability of diagnostic and treatment options and resources.

The AASM expects these guidelines to have a positive impact upon the practice of sleep medicine, patient treatment outcomes, and health care costs. These practice parameters reflect the state of knowledge at publication and will be reviewed, updated, and revised as new information becomes available.

A literature search was conducted for each of the sleep disorders described in this paper. For each sleep disorder, the methodology for the literature search, review of the literature, and grading of the evidence are discussed in the sections entitled, "Clinical indications for polysomnography and other sleep medicine procedures." Articles were assigned an evidence level based on Table 1. Evidence tables were developed for this paper; they list articles with evidence Levels I and II. Recommendations are designated as either standard, guideline, or option (Table 2).

3.0 BACKGROUND

In its 1992 assessment of PSG, the Agency for Health Care Policy and Research concluded that all PSG testing may not require the in-laboratory measurement of every one of the typical parameters.[5] Because it did not have sufficient peer-reviewed evidence to recommend tests other than standard PSG, however, the agency suggested that further research would be necessary to elucidate any situations in which testing other than in-laboratory standard PSG would be appropriate. The Board of Directors of the ASDA (now the AASM) charged a task force with reviewing the evidence (that was published both before and after the Agency for Health Care Policy and Research recommendations were made) and with formulating recommendations based upon that evidence. The subsequent sections of this paper present recommendations and highlight limitations and areas in need of further study. Section 4.0 (Diagnosis-Based Recommendations) summarizes the evidence-based indications for PSG in various clinical conditions. General evaluation procedures, additional validated stratification factors, clinical indications for the use of sleep testing procedures, alternative tools, and specific technical considerations for sleep medicine procedures are presented for each disorder. Section 5.0 discusses areas for future research.

4.0 DIAGNOSIS-BASED RECOMMENDATIONS

Unless otherwise specified, these recommendations refer to attended PSG and attended portable (Type 3) cardiorespiratory sleep studies.

4.1 Sleep related breathing disorders

Abnormal breathing events commonly encountered in sleep include snoring, apneas, hypopneas, and respiratory effort related arousals (RERAs). Over the last decade, the evolution of the technological means of measuring airflow and other respiratory parameters combined with the changing understanding of the pathophysiology of sleep related breathing disorders (SRBDs) has resulted in various definitions of hypopneas and RERAs. Since the previous practice parameter of 1997, a workshop was convened to promote consensus regarding definition of these respiratory events during sleep.[6] The workshop sought to reach definitions that could be agreed upon for ongoing research efforts. The recommended definitions for apneas, hypopneas, and RERAs are detailed in Table 3. These represent consensus opinions, and these definitions are not universally accepted. When reviewing data for this paper, definitions for events used in individual publications were accepted for review.

Since apneas, hypopneas, and RERAs are seen, albeit uncommonly, in normal sleepers, SRBDs are syndromes where the frequency of the breathing events noted above are pathophysiologically linked to symptoms or adverse health outcomes. These

Table 1—AASM classification of evidence, with subscript:

Recommendation Grades	Evidence Levels	Study Design
A	I	Randomized well-designed trials with low alpha and beta error*
B	II	Randomized trials with high alpha and beta error*
C	III	Nonrandomized concurrently controlled studies
C	IV	Nonrandomized historically controlled studies
C	V	Case series

Adapted from Sackett[3]

*Alpha error refers to the probability (generally set at 95% or greater) that a significant outcome (e.g., $p<0.05$) is not a result of chance occurrence. Beta error refers to the probability (generally set at 80% to 90% or greater) that a nonsignificant result (e.g., $p>0.05$) is the correct conclusion of the study or studies. The estimation of beta error is generally the result of a power analysis. The power analysis includes a sample size analysis to project the size of the study population necessary to ensure that significant differences will be observed if actually present.

include Obstructive Sleep Apnea Syndrome (OSA), Central Sleep Apnea Syndrome (CSA), Cheyne-Stokes Respiration (CSR), and Alveolar Hypoventilation Syndrome (AHS).[6] The frequency of apneas and hypopneas per hour of sleep is expressed as the "apnea-hypopnea index" or the AHI (number of apneas plus hypopneas per hour of sleep). The respiratory disturbance index (RDI) has at times been used synonymously with AHI, but at other times has included the total of apneas, hypopneas, and RERAs per hour of sleep. When a portable monitor is used that does not measure sleep, the RDI refers to the number of apneas plus hypopneas per hour of recording. Finally, the total number of arousals per hour of sleep from apneas, hypopneas, and RERAs is the respiratory arousal index.

In OSA and Upper Airway Resistance Syndrome (UARS), an increase in respiratory effort to breathe against relative or absolute airway obstruction is identified by measuring an intrathoracic pressure (usually inferred from other signals) that is more negative than during non-obstructed breathing. In CSA, recurrent apneas during sleep occur in the absence of upper airway obstruction due to lack of effort. Patients with CSR show cyclic fluctuation in ventilatory pattern during sleep with periods of central apnea or hypopnea alternating with hyperpnea in a crescendo and decrescendo manner. Finally, patients with AHS have an abnormal increase in PaCO2 and hypoxemia during sleep due to inappropriate central hypoventilation and not due exclusively to obstructive apneas and hypopneas. Diurnal hypercapnia is often present.

The present reference or "gold" standard for evaluation of sleep and sleep related breathing is the polysomnogram (PSG). Possible forms of error involved in the measurement of sleep and breathing with polysomnography include data loss, artifact, intra- and inter-rater event recognition errors, and measurement errors. Since the PSG is considered the reference standard, the reliability and technical accuracy of PSG is generally accepted without question. However, PSG, even when accurately measured, recorded, and analyzed, may misclassify patients based upon night-to-night variability in measured parameters, the use of different types of leads that may lead to over- or underestimation of events (e.g., use of thermistors vs. nasal cannula), and the vagaries of the clinical definitions of disease. For example, estimates of the sensitivity of one night of PSG to detect an AHI $\geq$ 5 in patients with OSA range between 75 to 88%.[7-12]

4.1.1 General evaluation

The evaluation should include a thorough sleep history and a physical examination that includes the respiratory, cardiovascular, and neurologic systems. Although the examiner must pay particular attention to observations regarding snoring, apneas, nocturnal choking or gasping, restlessness, and excessive daytime

Table 2—AASM levels of recommendations

Term	Definition
Standard	This is a generally accepted patient-care strategy, which reflects a high degree of clinical certainty. The term standard generally implies the use of Level I Evidence, which directly addresses the clinical issue, or overwhelming Level II Evidence.
Guideline	This is a patient-care strategy, which reflects a moderate degree of clinical certainty. The term guideline implies the use of Level II Evidence or a consensus of Level III Evidence.
Option	This is a patient-care strategy, which reflects uncertain clinical use. The term option implies either inconclusive or conflicting evidence or conflicting expert opinion.

Adapted from Eddy.[4] Reprinted with permission from the American College of Physicians.

Table 3—Definitions of Breathing Events During Sleep

Obstructive Apnea	**Clinical Definition:** Apnea is defined as a cessation of airflow for at least 10 seconds. The event is obstructive if during apnea there is effort to breathe. **Research Definition*:** A clear decrease (>50%) from baseline in the amplitude of a valid measure of breathing during sleep lasting at least 10 seconds (note, little difference made between obstructive apnea or hypopnea)
Central Apnea	**Clinical Definition:** Apnea is defined as a cessation of airflow for at least 10 seconds. The event is central if during apnea there is no effort to breathe. **Research Definition*:** Same as above, but an esophageal balloon must verify lack of effort.
Mixed Apnea	**Clinical Definition:** Apnea is defined as a cessation of airflow for at least 10 seconds. The event is mixed if the apnea begins as a central apnea, but towards the end there is effort to breathe without airflow.
Hypopnea	**Clinical Definition:** Several clinical definitions of hypopnea are in clinical use and there is no clear consensus. A Centers for Medicare and Medicaid Services (CMS)-approved definition of hypopnea is an abnormal respiratory event with at least a 30% reduction in thoracoabdominal movement or airflow as compared to baseline lasting at least 10 seconds, and with $\geq$4% oxygen desaturation. Obstruction is often inferred from thoracoabdominal paradox, the shape of the flow signal, or when snoring intensity increases during the event. **Research Definition*:** A clear amplitude reduction of a validated measure of breathing during sleep (but less than a 50% reduction from baseline) that is associated with an oxygen desaturation of >3% or an arousal. Only an esophageal balloon can demonstrate the hypopnea to be obstructive vs. central.
Respiratory-Effort Related Arousal (RERA)	**Clinical Definition:** Not agreed upon. **Research Definition*:** Sequence of breaths with increasing respiratory effort leading to an arousal from sleep, as shown by progressively more negative esophageal pressure for at least 10 seconds preceding an arousal with resumption of more normal pressures.

From The Report of an American Academy of Sleep Medicine Task Force.[6]

sleepiness, other aspects of a sleep history cannot be neglected since many patients suffer from more than one sleep disorder (e.g., a concurrent SRBD and restless legs syndrome). In addition, some medical conditions have been associated with increased risk for SRBDs, such as obesity, hypertension, stroke, and congestive heart failure. Because PSG may be used for diagnosis and for titration or evaluation of various treatment modalities, the general evaluation should serve to establish a differential diagnosis of SRBDs, which can then be used to select the appropriate test(s). The general evaluation should therefore take place before any PSG is performed.

4.1.2 Additional validated stratification factors

4.1.2.1 Snoring, sleepiness, obesity, and witnessed apneas

Snoring occurs in up to 30-50% of adults over the age of 50 years, and subjective sleepiness occurs in more than 30% of adults.[13,14] Although snoring and excessive sleepiness in SRBDs are common, not all snorers or sleepy adults have a sleep disorder. In one study involving retrospective analysis of 250 consecutive referrals to a sleep disorders center, snoring was strongly associated with the presence of OSA, but had a positive predictive value (PPV) and negative predictive value (NPV) of only 0.63 and 0.56, respectively.[15] The diagnostic value of witnessed apneas and hypersomnia were evaluated in a study of 380 patients referred for sleep study with problem snoring.[16] Fifty-four percent of the 380 had OSA (AHI > 15). The PPV and NPV for these symptoms in predicting the presence of OSA, separately or together, ranged between 0.40 and 0.60. Pouliot et al.[17] evaluated self rated sleepiness, body mass index (BMI), and witnessed apneas alone or in combination to identify patients with an apnea index < 20. Using the Epworth Sleepiness Scale (ESS) for sleepiness, the area under the Receiver Operating Characteristic (ROC) curve was only 0.56, indicating very poor discriminative value. The area under the ROC for BMI was 0.72, and combining all three factors (BMI, ESS, and witnessed apneas) improved specificity, but the numbers were very small and confidence limits were not reported. Similarly, the Sleep Heart Health Study also found SRBDs associated with self-reported snoring and breathing pauses and that prevalence of patients with AHI $\geq$ 15 increased with increasing categories of snoring frequency, loudness of snoring, and breathing pauses. However, multiple regression modeling found that snoring of moderate or habitual severity had an odds ratio of only 1.28-2.87 for identifying patients with AHI $\geq$ 15. While male gender, age, and snoring were associated with AHI, individually the associations were far from predictive of OSA.[14] These findings are in agreement with the bulk of the literature evaluating clinical features of patients with OSA.[14-23] Additionally, several studies have documented sensitivities and specificities of clinical impression from experienced clinicians as 52-60% and 65-70%, respectively.[24, 25] In summary, clinical impression alone or categorization based upon symptoms alone lacks the accuracy needed to diagnose SRBDs, and objective testing is still needed.

