Sleep–Related Breathing Disorders in Adults: Recommendations for Syndrome Definition and Measurement Techniques in Clinical Research

The Report of an American Academy of Sleep Medicine Task Force

1.0 INTRODUCTION

OBSTRUCTIVE SLEEP APNEA is a condition characterized by repetitive obstruction of the upper airway often resulting in oxygen desaturation and arousals from sleep. The classic daytime manifestation is excessive sleepiness but other symptoms such as unrefreshing sleep, poor concentration and fatigue are commonly reported. Over the past thirty years many types of abnormal breathing during sleep have been described that are related to, but not accurately described as apneas. Partial airway obstruction can lead to a reduction in tidal volume, referred to as a hypopnea, with the same consequences as an apnea.[1] Even more subtle abnormalities have been described such as progressive increases in respiratory effort, reflecting increasing upper airway resistance, that terminate after an arousal.[2] Some patients have periods of hypoventilation during sleep, most commonly seen in REM, and not always associated with apneic events. These patients are often obese, usually have awake hypercapnia, and signs of cor pulmonale.[3] Still another type of breathing abnormality consists of those apneic events that are not associated with inspiratory effort, indicating reduced central respiratory drive, referred to as central apneas.[4] Central apneas can occur in otherwise healthy individuals but they are also a feature of Cheyne-Stokes breathing, which is commonly seen in patients with congestive heart failure. Mixed apneas refer to periods of absent airflow that are initially associated with an absence of respiratory effort and that persist upon resumption of respiratory effort indicating upper airway obstruction.

As different types of disordered breathing events during sleep have been described, it has been recognized that signs and symptoms could be used to describe several syndromes. Burwell used the term Pickwickian syndrome to describe patients with obesity, hypercapnia, cor pulmonale, erythrocytosis, and daytime hypersomnolence.[3] Guilleminault introduced the term *obstructive sleep apnea syndrome* (OSAS) with its central feature of daytime hypersomnolence and polysomnographically proven obstructive apneas.[5] *Hypopneas* were first described by Block, et al. as events of shallow breathing causing oxygen desaturation.[6] In 1988, cases with hypopneas and no or few apneas were described, with clinical symptoms similar to OSAS, and introduced the sleep hypopnea syndrome.[1] Subsequently the OSAS began to be referred to as the *obstructive sleep apnea-hypopnea syndrome* (OSAHS). In 1992 Guilleminault described a series of patients that had typical symptoms of obstructive sleep apnea but who did not have obstructive apneas or hypopneas on polysomnography. It was suggested that these events, characterized by increasing negative esophageal pressure during inspiration and terminating with an arousal, reflected an *upper airways resistance syndrome* (UARS).[2] The term *central sleep apnea syndrome* (CSAS) has also been discussed in the literature but it has never been established whether patients with Cheyne-Stokes breathing or those with high altitude periodic breathing should be included under this rubric.

The initial description of OSAS by Guilleminault included a criterion of a minimum duration of 10 seconds for an apnea to be scored. Based on a study of healthy subjects it was also suggested that more than 30 apneas per night should be considered abnormal.[5] This was later standardized as the apnea index, which reflects the number of apneas per hour of sleep. The apnea index cutoff for OSAS was set at 5.[7] Since the initial descriptions of these different types of abnormal breathing events and their related syndromes, technology has changed and original definitions have been modified to incorporate methods with uncertain validity and reliability. This has led to variable definitions of events and syndromes that are based on differing methodologies. The lack of uniform definitions as well as the clinical overlap between the Pickwickian syndrome, OSAHS, and CSAS has created confusion in clinical settings and has hindered comparisons of results from research studies. This publication addresses this issue by proposing a set of standard criteria for defining apnea events and syndromes.

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2.0 BACKGROUND

In 1995 the American Academy of Sleep Medicine (AASM) and the American Thoracic Society co-sponsored a conference to develop recommendations on important outcome measures for studies investigating the effects of sleep apnea.[8] It was difficult to compare results of different studies because of the lack of standard definitions of abnormal breathing events during sleep. Subsequently the AASM Board of Directors established a task force that was charged to recommend standard definitions, criteria and severity ratings for abnormal breathing events during sleep and their associated clinical syndromes. **The purpose of these definitions was to facilitate comparability of studies for research purposes. The task force's report is not intended to provide guidelines for routine clinical care, nor are these recommendations offered as an alternative to current patient care practices, although clinicians are encouraged to consider the issues raised by this report.** The limitations of methodologic research currently available should encourage more research in this area and also a rethinking of methods of clinical practice.

Participants of the task force were selected to represent a spectrum of clinical and research expertise. Support was obtained from the European Respiratory Society and the Australasian Sleep Association to sponsor representatives. Two organizing meetings were held to discuss specific goals of the task force and to discuss the methodology to be used by the group to work towards a consensus for these recommendations. Four working groups were established to work on specific syndromes: OSAHS, CSAS, UARS, and the sleep hypoventilation syndrome (SHVS), a broader term that encompassed the Pickwickian Syndrome. The task force met for two days in Chicago, November, 1997 to formulate the recommendations. A report was drafted by all of the participants to be forwarded to the sponsoring organizations for feedback. All participants signed an AASM conflict of interest form identifying any potential conflicts and specifically stating any affiliations they had with companies who made sleep apnea diagnostic equipment.

3.0 METHODS

The task force addressed the following goal and objectives:

Goal:
To develop standard definitions of abnormal breathing events during sleep in adults and their associated syndromes that would facilitate more reliable and accurate reporting in research studies and in clinical practice.

Objectives:
1. Define the key features of four separate syndromes associated with abnormal breathing events during sleep previously described in the literature: the obstructive sleep apnea-hypopnea syndrome, the central sleep apnea syndrome, the upper airway resistance syndrome, and the sleep hypoventilation syndrome (including the Pickwickian syndrome).
2. Describe and define the specific events or features of sleep related breathing disorders during sleep.
3. Recommend methods of measuring the key features of these syndromes.
4. Develop a standard system for rating the severity of these syndromes.
5. Distribute the task force's report to professional organizations for feedback regarding the recommendations.
6. Publish the recommendations to encourage the adoption of standard definitions by the research and clinical communities.

The task force decided to restrict its work to adult syndromes in the belief that different criteria would be required for children and that their development was beyond the scope of this task force.

