

### INDICATIONS AND STANDARDS FOR CARDIOPULMONARY SLEEP STUDIES<sup>1</sup>

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#### EXECUTIVE SUMMARY

##### Indications for Cardiopulmonary Sleep Studies

###### *Chronic Obstructive Pulmonary Disease*

1. Polysomnography is indicated in patients with COPD whose awake  $P_{aO_2}$  is greater than 55 mm Hg but whose illness is complicated by pulmonary hypertension, right heart failure, or polycythemia.
2. Overnight ear oximetry can be very useful in prescribing an appropriate  $O_2$  flow rate during sleep in patients in whom continuous  $O_2$  therapy is indicated (i.e., patients whose awake  $P_{aO_2}$  is less than 55 mm Hg).
3. Overnight ear oximetry or polysomnography are not indicated in patients with COPD whose awake  $P_{aO_2}$  is greater than 55 mm Hg and who are free of complications (pulmonary hypertension, right heart failure, or polycythemia).

###### *Restrictive Ventilatory Disorders*

4. Polysomnography is indicated in patients with restrictive ventilatory impairment secondary to chest wall and neuromus-

cular disturbances whose illness is complicated by chronic hypoventilation, polycythemia, pulmonary hypertension, disturbed sleep, morning headaches, or daytime somnolence and fatigue.

5. Overnight sleep studies are not indicated in patients with restrictive chest wall, neuromuscular, or interstitial lung diseases who are not chronically hypoventilating and who are free of polycythemia, pulmonary hypertension, disturbed sleep, morning headaches, or daytime somnolence and fatigue.

###### *Disorders of Respiratory Control*

6. Polysomnography is indicated in patients with disturbances of respiratory control whose awake  $P_{aCO_2}$  is greater than 45 mm Hg, or whose illness is complicated by pulmonary hypertension, polycythemia, disturbed sleep, morning headaches, or daytime somnolence and fatigue.

###### *Risk Factors for Sleep Apnea*

7. Although snoring and obesity are risk factors for obstructive sleep apnea, the mere presence of snoring or obesity, without other symptoms, is not an indication for a cardiopulmonary sleep study.

###### *Symptoms Arising from Sleep Apnea*

8. Polysomnography is indicated in patients with excessive daytime sleepiness or sleep maintenance insomnia.

###### *Cardiovascular Manifestations of Sleep Apnea*

9. Polysomnography is indicated in patients with nocturnal cyclic bradytachyarrhythmias, nocturnal abnormalities of atrioventricular conduction, and ventricular ectopy during sleep that appears increased relative to wakefulness.
10. In the absence of other features of sleep apnea, overnight sleep studies are not indicated in patients with systemic hypertension, or in patients with nocturnal nonspecific cardiac arrhythmias.

###### **Timing and Number of Studies**

###### *Daytime Nap versus Overnight Studies*

1. Overnight polysomnography is recommended for the diagnosis and management of sleep-related breathing disorders.

However, daytime nap studies may be adequate to establish a diagnosis of sleep apnea provided that 2 to 4 hours of documented sleep are achieved, including both non-REM and REM sleep.

2. Nap studies are inadequate to definitely exclude a diagnosis of sleep apnea.

###### *Number of Sleep Studies*

3. A single overnight polysomnographic study is sufficient to exclude clinically important sleep apnea.
4. In patients with severe and unambiguous obstructive sleep apnea, the initiation of treatment with nasal CPAP may be incorporated into the diagnostic study night.

###### **Technology and Methods**

###### *Measurement Techniques*

1. Sleep studies for evaluation of cardiopulmonary sleep disorders should include an assessment of sleep stages, respiratory airflow and effort, arterial oxygen saturation, body position, and periodic leg movements.
2. Several techniques for measurement of respiratory airflow and effort are in current use, but there are insufficient data available to permit recommendations regarding standardization of instrumentation. Although measurement of esophageal pressure is the optimal technique for detection of respiratory effort, noninvasive techniques are usually adequate for clinical purposes.
3. Currently used ear oximeters are adequate for measurement of arterial  $O_2$  saturation in patients with cardiopulmonary sleep disorders; finger pulse oximeters may be inadequate.

###### *Scoring and Interpretation of Data*

4. Formalized scoring of the entire polysomnographic record is recommended.
5. Quantitative data derived from polysomnographic studies should be incorporated into a report that includes information regarding sleep stages, types of respiratory patterns and their relationship

<sup>1</sup> Reprints may be requested from your state or local lung associations.

to sleep stage and sleeping posture, arterial oxygen saturation, cardiac rate and rhythm, and interpretation of the findings.

6. There is a need to develop standard definitions and criteria by which to analyze and report respiratory events during sleep.
7. The development of computer-assisted techniques for the automated analysis of polysomnographic data should be pursued, but at present there are no such systems commercially available that can be recommended for clinical purposes.
8. There is a need for epidemiologic studies to identify the polysomnographic findings that are of clinical importance in the definition of sleep apnea and other cardiopulmonary disturbances during sleep.

#### *Screening and Ambulatory Monitoring Techniques*

9. The role of screening tests for the diagnosis of sleep apnea has not been established.
10. Currently available ambulatory monitoring systems have not been validated for the diagnosis of sleep apnea. However, the development of such systems is encouraged.

#### **Introduction**

During the past decade, respiratory physicians have become increasingly aware of the need to consider an evaluation of ventilation and gas exchange during sleep in patients with a variety of established or suspected cardiorespiratory disorders, including the sleep apnea syndromes, chronic obstructive pulmonary disease (COPD), asthma, cystic fibrosis, interstitial lung disease, pulmonary hypertension, structural chest wall abnormalities, respiratory neuromuscular disorders, and central hypoventilation syndromes. This increasing awareness of the potentially important role of sleep studies in evaluating such disorders has contributed to a proliferation of diagnostic sleep laboratories throughout North America and has stimulated considerable research into breathing and its disorders during sleep. Because of the large number of potential patients in whom sleep monitoring may be useful, the physician is faced with the dilemma of deciding which patients should undergo sleep studies, and sleep laboratories are faced with an increasing demand for expensive and time-consuming services. As a result, there is a need for the development of practical guidelines regarding the indications for cardiopulmonary sleep studies and the standards that such studies should meet.

Based on these considerations, the American Thoracic Society convened a Consensus Conference in September 1986, the objectives of which were to: (1) define the indications for cardiopulmonary sleep studies in adults; (2) define the type of sleep studies that are appropriate for cardiorespiratory disturbances during sleep; (3) identify areas of further research that are required to allow more defini-

tive conclusions to be reached regarding the indications and standards for cardiopulmonary sleep studies.

To meet these objectives, participants were selected from within and without the adult medicine respiratory community, representing a broad geographic distribution and a cross-section of clinical and research sleep centers and laboratories in North America. The format of the conference consisted of the presentation of one or two position papers on each topic, followed by an in-depth discussion, and the formulation of a consensus statement. The following report summarizes the position papers and consensus statements. The statements are applicable to the adult patient population and are not intended to apply to pediatric disorders.

#### **Definitions**

*Polysomnography* refers to a method of identifying and evaluating sleep-state and several physiologic variables during sleep.

*Cardiopulmonary sleep studies*, as used in this report, refers to a polysomnographic study in which the focus of physiologic interest is on cardiac and respiratory variables. Studies in which only cardiac or respiratory variables are recorded (without an evaluation of sleep state) are often referred to as *screening studies* but are more correctly referred to as *simplified studies*.

#### **Indications for Cardiopulmonary Sleep Studies**

##### *Chronic Obstructive Pulmonary Disease*

**Background.** Several studies have documented that patients with stable COPD are at risk of experiencing a deterioration in arterial oxygenation during sleep (1-10). The severity of hypoxemia is generally most severe during REM sleep (3, 6, 8-10). The cause of sleep-induced hypoxemia in COPD probably involves both a decrease in alveolar ventilation and a deterioration in gas exchange due to an increased ventilation-perfusion mismatching (9-12). Low flow supplemental O<sub>2</sub> ameliorates or eliminates sleep-induced arterial O<sub>2</sub> desaturation (10, 13).

Decreases in arterial O<sub>2</sub> saturation during sleep in patients with COPD have been shown to be accompanied by acute increases in pulmonary arterial pressure (6, 8, 9, 13, 14). Furthermore, supplemental O<sub>2</sub>, sufficient to prevent desaturation, also prevents the acute increases in pulmonary arterial pressure (13, 14). Several investigators have postulated that over a period of years sleep-induced hypoxemia in patients with COPD may contribute to development of long-term complications, including sustained pulmonary hypertension, right heart failure, polycythemia, and central nervous system dysfunction (15-17). If so, supplemental nocturnal O<sub>2</sub> would be expected to delay the development of, or attenuate the progression of, such complications. Some support for this notion can be derived from previous trials of supplemental O<sub>2</sub> in patients whose daytime PaO<sub>2</sub> was below 55 mm Hg,

and in whom complications (cor pulmonale and polycythemia) were already present (18-20). However, it is not known whether nocturnal desaturation in patients whose awake PaO<sub>2</sub> is greater than 55 mm Hg (21) and who are free of complications leads to long-term complications, or whether supplemental nocturnal O<sub>2</sub> in such patients delays the development of complications. Such information is required in these particular patients because supplemental O<sub>2</sub> therapy is generally not considered to be indicated when PaO<sub>2</sub> is greater than 55 mm Hg and the patient is free of complications (18, 19). Furthermore, although there is a correlation between reduced daytime PaO<sub>2</sub> and nocturnal desaturation in patients with COPD (22), it is not possible to precisely predict from daytime assessments alone the severity of nocturnal desaturations (21). Therefore, if nocturnal O<sub>2</sub> were found to be beneficial in patients whose PaO<sub>2</sub> is greater than 55 mm Hg, overnight ear oximetry would be required to identify patients in whom nocturnal O<sub>2</sub> therapy might be expected to produce important long-term clinical benefits (even when daytime O<sub>2</sub> is not indicated).