4.1.2.2 Other factors, clinical prediction rules, and neural networks

Refinement of the use of clinical variables using multiple regression analysis or bootstrap statistical methodology has produced several clinical prediction models that may refine the estimate of likelihood of having OSA (1 Level I, 2 Level II, and 2 Level III studies). In grading the quality of evidence, higher levels were assigned to studies that evaluated models prospectively (highest), on data sets separate from the derivation set (minimal required for Level of II), or those with large numbers. In one study, four previously published clinical prediction models were prospectively evaluated for their ability to categorize prospective consecutive patients by threshold AHI and found sensitivities of 76-96% and specificities of only 13-54%.[26] Positive predictive values ranged between 69-77%, and even when optimized to detect the more severe AHI (AHI $\geq$ 20), had sensitivities of 85-98% but specificities of 33-39%.[26] Another study derived a clinical prediction rule on data collected consecutively on 102 patients, and then tested the rule on an additional 108 patients. The accuracy of their optimized model reached only 53%, with a PPV of 86.7% and NPV of 36.7%.[19] The addition of oximetry data did not appreciably affect accuracy. In the study of Deegan et al.,[15] a model combining the clinical features with oximetry data only correctly classified 32.4% of patients with (defined as AHI $\geq$ 15) or without OSA.

Clinical prediction models have also been formulated utilizing combinations of clinical data and measurements of pulmonary function, craniofacial measurements, or oximetry data (3 level 1, 1 level 2 studies).[19,20,27] Kushida et al.[27] derived a model using upper airway and body measurements from a small test population, and then prospectively tested the model's accuracy in an additional 300 patients evaluated with PSG. The model had a sensitivity of 97.6%, specificity of 100%, with PPV and NPV of 100% and 88.5% respectively. They tested and demonstrated excellent inter-rater reliability for the measurements, and in their test population, only 6 patients would have received a "false negative" categorization. In those 6/300 patients, the mean AHI was found to be 7.4. The study results appear to merit additional prospective testing at alternate locations to verify reproducibility. An algorithmic approach using first a validated sleep questionnaire and then oximetry data was developed retrospectively in 80% of a test population and validated in the remaining 20% of the test population and found to have good sensitivity, specificity and predictive values, but the oximetry was not ambulatory and was concurrent with the reference standard of PSG recording, and there was no prospective evaluation performed.[20] A model using pulmonary function data and BMI was developed on a test population of 168 patients and then prospectively evaluated in 101 consecutive patients being tested for OSA.[22] The PPV was 86% and NPV was 100%. However, the test population was restricted to those without history of alcoholism, regular use of hypnotics, patients with upper respiratory tract disorders, cardiopulmonary disease, airway obstructive disease, or neuromuscular disease.

Neural networks are computer applications that attempt to mimic the biological nervous system in analyzing volumes of data and forming a conclusion. Their application is useful in complex problems because they can analyze a large number of linear and nonlinear variables without the application programmer knowing or making assumptions about the relationships between the variables. Neural networks are "trained" by analyzing reference or training data set together with the outcomes that the trainer wishes the network to learn. The trained neural network can then be evaluated by inputting similar, but previously unseen, data. One neural network trained on data from 255 patients referred for evaluation of OSA was then tested on data from an additional 150 patients.[28] The mean accuracy of the network was 91.3% for both ruling in and ruling out OSA. Another neural network model

demonstrated excellent diagnostic discrimination (area under the ROC curve ≥ 0.93) for AHI thresholds of 10, 15, or 20.[29] However, neither model has been tested in a prospective fashion or at other sites. Neural networks do have the potential to improve accuracy or adapt to changes in measurement or event classifications over time, and seem promising for future use in refining clinical impressions prior to any needed testing.

4.1.2.2.1 Heart disease

Since the last practice parameter paper, several studies demonstrate that there is a high prevalence of SRBDs in patients with moderate-to-severe congestive heart failure.[30,31] Furthermore, asymptomatic left ventricular dysfunction has also been associated with increased prevalence of SRBDs.[31]

Identification of the existence of sleep apnea in a patient with cardiovascular disease is important for several reasons. First, treatment of sleep apnea itself may have benefits to the patient's sleep and quality of life.[32] Second, there is some evidence that the presence of Central Sleep Apnea or Cheyne-Stokes (CSA/CSR) signifies a worse prognosis.[33] Thus, a segment of patients deserving more intensive treatment may be identified by PSG. Third, there is evidence that treatment of OSA[34] or CSA/CSR type in patients with heart failure[35] may improve cardiac function. There is some evidence that treatment of patients with CSA/CSR and congestive heart failure (CHF) may improve survival.[36] Patients with CSA/CSR may report nocturnal dyspnea, orthopnea, or paroxysmal nocturnal dyspnea, symptoms which may be inappropriately attributed to CHF alone. Thus identification of SRBDs with PSG in patients with CHF has important implications.

4.1.2.2.1.1 Congestive heart failure

Several studies evaluated the frequency with which SDB is present in patients with systolic or diastolic CHF (1 Level I, 2 Level II, and 2 Level III studies). A limitation of many of the studies is that the method of selection was not specified. Javaheri and coworkers[30] prospectively evaluated 45 patients with stable congestive heart failure (ejection fraction [EF] < 45%). A history of sleep apnea complaints was not solicited. The patients underwent PSG and 45% had an AHI > 20/hr. The majority of abnormal breathing events in this group were central in nature. Of note, none of the patients were on beta-blockers. This study provided some of the first evidence of a high prevalence of SRBDs in CHF. However, as selection methods were not specified, selection bias is possible. Sin et al.[31] retrospectively evaluated 450 patients with systolic CHF referred to the sleep laboratory for either sleep complaints or continued dyspnea despite effective medical management. SRBDs were very common in patients with symptomatic CHF, present in 75% of the men and 47% of the women. The predictive factors for the presence of CSA were male gender, the presence of atrial fibrillation, daytime hypocapnia, or age > 60 years. The predictive factors for OSA included an increased BMI (BMI > 35) in men and age > 60 in women. Chan and coworkers[37] evaluated 20 patients with symptomatic diastolic CHF for the presence of SRBD. Echocardiography was used to define diastolic dysfunction. All patients had an EF > 45%. In this study 55% of the patients were found to have SRBDs defined by an AHI > 10/hr. The selection methods were not explicitly stated.

Sanner and coworkers[38] evaluated a group of patients suspected of having OSA for right ventricular impairment (RVI) defined as a right ventricular ejection fraction less that 45%. Patients with significant lung disease were excluded. RVI was present in 18%. Of note some of the patients with RVI did not have evidence of pulmonary hypertension. This study provides some evidence that patients with unexplained RVI should be evaluated for OSA. However, a study of a large group of patients with unexplained RVI is needed to determine how often OSA is present.

Tremel and coworkers[39] evaluated 46 consecutive patients who presented with pulmonary edema. They were studied after stabilization (sleep studies were performed one and two months after medical stabilization). In these populations 82% had SRBDs. Of those with SRBDs, 75% had CSA and 25% had OSA.

In summary, evidence suggests that a high percentage of patients with moderate to severe CHF have SRBDs. In this population, CSA is more common that OSA. CSA appears more common in those with atrial fibrillation and in men.

4.1.2.2.1.2 Coronary artery disease

Only a few studies have addressed the question of the prevalence of SRBDs in patients with coronary artery disease (CAD) (1 study each of Levels II, III, and IV). Andreas and coworkers[40] randomly selected patients with coronary artery disease undergoing angiography. Patients with a reduced EF were excluded. All 50 patients underwent a limited sleep study. Of these 25/50 had an apnea index > 10/hr. Of the 25, 19 had a complete sleep study and had a mean AHI of 32.4 events/hour. The method of randomly selecting patients for inclusion was not specified. Morruzi et al.[41] evaluated 3 groups: group 1 had an acute MI, group 2 had unstable angina that had undergone stabilization, and group 3 had stable coronary disease. The number of patients with SRBD defined as an AHI > 10/hr were group 1 (22%), group 2 (36%), and group 3 (9%). Interestingly in the patients with an AHI > 10/hr, the majority of events were central apneas. Sanner and coworkers[42] studied 68 consecutive patients referred because of possible angina. Of these patients 21 (31%) had an AHI > 10/hr. SRBDs were found to be present in 21% of the patients with CAD and 42.1% of those not found to have CAD (difference not statistically significant).

These data suggest that patients with coronary artery disease should be particularly questioned for symptoms and signs of SRBDs. If there is even the slightest suspicion of sleep apnea, the patients should undergo a PSG.

4.1.2.2.1.3 Significant arrhythmias

Fries and coworkers[43] evaluated 40 consecutive patients who had placement of an automatic internal cardioverter defibrillator. SDB defined as an AHI > 10/hr was present in 16/40 (40%). Of the 16 with SRBDs there were 9 with CSA and 7 with OSA. The patients were followed for two years. Of the patients with CSA, 4/9 died but none (0/9) with OSA died. The etiology of expiration in all cases was nonsudden cardiac events. Stegman et al.[44] evaluated eight patients referred for pacemaker evaluation secondary to brady-arrhythmia who also had symptoms of OSA. Of the eight patients studied, seven had OSA. An observational study by Mooe et al.[45] followed 121 consecutive patients from time of coronary artery bypass to discharge. They found that SRBDs, defined by an oxygen desaturation index or RDI (Type 3 portable monitor) ≥ 5, were associated with nearly twice the occurrence of atrial fibrilla-

tion requiring pharmacological intervention or cardioversion. Untreated OSA patients have a higher risk of recurrence of atrial fibrillation after successful cardioversion than patients without known sleep apnea.[46] OSA patients treated with CPAP had a significant reduction in arrhythmia recurrence, which was independent of age, BMI, hypertension, or diabetes. These limited studies suggest that a significant proportion of groups with tachy- or bradyarrhythmias may have SRBDs (for bradyarrhythmia, 1 Level IV study; for tachyarrhythmia, 2 Level II and 1 Level IV study). However, more studies in large populations need to be conducted, and at present it is not clear what the prevalence of SRBDs are in such patients without sleep complaints.

Patients referred for evaluation of significant tachyarrhythmia or bradyarrhythmia should thus be questioned about symptoms of SRBDs. A PSG is indicated if questioning results in a reasonable suspicion that OSA or CSA are present.

4.1.2.2.1.4 Stroke

Several studies have evaluated the prevalence of SRBDs in patients who have suffered ischemic or hemorrhagic stroke.[47-56] Most studies used a threshold of AHI > 10 and found SDB in 38-95% of patients. There were no Level I or II studies assessing the prevalence of AHI > 10, but considering only Level IV studies, the prevalence was 70.0 ± 1.41 (mean ± SD), and when considering only Level V studies the prevalence was 69.6 ± 17.26.

One Level IV study[57] and four Level V studies[58-61] evaluated the effect of SRBDs on prognosis. Harbison et al.[61] noted in a case series of 78 patients that pre-existing white matter disease was present in 63% of those presenting with acute stroke. The mean AHI in those with white matter disease was 35, whereas mean AHI was 23 in those without white matter disease ($p < 0.01$), suggesting that OSA may contribute to neuronal destruction over a chronic exposure. Two Level V studies[55,58] evaluated neurologic outcomes in patients with recent stroke and AHI > 10. Both studies found that SRBDs were associated with early deterioration in neurological status (delirium or depression). However, one of these studies found patients with SRBDs to have less independence with activities of daily living,[55] but the other did not find a difference in functional outcome at 6 months after stroke.[58] One Level V study[60] with incomplete use of PSG found that an oxygen desaturation index greater than ten correlated with higher one year mortality and poorer functional outcomes.

