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

Clete A. Kushida, MD, PhD; Michael R. Littner, MD; Timothy Morgenthaler, MD; Cathy A. Alessi, MD; Dennis Bailey, DDS; Jack Coleman, Jr., MD; Leah Friedman, PhD; Max Hirshkowitz, PhD; Sheldon Kapen, MD; Milton Kramer, MD; Teofilo Lee-Chiong, MD; Daniel L. Loube, MD; Judith Owens, MD; Jeffrey P. Pancer, DDS; Merrill Wise, MD

1Stanford University Center of Excellence for Sleep Disorders, Stanford, CA; 2VA Greater Los Angeles Healthcare System and David Geffen School of Medicine at UCLA, Sepulveda, CA; 3Mayo Sleep Disorders Center, Mayo Clinic, Rochester, MN; 4UCLA/Greater Los Angeles Healthcare System, Sepulveda, CA; 5Greenwood Dental Associates, Englewood, CO; 6Middle Tennessee ENT, Murfreesboro, TN; 7Stanford University School of Medicine, Stanford, CA; 8Baylor College of Medicine and VA Medical Center, Houston, TX; 9VA Medical Center and Wayne State University, Detroit, MI; 10Maimonides Medical Center, Psychiatry Department, Brooklyn, NY and New York University School of Medicine, New York, NY; 11National Jewish Medical and Research Center, Sleep Clinic, Denver, CO; 12Sleep Medicine Institute, Swedish Medical Center, Seattle, WA; 13Department of Pediatrics, Rhode Island Hospital, Providence, RI; 14Toronto, ON, Canada; 15Departments of Pediatrics and Neurology, Baylor College of Medicine, Houston, TX

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 neuro muscular 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.


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.

Disclosure Statement
Dr. Kushida has received research support from GlaxoSmithKline, Boehringer-Ingelheim, Xenopont, and Pfizer; has received honoraria from GlaxoSmithKline; has received consulting fees from New Millennium Diagnostics, Inc.; and has received royalties as a licensor of a patented oral measurement device from Respironics, Inc. Dr. Littner is the principal investigator in research studies supported by GlaxoSmithKline, AstraZeneca, and Boehringer-Ingelheim; is on the speakers’ bureaus for Boehringer-Ingelheim, Novartis, GlaxoSmithKline, and Pfizer; and has received honorarium from Boehringer-Ingelheim. Dr. Morgenthaler has received research support from Itamar Medical and ResMed. Dr. Alessi is a speaker for the Medical Education Speaker’s Network; and is a consultant for Prescription Solutions. Dr. Owens has received research support from Eli Lilly, Sepracor, Cephalon, and Sanofi-Aventis; is a speaker for Eli Lilly and Johnson & Johnson; and is a consultant for Eli Lilly, Johnson & Johnson, Sepracor, Cephalon, and Sanofi-Aventis. Dr. Littner is a consultant for Sanofi-Aventis and Cephalon; and has received honoraria from Sanofi-Aventis. Dr. Bailey is a partner in Dental Appliance Innovators Inc., this company developed the NORAD oral appliance. Drs. Friedman, Kapen, Kramer, Lee-Chiong, Loube, Wise, Coleman, and Pancer have indicated no financial conflicts of interest.

SLEEP, Vol. 28, No. 4, 2005
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. 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. 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:**

<table>
<thead>
<tr>
<th>Recommendation Grades</th>
<th>Evidence Levels</th>
<th>Study Design</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>I</td>
<td>Randomized well-designed trials with low alpha and beta error*</td>
</tr>
<tr>
<td>B</td>
<td>II</td>
<td>Randomized trials with high alpha and beta error*</td>
</tr>
<tr>
<td>C</td>
<td>III</td>
<td>Nonrandomized concurrently controlled studies</td>
</tr>
<tr>
<td>C</td>
<td>IV</td>
<td>Nonrandomized historically controlled studies</td>
</tr>
<tr>
<td>C</td>
<td>V</td>
<td>Case series</td>
</tr>
</tbody>
</table>

Adapted from Sackett³

*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). 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 it 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 airway obstruction is identified by measuring an intrathoracic pressure for at least 10 seconds preceding an arousal with resumption of more normal pressures. As shown by progressively more negative esophageal pressure for at least 10 seconds preceding an arousal with resumption of more normal pressures, 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 hypventilation 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 ≥ 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