Cardiac arrhythmias during sleep are common in patients with COPD (23, 24). However, the hazards of such arrhythmias are unknown, particularly in patients without cardiac symptoms. Furthermore, it has not been established that potentially dangerous arrhythmias are responsive to supplemental nocturnal O<sub>2</sub>.

When patients with COPD experience an acute exacerbation of their disease, hypoxemia usually becomes more severe and oxygen needs increase. No studies have evaluated changes in arterial oxygenation during the disturbed sleep that is typical of such exacerbations, but it is reasonable to assume that nocturnal oxygenation deteriorates. It is estimated that 4 weeks or more may be required to achieve stability of arterial oxygenation after acute exacerbations (25).

**Consensus statement.** Investigations on which decisions are based regarding long-term management of patients with COPD should be done in the context of clinical stability, which may require 4 weeks or more to be achieved after an acute exacerbation. In stable patients, if the awake supine or semirecumbent PaO<sub>2</sub> is less than 55 mm Hg, supplemental O<sub>2</sub> is indicated (18, 19). Therefore, polysomnography or overnight ear oximetry are not required to reach a decision regarding the need for O<sub>2</sub> therapy. However, overnight oximetry can be very useful in prescribing an appropriate O<sub>2</sub> flow rate during sleep. In patients whose awake supine PaO<sub>2</sub> is greater than 55 mm Hg and who are free of complications, routine overnight ear oximetry is not indicated because the long-term consequences of possible episodic nocturnal desaturation and the possible beneficial effects of O<sub>2</sub> are unknown. Accordingly, there is a need for a prospective trial to assess the possible benefits of nocturnal O<sub>2</sub> in such patients.

Based on the preceding recommendations,

it is apparent that in the majority of patients with COPD, overnight oximetry is unlikely to alter management except to insure sufficient supplemental  $O_2$  when such treatment is indicated. In contrast, in patients whose awake supine  $P_{aO_2}$  is greater than 55 mm Hg and who also have pulmonary hypertension, right heart failure, or polycythemia, polysomnography is indicated to exclude sleep-induced desaturation as a possible factor contributing to these complications, and if significant desaturation is demonstrated, to establish the cause and to exclude other concomitant disorders, particularly obstructive sleep apnea.

Although cardiac arrhythmias during sleep are common in patients with COPD, mass screening of such patients for arrhythmias (by Holter monitoring) cannot be recommended in the absence of cardiac symptoms. Rather, research studies are needed to determine the clinical importance of such arrhythmias. Patients with COPD who undergo overnight Holter monitoring because of known or suspected cardiac disease, and who are found to have nocturnal arrhythmias, should be investigated and managed the same way as other patients with cardiac arrhythmias.

#### *Restrictive Ventilatory Disorders*

**Background.** Patients with restrictive ventilatory impairment secondary to distortion of the chest wall (such as kyphoscoliosis) or weakness of the respiratory muscles may experience considerable deterioration of alveolar ventilation and gas exchange during sleep, resulting in arterial  $O_2$  desaturation and  $CO_2$  retention (26-29). The degree of deterioration is generally most severe during REM sleep. Several factors contribute to these sleep-induced changes (30): a decrease in functional residual capacity in the supine posture; loss of the stimulatory effect of wakefulness on breathing, which may be particularly prominent in patients with underlying abnormalities of respiratory control; decreases in activity of the intercostal and accessory respiratory muscles during REM sleep; and a rapid-shallow pattern of breathing typical of phasic REM sleep, which may increase dead space ventilation excessively. Both central and obstructive sleep apneas have also been described in such patients (26, 27, 29). The degree of sleep-induced deterioration in ventilation and gas exchange in patients with chest wall and neuromuscular disturbances cannot be accurately predicted from awake pulmonary function tests, resting  $P_{aCO_2}$ , or angle of spinal deformity (31). In most cases, the best predictors are the clinical features, with polycythemia, pulmonary hypertension, cor pulmonale, disturbed sleep, morning headache, and daytime somnolence or generalized fatigue generally indicating the presence of severe nocturnal disturbances. Supplemental nocturnal  $O_2$  can attenuate arterial desaturation during sleep in such patients, but usually aggravates the severity of  $CO_2$  retention and may not alleviate the patient's symptoms (29). In contrast, assisted overnight ventilation generally improves daytime performance as

well as daytime blood gases, thereby lending support to the notion that the natural progression of the clinical disorder relates to nocturnal deterioration in alveolar ventilation (29, 32).

In contrast to patients with chest wall and neuromuscular disturbances, patients with restrictive ventilatory impairment secondary to interstitial pulmonary disease do not hypoventilate during sleep. However, such patients may experience some degree of arterial  $O_2$  desaturation particularly during REM sleep (33, 34). Patients who also snore tend to have greater falls in saturation (34).

**Consensus statement.** There is no indication for routine overnight sleep studies in patients with kyphoscoliosis, previous poliomyelitis, or neuromuscular weakness who are asymptomatic and free of chronic hypoventilation. However, research studies are required in such patients to better define the natural history of these disorders. In contrast, in patients with chronic hypoventilation, polycythemia, pulmonary hypertension, disturbed sleep, morning headaches, daytime somnolence, or generalized fatigue, polysomnography is indicated. In addition to routine polysomnographic measurements, such studies should include an assessment of upper airway function and measurement of transcutaneous  $PCO_2$ . When such patients are treated with supplemental nocturnal  $O_2$  or assisted ventilation, polysomnography and transcutaneous  $PCO_2$  monitoring are indicated to determine the efficacy of treatment and to ensure that further  $CO_2$  retention or iatrogenic obstructive sleep apneas are not present (29). In patients with interstitial lung disease who are not hypoventilating during wakefulness, there is little evidence that overnight oximetry or polysomnography alters management, and therefore routine overnight studies are not indicated in such patients.

#### *Disorders of Respiratory Control*

**Background.** Disorders of respiratory control for which sleep studies may be indicated are those that result in alveolar hypoventilation syndromes. In the purest form, disorders of respiratory control arise because of defects in the sensors (peripheral and central chemoreceptors) or brainstem neurons that constitute the metabolic respiratory control system (35, 36). Defects in this system can be idiopathic in nature (primary alveolar hypoventilation syndrome) (37, 38) or secondary to brainstem infection (39, 40), vascular lesions (41), or other disorders (42). The clinical features resulting from such disorders are attributable to chronic hypercapnia and hypoxia and include lethargy and fatigue, difficulty concentrating attention, pulmonary hypertension, and right heart failure (37, 43-45). Typically, such patients also complain of restless sleep, morning headache, and daytime sleepiness, which suggests aggravation of the ventilatory disturbance during sleep. By definition, they have laboratory evidence of chronic alveolar hypoventilation, including hypercapnia, hypoxemia, respiratory acidosis, and polycythemia, and usually have evi-

dence of weak ventilatory drive, including diminished ventilatory responses to hypercapnia and hypoxia. Similar clinical and laboratory features can also result from disorders of the respiratory neuromuscular system (28), chest wall (46, 47), and airways and lungs (48). Frequently, defects in respiratory control coexist with these other disorders and contribute to their natural progression.

Without treatment, disorders of respiratory control that result in chronic alveolar hypoventilation are usually progressive and ultimately fatal. Furthermore, sleep typically aggravates the degree of hypoventilation (39, 49-52). Supplemental nocturnal  $O_2$  can attenuate the magnitude of sleep-induced desaturation (51, 52), and, in some patients, may increase ventilation (53). In others, nocturnal  $O_2$  further depresses ventilation and increases  $CO_2$  retention (51, 52). Similarly, nocturnal negative pressure ventilation (29) or electrophrenic diaphragm pacing (54, 55) can substantially improve nocturnal ventilation but run the risk of inducing obstructive apneas during sleep. This problem can be avoided by intermittent positive pressure ventilation through a nasal mask (56).

**Consensus statement.** Overnight polysomnography is indicated in patients with disturbances of respiratory control whose daytime awake  $P_{aCO_2}$  is greater than 45 mm Hg. Studies are also indicated in patients with unexplained pulmonary hypertension or unexplained polycythemia. Such studies should include measurements of standard polysomnographic variables, arterial  $O_2$  saturation, and transcutaneous or end-tidal  $PCO_2$  and should permit the identification or exclusion of obstructive apneas or hypopneas during sleep. Polysomnography is also indicated to establish that therapeutic interventions such as supplemental nocturnal  $O_2$ , negative pressure ventilation, or electrophrenic diaphragm pacing are of benefit and without detrimental side effects.

