

# Sleep Apnea and Commercial Motor Vehicle Operators:

Statement From the Joint Task Force of the American College of Chest Physicians, American College of Occupational and Environmental Medicine, and the National Sleep Foundation

**Natalie Hartenbaum, MD, MPH, FACOEM**  
**Nancy Collop, MD, FCCP**  
**Ilene M. Rosen, MD, MSCE, FCCP**  
**Barbara Phillips, MD, MSPH, FCCP**  
**Charles F. P. George, MD, FRCPC**  
**James A. Rowley, MD**  
**Neil Freedman, MD, FCCP**  
**Terri E. Weaver, PhD, RN, CS, FAAN**  
**Indira Gurubhagavatula, MD, MPH**  
**Kingman Strohl, MD**  
**Howard M. Leaman, MD**  
**Gary L. Moffitt, MD**  
**Mark R. Rosekind, PhD**

## Introduction

**M**edical research supports the finding that obstructive sleep apnea (OSA) is a significant cause of motor vehicle crashes (MVCs) resulting in two- to sevenfold increased risk.<sup>1-6</sup> Recent reports indicate OSA is present in a greater prevalence in operators of commercial motor vehicle (CMV) operators than in the general population.<sup>1,7</sup> Although U.S. commercial drivers are required by federal statute to undergo medical qualification examinations at least every 2 years, the most recent OSA recommendations for medical examiners were prepared during a 1991 conference sponsored by the Federal Highway Administration (FHWA).<sup>8</sup> Since then, the clinical diagnosis, evaluation, treatment, and follow-up criteria have changed significantly.

From OccuMedix, Inc. (Dr Hartenbaum), Dresher, Pennsylvania; the Department of Medicine, Division of Pulmonary/Critical Care Medicine (Dr Collop), Johns Hopkins University, Baltimore, Maryland; the Department of Medicine, Divisions of Sleep Medicine and Pulmonary, Allergy & Critical Care Medicine (Dr Rosen), University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania; the Division of Pulmonary Critical Care and Sleep Medicine (Dr Phillips), University of Kentucky College of Medicine, Lexington, Kentucky; the Department of Medicine, Division of Respiriology (Dr George), University of Western Ontario, and the Sleep Laboratory, London Health Sciences Centre, South Street Hospital, London, Ontario, Canada; the Department of Medicine, Division of Pulmonary, Critical Care & Sleep Medicine, Department of Internal Medicine (Dr Rowley), Wayne State University School of Medicine, Harper University Hospital, Detroit, Michigan; The Sleep and Behavior Medicine Institute and Pulmonary Physicians of the North Shore (Dr Freedman), Bannockburn, Illinois; Biobehavioral and Health Sciences Division (Dr Weaver), University of Pennsylvania School of Nursing, Philadelphia, Pennsylvania; the Department of Medicine, Divisions of Sleep, Pulmonary and Critical Care Medicine (Dr Gurubhagavatula), University of Pennsylvania Medical Center, Philadelphia, Pennsylvania; the Department of Medicine, Director (Dr Strohl), Center for Sleep Disorders Research, Case Western Reserve University School of Medicine, Louis Stokes DVA Medical Center, Cleveland, Ohio; the IHC Health Services to Business (Dr Leaman), Intermountain WorkMed, Salt Lake City, Utah; and Arkansas Occupational Health (Dr Moffitt), Springdale, Arkansas; Alertness Solutions (Dr Rosekind), Cupertino, CA.

Address correspondence to: Natalie Hartenbaum, MD, MPH, FACOEM, President and Chief Medical Officer, OccuMedix, Inc., P.O. Box 197, Dresher, PA 19025; E-mail: occumedix@comcast.net.

Copyright © 2006 by American College of Occupational and Environmental Medicine

DOI: 10.1097/01.jom.0000236404.96857.a2

Lacking current recommendations from the U.S. Department of Transportation (DOT), commercial driver medical examiners (CDMEs) must rely on outdated guidance and are thus forced to fill in the many existing gaps when evaluating CMV operators for this safety-sensitive type of work. In addition to causing difficulties for the medical examiner, the current guidelines, or lack thereof, foster an environment in which drivers who possibly have OSA are afraid to be evaluated because it might result in their removal from work. This set of circumstances may lead to the underrecognition of this condition and an increase in MVCs.

OSA is a risk factor for sudden or subtle performance impairment leading to accidents. This impairment goes beyond simply falling asleep. The driver must be able to survey the surrounding environment, stay in his or her driving lane, and make adjustments in speed and position—a divided-attention task.<sup>9</sup> Like with alcohol use or hypoglycemia, the driver tends to underestimate his or her degree of impairment. Healthcare providers are also imperfect at assessing impairment<sup>10</sup> or adequately identifying those who should be evaluated for OSA.<sup>11,12</sup> The situation is further complicated by the fact that not all patients with OSA are prone to accidents or performance decrements and some commercial drivers with OSA are known to have long, safe driving records despite having “severe” OSA.

When a medical examiner qualifies a CMV operator, he or she is required to determine whether that driver meets U.S. federal medical standards and can perform driving and nondriving tasks. Examiners cannot place work restrictions on the driver such as limiting hours, travel, or type of vehicle operated. Once a driver is medically certified, he or she can use that medical certificate to perform any task that may be required of a commercial driver for any company. The CDME must be aware that although OSA itself can affect safety, it also plays a key role in other medical conditions that may affect medical certification; these conditions include hypertension, heart failure, diabetes, and other cardiovascular and cerebrovascular diseases.

Due to public safety considerations, acceptable risk is more stringently defined for safety-sensitive positions than it is for the general public. This is quite different from the usual approach in clinical medicine. In assessing risk of accidents due to a medical condition, commercial drivers must be held to higher medical criteria than the general population<sup>13</sup> for several reasons: they

operate larger vehicles, may transport hazardous material or passengers, operate their vehicles for longer stretches of time, and have an economic incentive to continue driving when private drivers may choose to stop for a medical reason or road conditions. For workers in transportation and other safety-sensitive positions in which the risk of impairment or incapacitation puts others at risk, medical standards and guidelines exist for almost all medical conditions. For example, drivers with heart disease may be required to undergo biennial stress tests or echocardiograms to assess heart function and, if function is below a certain threshold, withholding of medical certification is recommended.<sup>14</sup> Drivers suspected of having a condition that may impair safe operation of the vehicle are required to undergo screening tests to determine whether the condition is present. Although exercise stress testing is not generally recommended for the asymptomatic individual, the U.S. Preventive Services Task Force explained that for certain occupations “considerations other than benefit to the patient may favor screening.” The Task Force also notes that although the testing cannot always identify those at risk of an acute event, it may “increase the margin of safety for the public.”<sup>15</sup>

The Americans with Disabilities Act (ADA) clearly states that federal medical standards take precedence over the ADA and this has been supported by case law.<sup>16</sup> In addition, for an individual to be considered covered under the ADA, he or she must be impaired in activities of daily living and a broad class of jobs. CMV operators with suspected or diagnosed OSA should be evaluated in the same manner as drivers with any other potentially impairing medical condition. Determination must then be made as to whether the driver’s risk of impairment is acceptable and the duration of his or her certification determined. The ideal system for determining which drivers

with OSA are at highest risk of accidents has yet to be identified. In discussing the need for guidelines, the Federal Motor Carrier Safety Administration (FMCSA) explained in the *Cardiovascular Advisory Panel Guidelines for the Medical Examination of a Commercial Motor Vehicle Operator* that acceptable risk is a medical and societal issue, and given the complex demands of operating a large truck or bus, coupled with the high fatality risk for occupants of the other vehicle in crashes involving CMVs, a conservative approach is required.<sup>14</sup>

The largest study to date performed to estimate the proportion of commercial drivers license holders with sleep apnea was conducted in a cohort of commercial drivers licensed in 2002 in Pennsylvania.<sup>17</sup> Drivers in this study were promised complete anonymity and guaranteed that any findings would be confidential. The study found that 17.6% of participants from Philadelphia and its suburbs had mild sleep apnea, defined by an apnea–hypopnea index (AHI) of  $\geq 5$  and  $< 15$  events per hour. Another 5.8% had moderate sleep apnea, with AHI  $\geq 15$  but  $< 30$  events per hour. An additional 4.7% had severe sleep apnea (AHI  $\geq 30$  events per hour). These findings are similar to those reported in other prevalence studies of sleep apnea in general populations<sup>18–20</sup> but not close to the extremely high prevalence values reported previously for commercial drivers.<sup>21</sup>

To address these issues, the American College of Chest Physicians, the American College of Occupational and Environmental Medicine, and the National Sleep Foundation (NSF) convened a Task Force to review the existing literature and the medical regulations/guidelines for CMV operators with or suspected of having OSA. Literature searches were conducted through Medline using search terms (alone and in combination) that included sleep apnea, driving, accidents, pretest probability, commercial drivers, drowsy driving, reg-

**TABLE 1***International Classification of Sleep Disorders, 2nd Edition: Obstructive Sleep\* Apnea in Adults*

A. At least one of the following applies:

- i. The patient complains of unintentional sleep episodes during wakefulness, daytime sleepiness, unrefreshing sleep, fatigue, or insomnia;
- ii. The patient wakes with breathholding, gasping, or choking; or
- iii. The bed partner reports loud snoring, breathing interruptions, or both during the patient's sleep.

B. Polysomnographic recording shows the following:

- i. Five or more scoreable respiratory events (ie, apneas, hypopneas, or respiratory-effort related arousals) per hour of sleep;
- ii. Evidence of respiratory effort during all or a portion of each respiratory event (in the case of a RERA, this is best seen with use of esophageal manometry).

OR

C) Polysomnographic recording shows the following:

- i. Fifteen or more scoreable respiratory events (ie, apneas, hypopneas, or respiratory-effort related arousals) per hour of sleep; or
- ii. Evidence of respiratory effort during all or a portion of each respiratory event (in the case of a RERA, this is best seen with use of esophageal manometry).

D) The disorder is not explained by another current sleep disorder, medical or neurological disorder, medication use, or a substance abuse disorder.

*Note:* For diagnosis, need A, B, and D or C and D.\* Adapted from Department of Health & Human Services, Centers for Medicare & Medicaid Services. *Medicare Coverage Issues Manual*. 2001. Available at: <http://new.cms.hhs.gov/transmittals/downloads/R150CIM.pdf>.

RERA indicates respiratory-effort related arousal.

ulations, policy, federal guidelines, crashes, medical risk, assessments, and driving fitness.

Medical standards from DOT agencies were reviewed for references to OSA as were standards and guidelines on drivers from several international groups. Reports and recommendations from the National Transportation Safety Board (NTSB) and FMCSA were also obtained and reviewed.

Once a preliminary document was prepared and reviewed, the Task Force met to develop recommendations based on that review. The main recommendation areas covered were:

- Screening;
- Diagnosis;
- Treatment;
- Return to work after removal from service for evaluation and/or treatment of OSA; and
- Follow up.

Additional topics discussed included assessing compliance, duration of certification, and other issues or research that would need to be considered. Although these recommendations are directed toward the CMV driver, they would also be relevant to others in positions in

which public safety is an issue such as those employed in safety-sensitive rail work (engineer, conductor, switchman, and so on), transit, maritime, military, or nuclear operations, in which recent official criteria does not exist.

The recommendations of this Task Force have been endorsed by the Board of Directors of each of the respective organizations. They have not been adopted by the FMCSA and therefore are not official guidelines.

### Definition of Obstructive Sleep Apnea

The term obstructive sleep apnea syndrome (OSAS) has been used synonymously with the term obstructive sleep apnea hypopnea syndrome. The *International Classification of Sleep Disorders, 2nd Edition* (ICSD-2) recommends using the term OSA to be consistent with the *International Classification of Diseases, 9th Revision* and *International Classification of Diseases, 10th Revision* classification scheme.<sup>22</sup> Patients with this disorder have repetitive partial or com-

plete obstruction of upper airway tissues during sleep that results in sleep disruption, gas exchange abnormalities, and cardiovascular changes. This type of sleep-disordered breathing is a spectrum, and the boundary between safe and unsafe levels is uncertain and changeable.

The AHI is the most commonly used criterion to establish the diagnosis of OSA and to quantify its severity. The definitions of the specific criteria for apneas and hypopneas are defined in the "Polysomnography" (PSG) section of this document. AHI is defined as the sum of apneas and hypopneas divided by the hours of sleep. The U.S. Centers for Medicare and Medicaid (CMS) operationally defines OSA as an AHI of five events or more per hour of sleep with sequelae (hypertension, stroke, sleepiness, ischemic heart disease, insomnia, mood disorders) or 15 or more events per hour of sleep without sequelae.<sup>23</sup> The ICSD-2 defines OSA as five or more obstructed breathing events per hour of sleep with the appropriate clinical presentation (see Table 1).

## Current Regulations, Recommendations, and Guidelines

### Regulations and Guidelines for Commercial Motor Vehicle Drivers in the United States

The U.S. federal medical standard for CMV operators that covers OSA is section 49 CFR 391.41(b)(5) of the Federal Motor Carrier Safety Regulations (FMCSRs). This section states that the driver must have “no established medical history or clinical diagnosis of a respiratory dysfunction likely to interfere with his ability to control and drive a motor vehicle safely.”

Until 1998, there was no specific mention of OSA in the regulation itself or in the FMCSA medical advisory criteria. The advisory criteria are a series of recommendations created to assist CDMEs in determining whether a driver meets the medical standards. The FHWA’s 1991 Conference on Respiratory/Pulmonary Disorders and Commercial Drivers<sup>8</sup> and 1998 Conference on Neurologic Disorders and Commercial Drivers<sup>24</sup> offered recommendations, but these have not been updated.

The 1991 respiratory/pulmonary conference report<sup>8</sup> suggested that drivers should be screened by asking if they snore and frequently fall asleep during the day. The report recommended that those with suspected or diagnosed but untreated sleep apnea should not be medically qualified to drive until the diagnosis was eliminated or the condition successfully treated. Once diagnosed, it was recommended that drivers not return to work for 1 month. Before returning to safety-sensitive work, the driver should have either a repeat sleep study showing resolution of the apneas or a normal Multiple Sleep Latency Test (MSLT). Yearly sleep studies or MSLTs were recommended for follow up.

The neurologic disorders report<sup>24</sup> recommended that CMV operators with sleep apnea and any of the symp-

toms related to excessive daytime sleepiness not be permitted to operate in interstate commerce. Only surgical treatment was addressed in this report and a 3-month wait and laboratory studies (MSLT or polysomnogram) was recommended before allowing operators to resume commercial driving.

In 1998, a proposed new medical examination reporting form was published in the *Federal Register*.<sup>25</sup> A new question was added to the driver medical history that asks whether the driver has a sleep disorder, pauses in breathing while asleep, daytime sleepiness, or loud snoring. Parents Against Tired Truckers (PATT) requested that the Epworth Sleepiness Scale (ESS) be added to the examination, but in a Final Rule announcement,<sup>26</sup> the FMCSA did not include the ESS. The agency indicated that research was ongoing and referred examiners to the respiratory/pulmonary report.

Although there has been no complete current guidance on diagnosis, treatment, or assessment of drivers suspected of having or diagnosed with OSA, the FMCSA did issue a statement in 2004 regarding modafinil, a psychostimulant used to enhance wakefulness. The agency pointed out that modafinil did not cure the sleep disorder and did have potential side effects, noting that “Many drugs may interact with modafinil which include over-the-counter medication, prescription medication, nutritional supplements, herbal products, alcohol-containing beverages and caffeine.”<sup>27</sup> The agency further stated “Until there is more information regarding modafinil, the Federal Motor Carrier Safety Administration is recommending that commercial motor vehicle drivers not use modafinil for obstructive sleep apnea/hypopnea syndrome and shift work sleep disorders.” Examiners were also reminded that narcolepsy disqualifies CMV operators.

Drivers that operate CMVs also drive private vehicles and many states also have medical criteria that address the medical fitness of operating a private vehicle. Most state driving regulations address medical

fitness, but some such as California and Texas specifically address sleep apnea, listing it as a condition that if uncontrolled, prohibits driving.<sup>28</sup>

### Sleep Apnea and Other U.S. Transportation Modes

Other transportation modes in the United States have their own specific regulations and guidelines concerning workers with OSA. For example, the Federal Aviation Administration (FAA) does not permit an aviation medical examiner (AME) to make an initial determination on a pilot of any class with OSA.<sup>29</sup> The pilot can apply for a special issuance from the FAA. All pertinent medical information and a current status report must be submitted. A polysomnogram, discussion on use of medications, and any treatment and continuous positive airway pressure (CPAP) titration study results must be included in this report. Once the FAA has issued an Authorization for a Special Issuance, the AME may reissue the airman medical certificate provided the pilot provides a current report (performed within the last 90 days) from the treating physician. The report must reference the present treatment and whether it has eliminated any symptoms and include specific comments regarding daytime sleepiness. If there is any question about the pilot’s response or compliance with treatment, a maintenance of wakefulness test (MWT) is required. The AME is also instructed to defer to the Aeromedical Certification Division or a regional flight surgeon if:

- There is any question concerning the adequacy of therapy;
- The applicant appears to be non-compliant with therapy;
- The MWT demonstrates sleep deficiency; or
- The applicant has developed some associated illness such as right-sided heart failure.

If a pilot undergoes a surgical procedure as treatment for OSA, a MWT is required before returning to flying. The FAA does not permit the



use of modafinil in commercial airmen (Warren Silberman, DO, MPH, personal communication, November 4, 2005).

Although the Federal Railroad Administration (FRA) does not have medical standards that specifically address sleep apnea, a safety advisory was issued in 2004 as the result of a 2001 collision between two trains near Clarkston, Michigan. The NTSB determined that the probable cause of the accident was crewmember fatigue, primarily due to the engineer's untreated and the conductor's insufficiently treated sleep apnea.<sup>30</sup> The FRA alerted the railroad community to the dangers associated with undiagnosed or unsuccessfully treated sleep disorders and offered recommended actions to promote fitness. In addition to the Clarkston rail accident, the NTSB has also indicated that undiagnosed or insufficiently treated OSA may have contributed to several highway,<sup>31–33</sup> marine,<sup>34</sup> and rail<sup>35,36</sup> accidents.

### International Guidelines for Commercial Drivers With Obstructive Sleep Apnea

There are several international guidelines to help determine the medical fitness of employees to operate motor vehicles. The Canadian Medical Association (CMA) published *Determining Medical Fitness to Drive: A Guide for Physicians*.<sup>37</sup> This guide, which covers both private and commercial vehicle operators, recommends that drivers who report excessive somnolence and a history consistent with a sleep disorder be evaluated in a sleep laboratory. Those who have been involved in an at-fault MVC within the preceding 12 months should be questioned by their physician about excessive somnolence. The presence or absence of the following risk factors should also be assessed: more than 40 years of age, chronic heavy snoring, witnessed apnea, uncontrolled hypertension, significant cardiovascular disease, morning headaches,

craniofacial abnormalities, or obesity. Those with excessive somnolence and one or more of these risk factors should be evaluated for OSA. A driver whom a physician feels is likely to have a sleep disorder and who refuses evaluation should not be permitted to drive any class of vehicle. The CMA indicated that patients with mild OSA could be treated through behavioral modification (such as weight reduction, change in sleeping position, eliminating alcohol and sedatives before sleep) or by the use of oral appliances, but they would require reassessment for efficacy of treatment before resumption of driving. If treatment with CPAP is undertaken, compliance should be objectively assessed at 1 to 2 months after diagnosis. If the treatment is uvulopalatopharyngoplasty (UPPP) or other surgery, a repeat sleep study should be considered.

Canadian drivers who have documented OSA through a sleep study and are compliant with CPAP or have had successful UPPP treatments are considered safe to drive any type of motor vehicle. Those with moderate to severe OSA documented by sleep study and who are *not* compliant with treatment should not drive any type of motor vehicle. Patients with a high AHI, especially if associated with right heart failure or excessive daytime somnolence, should be considered at high risk for MVCs and not permitted to drive. Drivers who are thought to be compliant with treatment but are in a MVC in which they were at fault should not drive for at least 1 month.

These recommendations have been adopted by the Canadian Council of Motor Transport Administrators (CCMTA) in their CCMTA Medical Standards for Drivers,<sup>38</sup> which are part of Canada's National Safety Code. These medical standards have been adopted by most, but not all, Canadian provinces. The standards state that a driver with OSA "may operate any class of vehicle after the con-

dition has been adequately treated and controlled, subject to continued medical surveillance."

Australia also has guidelines for both private and commercial drivers. Austroad, the association of Australian and New Zealand road transport and traffic authorities, recommends that patients in whom sleep apnea is suspected or those with excessive sleepiness should be referred to a sleep specialist for further assessment, including an overnight polysomnogram.<sup>39</sup> The ESS is included in the driver history questionnaire. Drivers, especially CMV operators, who are considered high risk, eg, those with severe daytime sleepiness, a history of frequent self-reported sleepiness while driving, MVCs caused by inattention or sleepiness, or an ESS score above 16, should be referred for evaluation. CMV drivers who are diagnosed with OSA and require treatment are advised to have annual review by a sleep specialist. They should not be permitted to drive until treatment is effective. It is also advised that consideration should be given to how the driver would be able to comply with treatment while on the road. For those on CPAP, it is recommended that they use a machine with a compliance meter. Objective assessment of sleepiness, either an MWT or an MSLT, should be considered, especially if there is a concern about residual sleepiness or compliance. Medical determinations for rail workers in safety-sensitive positions are found in the *Code of Practice for Health Assessment of Rail Safety Workers*<sup>40</sup> and essentially mirror the guidelines for drivers.