Finally, one Level IV population based study[57] involving 6,424 free-living individuals undergoing unattended PSG found that the relative odds (95% CI) of self-reported stroke (upper versus lower AHI quartile) was 1.58 (1.02- 2.46), and the lower limit of the upper quartile for AHI was 11.0.

From the available data, it seems that SRBDs are both a risk factor for and are common in patients with stroke, and that it may have adverse impact on survival and prognosis. Pathophysiologically, SRBDs have been hypothesized to influence the course of cerebrovascular disease via a variety of mechanisms, including the influence of recurrent hypoxemia and respiratory events on hypertension, increased platelet aggregation, decreased fibrinolysis, endothelial dysfunction, increases in intracranial pressure, decreases in cerebral blood flow, and localized brain ischemia.[49]

Evaluation and treatment of patients with CPAP may be more difficult due to delirium in the acute setting, and influence on outcomes are not certain.[62-64] In the rehabilitation setting, CPAP may be more easily tolerated and has resulted in improvement in visual analogue measures of well-being and mean nocturnal blood pressure.[64]

4.1.2.3 Portable monitoring devices

Practice parameters regarding the use of portable monitors (PM) were recently published.[65] Using a categorization of sleep monitoring procedures in which Type 1 is standard attended in-lab PSG, PMs are categorized into 3 types: Type 2 - comprehensive portable polysomnography; Type 3 - modified portable sleep apnea testing (also referred to as cardiorespiratory sleep studies); and Type 4 - continuous single or dual bioparameter recording. Type 2 PM devices are lacking evidence to recommend their clinical use at this time. Type 3 PM devices used in an attended setting may increase or decrease the probability that a patient has an AHI > 15, and may rule in or rule out OSA when conducted on suitable patients and interpreted by manual scoring by trained personnel. Appropriate patients for this use should be free from significant comorbid conditions, and symptomatic patients with negative PM studies should undergo attended PSG to truly exclude OSA. Use of Type 3 PM devices is not recommended in the unattended setting at this time. Type 4 PM devices were not recommended in either attended or unattended settings for diagnosis of OSA.[65]

Ambulatory overnight pulse oximetry is a Type 4 PM device. The sensitivity, specificity, likelihood ratios, and strength of evidence were recently reviewed.[66] Parameters derived from ambulatory oximetry have been reported with both OSA and CSA.[9,67-71] However, the utility of ambulatory oximetry varies depending on equipment, analysis methods, and patient population, and routine use is not recommended.[65]

4.1.3 Clinical indications for polysomnography and other sleep medicine procedures

Reports of PSG results should routinely include as a minimum the key items listed in Table 4. These include sleep, ECG, respiratory, and periodic leg movement data.

The recommendations for use of PSG in the diagnosis of SRBDs have been updated from the 1997 practice parameter. There is additional information available for use of unattended studies, titration of PAP therapy, and an increased awareness of the role of SRBDs in patients with cardiovascular disease and stroke, which were reviewed above. A MEDLINE search was conducted using the terms polysomnography or polysomnogram or sleep study, and limited to English language articles published from January 1996 through February 2003 (yield 3464 citations). The obtained articles were further limited to those with search terms as indicated:

1) For the indications of PSG in diagnosing SRBDs (key words of snoring or Sleep Apnea Syndromes or Obstructive Sleep Apnea Syndrome) and (key words of sensitivity or specificity or positive predictive value or negative predictive value) yielded 149 citations. We eliminated any series of less than 20 patients, reviews, case reports, and citations that did not directly relate to the diagnosis of SRBDs. Eliminating such studies returned 25 citations, 21 dealing with non-polysomnographic or portable monitoring tests related to the diagnosis of SRBDs. These were not reviewed, since this topic was the focus of a recent evidence-based review and practice parameter paper.[65,66] This left 4 citations that directly related to the role of PSG in diagnosing SRBDs.[10,47,72,73] These

Table 4—Key Items for Polysomnographic Reports

Recorded Parameters:	**Central Monopolar Recording**
	Occipital Mono- or Bipolar Recording
	Chin EMG
	R/LAT
	ROC and LOC
	EKG
	Snoring MIC
	Nasal/Oral Airflow
	Thoracic Effort
	Abdominal Effort
	SaO2
	Body Position
Lights Out	
Lights On	
Total Recording Time	
Total Sleep Time	
Total Sleep Period Time	
Sleep Efficiency Index (Total Sleep Time / Total Recording Time)	
Sleep Latency (first epoch of any sleep)	
Latency to Persistent Sleep (typically first 10 minutes of uninterrupted sleep)	
REM Sleep Latency (from sleep onset)	
Wake-Corrected REM Sleep Latency (from sleep onset)	
Number of Awakenings	
Wake After Sleep Onset	
Sleep Stages:	**Total time in each stage**
	Percent of total sleep time
	Latency from sleep onset
Number and Index of Obstructive/Mixed and Central Apneas[1]	
(recommended also by sleep state and body position)	
Number and Index of Hypopneas (recommended also by sleep state and body position)	
Number and Index of Obstructive/Mixed and Central Apneas Associated with Arousals	
(recommended also by sleep state and body position)	
Number and Index of Hypopneas Associated with Arousals	
(recommended also by sleep state and body position)	
Number and Index of Respiratory-Effort-Related Arousals (RERAs)[1,2]	
(recommended also by sleep state and body position)	
Apnea-Hypopnea Index (Total Apneas + Hypopneas per hour of sleep)	
(recommended also by sleep state and body position)	
Respiratory Arousal/Disturbance Index (Total Apneas + Hypopneas + RERAs per hour of sleep)	
(recommended also by sleep state and body position)	
Minimum Oxygen Saturation:	**During sleep**
	By body position
	NREM vs. REM
	Means and longest duration in NREM and REM
	Duration of SaO_2 in percentage ranges in wake, NREM, and REM
	Mean, minimum, and maximum of SaO_2 in wake, NREM, and REM
PLMS with and without arousals	
Hypnogram:	Sleep states
	Distribution of different types of abnormal respiratory events
	PLMS with and without arousals
	Oximetry trend
	body position
	CPAP or bi-level pressure trends
Summary or Impression:	**Diagnosis**
	Any EEG or EKG abnormalities
	Unusual behavior observed during study
	(e.g., snoring, nightmares, hypnopompic and hypnogogic hallucinations, sleepwalking, enuresis, bruxism, seizures, nocturnal eating, rhythmic movement disorder)

Note: Items in bold text are "essential"; items in non-bold text are "recommended".

[1]For positive pressure polysomnographic reports, the number and index of obstructive/mixed apneas, central apneas, and respiratory-effort-related arousals (RERAs) at the optimal CPAP or Bilevel pressure are recommended.

[2]A sequence of breaths characterized by increasing respiratory effort leading to an arousal from sleep, but which does not meet criteria for an apnea or hypopnea. These events must fulfill both of the following criteria: (1) Pattern of progressively more negative esophageal pressure, terminated by a sudden change in pressure to a less negative level and an arousal; (2) The event lasts 10 seconds or longer.

citations did not suggest any changes in the current practice of using PSG for the diagnosis of SRBDs, so they were not entered into evidence tables. A supplemental search was conducted from February 1, 2003 through August 15, 2004 using the identical search terms as the previous literature search (yield = 83 citations); no new articles were found that necessitated changes in the following recommendations.

2) For the indications of PSG in initiating therapy for SRBD, a practice parameter paper on titration of PAP therapy is currently being developed by the Standards of Practice Committee of the AASM.

3) For the indications for portable monitoring, including oximetry, we refer to the recent parameter paper dealing directly with that topic.[65]

4) For the stratification of risk (key words of snoring or Sleep Apnea Syndromes or Obstructive Sleep Apnea Syndrome) and (key words of sensitivity or specificity or positive predictive value or negative predictive value) or (key word of prevalence) were used. A review of the 149 citations obtained yielded 17 citations dealing with clinical prediction models or prevalence studies for SRBDs. These were further reviewed by abstracts; reviews, studies with less than 20 subjects, and studies not employing PSG were rejected for grading, leaving 12 articles for evidence grading by the task force. There are three Level I,[19,22,27] five Level II,[17,20,26,28,29] and four Level III[14-16,21] studies, of which the Level I and II studies are summarized in Table 5.

5) For the indications of PSG in evaluating patients with heart disease, a special MEDLINE search was performed from 1996 to November 2002 using the terms polysomnography or sleep tests and heart diseases; heart failure, congestive; heart failure; ventricular dysfunction, left; cardiomyopathy; congestive; arrhythmia; with emphasis on articles using concepts of sensitivity, specificity, incidence, prognosis, prediction, course, mortality, or follow-up. Articles were limited to those with human adults published in English. This yielded 110 citations. Two reviewers independently excluded reviews, case studies, series of less than 10 patients, and citations that did not directly relate to the diagnosis or therapy of sleep related breathing disorders. This left 11 articles. The review of bibliographies from these yielded an additional article for a total of 12 articles. Data was then extracted by two reviewers into standardized data collection tables and graded for evidence quality. Disagreement in evidence grading was referred to a third examiner and the majority opinion was accepted. Two task force members analyzed each article for design, inclusion and exclusion criteria, outcome measures, biases, and conclusions. There are one Level I,[39] five Level II,[31,33,38,42,43] four Level III,[30,37,41,74] and two Level IV[40,44] studies, of which the Level I and II studies are depicted in the Table 6. A supplemental search was conducted from November 1, 2002 through August 15, 2004 using the identical search terms as the previous literature search (yield = 44 citations); no new articles were found that warranted inclusion in the evidence tables or necessitated change in the following recommendations.

6) For the indications of polysomnography in evaluating patients with stroke, a special MEDLINE search was performed from 1996 to August Week 4 2002 using cerebrovascular accident or stroke and sleep disorders; sleep apnea, central; sleep apnea, obstructive; or sleep apnea syndromes; with emphasis on articles using concepts of sensitivity, specificity, incidence, prognosis, prediction, course, mortality, or follow-up (yield = 85 citations). These were then restricted to those retrieved with the key word polysomnography and limited to those with human adults published in English (yield = 42 citations). Two reviewers independently excluded reviews, case studies, series of less than 5 patients, and citations that did not directly relate to the diagnosis or therapy of SRBDs (yield = 14 citations) for analysis of study design, inclusion and exclusion criteria, outcome measures, biases, and conclusions. There are one Level III,[75] four Level IV,[50,57,62,76] and thirteen Level V[48,49,51-56,58-61,77] studies. Since there were no Level I or II studies, no evidence tables are included. A supplemental search was conducted from August 1, 2002 through August 15, 2004 using the identical search terms as the previous literature search (yield = 70 citations); no new articles were found that necessitated changes in the following recommendations.

4.1.3.1 Polysomnography is routinely indicated for the diagnosis of sleep related breathing disorders. (Standard)

This recommendation is a modification of the recommendation of the previous practice parameter paper to include the distinction in the use between attended and unattended cardiorespiratory sleep studies, and the possible need for a second diagnostic PSG night.

1) Full-night PSG is recommended for the diagnosis of SRBDs [4.3.2.3.3].