#### *Risk Factors for Sleep Apnea*

**Background.** Snoring is a coarse, harsh respiratory sound typically caused by vibration of the uvula and soft palate. The sound is a manifestation of a process that causes mechanical loading of the respiratory system, which, in severe cases, can produce hypoventilation during sleep. Statistically, snorers are at risk for obstructive sleep apnea (OSA), hypertension, and cerebrovascular accidents (57). Considerable clinical importance attaches to a history of periodic pauses in snoring pattern.

Obesity is common among patients with OSA, but the prevalence of OSA in obese individuals has not been established. In one study, 36% of males referred for weight loss (with weights of 150 to 200% of predicted) were found to have greater than 30 apneas per night; few of these persons were completely asymptomatic (58). In contrast, the prevalence of OSA among asymptomatic obese males has not been defined. Common clinical experience indicates that weight loss can

ameliorate OSA, but thresholds for such an effect cannot be predicted (59, 60).

**Consensus statement.** Snoring and obesity are risk factors for sleep apnea. Snoring is indicative of upper airway narrowing and may be associated with obstructive apnea or hypopnea. Obesity appears to play a pathogenetic role in OSA. However, the mere presence of snoring or obesity alone, without other symptoms, is not an indication for a cardiopulmonary investigation during sleep. There is a need for additional studies on the relationship of these risk factors to the development of OSA.

#### *Symptoms Arising from Sleep Apnea*

**Background.** The symptom of excessive daytime sleepiness (EDS) is serious and frequently is a manifestation of sleep apnea (61, 62). It has been estimated that EDS is present in as much as 5% of the population, and of these persons, 30 to 40% have OSA (63). The multiple sleep latency test (MSLT) measures the tendency to fall asleep. Although the majority of patients whose EDS is due to OSA have a reduced sleep latency (64), the results of this test do not always correlate with the clinical history because some patients with daytime somnolence are unaware of it.

Patients with sleep maintenance insomnia (SMI) (frequent awakening during sleep) may exhibit sleep apnea; the probability of sleep apnea increases with age (65). If other causes of SMI (such as excessive time in bed, ingestion of medications or alcohol) are excluded, complaints of SMI in older persons are associated with sleep apnea in a large fraction of cases (66).

**Consensus statement.** Excessive daytime sleepiness is a serious symptom and deserves investigation with a polysomnographic study because a substantial fraction of patients with EDS have sleep apnea. Sleep maintenance insomnia is also associated with breathing disturbances, particularly in older persons. Therefore, if other causes of this symptom are excluded, polysomnography is indicated to exclude sleep apnea. Further research is needed to determine the prevalence of sleep apnea in patients with EDS and SMI.

#### *Cardiovascular Manifestations of Sleep Apnea*

**Background.** During sleep, patients with OSA display a rise in systemic blood pressure, in contrast to the normal decrease in pressure. Furthermore, many patients with OSA have systemic hypertension which normalizes after treatment of the sleep apnea. Of patients with essential hypertension, 30% appear to have breathing disorders during sleep (67-70). However, this association is not sufficiently strong to mandate a cardiopulmonary evaluation during sleep in otherwise asymptomatic hypertensive individuals, particularly in view of the fact that therapy for such patients will probably not be influenced by the results of this study.

The detection of cardiac arrhythmias during the night does not necessarily indicate that

they are occurring during sleep. While sleep is usually a time of increased bradyarrhythmias and decreased ventricular ectopy, the clinical implications of such findings are unknown. Periodic bradyarrhythmias during sleep suggest the presence of OSA except when they occur in the setting of a patient with an inferior myocardial infarction or in a young, healthy subject (71). Ventricular ectopy occurring almost exclusively during sleep should alert the clinician to the possibility that sleep apnea is the underlying cause of the arrhythmia (72-74).

**Consensus statement.** OSA can be associated with systemic hypertension and nocturnal nonspecific cardiac arrhythmias. However, in the absence of other signs or symptoms of OSA, the presence of hypertension or nocturnal nonspecific cardiac arrhythmias does not warrant a cardiopulmonary evaluation during sleep. In contrast, certain cardiac arrhythmias occurring principally during sleep (marked cyclic bradycardia, abnormalities of A-V conduction, and ventricular ectopy) may reflect a sleep-related cardiorespiratory disturbance and deserve evaluation. Additional research is needed into the natural history of these disturbances.

#### **Timing and Number of Sleep Studies**

##### *Daytime Nap Versus Overnight Studies*

**Background.** Daytime naps, by definition, differ from overnight studies both in the duration and timing of sleep. The validity of a nap study for the diagnosis of sleep apnea is based on the assumption that breathing disturbances associated with a given stage of sleep do not demonstrate a circadian variation. Only one publication (an abstract) has compared daytime nap results to overnight study results (75). The study concluded that afternoon nap recordings are often inadequate for evaluation of sleep-related breathing disturbances. In contrast, another study (76) compared the first 4 h of an overnight study with the entire night and found the two analyses to be highly correlated.

Patients with severe daytime sleepiness and suspected OSA (who have no symptoms suggestive of other sleep disorders) are the best candidates for daytime nap studies because of the ease with which they fall asleep. However, for an adequate nap study, 2 to 4 hours of sleep should be obtained, including both non-REM and REM sleep, and sleep in the supine posture. Otherwise, the severity of sleep apnea may be underestimated, which may influence therapeutic decisions. If the results of the nap study do not confirm the clinical diagnosis, an all-night sleep study is necessary. It has not been demonstrated that because nap studies are shorter and take place in the daytime, they are necessarily more cost effective.

**Consensus statement.** Although minimal systematic data exist on the value of nap recordings, nap studies of 2 to 4 hours' duration may be used to confirm the diagnosis of sleep apnea, provided that all routine poly-

somnographic variables are recorded, that both non-REM and REM sleep are sampled, and that the patient spends at least part of the time in the supine posture. Sleep deprivation or the use of drugs to induce a nap are contraindicated. Nap studies are inadequate to definitively exclude a diagnosis of sleep apnea.

##### *Number of Sleep Studies*

**Background and consensus statement.** The first night of sleep in a sleep laboratory differs from sleep on subsequent nights, being characterized by decreased sleep efficiency and prolonged REM latency. These differences are referred to as "the first night effect" (77). However, the differences have little clinical significance in the evaluation of sleep-related breathing disorders. Similarly, although there is night-to-night variability in the frequency of respiratory disturbances in patients in whom such events are infrequent (78), this variability is of little consequence in patients with clinically important respiratory disturbances during sleep. Based on these considerations, one night of polysomnography is considered sufficient to exclude clinically important sleep apnea.

When patients with sleep apnea undergo treatment, follow-up evaluation, which may include polysomnography, is required to determine the adequacy of treatment. The timing of follow-up studies depends on the nature of the treatment (weight loss, medications, CPAP, surgery, assisted ventilation, phrenic pacing). In patients with severe and unambiguous OSA, initiation of nasal CPAP treatment may be incorporated into the diagnostic study night, and if successful in abolishing obstructive events, may be expected to yield long-term improvement that is comparable to that achieved after a separate nasal CPAP trial night (79, 80).

#### **Technology and Methods**

##### *Measurement Techniques*

**Background.** Studies of patients with suspected cardiopulmonary sleep disorders require the use of methods to simultaneously and continuously monitor sleep state, respiratory pattern (both effort and airflow), oxygen saturation, the electrocardiogram, and the presence of snoring (81, 82). In addition, methods to record the electromyogram of the anterior tibialis muscle for detection of periodic movements in sleep (83) and body position should be included. The severity of sleep apnea can vary with body posture, being more severe in the supine as compared to the lateral decubitus position (84).

(1) *Sleep staging.* Techniques for monitoring sleep state are now relatively standard. They involve simultaneous recording of the electroencephalogram, electrooculogram for recording eye movements, and the electromyogram for monitoring of muscle tone. The latter two are particularly used to detect the presence of rapid eye movement (REM) sleep in which phasic eye movements and muscle at-

nia occur. Apneas may be most severe in REM sleep (85), and oxygen desaturations in patients with intrinsic lung disease are usually most marked in REM sleep (6-10, 26, 27, 29). Thus, complete assessment of the severity of sleep-disordered breathing requires measurement of respiration during REM sleep.

For recording of the electroencephalogram, electrodes are placed at the C3, C4, A1, and A2 positions following the International 10-20 system (86). Sleep stage should be recorded from the monopolar derivation C3/A2, although C4/A1 can be used as an alternative if recording difficulties develop. For recording the electrooculogram, one electrode is applied to the outer canthus of each eye with the electrode on the right outer canthus being 1 cm above the horizontal, and that on the left being 1 cm below the horizontal. Similar electrodes are used for recording the electromyogram of the submental (chin) muscles. One electrode is applied at the center of the chin, with two others beneath the chin. Although only two electrodes are required for recording purposes, the third acts as a back-up in the event of recording difficulties. The electromyogram provides information relevant to the loss of muscle tone in REM sleep, and movements with arousal such as those at apnea termination. (For further details of these techniques, see [81], [82], [87].)

(2) *Respiration.* While methods of monitoring sleep state are standard, the optimal techniques for monitoring respiration are less certain. Because differentiation of loss of activity of respiratory pump muscles (central apnea) from intermittent upper airway obstruction (obstructive apnea) is required, measurements must include some method to monitor respiratory effort combined with some method to monitor airflow. In each category, both quantitative and semiquantitative methods are employed. Semiquantitative methods which permit differentiation of central from obstructive events are considered adequate for clinical purposes. To detect mouth and nasal airflow, various devices have been employed. These include rapid response CO<sub>2</sub> analyzers, thermistors (88), laryngeal and tracheal microphones (89-91), and impedance pneumography (92, 93).