A leader was assigned to each of four syndrome working groups: upper airway resistance, obstructive sleep apnea-hypopnea, central sleep apnea-hypopnea, and sleep hypoventilation. After review of the available literature the task force felt that there was not enough evidence to suggest that the upper airway resistance syndrome was a distinct syndrome with unique pathophysiology. Therefore, the task force defined a specific event, the respiratory effort related arousal (RERA), but elected to include this under the definition of the obstructive sleep apnea-hypopnea syndrome. [1] Further, it was felt that Cheyne-Stokes breathing and central sleep apnea were distinct enough to justify describing them as separate syndromes.

It is recognized by this task force that OSAHS, CSAS, CSBS, and SHVS are clinical syndromes influenced by multiple pathogenic mechanisms which are incompletely understood. Evidence exists suggesting that these syndromes share at least some common pathogenic mechanisms. For example, it is common clinical experience that some patients exhibit predominantly mixed apneas, while other patients exhibit obstructive apneas that seem to change to central events by alterations in body position or application of positive airway pressure; observations suggesting possible coexistence of central neural dysregulation and an inadequately defended upper airway. It may be argued from such evidence that OSAHS, CSAS, CSBS, and SHVS should be treated as a single "sleep-disordered breathing syndrome". However, it was felt that such an aggregation would be premature at this time. A central goal of the task force was to focus and stimulate research that will help to clarify pathogenic mechanisms of these syndromes, allowing more informed decisions to be made

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regarding which syndromes should be appropriately combined.

The key features and possible severity ratings of each syndrome were based on a comprehensive review of the literature, conducted by each working group (Section 4.0). The quality of evidence on which to base a recommendation of a severity rating for a syndrome was variable and in some circumstances was nonexistent. Three levels of evidence were identified to support a recommendation of how to rate syndrome severity (Table 1). The highest level of evidence was felt to be prospective cohort studies demonstrating a relationship between a certain severity of breathing disorder during sleep and mortality (level 1). Currently there are no published studies with level 1 evidence. The second level of evidence was established as prospective, cohort studies that demonstrated a significant relationship between a certain severity of breathing disorder during sleep and morbidity (level 2). In the absence of this type of evidence from the literature the task force relied on consensus opinion among members of the committee (level 3).

The task force reviewed all available literature about measurement methods for the defining event(s) of each syndrome to determine if there was a well-accepted "reference standard" method for measuring each feature or event and to assess other possible, commonly used, methods of measurement for their reported accuracy and precision. The task force then generated recommendations about the commonly used methods for measuring these defining events. The quality of research on which to base the recommendations was variable and in some circumstances there was no acceptable research data. The task force agreed on four grades of recommendations (Table 2), and generated a method for rating the evidence that was available which supported the recommendations (Table 3).

Although there are published guidelines about how to rate the level of evidence on the treatment of medical conditions,[9] similar guidelines for how to evaluate literature about syndrome severity ratings and measurement methodology do not exist. Thus the task force had to generate a unique methodology for this based on the literature that was published on various breathing disorders during sleep (Tables 1, 2, and 3).

Each working group presented their findings and recommendations to the entire task force for discussion and final decisions regarding syndrome definitions and severity ratings, breathing event definitions, and measurement methodology. A preliminary draft of the report was distributed to 20 experts in the field of sleep medicine for their comments. Following revisions to the report, the findings were presented at the annual meetings of the American Thoracic Society and the Association of Professional Sleep Societies in 1998. After further revisions based on feedback from participants at these meetings, the report was sent out for a separate review process, commissioned by the American Academy of Sleep Medicine Board of Directors.

Table 1. Levels of Evidence of Syndrome Severity Ratings

Level	Type of Evidence
1	Variable(s) on which the severity rating is based have been demonstrated to have a statistically significant relationship with mortality in a prospective cohort study that has properly controlled for important covariates.
2	Variable(s) on which the severity rating is based have been demonstrated to have a statistically significant relationship with excess morbidity in a prospective cohort study that has properly controlled for important covariates.
3	Variable(s) on which the severity rating is based were determined by consensus; no prospective evidence currently validates this recommendation. Future research is required and is expected to demonstrate a relationship between the variable and excess mortality or morbidity.

4.0 SYNDROME DEFINTIONS AND SEVERITY RATINGS

4.1 Obstructive sleep apnea-hypopnea syndrome (OSAHS)

4.1.1 Essential features

OSAHS is characterized by recurrent episodes of partial or complete upper airway obstruction during sleep. This manifests as a reduction in (hypopnea) or complete cessation (apnea) of airflow despite ongoing inspiratory efforts. The lack of adequate alveolar ventilation usually results in oxygen desaturation and in cases of prolonged events, a gradual increase in $PaCO_2$. The events are often terminated by arousals. Daytime symptoms such as excessive sleepiness are thought to be related to sleep disruption (recurrent arousals) and possibly also to recurrent hypoxemia. Patients may demonstrate a lack of respiratory effort during the initial apnea period followed by gradually increasing effort against an occluded upper airway; such an events are referred to as mixed apneas. These events are felt to be pathophysiologically related to obstructive apneas and are considered to be part of the OSAHS.

4.1.2 Diagnostic criteria

The individual must fulfill criterion A or B, plus criterion C.

A. Excessive daytime sleepiness that is not better explained by other factors;

B. Two or more of the following that are not better explained by other factors:

-choking or gasping during sleep,
-recurrent awakenings from sleep,
-unrefreshing sleep,
-daytime fatigue,
-impaired concentration; and/or

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C. Overnight monitoring demonstrates five or more obstructed breathing events per hour during sleep.[1,4] These events may include any combination of obstructive apneas/hypopneas or respiratory effort related arousals, as defined below.