2) For patients in the high-pretest-probability stratification group (see Sections 4.1.2.1, 4.1.2.2, and 4.1.3(4)), an attended cardiorespiratory (Type 3) 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. In the unattended setting, or in patients without qualifications of a high pretest probability stratification, the data does not support the use of these devices. (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 as well [4.3.2.2, 4.3.2.3.5.1]).

3) In patients where there is strong suspicion of OSA, if other causes for symptoms have been excluded, a second night of diagnostic PSG may be necessary to diagnose the disorder.

4.1.3.2 Polysomnography is indicated for positive airway pressure (PAP) titration in patients with sleep related breathing disorders. (Standard)

This recommendation is a modification of the recommendation of the previous practice parameter paper to include bi-level PAP and auto-titrating PAP (APAP),[78] in addition to continuous PAP (CPAP). The RDI criteria for CPAP titration are also updated.

1) A full night of PSG with CPAP titration is recommended for patients with a documented diagnosis of a SRBD for whom PAP is warranted [4.1, 4.3.2.1.2, 4.3.2.3.3, 4.3.2.3.5.1, 4.3.3]

2) PSG with CPAP titration is appropriate for patients with any of the following results:

a) An RDI of at least 15 per hour, regardless of the patient's symptoms.

b) An RDI of at least 5 per hour in a patient with excessive daytime sleepiness.

3) A cardiorespiratory (Type 3) sleep study without EEG recording is not recommended for CPAP titration. CPAP titration should include the ability to perform sleep staging (including

documenting rapid eye movement [REM] sleep) and to identify and treat arousals. Even when apnea is eradicated by CPAP, residual hypopneas and RERAs may require additional titration to determine optimal therapeutic pressures. These additional adjustments require EEG recording.

4) For CPAP titration, a split-night study (initial diagnostic PSG followed by CPAP 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 the following four criteria are met:

a) An AHI of at least 40 is documented during a minimum of 2 hours of diagnostic PSG [4.3.2.3.3]. Split-night studies may sometimes be considered at an AHI of 20 to 40, based on clinical judgment (e.g., if there are also repetitive long obstructions and major desaturations). However, at AHI values below 40, determination of CPAP pressure requirements, based on split-night studies, may be less accurate than in full-night calibrations.

b) CPAP titration is carried out for more than 3 hours (because respiratory events can worsen as the night progresses) [4.3.2.3.3].

c) PSG documents that CPAP eliminates or nearly eliminates the respiratory events during REM and non-REM (NREM) sleep, including REM sleep with the patient in the supine position [4.3.2.3.3].

d) A second full night of PSG for CPAP titration is performed if the diagnosis of a SRBD is confirmed but criteria b and c are not met [4.3.2.3.3].

4.1.3.3 A preoperative clinical evaluation that includes polysomnography or an attended cardiorespiratory (Type 3) sleep study is routinely indicated to evaluate for the presence of obstructive sleep apnea in patients before they undergo upper airway surgery for snoring or obstructive sleep apnea. (Standard)

This recommendation is a modification of the recommendation of the previous practice parameter paper to include all forms of upper airway surgery for snoring or OSA. An attended cardiorespiratory (Type 3) sleep study may be an acceptable alternative to full-night PSG in the circumstances described in Section 4.1.3.1(2).

4.1.3.4 Follow-up polysomnography or an attended cardiorespiratory (Type 3) sleep study is routinely indicated for the assessment of treatment results in the following circumstances: (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper. An attended cardiorespiratory (Type 3) sleep study may be an acceptable alternative to full-night PSG in the circumstances described in Section 4.1.3.1(2).

1) After good clinical response to oral appliance treatment in patients with moderate to severe OSA, to ensure therapeutic benefit.[79]

2) After surgical treatment of patients with moderate to severe OSA, to ensure satisfactory response.[80]

3) After surgical or dental treatment of patients with SRBDs whose symptoms return despite a good initial response to treatment.[80]

4.1.3.5 Follow-up polysomnography is routinely indicated for the assessment of treatment results in the following circumstances: (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper.

1) After substantial weight loss (e.g., 10% of body weight) has occurred in patients on CPAP for treatment of SRBDs to ascertain whether CPAP is still needed at the previously titrated pressure [4.3.2.1.3]

2) After substantial weight gain (e.g., 10% of body weight) 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 [4.3.2.1.3]

3) When clinical response is insufficient or when symptoms return despite a good initial response to treatment with CPAP. In these circumstances, testing should be devised with consideration that a concurrent sleep disorder may be present (e.g., OSA and narcolepsy) [4.3.2.1.3]

4.1.3.6 Follow-up polysomnography or a cardiorespiratory (Type 3) sleep study is not routinely indicated in patients treated with CPAP whose symptoms continue to be resolved with CPAP treatment. (Option)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.1.3.7 A multiple sleep latency test is not routinely indicated for most patients with sleep related breathing disorders. A subjective assessment of excessive daytime sleepiness should be obtained routinely. When an objective measure of daytime sleepiness is also required, previously published practice parameters[81] should be consulted. (Standard)

This recommendation is a modification of the recommendation of the previous practice parameter paper, to refer to the recently published practice parameters.

4.1.3.8 Patients with systolic or diastolic heart failure should undergo polysomnography if they have nocturnal symptoms suggestive of sleep related breathing disorders (disturbed sleep, nocturnal dyspnea, snoring) or if they remain symptomatic despite optimal medical management of congestive heart failure. (Standard)

This is a new recommendation. There are one Level I,[39] two Level II,[31,38] and two Level III[30,37] studies indicating an association between SRBD by PSG and heart failure.

Patients with coronary artery disease should be evaluated for symptoms and signs of sleep apnea. If there is suspicion of sleep apnea, the patients should undergo a sleep study. (Guideline)

This is a new recommendation. There are one Level II,[42] one Level III,[41] and one Level IV[40] studies indicating an association between SRBD by PSG and coronary artery disease.

4.1.3.10. Patients with history of stroke or transient ischemic attacks should be evaluated for symptoms and signs of sleep apnea. If there is suspicion of sleep apnea, the patients should undergo a sleep study. (Option)

This is a new recommendation. There are one Level III,[75] four Level IV,[50,57,62,76] and thirteen Level V[48,49,51-55,58-61,77] studies indi-

cating an association between OSA by PSG and stroke.

4.1.3.11 Patients referred for evaluation of significant tachyarrhythmias or bradyarrhythmias should be questioned about symptoms of sleep apnea. A sleep study is indicated if questioning results in a reasonable suspicion that OSA or CSA are present. (Guideline)

This is a new recommendation. There is one Level IV[44] indicating an association between OSA by PSG and bradyarrhythmia, and two Level II[43,46] and one Level IV[45] study indicating an association between OSA by PSG for tachyarrhythmia.

4.1.4 Technical considerations

4.1.4.1 The use of polysomnography for evaluating sleep related breathing disorders requires a minimum of the following recordings: EEG, EOG, chin EMG, airflow, arterial oxygen saturation, respiratory effort, and ECG or heart rate. Anterior tibialis EMG is useful to assist in detecting movement arousals and may have the added benefit of assessing periodic limb movements, which coexist with sleep related breathing disorders in many patients (9). (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.1.4.2 A cardiorespiratory (Type 3) sleep study requires a minimum of the following four channels: respiratory effort, airflow, arterial oxygen saturation, and ECG or heart rate. (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper for Type 3 attended tests.

4.1.4.3 An attended study requires the constant presence of a trained individual who can monitor for technical adequacy, patient compliance, and relevant patient behavior. (Guideline)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.1.5 Alternative tools

4.1.5.1 Oximetry lacks the specificity and sensitivity to be used as an alternative to polysomnography or an attended cardiorespiratory (Type 3) sleep study for diagnosing sleep related breathing disorders. [4.3.2.4.4] (Guideline)

This recommendation is the same as the recommendation of the previous practice parameter paper. Additionally, oximetry must be used with caution when applied to estimates of increased or decreased probability of OSA.[65]

4.1.5.2 In-laboratory studies have validated the use of attended cardiorespiratory sleep studies for the diagnosis of sleep related breathing disorders. However, only a few peer reviewed articles specifically examined unattended cardiorespiratory sleep studies. In selected circumstances—for example, for patients with severe symptoms of obstructive sleep apnea (i.e., high-pretest-probability stratification group) and when initiation of treatment is urgent and an attended study is not available—an unattended study may be an alternative based on prior recommendations.[65] However, the routine use of unattended cardiorespiratory studies (or even unattended polysomnography) cannot be supported, at least until there has been clear validation of such studies conducted without a technologist providing ongoing observations and interventions to ensure accurate recording and interpretation. Further research is needed to clarify this issue. [4.3.2.3.5.1, 4.3.2.3.5.2] (Guideline)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.1.5.3 No clinical model is recommended for use to predict severity of obstructive sleep apnea (Option)

This is a new recommendation. There are three Level I,[19,22,27] five Level II,[17,20,26,28,29] and four Level III[14-16,21] studies describing the use of various models to predict or stratify OSA severity. However, no one clinical model has been adopted for widespread clinical use.

4.2 Other respiratory disorders

This diagnostic category includes breathing disorders that are not principally defined by OSA, CSA or UARS. It includes chronic lung diseases and disorders associated with chronic alveolar hypoventilation and hypoxemia.

4.2.1 General evaluation

A clinical history and physical evaluation are needed to establish the presence and severity of the underlying medical disorder.

4.2.2 Additional validated stratification factors

There are no stratification factors generalized to all the respiratory disorders addressed in this section. However, several studies have addressed prediction of nocturnal hypoxemia and/hypoventilation in patients with chronic obstructive pulmonary disease (COPD) and neuromuscular diseases using measures of pulmonary function.

Patients with severe COPD have disrupted sleep and often desaturations during NREM sleep, and especially during REM sleep.[82-84] Wake oxygen saturation at rest is one of the best predictors of desaturation during sleep.[85,86] Nocturnal desaturation is rare when resting SaO_2 is greater than 95%.[85] Lung function is also somewhat predictive. In a population based study of over 6000 subjects, desaturation was more common in those whose ratio of FVC/FEV_1 was less than 65%,[87] and the degree of desaturation was proportional to lung function abnormality. Other factors associated with the degree of nocturnal desaturation in patients with COPD include obesity and hypercapnia. Similar factors appear to apply to patients with cystic fibrosis, with hypoxemia during sleep more common when resting wake $SaO_2 < 94\%$ and $FEV_1 < 65\%$.[88]

The prevalence of OSA among those with COPD is similar to that in the general population.[87,89] When OSA and COPD coexist, patients usually have typical symptoms of OSA.[86] However, gas exchange abnormalities may be more profound, and have led to the recognition of the "Overlap Syndrome", where obstructive lung disease combined with OSA lead to chronic respiratory failure, and when severe, to *cor pulmonale*.[90,91] An arterial blood gas shows diurnal hypercapnia.[90]

Patients with neuromuscular disease have declines in respiratory function to variable degrees.[92-95] Pulmonary function tests may

predict nocturnal hypoventilation before patients or their physicians recognize symptoms. Sleep related hypoxemia is most often observed when FVC is below 50% or when maximal inspiratory pressures are less than 60 cm H_2O.[96-98]

4.2.3 Clinical indications for the use of polysomnography

The diagnosis of respiratory disorders other than OSAS or UARS are based on clinical findings revealed through history, physical examination, chest radiography and pulmonary function tests, including arterial blood gases. This evidence-based review was established to answer the question of whether PSG, when routinely performed in patients with these diseases, added clinically significant information to care not otherwise obtainable. Although 21 articles were specifically evaluated for SRBDs in the various patient groups, PSG was not the test establishing the primary diagnosis. The prior review, along with this update, highlights that "although polysomnography has produced insights into the interaction between sleep and breathing in these disorders, polysomnography is not essential to the diagnosis of these conditions" [5.3].