In the United Kingdom, commercial drivers with OSA are required to stop driving until symptoms are satisfactorily controlled and ongoing compliance with treatment is confirmed by a specialist.<sup>41</sup> An annual review is required, but there are no other specific criteria.

## Identification and Diagnosis of Patients at Risk for Sleep Apnea

### History and Physical Predictors of Obstructive Sleep Apnea

There are some physical findings and a few historical facts that would normally be elicited during the commercial driver medical examination that might suggest the diagnosis of OSA. The only portion of the examination directly related to OSA is the question of whether the driver has any “sleep disorders, pauses in breathing while asleep, daytime sleepiness, or loud snoring.”<sup>25</sup> Unfortunately, in informal surveys conducted at multiple CDME educational offerings over that past 5 years by one of the authors, it was found that few drivers are currently responding “yes” to this question. If the driver is accompanied by a bed partner, the examiner should also ask that person if they have noticed any snoring or any gasping or pauses in breathing. In any driver, but especially in those with history or physical findings suggestive of OSA, the examiner should inquire about MVCs and try to determine if these crashes might have been due to fatigue or inattention because a history of sleep-related crashes is a high predictor of future sleep-related crashes.

There are several risk factors for OSA, the strongest being obesity and age.<sup>42,43</sup> The prevalence of OSA increases with age, with a two- to threefold higher prevalence in individuals over 65 compared with those in middle age.<sup>44</sup> The body mass index (BMI)—weight in kg/height in m<sup>2</sup>—should be determined in all commercial drivers because there is a relationship between a BMI of >26 kg/m<sup>2</sup> and OSA.<sup>45</sup> A recent study suggested that BMI should be a first-line screening criteria in professional drivers.<sup>46</sup> In this study, a relationship between BMI and AHI as well as between BMI and excessive daytime sleepiness, as measured by an

MSLT, was found. The study also found that obese truck drivers in their 20s did not have apnea, whereas individuals with class 2 or 3 obesity (BMI  $\geq 35$  kg/m<sup>2</sup>) who were 50 to 59 years of age had a higher prevalence of OSA. In this population, it appears that the prevalence of severe apnea was a function of both age and BMI.<sup>17</sup>

Examiners should also assess neck circumference because it has also been associated with increase risk of OSA.<sup>47,48</sup> In males, a neck circumference of >17 inches has been highly correlated with OSA<sup>49,51</sup> as has a neck circumference of >16 inches in a woman.<sup>50,51</sup> Neck circumference may correlate better with OSA than BMI, and there may be a relationship between neck circumference and the severity of OSA.<sup>51</sup> Examiners should also examine craniofacial features. Those features that have been shown to increase the risk for OSA include a high and narrow hard palate, an elongated soft palate, small chin, and an abnormal distance between upper and lower incisors. Tonsillar enlargement and narrowing of the airway by the lateral pharyngeal walls and a receding chin are also predictive of OSA.<sup>49</sup> Risk may increase with a positive family history of OSA.<sup>52,53</sup>

Although male gender is considered a risk for OSA, the diagnosis is often not considered in women,<sup>54</sup> especially if the BMI is <30 kg/m<sup>2</sup>. The male to female ratio in OSA patient populations (8:1) differs significantly from that in population-based studies in which OSA has not been diagnosed (2:1), suggesting that women with OSA are less likely to be evaluated and diagnosed.<sup>47,50</sup> The likelihood of OSA rises significantly after menopause to a 1:1 ratio.<sup>55,56</sup>

OSA appears to be an independent risk factor for hypertension,<sup>57–64</sup> but this relationship may be related to age, gender, BMI, smoking, or alcohol use. The Sleep Heart Health Study found a linear relationship between hypertension and the severity of OSA.<sup>60</sup> In their seventh report

(JNC VII), the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure listed OSA first on the list of identifiable causes of hypertension.<sup>63</sup> In one study of patients with resistant hypertension (poorly controlled despite the use of 3 or more antihypertensive agents), unsuspected sleep apnea was noted in 83%.<sup>64</sup> Because of this relationship, drivers who are hypertensive and have other risk factors should be considered to be at increased risk of having OSA.

OSA is also associated with diabetes, coronary artery disease, myocardial infarction, congestive heart failure, and stroke,<sup>65</sup> and the CDME should be highly suspicious of an increased risk for OSA in drivers with these conditions. Although, based on history and physical findings, an examiner may have a high suspicion that a driver has OSA, more definitive evaluations should be performed.

### Screening Questionnaires and Algorithms

Numerous tools exist for identifying those at risk for sleep apnea. The majority of these instruments use self-reported symptoms combined with demographic and anthropometric variables to discriminate between patients with and without OSA,<sup>66–81</sup> although two instruments use only anthropometric/physical examination findings<sup>78,79</sup> (details of evaluations of such instruments can be found in Table 2). In general, when compared with polysomnography, the models have reasonable sensitivities (generally >80%) but poor specificities (generally <60%). Therefore, the likelihood ratios are in a range (0.2–5.0) that does not significantly increase or decrease the posttest probability of the presence of OSA, particularly in populations with a high pretest probability of disease,<sup>77</sup> eg, truck drivers.

A significant problem with the studies investigating these tools is

**TABLE 2**  
Articles on Use of Predictive Models to Detect Obstructive Sleep Apnea

Author, Year	No. Subjects (men/women)	Apnea-Hypopnea Cutoff	Percent of Subjects With Obstructive Sleep Apnea	Factors in Predictive Formula	LR(+)/LR(-)	Comments
Crocker et al, 1990 <sup>66</sup>	Development group: 100 not given Validation group: 105 not given	15	Development group: 27 Validation group: 34	Witnessed apneas, hypertension, BMI, age	Development group: 2.18/0.25 Validation group: 1.9/0.2	1. Unclear if any women tested 2. States patients were consecutive; no exclusion criteria given
Williams et al, 1991 <sup>67</sup>	36 Not given	10 (see #3 in comments)	64	Snoring, witnessed apneas, excessive daytime sleepiness, BMI, hypertension	3.22/0.34	1. Unclear if any women tested 2. Not clear if patients were consecutive or if exclusion criteria applied 3. Used apnea index >10 as cutoff; hypopneas not scored 4. Index derived empirically 5. Study also included oximetry component; LR(+) = 0.0; LR(-) = 0.25
Viner et al, 1991 <sup>68</sup>	410 338/72	10	46	Snoring, BMI, age, gender	1.31/0.21	1. Few women included in study 2. States patients were consecutive; no exclusion criteria given 3. Tested clinician impression of presence of OSA: LR(+) = 1.73, LR(-) = 0.69; indicates physicians were less sensitive but more specific in their impression of OSA 4. AUC of the ROC = 0.77.
Gyulay et al, 1993 <sup>71</sup>	98 77/23	15	44	Witnessed apneas, hypertension, BMI, age	1.51/0.58	1. Compared clinical assessment, Crocker equation, and home oximetry 2. Few women studied 3. Not clear if patients consecutive; did exclude patients with significant chronic lung disease 4. Clinical assessment: LR(+) = 1.58, LR(-) = 0.46 5. Oximetry assessment showed that different indices could either exclude or include OSA
Kump et al, 1994 <sup>70</sup>	465 228/237	5 if age <15 10 if age 15-50 15 if age >50	34	Snoring intensity, witnessed apneas, fallen asleep while driving, BMI, gender, age	Only ROC data given	1. Participants were from an ongoing genetic-epidemiologic study of OSA 2. Included large numbers of women and blacks 3. Sleep studies were home portable studies, not in-laboratory studies 4. Factor analysis performed for 44 items related to sleep and OSA. 5. ROC for snoring intensity alone, 0.76; for snoring/ witnessed apneas/fallen asleep driving, 0.78, not significantly different from snoring alone; ROC for symptoms with demographics, 0.87, significantly increased from symptoms alone 6. Cannot be used clinically as exact predictive equation(s) not given
Flemons et al, 1994 <sup>69</sup>	180 133:67	10	46	Snoring, gasping/ choking, hypertension, neck circumference	5.17/0.25	1. Few women included in study 2. 63 subjects excluded for specific criteria given in report 3. Model calculates a sleep apnea clinical score (SACS), which predicts the PSG-derived AHI 4. Different SACS scores used to calculate the LR(+) (SACS ≥15) and LR(-) (SACS ≤5)

(Continued)

TABLE 2.  
(Continued)

Author, Year	No. Subjects (men/women)	Apnea-Hypopnea Cutoff	Percent of Subjects With Obstructive Sleep Apnea	Factors in Predictive Formula	LR(+)/LR(-)	Comments
Maislin et al, 1995 <sup>72</sup>	427 not given	10	60	Snoring, gasping, witnessed ap- neas, BMI, age, gender	1.96/0.22	1. Multicenter trial (3 sites) 2. Patients only consecutive from one site, not indicated if exclusion criteria applied 3. Women were clearly included but the exact number of included women cannot be de- termined from the paper 4. Blacks well-represented in group 5. Study included a confirmatory factor analysis of responses from 928 subjects to determine that the questionnaire was reliable and valid 6. Most of the predictive ability of the model came from the BMI (AUC of ROC for symp- toms alone, 0.695; for BMI alone, 0.734; for whole model, 0.786) 7. Model has been subsequently combined with oximetry to create a two-step algorithm for predicting the presence of OSA
Pradhan et al, 1996 <sup>73</sup>	150 107/43	10	57	Snoring, age, gen- der, BMI	1.19/0.0	1. Not clear if patients were consecutive or if exclusion criteria applied 2. Few women included 3. Specifically maximized sensitivity to 100% to determine cutoff value 4. Study also included oximetry component; combined model LR(+) = 1.45; LR(-) = 0.0
Kushida et al, 1997 <sup>79</sup>	300 224/76	5	85	Neck circumference, BMI, palatal height, maxillary intermolar dis- tance, mandibular intermolar dis- tance, overjet	∞/0.02	1. Morphometric model only; no symptoms included in development or validation of model 2. Model developed in 30 patients: 15 with OSA 3. Model tested in consecutive patients; no apparent exclusion criteria for comorbidities 4. Women and minorities included but in low numbers 5. Most of the predictive ability came from the BMI: AUC for the model was 0.996; for BMI alone, 0.938; for NC alone, 0.898. AUC values for BMI and NC higher than in other investigations
Netzer et al, 1999 <sup>74</sup>	100 not given	5		Snoring, excessive daytime sleepi- ness, BMI, or hy- pertension	3.79/0.18	1. Primary care population, not sleep center population 2. 774 patients for analysis of the questionnaire; sleep studies performed on 100 patients, with enrichment for high-risk group 3. Genders equally represented for the questionnaire component; article states that gen- der representation is similar for the sleep study portion, but exact data not given 4. Criteria for risk groups based on results of previous studies (including Flemmons, Kump, Maislin) not from an analysis of responses compared with sleep study results 5. Criteria for high- vs low-risk groups based on number of positive responses to symp- tom-based questions; predictive equation not created 6. Sleep studies were portable studies, not standard sleep studies

(Continued)



**TABLE 2.**  
(Continued)

Author, Year	No. Subjects (men/women)	Apnea– Hypopnea Cutoff	Percent of Subjects With Obstructive Sleep Apnea	Factors in Predictive Formula	LR(+)/LR(-)	Comments
Rowley et al, 2000 <sup>77</sup>	370 191/179	10	67	Witnessed apneas, snoring, gasping/ choking, hyper- tension, BMI, age gender, neck circumference	See comments	1. Study comparing prediction models from Crocker, Viner, Flemmons, and Maislin 2. Consecutive patients without exclusion criteria, but 13% of enrolled 425 patients did not have PSG 3. Women and minorities well represented 4. LR(+) ranged from 1.1–1.7; LR(-) ranged from 0.3–0.4 5. All models performed better in men than women as measured by the AUC of the ROC (men, 0.707–0.801 vs women, 0.611–0.648). 6. Also tested the models for AHI $\geq 20$ ; LR(+) ranged from 2.6–5.6; LR(-) ranged from 0.7–0.8
Tsai et al, 2003 <sup>78</sup>	75 56/19	10	48	Pharyngeal grade, cricomental space, overbite	10.0/0.63	1. Not clear if patients were consecutive 2. Low percentage of women 3. Sleep studies were portable studies, not standard sleep studies 4. Cricomental space alone: LR(+) = 1.85, LR(-) = 0.0 5. Created a decision rule starting with the cricomental space: if $>1.5$ cm, 100% negative predictive value, cannot exclude OSA; if $\leq 1.5$ cm, proceed to pharyngeal grade/overbite 5. Validated in an additional 50 patients with a positive predictive value of 100%
Hussain/ Fleetham, 2003 <sup>76</sup>	30 15/15	15	40	Snoring, gasping/ choking, hypertension, neck circumfer- ence	1.74/0.26	1. Small study comparing the Flemmons prediction rule with oximetry 2. Study designed to look at value of negative oximetry (oxygen desaturation index $<10$ ) to rule out significant OSA (AHI $\geq 15$ ) 3. Equal number of men and women 4. Oximetry: LR(+) = 2.75; LR(-) = 0.76
Rodsutti et al, 2004 <sup>75</sup>	Development group: 837 571/266 Validation group: 243 153/90	5	Development group: 68 Validation group: 72	Gender, age, BMI, snoring, stopped breathing	Only ROC data given	1. Large number of women compared with other studies 2. Patients consecutive; no exclusion criteria given 3. AUC of ROC for development phase: 0.809; for validation phase: 0.789 4. Few patients placed into the low-risk group by the model; however, most were true-negatives for OSA 5. Most patients placed into high-risk group by model; most were true-positives for OSA 6. Final rule provided as a simple table with color coding for the different risk groups (low, moderate, high)

AHI indicates apnea-hypopnea index; OSA, obstructive sleep apnea; BMI, body mass index; NC, neck circumference; LR(+), likelihood ratio for a positive test; LR(-), likelihood ratio for a negative test; AUC, area under the curve; ROC, receiver operator characteristic; PSG, polysomnography.

that the instruments have typically been compared with polysomnography rather than with functional outcomes. Another limitation is that the majority of models were created in sleep clinic populations; therefore, they were specifically created to determine which patients who had symptoms suggestive of OSA were most likely to have OSA. Thus, the models may have limited use and validity in broader populations of patients, including CMV drivers. (Two screening tools were created using data from nonsleep clinic populations, one from an epidemiologic cohort<sup>70</sup> and one from a primary care population.<sup>74</sup>) Other limitations of the studies include single-center studies in the majority, data from limited numbers of women and minorities included in the model, and uncertainty about patient recruitment methods. The limitations of screening models were shown in a study by Rowley in which four of the models were prospectively tested in a consecutive group of 370 patients referred to a sleep center.<sup>77</sup> In this study, no patients were excluded, a large number of women were enrolled, and the pretest probability was high (67%). In general, the models had lower likelihood ratios than in the original studies (positive likelihood ratios 1.1–1.7). Therefore, the clinical prediction models tested may not be sufficiently accurate to discriminate between individuals with or without OSA. Lower likelihood ratios have also been found in other studies in which a prediction model was used in a population other than the original.<sup>71,76</sup> Of note, however, is that Rowley found that prediction models may be more accurate in identifying individuals with more severe sleep apnea (AHI >20; positive likelihood ratios 2.6–5.6). Thus, one application of prediction models could be to identify patients at the highest risk of OSA with further testing or empiric treatment performed just on the high-risk group. This approach has been studied by one group in CMV drivers in Pennsylvania,

which combined the questionnaire prediction model with overnight oximetry, resulting in improved sensitivity and specificity.<sup>80,81</sup>

*The Berlin Questionnaire.* One well-studied, self-report instrument is the Berlin Questionnaire, which resulted from a 1996 consensus conference of primary care physicians who were presented with the risk factors for sleep apnea.<sup>42,74</sup> The Questionnaire addresses three known risk factors for sleep apnea: snoring history, tiredness, and history of high blood pressure and/or a BMI of >30 kg/m<sup>2</sup>. The determination of a “high risk” (high pretest probability) and “lower risk” (low pretest probability) for sleep apnea is based on the frequency of these symptoms and on traits. To be “high risk,” a person must have reported persistent symptoms (>3–4 times/week or every day) on two or more questions about snoring (category 1) or waketime sleepiness and/or drowsy driving (category 2) or persistent symptoms in either category 1 or category 2 and at least one feature (history of high blood pressure or BMI >31 kg/m<sup>2</sup>) in category 3. Respondents who deny chronic symptoms or have frequent symptoms or signs in only one category are placed in the “lower risk” group. In combination with portable monitoring, one study of a primary care population reported a sensitivity of 0.77, a specificity of 0.89, and a positive predictive value of 0.71.<sup>74</sup> In an assessment comparing the Berlin Questionnaire with polysomnography in a population of cardiology patients, the sensitivity was 0.86, specificity 0.87, and positive predictive value 0.97.<sup>82</sup>

The Berlin Questionnaire was also used in a survey of 10,101 truck drivers.<sup>7</sup> Approximately 26% of these truck drivers were found to be at high risk for sleep apnea, although this was not confirmed with objective testing. Approximately 15% of the drivers self-reported falling asleep while driving. In an adjusted multiple logistic model, smoking and drug use were associated with high

risk for OSA, whereas self-reported occasional and regular physical activity was an independent factor protective of OSA. The authors concluded that programs aimed at engagement in physical exercise could be considered to reduce OSA risk. It is important to note that this study was conducted independent of evaluation for medical certification and thus did not affect work status.

*Screens of Sleepiness and Sleep Efficacy.* Two commonly used screens for sleepiness and sleep efficacy are the ESS and the Functional Outcomes of Sleep Questionnaire (FOSQ). Some studies have found that these tools have predictive value for car crashes, at least when applied to individuals in research conditions.<sup>1</sup> Subjective sleepiness is commonly assessed clinically with an ESS.<sup>83</sup> The ESS correlates with measures of sleep-disordered breathing and is more likely to be elevated in individuals with sleep disorders than in those with no sleep pathology. A total score below 10 points (out of 24) is typically reported as normal. Although subjective, ESS scores correlate with pathology and improve with effective treatment of sleep apnea.<sup>84</sup> Recent retrospective data from the Sleep Heart Health Study found that more than 50% of individuals with moderate to severe OSA (AHI >15) were not subjectively sleepy as assessed by the ESS.<sup>60</sup> Therefore, a normal ESS score (<10) does not rule out OSA.

The FOSQ is a 30-item, self-report instrument to assess the functional consequences of daytime sleepiness.<sup>85,86</sup> A total score can be calculated from five subscale scores. The measure has demonstrated internal reliability ( $\alpha = 0.95$  for the total scale and 0.86–0.91 for the five subscales) and test-retest reliability ( $r = 0.90$  for the total measure and 0.81–0.90 for the five subscales). The FOSQ also predicted driving accidents in CMV drivers.<sup>1</sup> A number of controlled studies have demonstrated the ability of the FOSQ to detect treatment response. In patients

with OSA, this instrument measured differences between interventions with CPAP and placebo<sup>87,88</sup> conservative treatment,<sup>89</sup> and oral appliances.<sup>90,91</sup> The FOSQ also demonstrated differences in treatment response between placebo and radiofrequency ablation treatment.<sup>92</sup>

**Multivariate Apnea Predictor.** The Multivariable Apnea Prediction (MAP) Questionnaire is a tool used to identify persons at high risk for OSA. This tool was validated in a sleep clinic population<sup>71,81</sup> and also in general groups, particularly CMV drivers.<sup>80</sup> This tool relies on a combination of sources of information: the frequency of having apnea-related symptoms, BMI, age, and gender. Because BMI is in itself such a powerful predictor of the presence of OSA, the MAP score is particularly useful in situations when obesity is not present.

In the MAP questionnaire, subjects are asked three questions in the following way: "During the past month, have you had, or have you been told about, the following symptoms: 1) snorting or gasping; 2) loud snoring; and 3) breathing stops, choking, or struggling for breath?" Respondents rate symptom occurrence as: never (0); rarely, less than once/week (1); once or twice/week (2); three or four times/week (3); five to seven times/week (4); or don't know. Each rating is associated with a numeric score. These symptom-frequency scores are averaged together with a potential range of 0 to 4. For example, when all symptoms are present five to seven times a week, the average symptom score is 4. This symptom score is then combined with BMI, age, and gender to determine the multivariable prediction score.<sup>72</sup> These scores range from 0 to 1, with 0 representing no risk and 1 representing maximal risk for OSA.

Questionnaire-based tools rely heavily not only on subjective experience, but also on the willingness of the person queried to report symptoms with full accuracy. Concerns

that such reporting may not be accurate in an occupational setting may limit the usefulness of the symptom-based MAP. However, in the research setting, the MAP data were quite useful in predicting apnea frequency with reasonable accuracy<sup>80</sup> because privacy and confidentiality were guaranteed. An alternative MAO tool that does not rely on symptom data is currently being developed for the CMV driver population. It should be noted that the MAP was not useful in discriminating between patients with and without OSA in a population with a high pretest probability of OSA and, therefore, should be used cautiously in such a population.<sup>78</sup>

**Comment.** It is important to remember that the screening tests and algorithms discussed were based on sleep center validation studies, which by their nature are enriched for sleep-disordered breathing and are less common than those encountered in the general population or in the trucking community.