4.1.2.1 Obstructive apnea/hypopnea event

An event characterized by a transient reduction in, or complete cessation of, breathing. In routine clinical practice it is not considered necessary to distinguish obstructive hypopneas from apneas because both types of events have similar pathophysiology. These events must fulfill criterion 1 or 2, plus criterion 3 of the following:

1. A clear decrease (>50%) from baseline in the amplitude of a valid measure of breathing during sleep. Baseline is defined as the mean amplitude of stable breathing and oxygenation in the two minutes preceding onset of the event (in individuals who have a stable breathing pattern during sleep) or the mean amplitude of the three largest breaths in the two minutes preceding onset of the event (in individuals without a stable breathing pattern).
2. A clear amplitude reduction of a validated measure of breathing during sleep that does not reach the above criterion but is associated with either an oxygen desaturation of >3% or an arousal.
3. The event lasts 10 seconds or longer.[2]

4.1.2.2 Respiratory effort-related arousal (RERA) event

A sequence of breaths characterized by increasing respiratory effort leading to an arousal from sleep, but which does not meet criteria for an apnea or hypopnea. These events must fulfill both of the following criteria:

1. Pattern of progressively more negative esophageal pressure, terminated by a sudden change in pressure to a less negative level and an arousal
2. The event lasts 10 seconds or longer[1]

4.1.2.3 Justification for the diagnostic criteria

The use of an event frequency of five per hour as a minimal threshold value was based on epidemiological data that suggest minimal health effects such as hypertension, sleepiness, and motor vehicle accidents,[10-12] may be observed at an apnea-hypopnea index (AHI) threshold of five. Additionally, limited data from intervention studies suggest treatment associated improvements in vitality, mood, and fatigue in subjects with AHIs between 5 and 30[13] and improvements in sleepiness and neurocognitive function in subjects with AHI levels of 5 to 15.[14,15]

4.1.3 Severity criteria

Severity of the OSAHS has two components: severity of daytime sleepiness and of overnight monitoring. A severity level should be specified for both components. The rating of severity for the syndrome should be based on the most severe component.

A. Sleepiness [Level of Evidence - 3]
1. Mild: Unwanted sleepiness or involuntary sleep episodes occur during activities that require little attention. Examples include sleepiness that is likely to occur while watching television, reading, or traveling as a passenger. Symptoms produce only minor impairment of social or occupational function.
2. Moderate: Unwanted sleepiness or involuntary sleep episodes occur during activities that require some attention. Examples include uncontrollable sleepiness that is likely to occur while attending activities such as concerts, meetings, or presentations. Symptoms produce moderate impairment of social or occupational function.
3. Severe: Unwanted sleepiness or involuntary sleep episodes occur during activities that require more active attention. Examples include uncontrollable sleepiness while eating, during conversation, walking, or driving. Symptoms produce marked impairment in social or occupational function.

B. Sleep Related Obstructive Breathing Events [Level of Evidence - 2]
1. Mild: 5 to 15 events per hour
2. Moderate: 15 to 30 events per hour
3. Severe: greater than 30 events per hour

4.1.3.2 Justification for the severity criteria

There are currently no adequate prospective studies that have validated severity criteria for sleepiness. The criteria are suggested by the task force as an operational definition. The data to justify a severity index based on event frequency are derived from the Wisconsin Sleep Cohort data that show an increased risk of hypertension that becomes substantial at an AHI of approximately 30.[10] Currently there is no data available to indicate an appropriate distinction between mild and moderate degrees of obstructed breathing events during sleep. The recommended level of 15 reflects a consensus opinion of the Task Force (level 3). Additional data are needed to characterize changing risk profiles with changing frequency of hypopneas/apneas.

4.1.4 Associated features

1. Snoring
2. Obesity
3. Systemic hypertension

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4. Pulmonary hypertension
5. Sleep fragmentation
6. Sleep-related cardiac dysrhythmias
7. Nocturnal angina
8. Gastroesophageal reflux
9. Impaired quality of life[3]
10. Insomnia

4.1.5 Predisposing factors

1. Obesity, particularly upper body adiposity
2. Male gender
3. Craniofacial abnormalities including mandibular/maxillary hypoplasia
4. Increased pharyngeal soft or lymphoid tissue including tonsillar hypertrophy
5. Nasal obstruction
6. Endocrine abnormalities: hypothyroidism, acromegaly
7. Familial history

4.1.6 Prevalence

OSAHS encompasses a wide spectrum of airflow obstruction and associated morbidity. Prevalence varies with levels of severity. Snoring is reported by 40-60% of adults.[16,17] The combination of snoring and "breathing pauses during sleep" have been reported in 2.5% of adults.[18] Using AHI threshold values to define prevalence is problematic because of the previous use of disparate definitions of hypopneas, limiting both comparisons among studies and estimation of prevalence based on the current recommended procedures for measuring hypopneas. AHI levels >5, based on identifying hypopneas using a breathing amplitude criteria in conjunction with desaturation, may be present in 24% of men and 9% of women.[19] Similarly measured AHI levels of >15 may be found in 9% and 4% of men and women, respectively.[19] Using that definition of AHI and requiring sleepiness as another disease defining criterion reduces prevalence estimates to 2 and 4% for women and men respectively. Prevalence may be higher among racial and ethnic minorities.[20,21]

4.1.7 Associated polysomnographic features

Apneas and hypopneas typically are 10 to 50 seconds in duration, although hypopneas lasting several minutes may occur in REM sleep. Episodes may be most prominent when the patient is in the supine position. Apneas and hypopneas often result in oxyhemoglobin desaturation (usually reaching nadir levels within 30 seconds of the termination of the obstructed breath) and sleep fragmentation (with an EEG arousal occurring within three seconds of the termination of the event). Oxygen saturation monitoring usually shows recurrent episodes of desaturation and resaturation in a "saw-tooth" pattern. Sleep monitoring often demonstrates increased stage 1 sleep, reduced 3/4 and REM sleep, and recurrent arousals.

4.1.8 Laboratory features

Laboratory features are non-specific. Severe OSAHS may be accompanied by ECG, chest x-ray, and echocardiography evidence of pulmonary hypertension and right ventricular hypertrophy.

4.1.9 Differential diagnosis

OSAHS should be distinguished from simple snoring which is associated with few episodes of airflow obstruction and no symptoms of disruptive sleep/impaired daytime performance. OSAHS may be present in some patients with an elevated, awake $PaCO_2$ but is distinguished from chronic hypoventilation syndromes by the failure of the $PaCO_2$ to return to normal after relief of the upper airway obstruction with continuous positive airway pressure. OSAHS should be distinguished from central sleep apnea and Cheyne-Stokes Respiration by evidence of continued respiratory effort in the former but not in the latter two. Sleepiness should not be considered a diagnostic feature if it can be attributed to narcolepsy, insufficient sleep, periodic leg movements or non-respiratory arousal disorders, or alcohol and drug use.