The 1997 review article outlines the methods [3.1] for performing the literature search for the indications for PSG, with specific reference to other respiratory disorders [5.2]. The additional MEDLINE search terms for this section included obstructive and interstitial lung disease, asthma, neuromuscular disease, amyotrophic lateral sclerosis, hypoventilation, ankylosis, cystic fibrosis, post-poliomyelitis syndrome, physiologic monitoring, polysomnography, sleep study, spondylitis, oximetry, cardiorespiratory study. The MEDLINE search used the OVID database from 1966 to September 2002 and yielded 250,532 articles within the specified disease categories, resulting in 529 articles pertaining to the key words for monitoring. The inclusion criteria for selection of literature required that only articles from 1996 to the present would be reviewed as the prior literature was included in the 1997 review. Search restrictions further required that research be reported in English and involve humans greater than 18 years of age. The restricted search led to 85 articles. Review of abstracts eliminated 13 articles which reported on 5 or fewer subjects as per criteria established by the 1997 AASM review. The same search strategy was employed to update the data published through September 2003, yielding 83 additional references. Using the criteria above, only 1 of these 83 citations involved evaluations that had implications for use of PSG in evaluation of respiratory disorders other than OSA.

Chronic obstructive pulmonary disease

Twenty-seven articles from the updated review involved patients with COPD. Hypoxemia during sleep was the major abnormality under evaluation. Several very large studies [90,99,100] involved over 200 patients each but were not designed to answer the question posed by the Standards of Practice Committee as stated above. Chaouet et al.[82] evaluated 265 COPD patients with mild hypoxemia and found a significant number with OSA. Resta et al.[99] included patients with alveolar hypoventilation and in their separate report[100] included evaluation of bi-level PAP in their evaluation of 286 patients when they were found to be CPAP intolerant. This review will not focus on the use of bi-level PAP or other forms of nocturnal ventilation, which will be reviewed in a future practice parameters paper and do not specifically apply to the question posed. In another study by Chaouet et al.,[82] PSG was used to rule out OSA as a contributing cause of pulmonary hypertension in 94 patients with COPD. Brijker et al.[101] evaluated the advantages of PSG over routine oximetry in 14 patients. As with other studies,[102-104] PSG did not offer a significant advantage over less expensive oximetry alone in the assessment and therapy of nocturnal hypoxemia in patients with COPD. Of the remaining articles, eleven[104-114] did not specifically evaluate PSG in a manner necessary to answer the questions above.

Asthma

Sleep disturbances are quite prevalent in patients with asthma [5.3.2]. This may be related to diverse etiologies such as bronchospasm, medication side effects, or gastroesophageal reflux, to name a few. Sleep disruption has been further documented to occur in all stages of sleep and can add to daytime impairment and patient complaints. Hypoxemia may occur in these patients, but not to the degree observed in patients with COPD or cystic fibrosis. Cibella[115] and Cuttitta[116] specifically showed effects of asthma from gastroesophageal reflux in small groups of seven subjects per each report. Lin and Lin[117] reported an increased number of patients (N = 48) with asthma and OSA using methacholine challenge testing.

Other chronic lung diseases

With the improvement in care for children with cystic fibrosis, many survive to adulthood. A study[118] evaluated patients for nocturnal effects of their illness as well as oxygen vs. nocturnal mechanical ventilation. As with the prior 1997 review [5.3.5], there is little evaluation of patients with interstitial lung disease with PSG. Hira et al.[119] evaluated interstitial lung disease in 27 patients, and found that these patients, compared to controls, showed a significantly greater maximum fall in SaO_2 during sleep, more disturbed sleep, less respiratory drive during sleep, and asynchronous breathing during sleep. Observed respiratory events did not qualify for a diagnosis of a SRBD in any case.

Neuromuscular disease

There were 35 articles which dealt with patients with either neurological diseases or other musculoskeletal abnormalities. Patients often develop respiratory insufficiency associated with these disorders, especially when they reach more severe stages. This may be associated with significant sleep disruption [5.3.4], hypoxemia, alveolar hypoventilation or muscle fatigue. Furthermore, nocturnal mechanical ventilation may help alleviate symptoms and complications, making an accurate diagnosis important.[120-123] The recent review exemplifies this view with a large amount of data reputedly concerning patients with amyotrophic lateral sclerosis (ALS) and post-polio syndrome. David et al.[124] reported that in their retrospective review of 17 patients with amyotrophic lateral sclerosis, PSG was recommended (criteria were not specified). Other studies in large patient groups were not specifically PSG studies and could not address the question of indications for PSG directly.[96,108,125-131] Similarly, Bruno et al.[132] described significant sleep disruption in 7 post-polio patients. However, the study design did not allow for the evaluation of PSG. Hsu[133] evaluated a larger number (108) of patients

retrospectively, but the study was designed to describe patients with post-polio syndrome and to evaluate effects of intermittent positive pressure ventilation, not PSG. One paper compared ALS patients started on non-invasive positive pressure ventilation (NIPPV) based on ambulatory oximetry criteria with those started after ventilatory failure was evident, and found that the former group had a longer survival time after ventilator initiation.[134] Lead time biases were not addressed, numbers were small, and quality of life was not monitored.

4.2.3.1 For patients with neuromuscular disorders and sleep related symptoms, polysomnography is routinely indicated to evaluate symptoms of sleep disorders that are not adequately diagnosed by obtaining a sleep history, assessing sleep hygiene, and reviewing sleep diaries. [5.3.4] (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.2.3.2 Polysomnography is not indicated to diagnose chronic lung disease. [5.3] (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper. Nocturnal hypoxemia in patients with chronic obstructive, restrictive, or reactive lung disease is usually adequately evaluated by oximetry and does not require PSG [5.3.1, 5.3.3]. However, if the patient's symptoms suggest a diagnosis of OSA or periodic limb movement sleep disorder, indications for PSG are the same as for those disorders in patients without chronic lung disease. [4.3.2, 5.3.1, 8.3.2].

4.2.4 Technical considerations

PSG recording for evaluating breathing disorders requires a minimum of EEG, EOG, chin EMG, airflow, arterial oxygen saturation, respiratory effort, and heart rate or ECG channels. Measurement of end-tidal carbon dioxide is often very important in clarifying the patient's respiratory adequacy. Anterior tibialis EMG is useful to assist in detecting movement arousals and may also allow for the assessment of periodic limb movement sleep disorder, which may coexist with respiratory disorders in many patients.

4.2.5 Alternative tools

4.2.5.1 Nocturnal oximetry may be helpful or sufficient in assessing a disorder in which the only or principal clinical issue is the level of hypoxemia and when determining sleep stages or assessing sleep apnea is not necessary. [5.3.1] (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.2.5.2 Pulmonary function tests and arterial blood gases also may be used to help assess the patient's level of respiratory dysfunction. (Option)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.3 Narcolepsy

Narcolepsy is a neurologic disorder characterized predominantly by abnormalities of REM sleep, some abnormalities of NREM sleep, and the presence of excessive daytime sleepiness. The classic tetrad of narcolepsy symptoms includes hypersomnolence, cataplexy, sleep paralysis, and hypnagogic hallucinations, although 30-50% of patients with narcolepsy do not have all of these symptoms [6.1]. Cataplexy refers to the total or partial loss of muscle tone in response to sudden emotion. Narcoleptic patients often report disrupted sleep, and PSG often confirms fragmented sleep patterns.

The prevalence of narcolepsy is estimated at .05% in the industrialized world. It is a condition that generally begins after puberty with a peak onset in the late teens or twenties although perhaps 5% of cases have their onset prior to the age of ten. It is uncommon for narcolepsy to begin after the age of 35 but the oldest reported age of onset is 67. Over 90% of patients with narcolepsy carry the DQB1-0602 marker on HLA testing and the majority of patients with cataplexy have abnormally low levels of hypocretin-1 (orexin A) in the cerebrospinal fluid.[135]

PSG followed by a multiple sleep latency test (MSLT) is helpful in confirming the clinical impression but these tests assume greater significance if cataplexy is lacking. The PSG may show an early sleep-onset REM episode (SOREMP), i.e., a REM latency < 20 minutes. The MSLT is indicated as part of the evaluation of patients with suspected narcolepsy to confirm the diagnosis [6.3].

The usefulness of the mean sleep latency value in the evaluation of patients with possible narcolepsy is supported by evidence reported in 13 papers described in the updated practice parameter paper on the MSLT and maintenance of wakefulness test,[81] which indicated that most patients with narcolepsy have objective evidence of hypersomnia as determined by a mean sleep latency less than 5 minutes. However, MSLT data suggest that approximately 16% of patients with narcolepsy would have a mean sleep latency above the 5 minute cutoff, and approximately 16% of normal controls would have a mean sleep latency below the 5 minute cutoff.[81] The presence of two or more SOREMPs was associated with a sensitivity of 0.78 and a specificity of 0.93.[81] SOREMPs did not occur exclusively in patients with narcolepsy, and thus it is important to rule out or treat other sleep disorders before evaluating SOREMPs in the diagnosis of narcolepsy. In the absence of cataplexy and when there is one or more of the other symptoms, the laboratory criteria are required to establish the diagnosis of narcolepsy.

4.3.1 General evaluation

A clinical history, sleep diaries, PSG, and a MSLT are key items in the evaluation of narcolepsy.

4.3.2 Additional validated stratification factors

There are no additional validated stratification factors.

4.3.3 Clinical indications for the use of polysomnography and other sleep medicine procedures

The recommendations for use of PSG in the diagnosis of narcolepsy have been updated from the 1997 practice parameter. A MEDLINE search was conducted using the terms narcolepsy and indications; diagnosis; electroencephalography; polysomnography; sleep study; or overnight study; and limited to English language articles describing adult human studies published from

January 1997 through September 2001 (yield = 67 citations). We eliminated any series of less than 20 patients, reviews, case reports, and citations that did not directly relate to the diagnosis of narcolepsy. No articles were found that justified changes in the current practice of using PSG for the diagnosis of narcolepsy, so they were not entered into evidence tables. A supplemental search was conducted from September 1, 2001 through August 15, 2004 using the identical search terms as the previous literature search (yield = 108 citations); no new articles were found that necessitated changes in the following recommendations.

4.3.3.1. Polysomnography and a multiple sleep latency test performed on the day after the polysomnographic evaluation are routinely indicated in the evaluation of suspected narcolepsy [6.3]. (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.3.4 Technical considerations

4.3.4.1 The minimum channels required for the diagnosis of narcolepsy include EEG, EOG, chin EMG, and ECG. (Standard)

This recommendation is a modification of the recommendation of the previous practice parameter paper to include ECG.