### Polysomnography

The current gold standard to confirm the diagnosis of OSA is nocturnal polysomnography (PSG). This test monitors physiological signals from various organs and transduces those signals to a recording device. Although it is the current standard, errors can occur and these include data loss, artifacts, and intra- and interrater variability in scoring events. Additionally, different sleep laboratories use different types of monitoring equipment, which may change the sensitivity of event recognition. However, given these limitations, most sleep laboratories use similar montages for baseline PSG, which include central and occipital electroencephalogram (EEG) leads based on the International 10/20 system; electrooculographic (EOG) leads to monitor eye movements; chin (genioglossus) and leg (anterior tibialis) electromyography leads to monitor activity of those muscles; a modified lead II of a standard elec-

trocardiogram to monitor the heart signal; an airflow signal, with most sleep laboratories using either or both a nasal thermistor and a nasal cannula pressure transducer; a measure of respiratory effort of the chest and abdomen with either respiratory inductance plethysmography or impedance pneumography; and a pulse oximeter to monitor oxygen saturation continuously. In addition, the American Academy of Sleep Medicine (AASM) Practice Parameters on PSG<sup>93</sup> recommend a measure of body position, which may be a sensor placed on the patient or the more commonly used continuous video recording to observe the patient. Infrared lighting is used to allow adequate viewing with maintenance of a dark environment. These 11 items are considered the minimum items required to perform PSG for diagnosis of OSA.<sup>93</sup> A 6-hour minimum duration of a diagnostic PSG is preferred, allowing for the assessment of variability related to sleep stage and position with respect to the frequency of obstructive respiratory events.<sup>94</sup> The exception to this is in the case of a split-night study in which a patient demonstrating severe OSA may be initiated on nasal CPAP after only 2 hours of study.

Once acquired, PSG should be analyzed first by a person trained in the technical aspects of PSG scoring and then by a physician with experience in sleep medicine. Sleep stages are scored in 30-second epochs according to the criteria set forth by Rechtschaffen and Kales.<sup>95</sup> All the respiratory signals should be reviewed and breathing events counted. Breathing events can be broken down into apneas, hypopneas, and respiratory-effort related arousals (RERA). Apneas are further broken down into obstructive (continued respiratory effort), central (no respiratory effort), or mixed (both obstructive and central components) events. Although most sleep laboratories agree on a standard definition for an apnea (greater than or equal to 10 seconds of absent airflow), the defi-

nition of a hypopnea remains controversial. The AASM's Clinical Practice Review Committee criteria for an hypopnea is a 30% reduction in effort or airflow lasting for 10 or more seconds occurring with a 4% desaturation.<sup>96</sup> Many laboratories however, will also count hypopneas if the reduced effort or airflow is accompanied by an arousal from sleep; some laboratories count such events separately and call them RERAs. Therefore, when reviewing studies from sleep laboratories, it is helpful to understand their scoring criteria.

Once a PSG is scored, the frequency of disordered breathing events is usually quantified by creating an index. This index may be referred to in a variety of ways; however, the most common index used is the AHI, which is derived by adding the total number of apneas plus the total number of hypopneas and dividing it by the total sleep time in hours. Other terms used include the respiratory disturbance index (RDI), disordered breathing index, and an oxygen desaturation index.

Because there is variability in acquisition and scoring of PSG, there is also no consensus on what minimum AHI constitutes disease. In a 1999 consensus statement, it was suggested that CPAP treatment is indicated for all patients with OSA with an AHI of  $\geq 30$  events per hour and for patients with an AHI of 5 to 30 if accompanied by symptoms of excessive daytime sleepiness, impaired cognition, mood disorders, insomnia, or documented cardiovascular diseases to include hypertension, ischemic heart disease, or stroke.<sup>94</sup> The AASM Practice Parameter states that CPAP treatment is indicated for all patients with OSA with an AHI of  $\geq 15$  and for those with an AHI of 5 to 14 if excessive daytime sleepiness is present.<sup>94</sup> The CMS criteria to provide CPAP is an AHI of  $\geq 15$  or an AHI of 5 to 14 with documented symptoms of excessive daytime sleepiness, impaired cognition, mood disorders or insomnia, documented

hypertension, ischemic heart disease, or a history of stroke.<sup>23</sup>

Studies reviewed within this document use varying criteria as well. The problems with using AHI criteria to define severity include the variability between sleep laboratories in defining/scoring respiratory events<sup>97,98</sup>; the fact that the AHI does not account for length of events or event association with differing degrees of oxygen desaturation; and that AHI does not correlate well with degree of sleepiness or other measures of driving risk such as number of crashes,<sup>99</sup> "Steer Clear" scores,<sup>100</sup> tracking errors, and response time.<sup>101</sup> In contrast, the degree of AHI has been shown to correlate with the prevalence of cardiovascular disease such as hypertension and stroke.<sup>61,102</sup>

### Portable Monitoring

Portable monitoring, defined as a sleep study done outside of the sleep laboratory, can be done with the same leads that in-laboratory PSG uses. This type of monitoring, full unattended PSG in the home, was performed in the Sleep Heart Health study, a National Institutes of Health-sponsored study with more than 6000 participants. However, the emphasis for home diagnosis of sleep apnea has focused on limited channel monitoring. These monitors center on the respiratory leads and do not include the EEG or leg leads. Some of the considerations for using limited channel systems include ease of attachment (must be easy if a patient is to attach themselves to the leads), permanence of attachment (so there is no data loss), likelihood of technical problems because the study is unattended, and absence of data on sleep (unsure about how much of the night the patient was actually asleep).

A key difference between PSG and portable monitors is the calculation of disordered breathing events. PSG-derived AHI is divided by the total sleep time. Portable monitors

deviate from the standard polysomnographically defined AHI because the total sleep time is not able to be ascertained. Therefore, total recording time is the denominator, which dilutes the severity, and arousals from sleep cannot be determined. Thus, if arousals are used to score respiratory events, the definition of hypopnea is changed.

Portable monitoring was first examined in a systematic way by the AASM (then known as the American Sleep Disorders Association). In its 1994 standards of practice,<sup>103</sup> the AASM divided sleep monitoring into four levels:

- Level 1: attended PSG in the sleep laboratory setting;
- Level 2: portable PSG outside the laboratory setting (monitors the same channels as level 1 but not in a sleep laboratory);
- Level 3: limited channel PSG ( $\geq 4$  leads, but usually  $< 8$ ); and
- Level 4: one or two channels (usually oximetry alone or with one other channel such as airflow).

*Level 2 Monitors.* These portable monitors will not be discussed because they are not commonly used outside of research studies. In 1998, a literature review and meta-analysis was done under the sponsorship of the Agency for Health Care Quality and Research.<sup>104</sup> This review also found insufficient data to support the use of portable monitoring in the diagnosis of OSA.

*Level 3 Monitors.* Most recently, a trisociety committee performed an evidence review and prepared practice parameters.<sup>105,106</sup> In the evidence review, the level 3 and 4 monitors were grouped by whether the study was attended by a technician or unattended (as would likely occur in the home). The committee concluded that attended level 3 monitors are acceptable to both rule in and rule out OSA but with limitations such as: raw data analysis is recommended, limitation of different devices must be understood, and patients with comorbidities must be



handled with caution. Unattended level 3 monitors were not recommended to rule in or rule out OSA. A supplemental literature review later conducted by the AASM to capture any studies published through May 2004 (after the literature review) did not change any of these conclusions.

**Level 4 Monitors.** The most widely used level 4 monitoring system is oximetry. In the trisociety documents, it was suggested that studies used a variety of criteria to detect sleep-disordered breathing (SDB), including desaturation indices at different  $\Delta\text{SaO}_2$  cutoffs (2–4%), time spent with a saturation <90%, and a delta index, which describes the variability of the oxyhemoglobin saturation over the course of the night.<sup>103,104</sup> In addition, the primary limitation of oximetry is the devices themselves. Different oximeters have different sampling frequencies (range from 0.08–10 Hz) and algorithms used to record oxygen saturation. Memory storage and methods of automated analysis of the oxygen saturation also vary between devices.<sup>107,108</sup> Because of these limitations and the fact that there is limited data for use of oximetry in the unattended environment, recent guidelines state that oximetry is not an acceptable diagnostic modality for diagnosing or excluding OSA.<sup>105</sup>

### Summary: Polysomnography and Portable Monitoring

PSG is the current standard for making an objective diagnosis of OSA. The severity of OSA as measured by a polysomnographically derived AHI does not correlate well with measures typically associated with poor driving. Portable monitoring is not yet accepted in the United States as a surrogate diagnostic strategy for OSA, but ongoing efforts will likely result in its acceptance in the near future for some patients with utilization of diagnostic algorithms.

## Objective Assessment of Sleepiness and Performance

### Multiple Sleep Latency Test

Objective measures of sleepiness such as the MSLT attempt to quantify the physiological tendency to fall asleep. Sleepiness is tightly regulated by both homeostatic factors (the duration of prior sleep) and circadian factors (timing of sleep across the 24-hour day). MSLT is based on the premise that the more sleepy a person is, the faster she or he will fall asleep and, recognizing the circadian variation in sleepiness, it measures latency to sleep at preset intervals across the day. The test context controls for and removes/reduces alerting factors such as temperature, noise, ambient light, activity, and motivation. MSLT can be affected by physiological factors such as age,<sup>109–111</sup> circadian rhythm,<sup>112</sup> quantity<sup>113,114</sup> and/or quality of previous sleep,<sup>115–118</sup> and the use of medications.<sup>119–124</sup> Psychologic factors such as anxiety, stress,<sup>125–127</sup> and depression<sup>128,129</sup> can also influence MSLT.

Although there is no known measurable biologic substrate of sleepiness, MSLT is the de facto standard objective measure of sleepiness and has face validity given its robust response to sleep deprivation and sleep extension and its sensitivity to disordered sleep as in sleep apnea. Standardized protocols exist for MSLT in both clinical and research settings.<sup>130,131</sup> There are very few studies that measure MSLT in relation to driving. Some simulator studies<sup>101,132,133</sup> show a modest relationship between MSLT and performance, but none address the issue of fitness to drive.

### Maintenance of Wakefulness Test

MWT measures the ability to stay awake for a defined period of time under conditions that promote sleep. Although both MSLT and MWT are ostensibly measuring pathologic sleepiness, it can be argued that the

ability to stay awake is a different behavioral component of sleepiness compared with the tendency to fall asleep, and the former is more important in certain occupations or conditions (ie, driving). Subjects are asked to remain awake in a quiet, darkened room and EEG is continually monitored for onset of sleep. Moreover, subjects have been studied sitting upright in a chair, lying in bed, or semirecumbent in bed. Despite the lack of uniformity in the literature, the AASM suggests a 40-minute protocol with the subject sitting upright in bed. Still, the MWT is susceptible to the same confounding factors as the MSLT (*vide supra*).

Because MWT assesses the ability to remain awake, this test has been suggested as one of the most important in areas of public transportation. For example, the FAA requires a normal MWT for pilots to be licensed after treatment of sleep apnea. However, when assessing fitness to fly, drive, or operate heavy equipment, there are no studies that use MWT. There are a few research studies that assess MWT in drivers as a measure of sleepiness. A study of 10 Finnish bus drivers with mild OSA ( $\text{RDI } 16 \pm 9.9$ ) reported a shorter MWT for untreated drivers compared with control drivers ( $23.2 \pm 10.2$  vs  $31.8 \pm 6.03$  minutes) and this correlated with eyeblink duration while driving, a measure of sleepiness.<sup>134</sup> Although both measures improved with CPAP treatment, this study did not directly address the issue of MVCs or fitness to drive. A case-control study of drivers involved in MVCs evaluated the role of sleepiness and sleep-disordered breathing in these crashes. Sleepiness was assessed both subjectively (using ESS) and by MWT.<sup>135</sup> Crash drivers demonstrated significantly more driver (subjective) sleepiness and a trend for greater objective sleepiness compared with well-matched controls. Again, this study did not specifically address the use of a particular cutoff level for sleep-

iness on the MWT as predictive of crashes or driving ability.

Determining ability to stay awake at any time on the MWT may have no relationship to when the subject is actually driving. Sleepiness can fluctuate hour-to-hour or day-to-day and can be influenced by multiple factors, including prior sleep, shift work, medications, and compliance with medical treatments (including CPAP). Therefore, it is not surprising that the predictive value of a MWT is poor. Moreover, although the MWT typically shows a high sensitivity to acute sleep loss, its relationship to less severe or chronic sleep loss may not be predictable.<sup>136</sup>

### Oxford SLEep Resistance Test

MSLT and MWT are labor-intensive and require continuous technician observation of EEG recording, making both tests expensive and cumbersome. The Oxford SLEep Resistance (OSLER) test is a low-cost alternative designed to reproduce many of the features of the MWT. Subjects respond to a light-emitting diode mounted on the wall which flashes for 1 second every 3 seconds. If there is no response after seven consecutive stimuli, the subject is deemed to be asleep and the test is ended. Limited data are available for this metric. The original study compared OSLER test sleep latency with MWT latency in 10 patients with OSA and 10 control subjects on separate days.<sup>137</sup> Two other studies involving 11 sleep disorders center patients<sup>138</sup> and 10 normal subjects before and after sleep deprivation<sup>139</sup> have demonstrated excellent agreement between the two measures and suggest that the OSLER test could be an alternative to measuring sleepiness. The main limitation of this test is its dependence on patient cooperation. In a study of 30 patients with heart failure receiving adaptive ventilation for treatment of Cheyne-Stokes respiration,<sup>140</sup> improvement in OSLER test scores followed improvement in nighttime sleep. Despite these promising results, there

are no large-scale studies using the OSLER test and none specifically involving drivers.

### Identifying Drivers With Sleep Apnea Who Are at High Risk for Crashes

Although the evidence strongly suggests that drivers with OSA are at an increased risk of MVCs, the majority of drivers with OSA are not involved in crashes. The challenge is identifying those that are most likely to be at higher risk. Studies have not been able to definitively identify those drivers with OSA who are at highest risk of crashes.<sup>3</sup> A recent small study from the FMCSA did not find an increased risk among commercial drivers, but that study was acknowledged to have several limitations, including incomplete data, self-reporting by participants, and a majority of the drivers working in short haul or local settings.<sup>141</sup> In the most comprehensive study of accidents and CMV drivers to date, Howard estimated, using both a screening questionnaire and PSG, that 50% of the more than 3000 CMV drivers surveyed were at risk for sleep apnea.<sup>1</sup> Scores above 18 on the ESS, scores below 18 on the FOSQ, time spent driving per week, interstate and country driving, narcotic use, antihistamine use, and younger age were associated with increased risk of crashes. Sleepiness increased the risk of accidents, but neither sleep apnea diagnosed by PSG nor suspected by MAP predicted increased risk of crashes. Teran-Santos found a significant relationship between an AHI of >10 and the risk of motor vehicle accidents.<sup>5</sup> This remained after adjustment for potential confounders such as alcohol consumption, visual-refraction disorders, BMI, years of driving, age, history with respect to traffic accidents, use of medications causing drowsiness, and sleep schedule. Hypopnea for this study was defined as a

“substantial decrease in oronasal airflow with desaturation (a decrease of at least 4% in saturation), arousal, or both.”

Drivers who have been in previous sleep-related crashes are at increased risk of recurrent accidents unless the risk is lowered through treatment. Some research suggests that an elevated AHI (>40) may be associated with a higher risk of crashes,<sup>142</sup> whereas others have found no correlation between AHI and driving simulator tests<sup>100,101</sup> or crashes.<sup>99</sup> Drivers who snore and report being sleepy appear to be at an increased risk of accidents,<sup>143</sup> but this would depend on an accurate reporting of subjective sleepiness. CDMEs commonly report that responses to the medical examination history questions often tend to underestimate or underreport medical conditions.

A literature review of the clinical use of MSLT and MWT concluded that sleep latencies were not correlated with crash history for most groups.<sup>131</sup> Drivers with OSA who had been treated with CPAP did perform better on both MSLT and MWT than they did before treatment and reported better driving performance. The AASM's practice parameters indicated that a MWT 40-minute protocol could be used in addition to clinical history and compliance with treatment to evaluate an individual's ability to remain awake when personal or public safety was at issue.<sup>130</sup>

### Psychomotor Vigilance Task

The psychomotor vigilance task (PVT) measures the ability to sustain attention, thus reflecting the arousal and attention state of the subject.<sup>144</sup> The 10-minute task measures repeated response to a small, bright-red light stimulus (light-emitting diode digital counter). Stimuli are displayed at 2- to 10-second intervals and an incrementing time counter for each stimulus is stopped by a press of a button. The PVT is sensitive to sleep loss with changes in reaction time and lapses in performance cor-

relating with increasing objective sleepiness.<sup>145</sup> Many studies use this metric to assess performance in a number of settings, and although PVT lapses after sleep deprivation followed the same temporal distribution as highway crashes,<sup>146,147</sup> few include it as an index of driving performance. In one of the few studies to relate PVT to crashes, 60 patients with sleep apnea had both clinical measures and laboratory performance measures (including PVT) assessed in an effort to predict motor vehicle accidents.<sup>99</sup> The study found that neither clinical data (depression, anxiety, or referred daytime sleepiness) nor markers of disease severity (AHI time below 90% SaO<sub>2</sub> at night and mean nocturnal SaO<sub>2</sub>) were related to the number of accidents. Patients with a worse reaction time or a higher reaction fatigue (inverse of reaction time vs time on PVT) appeared to have a slightly higher number of accidents, but differences did not reach statistical significance.

### Driving Simulators

Considerable research has focused on a variety of impairment measures in an effort to characterize and quantify impairments as a means to predict (and thereby prevent) motor vehicle collisions. The use of driving simulators as a means for measuring impairment has become popular because they provide a safe, controllable, and low-cost environment in which to assess the effects of sleepiness on driving. The literature on simulators and sleep disorders is relatively large, but the subjects and the degree of simulator complexity vary widely from one report to another.

One of the first reports of a “driving simulator” test in patients with sleep apnea was the Steer Clear test.<sup>148</sup> This is actually a choice reaction computer test, which evaluates the subject’s ability to maintain vigilance. Performance on this test is typically reduced in studies with small number of patients with sleep apnea or narcolepsy,<sup>149–151</sup> but the magnitude of the difference in per-

formance between patients and controls varies quite widely. None of these reports have shown any correlation between Steer Clear performance and accidents.

The Divided Attention Driving Test (DADT) includes a tracking task controlled by a steering wheel and a secondary visual search task. The tracking task is a variation of the subcritical tracking task, one of many psychomotor tasks developed to study performance and detect impairment due to fatigue, stress, or drug effects. This task has been shown to be sensitive to fatigue among truck drivers due to hours of work.<sup>41</sup> Studies using DADT have found variably reduced driving performance in patients with sleep apnea<sup>101</sup> and/or narcolepsy<sup>152</sup> and improvements with CPAP therapy.<sup>132</sup>

The Divided Attention Steering Simulator (DASS) is similar to DADT and is based on a tracking task and visual detection of digits located at the 4 points near the sides of a computer screen. The software reproduces a winding road shown as lines on the screen, and the tasks are to use the steering wheel to keep the front of the car in the middle of the road and not to go over the edge of the road. There are four studies in patients with OSA<sup>153–156</sup> and one study in normal subjects using DASS.<sup>157</sup>

Like with other tests, performance is worse in patients with OSA than in controls and improvement is achieved with CPAP therapy. The rate of improvement in DASS performance was formally assessed in one study of CPAP treatment in 18 patients and suggests that safe driving may be resumed after 7 days of successful treatment.<sup>156</sup> Only one study showed a very weak correlation with self-reported accidents.<sup>155</sup> The most recent study with this instrument compared on-road driving with DASS driving in a small ( $n = 12$ ) group of normal subjects under usual and sleep-restricted conditions.<sup>157</sup> This is the only study to suggest that deficits in driving performance are more pronounced in a

simulated environment than on-road, which may make simulators less useful in predicting real-world driving performance.

A number of other simulators (of varying complexity) have been tested either in patients or normal subjects under a variety of sleep conditions (see Table 3). In general, these tests provide consistent results: performance is worse under usual or sleep-deprived conditions, worse after using drugs or alcohol, and in the case of sleep apnea, better after treatment with CPAP or surgery (UPPP). Although these data show a consistent effect of sleepiness on simulated driving performance, there is still no good data link with on-road driving. As such, the clinical use of these simulators in day-to-day practice is unproven. A review of the literature on medical conditions and driving by the National Highway Traffic Safety Administration (NHTSA) concluded that “the results reveal that measures commonly used to measure disease severity in sleep apnea are not very useful in discriminating between individuals who are likely to perform poorly on laboratory based measures putatively related to driving performance or who are at risk for crashes.”<sup>158</sup>

### Management of Sleep Apnea in the Commercial Motor Vehicle Driver

#### Continuous Positive Airway Pressure

Since the initial description of nasal CPAP therapy as a treatment for OSA,<sup>170</sup> it has become and remains the mainstay of treatment for patients with OSA.<sup>171</sup> CPAP therapy has been demonstrated to resolve sleep-related breathing disorders and improve several clinical outcomes.