4.2 CENTRAL SLEEP APNEA-HYPOPNEA SYNDROME (CSAHS)

4.2.1 Essential features

Idiopathic central sleep apnea-hypopnea syndrome is characterized by recurrent apneic episodes in the absence of upper airway obstruction during sleep, which usually result in oxygen desaturations, recurrent arousals, and daytime symptoms. Central apneas during sleep may occur in a number of circumstances.[22,23] Broadly one can distinguish those which occur in individuals with alveolar hypoventilation, i.e., those who are hypercapnic, and those who are normocapnic or hypocapnic.[22,24] Hypercapnic central sleep apnea overlaps with hypoventilation syndromes and is therefore considered as part of the Sleep Hypoventilation Syndrome (see 4.4). This can be the result of metabolic or neuromuscular disorders. Normocapnic or hypocapnic central sleep apnea can arise in a number of forms including: a) Idiopathic Central Sleep Apnea; b) Cheyne-Stokes Breathing (see 4.3); and c) High Altitude Sleep Apnea. The description of the central sleep apnea syndrome in this report will refer only to the idiopathic form of normocapneic/hypocapneic central sleep apnea.

Idiopathic central sleep apnea is likely to be an uncommon condition. It is believed that individuals with this disorder may have increased ventilatory response to CO_2[25] that causes them to hyperventilate[26] and become hypocap-

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nic.[25] This results in their PCO_2 being closer to the sleep-induced PCO_2 apnea threshold[27] that results from withdrawal of the wakefulness stimulus to respiration.[28]

4.2.2 Diagnostic criteria

The individual must fulfill criteria A, B, and C.

A. At least one of the following symptoms that is not better explained by other factors:
-excessive daytime sleepiness
-frequent nocturnal arousals/ awakenings

B. Overnight monitoring demonstrates 5 or more central apneas plus hypopneas per hour of sleep.[4]

C. Normocarbia while awake ($PaCO_2$ < 45 torr).

4.2.2.1 Central apnea/hypopnea event

An event characterized by reduced or absent breathing and respiratory effort. These events must meet each of the following criteria:

1. Reduction of airflow (see 4.1.2.1 - criteria 1 and 2).
2. A clear reduction in esophageal pressure swings from baseline (see 4.1.2.1 for a definition of baseline). There is no relative or absolute reduction in esophageal pressure that can be used to distinguish central hypopneas from obstructive hypopneas. The reduction in esophageal pressure should parallel chronologically the reduction in airflow.
3. The event lasts 10 seconds or longer.[2]

4.2.3 Severity criteria

There are no outcome studies of patients with the CSAS on which to base a recommendation of severity. At this time it is recommended that the severity of this syndrome not be rated but that further research be conducted to determine if the number of central apneas and hypopneas or some other parameter predicts morbidity or mortality.

4.2.4 Associated features

1. Sleep fragmentation
2. Excessive daytime sleepiness
3. Insomnia

4.2.5 Predisposing factors

The main predisposing factor is an increase in ventilatory response to PCO_2[25] that leads to hyperventilation[26] and a low arterial PCO_2.[25] The basis for this is unknown.

4.2.6 Prevalence

Unknown.

4.2.7 Polysomnographic features

The characteristic finding in patients with idiopathic CSAHS is the presence of recurrent episodes of central apneas or hypopneas associated with the transition from wakefulness to sleep. These alternate with episodes of hyperpnea associated with arousals.[22] Such episodes of hyperpnea trigger a reduction in $PaCO_2$ and hence a central apnea.[26] These episodes occur most frequently in the lighter stages of sleep. They are less frequent in stage 2 and REM sleep and distinctly uncommon in stage 3-4 sleep.[22] These episodes are usually associated with only minor degrees of oxygen desaturation.

4.2.8 Laboratory features

This syndrome is associated with an increased ventilatory response to CO_2.[25] However, evaluation of the ventilatory response is seldom performed for clinical purposes. Arterial PCO_2 is usually normal to low.

4.2.9 Differential diagnosis

CSAHS should be distinguished from OSAHS by demonstrating that there is reduced or absent respiratory effort in the former and maintained or increased respiratory effort in the latter. It is related but distinct from Cheyne-Stokes breathing since there isn't a pattern of crescendo/decrescendo breathing and respiratory effort that is characteristic of Cheyne-Stokes breathing. It may occur in conjunction with the SHVS (4.4) and in this circumstance the $PaCO_2$ is elevated.

4.3 CHEYNE-STOKES BREATHING SYNDROME (CSBS)

4.3.1 Essential features

CSBS is characterized by a cyclical fluctuation in breathing with periods of central apneas or hypopneas alternating with periods of hyperpnea in a gradual waxing and waning fashion. It occurs in patients with cardiac dysfunction usually in association with severe congestive heart failure[29-31] or neurologic disease/dysfunction; usually cerebrovascular.[32] Cheyne-Stokes breathing (CSB) is present during sleep, and in more severe cases may also be observed during wakefulness.

4.3.2 Diagnostic criteria

The individual must fulfill criteria A and B.

A. Presence of congestive heart failure or cerebral neurologic disease.
B. Respiratory monitoring demonstrates:

1. At least three consecutive cycles of a cyclical crescendo

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and decrescendo change in breathing amplitude. Cycle length is most commonly in the range of 60 seconds, although the length may vary.
2. One or both of the following:

a. 5 or more central sleep apneas or hypopneas per hour of sleep.
b. The cyclic crescendo and descrescendo change in breathing amplitude has a duration of at least 10 consecutive minutes.

4.3.3 Severity criteria

The extent of CSB can be documented as the number of events per hour of sleep or the proportion of total sleep time spent with the patient having CSB. There are no large scale studies documenting a link between the extent of CSB and morbidity or mortality on which a severity rating can be confidently based, although there is some evidence that suggests that CSB while awake is more predictive of mortality than CSB confined to sleep.[33] At this time it is recommended that the severity of this syndrome not be rated but that further research be conducted to determine if the number of CSB events predict morbidity or mortality.

4.3.4 Associated features

1. Changes in heart rate, blood pressure, and cerebral circulation which parallel the changes in respiration[34]
2. Transient arousals at the peak of hyperpnea
3. Sleep fragmentation
4. Excessive daytime somnolence
5. Increased ventilatory response to CO_2
6. Awake low or low normal $PaCO_2$
7. Prolonged circulation time

4.3.5 Predisposing factors

1. Congestive heart failure
2. Neurologic disease, in particular cerebrovascular disease

4.3.6 Prevalence

Thirty to 50% of CHF patients with a left ventricular ejection fraction <40% have CSB.[35] The prevalence of CSR among patients with neurological disease is not clear.