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard</td>
<td>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.</td>
</tr>
<tr>
<td>Guideline</td>
<td>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.</td>
</tr>
<tr>
<td>Option</td>
<td>This is a patient-care strategy, which reflects uncertain clinical use. The term option implies either inconclusive or conflicting evidence or conflicting expert opinion.</td>
</tr>
</tbody>
</table>

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

Table 3—Definitions of Breathing Events During Sleep

<table>
<thead>
<tr>
<th>Breathing Event</th>
<th>Clinical Definition</th>
<th>Research Definition*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstructive Apnea</td>
<td>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.</td>
<td>A clear decrease (&gt;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)</td>
</tr>
<tr>
<td>Central Apnea</td>
<td>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.</td>
<td>Same as above, but an esophageal balloon must verify lack of effort.</td>
</tr>
<tr>
<td>Mixed Apnea</td>
<td>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.</td>
<td>ghosts of the blood signal, or when snoring intensity increases during the event.</td>
</tr>
<tr>
<td>Hypopnea</td>
<td>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 ≥4% oxygen desaturation. Obstruction is often inferred from thoracoabdominal paradox, the shape of the flow signal, or when snoring intensity increases during the event.</td>
<td>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 &gt;3% or an arousal. Only an esophageal balloon can demonstrate the hypopnea to be obstructive vs. central.</td>
</tr>
<tr>
<td>Respiratory-Effort Related Arousal (RERA)</td>
<td>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.</td>
<td>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 &gt;3% or an arousal. Only an esophageal balloon can demonstrate the hypopnea to be obstructive vs. central.</td>
</tr>
</tbody>
</table>

From The Report of an American Academy of Sleep Medicine Task Force.4
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.\textsuperscript{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.\textsuperscript{15} The diagnostic value of witnessed apneas and hypersomnia were evaluated in a study of 380 patients referred for sleep study with problem snoring.\textsuperscript{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 from 0.40 and 0.60. Pouliot et al.\textsuperscript{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 ≥ 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 ≥ 15. While male gender, age, and snoring were associated with AHI, individually the associations were far from predictive of OSA.\textsuperscript{14} These findings are in agreement with the bulk of the literature evaluating clinical features of patients with OSA.\textsuperscript{14-23} Additionally, several studies have documented sensitivities and specificities of clinical impression from experienced clinicians as 52-60% and 65-70%, respectively.\textsuperscript{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%.\textsuperscript{26} Positive predictive values ranged between 69-77%, and even when optimized to detect the more severe AHI (AHI ≥ 20), had sensitivities of 85-98% but specificities of 33-39%.\textsuperscript{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%.\textsuperscript{19} The addition of oximetry data did not appreciably affect accuracy. In the study of Deegan et al.,\textsuperscript{15} a model combining the clinical features with oximetry data only correctly classified 32.4% of patients with (defined as AHI ≥ 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).\textsuperscript{19,20,27} Kushida et al.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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.\textsuperscript{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. 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. Furthermore, asymptomatic left ventricular dysfunction has also been associated with increased prevalence of SRBDs. 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. Second, there is some evidence that the presence of Central Sleep Apnea or Cheyne-Stokes (CSA/CSR) signifies a worse prognosis. Thus, a segment of patients deserving more intensive treatment may be identified by PSG. Third, there is evidence that treatment of OSA or CSA/CSR type in patients with heart failure may improve cardiac function. There is some evidence that treatment of patients with CSA/CSR and congestive heart failure (CHF) may improve survival. 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 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. 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 hypopnea, 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 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 evaluated a group of patients suspect-
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. 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 patients 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.

4.1.2.1.4 Stroke

Several studies have evaluated the prevalence of SRBDs in patients who have suffered ischemic or hemorrhagic stroke. 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 and four Level V studies evaluated the effect of SRBDs on prognosis. Harbison et al. 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 evaluated neurologic outcomes in patients with recent stroke and AHI > 10. Both studies found that SRBDs were associated with early deterioration in neurological status (dementia or depression). However, some of these studies found patients with SRBDs to have less independence with activities of daily living, but the other did not find a difference in functional outcome at 6 months after stroke. One Level V study 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 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.

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. 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.

4.1.2.3 Portable monitoring devices

Practice parameters regarding the use of portable monitors (PM) were recently published. 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.

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

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. This left 4 citations that directly related to the role of PSG in diagnosing SRBDs.
### 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

#### Recorded Parameters:
- 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:
- Number and Index of Obstructive/Mixed and Central Apneas
  - (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 Respiratory-Effort-Related Arousals (RERAs)
  - (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 SaO2 in percentage ranges in wake, NREM, and REM
  - Mean, minimum, and maximum of SaO2 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 hypnagogic hallucinations, sleepwalking, enuresis, bruxism, seizures, nocturnal eating, rhythmic movement disorder)

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

1For 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.