CPAP is conventionally delivered through a nasal mask at a fixed pressure, typically in the range of 8 to 12 cm H<sub>2</sub>O, which remains constant throughout the respiratory cycle. This pressure is applied by

**TABLE 3**

Driving Simulator Studies of Performance

Simulator Test	Study Author	Patient Type	No. of Subjects	Therapy	Outcome
Steer Clear	Findley, 1989 <sup>148</sup>	OSA; controls	OSA = 12; controls = 12	CPAP in 6 patients	OSA hit more obstacles than controls; fewer obstacles hit on CPAP
	Munoz, 2000 <sup>150</sup>	OSA; controls	OSA = 80; controls = 80	CPAP in 74 patients	OSA hit more obstacles than controls; fewer obstacles hit on CPAP
DADT	Kingshott, 2000 <sup>151</sup>	OSA	OSA = 62	CPAP	Fewer obstacles hit on CPAP
	George, 1996 <sup>101</sup>	OSA	OSA = 21; controls = 6	None	Tracking and reaction time worse in OSA
	George, 1996 <sup>152</sup>	OSA; narcolepsy	OSA = 21; narcolepsy = 16	None	Narcolepsy worse than OSA on all DADT measures
	George, 1997 <sup>132</sup>	OSA; controls	OSA = 17; controls = 18	CPAP	Improvement in tracking and correct responses but not reaction time after CPAP treatment
DASS	Hack, 2000 <sup>153</sup>	OSA	CPAP = 26; sham = 33	CPAP, subtherapeutic CPAP	Improvement in tracking and reaction time after active CPAP treatment
	Juniper, 2000 <sup>154</sup>	OSA; controls	OSA = 12; controls = 12	None	OSA worse on steering, off-road events and target response time
	Turkington, 2001 <sup>155</sup>	OSA	OSA = 150	None	Weak association of simulator off-road events and previous road traffic accident
	Turkington, 2004 <sup>156</sup>	OSA	OSA = 36	18 on/off CPAP; 18 untreated; both repeated testing over 2 wk	Improvement in simulator performance by 7 d; maintained for up to 7 d post-CPAP withdrawal
	Philip, 2005 <sup>157</sup>	Normals	Normals = 12	Comparison of simulator with on-road, observed driving after 2 or 8 hrs sleep	Changes in performance greater in simulated environment
STISIM	Risser, 2000 <sup>159</sup>	OSA; controls	OSA = 15; controls = 15	None	Lane position variability increases with time in OSA; correlates with EEG lapses rather than overt sleep onset
York Driving Simulator	Arnedt, 2000 <sup>160</sup>	Normals	Normals = 22	Conditions of prolonged wakefulness, ethanol, or both	Prolonged wakefulness and alcohol consumption produced greater decrements in performance than either alone
	Arnedt, 2001 <sup>161</sup>	Normals	Normals = 18	Conditions of prolonged wakefulness, ethanol, or both	Even 3 hrs of extra wakefulness may impair performance
	Arnedt, 2005 <sup>162</sup>	Residents	Pediatric residents = 34	Conditions of prolonged wakefulness or ethanol	On-call and BAC 0.05% had similar effects of simulated driving
Loughborough Simulator	Horne, 1996 <sup>163</sup>	Normals	Normals = 10	Caffeine vs nap in sleepy subjects	Caffeine or nap significantly reduced driving impairments

(Continued)



**TABLE 3***(Continued)*

Simulator Test	Study Author	Patient Type	No. of Subjects	Therapy	Outcome
	Reyner, 1997 <sup>164</sup>	Normals	Normals = 12	Caffeine + nap in sleepy subjects	Combined countermeasures better than either alone
	Horne, 2003 <sup>165</sup>	Normals	Normals = 12	Ethanol or placebo in rested or sleep-deprived conditions	Prolonged wakefulness and alcohol consumption produced greater decrements in performance than either alone
	Barrett, 2004 <sup>166</sup>	Normals	Normals = 20	Ethanol before 2-hr drive, sleep-deprived conditions	Alcohol continued to interact with sleepiness-related driving impairment after BAC reached zero
Hi-Fidelity Simulator(s)	Haraldsson, 1990 <sup>167</sup>	OSA; controls	OSA = 15; controls = 10	None	OSA performance worse than controls (braking time, lane position)
	Haraldsson, 1991 <sup>168</sup>	OSA; controls	OSA = 15; controls = 10	UPPP for OSA	12 of 15 patients improved in all measures of simulator performance after UPPP
	Haraldsson, 1995 <sup>169</sup>	OSA; controls	OSA = 13; controls = 5	UPPP for OSA	Improvements in performance sustained 4 yr after UPPP

The Task Force recommends that the commercial driver medical examiner (CDME) evaluate each driver individually and make a judgment about his or her fitness for duty based on specific criteria, including those listed in the other tables. These criteria cannot predict every situation faced by the examiner, and the final judgment belongs to the CDME. Additional testing is optional based on clinical judgment to document absence of excessive somnolence.

DADT indicates Divided Attention Driving Test; DASS, Divided Attention Steering Simulator; OSA, obstructive sleep apnea; CPAP, continuous positive airway pressure; UPPP, uvulopalatopharyngoplasty; EEG, electroencephalography.

means of flow through a face mask, which can be a nasal or oronasal (full face) mask. CPAP therapy exerts its beneficial effects by acting as a pneumatic splint to prevent the upper airway soft tissue from collapsing. It does not exert its effects by increasing upper airway muscle activity.<sup>172</sup> Compliance remains the most difficult part of CPAP treatment. Probably only approximately 50% to 60% of patients comply with CPAP.<sup>173–175</sup> CPAP use of greater than 6 hours has been associated with reducing the ESS score below the normal value of 10.<sup>176,177</sup>

**Optimal Continuous Positive Airway Pressure Setting.** The optimal CPAP settings for home use may be defined as the minimal pressure required to resolve all apneas, hypopneas, snoring, and arousals related to these events in all stages of sleep and

in all positions.<sup>78,94</sup> Simply stated, the optimal CPAP setting should resolve all SDB in supine rapid eye movement (REM) sleep to account for the effects of gravity and changes in muscle tone that may occur in different sleep stages and positions.<sup>178–180</sup> CPAP requirements vary with airway resistance and tend to be higher in more obese patients, in REM sleep, in the supine position, and after use of alcohol or muscle relaxants. CPAP historically has been “titrated” in a sleep laboratory. A CPAP titration study involves a mask fitting and subsequent CPAP adjustment to eliminate respiratory events. Recently, “autotitrating” CPAP machines have entered the market.<sup>181</sup> The AASM Practice Parameters Committee currently recommends a full night of CPAP titration to determine the optimal CPAP

pressure setting.<sup>78</sup> A repeat CPAP titration need only be performed if symptoms of OSA reappear despite compliance with CPAP therapy or if a patient sustains a significant weight loss either through diet or bariatric surgery.

A split-night sleep study in which the initial portion of the study is used to objectively document an individual's SDB followed by a CPAP titration during the second portion of the night may be indicated for patients with severe SDB (AHI >40 events per hour).<sup>78</sup> Although split-night studies are usually adequate to determine optimal positive airway pressure (PAP), full-night attended PSG is the preferred approach.<sup>171</sup>

Although current recommendations warrant that CPAP titrations occur during a full overnight in-laboratory PSG, some data suggest

that conventional fixed-pressure CPAP as well as auto-CPAP (APAP) therapy can be successfully initiated in an unattended home setting in patients with uncomplicated OSAS.<sup>182–186</sup> New evidence suggests that arbitrary<sup>180</sup> or algorithm-based CPAP pressures<sup>186</sup> can also effectively treat SDB. Given AASM's current recommendations and the limited amount of data supporting these unattended approaches, these alternative formats for titrating CPAP cannot be recommended for general use at this time.

### Additional Modes of Positive Airway Pressure Delivery

CPAP therapy is difficult for many patients to tolerate despite its clinical efficacy. Clinicians, patients, and industry are continually evaluating better methods to make CPAP therapy more acceptable. The next part of this review summarizes new advances in technology related to treating OSA, specifically focusing on advances in the delivery of PAP.

*Autocontinuous Positive Airway Pressure.* Automatic (also known as auto-, automated, autoadjusting, or autotitrating) continuous positive airway pressure further advances CPAP therapy because it has the ability to detect and respond to changes in upper airway resistance in real time.<sup>187</sup> Although this technology may be used to detect and diagnose patients with OSA, this section focuses on the literature related to APAP's ability to treat patients already diagnosed with OSA.

APAP noninvasively detects variations of upper airway obstruction and airflow limitation, including snoring, hypopneas, and apneas. Once upper airway flow limitation has been detected, the APAP devices automatically increase the pressure until the flow limitation has been resolved. Once a therapeutic pressure has been achieved, the APAP devices reduce pressure until flow limitation resumes. Most APAP devices have a therapeutic pressure range

between 3 and 20 cm H<sub>2</sub>O with the ability to adjust the upper and lower pressure limits based on the clinical conditions.

In a meta-analysis comparing the effectiveness of APAP versus standard CPAP with regard to several clinical parameters and outcomes,<sup>181</sup> it was concluded that compared with standard CPAP, APAP is almost always associated with a reduction in mean pressure. Aside from this difference, APAP and standard CPAP were similar in objective compliance, their ability to eliminate respiratory events, and their ability to improve subjective daytime sleepiness in uncomplicated moderate to severe OSA. Currently available machines have several limitations, including inability to recognize central apneas and hypoventilation. Therefore, their use is limited in populations that may be at risk for these conditions.

Currently, there is not enough data to support the use of unattended APAP titrations to determine a fixed CPAP setting despite the fact that several studies demonstrated that APAP and conventional CPAP therapy result in similar treatment outcomes. Although it is possible that APAP therapy may take the place of the standard in-laboratory CPAP titration in the not-so-distant future, these devices cannot be recommended as initial treatment for OSA in CMV drivers.

*Bilevel Positive Pressure Therapy.* Bilevel therapy's potential benefits in treating patients with OSA were first described in 1990.<sup>188</sup> As opposed to CPAP therapy, which allows a fixed pressure throughout the respiratory cycle, bilevel therapy allows for the independent adjustment of the expiratory positive airway pressure (EPAP) and the inspiratory positive airway pressure. In its initial description, bilevel therapy demonstrated that obstructive events could be eliminated at a lower EPAP compared with conventional CPAP pressures.<sup>188</sup> Although intuitively one would predict that bilevel therapy

would increase compliance by reducing unwanted pressure-related side effects, there is no convincing data to date that it improves compliance.<sup>189</sup> Bilevel therapy remains a viable option for CPAP-intolerant patients who have OSA, OSA with concurrent respiratory disease, and/or obesity hypoventilation syndrome.<sup>171</sup> The role of bilevel therapy in otherwise uncomplicated OSA remains unclear.

*C-Flex.* C-Flex is a relatively new technology that allows pressure relief during exhalation in an attempt to make CPAP therapy more comfortable. In simple terms, the C-Flex technology briefly reduces the CPAP pressure during exhalation before returning the pressure to its baseline CPAP setting before the initiation of inspiration. In a nonrandomized study that followed CPAP-naïve patients with OSA over a 3-month period, C-Flex compliance was greater by an average of 1.7 hours per night when compared with conventional CPAP users.<sup>190</sup> Subjective sleepiness and objective outcomes were otherwise similar between the groups. Although these results imply that C-Flex technology may improve CPAP compliance, further randomized, controlled trials (RCTs) will be necessary to confirm these findings. Based on the paucity of data currently available on C-Flex technology, no recommendations can be made regarding its use in clinical practice.

### Effects of Continuous Positive Airway Pressure

CPAP has been shown to reverse many of the sequelae of significant SDB, including mortality,<sup>191</sup> automobile accidents,<sup>31,153,192</sup> hypertension and other cardiovascular sequelae,<sup>193–199</sup> diabetic control,<sup>200,201</sup> increased healthcare costs,<sup>202</sup> and impaired quality of life.<sup>176,203,204</sup> The association among neurocognitive impairment, sleepiness, and depression and their potential improvement and reversibility with CPAP

treatment is more variable.<sup>189</sup> Most studies documenting improved outcomes with CPAP treatment have shown benefit with an intention-to-treat model. In other words, if the patient accepts CPAP, benefit occurs even in the absence of well-documented compliance. Complications of CPAP treatment are relatively minor and include epistaxis, ulcer, rashes or irritation on the bridge of the nose, rhinorrhea, chest or sinus discomfort, claustrophobia, nasal congestion, and conjunctivitis.

Healthcare costs are reported to be higher in patients with untreated OSA syndrome and appear to decrease after the introduction of CPAP therapy.<sup>202,205</sup> These savings are not readily apparent after 6 months of treatment<sup>206</sup> and may take up to 2 years to be realized. The incremental cost-effectiveness ratio of nasal CPAP has been reported as an approximately \$7000 quality-adjusted life-year.<sup>207</sup> This cost-effectiveness is comparable to that of many routine practices, including the use of antihypertensive medications.

**Daytime Sleepiness.** Although not all patients with OSA have symptoms of daytime sleepiness, a review of placebo-controlled studies shows that CPAP therapy usually improves or resolves symptoms of daytime sleepiness in patients with OSA who have this complaint.<sup>171</sup> The minimal and optimal amounts of nocturnal use necessary to improve symptoms of daytime sleepiness are not well defined, because even partial nocturnal use (as little as 4 hours per night) has been associated with significant improvements in daytime symptoms.<sup>174</sup> Although the minimal amount of time required on a nightly basis to improve symptoms of daytime sleepiness is not well established, there is evidence to suggest that use greater than 6 hours decreases ESS values to normal levels.<sup>176,177</sup> It has also been suggested that symptoms of daytime sleepiness reappear when CPAP therapy is discontinued for as little as one night.<sup>208</sup>

Although most patients with daytime sleepiness related to OSA will achieve significant improvements in symptoms after CPAP therapy has been instituted, this is not the case for all patients. There remains an OSA patient subgroup that continues to have symptoms of residual daytime sleepiness despite adequate compliance with CPAP therapy.<sup>209</sup> The actual prevalence of residual daytime sleepiness in CPAP-compliant patients remains undefined. The mechanisms responsible for this syndrome of residual daytime sleepiness also remain unclear but may be related to the oxidative injury effects of long-term intermittent hypoxemia on sleep-wake regions in the brain.<sup>210</sup> This raises an important point in that documenting objective compliance with CPAP therapy does not guarantee that a patient with OSA will be free from symptoms of daytime sleepiness. Furthermore, individuals who remain sufficiently sleepy despite documented adherence to CPAP are typically candidates for modafinil therapy.<sup>209</sup> However, as previously noted, FMCSA has precluded CMV drivers from using modafinil.<sup>27</sup>

**Neurocognitive/Psychologic Functioning.** Numerous studies have assessed the effects of SDB on neurocognitive/psychological functioning.<sup>211–213</sup> Altered memory, concentration, and reaction time may occur, but the levels of cognitive dysfunction vary over a wide range of performance areas in patients with different degrees of OSA.

Few studies on the effect of CPAP treatment for OSA on neurocognitive function are robust. Prospective studies evaluating patients with severe SDB for up to 12 months have demonstrated improvements in neurocognitive function.<sup>189</sup> These studies have demonstrated significant improvements in certain parameters of alertness and concentration, memory, and vigilance<sup>189</sup> as well as reaction time.<sup>214</sup>

Several small randomized, placebo-controlled trials have evaluated the effect of CPAP therapy on the

reversibility of neurocognitive deficits in patients with a broader spectrum of OSA severity.<sup>189</sup> Compared with the oral placebo arms of these studies, at least 1 month of CPAP therapy was required to see trends or significant improvements in various parameters of cognitive performance scores.<sup>215–218</sup> Studies looking at 3 weeks or less of therapy did not show evidence of neurocognitive improvements.<sup>219,220</sup> Although there were several statistically significant improvements with CPAP therapy, the effect size of these changes was relatively small. This finding may be secondary to a significant proportion of the study patients having relatively mild sleep apnea. Another possible explanation is that 1 month of CPAP therapy may be insufficient to reverse changes having accumulated over long periods of time (months to years). Also, the mean nocturnal CPAP use in these studies was only 3.3 hours per night, which is well below the typical average of 4.5 hours per night. Finally, it is possible that some of the cognitive deficits seen in patients with OSAS are irreversible, possibly related to repeated episodes of nocturnal hypoxemia.<sup>210,221</sup>

Further research is required to better define the mechanisms responsible for the neurocognitive deficits observed in some patients with OSA and to clarify the potential reversibility of these deficits. As is the case with daytime sleepiness, in those patients with OSA who have neurocognitive changes, objective compliance with CPAP therapy does not necessarily guarantee improvement or complete reversibility of these neurocognitive deficits.

**Driving Risk (Crashes) in the 'Real World.'** Very limited data exist on the risk of MVCs in patients who are started on nasal CPAP. In a self-report questionnaire, individuals reported fewer traffic accidents in the year after being started on CPAP as opposed to the year prior.<sup>222</sup> Subsequently, in a prospective cohort study of patients with known OSA

treated with PAP therapy, the risk of MVCs decreased to that of individuals without a sleep disorder in the 3 years after the initiation of treatment.<sup>3</sup> These data are supported by the improved performance on driving simulator tasks noted on several studies.<sup>148,153,156</sup>

**Timeline to Benefit.** There is no consensus on the time to improvement in performance parameters in patients who are treated with CPAP. This is due mostly to the paucity of RCTs looking at the specific question of time to improvement in performance as an outcome. For example, driving performance on a simulator task has been reported to improve within 3 days of starting CPAP therapy,<sup>156,223</sup> but a look at a randomized, placebo-controlled trial showed driving performance was improved at 1 month.<sup>153</sup> Similarly, a recent placebo-controlled trial showed that sleepiness as measured by ESS improved after 3 weeks of treatment.<sup>221</sup> Lastly, vigilance aspects of cognitive functioning seem to require 6 weeks to recover.<sup>88</sup> Placebo-controlled trials of CPAP treatment for lesser duration did not show a significant effect on cognitive function.<sup>219,224</sup> Overall, it appears that 1 month of modest amounts of adherence to CPAP therapy is associated with improvements in sleepiness, driving simulator tasks, and vigilance performance.

**Importance of Compliance and How to Measure.** Although CPAP is considered highly effective, there is evidence that approximately half the patients prescribed CPAP treatment fail to use it as prescribed, ie, throughout the night, every night.<sup>225</sup> Of patients initially offered CPAP, 5% to 50% refuse it with another 12% to 25% abandoning this therapy within 3 years.<sup>226</sup> Using a definition of nonadherence commonly used in the literature of use less than 4 hours and applied less than 70% of the nights, 21% to 46% of patients would be nonadherent by 3 months.<sup>226</sup> The longer the nightly duration of CPAP use, the greater the

likelihood of achieving normal functioning with the best outcomes obtained with use of 7 or more hours per night.<sup>176,177</sup> Consistency of use is equally as important as the nightly duration. Indeed, those patients who skip nights of treatment also have shorter duration of nightly use, averaging 5 hours of use.<sup>225,227</sup> Although some evidence suggests that failure to use CPAP for even 1 night of treatment returns the individual to pretreatment levels of sleep apnea severity,<sup>208,228,229</sup> other evidence suggests that CPAP has beneficial effect beyond its period of use.<sup>230,231</sup> The pattern of use is established early, within the first week of treatment.<sup>225,230</sup> Thus, it is critical to assess CPAP adherence early in the treatment regimen with periodic checks to assure maintenance of the therapy.

Patient reports of their application of CPAP treatment have been shown to be unreliable and generally approximately 1 hour less than actual use.<sup>200,227</sup> Meters that record the time the CPAP machine is on are available in most devices but are unable to differentiate between when the mask, the primary pressure delivery interface, is applied versus the time the machine is turned on but the mask is off. More sophisticated CPAP machines have embedded technology to differentiate machine-on from mask-on time. Thus, the practitioner is able to obtain valid and reliable data regarding the actual time prescribed pressure is applied. Using this technology, it has been determined that there is a 10% difference between the time the machine is turned on versus the length of time the mask was applied.

There have been considerable advances in the ease in obtaining data regarding CPAP use. Adherence data can be obtained and transmitted to the provider's office through a smart card, modem, or wireless upload to a web site. Most manufacturers have developed software to accompany these utilities that can construct provider-specific reports including

graphic displays, nightly device application over a set period of time, mean use, and number of days with a specific pattern of use, eg, how many days per week CPAP was used with an explicit duration of use. These advances in data generation and transmission should enable the provider to monitor CPAP use routinely and to intervene immediately when there is a detrimental change in the pattern.

There remains no clear understanding of why patients abandon treatment so early in the treatment regimen. Several factors have been explored with mixed results, including demographic characteristics, disease severity, technologic interface, and psychologic factors.<sup>227</sup> There is a weak relationship between CPAP use and disease severity.<sup>227</sup> However, there is stronger evidence associating an ESS of >10 and moderate to severe OSA with long-term use. Although recent parameters recommend the addition of heated humidification of inspired air,<sup>171</sup> two subsequent RCTs did not find evidence for improved adherence with routine use of heated humidification.<sup>232,233</sup> Utilization of flexible pressure devices remains controversial, but one study demonstrated improved application of PAP when such a device was used.<sup>190</sup> Recent evidence also suggests that psychologic factors, including claustrophobic tendencies,<sup>234</sup> active problem-solving,<sup>235</sup> and belief in self-efficacy,<sup>236</sup> affect confidence in the ability to engage in CPAP treatment and overcome obstacles to use.

The lack of clearly identified risk factors for nonadherence makes it difficult to develop interventions to promote adherence. Studies that have demonstrated statistically robust improvement in adherence have used intensive support, including several days of hospitalization, close follow up, and home visits,<sup>237</sup> altering confidence in the ability to apply the treatment by goal setting and immediate follow up to troubleshoot problems.<sup>238</sup> However, these studies have



not been replicated and may incur considerable cost if used in routine practice. Therefore, additional research is needed to provide a better understanding of how the initial experience with CPAP affects adherence as well as practical interventions to promote its use.