Quantitative measurements of airflow can be obtained by use of a face mask with an attached pneumotachograph. This method is more invasive and may be less well tolerated by certain subjects. In young volunteer subjects, use of pneumotachography results in some modifications of sleep pattern (increased duration of awakenings, reduced total sleep time, longer latency to REM sleep, reduced duration of REM sleep), but the effects are not major (94). Whether more marked disturbances in sleep pattern occur in other subjects, e.g., the elderly, is unknown.

Ventilation can also be measured quantitatively using magnetometers (95, 96) or the respiratory inductance plethysmograph (97, 98). Both of these are based on the principle that the respiratory system has two degrees of freedom, i.e., chest and abdominal motion (99).

Debate continues, however, as to the optimal method to calibrate the respiratory inductance plethysmograph (100, 101) and indeed whether quantitative measurements of ventilation can be obtained in unrestrained humans (102). Difficulties in the measurement relate in part to alterations in the relationship between chest and abdominal motion and volume change (calibration factors) with changes in body position (103). Also, the bands of the inductance plethysmograph may tend to move during sleep, a problem that is minimized by use of a body jerkin into which the inductance coils are sewn or glued (104). Recent improvements in this technology have been reported. These include description of a new, more robust sensor (105) and a microprocessor-based system that incorporates a new automated calibration method (106). There are, however, few data evaluating the application of these improved systems in clinical testing.

Although doubts continue to be expressed about the use of the respiratory inductance plethysmograph to quantify ventilation during sleep (102), there is no doubt that the instrument can be used in a qualitative or semiquantitative fashion to monitor surface movements, thereby providing information about respiratory effort. Absence of movements (effort) implies central apneas, whereas in obstructive apneas paradoxical movements of rib cage and abdomen occur. Other techniques can also be employed for semiquantitative assessment of effort based on similar principles. These include magnetometers (96), bellows pneumography, and mercury in silastic strain gauges (107). The latter is probably the most commonly used device in clinical testing (81, 108). In addition, the electromyogram of the inspiratory muscles (intercostal, costal diaphragm, or crural diaphragm by means of an esophageal balloon) can be directly recorded. These techniques are, however, mostly used for research purposes (see for example [109]).

Quantitative techniques to measure effort also exist. Measurement of esophageal pressure by esophageal balloon or pressure catheter can be employed. This technique may produce discomfort and interfere with sleep by reducing total sleep time and sleep efficiency in nonapneic patients (110). However, in a small sample of apneic subjects, sleep profiles did not appear to be significantly affected (110).

Thus, a large number of measurement techniques can be employed for monitoring respiration. There are, however, few studies addressing the relative efficacy of each in clinical testing and their validity in the obese subjects that are studied commonly in cardiopulmonary sleep laboratories. Nevertheless, evidence exists that monitoring of thoracoabdominal motion in such cases by magnetometers is as reliable as measurement of esophageal pressure in detecting central and obstructive apneas (96). Respiratory inductive plethysmography is also a reliable alternative, although comparisons with esophageal pressure indicate that obstructive apneas with fee-

ble inspiratory efforts may be incorrectly labeled as purely central (111). Differences are also reported among the different devices used to monitor thoracoabdominal motion. Some preliminary data suggest that strain gauges are less reliable than the respiratory inductance plethysmograph because they are more sensitive to changes in body position that may produce reductions in transducer tension (112).

(3) *Oxygen saturation.* A number of oximeters are now available for the noninvasive monitoring of arterial oxygen saturation. Spectrophotometric oximetric and photoelectric plethysmographic techniques are employed. Several studies have addressed the steady-state response of these instruments (113-121). These studies show good correlation between such measurements of saturation and independent estimates, down to saturations of the order of 65 to 70%. However, in sleep apnea it is not just the steady-state characteristics that are important but also the dynamic response because in such patients there are oscillatory changes in oxygenation. The dynamic responses of the earliest ear oximeters (Biox, Hewlett-Packard HP47201A) were measured and the 50% response times were of the order of 3 seconds in the fast mode of operation (115, 116). Recent studies have examined the response of a number of oximeters in sleep apnea patients (122) and during induced brief, but profound, hypoxia (123). These studies show that during transient hypoxemia certain oximeters may underestimate oscillations in SaO<sub>2</sub>, whereas others may overestimate. In some cases, the errors are large. In addition, finger pulse oximeters show a delay in response that, not unexpectedly, is correlated with heart rate.

*Consensus statement.* Measurements for evaluation of cardiopulmonary sleep disorders should include an assessment of sleep stage (electroencephalogram, electrooculogram, and electromyogram of a skeletal muscle), respiratory airflow and effort, arterial oxygen saturation, body position, and anterior tibialis EMG to detect periodic movements in sleep. Although there is a wide variety of techniques for measurement of respiratory airflow and effort, there is, at present, insufficient data to develop recommendations about which instrumentation is optimal. The situations in which measurements of esophageal pressure should be used as the preferred method to assess quantitatively respiratory effort are uncertain. There is a need for studies examining the error rates that are found in identification and typing of respiratory events with different currently employed methodologies.

Although all studies should include measurement of oxygen saturation by oximetry, there are important differences in the response of different oximeters that affect their ability to measure the oscillatory changes in saturation that are typical of patients with sleep apnea. Currently used ear oximeters have adequate responses for this purpose. Clinicians should investigate both the steady-state and transient responses of oximeters before em-

ploying them for cardiopulmonary sleep studies. There is a need to develop standards for such instrumentation in a similar fashion to the standards developed for spirometry (124).

### *Scoring and Interpretation of Data*

#### *Background.*

(1) *Scoring of nonrespiratory variables.* Recordings obtained by polysomnography need to be analyzed to obtain measures of the number of respiratory events during sleep, distribution of sleep stages including number of arousals, as well as the frequency of occurrence of nocturnal myoclonus. Currently, sleep staging is performed in fixed intervals (typical epoch of 30 seconds) using the criteria proposed by Rechtschaffen and Kales (125). However, such conventional criteria may be insufficient for scoring the records of patients with sleep apnea (126). In these patients, there are oscillations in the frequency content of the electroencephalogram in synchrony with the periodicities in ventilation (127). Indeed, some have proposed a modification of the standard sleep scoring system for use in sleep apnea patients (126).

Apart from scoring of sleep state, analysis of polysomnographic records can reveal the number of arousals during sleep and the frequency of occurrence of periodic movements in sleep. For the latter, the total number of movements, total number of movements associated with arousal or awakenings, and the movement index, i.e., number of movements per hour of sleep, can all be calculated (for full discussion, see [83]). There is good reliability in scoring these measures as revealed by a relatively low variation in results when the same records are scored by different persons (128).

(2) *Scoring of respiratory variables.* Scoring of respiratory events is at present more problematic. Although most, but not all (129), would agree with the "standard" definition of apnea, i.e., a cessation of airflow for greater than 10 seconds (130), there is considerable variation in the criteria used to identify a hypopnea. Hypopneas have been identified by reductions in airflow measured by thermistors (129, 131); by reductions in ventilation measured by a calibrated respiratory inductance plethysmograph (132); and by the combination of reductions in airflow and decrements in oxygen saturation that are greater than 4% (133). There are, moreover, differences in the quantitative criterion employed to define the reduction in airflow that is required for a hypopnea: some have used a 50% reduction (131, 132) and others a two-thirds reduction (134). However, such precise quantification of decrements in airflow is difficult using the relatively imprecise and essentially semiquantitative measurement system employed in these studies.

Although there is variation in the definitions of apnea and hypopnea employed by different laboratories, there is good agreement among different observers in identifying re-

spiratory events when fixed rules are employed. Relatively inexperienced observers perform as well as experienced ones (129). Agreement, however, was not good in the identification of events as being central or obstructive when thermistors were used to monitor airflow, and strain gauges or a single-channel inductive plethysmograph were used to measure respiratory effort (129).

Once identified, events are counted and indices of abnormality computed. Originally, only the number of apneas was counted, and an apnea index, i.e., number of apneas/hour of sleep, was derived (130). Recently, many investigators have summated the number and hypopneas to yield a respiratory disturbance index, i.e., the number of apneas plus hypopnea/hour of sleep. Although these indices are derived by counting events over the whole night of study, there is recent evidence that essentially identical estimates are obtained by analyzing every fourth page of the record (135). This approach will greatly reduce the amount of time required for scoring of sleep records. It is estimated that scoring a sleep apnea study by conventional methods takes between 240 and 360 minutes (136).

(3) *Indices of oxygen desaturation.* A large number of measures of oxygen desaturation have been employed. Perhaps the most frequently used is the O<sub>2</sub> saturation nadir, but this value may not be representative because it may relate to a brief and infrequent event. Other measures are mean oxygen saturation or mean oxygen saturation per sleep stage. A convenient graphical format is the cumulative oxygen saturation histogram, in which the total percentage of sleep time spent at each saturation is demonstrated (137). From this format, a number of parameters are derived: percentage of time below 90, 80, 70, and 60% and the saturation that is at the 50% time point.