4.3.7 Polysomnographic features

Overnight sleep recordings demonstrate the typical pattern of a cyclical fluctuation in ventilation, with periods of central apnea or hypopnea alternating with periods of hyperpnea in a gradual waxing and waning fashion. This breathing pattern predominates in NREM sleep, and arousals occur coincident with the peak of hyperpnea. Parallel changes are also seen in heart rate and blood pressure.

4.3.8 Laboratory features

Awake $PaCO_2$ is usually low or low normal and ventilatory response to CO_2 when measured is usually increased. Left ventricular ejection fraction is usually low in patients with underlying heart disease unless significant mitral regurgitation is present.

4.3.9 Differential diagnosis

CSBS and OSAHS may coexist, and occasionally patients with OSAHS may have a waxing and waning pattern to their breathing. Furthermore, the cyclical fluctuation in respiratory effort may predispose to upper airway obstruction because of a diminished drive to the upper airway dilating muscles during the apneic/hypopneic phase of the cycle.[36] Obstructive apneas/hypopneas have a constant or increasing respiratory effort during the event, whereas in CSB increasing respiratory effort is associated with an increase in ventilation and periods of apnea/hypopnea are associated with absent or reduced respiratory effort. Additionally, arousals typically terminate an obstructed breathing event but if they occur in CSB, they are associated with the peak of ventilation in any cycle of breathing. CSBS should also be distinguished from other forms of central sleep apnea, primarily the idiopathic form by its characteristic pattern of crescendo/decrescendo breathing and respiratory effort.

4.4 SLEEP HYPOVENTILATION SYNDROME (SHVS)

4.4.1 Essential features

The central feature of SHVS is an abnormal increase in $PaCO_2$ during sleep which results in severe hypoxemia. Hypoxemia leads to clinical sequelae such as erythrocytosis, pulmonary hypertension, cor pulmonale, or respiratory failure. The feature of severe hypoxemia during sleep is usually present to some degree throughout the monitoring period with oxygen desaturation episodes that occur in addition to, and which are not associated with, distinct apneas and hypopneas. The oxygen desaturation episodes may be prolonged (>1 minute) and are most severe (in degree and duration) during REM sleep.

4.4.2 Diagnostic criteria

The individual must fulfill criteria A and B:
A. One or more of the following:
-Cor pulmonale
-Pulmonary hypertension
-Excessive daytime somnolence that is not better explained by other factors
-Erythrocytosis

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-Hypercapnia during wakefulness ($PaCO_2$ > 45 torr)
B. Overnight monitoring demonstrates one or both of the following:

1. An increase in $PaCO_2$ during sleep > 10 torr from awake supine values
2. Oxygen desaturation during sleep not explained by apnea or hypopnea events.

4.4.3 Severity criteria [Level of evidence - 3]

SHVS is described as severe if at least one of the following are present:

1. Oxygen saturation <85% for more than 50% of the sleep time
2. Cor pulmonale or biventricular failure

4.4.4 Associated features

1. Biventricular heart failure
2. Systemic hypertension
3. Sleep-related cardiac dysrhythmias
4. Impaired quality of life[3]
5. Central apnea

4.4.5 Predisposing factors

1. Morbid Obesity (BMI > 35)
2. Chest wall restrictive disorders
3. Neuromuscular weakness or disorder (e.g. amyotrphic lateral sclerosis)
4. Brainstem or high spinal cord lesions
5. Idiopathic central alveolar hypoventilation
6. Obstructive lung disease[5]
7. Hypothyroidism

4.4.6 Prevalence

Prevalence is currently unknown. The idiopathic form is rare. It is likely that sleep hypoventilation is common in patients who have abnormal mechanical properties of the lungs or chest wall or neuromuscular weakness.

4.4.7 Polysomnographic features

Periods of hypoventilation are detected by measuring an increase in arterial CO_2 levels. There is insufficient evidence that increases in $PaCO_2$ can be reliably detected noninvasively (see 5.5). Periods of hypoventilation are usually evident by sustained arterial desaturation lasting up to several minutes. However this is not a specific finding since it may also be caused by a change in lung or closing volume that can cause a worsening in ventilation perfusion homogeneity. Hypoventilation periods are much more common and severe in REM than in NREM sleep.

4.4.8 Laboratory features

There may be ECG, chest x-ray, and echocardiography evidence of pulmonary hypertension and right ventricular hypertrophy. An elevated hematocrit and hemoglobin may be present as a complication of severe hypoxemia. There may be a reduction in respiratory muscle strength and impaired pulmonary function.

4.4.9 Differential diagnosis

Apneas and hypopneas may occur in association with hypoventilation; however SHVS should only be diagnosed if the clinical sequelae can be attributed to sleep hypoventilation separate from the apneas and hypopneas. In the case of obstructive apneas and hypopneas this requires demonstration that the increase in $PaCO_2$ or the prolonged periods of hypoxemia are not corrected by adequate treatment of the upper airway obstruction.

Almost all disorders that result in awake hypercapnia are complicated by sleep hypoventilation. Sleep hypoventilation likely precedes awake hypercapnia in most cases. An exception may be chronic airflow limitation which is a common cause of awake hypercapnia but is not necessarily complicated by an additional abnormal increase (>10 torr) during sleep. There is a moderate correlation between the severity of airflow limitation and the severity of hypercapnia. When two or more predisposing factors for sleep hypoventilation are present, the severity of hypercapnia awake and during sleep is increased.

5.0 RECOMMENDATIONS FOR MEASUREMENT TECHNIQUES

For each type of sleep-related breathing event the task force determined what the appropriate reference standard was. The task force then attempted to determine the utility of alternative methods of measuring an event or feature by evaluating available research for information on its precision and accuracy. Recommendations for a particular method were based on its ability to detect an event without corroborating data (such as oxygen desaturation or arousal). The task force recognized, however, that where there was uncertainty about an event, other information like oxygen desaturation or an arousal could be a useful arbiter.