## Oral Appliances

Oral appliances are frequently considered as treatment options for patients with snoring and OSA. These devices are inserted into the mouth and are anchored by the teeth or the tongue in an effort to increase airway size and airway muscle tone. Two major types of appliances are currently in use: mandibular repositioning appliances (MRAs) and tongue repositioners. MRAs position the mandible to a forward and slightly opened position. Many of these devices have the option for adjustment of the degree of mandibular advancement. By contrast, tongue repositioning devices have an anterior plastic bulb. Negative suction pressure is used to hold the tongue inside the bulb in an anterior position. There are little scientific data available on this device.

A recent AASM review and practice parameter recommends that oral appliances be used for patients with simple snoring or mild to moderate OSA.<sup>239</sup> In trials comparing treatment with an oral appliance to CPAP therapy, CPAP therapy was more effective in reducing snoring and improving oxygenation,<sup>240–242</sup> and decreasing AHI.<sup>90,240–243</sup> Oral appliances and CPAP treatments were noted to be equally effective in reducing subjective sleepiness in most of these studies.<sup>90,241</sup> Although the majority of subjects favored the MRAs,<sup>240–242</sup> preference for CPAP was noted in one study in patients with a higher BMI and greater daytime impairment.<sup>90</sup> One study has shown a reduction in blood pressure with use of a mandibular advancement splint.<sup>244</sup> Nonetheless, the efficacy of oral appliances are well stud-

ied<sup>245</sup> and appear to be more effective than surgery for OSA.<sup>246</sup>

Unfortunately, no objective compliance monitors are presently available for oral appliances. Self-reports have been used and appear to be equivalent across therapies.<sup>4,5</sup> However, now that objective monitors for CPAP are widely available, the biases of self-reporting will be unequally distributed across these treatment arms.

Patients who use oral appliances often report excessive salivation and temporomandibular joint discomfort after awakening. The MRAs may also damage the teeth, cause occlusal changes, and affect the temporomandibular joint due to mechanical stress.<sup>247</sup> Advancement of the oral appliance to an optimal position to relieve SDB may take some time. A team approach among sleep specialists, dentists, and oral surgeons is often required. Anterior adjustment of the mandibular repositioning is done initially by symptoms and ultimately is guided by repeat PSG. In the future, overnight titrations of these devices may decrease the time to optimal benefit.<sup>248,249</sup>

## Surgical Treatment of Sleep Apnea

A variety of surgical approaches have been applied to SDB. The goal of these surgical treatments is to bypass the site of upper airway obstruction either by increasing airway caliber or by decreasing the collapsibility of the airway.<sup>250</sup>

Identifying the site of anatomic airway obstruction is an important step when considering possible surgical intervention for SDB.<sup>251,252</sup> Obstruction in the nasal passages due to conditions such as polyps or a deviated septum has been associated with the occurrence of apneas and hypopneas.<sup>253–256</sup> However, surgery to correct obstruction in the nasal passages has not been shown to reduce AHI.<sup>257,258</sup> Nonetheless, significant nasal obstruction may hinder attempts at nasal CPAP therapy. Therefore, aggressive treatment of

nasal obstruction may need to be considered.

Tonsillectomy alone should be reserved for a small subset of patients with isolated tonsillar hypertrophy.<sup>259</sup> In persons with retropalatal collapse or obstruction, UPPP with or without tonsillectomy is the most commonly performed and best studied of the surgical procedures used to treat sleep apnea. Surgical success is often defined by some ratio of preoperative to postoperative RDI *plus* a postoperative RDI or AHI of <10 to 20 events per hour of sleep. Using this type of definition, UPPP has a success rate of approximately 50% and appears to be even less effective in more obese patients who tend to have more severe apnea<sup>260</sup> with relapse occurring in as many as half.

Other treatments for retropalatal obstruction include laser-assisted uvulopalatopharyngoplasty (LAUP), radiofrequency volumetric tissue reduction (RFVTR), the Pillar Procedure, and coblation therapy. None of these has a better success rate than UPPP, and there are very few long-term results or data about complications available. In fact, LAUP has been associated with an acute exacerbation of SDB,<sup>261</sup> and RFVTR (also known as somnoplasty) is often associated with relapse.<sup>262</sup> The other two procedures have minimal long-term data to support their use.<sup>19,263</sup>

For severe OSA and for cases with craniofacial dysmorphism not amenable to CPAP, maxillofacial surgery may be a suitable alternative. This group of procedures includes maxillary, mandibular, or hyoid advancement in carefully preselected patients.<sup>264</sup> These procedures are designed to advance the ventral wall of the pharynx. Success rates can be very high, but there are no controlled trials and it is unclear which patients are suited to this approach.<sup>250</sup>

Tracheostomy is the only surgical procedure that is consistently effective even when performed alone, ie, without other surgical procedures,<sup>265</sup> and, as such, could be considered the “gold standard.” The goal with this

procedure is to create an artificial area distal to all areas of obstruction and bypass all areas of collapse. However, tracheostomy is rarely the first surgical option pursued due to its disfiguring nature.<sup>250</sup> Several studies indicate that it is useful for severe cases of OSA when nasal CPAP therapy cannot be used.<sup>260,266–269</sup>

Given that body weight, fat skin folds, and BMI are predictive of the degree of OSA,<sup>270</sup> it is not surprising that there is significant interest in bariatric surgery as a possible treatment for this condition. Overall, perioperative morbidity and mortality are substantial and higher in men.<sup>271</sup> In one small study of consecutive patients undergoing gastric bypass surgery, bariatric procedures were associated with a significant reduction in subjective sleepiness and AHI.<sup>272</sup> However, on average, the residual indices of SDB remain in the moderate range and may relapse over time.<sup>273</sup>

## Effects of Surgery

A 2002 study found that approximately one third of patients who received either UPPP or diet treatment for sleep apnea were cured, but that any treatment—including surgery, diet, or CPAP—improved mortality rates compared with the non-treated group.<sup>274</sup> Notably, the control group was composed largely of those who refused treatment, and such refusal may in itself be a marker of poor health outcomes. In patients who undergo tracheostomy, sleepiness resolves in 82% and hypertension resolves in 40%.<sup>260</sup> Critical general health outcome measures for all types of surgical treatments of SDB—such as quality of life, blood pressure, and healthcare costs—have been inadequately investigated.<sup>260,275</sup>

## Vigilance/Performance/Driving

In general, outcome data on automobile accidents, vigilance, and cognitive performance in patients who undergo surgical treatment for OSA

are lacking and must be extrapolated from studies done in nonoccupational settings.<sup>260,275</sup> Even among these studies, the lack of RCTs means that the benefit from most surgical approaches is only theoretical or experimental. Furthermore, reports in the surgical literature define a successful outcome as a proportion reflecting the reduction of SDB (eg, reducing AHI by 50%). Such a definition of outcome is clearly inadequate for many patients, because it still leaves them with clinically important sleep apnea. Surgery cannot be recommended for the vast majority of patients with OSA given its lack of long-term efficacy.

## Weight Loss

Obesity is an important risk factor for OSA.<sup>276</sup> Approximately 70% of patients with OSA are obese.<sup>277</sup> Conversely, the risk of OSA is increased more than 10-fold in obese individuals compared with the general population.<sup>277</sup> There is a significant correlation between a BMI of  $>30 \text{ m/kg}^2$  and AHI.<sup>278</sup> It has been well established that weight gain is positively correlated with the development of moderate to severe OSA independent of baseline BMI.<sup>279–283</sup> In the Wisconsin Sleep Cohort Study, a 10% weight gain was associated with a sixfold increase in the odds of being newly classified as having an AHI  $\geq 15$  events per hour.<sup>281</sup> This phenomenon has been shown to be strongest in men.<sup>278,280</sup>

Weight loss has been shown to reduce the amount of SDB.<sup>284–288</sup> Unfortunately, it appears that the effect of weight loss on decreasing AHI is not as dramatic as the effect of weight gain on increasing AHI.<sup>280</sup> Nonetheless, individuals who lose weight starting from extremes of BMI can have significant reductions in SDB.<sup>272,284,288</sup> However, the potential effect that SDB has on weight gain remains unanswered.<sup>280</sup>

## Practical Considerations

For recommendations to have practical applicability, “stakeholder”

acceptance is important, particularly among CMV drivers and their employers. For employers, testing and treatment expense, delays in evaluation and treatment, competing business priorities, driver shortages, and inconsistent CDME examination standards are concerns. For many drivers, the lack of health insurance is perhaps the most significant barrier to receiving testing and treatment for sleep disorders. However, some sleep centers and national home care companies are able to access indigent funding sources to enable drivers to be tested and treated. A driver without a medical certificate is “out of work” and often receives no disability insurance benefits.

Drivers’ acceptance of medical qualification is further complicated by their limited awareness and/or acceptance of sleep disorder symptoms. Driver education is available for fatigue prevention and management and is an important part of gaining “stakeholder acceptance.” FMCSA and NSF provide training materials for drivers and employers on sleep apnea and staying alert while driving.

Despite well-documented access limitations, public safety requires CMV drivers to have rapid access to sleep evaluation and treatment. Unfortunately, this is not consistently true at present. Sleep medicine professionals *must* develop ways to streamline the care of CMV drivers with sleep apnea for any approach to this problem to be successful. Furthermore, sleep specialists must understand the safety implications for CMV drivers whose other medical conditions put them and others on the road in danger. For many other medical conditions, the decision point is whether the driver is at higher risk than the general public for a motor vehicle crash. Follow up and continued evaluation of compliance by the sleep specialist is important because the driver is only evaluated by the CDME when his or her medical certificate expires, generally

every 1 to 2 years. This integrated care could be facilitated by forming relationships between CDMEs and sleep medicine specialists to promote bidirectional understanding of public health issues involved with driver medical certification as well as facilitate appropriate access to consultative evaluation, testing, and follow up of commercial drivers.

## Research Questions

The Task Force recognizes that there are many additional questions to be answered before the ideal method to properly identify those CMV drivers who are most likely to be involved in a MVC contributed to by their sleep apnea. Revisions to these recommendations may still be indicated as additional research is conducted on how to best ensure that treatment for OSA is and continues to be adequate to reduce the risk of a MVC. Key research that needs to be conducted includes:

1. Correlating screening tests with functional outcomes such as crashes, cardiovascular morbidity, and mortality;
2. Comparing screening algorithms, oximetry, and portable monitoring to each other with regard to predicting crashes;
3. Incorporating ethnic, gender, and age-related factors into screening and prediction models;
4. Gaining a better understanding of individual or other factors that predict which drivers with OSA will crash; and
5. Assessing factors related to CPAP use (nightly duration and time to benefit) that predict reduction in crashes.

In addition, given that the current published data are limited in scope and in level of experimental design, prospective data of better design are needed to link the recommendations for CDME assessments for sleep apnea risk to subsequent driver behavior and safety records.

In the course of producing this review, the Task Force also encoun-

tered the need to assess interactions among the CDME, the driver, a sleep assessment facility, and the trucking company. What communication skills and processes are needed in the interim between license renewals should be an area of focus. Best practice research might address how such communications can be done in the best interest of both the driver and public safety.

- Evaluate the cost of screening and subsequent management needs arising from driver assessments initiated by the CDME. Because sleep apnea is rather common, but medical recognition rather low, questions arose regarding medical costs to drivers and medical liability for sleep center referrals arising from a CDME administrative referral for testing and/or treatment. These policy issues are beyond the scope of this review.
- Encourage further research that addresses the issue of driver honesty regarding symptoms that may result in his or her being taken out of service by a CDME. In particular, what is the frequency of this problem? What attitudes and/or education regarding sleepiness and motor vehicle accidents influence this decision? Does an ongoing relationship with a CDME make a difference? Does driver (dis)honesty have an impact on outcomes such as crashes?
- An assessment of outcomes, including treatment modalities used, adherence, as well as days worked and number of crashes should be performed.

## Recommendation Statements Regarding Evaluation and Fitness for Duty for Commercial Drivers With Sleep Apnea

### Screening

The current examination form asks the CMV driver to respond to questions about sleep disorders, pauses in breathing while asleep, daytime sleepiness, and loud snoring.

CDMEs should, for clarification purposes, also inquire whether the driver has ever been told by *any* healthcare provider that he or she has sleep apnea, should be evaluated for sleep apnea, or has any other sleep disorders. Drivers should also be questioned about their history of MVCs, and the CDME should attempt to assess whether the crashes might have been related to sleepiness and/or a sleep disorder. One of the highest correlations for a future sleep fatigue-related crash is a history of a sleep/fatigue-related near miss or crash. The presence of these symptoms should prompt an evaluation for sleep apnea and/or other sleep disorders whether or not the CMV driver is overweight or obese.

In addition, as part of the history, considering the relationship between hypertension and OSA, CDMEs need to review with the driver whether he or she has a diagnosis of hypertension, and if so, if it has been difficult to manage and requires two or more medications to control. Objective assessments through the physical examination should also include a calculation of the driver's BMI (height and weight is already included on the examination form).

Examination of the upper airway should assess whether there is obstruction due to enlarged tonsils or uvula or whether the uvula is absent (evidence of an uvulopalatoplasty). Neck circumference should also be measured.

Inappropriate sleepiness during normal waking hours should be determined through a general question about wake-time alertness as well as by asking about excessive sleepiness during activities that are usually associated with vigilance such as talking to others. CDMEs should also note whether the driver falls asleep in either the waiting area or the examination room. Some self-report questionnaires might be useful such as ESS or FOSQ, depending on history and physical examination. If abnormal, both ESS and FOSQ have been shown to be related to an in-

TABLE 4

Screening Recommendation for Commercial Drivers With Possible or Probable Sleep Apnea

Medically Qualified to Drive Commercial Vehicles If Driver Meets Either of the Following	In-Service Evaluation (ISE) Recommended If Driver Falls Into Any One of the Following Five Major Categories (3 mo maximum certification)	Out-of-Service Immediate Evaluation Recommended If Driver Meets Any One of the Following Factors
1. No positive findings or any of the numbered in-service evaluation factors	1. Sleep history suggestive of OSA (snoring, excessive daytime sleepiness, witnessed apneas)	1. Observed unexplained excessive daytime sleepiness (sleeping in examination or waiting room) or confessed excessive sleepiness
2. Diagnosis of OSA with CPAP compliance documented	2. Two or more of the following: a) BMI $\geq 35$ kg/m <sup>2</sup> ; b) Neck circumference greater than 17 inches in men, 16 inches in women; c) Hypertension (new, uncontrolled, or unable to control with less than 2 medications). 3. ESS $>10$ 4. Previously diagnosed sleep disorder; compliance claimed, but no recent medical visits/compliance data available for immediate review (must be reviewed within 3-mo period); if found not to be compliant, should be removed from service (includes surgical treatment) 5. AHI $>5$ but $<30$ in a prior sleep study or polysomnogram and no excessive daytime somnolence (ESS $<11$ ), no motor vehicle accidents, no hypertension requiring 2 or more agents to control	2. Motor vehicle accident (run off road, at-fault, rear-end collision) likely related to sleep disturbance, unless evaluated for sleep disorder in the interim 3. ESS $\geq 16$ or FOSQ $<18$ 4. Previously diagnosed sleep disorder: d) Noncompliant (CPAP treatment not tolerated); e) No recent follow up (within recommended time frame); f) Any surgical approach with no objective follow up. 5. AHI $>30$

AHI indicates apnea-hypopnea index; BMI, body mass index; CPAP, continuous positive airway pressure; ESS, Epworth Sleepiness Scale; FOSQ, Functional Outcomes of Sleep Questionnaire; OSA, obstructive sleep apnea.

creased risk of crashes in CMV drivers, although this finding occurred outside the context of fitness-for-duty examination. FOSQ is the only measurement that has been shown to correlate with effectiveness of treatment. However, it may be less valuable in the CMV driver population because it relies significantly on subjective reporting.

The decision the CDME must make at this point is whether the driver is at sufficient enough risk of OSA to have his or her medical certification withheld or limited.

**Out-of-Service Pending Evaluation.** The driver should have his or her medical certification withheld pending an evaluation for a sleep disorder if any one of the following major criteria are met:

1. If the driver provides a history of excessive sleepiness, unexplained observed sleepiness on examination, a history of a sleep/fatigue

related MVC (if not treated since crash), or has an ESS of  $\geq 16$  or an FOSQ of  $\leq 18$ ; or

2. A previous diagnosis of OSA with no recent evaluation or a history of noncompliance with treatment as well as *any* surgical approach without objective follow up.

**In-Service With Evaluation.** The driver should have an evaluation for a sleep disorder if any one of the following major criteria are met:

1. A sleep history suggestive of OSA, snoring, excessive daytime somnolence, or witnessed apneas; or
2. Two or more of the following:  
a. BMI  $\geq 35$  kg/m<sup>2</sup>;  
b. Hypertension (new, uncontrolled, or unable to be controlled on less than 2 medications);

- c. A neck circumference of  $\geq 16$  inches in a woman or  $\geq 17$  inches in men;
- d. ESS score  $>10$ ;
- e. A history of a previously diagnosed sleep disorder with no recent evaluation; or
- f. AHI  $>5$  but  $<30$  on a prior sleep study or PSG and does not meet any of the previously mentioned criteria.

If the driver meets any of the in-service with evaluation (ISE) criteria, he or she could be certified for no longer than 3 months pending evaluation for OSA.

A BMI cutoff of 35 kg/m<sup>2</sup> was chosen instead of 30 kg/m<sup>2</sup> to maximize the specificity. It was believed that use of a lower cutoff such as a BMI of 30 kg/m<sup>2</sup>, which defines obesity, could result in the need for a



**TABLE 5.**

Recommendation Regarding the Evaluation for Fitness-for-Duty for Commercial Drivers With Possible or Probable Sleep Apnea

Category	Recommendation
Diagnosis	<ol style="list-style-type: none"> <li>1. Diagnosis should be determined by a physician and confirmed by polysomnography, preferably in an accredited sleep laboratory or by a certified sleep specialist</li> <li>2. A full-night study should be done unless a split-night study is indicated (severe OSA identified after at least 2 hours of sleep)</li> </ol>
Treatment	<ol style="list-style-type: none"> <li>1. First-line treatment for CMV drivers with OSA should be delivered by positive airway pressure (CPAP, Bilevel PAP)</li> <li>2. All CMV drivers on PAP <i>must</i> use a machine that is able to measure time on pressure</li> <li>3. A minimum acceptable average use of CPAP is 4 hours within a 24-hour period, but drivers should be advised that longer treatment would be more beneficial</li> <li>4. Treatment should be started as soon as possible but within 2 weeks of the sleep study</li> <li>5. Follow up by a sleep specialist should be done after 2-4 weeks of treatment</li> </ol>
Return to work after treatment	<ol style="list-style-type: none"> <li>1. After approximately 1 week of treatment, contact between the patient and personnel from the durable medical equipment supplier, treating provider, or sleep specialist</li> </ol>
Treatment with PAP	<ol style="list-style-type: none"> <li>2. AHI <math>\leq 5</math> documented with CPAP at initial titration (full night or split night) or after surgery or with use of oral appliance; AHI <math>\leq 10</math> depending on clinical findings</li> <li>3. Query driver about mask fit and compliance and remind to bring card (if used) or machine to next session</li> <li>4. At a minimum of 2 weeks after initiating therapy, but within 4 weeks, the driver should be reevaluated by the sleep specialist and compliance and blood pressure assessed</li> <li>5. If driver is compliant and blood pressure is improving (must meet FMCSA criteria), the driver can return to work but should be certified for no longer than 3 months</li> </ol>
Return to work after treatment	<ol style="list-style-type: none"> <li>1. Oral appliances should only be used as a primary therapy if AHI <math>&lt; 30</math></li> <li>2. Before returning to service, must have follow-up sleep study demonstrating AHI ideally <math>&lt; 5</math>, but <math>\leq 10</math> while wearing oral appliance</li> </ol>
Treatment with oral appliances	<ol style="list-style-type: none"> <li>3. All reported symptoms of sleepiness must be resolved and blood pressure must be controlled or improving (must meet FMCSA criteria)</li> </ol>
Return to work after treatment	Follow-up sleep study—AHI ideally $< 5$ but $\leq 10$ required to document efficacy
Treatment with surgery or weight loss	

AHI indicates apnea-hypopnea index; CPAP, continuous positive airway pressure; FMCSA, Federal Motor Carrier Safety Administration; PAP, positive airway pressure; OSA, obstructive sleep apnea; CMV, commercial motor vehicle.

majority of truck drivers to be evaluated for sleep apnea despite the fact that the majority do not have this condition.

Abnormal upper airway examination, FOSQ, MAP, or ESS may cause the CDME to only certify for up to 3 months pending evaluation or to remove the driver from service pending evaluation. If the sleep specialist finds that the driver has an AHI  $> 30$ , he or she should be removed from service, treated expeditiously, and returned to work as per the recommendation stated subsequently.

**Recertification.** For recertification, the driver should have no positive finding or numbered ISE criteria unless previously evaluated and addressed.

## Diagnosis

Diagnosis should be made by a physician and confirmed by PSG preferably in an accredited sleep laboratory or by a certified sleep specialist. A full-night study should be done unless a split-night study is indicated (severe OSA identified after at least 2 hours of sleep).