(4) *Applications of computers to scoring of polysomnographic data.* With the intense time commitment involved in scoring of polysomnographic records, it is natural to consider whether the process can be automated, at least to some degree, by application of computers. Of the variables measured, the most simple to adapt to such technology is arterial oxygen saturation, which has been analyzed on-line using microcomputers (138, 139). The systems produce graphic representations of oxygen saturation as a function of time, and cumulative time spent at various oxygen saturations (137). Indices such as the number of desaturations > 4%, average desaturation per episode, percentage of time below 90, 80, 70, and 60%, as well as the 50% sleep saturation point are simply produced.

For the other variables, application of computers is more problematic. One approach is to simply use a computer to expedite manual scoring by keeping track of respiratory events, arousals, etc. This approach reduces the time involved, improves accuracy, and provides a more adequate summary of the data (140). Data processed in this way can be used to pro-

duce a detailed graphic record (sleep histogram) of the events during the sleep study (141).

More automated approaches to scoring of records have also been described, including computer analysis for sleep staging (for reviews, see [142], [143]). While such techniques satisfactorily classify normal sleep, disturbed sleep remains a problem (143). As discussed previously, sleep in patients with sleep apnea is extremely disturbed and there are doubts as to whether conventional criteria can be applied (126).

Automated approaches to assessment of respiration during sleep, i.e., detection of apneas and hypopneas, have also been described (134, 144, 145). Problems are presented, however, by body movements or changes in posture such that careful operator interaction is required. Even with such interaction, invalid data due to signal artifact will sometimes be stored (134). Because sleep staging is still scored manually, use of such systems requires careful synchronization of the computer and polygraph records. Given these problems, such automated systems are not in widespread use.

(5) *Reports of polysomnography and interpretation of results.* Currently, different laboratories use different reporting formats. There have, however, been recent recommendations about the data that should be included in the laboratory report (82). Although initially an apnea index of greater than 5 apneas/hour was considered abnormal (130), this figure can no longer be considered valid (146). This definition was based on a small sample of young to middle-aged adults, but when applied to the elderly it results in an alarming number of subjects being diagnosed as having sleep apnea, i.e., of the order of 30 to 35% (147, 148).

*Consensus statement.* Measurements of data obtained during polysomnography require that formalized scoring be performed on the entire nocturnal record. Therefore, scoring of each epoch of the record is currently recommended. Sampling of every fourth epoch appears to be adequate for analysis of respiratory variables (135), but before this approach can be recommended for routine clinical purposes, there is a need for further studies to confirm these results.

Quantitative data extracted from scoring should be incorporated into a report of the sleep study. However, there is currently lack of standardization of definitions in this field. Thus, standardization of reports at this time is not feasible. As recommended by the American College of Chest Physicians and the Association of Sleep Disorders Centers (82), reports should contain the following:

1. What variables were measured and by what method(s).
2. Sleep staging—the percentage of each sleep stage and the relationship to age-matched normals. The total sleep time, sleep efficiency, and sleep latency should be noted.
3. Type(s) of respiratory patterns, as well as the total number, number per hour of sleep

time, and range and mean duration of patterns, and relationship to sleep stage. The patterns should be defined.

4. Relationship of body position to disordered breathing, if pertinent.

5. Oxygen saturation—the awake baseline level; arterial oxygenation should be described in quantitative terms, using either a continuous saturation versus time technique, or discrete intervals, e.g., amount or percentage of time spent between 80 and 90%, 70 and 79%, 60 and 69%, < 60%; lowest saturation level; mean saturation for abnormal respiratory patterns.

6. Cardiac rate and rhythm should be described, and the relationship of any abnormalities to other cardiopulmonary events noted.

7. Technician's comments.

8. Interpretation.

Difficulties are encountered in comparing records because different laboratories use different definitions of what constitutes a respiratory event. In particular, there are differences in the criteria used to define a hypopnea. The field would be advanced if standard definitions of indices used to quantify abnormality were developed. It is recommended that a subcommittee of the American Thoracic Society be impaneled to address this issue. This group should work with other professional organizations that are involved in cardiopulmonary sleep disorders.

The lack of standardization also hampers development of computerized systems for automated analysis of polysomnographic data. Currently, there are no such systems that can be recommended for clinical purposes. However, development of such systems should be encouraged. Before any such system can be recommended, there is a need for adequate validity data to prove the performance of the system. Particular problems with automation will be in automatic scoring of sleep stages because the concepts developed by Rechtschaffen and Kales (125) may not be applicable to sleep apnea patients in whom sleep is extremely disrupted.

There are also problems at present in defining what is normal. Although the original definition of sleep apnea (greater than 5 apneas/hour) has helped advance this field, it is now outmoded. New definitions will need to be developed in the light of new information derived from population studies. There is an urgent need for epidemiologic studies to address issues related to sleep apnea syndromes.

#### Screening and Ambulatory Monitoring Techniques

**Background.** Given the expense and time commitment involved in performing polysomnography, there have been approaches to simplify the diagnostic procedure. These approaches have taken two directions. First, simplified techniques have been suggested that could serve to screen individuals, thereby reducing the number of subjects that need polysomnography. Second, equipment is be-

ing developed to allow performance of a study equivalent to polysomnography in the home setting. This may also be used for screening purpose.

For screening purposes, a number of simplified approaches have been suggested. They include use of tracheal sound recordings (89–91), for which automated computer techniques can be used to detect apneas and hypopneas (91). A computer has also been employed with a CO<sub>2</sub> detector to monitor for apnea (149). Analysis of oximetry data alone can be used to detect apneas and hypopneas associated with desaturations and has some utility in monitoring sleep states (150). However, the predictive value with respect to apneas is relatively low (0.56). Screening studies that combine continuous measurement of arterial oxygen saturation and ventilatory effort may be adequate for the presumptive diagnosis of sleep apnea if cyclic breathing and saturation abnormalities are detected and then abolished by the application of nasal CPAP. However, the absence of abnormalities in such screening tests does not prove that the patient is free of sleep apnea. Finally, the static charge-sensitive bed has also been used as a screening technique for sleep apnea (151, 152). After filtering, three separate signals are obtained that relate to movements of the heart (ballistocardiography), respiratory movements, and other movements. When combined with oximetry, this technique may have some utility in detecting apneas, although validation of this device is at the preliminary stage (152).

The question of screening tests for sleep apnea raises economic as well as scientific issues. For a screening test to be valuable economically, the number of definitive tests not done as a result of the screen should represent a savings in money or time as compared to the cost of the screen. To date, there is no information on the relative costs of these two approaches and, therefore, no data on the potential savings or increased costs associated with screening tests.

In addition to screening tests, several approaches to ambulatory monitoring have been developed recently, using both digital and analog recording techniques. Such devices are being continuously upgraded, as is the software for detection of respiratory events. The devices employ inductance plethysmography with calibrated signals from the chest and abdomen to detect apneas and hypopneas (153, 154). Obstruction is detected by examining the phase relationship between rib cage and abdomen, i.e., to determine when paradoxical respiration is present (154). Simultaneous with the respiratory measurements, recordings are made of other important variables, e.g., ear oximetry, heart rate determined by the R-R interval of the electrocardiogram, tibialis EMG, and body movement with an activity monitor strapped to the subject's wrist. The latter is used to distinguish sleep from wakefulness (155).

At the present stage of development, the number of apneas detected by these instruments shows a good correlation with those

detected by simultaneous polysomnography (153, 154, 156, 157). Thus, in general the instruments correctly identify persons who have sleep apnea. However, the devices are not as proficient in classifying apneas as being obstructive or central. In particular, with current digital systems, central apneas are frequently classified as obstructive (154, 157). Thus, the positive predictive rate for obstructive apneas is 62%, but only 11% for central apneas (154). Improvements in the system to obviate this problem are evidently in hand.

**Consensus statement.** The role of screening tests in clinical practice for the diagnosis of sleep apnea is uncertain. Before any approach can be recommended for widespread use, further validity data are required. Data are currently available for ambulatory monitoring systems that are based on use of respiratory inductance plethysmography. While these instruments have reasonable predictive value in detecting apneas, there are problems in distinguishing central and obstructive events. It is anticipated that there will be further improvements in technology, which are encouraged, and with such improvements, the precise role of ambulatory instrumentation in clinical practice will become increasingly apparent. Such instrumentation will greatly facilitate the epidemiologic studies that are required.