2A 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 III14,16,21 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,30,37,41,75 and two Level IV,40,44 studies, of which the Level I and II studies are depicted in 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,74 four Level IV,50,57,62,76 and thirteen Level V48,49,51-56,58-61,77 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),8 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
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 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, two Level II, and two Level III 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, one Level III, and one Level IV 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, four Level IV, and thirteen Level V studies indicating...
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 study indicating an association between OSA by PSG and bradyarrhythmia, and two Level II and one Level IV 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 with 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 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 SaO2 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/FEV1 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 SaO2 < 94% and FEV1 < 65%.88,89 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.90 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 hypventilation 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 H₂O.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 OSAS.

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 studies90,99,100 involved over 200 patients each but were not designed to answer the question posed by the Standards of Practice Committee as stated above. Chauuet 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 report100 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 Chauuet et al.,82 PSG was used to rule out OSA as a contributing cause of pulmonary hypertension in 94 patients with COPD. Bijkerk 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, eleven104-114 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. Cibella115 and Cuttitta116 specifically showed effects of asthma from gastroesophageal reflux in small groups of seven subjects per each report. Lin and Lin117 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 study118 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 SaO₂ 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 fatique. 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. Hsu133 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. 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. 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, 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. The presence of two or more SOREMPs was associated with a sensitivity of 0.78 and a specificity of 0.93. 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, sleepwalking, 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”.  

SLEEP, Vol. 28, No. 4, 2005
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. The validated RLS rating scale 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, so PSG may be helpful in increasing the confidence in the RLS diagnosis.

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. [8.3.2] (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, polysonomograms, 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 article144 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 to 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. 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.

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) Paroxysmal nocturnal dyspnea. The use of digital PSG in the recording and assessment of paroxysmal dyspnea and sleep related 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.

REFERENCES


139. Silber MH. Commentary on controversies in sleep medicine. Montplaisir et al.: Periodic leg movements are not more prevalent in insomnia or hypersomnia but are specifically associated with sleep disorders involving a dopaminergic mechanism. Sleep Med. 2001;2(4):367-9.