4.3.4.2 Additional cardiorespiratory channels and anterior tibialis recording is recommended because obstructive sleep apnea, upper-airway resistance syndrome, and periodic limb movement sleep disorder are common co-existing conditions in patients with narcolepsy or may be independent causes of sleep fragmentation that lead to short sleep latencies and sleep-onset REM periods. The diagnosis of narcolepsy (or idiopathic hypersomnolence) requires documentation of the absence of other untreated significant disorders that cause excessive daytime sleepiness. [6.1.2, 6.3] (Option)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.3.4.3 Recommendations for the multiple sleep latency test protocol should be followed whenever possible to allow standardization of the administration of the test. (Standard)

This recommendation is a modification and combination of three of the recommendations of the previous practice parameter paper. Recommendations for the MSLT protocol were made previously by the Standards of Practice Committee of the AASM.[81]

4.3.5 Alternative tools

4.3.5.1 No alternatives to the polysomnogram and multiple sleep latency test have been validated for making the diagnosis of narcolepsy. Although the maintenance of wakefulness test may be useful in assessing treatment adequacy (by measuring the ability to stay awake), it has not been shown to be as valid as the multiple sleep latency test for confirmation of excessive daytime sleepiness and the demonstration of sleep-onset REM periods. (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.3.5.2 HLA (human leukocyte antigen) typing is not routinely indicated as a replacement for polysomnography and the multiple sleep latency test because HLA typing lacks specificity in the diagnosis of narcolepsy. Its use in providing supplementary information depends on the clinical setting. (Option)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.4 Parasomnias and seizure disorders

Parasomnias are undesirable physiologic phenomena that occur predominantly during sleep. These sleep related events can be injurious to the patient and others and can produce a serious disruption of sleep-wake schedules and family functioning. Parasomnias may reflect, be associated, or confused with, several diagnoses, including disorders of arousal from NREM sleep (confusional arousals, sleepwalking, sleep terrors), REM sleep behavior disorder, sleep related seizure disorders, and sleep related psychiatric disorders [7.1].

Epilepsy is a chronic condition characterized by the occurrence of paroxysmal electrical discharges in the brain and manifested by changes in consciousness, motor control, or sensory function. Seizures and epilepsy can be categorized into many clinical types and syndromes, often requiring different yet specific approaches to diagnosis and treatment. The term “sleep related seizure disorders” encompasses conditions with recurrent seizures during sleep, including sleep related epilepsy. In 15-20% of patients with epilepsy, seizures occur mostly or exclusively during sleep [7.1]. In the largest reported case series of difficult to diagnose paroxysmal nocturnal behaviors, approximately 50% of patients were ultimately diagnosed with sleep related epilepsy [7.3.2].

4.4.1 General evaluation

4.4.1.1 A clinical history of any parasomnia must describe and characterize the behaviors in detail with special emphasis on age of onset, time of night, frequency, regularity, and duration of episodes. (Standard)

This recommendation is a modification of the recommendation of the previous practice parameter paper to include additional items of emphasis in the clinical evaluation of parasomnias.

4.4.1.2 Common, uncomplicated, noninjurious parasomnias, such as typical disorders of arousal, nightmares, enuresis, sleeptalking, and bruxism, can usually be diagnosed by clinical evaluation alone. [7.3.1, 7.3.3] (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.4.1.3 A clinical history, neurologic examination, and a routine EEG obtained while the patient is awake and asleep are often sufficient to establish the diagnosis and permit the appropriate treatment of a sleep related seizure disorder. The need for a routine EEG should be based on clinical judgment and the likelihood that the patient has a sleep related seizure disorder. (Option)

This recommendation is a modification of the recommendation of the previous practice parameter paper by using the term “seizure disorder” instead of “epilepsy”.

4.4.2 Additional validated stratification factors

There are no additional validated stratification factors.

4.4.3 Clinical indications for polysomnography and other sleep medicine procedures

The recommendations for use of PSG in the diagnosis of parasomnias and sleep related seizure disorders have been updated from the 1997 practice parameter. A MEDLINE search was conducted using the terms parasomnia, partial arousal, or disorders of arousal and sleep studies, sleep EEG, electroencephalography, EEG, polysomnography, abnormal EEG, seizure, epilepsy, indications, diagnosis, sleep deprivation, or sleep-related, and limited to English language articles describing adult human studies published from January 1997 through September 2001 (yield 2,037 citations). We eliminated any series of less than 20 patients, reviews, case reports, and citations that did not directly relate to the diagnosis of parasomnias and sleep related seizure disorders. No articles were found that justified changes in the current practice of using PSG for the diagnosis of parasomnias and sleep related seizure disorders, so they were not entered into evidence tables. A supplemental search was conducted from September 1, 2001 through August 15, 2004 using the identical search terms as the previous literature search (yield 837 citations); no new articles were found that necessitated changes in the following recommendations.

4.4.3.1 Polysomnography, with additional EEG derivations in an extended bilateral montage, and video recording, is recommended to assist with the diagnosis of paroxysmal arousals or other sleep disruptions that are thought to be seizure related when the initial clinical evaluation and results of a standard EEG are inconclusive. [7.3.1, 7.3.3] (Option)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.4.3.2 Polysomnography, with additional EEG derivations and video recording, is indicated in evaluating sleep related behaviors that are violent or otherwise potentially injurious to the patient or others. [7.3.1] (Option)

This recommendation is a modification of the recommendation of the previous practice parameter paper to include additional EEG derivations and video recording in the PSG evaluation.

4.4.3.3 Polysomnography is indicated when evaluating patients with sleep behaviors suggestive of parasomnias that are unusual or atypical because of the patient's age at onset; the time, duration, or frequency of occurrence of the behavior; or the specifics of the particular motor patterns in question (e.g., stereotypical, repetitive, or focal). [7.3.1] (Guideline)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.4.3.4 Polysomnography 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). [7.3.1] (Option)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.4.3.5 Polysomnography may be indicated when the presumed parasomnia or sleep related seizure disorder does not respond to conventional therapy. [7.3.2] (Option)

This recommendation is a modification of the recommendation of the previous practice parameter paper by using the term "seizure disorder" instead of "epilepsy".

4.4.3.6 Polysomnography is not routinely indicated in cases of typical, uncomplicated, and non-injurious parasomnias when the diagnosis is clearly delineated. [7.3.1] (Option)

This recommendation is the same as the recommendation of the previous practice parameter paper. An example of this situation would be a 6-year-old child brought in by the parents who has occasional episodes of sleepwalking without injury.

4.4.3.7 Polysomnography is not routinely indicated for patients with a seizure disorder who have no specific complaints consistent with a sleep disorder. [7.3.2, 7.3.5] (Option)

This recommendation is a modification of the recommendation of the previous practice parameter paper by using the term "seizure disorder" instead of "epilepsy".

4.4.4 Technical considerations

4.4.4.1 The minimum channels required for the diagnosis of parasomnia or sleep-related seizure disorder include sleep-scoring channels (EEG, EOG, chin EMG); EEG using an expanded bilateral montage; and EMG for body movements (anterior tibialis or extensor digitorum). Audiovisual recording and documented technologist observations during the period of study are also essential. [7.3.4] (Option)

This recommendation is a modification of the recommendation of the previous practice parameter paper by using the term "seizure disorder" instead of "epilepsy".

4.4.4.2 Interpretation of polysomnography with video and extended EEG montage requires skills in both sleep medicine and seizure recognition. Polysomnographers and electroencephalographers who are not experienced or trained in recognizing and interpreting both polysomnographic and electroencephalographic abnormalities should seek appropriate consultation or should refer patients to a center where this expertise is available. [7.3.4] (Option)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.4.4.3 A paper speed of at least 15 mm/second and preferably 30 mm/second is recommended to enhance the recognition of seizure activity. In digital EEG recordings, the sampling rate must be adequate to identify brief paroxysmal discharges. [7.3.4] (Option)

This recommendation is a modification of the recommendation of the previous practice parameter paper to eliminate outdated digital sampling rate criteria.

4.4.5 Alternative tools

The diagnosis of a sleep related seizure disorder can often be made with EEG or video EEG recording alone. There is no alternative to PSG for the electrophysiologic diagnosis of the para-

somnias noted in Sections 4.4.3.2 and 4.4.3.3, e.g., sleep related behaviors that are violent or otherwise potentially injurious to the patient or others and parasomnias that are unusual or atypical because of the patient's age at onset; the time, duration, or frequency of occurrence of the behavior; or the specifics of the particular motor patterns in question.

4.5 Restless legs syndrome and periodic limb movement disorder

Restless legs syndrome (RLS) is a neurologic disorder characterized by disagreeable leg sensations that usually occur at rest or before sleep and are temporarily relieved by movement. Periodic limb movements (PLMs) are involuntary, stereotypic, repetitive limb movements that may occur during sleep and usually involve the legs and, occasionally, the arms. Periodic limb movements during sleep often accompany RLS. Periodic limb movement disorder (PLMD) is characterized by PLMs that cause frequent arousals and lead to insomnia or excessive daytime sleepiness.

The results of PSG studies from patients with severe RLS often show prolonged sleep latencies, decreased sleep efficiency, increased number of awakenings, significant reductions in total sleep time, and decreased amounts of slow-wave sleep [8.3.1]. Patients with PLMD often have frequent PLMs that are associated with arousals and awakenings, reduced total sleep time, and decreased sleep efficiency [8.3.2].

4.5.1 General evaluation

The evaluation should include a clinical history and physical examination. A ferritin level, complete blood count, urinalysis, and screening chemistries to assess secondary causes of RLS (e.g., anemia, uremia) and to rule out other conditions that can mimic RLS or PLMD (e.g., peripheral neuropathies). The clinical history should include bedpartner observation, if possible, with special emphasis on complaints of leg discomfort, the occurrence of leg or body jerks and restless sleep, and reports of insomnia or excessive daytime sleepiness.

4.5.2 Additional stratification factors

The validated NIH criteria can be used to establish the diagnosis of RLS.[136] The validated RLS rating scale[137] can be used to establish severity of patients' symptoms; this scale may be particularly useful for comparisons pre- and post-treatment.

4.5.3 Clinical indications for polysomnography and other sleep medicine procedures

The recommendations for use of PSG in the diagnosis of RLS and PLMD have been updated from the 1997 practice parameter. A MEDLINE search was conducted using the terms PLM, PLMD, PLMS, periodic leg movements, periodic limb movements, RLS, restless legs, or restless legs syndrome and sleep studies, PSG, polysomnograms, polysomnography, or overnight studies, and limited to English language articles describing adult human studies published from January 1997 through September 2001 (yield = 119 citations). We eliminated any series of less than 20 patients, reviews, case reports, and citations that did not directly relate to the diagnosis of RLS and PLMD. No articles were found that justified changes in the current practice of using PSG for the diagnosis of RLS and PLMD, so they were not entered into evidence tables. A supplemental search was conducted from September 1, 2001 through August 15, 2004 using the identical search terms as the previous literature search (yield = 92 citations); no new articles were found that necessitated changes in the following recommendations.

4.5.3.1 Polysomnography is indicated when a diagnosis of periodic limb movement disorder 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. [8.3.2] (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper. The diagnosis of PLMD can be established only by PSG. The diagnosis of PLMD requires quantification of PLMs and PLM related arousals, assessment of the impact of the movements upon sleep architecture, and identification and exclusion of other sleep disorders.