#### References

1. Trask CH, Cree EM. Oximeter studies on patients with chronic obstructive emphysema, awake and during sleep. *N Engl J Med* 1962; 266:639–42.
2. Pierce AK, Jarrett CE, Werkle G Jr, Miller WF. Respiratory function during sleep in patients with chronic obstructive lung disease. *J Clin Invest* 1966; 45:631–6.
3. Koo KW, Sax DS, Snider GL. Arterial blood gases and pH during sleep in chronic obstructive pulmonary disease. *Am J Med* 1975; 58:663–70.
4. Leitch AG, Clancy LJ, Leggett RJE, et al. Arterial blood gas tensions, hydrogen ion, and electroencephalogram during sleep in patients with chronic ventilatory failure. *Thorax* 1976; 31:730–5.
5. Flick MR, Block AJ. Continuous *in vivo* monitoring of arterial oxygenation in chronic obstructive lung disease. *Ann Intern Med* 1977; 86:725–30.
6. Coccagna G, Lugaresi E. Arterial blood gases and pulmonary and systemic arterial pressure during sleep in chronic obstructive pulmonary disease. *Sleep* 1978; 1:117–24.
7. Wynne JW, Block AJ, Hemenway J, et al. Disordered breathing and oxygen desaturation during sleep in patients with chronic obstructive lung disease. *Am J Med* 1979; 66:573–9.
8. Douglas NJ, Calverley PMA, Leggett RJE, et al. Transient hypoxaemia during sleep in chronic bronchitis and emphysema. *Lancet* 1979; 1:1–4.
9. Fletcher EC, Gray BA, Levin DC. Nonapneic mechanisms of arterial oxygen desaturation during rapid-eye-movement sleep. *J Appl Physiol* 1983; 54:632–9.
10. Goldstein RS, Ramcharan V, Bowes G, et al. Effect of supplemental nocturnal oxygen on gas exchange in patients with severe obstructive lung disease. *N Engl J Med* 1984; 310:425–9.
11. Littner MR, McGinty DJ, Arand DL. Determinants of oxygen desaturation in the course of ventilation during sleep in chronic obstructive pul-

- monary disease. *Am Rev Respir Dis* 1980; 122: 849-57.
12. Hudgel DW, Martin RJ, Copehart M, *et al*. Contribution of hypoventilation to sleep oxygen desaturation in chronic obstructive pulmonary disease. *J Appl Physiol* 1983; 55:669-77.
  13. Fletcher EC, Levin DC. Cardiopulmonary hemodynamics during sleep in subjects with chronic obstructive pulmonary disease: the effect of short and long term oxygen. *Chest* 1984; 85:6-14.
  14. Boysen PG, Block AJ, Wynne JW, *et al*. Nocturnal pulmonary hypertension in patients with chronic obstructive pulmonary disease. *Chest* 1979; 76:536-42.
  15. Flenley DC. Clinical hypoxia: causes, consequences, and correction. *Lancet* 1978; 1:542-6.
  16. Block AJ, Boysen PG, Wynne JW. The origins of cor pulmonale: a hypothesis. *Chest* 1979; 75:109-10.
  17. Demarco FJ Jr, Wynne JW, Block AJ, *et al*. Oxygen desaturation during sleep as a determinant of the "blue and bloated" syndrome. *Chest* 1981; 79:621-5.
  18. Nocturnal Oxygen Therapy Trial Group. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease: a clinical trial. *Ann Intern Med* 1980; 93:391-8.
  19. Medical Research Council Working Party. Long term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. *Lancet* 1981; 1:681-6.
  20. Timms RM, Khaja FU, Williams GW, *et al*. Hemodynamic response to oxygen therapy in chronic obstructive pulmonary disease. *Ann Intern Med* 1985; 102:29-36.
  21. Fletcher EC, Miller J, Divine GW, *et al*. Nocturnal oxyhemoglobin desaturation in COPD patients with arterial oxygen tensions above 60 torr. *Chest* 1987; 92:604-8.
  22. Stradling JR, Lane DJ. Nocturnal hypoxemia in chronic obstructive pulmonary disease. *Clin Sci* 1983; 64:213-22.
  23. Flick MR, Block AJ. Nocturnal vs. diurnal cardiac arrhythmias in patients with chronic obstructive pulmonary disease. *Chest* 1979; 75:8-11.
  24. Tirlapur VG, Mir MA. Nocturnal hypoxemia and associated electrocardiographic changes in patients with chronic obstructive airways disease. *N Engl J Med* 1982; 306:125-30.
  25. Levi-Valensi P, Weitzenblum E, Pedinielli JL, *et al*. Three-month follow-up of arterial blood gas determinations in candidates for long-term oxygen therapy. *Am Rev Respir Dis* 1986; 133:547-51.
  26. Guilleminault C, Kurlund G, Winkle R, Miles LE. Severe kyphoscoliosis, breathing and sleep: the "Quasimodo" syndrome during sleep. *Chest* 1981; 79:626-30.
  27. Mezon BL, West P, Israels J, Kryger M. Sleep breathing abnormalities in kyphoscoliosis. *Am Rev Respir Dis* 1980; 122:617-22.
  28. Newsom Davis J, Goldman M, Loh L, Casson M. Diaphragm function and alveolar hypoventilation. *Q J Med* 1976; 45:87-100.
  29. Goldstein RS, Molotiu N, Skrastins R, *et al*. Reversal of sleep-induced hypoventilation and chronic respiratory failure by nocturnal negative pressure ventilation in patients with restrictive ventilatory impairment. *Am Rev Respir Dis* 1987; 135:1049-55.
  30. Phillipson EA, Bowes G. Control of breathing during sleep. In: Cherniack NS, Widdicombe JG, eds. *Handbook of physiology*. Section 3: the respiratory system, Vol. 2, Control of breathing. Bethesda: American Physiological Society, 1986; 649-89.
  31. Kryger MH. Sleep in restrictive lung disorders. *Clin Chest Med* 1985; 6:675-7.
  32. Hoepfner VH, Cockcroft DW, Dosman JA, Cotton DJ. Nighttime ventilation improves respiratory failure in secondary kyphoscoliosis. *Am Rev Respir Dis* 1984; 129:240-3.
  33. Bye PTP, Issa F, Berthoin-Jones M, Sullivan CE. Studies of oxygenation during sleep in patients with interstitial lung disease. *Am Rev Respir Dis* 1984; 129:27-32.
  34. Perez-Padilla R, West P, Lertzman M, Kryger MH. Breathing during sleep in patients with interstitial lung disease. *Am Rev Respir Dis* 1985; 132:224-9.
  35. Plum F. Neurological integration of behavioral and metabolic control of breathing. In: Porter R, ed. *Breathing: Hering-Breuer centenary symposium*. London: Churchill, 1970; 159-75.
  36. Berger AJ, Mitchell RA, Severinghaus JW. Regulation of respiration. *N Engl J Med* 1977; 287:92-7, 138-43.
  37. Rhoads GG, Brody JS. Idiopathic alveolar hypoventilation: clinical spectrum. *Ann Intern Med* 1969; 71:271-8.
  38. Mellins RB, Balfour HH Jr, Turino GM, Winters RW. Failure of automatic control of ventilation (Ondine's Curse). *Medicine* 1970; 49:487-504.
  39. Plum F, Swanson AG. Abnormalities in central regulation of respiration in acute and convalescent poliomyelitis. *Arch Neurol Psychol* 1958; 80:267-85.
  40. Cohn JE, Kuida H. Primary alveolar hypoventilation associated with western equine encephalitis. *Ann Intern Med* 1962; 56:633-44.
  41. Devereaux MW, Keane JR, Davis RL. Automatic respiratory failure associated with infarction of the medulla. *Arch Neurol* 1973; 29:46-52.
  42. Reichel J. Primary alveolar hypoventilation. *Clin Chest Med* 1980; 1:119-24.
  43. Rodman T, Close HP. The primary hypoventilation syndrome. *Am J Med* 1959; 26:808-17.
  44. Bradley TD, McNicholas WT, Rutherford R, *et al*. Clinical and physiologic heterogeneity of the central sleep apnea syndrome. *Am Rev Respir Dis* 1986; 134:217-21.
  45. Phillipson EA. Hypoventilation syndromes. In: Murray JF, Nadel JA, eds. *Textbook of respiratory medicine*. Philadelphia: WB Saunders, 1988; 1831-40.
  46. Sharp J, Barrocas M, Chokroverty S. The cardiorespiratory effects of obesity. *Clin Chest Med* 1980; 1:103-18.
  47. Bergofsky EH. Respiratory failure in disorders of the thoracic cage. *Am Rev Respir Dis* 1979; 119:643-69.
  48. Gelb AF, Klein E, Schiffman P, *et al*. Ventilatory response and drive in acute and chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1977; 116:9-16.
  49. Naughton J, Block R, Welch M. Central alveolar hypoventilation. *Am Rev Respir Dis* 1971; 103:557-65.
  50. Farmer WC, Glenn WW, Gee JB. Alveolar hypoventilation syndrome. Studies of ventilatory control in patients selected for diaphragm pacing. *Am J Med* 1978; 64:39-49.
  51. Bubis MJ, Anthonisen NR. Primary alveolar hypoventilation treated by nocturnal administration of O<sub>2</sub>. *Am Rev Respir Dis* 1978; 118:947-53.
  52. Barlow PB, Bartlett D Jr, Hauri P, *et al*. Idiopathic hypoventilation syndrome: importance of preventing nocturnal hypoxemia and hypercapnia. *Am Rev Respir Dis* 1980; 121:141-5.
  53. McNicholas WT, Carter JL, Rutherford R, *et al*. Beneficial effect of oxygen in primary alveolar hypoventilation with central sleep apnea. *Am Rev Respir Dis* 1982; 125:773-5.
  54. Glenn WWL, Gee JBL, Cole DR, *et al*. Combined central alveolar hypoventilation and upper airway obstruction. *Am J Med* 1978; 64:50-60.
  55. Hyland RH, Hutcheon MA, Perl A, *et al*. Up- per airway occlusion induced by diaphragm pacing for primary alveolar hypoventilation: implications for the pathogenesis of obstructive sleep apnea. *Am Rev Respir Dis* 1981; 124:180-5.
  56. Ellis ER, Bye PTP, Bruderer JW, Sullivan CE. Treatment of respiratory failure during sleep in patients with neuromuscular disease: positive pressure ventilation through a nose mask. *Am Rev Respir Dis* 1987; 135:148-52.
  57. Lugaresi E, Cirignotta F, Coccagna G, *et al*. Some epidemiological data on snoring and cardiorespiratory disturbances. *Sleep* 1980; 3:221-4.
  58. Peiser J, Lavie P, Ovnat A, Charuzi I. Sleep apnea syndrome in the morbidly obese as an indication for weight reduction surgery. *Ann Surg* 1984; 199:112-5.
  59. Harman EM, Wynne JW, Block AJ. The effect of weight loss on sleep-disordered breathing and oxygen desaturation in morbidly obese men. *Chest* 1982; 82:192-4.
  60. Smith PL, Gold AR, Meyers DA, *et al*. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. *Ann Intern Med* 1985; 103:850-5.
  61. Guilleminault C. Disorders of excessive sleepiness. *Ann Clin Res* 1985; 17:190-8.
  62. Franceschi M, Samproni P, Crippa D, Smirne S. Excessive daytime sleepiness. A one year study in an unselected inpatient population. *Sleep* 1982; 5:239-47.
  63. Coleman RM, Roffwarg HP, Kennedy SJ, *et al*. Sleep-wake disorders based on polysomnographic diagnosis. *JAMA* 1982; 247:997-1003.
  64. Roth T, Hartse KM, Zorick F, Conway W. Multiple naps and the evaluation of daytime sleepiness in patients with upper airway sleep apnea. *Sleep* 1980; 3:425-9.
  65. Bliwise DL, Carey E, Dement WC. Nightly variation in sleep-related respiratory disturbances in older adults. *Exp Aging Res* 1983; 9:77-82.
  66. Coleman RM, Miles LE, Guilleminault C, *et al*. Sleep-wake disorders in the elderly: a polysomnographic analysis. *J Am Geriatr Soc* 1981; 29: 289-96.
  67. Lavie P, Ben-Yosef R, Rubin AE. Prevalence of sleep apnea syndrome among patients with essential hypertension. *Am Heart J* 1984; 108:373-6.
  68. Kales A, Bixler EO, Cadieux RJ, *et al*. Sleep apnea in a hypertensive population. *Lancet* 1984; 2:1005-8.
  69. Fletcher EC, DeBehnke RD, Lovoi MS, *et al*. Undiagnosed sleep apnea in patients with essential hypertension. *Ann Intern Med* 1985; 102:190-5.
  70. Williams AJ, Houston D, Finberg S, *et al*. Sleep apnea syndrome and essential hypertension. *Am J Cardiol* 1985; 55:1019-22.
  71. Zwillich C, Devlin T, White D, *et al*. Bradycardia during sleep apnea. *J Clin Invest* 1982; 69:1286-92.
  72. Guilleminault C, Connolly SJ, Winkle RA. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. *Am J Cardiol* 1983; 52:490-4.
  73. Guilleminault C, Connolly S, Winkle R, *et al*. Cyclical variation of the heart rate in sleep apnoea syndrome. *Lancet* 1984; 1:125-31.
  74. Shepard JW Jr, Garrison MW, Grither DA, Dolan GF. Relationship of ventricular ectopy to oxyhemoglobin desaturation in patients with obstructive sleep apnea. *Chest* 1985; 88:335-40.
  75. Silvestri R, Guilleminault C, Coleman R, *et al*. Nocturnal sleep versus daytime nap findings in patients with breathing abnormalities during sleep (abstract). *Sleep Res* 1982; 11:174.
  76. Roberts CJ, Hooper RG. Prediction of polysomnography results by abbreviated testing (abstract). *Chest* 1985; 88:4435.
  77. Agnew HW, Webb WB, Williams RL. The first