## Treatment

Drivers who must be treated for sleep apnea before returning to work as a CMV operator are those with an AHI  $\geq 30$  or an AHI  $\geq 5$  to 30 with: 1) difficult to manage hypertension (requiring two or more medications), 2) excessive sleepiness, or 3) a MVC due to fatigue or sleepiness. First-line treatment for CMV drivers with OSA should be PAP. All CMV driv-

ers on PAP *must* use a machine that is able to measure time on pressure. Data indicates that vigilance improves linearly with hours of CPAP use. A minimum acceptable average use of CPAP is 4 hours within a 24-hour period, but drivers should be advised that longer treatment would be more beneficial.<sup>176,177</sup> AHI during titration should be less than 5, but no greater than 10, for the driver to be considered able to return to duty. Treatment should be started as soon as possible to minimize time off work or risk while operating a CMV. Follow up by a sleep specialist should be done after 2 to 4 weeks of treatment.

Oral appliances should not be considered as first-line treatment as compliance cannot be monitored.

They could be used if AHI is  $<30$ , but before resuming commercial driving, the CMV operator must have a follow-up sleep study. AHI should be  $<5$ , although it could be up to 10 depending on clinical findings demonstrated while wearing the appliance. Any reported symptoms of sleepiness must be resolved. Upper airway surgery (UPPP, and so on) in the majority of cases is not as effective as sole treatment for OSA. If surgery is done, a follow-up sleep study is required to document efficacy. AHI should be  $<5$ , although it could be up to 10 depending on clinical findings. Weight loss or bariatric surgery may decrease the severity of OSA, but again, documentation of improvement with a sleep study is needed.

### Compliance and Efficacy

Because there is currently no objective method that reliably measures efficacy, the best determination of effective treatment is demonstration of compliance. Examiners should use clinical judgment based on improvement on individualized metrics to evaluate effective treatment. Repeat objective testing can be done but is not clinically indicated. Literature shows that FOSQ can be correlated with efficacy and decreased accident risk.

### Return to Work After Removal From Service to Obstructive Sleep Apnea

Close coordination among the sleep specialist, CDME, and other healthcare professionals is essential to ensure rapid evaluation and treatment in CMV drivers. The longer evaluation and treatment is delayed, the longer the driver will be out of work or at risk of accidents. If drivers know that they will be out of work for a prolonged period of time, the risk of denying symptoms or avoiding evaluation and treatment will increase. Once a driver is diagnosed with OSA and the appropriate pressure to be used determined (or

autotitrating machine to be used), the equipment should be provided as rapidly as possible. AHI ideally should be  $\leq 5$ , but could be up to 10 depending on clinical findings documented with CPAP at initial titration (full night or split night) or after surgery or with use of oral appliance. After approximately 1 week of treatment, there should be contact between the patient and personnel from either the CDME or sleep specialist. Drivers should be queried on fit compliance and reminded to bring the card (if used) or machine to their next appointment so that compliance can be assessed. At a minimum of 2 weeks, but within 4 weeks, the driver should be reevaluated by the sleep specialist and compliance and blood pressure assessed. At that point, the driver can return to work but should be certified for no longer than 3 months at which time compliance should again be evaluated. This is essential to ensure that the driver is compliant with treatment while on a normal work schedule.

### Follow Up

Drivers with OSA should be certified annually. Annual evaluation by a sleep specialist and assessment of compliance must be done for all OSA drivers, including for those who have had either upper airway surgery or weight reduction surgery. Retesting may not be required if sleep apnea is adequately controlled using both adherence data and subjective reports. Documentation must be provided to the CDME for review.

### Conclusion

It is recommended that the CDME evaluate each driver individually and make a judgment about fitness for duty based on these criteria. These criteria cannot predict every situation faced by the examiner, and the final judgment belongs to the CDME. Additional testing to document absence of excessive somnolence is optional based on the CDME's clinical judgment (see Tables 4 and 5 for screen-

ing and fitness-for-duty recommendations).

### Acknowledgment

The authors gratefully acknowledge the considerable commitment and contributions of Darrel Drobnich of the National Sleep Foundation to this project.

### References

1. Howard ME, Desai AV, Grunstein RR, et al. Sleepiness, sleep-disordered breathing, and accident risk factors in commercial vehicle drivers. *Am J Respir Crit Care Med*. 2004;170:1014–1021.
2. Shiomi T, Arita AT, Sasanabe R, et al. Falling asleep while driving and automobile accidents among patients with obstructive sleep apnea–hypopnea syndrome. *Psychiatry Clin Neurosci*. 2002; 56:333–334.
3. George CF. Reduction in motor vehicle collisions following treatment of sleep apnoea with nasal CPAP. *Thorax*. 2001; 56:508–512.
4. Horstmann S, Hess CW, Bassetti C, Gugger M, Mathis J. Sleepiness-related accidents in sleep apnea patients. *Sleep*. 2000;23:383–389.
5. Teran-Santos J, Jimenez-Gomez A, Cordero-Guevara J. The association between sleep apnea and the risk of traffic accidents. Cooperative Group Burgos-Santander. *N Engl J Med*. 1999;340: 847–851.
6. Young T, Blustein J, Finn L, Palta M. Sleep-disordered breathing and motor vehicle accidents in a population-based sample of employed adults. *Sleep*. 1997; 20:608–613.
7. Moreno CR, Carvalho FA, Lorenzi C, et al. High risk for obstructive sleep apnea in truck drivers estimated by the Berlin Questionnaire: prevalence and associated factors. *Chronobiol Int*. 2004;21: 871–879.
8. US Department of Transportation, Federal Highway Administration, Office of Motor Carriers. *Conference on Pulmonary/Respiratory Disorders and Commercial Drivers. Publication No. FHWA-MC-91-004*. Washington, DC: USDOT; 1991. Available at: [www.fmcsa.dot.gov/documents/pulmonary1.pdf](http://www.fmcsa.dot.gov/documents/pulmonary1.pdf).
9. George CF. Sleep. 5: driving and automobile crashes in patients with obstructive sleep apnoea/hypopnoea syndrome. *Thorax*. 2004;59:804–807.
10. Engleman HM, Hirst WS, Douglas NJ. Under reporting of sleepiness and driv-

- ing impairment in patients with sleep apnoea/hypopnoea syndrome. *J Sleep Res.* 1997;6:272–275.
11. Philip P. Sleepiness of occupational drivers. *Ind Health.* 2005;43:30–33.
  12. Reuveni H, Tarasiuk A, Wainstock T, Ziv A, Elhayany A, Tal A. Awareness level of obstructive sleep apnea syndrome during routine unstructured interviews of a standardized patient by primary care physicians. *Sleep.* 2004;27:1518–1525.
  13. McNicholas WT. Sleep apnoea and driving risk. European Respiratory Society Task Force on 'Public Health and Medicolegal Implications of Sleep Apnoea.' *Eur Respir J.* 1999;13:1225–1227.
  14. Blumenthal R, Braunstein J, Connolly H, Epstein A, Gersh BJ, Wittels EH. *Cardiovascular Advisory Panel Guidelines for the Medical Examination of Commercial Motor Vehicles Drivers.* Washington, DC: US Department of Transportation, Federal Motor Carrier Safety Administration, FMCSA-MCP-02–002. October 2002. Available at: [www.fmcsa.dot.gov/documents/cardio.pdf](http://www.fmcsa.dot.gov/documents/cardio.pdf).
  15. US Preventive Services Task Force. *Guide to Clinical Preventive Services*, 2nd ed. Baltimore: Williams & Wilkins; 1996.
  16. Sutton v. United Air Lines, Inc. (97–1943) 527 us 471 (1999).
  17. Pack AI, Dinges D, Maislin G. *A Study of Prevalence of Sleep Apnea Among Commercial Truck Drivers.* Federal Motor Carrier Safety Administration, Publication No. DOT-RT-02–030, Washington, DC; 2002. Tech Brief available at: [www.fmcsa.dot.gov/facts-research/research-technology/tech/Sleep-Apnea-TechBrief.pdf](http://www.fmcsa.dot.gov/facts-research/research-technology/tech/Sleep-Apnea-TechBrief.pdf).
  18. Bearpark H, Elliott L, Grunstein R, et al. Snoring and sleep apnea. A population study in Australian men. *Am J Respir Crit Care Med.* 1995;151:1459–1465.
  19. Bixler EO, Vgontzas AN, Ten Have T, Tyson, K, Kales A. Effects of age on sleep apnea in men: I. Prevalence and severity. *Am J Respir Crit Care Med.* 1998;157:144–148.
  20. Young T. Analytic epidemiology studies of sleep disordered breathing—what explains the gender difference in sleep disordered breathing? *Sleep.* 1993; 16(suppl):S1–2.
  21. Stoohs RA, Bingham LA, Itoi A, Guillemineault C, Dement WC. Sleep and sleep-disordered breathing in commercial long-haul truck drivers. *Chest.* 1995;107:1275–1282.
  22. *International Classification of Sleep Disorders, 2nd Ed. Diagnostic and Coding Manual.* Westchester, IL: American Academy of Sleep Medicine; 2005.
  23. Department of Health & Human Services, Centers for Medicare & Medicaid Services. *Medicare Coverage Issues Manual.* 2001. Available at: <http://new.cms.hhs.gov/transmittals/downloads/R150CIM.pdf>.
  24. US Department of Transportation, Federal Highway Administration. *Conference on Neurologic Disorders and Commercial Drivers.* Publication No. FHWA-MC-88–042. Washington, DC: US DOT, Federal Highway Administration, Office of Motor Carriers, 1988. Part I available at: [www.fmcsa.dot.gov/documents/neuro.pdf](http://www.fmcsa.dot.gov/documents/neuro.pdf). Part II available at: [www.fmcsa.dot.gov/documents/neuro2.pdf](http://www.fmcsa.dot.gov/documents/neuro2.pdf).
  25. Physical qualification of drivers: medical examination—certificate. *Federal Register.* 1998;63:4176941781.
  26. Physical qualification of drivers; medical examination; final rule. *Federal Register.* 2000;65:5936359380.
  27. Federal Motor Carrier Safety Administration. *Frequently Asked Questions.* Available at: [www.fmcsa.dot.gov/rules-regulations/topics/medical/faq.asp](http://www.fmcsa.dot.gov/rules-regulations/topics/medical/faq.asp).
  28. Pakola SJ, Dinges DF, Pack AI. Review of regulations and guidelines for commercial and noncommercial drivers with sleep apnea and narcolepsy. *Sleep.* 1995;18:787–796.
  29. Federal Aviation Administration. *Guide for Aviation Medical Examiners.* Available at: [www.faa.gov/about/office\\_org/headquarters\\_offices/avs/offices/aam/ame/guide/](http://www.faa.gov/about/office_org/headquarters_offices/avs/offices/aam/ame/guide/).
  30. National Transportation Safety Board. *Railroad Accident Report; Collision of Two Canadian National/Illinois Central Railway Trains Near Clarkston, Michigan. November 15, 2001.* NTSB RAR-02–04. PB2002–916304. Adopted November 19, 2002. Available at: [www.nts.gov/publictn/2002/RAR0204.pdf](http://www.nts.gov/publictn/2002/RAR0204.pdf).
  31. National Transportation Safety Board. *Highway Accident Report; Work Zone Collision Between a Tractor-Semi-trailer and a Tennessee Highway Patrol Vehicle. Jackson, Tennessee, July 26, 2000.* NTSB/Har-02/01. PB2002–916201. Adopted May 14, 2002. Available at: [www.nts.gov/Publictn/2002/HAR0201.pdf](http://www.nts.gov/Publictn/2002/HAR0201.pdf).
  32. National Transportation Safety Board. *Highway Accident Report. 15-Passenger Child Care Van Run-Off-Road Accident, Memphis, Tennessee. April 4, 2002.* NTSB/HAR-04/02. PB2004–916202. Adopted April 7, 2004. Available at: [www.nts.gov/publictn/2004/HAR0402.pdf](http://www.nts.gov/publictn/2004/HAR0402.pdf).
  33. National Transportation Safety Board. *Brief of Accident. NTSB Identification CH100LA076.* Washington, DC: NTSB; 2001. Summary available at: [www.nts.gov/ntsb/brief.asp?ev\\_id=20001212X20463&key=1](http://www.nts.gov/ntsb/brief.asp?ev_id=20001212X20463&key=1).
  34. National Transportation Safety Board. *Grounding of the Liberian Passenger Ship Star Princess on Poundstone Rock, Lynn Canal, Alaska, June 23, 1995. (NTSB/MAR-97/02).* Available at: [www.nts.gov/publictn/1997/MAR9702.pdf](http://www.nts.gov/publictn/1997/MAR9702.pdf).
  35. National Transportation Safety Board. *Maryland Transit Administration Light Rail Vehicle Accidents at the Baltimore–Washington International Airport Transit Station Near Baltimore, Maryland, February 13 and August 15, 2000.* NTSB/SIR-01/02. Available at: <http://ntl.bts.gov/lib/11000/11700/11786/SIR0102.pdf>.
  36. National Transportation Safety Board. *Railroad Accident Brief. Side Collision of Burlington Northern Santa Fe Railway Train and Union Pacific Railroad Train Near Kelso, Washington. November 15, 2003.* NTSB/RAB-05/03. Adopted June 6, 2005. Available at: [www.nts.gov/publictn/2005/RAB0503.pdf](http://www.nts.gov/publictn/2005/RAB0503.pdf).
  37. Canadian Medical Association. *Determining Medical Fitness to Drive: A Guide for Physicians*, 6th ed. Available at: [www.cma.ca/index.cfm/ci\\_id/18223/la\\_id/1.htm](http://www.cma.ca/index.cfm/ci_id/18223/la_id/1.htm).
  38. Canadian Council on Motor Transport Administrators. *CCMTA Medical Standards for Drivers.* July 2004. Available at: [www.ccmta.ca/english/pdf/medical\\_standards\\_july04.PDF](http://www.ccmta.ca/english/pdf/medical_standards_july04.PDF).
  39. Austroads. *Assessing Fitness to Drive*, 3rd ed, 2003. Austroads Publications No. AP-G56/03. Available at: [www.austroads.com.au/upload\\_files/docs/AFTD%202003-F\\_A-WEBREV1.pdf](http://www.austroads.com.au/upload_files/docs/AFTD%202003-F_A-WEBREV1.pdf).
  40. *Current Regulations, Guidelines and Transportation Reports. National Standard for Health Assessment of Rail Safety Workers. Volume 2: Assessment Procedures and Medical Criteria.* June 2004.
  41. Drivers Medical Group for Medical Practitioners. *At a Glance Guide to the Current Medical Standards of Fitness to Drive. DVLA.* February 2005. Available at: [www.dvla.gov.uk/medical/about\\_dri\\_med.aspx](http://www.dvla.gov.uk/medical/about_dri_med.aspx).
  42. Strohl KP, Redline S. Recognition of obstructive sleep apnea. *Am J Respir Crit Care Med.* 1996;154:279–289.
  43. Phillips B, Cook Y, Schmitt F, Berry D.



- Sleep apnea: prevalence of risk factors in a general population. *South Med J*. 1989;82:1090–1092.
44. Young T, Skatrud J, Peppard PE. Risk factors for obstructive sleep apnea in adults. *JAMA*. 2004;291:2013–2016.
  45. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. *Am J Respir Crit Care Med*. 2002;165:1217–1239.
  46. Dagan Y, Doljansky JT, Green A, Weiner A. Body mass index (BMI) as a first-line screening criterion for detection of excessive daytime sleepiness among professional drivers. *Traffic Inj Prev*. 2006;7:44–48.
  47. Katz I, Stradling J, Slutsky AS, Zamel N, Hoffstein V. Do patients with obstructive sleep apnea have thick necks? *Am Rev Respir Dis*. 1990;141:1228–1231.
  48. Hoffstein V, Mateika S. Differences in abdominal and neck circumferences in patients with and without obstructive sleep apnoea. *Eur Respir J*. 1992;5:377–381.
  49. Schellenberg JB, Maislin G, Schwab RJ. Physical findings and the risk for obstructive sleep apnea. The importance of oropharyngeal structures. *Am J Respir Crit Care Med*. 2000;162:740–748.
  50. Young T, Palta M, Badr MS. Sleep-disordered breathing [Letter]. *N Engl J Med*. 1993;329:1429–1430.
  51. Davies RJ, Stradling JR. The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apnoea syndrome. *Eur Respir J*. 1990;3:509–514.
  52. Redline S, Tishler PV, Tosteson TD, et al. The familial aggregation of obstructive sleep apnea. *Am J Respir Crit Care Med*. 1995;151:682–687.
  53. Pillar G, Lavie P. Assessment of the role of inheritance in sleep apnea syndrome. *Am J Respir Crit Care Med*. 1995;151:688–691.
  54. Guilleminault C, Stoohs R, Kim YD, et al. Upper airway sleep-disordered breathing in women. *Ann Intern Med*. 1995;122:493–501.
  55. Bixler EO, Vgontzas AN, Lin HM, et al. Prevalence of sleep-disordered breathing in women: effects of gender. *Am J Respir Crit Care Med*. 2001;163:608–613.
  56. Dancy DR, Hanly PJ, Soong C, Lee B, Hoffstein V. Impact of menopause on the prevalence and severity of sleep apnea. *Chest*. 2001;120:151–155.
  57. Kales A, Bixler EO, Cadieux RJ, et al. Sleep apnoea in a hypertensive population. *Lancet*. 1984;2:1005–1008.
  58. Fletcher EC, DeBehnke RD, Lovoi MS, Gorin AB. Undiagnosed sleep apnea in patients with essential hypertension. *Ann Intern Med*. 1985;103:190–195.
  59. Silverberg DS, Oksenberg A. Essential hypertension and abnormal upper airway resistance during sleep. *Sleep*. 1997;20:794–806.
  60. Nieto FJ, Young TB, Lind BK, et al. Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep Heart Health Study. *JAMA*. 2000;283:1829–1836.
  61. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med*. 2000;342:1378–1384.
  62. Lavie P, Herer P, Hoffstein V. Obstructive sleep apnoea syndrome as a risk factor for hypertension: population study. *BMJ*. 2000;320:479–482.
  63. Chobanian AV, Bakris GL, Black HR, et al. National Heart, Lung, and Blood Institute Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; National High Blood Pressure Education Program Coordinating Committee. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA*. 2003;289:2560–2572.
  64. Logan AG, Perlikowski SM, Mente A, et al. High prevalence of unrecognized sleep apnoea in drug-resistant hypertension. *J Hypertens*. 2001;19:2271–2277.
  65. Kryger M, Roth T, Dement WC. *Principles and Practice of Sleep Medicine*, 4th ed. Philadelphia: Elsevier Saunders; 2005.
  66. Crocker BD, Olson LG, Saunders NA, et al. Estimation of the probability of disturbed breathing during sleep before a sleep study. *Am Rev Respir Dis*. 1990;142:14–18.
  67. Williams AJ, Yu G, Santiago S, Stein M. Screening for sleep apnea using pulse oximetry and a clinical score. *Chest*. 1991;100:631–635.
  68. Viner S, Szalai JP, Hoffstein V. Are history and physical examination a good screening test for sleep apnea? *Ann Intern Med*. 1991;115:356–359.
  69. Flemons WW, Whitelaw WA, Brant R, Remmers JE. Likelihood ratios for a sleep apnea clinical prediction rule. *Am J Respir Crit Care Med*. 1994;150:1279–1285.
  70. Kump K, Whalen C, Tishler PV, et al. Assessment of the validity and utility of a sleep-symptom questionnaire. *Am J Respir Crit Care Med*. 1994;150:735–741.
  71. Gyulay S, Olson LG, Hensley MJ, King MT, Allen KM, Saunders NA. A comparison of clinical assessment and home oximetry in the diagnosis of obstructive sleep apnea. *Am Rev Respir Dis*. 1993;147:50–53.
  72. Maislin G, Pack AI, Kribbs NB, et al. A survey screen for prediction of apnea. *Sleep*. 1995;18:158–166.
  73. Pradhan PS, Gliklich RE, Winkelman J. Screening for obstructive sleep apnea in patients presenting for snoring surgery. *Laryngoscope*. 1996;106:1393–1397.
  74. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Ann Intern Med*. 1999;131:485–491.
  75. Rodsutti J, Hensley M, Thakkinian A, D'Este C, Attia J. A clinical decision rule to prioritize polysomnography in patients with suspected sleep apnea. *Sleep*. 2004;27:694–699.
  76. Hussain SF, Fleetham JA. Overnight home oximetry: can it identify patients with obstructive sleep apnea–hypopnea who have minimal daytime sleepiness? *Respir Med*. 2003;97:537–540.
  77. Rowley JA, Aboussouan LS, Badr MS. The use of clinical prediction formulas in the evaluation of obstructive sleep apnea. *Sleep*. 2000;23:929–938.
  78. Tsai WH, Remmers JE, Brant R, Flemons WW, Davies J, Macarthur C. A decision rule for diagnostic testing in obstructive sleep apnea. *Am J Respir Crit Care Med*. 2003;167:1427–1432.
  79. Kushida CA, Efron B, Guilleminault C. A predictive morphometric model for the obstructive sleep apnea syndrome. *Ann Intern Med*. 1997;127:581–587.
  80. Gurubhagavatula I, Maislin G, Nkwuo JE, Pack AI. Occupational screening for obstructive sleep apnea in commercial drivers. *Am J Respir Crit Care Med*. 2004;170:371–376.
  81. Gurubhagavatula I, Maislin G, Pack AI. An algorithm to stratify sleep apnea risk in a sleep disorders clinic population. *Am J Respir Crit Care Med*. 2001;164:1904–1909.
  82. Gami AS, Pressman G, Caples SM, et al. Association of atrial fibrillation and obstructive sleep apnea. *Circulation*. 2004;110:364–367.
  83. Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep*. 1991;14:540–545.
  84. Redline S, Adams N, Strauss ME, Roebuck T, Winters M, Rosenberg C. Improvement of mild sleep-disordered