Precision should be assessed by determining the test-retest variability. The method of analyzing this type of data proposed by Bland and Altman (coefficient of repeatibility) is preferred to the more commonly reported correlation analysis.[37] Where there is an element of judgment required to score an event, such as a hypopnea, an analysis of agreement of the method both within scorers (intrascorer agreement) and between scorers (interscorer agreement) should

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be determined. This requires an event by event analysis and the recommended method of reporting the degree of agreement is Cohen's kappa statistic.[38]

Accuracy or validity of a method of measurement is an indication of how closely the method can estimate the true value of whatever is being measured. This can be evaluated in several ways.[39] In circumstances where there is a recognized reference standard, the degree of agreement between the two methods should be assessed again using the Bland and Altman method[37] and not with a correlation coefficient. This is commonly referred to as criterion validity. Cohen's kappa statistic can be used to assess the degree of agreement between methods when an event by event comparison is being made as another measure of criterion validity. Predictive validity can be used in evaluating a new metric comparing the degree that it agrees with other related metrics or with important outcomes. This represents indirect evidence and requires several comparisons to be made to determine if the metric relates to other metrics or to important outcomes in an understandable and predictable way. Another type of validity, referred to as face validity, involves assessment of the method by experts to determine if there is a rational theoretical framework on which to base an opinion that the metric is a reasonable method of measurement that records valuable information.

None of the commonly used methods for detecting breathing disorders during sleep have been rigorously evaluated. The task force believed that any method of measurement should be evaluated in the setting for which it is intended, that is in sleeping, unrestrained subjects undergoing full overnight polysomnography. There was little or no data reported on the precision of most methods in this setting and only indirect evidence of validity. In some circumstances available evidence indicated that a particular metric correlated poorly with a reference standard. The task force accepted such evidence to support a lower grade of recommendation for such a method. However, it was not possible to find enough supportive evidence that indicated that any metric was as accurate and precise as the reference standard. Thus in most circumstances the task force relied on face validity and assigned recommendations based on a consensus opinion of its members (Table 2). A recommendation of D was given if there was evidence available that the method in question had been shown not to agree with a reference standard. An intermediate grade of B or C was assigned to methods that had face validity in the opinion of the task force. Methods that had a quantitative or at least a semi-quantitative relationship with the reference standard were felt to be superior to those that did not and were given a B recommendation. The task force believed that basic research on measurement methodology should be a high research priority so that in the future there is a greater amount of empirical evidence on which to base these recommendations.

When there was supporting evidence for the recommendations it was stated along with the level of recommendation (see Table 3). Different types of indirect evidence (Level 2) were labeled separately (2a, 2b, 2c), but no type of evidence was considered superior to another type.

Table 2. Grades of Measurement Methodology

Grade	Interpretation
A	Good to excellent agreement with the reference standard.
B	Limited information available on which to base a firm recommendation. There is a good theoretical framework and clinical experience with the method that indicates it is valid. More research is required to confirm the utility of the method.
C	Very little or no research available to indicate whether the method is valid. There is a weak theoretical framework and/or clinical experience that suggests the method is not as good as others with a higher rating.
D	Research and/or clinical experience suggest that the method is not valid for this application.

Table 3. Quality of Evidence for Recommendations of Measurement Methodology

Level	Type of Evidence
1	i) Reference standard (RS) measurement or ii) Method has been compared to a RS in an appropriately designed study.
2a	Method has been compared with a RS measurement but analysis of precision or accuracy was based only on correlation or similar analysis. If a moderate to high agreement was found, the method would appear valid but further research is required to confirm. If poor agreement is found the method is likely not valid.
2b	Method shows moderate to high correlation with a short-term outcome of importance (e.g.; oxygen desaturation or arousal).
2c	Method correlates with a long-term outcome of importance (e.g.; hypertension, motor vehicle accidents, or quality of life).
3	No systematic study of the method or its correlation with morbidity has been published or published studies show conflicting results.

5.1 Obstructive hypopnea/apnea

In routine clinical practice it is not necessary to differentiate apneas from hypopneas because both types of events have a similar pathophysiology and consequences. Both apneas and hypopneas usually end in arousal and produce desaturation.[1,6,40] There are currently no data to suggest different long or short term outcomes in patients with predominantly apneas as compared to hypopneas. For research studies that aim to differentiate risk profiles or elucidate pathogenic mechanisms, it may be useful to score and report hypopneas and apneas separately (see Appendix 1). Methods for the measurement of hypopneas are adequate to measure apneas; however the converse is not necessarily true. Therefore, the recommendations and quality of evidence listed below relate to the ability of a particular method to detect the presence of a hypopnea.

Since the initial description of these events, criteria for measuring hypopneas have varied widely.[41] Sources of

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variability include differences in sensors for detecting breathing amplitude change, the absolute amplitude criteria considered sufficient to identify a breathing pattern as "reduced," and the use of corroborative data (oxygen desaturation and arousal) in the hypopnea definition.

Because most apneas/hypopneas are caused by upper airway obstruction/narrowing, it is assumed that a particular event is obstructive unless it is clearly demonstrated to be central in origin (see 5.3).

5.1.1 Reference Standard Measurement: Reduction of air flow detected by pneumotachometer.

Tidal volume measurement requires continuous monitoring of total oronasal airflow, which in most circumstances requires a snug-fitting face mask with the pneumotachometer. If a pneumotachometer is not used, sleep studies should include two independent techniques for the measurement of hypopneas, in order to allow for sensor failure.

5.1.2.1 Nasal Pressure

Recommendation - B
Evidence - 2a & 2b

Detection of fluctuation in nasal pressure during inspiration and expiration reflects changes in inspiratory and expiratory airflow and therefore is a promising method for the detection of hypopneas. Moderate accuracy measuring ventilation while awake has been demonstrated for this method.[42] When used during wakefulness, nasal pressure has a better negative predictive value and a poorer positive predictive value than RIP (when each is compared to body plethysmography).[42] In healthy subjects studied during wakefulness, there is a curvilinear relationship between flow measured by a nasal cannula compared to flow measured with a full face mask by a pneumotachometer.[43] There is only preliminary data on the accuracy of measuring ventilation during sleep with nasal pressure compared to a pneumotachometer suggesting that the former is not as sensitive as the latter for detecting hypopneas.[44] However, other studies have shown nasal pressure to be more sensitive than thermal sensors for detecting hypopneas.[45]

The impact of several potential limitations in the use of nasal pressure/flow on the accuracy and precision of the diagnosis of the OSAHS require clarification. These include the non-linear relationship between pressure changes detected at the nostrils and actual airflow,[43] which may result in underestimation of airflow, the occurrence of false positive detection of hypopneas as a result of nasal obstruction or mouth breathing, and the feasibility of obtaining an adequate signal in patients with nasal obstruction.