- night effect: an EEG study of sleep. *Psychophysiol* 1966; 2:263-6.
78. Wittig RM, Romaker A, Zorick FJ, *et al*. Night-to-night consistency of apneas during sleep. *Am Rev Respir Dis* 1984; 129:244-6.
79. O'Brien CF, Mahowald MW, Schleuter J, Iber C. Continuous positive airway pressure in obstructive sleep apnea: initial response and long-term follow-up (abstract). *Am Rev Respir Dis* 1986; 133(Suppl:A342).
80. Sullivan CE, Issa FG, Berthoin-Jones M, *et al*. Home treatment of obstructive sleep apnea with continuous positive airway pressure through a nose-mask. *Bull Eur Physiopathol Respir* 1984; 20:49-54.
81. Bornsten SK. Respiratory monitoring during sleep: polysomnography. In: Guilleminault C, ed. *Sleeping and waking disorders: indications and techniques*. Boston: Butterworth, 1982; 183-212.
82. Martin RJ, Block AJ, Cohn MA, *et al*. Indications and standards for cardiopulmonary sleep studies. *Sleep* 1985; 8:371-9.
83. Coleman RM. Periodic movements in sleep (nocturnal myoclonus) and restless legs syndrome. In: Guilleminault C, ed. *Sleeping and waking disorders: indications and techniques*. Boston: Butterworth, 1982; 265-95.
84. Cartwright RD. Effect of sleep position on sleep apnea severity. *Sleep* 1984; 7:110-4.
85. Findley LJ, Wilhoit SC, Suratt PM. Apnea duration and hypoxemia during REM sleep in patients with obstructive sleep apnea. *Chest* 1985; 87:432-6.
86. Jasper HH. The ten twenty electrode system of the International Federation. *Electroencephalogr Clin Neurophysiol* 1985; 10:371-5.
87. Carskadon MA. Basis for polygraphic monitoring of sleep. In: Guilleminault C, ed. *Sleeping and waking disorders: indications and techniques*. Boston: Butterworth, 1982; 1-16.
88. Fisher JG, Garza G, Flickinger R, de la Pena A. An alternate method of recording airflow during sleep. *Sleep* 1980; 21:461-3.
89. Krumpke PE, Cummiskey JM. Use of laryngeal sound recordings to monitor apnea. *Am Rev Respir Dis* 1980; 122:797-801.
90. Cummiskey JM, Williams TC, Krumpke PE, Guilleminault C. The detection and quantification of sleep apnea by tracheal sound recordings. *Am Rev Respir Dis* 1982; 126:221-4.
91. Peirick J, Shepard JW Jr. Automated apnea detection by computer: analysis of tracheal breath sounds. *Med Biol Eng Comput* 1983; 21:632-5.
92. Baker LE, Geddes LA. The measurement of respiratory volumes in animals and man with use of electrical impedance. *Ann NY Acad Sci* 1970; 170:667-88.
93. Larsen VH, Christensen P-H, Oxhojand H, Brask T. Impedance pneumography for long-term monitoring of respiration during sleep in adult males. *Clin Physiol* 1984; 4:333-42.
94. Krieger J, Kurtz D. Effects of pneumotachographic recording of breathing on sleep and respiration during sleep. *Bull Eur Physiopathol Respir* 1983; 19:641-4.
95. Mead J, Peterson N, Grimby G, Mead J. Pulmonary ventilation measured from body surface movements. *Science* 1967; 156:1383-4.
96. Sharp JT, Druz WS, Foster JR, *et al*. Use of the respiratory magnetometer in diagnosis and classification of sleep apnea. *Chest* 1980; 77:350-3.
97. Sackner MA. Monitoring of ventilation without a physical connection to the airway. In: Sackner MA, ed. *Diagnostic techniques in pulmonary disease*. New York: Marcel Dekker, 1980; 503-37.
98. Cohn MA, Roa ASV, Broudy M, *et al*. The respiratory inductive plethysmograph: a new noninvasive monitor of respiration. *Bull Eur Physiopathol Respir* 1982; 18:643-58.
99. Konno K, Mead J. Measurement of the separate volume changes of rib cage and abdomen during breathing. *J Appl Physiol* 1967; 22:407-22.
100. Chadha TS, Watson H, Birch S, *et al*. Validation of respiratory inductance plethysmography using different calibration procedures. *Am Rev Respir Dis* 1982; 125:644-9.
101. Stradling JR, Chadwick GA, Quirk C, Phillips T. Respiratory inductance plethysmography: calibration techniques, their validation and the effects of posture. *Bull Eur Physiopathol Respir* 1985; 21:317-24.
102. Guggen M, Gould GA, Whyte KF, *et al*. Inductive plethysmographs do not accurately measure ventilation during sleep in unrestrained subjects (abstract). *Am Rev Respir Dis* 1987; 135(Suppl:A50).
103. Zimmerman PV, Connellan SJ, Middleton HC, *et al*. Postural changes in rib cage and abdominal motion coefficients and their effect on the calibration of a respiratory inductance plethysmograph. *Am Rev Respir Dis* 1983; 127:209-14.
104. Spier S, England S. The respiratory inductive plethysmograph: bands versus jerkins. *Am Rev Respir Dis* 1983; 127:784-5.
105. Miles LE, Herekar BV, Rule RB. An improved sensor for recording respiration by inductive plethysmography (abstract). *Sleep Res* 1986; 15:249.
106. Sackner MA, Belsito AS, Nay N, *et al*. Apnea diagnosis with respisomnograph (abstract). *Sleep Res* 1987; 16:576.
107. Shapiro A, Cohen HD. The use of mercury capillary length groups for the measurement of the volume of thoracic and diaphragmatic components of human respiration: a theoretical analysis and practical method. *Trans NY Acad Sci* 1965; 27:634-49.
108. Guilleminault C, Dement WC. Polysomnography. In: Sackner MA, ed. *Diagnostic techniques in pulmonary disease*. New York: Marcel Dekker, 1981; 849-60.
109. Onal E, Lopata M, O'Connor T. Pathogenesis of apneas in hypersomnia-sleep apnea syndrome. *Am Rev Respir Dis* 1982; 125:167-74.
110. Sampson MG, Walsleben JA, Gujavarty KS, *et al*. Effect of esophageal balloon on sleep structure (abstract). *Sleep Res* 1984; 13:211.
111. Staats BA, Bonekat HW, Harris CD, Offord KP. Chest wall motion in sleep apnea. *Am Rev Respir Dis* 1984; 130:59-63.
112. Suarez M, Bizousky F, Befeler A, Sackner MA. Performance of mercury in silastic strain gauges and respiratory inductive plethysmograph as assessed with spirometry (abstract). *Am Rev Respir Dis* 1987; 135(Suppl:A49).
113. Saunders NA, Powles ACP, Rebeck AS. Ear oximetry: accuracy and practicability in the assessment of arterial oxygenation. *Am Rev Respir Dis* 1976; 113:745-9.
114. Chaudhary BA, Burki NK. Ear oximetry in clinical practice. *Am Rev Respir Dis* 1978; 117:173-5.
115. Douglas NJ, Brash HM, Wraith PK, *et al*. Accuracy, sensitivity to carboxyhemoglobin, and speed of response of the Hewlett-Packard 47201A ear oximeter. *Am Rev Respir Dis* 1979; 119:311-3.
116. Rebeck AS, Chapman KR, D'Urzo A. The accuracy and response characteristics of a simplified ear oximeter. *Chest* 1983; 83:860-4.
117. Yelderman M, New W. Evaluation of pulse oximetry. *Anesthesiol* 1983; 59:349-62.
118. Tweeddale PM, Douglas NJ. Evaluation of Biox IIA ear oximeter. *Thorax* 1985; 40:825-7.
119. Ries AL, Farrow JT, Clausen JL. Accuracy of two ear oximeters at rest and during exercise in pulmonary patients. *Am Rev Respir Dis* 1985; 132:685-9.
120. Mackenzie N. Comparison of a pulse oximeter with an ear oximeter and *in vitro* oximeter. *J Clin Monitor* 1985; 1:156-60.
121. Chapman KR, Liu FLW, Watson RM, Rebeck AS. Range of accuracy of two wavelength oximetry. *Chest* 1986; 89:540-2.
122. West P, George CF, Kryger MH. Dynamic *in vivo* response characteristics of three oximeters: Hewlett-Packard 47201A, Biox III and Nellcor N-100. *Sleep* 1987; 10:263-71.
123. Naifeh KH, Severinghaus JW. How accurate are pulse oximeters to profound brief hypoxia? (abstract). *Sleep Res* 1987; 16:1569.
124. Gardner RM. Snowbird workshop on standardization of spirometry. *Am Rev Respir Dis* 1979; 119:831-8.
125. Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring systems for sleep stages of human subjects. Washington, DC: National Institute of Health, 1968; Publ. No. 204.
126. Schmidt-Nowara WW, Sano J, Appel D. Stage T: a scoring modification for breathing disturbed sleep (abstract). *Sleep Res* 1983; 12:356.
127. Pack AI, Cola M, Goldszmidt A, *et al*. Coherence between periodic oscillations in ventilation and frequency content of the electroencephalogram (abstract). *Am Rev Respir Dis* 1987; 135(Suppl:A371).
128. Campbell PI, Reynolds CF, Berman SB, *et al*. Assessment of sleep fragmentation in the elderly: a reliability study (abstract). *Sleep Res* 1987; 16:547.
129. Bliwise D, Bliwise NC, Kramer HC, Dement W. Measurement error in visually scored electrophysiological data: respiration during sleep. *J Neurosci Methods* 1984; 12:49-56.
130. Guilleminault C, van den Hoed J, Mitler MM. Clinical overview of the sleep apnea syndromes. In: Guilleminault C, Dement WC, ed. *Sleep apnea syndromes*. New York: Alan R. Liss, 1978; 1-12.
131. Catterall JR, Calverley PMA, Shapiro CM, *et al*. Breathing and oxygenation during sleep are similar in normal men and normal women. *Am Rev Respir Dis* 1985; 132:86-8.
132. Bradley TD, Brown IG, Zamel N, *et al*. Differences in pharyngeal properties between snorers with predominantly central sleep apnea and those without sleep apnea. *Am Rev Respir Dis* 1987; 135:387-91.
133. Block AJ, Boysen PG, Wynne JW, Hunt LA. Sleep apnea, hypopnea and oxygen desaturation in normal subjects. A strong male predominance. *N Engl J Med* 1979; 300:513-7.
134. West P, Kryger MH. Continuous monitoring of respiratory variables during sleep by microcomputer. *Methods Inf Med* 1983; 22:198-203.
135. Steyer BJ, Quan SF, Morgan WJ. Polysomnography scoring for sleep apnea: use of a sampling method. *Am Rev Respir Dis* 1985; 131:592-5.
136. Guilleminault C, Cummiskey J, Dement WC. Sleep apnea syndrome: recent advances. *Adv Intern Med* 1980; 26:347-72.
137. Slutsky AS, Strohl KP. Quantification of oxygen saturation during episodic hypoxemia. *Am Rev Respir Dis* 1980; 121:893-5.
138. Evans RJ, Wilhoit SC, Suratt PM. Computer analysis of oxyhemoglobin saturation during sleep (abstract). *Sleep Res* 1984; 13:202.
139. Timms RM, Dawson A, Taft R, Mitler MM. A technique for microcomputer analysis of bedside ear oximetry (abstract). *Sleep Res* 1986; 15:259.
140. Ware JC, Brown FW, Bellamy M, Bell L. Use of a microcomputer system for assistance in scoring and summarizing polysomnographic data (abstract). *Sleep Res* 1981; 10:287.
141. Broughton R, Barker D, Roberts J. A computer generated sleep histogram (abstract). *Sleep Res* 1984; 13:197.
142. Smith JR. Computers in sleep research. *CRC*