4.5.3.2 Polysomnography is not routinely indicated to diagnose or treat restless legs syndrome, except where uncertainty exists in the diagnosis. [8.3.1] (Standard)

This recommendation is a modification of the recommendation of the previous practice parameter paper to include the exception for performing PSG. Although PLMD can exist independent of RLS, it is estimated that 80.2% of individuals with RLS have evidence of PLMS on PSG,[138] so PSG may be helpful in increasing the confidence in the RLS diagnosis.[139]

4.5.4 Technical considerations

4.5.4.1 The minimum channels required for the evaluation of periodic limb movements and related arousals include EEG, EOG, chin EMG, and left and right anterior tibialis surface EMG. Respiratory effort, airflow, and oximetry should be used simultaneously if sleep apnea or upper-airway resistance syndrome is suspected to allow a distinction to be made between inherent periodic limb movements and those limb movements associated with respiratory events. [8.3.3] (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.5.4.2 Intra-individual night-to-night variability exists in patients with periodic limb movement sleep disorder, and a single study might not be adequate to establish this diagnosis. [8.3.2] (Option)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.5.5 Alternative tools

4.5.5.1 Actigraphy is not indicated for the routine diagnosis, assessment of severity, or management of restless legs syndrome or periodic limb movement sleep disorder. However, it may be useful in the assessment of treatment effects of these disorders. (Option)

This is a new recommendation. It is reproduced from the practice parameters for the role of actigraphy in the study of sleep and

circadian rhythms.[140]

4.5.5.2 The suggested immobilization test (SIT) and forced immobilization test (FIT) may be an aid in the diagnosis of restless legs syndrome and for pre- and post-treatment comparisons. (Option)

This is a new recommendation. The SIT and FIT have been used primarily in research applications,[141,142] but may have usefulness in establishing the diagnosis of RLS and also may provide an objective means of comparison for pre- and post-treatment.

4.6 Depression with insomnia

Depression with insomnia is characterized by the complaint of difficulty with sleep associated with a psychiatric diagnosis of unipolar or bipolar illness. Difficulty with sleep maintenance, difficulty with sleep onset, and early morning awakenings may all be present. Daytime fatigue may also be present, although there is little evidence to suggest that true physiologic sleepiness is present, except in depression with hypersomnia (seasonal affective disorder or bipolar depression). During the manic phase of a bipolar disorder, sleep may be markedly reduced in amount without the patient having a concurrent complaint of insomnia. Most studies on sleep in depression focus on patients with unipolar depression or patients in the depressed phase of bipolar illness.

4.6.1 General evaluation

A clinical history is essential in establishing the characteristics of the patient's insomnia. A psychiatric evaluation provides information for the diagnosis of depression. Previously published practice parameters address the use of PSG in the evaluation of insomnia.[143]

4.6.2 Additional validated stratification factors

Structured psychiatric interviews as well as paper-and-pencil tests, including the Beck Depression Inventory and the Hamilton Rating Scale for Depression, help establish the diagnosis of depression.

4.6.3 Clinical indications for polysomnography and other sleep medicine procedures

The recommendations for use of PSG in the diagnosis of depression with insomnia have been updated from the 1997 practice parameter. A MEDLINE search was conducted using the terms depression or insomnia and sleep studies, PSG, polysomnograms, or polysomnography, and limited to English language articles describing adult human studies published from January 1997 through September 2002 (yield = 707 citations). Forty articles from these searches were obtained that were potentially relevant to the question of whether the PSG is useful in diagnosing depression. Twenty-four articles, in addition, were obtained. These articles were thought to address issues such as: (1) the use of PSG to be able to predict early on which depressed patient was more likely to respond to treatment; and (2) whether alterations in the PSG shed light on the changes in sleep that might underlie the recovery from depression. Thirty-nine articles were rejected because: (1) they did not include a PSG in the study, (2) the number of subjects studied was too small, or (3) the study was not focused on diagnostic discrimination (i.e., sensitivity and specificity were not provided or could not be calculated). Four articles were selected for review from the 24 that were thought to be relevant to predicting an early response to treatment from the PSG or which addressed changes in the sleep EEG that might help explain the recovery from depression. Of the articles reviewed since 1997, there was only one article[144] that provided data from which specificity and sensitivity data could be calculated. The remaining data were from studies that distinguished depressed inpatients and outpatients with Major Depressive Disorder from normals. The authors of the article recognized the limited clinical utility of the PSG to make the diagnosis of depression because of cost and inconvenience. A supplemental search was conducted from September 1, 2001 through August 15, 2004 using the identical search terms as the previous literature search (yield 308 citations); no new articles were found that necessitated changes in the following recommendations.

There has been an effort to utilize the PSG in depression: (1) to be able to predict the response to treatment and (2) to examine the changes in sleep that may underpin the therapeutic response in depression. One study found that the movement of SWS to earlier in the night, into the first NREM period, occurs early in treatment and co-varies with later recovery from depression.[145] The nature of the change in sleep that co-varies with recovery from a depression is the presence of more delta sleep in the first NREM period as well as changes in phasic aspects of REM sleep.[146-148]

4.6.3.1 Neither a polysomnogram nor a multiple sleep latency test is routinely indicated in establishing the diagnosis of depression. [9.4.2] (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper. No characteristics of sleep architecture are specific for the diagnosis of depression. A diagnosis of depression does not in and of itself preclude PSG evaluation if the patient's symptoms and history are indicative of a diagnosis that requires PSG evaluation. Other common sleep disorders can also produce fatigue, tiredness, or sleepiness, symptoms that may suggest depression.

4.6.4 Technical considerations

4.6.4.1 A number of pharmacologic agents used to treat depression can affect sleep [9.4.2]. The clinician must consider these effects when interpreting a polysomnogram or multiple sleep latency test performed on a patient who takes these medications. (Guideline)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.6.4.2 Except for those patients who are being evaluated for narcolepsy, patients who have depression and are being evaluated for a coexisting sleep disorder, e.g., a sleep related breathing disorder, usually do not need to stop taking antidepressant medications. Because the diagnosis of narcolepsy is dependent upon the observation of pathologic alterations in REM sleep, however, the outcome of the evaluation may be inaccurate if polysomnography is performed while the patient is taking these REM-altering medications. Although antidepressants can affect sleep architecture in other sleep disorders and may affect the occurrence of parasomnias and periodic limb movements, patients may face significant risks in controlling depression if antidepressant medications are

discontinued. In addition, because patients with depression often require the use of antidepressant medications for a long period of time, the results of a study performed with the patient off medications may not be representative of the patient's usual circumstances and sleep symptoms. (Guideline)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.6.5 Alternative tools

For the diagnosis of depression, with or without insomnia, a variety of other diagnostic psychiatric tests exist.

4.7 Circadian rhythm sleep disorders

Circadian rhythm sleep disorders result from a mismatch between an individual's sleep pattern and the timing and amount of sleep that the person desires, needs, requires, or expects. Specific diagnoses are: circadian rhythm sleep disorder, delayed sleep phase type; circadian rhythm sleep disorder, advanced sleep phase type; circadian rhythm sleep disorder, irregular sleep-wake type; circadian rhythm sleep disorder, free running (non-entrained) type; circadian rhythm sleep disorders due to medical condition; primary (organic) circadian rhythm sleep disorders, unspecified other physiological (organic) circadian rhythm, unspecified (organic circadian rhythm disorder, no other symptoms); circadian rhythm sleep disorder not due substance or known physiological condition, jet lag type; circadian rhythm sleep disorder not due to substance or known physiological condition, shift-work type; circadian rhythm sleep disorder not due to substance or known physiological condition, delayed sleep phase type; circadian rhythm sleep disorder not due to substance or known physiological condition, unspecified (nonorganic circadian rhythm sleep disorder, no other symptoms); other circadian rhythm sleep disorder not due to substance or known physiological condition; and other circadian rhythm sleep disorder due to drug or substance.

4.7.1 General evaluation

A clinical history in conjunction with a multiweek sleep diary should be obtained to assess the consistency and patterns of sleep and to identify details suggesting other etiologies.

4.7.2 Additional validated stratification factors

There are no additional validated stratification factors.

4.7.3 Clinical indications for polysomnography and other sleep medicine procedures

The recommendations for use of PSG in the diagnosis of circadian rhythm sleep disorders have been updated from the 1997 practice parameter. A MEDLINE search was conducted using the terms circadian rhythm sleep disorder, time zone change syndrome, shift work sleep disorder, irregular sleep-wake pattern, delayed sleep phase syndrome, advanced sleep phase syndrome, or non-24 hour sleep-wake disorder and polysomnography, sleep studies, diagnosis, or indications, and limited to English language articles describing adult human studies published from January 1997 through September 2001 (yield = 69 citations). We eliminated any series of less than 20 patients, reviews, case reports, and citations that did not directly relate to the diagnosis of circadian rhythm sleep disorders. No articles were found that justified changes in the current practice of using PSG for the diagnosis of circadian rhythm sleep disorders, so they were not entered into evidence tables. A supplemental search was conducted from September 1, 2001 through August 15, 2004 using the identical search terms as the previous literature search (yield = 82 citations); no new articles were found that necessitated changes in the following recommendations.

4.7.3.1 Polysomnography is not routinely indicated for the diagnosis of circadian rhythm sleep disorders [10.3]. (Standard)

This recommendation is the same as the recommendation of the previous practice parameter paper.

4.7.4 Technical considerations

There are no technical considerations.

4.7.5 Alternative tools

4.7.5.1 Actigraphy may be useful in characterizing and monitoring circadian rhythm patterns or disturbances in the following special populations: (a) the elderly and nursing home patients with and without dementia; (b) newborns, infants, children and adolescents; (c) hypertensive individuals; (d) depressed or schizophrenic patients; and (e) individuals in inaccessible situations (e.g., space flight). (Option).

This recommendation is reproduced from the practice parameters for the role of actigraphy in the study of sleep and circadian rhythms.[140] Actigraphy may be a useful adjunct to a clinical history, physical examination, and subjective sleep diary in the evaluation of circadian rhythm disorders in select circumstances.

4.7.5.1 Serum and urinary melatonin levels and twenty-four hour core body temperature levels have also been used as alternative methods for detecting circadian rhythm disorders in research settings. (Option)

This is a new recommendation. There is limited evidence for melatonin and temperature measures for detection of circadian rhythm disorders.[149,150]

5.0 FUTURE DIRECTIONS

1) Sleep related breathing disorders. The indications for PSG in patients with OSA and risk factors for cardiac disease and stroke need to be further explored. Models for predicting and stratifying severity of SRBDs as a cost-effective method for screening patients should be developed.

2) Other respiratory disorders. Future studies should be designed to specifically address whether the use of PSG in the evaluation of patients with respiratory insufficiency of any cause may affect diagnosis and treatment. Specific outcomes, variables, and cost analyses are needed. Studies to determine the role of PSG in the titration of NIPPV are particularly needed.

3) Narcolepsy. The specific utility of PSG in the diagnosis of narcolepsy needs to be explored. In addition, further studies using the MSLT, particularly with respect to additional normative data, need to be conducted.

4) Parasomnias and sleep related seizure disorders. The use of digital PSG in the recording and assessment of parasomnias and sleep related seizure disorders needs to be standardized.

5) RLS and PLMD. The use of actigraphy and the SIT or FIT should be explored as alternative methods for the diagnosis of these conditions.

6) Depression with insomnia. The specific utility of PSG in the diagnosis of depression with insomnia needs to be further explored.

7) Circadian rhythm sleep disorders. The use of actigraphy, melatonin, and core body temperature measures for the diagnosis of these disorders should be evaluated.