<table>
<thead>
<tr>
<th>Author Year</th>
<th>Study Design, Blinded?</th>
<th>Protocol, Monitoring channels</th>
<th># of Patients, Pt selection, range of Pts</th>
<th>Outcome</th>
<th>Conclusions</th>
<th>Biases</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roche 2002</td>
<td>Cohort, Not stated</td>
<td>Diagnostic, Type 1 (PSG)</td>
<td>102/108, consecutive, select</td>
<td>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</td>
<td>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.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guruhagavatula 2001</td>
<td>Cohort, Single blind</td>
<td>Diagnostic, Type 1 (PSG)</td>
<td>243, convenience, select</td>
<td>Sens, Spec, PPV, NPV for AHI&gt;5 was 0.941, 0.667, 0.857, and 0.842 respectively. For AHI&gt;5%, values were 0.833, 0.947, 0.833, and 0.947. Population bias</td>
<td>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.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zerah-Lancner 2000</td>
<td>Cohort, Double blinded</td>
<td>Diagnostic, Type 1 (PSG)</td>
<td>168/101, consecutive, select</td>
<td>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 NPV of 100% when used prospectively in 101 consecutive patients. Pt selection bias</td>
<td>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; cardiodiaphragmatic disease; or airway obstruction, or neuromuscular disease excluded.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rowley 2000</td>
<td>Cohort, Not stated</td>
<td>Diagnostic, Type 1 (PSG)</td>
<td>425/370, consecutive, broad</td>
<td>The four models were moderately sensitive (sensitivities 76%—96%) but poorly specific (specificities 13-54%) for an AHI threshold of ≥ 10. The positive predictive values ranged between 69%—77% For AHI ≥ 20, the specificity was optimized at 85-90%, but sensitivity was only 33-39%. Population bias</td>
<td>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.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kirby 1999</td>
<td>Cohort, Not stated</td>
<td>Diagnostic, Type 1 (PSG)</td>
<td>255/150, random, select</td>
<td>Neural network trained on set of 255 pts, tested on set of 150 pts retrospectively. Prevalence of OSA (defined as AHI ≥ 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</td>
<td>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.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>el-Solh 1999</td>
<td>Cohort, Not stated</td>
<td>Diagnostic, Type 1 (PSG)</td>
<td>293/189, random, select</td>
<td>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 &gt;10, &gt;15, and &gt;20/hour, respectively. Population bias</td>
<td>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.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kushida 1997</td>
<td>Cohort, Not stated</td>
<td>Diagnostic, Type 1 (PSG)</td>
<td>30/423/300, consecutive, broad</td>
<td>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 ≥ 5. Authors tested interrater reliability for measurements and found it excellent. Population bias</td>
<td>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.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pouliot 1997</td>
<td>Cohort, Not stated</td>
<td>Diagnostic, Type 1 (PSG)</td>
<td>354, consecutive, broad</td>
<td>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 ≤ 28), and for ESS was 0.56 (sens=0.424, spec=0.675 for LESS &lt; 12). Combining all 3 factors improved sens to 0.047, spec to 1.00, but numbers were very small. Population bias</td>
<td>Reference standard applied to all. Limited by relatively small numbers fitting criteria. Data analysis not generalizable.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Author Year</td>
<td>Study Design</td>
<td>Study Design</td>
<td>Protocol</td>
<td># of Patients</td>
<td>Outcome Conclusions</td>
<td>Comments</td>
<td></td>
</tr>
<tr>
<td>------------------</td>
<td>--------------------</td>
<td>-------------------------</td>
<td>-------------------------------</td>
<td>---------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Fries 199913</td>
<td>Prospective</td>
<td>Cross-sectional</td>
<td>Full PSG Hypopnea = 40% decrease in airflow + 4% desat SRBD = AHI &gt; 10/hr Recurrence of artrhythmia and time of occurrence noted</td>
<td>N=40 Consecutive</td>
<td>Inclusions:Pts had PSG following AICD Exclusions: none</td>
<td>In pts with severe stable CHF the prevalence of CSR implies worse prognosis. Group prospectively followed for 2 years: for mortality, for arrhythmia. The exact criteria separating CSR from CSA were not given - i.e., what % of events must be central to be considered CSR.</td>
<td></td>
</tr>
<tr>
<td>Hanly 199613</td>
<td>Prospective</td>
<td>Cohort</td>
<td>Initial PSG determines if CSR (Cheyne-Stokes respiration) Present Patients followed by telephone questionnaire.</td>
<td>N=16 M=16 F=0</td>
<td>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.</td>
<td>CSR and no CSR groups have equivalent LVEF, age, BMI Transcutaneous PCO2 lower in CSR group.</td>
<td></td>
</tr>
<tr>
<td>Sanner 199738</td>
<td>Prospective</td>
<td>Cross-sectional</td>
<td>RV evaluated for RV impairment (RVd, RVI) defined as RVEF ≤ 45% by radionuclide ventriculogram (evaluation blind to result of PSG). FTT, ABG right heart catheterization also determined to perform wedge pressure at rest and exercise.</td>
<td>N=107 M=94 F=13</td>
<td>Conclusions: 1. RV impaired in 19/107 (18%) (pts had normal lung function). 2. LVEF only mildly impaired (38%) in this group. 3. No difference in BMI, gender, PPT, PO2, LVEF, or wedge pressure between pts with and without RV. 4. RV group had higher AHI and nocturnal desaturations. Only 52% of the pts with RV had pulmonary HTN, and 48% of patients with RV had pulmonary HTN. Conclusions: OSA can be cause of unexplained RVI.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sin 199931</td>
<td>Retrospective</td>
<td>Cross-sectional</td>
<td>Retrospective analysis for CSA and OSA of 450 consecutive patients 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.</td>
<td>N=450 M=382 F=68</td>
<td>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.</td>
<td>SRBDs very common in pts with symptomatic CHF: Men 75% (46% CSA, 48% OSA); Women 47% (15% CSA, 31% OSA). 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&lt;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. 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.</td>
<td></td>
</tr>
<tr>
<td>Tremel 199939</td>
<td>Cohort study</td>
<td>Cohort</td>
<td>Prospective study involving consecutive patients with CHF who had PSG at 1 and 2 months after optimization of medical therapy.</td>
<td>N=34 M=28 F=6</td>
<td>Inclusions: Pts with initial pulmonary edema, who improved after 1 month of medical treatment to NYHA II or III, age &gt; 75 years, LVEF &lt; 45% Exclusion: Unspecified.</td>
<td>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.</td>
<td></td>
</tr>
</tbody>
</table>