It is not clear whether the sensitivity of this method could be improved if criteria that included an oxygen desaturation or an arousal were included to arbitrate equivocal events (see section 5.1.2.8).

5.1.2.2 Respiratory Inductance Plethysmography (RIP) - Sum of Chest and Abdominal Signals.

Recommendation - B
Evidence - 2a & 2b

RIP measurement is based on detection of changes in volume of the chest and abdomen during inspiration and expiration. When properly calibrated the sum of these two signals can provide a measure of tidal volume. If uncalibrated the amplification should be such that both signals have similar amplitudes. It may be difficult to maintain a calibrated sum signal throughout an entire sleep study, so it is often not practical to base hypopnea detection on an absolute reduction in tidal volume as reflected by the sum signal. Therefore, a relative reduction of 50% from baseline in the sum signal of calibrated or uncalibrated RIP is recommended (see 4.1.1).

The literature suggests that RIP is acceptable for semiquantitative measurements of ventilation. Moderate accuracy for measuring ventilation awake was demonstrated by using body plethysmography or spirometry as reference standards.[42,46] During wakefulness the sensitivity and specificity of detecting hypopneas (defined as a 50% reduction in breathing amplitude) was 0.86 and 0.65.[42] RIP provides moderate accuracy for measuring ventilation during sleep (mean variation, 10%), with no consistent bias.[47,48] Good to excellent reproducibility has been demonstrated between observers for scoring hypopneas on the basis of 50% reduction in RIP sum from sleeping baseline.[48] Moderate agreement has been measured for hypopneas using a 50% reduction in sum signal (uncalibrated) from sleeping baseline and the short term outcome measures of arousals and desaturations.[1[6]] There are no data on the association between hypopnea detection by RIP alone and long term outcomes.

5.1.2.3 RIP - Dual Channel.

Recommendation - C
Evidence - 3

In circumstances where a sum signal is unavailable, hypopneas can be defined by a ≥50% decrease in both chest and abdominal signals from baseline.

There are no data currently available to correlate this type of measurement with the gold standard or short or long term outcomes. However, the task force consensus was that in the absence of a sum signal, analysis of the RIP chest and abdominal channels can indicate a hypopnea, provided that there is a good baseline from which to make this judgment (see 4.1.2.1).

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5.1.2.4 Single Channel RIP

Recommendation - C
Evidence - 3

In circumstances where one of the 2 RIP channels has failed, a hypopnea can be scored if the remaining channel shows a > 50% decrease from baseline (as per 4.1.2.1 and 5.1.2.2) or a < 50% decrease from baseline associated with oxygen desaturation of ≥3% or an arousal (as per 4.1.2.1 and 5.1.2.3).

There are currently no data that have independently evaluated a single RIP channel compared with dual channels or a sum signal in the detection of hypopneas during sleep. However, there are times when one of the RIP channels fail and the task force consensus was that the remaining channel can still indicate a hypopnea provided there is a good baseline from which to base this judgment.

5.1.2.5 Piezo Sensors, Strain Gauges, and Thoracic Impedance

Recommendation - C
Evidence - 3

Piezo sensors, strain gauges, and impedance sensors are commonly used to detect hypopneas; however, they provide only qualitative information on changes in ventilation or airflow. There is little data on which to judge the accuracy of these sensors to quantitatively or semi-quantitatively record flow or volume changes compared with reference standards. Therefore, it is felt that a relative reduction in amplitude (e.g. > 50%) of this signal may not be as reliable to indicate the presence or absence of a hypopnea as some other methods. It is likely that these sensors will be more accurate in recording hypopneas if they are used according to section 5.1.2.8 (i.e. accompanied by oxygen desaturation or an arousal). However, the Task Force discourages the use of signals that routinely require additional channels like oxygen saturation or electroencephalography in order to score events.

5.1.2.6 Thermal sensors

Recommendation - D
Evidence - 2a

There are limited published data that document the accuracy and precision of these devices. In laboratory models that have compared thermocouples and thermistors to a pneumotachograph, the signals from the thermal sensors have been shown to be non-linearly related to actual airflow, while generally resulting in overestimation of ventilation.[49] Thus the detection of airflow by thermal sensors provides only qualitative information that is not well correlated with breath amplitude. Therefore, a relative reduction in amplitude (e.g. > 50%) of this signal can not be used to reliably indicate the presence or absence of a hypopnea.

Thermal sensors have poor accuracy in recording hypopneas in awake subjects under ideal conditions.[42] The limited data available assessing hypopneas detected by thermal sensors with short term outcome data has shown poor agreement.[1]

Due to the nature of the signal, it is unlikely that any further research on thermal sensors would yield acceptable data on accuracy or precision.

5.1.2.7 Expired CO_2

Recommendation - D
Evidence - 3

Expired CO_2, like thermal sensors, is a qualitative indicator of flow. There are no data available that evaluate the accuracy or precision of expired CO_2 nor is there any data concerning this method of detecting hypopneas and its correlation with outcomes. The task force consensus was that, like thermal sensors, expired CO_2 was a technically inadequate signal.

5.1.2.8 Breathing Measurement Signal - < 50% Decrease with Desaturation or Arousal

Recommendation - B
Evidence - 2b

A discernible but less than 50% decrease in the breathing measurement signal, can be scored as a hypopnea. However, the event must be associated with oxygen desaturation ≥3% (see 4.1.1) or terminate with an arousal. The Task Force recommends that this definition be used to arbitrate those events that are felt to be equivocal by the person scoring the sleep study. It should not be used as a primary definition of a hypopnea. It is recommended to be used in conjunction with those methods given an A, B, or C recommendation.

The justification for considering lesser reductions in signals recording breathing, when associated with an oxygen desaturation, in event identification is based on two studies that used RIP as the primary method of recording disturbances in breathing. In the Wisconsin Sleep Cohort study,[19] hypopneas were defined as a clear decrease in amplitude of calibrated RIP accompanied by a ≥4% oxygen desaturation. The severity measure (AHI) based on this definition correlated with blood pressure,[10] neuropsychologic test scores,[50] quality of life (SF-36),[51] sleepiness, and automobile accidents.[11][7] These data have been reanalyzed using hypopnea definitions based on the criteria in 5.1.2.2 and 5.1.2.8 (≥50% decrease in RIP sum or a clear

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decrease in RIP sum (>20%) plus a ≥3% O_2 desaturation), and the correlations with the important outcomes did not change appreciably (data available on request from T. Young). In a clinical trial of CPAP, hypopneas were defined on the basis of lesser reductions in thermistry and/or impedance that were associated with desaturations of 3%.[52,53] This approach successfully classified groups who differed in relevant outcomes such as level of vigilance and quality of life. Based on these data the Task Force concluded that the hypopnea definition in this section was valid.