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Table 5—Evidence table for stratification of risk for SRBD

Author Year (Ref#) Level	Study Design, Blinded?	Protocol, Monitoring channels	# of Patients, Pt selection, range of Pts	Outcome Conclusions Biases	Comments
Roche 2002[19] I	Cohort, Not stated	Diagnostic, Type I (PSG)	102/108, consecutive, select	Logistic regression was used on 102 pts to develop clinical prediction rule, and then tested on 108 pts. NPV in group 2 was only 36.7%, PPV 86.7%, accuracy 53%. Population bias	Reference standard applied to all. Carefully done study. Group 1 had lower prevalence of OSA than Group 2. Authors used models with and without oximetry data-emphasize the importance of validating prediction rules in other populations.
Gurubhagavatula 2001[20] II	Cohort, Single blind	Diagnostic, Type 1 (PSG)	243, convenience, select	Sens, Spec, PPV, NPV for AHI>5 was 0.941,0.667, 0.857, and 0.842 respectively. For AHI>%, values were 0.833, 0.947, 0.833, and 0.947. Population bias	Reference standard applied to all. Parameters used were MAP questionnaire plus oximetry. Oximetry was done concurrently, and the model was developed on 80% of the select population, and tested retrospectively on 20% of the population. Potential for bias in homogeneous population, lack of prospective validation, and concurrent oximetry and PSG testing.
Zerah-Lancner 2000[22] I	Cohort, Double blinded	Diagnostic, Type 1 (PSG)	168/101, consecutive, select	The model developed on 168 pts utilizing PFT data plus BMI showed a sensitivity of 100%, a specificity of 84%, a PPV of 86%, and an NPVof 100% when used prospectively in 101 consecutive patients. Pt selection bias	Reference standard applied to all. AHI diagnostic threshold=15.Pts with history of alcoholism, regular use of hypnotic medication; upper respiratory tract disorders; previous treatment for sleep apnea; cardiopulmonary disease; or airway obstruction, or neuromuscular disease excluded.
Rowley 2000[26] II	Cohort, Not stated	Diagnostic, Type 1 (PSG)	425/370, consecutive, broad	The four models were moderately sensitive (sensitivities 76%—96%) but poorly specific (specificities 13-54%) for an AHI threshold of $\geq$ 10. The positive predictive values ranged between 69%—77% For AHI $\geq$ 20, the specificity was optimized at 85-90%, but sensitivity was only 33-39%. Population bias	Reference standard applied to all. Prospectively evaluated other clinical prediction models in a population referred for suspected OSA. Test population with a greater percent female gender and heavier. Not tested in general population.
Kirby 1999[28] II	Cohort, Not stated	Diagnostic, Type 1 (PSG)	255/150, random, select	Neural network trained on set of 255 pts, tested on set of 150 pts retrospectively. Prevalence of OSA (defined as AHI $\geq$ 10) in this series was 69% (53% in women and 76% in men). The neural network model had a mean predictive accuracy of 91.3% (95% confidence interval [CI], 86.8 to 95.8) for both ruling in and out OSA. Population bias	Reference standard applied to all. Limited by retrospective nature of the data review, the lack of prospective validation, and the relatively small numbers, but population was general.
el-Solh 1999[29] II	Cohort, Not stated	Diagnostic, Type 1 (PSG)	293/189, random, select	Neural network trained on 9/10th of pts, tested on additional 1/10 retrospectively. The area under the ROC was 0.96±0.0191 SE, 0.951± 0.0203 SE, and 0.935±0.0274 SE when OSA was defined as an AHI of >10, >15, and >20/hour, respectively. Population bias	Reference standard applied to all. Limited by retrospective nature of the data review, the lack of prospective validation, and the relatively small numbers, but population was general.
Kushida 1997[27] I	Cohort, Not stated	Diagnostic, Type 1 (PSG)	30/423/300, consecutive, broad	Model derived from upper airway and body measurements in test population of 30, and then applied to 300 of 423 qualifying patients. Model had sensitivity of 97.6% (95% CI, 95% to 98.9%), to specificity of 100% (CI, 92% to 100%), positive predictive value of 100% (CI, 98.5% to 100%), and negative predictive value of 88.5% (CI, 77% to 96%) using AHI $\geq$ 5. Authors tested interrater reliability for measurements and found it excellent. Population bias	Reference standard applied to all. Model had only 6 false negative results, with mean AHI of 7.4. The model appears applicable to a wide range of pts but only tested in referred population.
Pouliot 1997[17] II	Cohort, Not stated	Diagnostic, Type 1 (PSG)	354, consecutive, broad	Evaluated self rated ESS, BMI, witnessed apneas alone or in combination to identify Apnea Index (AI)<20. AUC of ROC for BMI was 0.72 (sens=0.393, spec=0.920 for BMI $\leq$ 28), and for ESS was 0.56 (sens=0.424, spec=0.675 for LESS $\leq$ 12). Combining all 3 factors improved sens to 0.047, spec to 1.00, but numbers were very small. Population bias	Reference standard applied to all. Limited by relatively small numbers fitting criteria. Data analysis not generalizable.

Table 6—Evidence table for cardiac disease and OSA

Selection (consecutive, unspecified)
Abbreviations: M = male, F= female, LVEF = left ventricular ejection fraction, desats = desaturations, PSG = polysomnography, NHYA = New York Heart Association, AICD = automatic internal cardiac defibrillator. CSB = Cheyne-Stokes breathing, CSA = central sleep apnea, OSA = obstructive sleep apnea, TMST = treadmill stress test, OR = odds ratio, RVI = right ventricular impairment, RVEF = right ventricular ejection fraction, SDB = sleep disordered breathing

Author Year (Ref#) Grade	Study Design	Protocol	# of Patients, Pt selection	Outcome Conclusions	Comments
Fries 1999[43] II	Prospective Cross-sectional Prospective Cohort for mortality of CSA versus OSA	Full PSG Hypopnea = 40% decrease in airflow + 4% desat SRBD = AHI > 10/HR Recurrence of arrhythmia and time of occurrence noted.	N =40 Consecutive Inclusions: Pts had PSG following AICD Exclusions: none	SRBDs in 40% (16/40); Of these 16: CSA in 9/16 (8 of those with CSA had CSB); OSA in 7/16. Pts with and without SRBD did not differ in LVEF, or use of beta blockers. Most recorded arrhythmia occurrences were NOT during sleep. Mortality (all were non-sudden cardiac deaths): CSA 4/9 died (44%); OSA 0/9; no SRBDs (3/24 = 12.5%) Conclusions: SRBDs common in pts with arrhythmia. Mortality highest over 2 years in those with CSA.	Group prospectively followed for 2 years: for mortality, for arrhythmia. The exact criteria separating CSA from OSA were not given - i.e., what % of events must be central to be considered CSA.
Hanly 1996[33] II	Prospective Cohort	Initial PSG determines if CSR (Cheyne-Strokes respiration) Present. Patients followed by telephone questionaire.	N = 16 M = 16 F = 0 Inclusions: NYHA 3,4 stable severe CHF LVEF 22.9%. No pts treated with CPAP or oxygen. Exclusions: pulmonary, renal, neurological disease, coexisting disease with poor prognosis.	9 with CSR 7 no CSR. Deaths: 7/9 with CSR 1/7 with no CSR. In pts with severe stable CHF the presence of CSR implies worse prognosis.	CSR and no CSR groups have equivalent LVEF, age, BMI Transcutaneous PCO2 lower in CSR group.
Sanner 1997[38] II	Prospective cross-sectional	RV evaluated for RV impairment (RVI). RVI defined as RVEF ≤45% by radionuclide ventriculogram (evaluation blind to result of PSG). PFT, ABG, right heart catheterization also performed to determine wedge pressure at rest and with exercise.	N=107 M=94 F=13 Consecutive Inclusion: snoring suspected OSA, or EDS; and AHI>10 on PSG. Exclusions: Clinical or lab evidence of chronic lung disease, LV failure (= radiographic LV enlargement or pulmonary venous HTN), hypoventilation from CNS disorders.	1. RV impaired in 19/107 (18%) (pts had normal lung function). 2. RVEF only mildly impaired (38%) in this group.3. No difference in BMI, gender, PFT, pO2, LVEF, or wedge pressure between pts with and without RVI 4. RVI group had higher AHI and nocturnal desaturation. 5. Only 52% of the pts with RVI had pulmonary HTN, and 48% of patients with nl RV had pulmonary HTN. Conclusions: OSA can be cause of unexplained RVI.	No matched obese population without OSA studied. True incidence of RVI patients with OSA not determined. RVI patients did not have more pulmonary HTN than non RVI group. RVEF only mildly impaired in the RVI group.
Sanner 2001[42] II	Prospective Cohort	Consecutive pts referred for angina evaluation. Studies: coronary angiography, selective left ventriculography, and portable polygraphic study, sif AHI > 10, went on to have PSG (16/21 had full PSG).	N=68 M=53 F=15 Consecutive Inclusions: referred for angina evaluation. Exclusions: alcohol or sedatives the day day prior or night of recording.	1. 21 patients (31%) had AHI > 10 2. 6/21 had CSB 3. Incidence of SRBDs not increased in patients with (26.5%) versus those without CAD (42.1%). 4. Severity of SRBDs were significantly and independently associated with LVEF (r=-0.38; p=0.002) but not with age, BMI, gender, DM, HTN, hyperuricemia, hypercholesterolemia, smoking and CAD 5. Sleep disordered breathing was not an independent predictor of CAD.	70 eligible, 68 completed study. Most pts had normal LVEF. 49 had CAD 19 no CAD –but 17/19 had non-ischemic heart disease (nearly all the patients had some form of heart disease).
Sin 1999[31] II	Retrospective Cross-sectional	Retrospective analysis for CSA and OSA of 450 consecutive pts with CHF referred to Sleep research laboratory: Reasons for referral to sleep lab: 1) suspected sleep apnea syndrome (excessive daytime sleepiness, snoring, nocturnal dyspnea or 2) persistent dyspnea or exercise limitation despite optimal medical management.	N=450 M=382 F=68 Inclusions: 1. Diagnosis of CHF by cardiologist required at least 6 months of symptoms, at least one prior episode of symptomatic CHF (dyspnea at rest/exertion, chest x-ray showing cardiomegaly and congestion, 2. continued dyspnea NYHA class 2-4 despite optimal medical therapy, 3. stable clinical status - no change in meds for 2 weeks, 4. at least 30 min of sleep in the lab. Exclusions: unstable angina, or MI within 4 wks of study.	SRBDs very common in pts with symptomatic CHF: Men 75% (46% CSA, 48% OSA); Women 47% (15% CSA, 31% OSA), mean RDI for CSA=40.5, OSA=35.0. Predictive for CSA was male gender (OR 3.5), a-fib (OR 4.13), age>60 (OR 2.37), hypocapnia (OR 4.33). Predictive for OSA was BMI>35 in men, and age>60 in women.	SRBD≥10/hr, CSA if ≥50% of events central, OSA if ≥50% obstructive. As all patients referred for suspected sleep apnea, likely biased sample may not represent other samples of CHF patients.
Tremel 1999[39] I	Cohort study	Prospective study involving consecutive patients with CHF who had PSG at 1 and 2 months after optimization of medical therapy.	N=34 M=28 F=6 Consecutive. Inclusion: Pts with initial pulmonary edema, who improved after 1 month of medical treatment to NYHA II or III; age < 75 years; LVEF < 45% Exclusion: Unspecified.	SRBDs in this population were 82% -75% had CSA and 25% had OSA. After medical therapy, only a few (those with CSA) showed improvements in AHI. .	Patients with CSA had lower PCO2s than OSA. There are significant correlations between AHI and VO2max, and between AHI and PCO2. In those with CSA, AHI correlated with LVEF.