- breathing with CPAP compared with conservative therapy. *Am J Respir Crit Care Med.* 1998;157:858–865.
85. Weaver TE, Laizner AM, Evans LK, et al. An instrument to measure functional status outcomes for disorders of excessive sleepiness. *Sleep.* 1997;20:835–843.
  86. Weaver TE, Chugh DK, Maislin G, et al. Impact of obstructive sleep apnea on the conduct of daily activities. *Sleep Res.* 1997;26:530.
  87. Faccenda JF, Mackay TW, Boon NA, Douglas NJ. Randomized placebo-controlled trial of continuous positive airway pressure on blood pressure in the sleep apnea–hypopnea syndrome. *Am J Respir Crit Care Med.* 2001;163:344–348.
  88. Montserrat JM, Ferrer M, Hernandez L, et al. Effectiveness of CPAP treatment in daytime function in sleep apnea syndrome: a randomized controlled study with an optimized placebo. *Am J Respir Crit Care Med.* 2001;164:608–613.
  89. McFadyen TA, Espie CA, McArdle N, Douglas NJ, Engleman HM. Controlled, prospective trial of psychosocial function before and after continuous positive airway pressure therapy. *Eur Respir J.* 2001;18:996–1002.
  90. Engleman HM, McDonald JP, Graham D, et al. Randomized crossover trial of two treatments for sleep apnea/hypopnea syndrome: continuous positive airway pressure and mandibular repositioning splint. *Am J Respir Crit Care Med.* 2002;166:855–859.
  91. Blanco J, Zamarron C, Abeleira Pazos MT, Lamela C, Suarez Quintanilla D. Prospective evaluation of an oral appliance in the treatment of obstructive sleep apnea syndrome. *Sleep Breath.* 2005;9:20–25.
  92. Woodson BT, Steward DL, Weaver EM, Javaheri S. A randomized trial of temperature-controlled radiofrequency, continuous positive airway pressure, and placebo for obstructive sleep apnea syndrome. *Otolaryngol Head Neck Surg.* 2003;128:848–861.
  93. Kushida C, Littner M, Morgenthaler T, et al. Practice parameters for the indication for polysomnography and related procedures: an update for 2005. *Sleep.* 2005;28:499–521.
  94. Loube D, Gay P, Strohl K, Pack A, White D, Collop N. Indications for positive airway pressure treatment of adult obstructive sleep apnea patients: a consensus statement. *Chest.* 1999;115:863–866.
  95. Rechtschaffen A, Kales A. *A Manual of Standardized Terminology, Techniques and Scoring Systems for Sleep Stages of Human Subjects.* Los Angeles: UCLA Brain Information Service/Research Institute; 1968.
  96. Meoli A, Casey K, Clark R, et al. Clinical Practice Review Committee. Hypopnea in sleep-disordered breathing in adults. *Sleep.* 2001;24:469–470.
  97. Collop N. Scoring variability between polysomnography technologists in different sleep laboratories. *Sleep Med.* 2002;3:43–47.
  98. Redline S, Kapur V, Sanders M, et al. Effects of varying approaches for identifying respiratory disturbances on sleep apnea assessment. *Am J Respir Crit Care Med.* 2000;161:369–374.
  99. Barbe F, Pericas J, Munoz A, et al. Automobile accidents in patients with sleep apnea syndrome. An epidemiological and mechanistic study. *Am J Respir Crit Care Med.* 1998;158:18–22.
  100. Flemons W, Remmers J, Whitelaw W. The correlation of a computer simulated driving program with polysomnographic indices and neuropsychological tests in consecutively referred patients for assessment of sleep apnea. *Sleep.* 1993;16(suppl 8):S71.
  101. George C, Boudreau A, Smiley A. Simulated driving performance in patients with obstructive sleep apnea. *Am J Respir Crit Care Med.* 1996;154:175–181.
  102. Yaggi H, Concato J, Kernan W, Lichtman J, Brass L, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. *N Engl J Med.* 2005;353:2034–2041.
  103. Ferber R, Millman R, Coppola M, et al. Portable recording in the assessment of obstructive sleep apnea. ASDA Standards of Practice. *Sleep.* 1994;17:378–392. Available at: [www.aasmnet.org/PDF/PortableReview.pdf](http://www.aasmnet.org/PDF/PortableReview.pdf).
  104. Ross S, Sheinhardt I, Harrison K, et al. Systematic review and meta-analysis of the literature regarding the diagnosis of sleep apnea. *Sleep.* 2000;23:519–532.
  105. Chesson AL Jr, Berry R, Pack A; American Academy of Sleep Medicine; American Thoracic Society; American College of Chest Physicians. Practice parameters for the use of portable monitoring devices in the investigation of suspected obstructive sleep apnea in adults. *Sleep.* 2003;26:907–913.
  106. Flemons W, Littner M, Rowley J, et al. Home diagnosis of sleep apnea: a systematic review of the literature. An evidence review cosponsored by the American Academy of Sleep Medicine, the American College of Chest Physicians, and the American Thoracic Society. *Chest.* 2003;124:1543–1579.
  107. Zafar S, Ayappa I, Norman RG, Krieger AC, Walsleben JA, Rapoport DM. Choice of oximeter affects apnea–hypopnea index. *Chest.* 2005;127:80–88.
  108. Davila DG, Richards KC, Marshall BL, et al. Oximeter's acquisition parameter influences the profile of respiratory disturbances. *Sleep.* 2003;26:91–95.
  109. Levine B, Roehrs T, Zorick F, Roth T. Daytime sleepiness in young adults. *Sleep.* 1988;11:39–46.
  110. Palm L, Persson E, Elmqvist D, Blennow G. Sleep and wakefulness in normal preadolescent children. *Sleep.* 1989;12:299–308.
  111. Valencia-Flores M, Campos RM, Mendez J, et al. Multiple sleep latency test (MSLT) and sleep apnea in aged women. *Sleep.* 1993;16:114–117.
  112. Richardson GS, Carskadon MA, Orav EJ, Dement WC. Circadian variation of sleep tendency in elderly and young adult subjects. *Sleep.* 1982;5(suppl 2):S82–94.
  113. Carskadon MA, Dement WC. Nocturnal determinants of daytime sleepiness. *Sleep.* 1982;5(suppl 2):S73–81.
  114. Rosenthal L, Roehrs TA, Rosen A, Roth T. Level of sleepiness and total sleep time following various time in bed conditions. *Sleep.* 1993;16:226–232.
  115. Carskadon MA, Brown ED, Dement WC. Sleep fragmentation in the elderly: relationship to daytime sleep tendency. *Neurobiol Aging.* 1982;3:321–327.
  116. Levine B, Roehrs T, Stepanski E, Zorick F, Roth T. Fragmenting sleep diminishes its recuperative value. *Sleep.* 1987;10:590–599.
  117. Philip P, Stoohs R, Guilleminault C. Sleep fragmentation in normals: a model for sleepiness associated with upper airway resistance syndrome. *Sleep.* 1994;17:242–247.
  118. Roehrs T, Merlotti L, Petrucelli N, Stepanski E, Roth T. Experimental sleep fragmentation. *Sleep.* 1994;17:438–443.
  119. Roehrs T, Lumley M, Asker D, Zorick F, Roth T. Ethanol and caffeine effects on daytime sleepiness. *Sleep Res.* 1986;15:41.
  120. Roehrs T, Kribbs N, Zorick F, Roth T. Hypnotic residual effects of benzodiazepines with repeated administration. *Sleep.* 1986;9:309–316.
  121. Roth T, Roehrs T, Koshorek G, Sickelsteel J, Zorick F. Central effects of antihistamine. *Sleep Res.* 1986;15:43.
  122. Carskadon MA, Seidel WF, Greenblatt DJ, Dement WC. Daytime carryover of triazolam and flurazepam in elderly insomniacs. *Sleep.* 1982;5:361–371.

123. Bishop C, Roehrs T, Rosenthal L, Roth T. Alerting effects of methylphenidate under basal and sleep-deprived conditions. *Exp Clin Psychopharmacol*. 1997; 5:344–352.
124. Mitler MM, Hajdukovic R. Relative efficacy of drugs for the treatment of sleepiness in narcolepsy. *Sleep*. 1991; 14:218–220.
125. Bonnet MH, Arand DL. 24-hour metabolic rate in insomniacs and matched normal sleepers. *Sleep*. 1995;18:581–588.
126. Bonnet MH, Arand DL. Caffeine use as a model of acute and chronic insomnia. *Sleep*. 1992;15:526–536.
127. Bonnet MH, Arand DL. The use of lorazepam TID for chronic insomnia. *Int Clin Psychopharmacol*. 1999;14:81–89.
129. Kayumov L, Rotenberg V, Buttoo K, Auch C, Pandi-Perumal SR, Shapiro CM. Interrelationships between nocturnal sleep, daytime alertness, and sleepiness: two types of alertness proposed. *J Neuropsychiatry Clin Neurosci*. 2000; 12:86–90.
129. Benca RM. Mood disorders. In: Kryger MH, Roth T, Dement W, eds. *Principles and Practice of Sleep Medicine*, 4th ed. Philadelphia: Elsevier Saunders; 2005: 1311–1326.
130. Littner MR, Kushida C, Wise M, et al. Practice parameters for clinical use of the multiple sleep latency test and the maintenance of wakefulness test. *Sleep*. 2005;28:113–121.
131. Arand D, Bonnet M, Hurwitz T, Mitler M, Rosa R, Sangal RB. The clinical use of the MSLT and MWT. *Sleep*. 2005; 28:123–144.
132. George CF, Boudreau AC, Smiley A. Effects of nasal CPAP on simulated driving performance in patients with obstructive sleep apnoea. *Thorax*. 1997; 52:648–653.
133. Pizza F, Contardi S, Mostacci B, Mondini S, Cirignotta F. A driving simulation task: correlations with Multiple Sleep Latency Test. *Brain Res Bull*. 2004;63:423–426.
134. Hakkanen H, Summala H, Partinen M, Tiitonen M, Silvo J. Blink duration as an indicator of driver sleepiness in professional bus drivers. *Sleep*. 1999;22: 798–802.
135. Kingshott RN, Cowan JO, Jones DR, et al. The role of sleep-disordered breathing, daytime sleepiness, and impaired performance in motor vehicle crashes—a case control study. *Sleep Breath*. 2004;8:61–72.
136. Harma M, Suvanto S, Popkin S, Pulli K, Mulder M, Hirvonen K. A dose–response study of total sleep time and the ability to maintain wakefulness. *J Sleep Res*. 1998;7:167–174.
137. Bennett LS, Stradling JR, Davies RJ. A behavioural test to assess daytime sleepiness in obstructive sleep apnoea. *J Sleep Res*. 1997;6:142–145.
138. Krieger AC, Ayappa I, Norman RG, Rapoport DM, Walsleben J. Comparison of the maintenance of wakefulness test (MWT) to a modified behavioral test (OSLER) in the evaluation of daytime sleepiness. *J Sleep Res*. 2004;13: 407–411.
139. Priest B, Brichard C, Aubert G, Liistro G, Rodenstein DO. Microsleep during a simplified maintenance of wakefulness test. A validation study of the OSLER Test. *Am J Respir Crit Care Med*. 2001; 163:1619–1625.
140. Pepperell JC, Maskell NA, Jones DR, et al. A randomized controlled trial of adaptive ventilation for Cheyne-Stokes breathing in heart failure. *Am J Respir Crit Care Med*. 2003;168:1109–1114.
141. Federal Motor Carrier Safety Administration. *Sleep Apnea Crash Risk Study*. September 2004. Publication No. FMCSA-RT-04–007. Available at: [www.fmcsa.dot.gov/facts-research/briefs/SleepApneaCrash-RiskStudy-TechBrief.pdf](http://www.fmcsa.dot.gov/facts-research/briefs/SleepApneaCrash-RiskStudy-TechBrief.pdf).
142. George CF, Smiley A. Sleep apnea & automobile crashes. *Sleep*. 1999;22: 790–795.
143. Lindberg E, Carter N, Gislason T, Jansson C. Role of snoring and daytime sleepiness in occupational accidents. *Am J Respir Crit Care Med*. 2001;164: 2031–2035.
144. Dinges DF, Powell JW. Microcomputer analyses of performance on a portable, simple visual RT task during sustained operations. *Beh Res Meth Instr Comp*. 1985;17:652–655.
145. Dinges DF, Pack F, Williams K, et al. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. *Sleep*. 1997;20:267–277.
146. Konowal NM, Van Dongen HP, Powell JW, Mallis MM, Dinges DF. Determinants of microsleeps during experimental sleep deprivation. *Sleep*. 1999; 22(suppl 1):S328.
147. Dorrian J, Rogers NL, Dinges DF. Psychomotor vigilance performance: a neurocognitive assay sensitive to sleep loss. In: Kushida C, ed. *Sleep Deprivation: Clinical Issues, Pharmacology and Sleep Loss Effects*. New York: Marcel Dekker, Inc; 2005:39–70.
148. Findley LJ, Fabrizio MJ, Knight H, Norcross BB, LaForte AJ, Suratt PM. Driving simulator performance in patients with sleep apnea. *Am Rev Respir Dis*. 1989;140:529–530.
149. Findley LJ, Suratt PM, Dinges DF. Time-on-task decrements in ‘steer clear’ performance of patients with sleep apnea and narcolepsy. *Sleep*. 1999;22:804–809.
150. Munoz A, Mayoralas LR, Barbe F, Pericas J, Agusti AG. Long-term effects of CPAP on daytime functioning in patients with sleep apnoea syndrome. *Eur Respir J*. 2000;15:676–681.
151. Kingshott RN, Vennelle M, Hoy CJ, Engleman HM, Deary IJ, Douglas NJ. Predictors of improvements in daytime function outcomes with CPAP therapy. *Am J Respir Crit Care Med*. 2000;161: 866–871.
152. George CF, Boudreau AC, Smiley A. Comparison of simulated driving performance in narcolepsy and sleep apnea patients. *Sleep*. 1996;19:711–717.
153. Hack M, Davies RJ, Mullins R, et al. Randomised prospective parallel trial of therapeutic versus subtherapeutic nasal continuous positive airway pressure on simulated steering performance in patients with obstructive sleep apnoea. *Thorax*. 2000;55:224–231.
154. Juniper M, Hack MA, George CF, Davies RJ, Stradling JR. Steering simulation performance in patients with obstructive sleep apnoea and matched control subjects. *Eur Respir J*. 2000;15: 590–595.
155. Turkington PM, Sircar M, Allgar V, Elliott MW. Relationship between obstructive sleep apnoea, driving simulator performance, and risk of road traffic accidents. *Thorax*. 2001;56:800–805.
156. Turkington PM, Sircar M, Saralaya D, Elliott MW. Time course of changes in driving simulator performance with and without treatment in patients with sleep apnoea hypopnoea syndrome. *Thorax*. 2004;59:56–59.
157. Philip P, Sagaspe P, Taillard J, et al. Fatigue, sleepiness, and performance in simulated versus real driving conditions. *Sleep*. 2005;28:1511–1516.
158. Dobbs BM. *Medical Conditions and Driving; A Review of the Literature (1960–2000)*. NHTSA Report No. DOT HS 809–690. September 2005. Available at: [www.nhtsa.dot.gov/people/injury/research/MedicalConditions\\_Driving.pdf](http://www.nhtsa.dot.gov/people/injury/research/MedicalConditions_Driving.pdf).
159. Risser MR, Ware JC, Freeman FG. Driving simulation with EEG monitoring in normal and obstructive sleep apnea patients. *Sleep*. 2000;23:393–398.
160. Amedt JT, Wilde GJ, Munt PW, MacLean AW. Simulated driving performance

- following prolonged wakefulness and alcohol consumption: separate and combined contributions to impairment. *J Sleep Res.* 2000;9:233–241.
161. Arnedt JT, Wilde GJ, Munt PW, MacLean AW. How do prolonged wakefulness and alcohol compare in the decrements they produce on a simulated driving task? *Accid Anal Prev.* 2001;33:337–344.
  162. Arnedt JT, Owens J, Crouch M, Stahl J, Carskadon MA. Neurobehavioral performance of residents after heavy night call vs after alcohol ingestion. *JAMA.* 2005;294:1025–1033.
  163. Horne JA, Reyner LA. Counteracting driver sleepiness: effects of napping, caffeine, and placebo. *Psychophysiology.* 1996;33:306–309.
  164. Reyner LA, Horne JA. Suppression of sleepiness in drivers: combination of caffeine with a short nap. *Psychophysiology.* 1997;34:721–725.
  165. Horne JA, Reyner LA, Barrett PR. Driving impairment due to sleepiness is exacerbated by low alcohol intake. *Occup Environ Med.* 2003;60:689–692.
  166. Barrett PR, Horne JA, Reyner LA. Alcohol continues to affect sleepiness related driving impairment, when breath alcohol levels have fallen to near-zero. *Hum Psychopharmacol.* 2004;19:421–423.
  167. Haraldsson PO, Carenfelt C, Laurell H, Tornros J. Driving vigilance simulator test. *Acta Otolaryngol.* 1990;110:136–140.
  169. Haraldsson PO, Carenfelt C, Persson HE, Sachs C, Tornros J. Simulated long-term driving performance before and after uvulopalatopharyngoplasty. *ORL J Otorhinolaryngol Relat Spec.* 1991;53:106–110.
  169. Haraldsson PO, Carenfelt C, Lysdahl M, Tornros J. Long-term effect of uvulopalatopharyngoplasty on driving performance. *Arch Otolaryngol Head Neck Surg.* 1995;121:90–94.
  170. Sullivan C, Issa F, Berthon-Jones M, et al. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. *Lancet.* 1981;1:862–865.
  171. Kushida CA, Littner MR, Hirshkowitz M, et al. Practice parameters for the use of continuous and bilevel positive airway pressure devices to treat adult patients with sleep-related breathing disorders. *Sleep.* 2006;29:375–380.
  172. Strohl K, Redline S. Nasal CPAP therapy, upper airway muscle activation, and obstructive sleep apnea. *Am Rev Respir Dis.* 1986;134:555–558.
  173. Janson C, Nöges E, Svedberg-Brandt S, Lindberg E. What characterizes patients who are unable to tolerate continuous positive airway pressure (CPAP) treatment? *Respir Med.* 2000;94:145–149.
  174. Kribbs NB, Pack AI, Kline LR, et al. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am Rev Respir Dis.* 1993;147:887–895.
  175. McArdle N, Devereux G, Heidarnajad H, Engleman HM, Mackay TW, Douglas NJ. Long-term use of CPAP therapy for sleep apnea/hypopnea syndrome. *Am J Respir Crit Care Med.* 1999;159:1108–1114.
  176. Stradling J, Davies R. Is more NCPAP better? *Sleep.* 2000;23(suppl 4):S150–153.
  177. Weaver TE, Maislin G, Dinges D, Pack AI, Group MS. CPAP dose duration for effective outcome response [Abstract]. *Am J Respir Crit Care Med.* 2003;167:A234.
  178. Pevernagie DA, Shepard JW Jr. Relations between sleep stage, posture and effective nasal CPAP levels in OSA. *Sleep.* 1992;15:162–167.
  179. Series F, Marc I. Importance of sleep stage-and body position-dependence of sleep apnoea determining benefits of auto-CPAP therapy. *Eur Respir J.* 2001;18:170–175.
  180. Oksenberg A, Silverberg D, Arons E, Radwan H. The sleep supine position has a major effect on optimal nasal continuous positive airway pressure: relationship with rapid eye movements and non-rapid eye movements sleep, body mass index, respiratory disturbance index, and age. *Chest.* 1999;116:1000–1006.
  181. Ayas NT, Patel SR, Malhotra A, et al. Auto-titrating versus standard continuous positive airway pressure for the treatment of obstructive sleep apnea: results of a meta-analysis. *Sleep.* 2004;27:249–253.
  182. Fitzpatrick MF, Alloway CE, Wakeford TM, MacLean AW, Munt PW, Day AG. Can patients with obstructive sleep apnea titrate their own continuous positive airway pressure? *Am J Respir Crit Care Med.* 2003;167:716–722.
  183. Masa JF, Jimenez A, Duran J, et al. Alternative methods of titrating continuous positive airway pressure: a large multicenter study. *Am J Respir Crit Care Med.* 2004;170:1218–1224.
  184. Planes C, D'Ortho M, Foucher A, et al. Efficacy and cost of home-initiated auto-nCPAP versus conventional nCPAP. *Sleep.* 2003;26:156–160.
  185. Hukins CA. Arbitrary-pressure continuous positive airway pressure for obstructive sleep apnea syndrome. *Am J Respir Crit Care Med.* 2005;171:500–505.
  186. Stradling JR, Hardinge M, Paxton J, Smith DM. Relative accuracy of algorithm-based prescription of nasal CPAP in OSA. *Respir Med.* 2004;98:152–154.
  187. Roux F, Hilbert J. Continuous positive airway pressure: new generations. *Clin Chest Med.* 2003;24:315–342.
  188. Sanders M, Kern N. Obstructive sleep apnea treated by independently adjusted inspiratory and expiratory positive airway pressures via nasal mask. Physiologic and clinical implications. *Chest.* 1990;98:317–324.
  189. Gay P, Weaver T, Loubé D, Iber C; Positive Airway Pressure Task Force; Standards of Practice Committee; American Academy of Sleep Medicine. Evaluation of positive airway pressure treatment for sleep related breathing disorders in adults. *Sleep.* 2006;29:381–401.
  190. Aloia MS, Stanchina M, Arnedt JT, Malhotra A, Millman RP. Treatment adherence and outcomes in flexible vs standard continuous positive airway pressure therapy. *Chest.* 2005;127:2085–2093.
  191. Campos-Rodriguez F, Pena-Grinan N, Reyes-Nunez N, et al. Mortality in obstructive sleep apnea–hypopnea patients treated with positive airway pressure. *Chest.* 2005;128:624–633.
  192. Findley L, Smith C, Hooper J, Dineen M, Suratt PM. Treatment with nasal CPAP decreases automobile accidents in patients with sleep apnea. *Am J Respir Crit Care Med.* 2000;161:857–859.
  193. Becker HF, Jerrentrup A, Ploch T, et al. Effect of nasal continuous positive airway pressure treatment on blood pressure in patients with obstructive sleep apnea. *Circulation.* 2003;107:68–73.
  194. Kanagala R, Murali N, Friedman PA, et al. Obstructive sleep apnea and the recurrence of atrial fibrillation. *Circulation.* 2003;107:2589–2594.
  195. Logan AG, Tkacova R, Perlikowski SM, et al. Refractory hypertension and sleep apnoea: effect of CPAP on blood pressure and baroreflex. *Eur Respir J.* 2003;21:241–247.
  196. Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea–hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet.* 2005;365:1046–1053.
  197. Peker Y, Hedner J, Norum J, Kraiczi H, Carlson J. Increased incidence of cardiovascular disease in middle-aged men