Crit Rev Bioeng 1978; 3:93-148.

143. Hasan J. Differentiation of normal and disturbed sleep by automatic analysis. *Acta Physiol Scand [Suppl]* 1983; 526:3-103.
144. Nino-Murcia G, Greenleaf W, Keenan S, Hook J. A real time sleep apnea detector and respiration scoring system (abstract). *Sleep Res* 1985; 14:275.
145. Yu FS, Principe JC, Smith JR. Automated respiration monitoring (abstract). *Sleep Res* 1986; 15:263.
146. Berry DTR, Webb WB, Block AJ. Sleep apnea syndrome. A critical review of the apnea index as a diagnostic criterion. *Chest* 1984; 86:529-31.
147. Carskadon MA, Dement WC. Respiration during sleep in the aged human. *J Gerontol* 1981; 36:420-3.
148. Ancoli-Israel S, Kripke DF, Mason W, Kaplan OJ. Sleep apnea and periodic movements in an aging sample. *J Gerontol* 1985; 4:419-25.
149. Schmidt-Nowara WW. The utility of a CO<sub>2</sub> home monitor respiromnograph in the diagnosis of sleep apnea syndrome (abstract). *Sleep Res* 1985; 14:279.
150. Farney RJ, Walker LE, Jensen RL, Walker JM. Ear oximetry to detect apnea and differentiate rapid eye movement (REM) and non-REM (NREM) sleep. *Chest* 1986; 89:533-9.
151. Salmi T, Leinonen L. Automatic analysis of sleep records with static charge sensitive bed. *Electroencephal Clin Neurophysiol* 1986; 64:84-7.

152. Svanborg E, Larsson H, Nordlander B, *et al.* Screening of obstructive sleep apnea with respiration movement and SaO<sub>2</sub> monitoring: high diagnostic accuracy in comparison with polygraphic recordings (abstract). *Sleep Res* 1987; 16:587.
153. Ancoli-Israel S, Kripke DF, Mason W, Messin S. Comparison of home sleep recordings and polysomnograms in older adults with sleep disorders. *Sleep* 1981; 4:283-91.
154. Gyulay S, Gould D, Sawyer B, *et al.* Evaluation of a microprocessor-based portable home monitoring system to measure breathing during sleep. *Sleep* 1987; 10:130-42.
155. Webster JB, Kripke DF, Messin S, *et al.* An activity-based sleep monitor system for ambulatory use. *Sleep* 1982; 5:389-99.
156. Nino-Murcia G, Bliwise D, Kennan S, *et al.* Respiration monitoring in sleep: comparison of judgements based on conventional polysomnography (PSG) and an ambulatory microprocessor derived recording (AMD) (abstract). *Sleep Res* 1985; 14:274.
157. Walker LE, Walker JM, Farney RJ, Kramer J. A comparison of polysomnography with a portable home monitoring system in the detection of sleep apnea (abstract). *Am Rev Respir Dis* 1986; 133(Suppl:A54).

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