- with obstructive sleep apnea: a 7-year follow-up. *Am J Respir Crit Care Med*. 2002;166:159–165.
198. Pepperell JC, Ramdasssingh-Dow S, Crosthwaite N, et al. Ambulatory blood pressure after therapeutic and subtherapeutic nasal continuous positive airway pressure for obstructive sleep apnoea: a randomised parallel trial. *Lancet*. 2002; 359:204–210.
  199. Suzuki M, Otsuka K, Guilleminault C. Long-term nasal continuous positive airway pressure administration can normalize hypertension in obstructive sleep apnea patients. *Sleep*. 1993;16:545–549.
  200. Babu AR, Herdegen J, Fogelfeld L, Shott S, Mazzone T. Type 2 diabetes, glycemic control, and continuous positive airway pressure in obstructive sleep apnea. *Arch Intern Med*. 2005;165:447–452.
  201. Harsch I, Schahin P, Radespiel-Troger M, et al. Continuous positive airway pressure treatment rapidly improves insulin sensitivity in patients with obstructive sleep apnea syndrome. *Am J Respir Crit Care Med*. 2004;169:156–162.
  202. Bahammam A, Delaive K, Manfreda RJ, Roos L, Kryger MH. Health care utilization in males with obstructive sleep apnea syndrome two years after diagnosis and treatment. *Sleep*. 1999;22: 740–747.
  203. Sanner BM, Klewer J, Trumm A, Randerath W, Kreuzer I, Zidek W. Long-term treatment with continuous positive airway pressure improves quality of life in obstructive sleep apnoea syndrome. *Eur Respir J*. 2000;16:118–122.
  204. Jenkinson C, Davies RJ, Mullins R, Stradling JR. Long-term benefits in self-reported health status of nasal continuous positive airway pressure therapy for obstructive sleep apnoea. *QJM*. 2001; 94:95–99.
  205. Peker Y, Hedner J, Johansson A, Bende M. Reduced hospitalization with cardiovascular and pulmonary disease in obstructive sleep apnea patients on nasal CPAP treatment. *Sleep*. 1997;20:645–653.
  206. Pelletier-Fleury N, Meslier N, Gagnadoux F, et al. Economic arguments for the immediate management of moderate-to-severe obstructive sleep apnoea syndrome. *Eur Respir J*. 2004;23:53–60.
  207. Mar J, Rueda JR, Duran-Cantolla J, Schechter C, Chilcott J. The cost effectiveness of nCPAP treatment in patients with moderate-to-severe obstructive sleep apnea. *Eur Respir J*. 2003;21:515–522.
  208. Kribbs NB, Pack AI, Kline LR, et al. Effects of one night without nasal CPAP treatment on sleep and sleepiness in patients with obstructive sleep apnea. *Am Rev Respir Dis*. 1993;147:1162–1168.
  209. Black J, Hirshkowitz M. Modafinil for treatment of residual excessive sleepiness in nasal continuous positive airway pressure-treated obstructive sleep apnea/hypopnea syndrome. *Sleep*. 2005; 28:464–471.
  210. Veasey S, Davis C, Fenik P, et al. Long-term intermittent hypoxia in mice: protracted hypersomnolence with oxidative injury to sleep-wake brain regions. *Sleep*. 2004;27:194–201.
  211. Kim H, Young T, Matthews CG, Weber SM, Woodard AR, Palta M. Sleep-disordered breathing and neuropsychological deficits. A population-based study. *Am J Respir Crit Care Med*. 1997;156:1813–1819.
  212. Engleman HM, Douglas NJ. Sleep. 4: Sleepiness, cognitive function, and quality of life in obstructive sleep apnoea/hypopnoea syndrome. *Thorax*. 2004;59:618–622.
  213. Redline S, Strauss ME, Adams N, et al. Neuropsychological function in mild sleep-disordered breathing. *Sleep*. 1997; 20:160–167.
  214. Sanchez AI, Buela-Casal G, Paz Bermudez M, Cabello-Salas R. Effects of nCPAP treatment over reaction time and sleepiness levels during vigilance. *Clin Neuropsychol*. 2004;18:277–283.
  215. Engleman HM, Martin SE, Deary IJ, Douglas NJ. Effect of continuous positive airway pressure treatment on daytime function in sleep apnoea/hypopnoea syndrome. *Lancet*. 1994;343:572–575.
  216. Engleman HM, Martin SE, Dreary IJ, Douglas NJ. Effect of CPAP therapy on daytime function in patients with mild sleep apnoea/hypopnoea syndrome. *Thorax*. 1997;52:114–119.
  217. Engleman HM, Martin SE, Kingshott RN, Mackay TW, Deary IJ, Douglas NJ. Randomised placebo controlled trial of daytime function after continuous positive airway pressure (CPAP) therapy for the sleep apnoea/hypopnoea syndrome. *Thorax*. 1998;53:341–345.
  218. Engleman HM, Kingshott RN, Wraith PK, Mackay TW, Deary IJ, Douglas NJ. Randomized placebo-controlled crossover trial of continuous positive airway pressure for mild sleep apnea/hypopnea syndrome. *Am J Respir Crit Care Med*. 1999;159:461–467.
  219. Bardwell WA, Ancoli-Israel S, Berry CC, Dimsdale J. Neuropsychological effects of one-week continuous positive airway pressure treatment in patients with obstructive sleep apnea: a placebo-controlled study. *Psychosom Med*. 2001;63:579–584.
  220. Marshall NS, Neill AM, Campbell AJ, Sheppard DS. Randomised controlled crossover trial of humidified continuous positive airway pressure in mild obstructive sleep apnoea. *Thorax*. 2005; 60:427–432.
  221. Kingshott R, Engleman HM, Deary IJ, Douglas NJ. Does arousal frequency predict daytime function? *Eur Respir J*. 1998;12:1264–1270.
  222. Cassel W, Ploch T, Becker C, Dugnus D, Peter JH, von Wichert P. Risk of traffic accidents in patients with sleep-disordered breathing: reduction with nasal CPAP. *Eur Respir J*. 1996;9:2606–2611.
  223. Orth M, Duchna HW, Leidag M, et al. Driving simulator and neuropsychological testing in OSAS before and under CPAP therapy. *Eur Respir J*. 2005;26: 898–903.
  224. Valencia-Flores M, Bliwise DL, Guilleminault C, Cilveti R, Clerk A. Cognitive function in patients with sleep apnea after acute nocturnal nasal continuous positive airway pressure (CPAP) treatment: sleepiness and hypoxemia effects. *J Clin Exp Neuropsychol*. 1996;18:197–210.
  225. Weaver TE, Kribbs NB, Pack AI, et al. Night-to-night variability in CPAP use over the first three months of treatment. *Sleep*. 1997;20:278–283.
  226. Engleman HM, Wild MR. Improving CPAP use by patients with the sleep apnoea/hypopnoea syndrome (SAHS). *Sleep Med Rev*. 2003;7:81–99.
  227. Weaver TE. Adherence to CPAP treatment and functional status in adults obstructive sleep apnea. In: Pack AI, ed. *Sleep Apnea: Pathogenesis, Diagnosis and Treatment*. New York: Marcel Dekker; 2002:523–554.
  228. Grunstein RR, Stewart DA, Lloyd H, Akinci M, Cheng N, Sullivan CE. Acute withdrawal of nasal CPAP in obstructive sleep apnea does not cause a rise in stress hormones. *Sleep*. 1996;19:774–782.
  229. Sforza E, Lugaresi E. Daytime sleepiness and nasal continuous positive airway pressure therapy in obstructive sleep apnea syndrome patients: effects of chronic treatment and 1-night therapy withdrawal. *Sleep*. 1995;18:195–201.
  230. Collop N, Block A, Hellard D. The effect of nightly nasal CPAP treatment on underlying obstructive sleep apnea and pharyngeal size. *Chest*. 1991;99: 855–860.



231. Hers V, Liistro G, Dury M, Collard P, Aubert G, Rodenstein DO. Residual effect of nCPAP applied for part of the night in patients with obstructive sleep apnoea. *Eur Respir J*. 1997;10:973-976.
232. Mador MJ, Krauzza M, Pervez A, Pierce D, Braun M. Effect of heated humidification on compliance and quality of life in patients with sleep apnea using nasal continuous positive airway pressure. *Chest*. 2005;128:2151-2158.
233. Duong M, Jayaram L, Camfferman D, Catcheside P, Myktyyn I, McEvoy RD. Use of heated humidification during nasal CPAP titration in obstructive sleep apnoea syndrome. *Eur Respir J*. 2005;26:679-685.
234. Chasens ER, Pack AI, Maislin G, Dinges DF, Weaver TE. Claustrophobia and adherence to CPAP treatment. *West J Nurs Res*. 2005;27:307-321.
235. Stepnowsky CJ Jr, Bardwell WA, Moore PJ, Ancoli-Israel S, Dimsdale JE. Psychologic correlates of compliance with continuous positive airway pressure. *Sleep*. 2002;25:758-762.
236. Stepnowsky CJ Jr, Marler MR, Ancoli-Israel S. Determinants of nasal CPAP compliance. *Sleep Med*. 2002;3:239-247.
237. Hoy CJ, Vennelle M, Kingshott RN, Engleman HM, Douglas NJ. Can intensive support improve continuous positive airway pressure use in patients with the sleep apnea/hypopnea syndrome? *Am J Respir Crit Care Med*. 1999;159:1096-1100.
238. Aloia MS, Di Dio L, Ilniczky N, Perlis ML, Greenblatt DW, Giles DE. Improving compliance with nasal CPAP and vigilance in older adults with OAHs. *Sleep Breath*. 2001;5:13-21.
239. Kushida CA, Morgenthaler TI, Littner MR, et al. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances: an update for 2005. *Sleep*. 2006;29:240-243.
240. Ferguson KA, Ono T, Lowe AA, Al-Majed S, Love LL, Fleetham JA. A short-term controlled trial of an adjustable oral appliance for the treatment of mild to moderate obstructive sleep apnoea. *Thorax*. 1997;52:362-368.
241. Ferguson KA, Ono T, Lowe AA, Keenan SP, Fleetham JA. A randomized crossover study of an oral appliance vs nasal-continuous positive airway pressure in the treatment of mild-moderate obstructive sleep apnea. *Chest*. 1996;109:1269-1275.
242. Randerath WJ, Heise M, Hinz R, Ruehle KH. An individually adjustable oral appliance vs continuous positive airway pressure in mild-to-moderate obstructive sleep apnea syndrome. *Chest*. 2002;122:569-575.
243. Gotsopoulos H, Chen C, Qian J, Cistulli PA. Oral appliance therapy improves symptoms in obstructive sleep apnea: a randomized, controlled trial. *Am J Respir Crit Care Med*. 2002;166:743-748.
244. Gotsopoulos H, Kelly JJ, Cistulli PA. Oral appliance therapy reduces blood pressure in obstructive sleep apnea: a randomized, controlled trial. *Sleep*. 2004;27:934-941.
245. Lim J, Lasserson TJ, Fleetham J, Wright J. Oral appliances for obstructive sleep apnoea. *Cochrane Database Syst Rev*. 2004;4:CD004435.
246. Walker-Engstrom ML, Tegelberg A, Wilhelmsson B, Ringqvist I. 4-year follow-up of treatment with dental appliance or uvulopalatopharyngoplasty in patients with obstructive sleep apnea: a randomized study. *Chest*. 2002;121:739-746.
247. Ferguson KA. The role of oral appliance therapy in the treatment of obstructive sleep apnea. *Clin Chest Med*. 2003;24:355-364.
248. Petelle B, Vincent G, Gagnadoux F, Rakotonanahary D, Meyer B, Fleury B. One-night mandibular advancement titration for obstructive sleep apnea syndrome: a pilot study. *Am J Respir Crit Care Med*. 2002;165:1150-1153.
249. Raphaelson MA, Alpher EJ, Bakker KW, Perlstrom JR. Oral appliance therapy for obstructive sleep apnea syndrome: progressive mandibular advancement during polysomnography. *Cranio*. 1998;16:44-50.
250. Ryan CF. Sleep  $\times$  9: an approach to treatment of obstructive sleep apnoea/hypopnoea syndrome including upper airway surgery. *Thorax*. 2005;60:595-604.
251. Launois SH, Feroah TR, Campbell WN, et al. Site of pharyngeal narrowing predicts outcome of surgery for obstructive sleep apnea. *Am Rev Respir Dis*. 1993;147:182-189.
252. Shepard JW Jr, Thawley SE. Evaluation of the upper airway by computerized tomography in patients undergoing uvulopalatopharyngoplasty for obstructive sleep apnea. *Am Rev Respir Dis*. 1989;140:711-716.
253. Lavie P, Gertner R, Zomer J, Podoshin L. Breathing disorders in sleep associated with 'microarousals' in patients with allergic rhinitis. *Acta Otolaryngol*. 1981;92:529-533.
254. McNicholas WT, Tarlo S, Cole P, et al. Obstructive apneas during sleep in patients with seasonal allergic rhinitis. *Am Rev Respir Dis*. 1982;126:625-628.
255. Olsen KD, Kern EB, Westbrook PR. Sleep and breathing disturbance secondary to nasal obstruction. *Otolaryngol Head Neck Surg*. 1981;89:804-810.
256. Taasan V, Wynne JW, Cassisi N, Block AJ. The effect of nasal packing on sleep-disordered breathing and nocturnal oxygen desaturation. *Laryngoscope*. 1981;91:1163-1172.
257. Series F, St Pierre S, Carrier G. Effects of surgical correction of nasal obstruction in the treatment of obstructive sleep apnea. *Am Rev Respir Dis*. 1992;146:1261-1265.
258. Verse T, Maurer JT, Pirsig W. Effect of nasal surgery on sleep-related breathing disorders. *Laryngoscope*. 2002;112:64-68.
259. Verse T, Kroker BA, Pirsig W. Tonsillectomy as a treatment of obstructive sleep apnea in adults with tonsillar hypertrophy. *Laryngoscope*. 2000;110:1556-1559.
260. Sher AE. Upper airway surgery for obstructive sleep apnea. *Sleep Med Rev*. 2002;6:195-212.
261. Finkelstein Y, Stein G, Ophir D, Berger R, Berger G. Laser-assisted uvulopalatoplasty for the management of obstructive sleep apnea: myths and facts. *Arch Otolaryngol Head Neck Surg*. 2002;128:429-434.
262. Li KK, Powell NB, Riley RW, Troell RJ, Guilleminault C. Radiofrequency volumetric reduction of the palate: an extended follow-up study. *Otolaryngol Head Neck Surg*. 2000;122:410-414.
263. St. Paul, MN: Restore Medical Incorporated; 2005 [cited 2005 Dec 29]. Available at: [www.restoremedical.com/](http://www.restoremedical.com/).
264. Riley RW, Powell NB, Guilleminault C, Nino-Murcia G. Maxillary, mandibular, and hyoid advancement: an alternative to tracheostomy in obstructive sleep apnea syndrome. *Otolaryngol Head Neck Surg*. 1986;94:584-588.
265. American Sleep Disorders Association. Practice parameters for the treatment of obstructive sleep apnea in adults: the efficacy of surgical modifications of the upper airway. *Sleep*. 1996;19:152-155.
266. Guilleminault C, Cumiskey J. Progressive improvement of apnea index and ventilatory response to CO<sub>2</sub> after tracheostomy in obstructive sleep apnea syndrome. *Am Rev Respir Dis*. 1982;126:14-20.
267. Guilleminault C, Simmons FB, Motta J, et al. Obstructive sleep apnea syndrome and tracheostomy. Long-term follow-up experience. *Arch Intern Med*. 1981;141:985-988.

268. Motta J, Guilleminault C, Schroeder JS, Dement WC. Tracheostomy and hemodynamic changes in sleep-inducing apnea. *Ann Intern Med.* 1978;89:454–458.
269. Thatcher GW, Maisel RH. The long-term evaluation of tracheostomy in the management of severe obstructive sleep apnea. *Laryngoscope.* 2003;113:201–204.
270. Schafer H, Pauleit D, Sudhop T, Gouni-Berthold I, Ewig S, Berthold HK. Body fat distribution, serum leptin, and cardiovascular risk factors in men with obstructive sleep apnea. *Chest.* 2002;122:774–778.
271. Flum DR, Salem L, Elrod JA, Dellinger EP, Cheadle A, Chan L. Early mortality among Medicare beneficiaries undergoing bariatric surgical procedures. *JAMA.* 2005;294:1903–1908.
272. Rasheid S, Banasiak M, Gallagher SF, et al. Gastric bypass is an effective treatment for obstructive sleep apnea in patients with clinically significant obesity. *Obes Surg.* 2003;13:58–61.
273. Pillar G, Peled R, Lavie P. Recurrence of sleep apnea without concomitant weight increase 7.5 years after weight reduction surgery. *Chest.* 1994;106:1702–1704.
274. Marti S, Sampol G, Munoz X, et al. Mortality in severe sleep apnoea/hypopnoea syndrome patients: impact of treatment. *Eur Respir J.* 2002;20:1511–1518.
275. Li KK. Surgical management of obstructive sleep apnea. *Clin Chest Med.* 2003;24:365–370.
276. Young T, Shahar E, Nieto FJ, et al. Predictors of sleep-disordered breathing in community-dwelling adults: the Sleep Heart Health Study. *Arch Intern Med.* 2002;162:893–900.
277. Kyzer S, Charuzi I. Obstructive sleep apnea in the obese. *World J Surg.* 1998;22:998–1001.
278. Namyslowski G, Scierski W, Mrowka-Kata K, Kawecka I, Kawecki D, Cze-cior E. Sleep study in patients with overweight and obesity. *J Physiol Pharmacol.* 2005;56(suppl 6):59–65.
279. Lambert MV, Bird JM. Obstructive sleep apnoea following rapid weight gain secondary to treatment with viga-batrin (Sabril). *Seizure.* 1997;6:233–235.
280. Newman AB, Foster G, Givelber R, Nieto FJ, Redline S, Young T. Progression and regression of sleep-disordered breathing with changes in weight: the Sleep Heart Health Study. *Arch Intern Med.* 2005;165:2408–2413.
281. Peppard PE, Young T, Palta M, Dempsey J, Skatrud J. Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA.* 2000;284:3015–3021.
282. Phillips BG, Hisel TM, Kato M, et al. Recent weight gain in patients with newly diagnosed obstructive sleep apnea. *J Hypertens.* 1999;17:1297–1300.
283. Redline S, Schluchter MD, Larkin EK, Tishler PV. Predictors of longitudinal change in sleep-disordered breathing in a nonclinic population. *Sleep.* 2003;26:703–709.
284. Dixon JB, Schachter LM, O'Brien PE. Sleep disturbance and obesity: changes following surgically induced weight loss. *Arch Intern Med.* 2001;161:102–106.
285. Karason K, Lindroos AK, Stenlof K, Sjostrom L. Relief of cardiorespiratory symptoms and increased physical activity after surgically induced weight loss: results from the Swedish Obese Subjects study. *Arch Intern Med.* 2000;160:1797–1802.
286. Sampol G, Munoz X, Sagales MT, et al. Long-term efficacy of dietary weight loss in sleep apnoea/hypopnoea syndrome. *Eur Respir J.* 1998;12:1156–1159.
287. Smith PL, Gold AR, Meyers DA, Haponik EF, Bleecker ER. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. *Ann Intern Med.* 1985;103:850–855.
288. Strobel RJ, Rosen RC. Obesity and weight loss in obstructive sleep apnea: a critical review. *Sleep.* 1996;19:104–115.