There are numerous approaches for identifying arousals but insufficient data to recommend a specific approach for use in hypopnea evaluation. Since arousal scoring may reduce the precision of hypopnea scoring it is recommended that all reports of hypopneas using this criterion contain the following information: 1) A clear arousal definition; 2) The percentage of total hypopneas scored using the arousal criteria; 3) for research studies, a Kappa statistic[38] for inter-rater and intra-rater reliability for scoring hypopnea events defined by an arousal. If the kappa statistic is less than 0.6 the method of scoring hypopneas may not be reliable.[54]

5.1.3 Recommendations for Further Methodologic Research

Standard criteria need to be established for quantifying and interpreting data collected from sensors used to detect hypopneas. The adequacy of these criteria should be judged by the accuracy of any measurement or set of measurements compared with a gold standard, or by the ability to predict clinically relevant short and long term outcomes. The method should have demonstrated test-retest reliability and, if appropriate, inter-rater and intra-rater reliability. Ideally, evaluations should be in unrestrained subjects during sleep and in subjects (males and females) with a range of disease severity and obesity.

The pathophysiological consequences of recurrent hypopneas are not likely to result from absolute changes in ventilation that occur with obstructed breathing events. Rather, the consequences are more likely to be related to cortical or sub-cortical arousals caused by increased respiratory effort during hypopneas,[55] or to oxyhemoglobin desaturations. The emphasis on identifying events based on amplitude changes in ventilation are based on the premise that this approach may most consistently and noninvasively detect events that are associated with physiological stresses. However, more direct methods for detecting and quantifying inspiratory effort and arousal may improve the ability to detect clinically meaningful changes in breathing. Additional work is needed to identify optimal methods for detecting increased inspiratory effort and cortical and subcortical arousal. Methods need to be widely applicable outside of research laboratories. The ability of any measurement or set of measurements to predict short and long term, clinically relevant outcomes, is of utmost importance.

5.2 Respiratory effort related arousal (RERA)

The upper airway resistance syndrome (UARS) was described by Guilleminault et al.[2,56] as an abnormal breathing pattern during sleep associated with daytime sleepiness unexplained by any other cause, including obstructive sleep apnea. The essential feature proposed for the diagnosis of the UARS was episodes of increased respiratory effort resulting in an arousal index of more than 10 events per hour of sleep. These episodes occurred in the absence of obstructive sleep apnea but were associated with the clinical complaint of excessive daytime sleepiness. It remains unclear whether the new criteria for hypopnea measurement will improve the sensitivity for detecting these events. Thus, there may be obstructive events that occur during sleep, that do not fulfill criteria for an apnea or hypopnea, but that result in significant symptoms due to arousal and sleep fragmentation. The detection of such events requires specialized diagnostic monitoring techniques.

5.2.1 Reference Standard Measurement: Esophageal pressure

The measurement of esophageal pressure with continuous overnight monitoring is the reference standard for measuring respiratory effort.[2,56-63] The main finding reported is that of increasing negativity of esophageal pressure prior to an EEG arousal. The arousal is associated with a rapid increase in the esophageal pressure back to resting levels. This rapid increase of esophageal pressure is an essential feature of the measurement and helps to distinguish an event from a nonphasic increase in pressure that may occur during stages 3 and 4 of NREM sleep. There is no absolute level of esophageal pressure that is known to be abnormal.

No studies have compared alternative techniques of measuring respiratory effort or flow limitation with the reference standard. Therefore, no other techniques have demonstrated accuracy and precision, or have shown a correlation with clinically important outcomes.

5.2.2.1 Nasal Pressure

Recommendation - C
Evidence - 3

The detection of flow limitation by nasal pressure monitoring may be possible. The shape of a continuous recording of inspiratory and expiratory pressure can detect flow limitations with either a full face mask[64] or nasal pressure cannulae.[65] This technique may lack specificity under certain clinical circumstances. If only nasal pressure cannulae are used, the technique may lack sensitivity if the patient employs predominantly mouth breathing. Currently there

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are no data linking measurement of events recorded by measuring nasal pressure with important clinical outcomes. Thus although this technique appears promising it is not recommended as a measurement of a RERA until further methodologic research has been published.

5.2.2.2 Supraglottic Pressure

Recommendation - C
Evidence - 3

Measurement of the pressure difference between the mouth and hypopharynx just proximal to the glottis, when combined with quantitative airflow measurement, provides a measurement of upper airway resistance. This measurement technique has not been investigated rigorously as a method for detecting events distinct from apneas and hypopneas.[66] It should have similar advantages and may have the same drawbacks (could disturb sleep), as esophageal pressure measurement.

5.2.2.3 Diaphragmatic EMG (Surface)

Recommendation - D
Evidence - 3

Diaphragm EMG is an indirect measurement of respiratory effort. It is a difficult signal to record reliably and continuously, and there is no direct way to correlate it with esophageal pressure or upper airway resistance. There are no data on accuracy, reliability, or correlation with long term outcome in relation to this technique.

5.2.3 Recommendations for Further Methodologic Research

Further studies are needed to determine whether RERAs are a distinct event from hypopneas or merely reflect a form of hypopnea that requires more sensitive methods for detection. The relative prevalence of RERAs compared with hypopneas within individual patients and between patient populations also remains to be determined. If RERAs are relatively uncommon it may not be worthwhile to employ sophisticated techniques for their measurement as a distinct event. The short and long term outcomes of treated and untreated patients with RERAs, and possible associations between the level of negative pressure and clinically-important outcomes, also await further study. Careful validation studies are needed to adequately document the relationship of any proposed measurement of these events with the reference standard and important clinical outcomes. Finally, as with all measurements there needs to be adequate reporting of test-retest reliability, as well as inter-rater and intra-rater reliability.

5.3 CENTRAL HYPOPNEA/APNEA

Central sleep apnea is much less common than obstructive sleep apnea.[22] Identification of central apnea requires demonstration that respiratory effort is absent. In one apnea event both increased airway obstruction and reduced (but not absent) respiratory effort may coexist. In current parlance this would be called an obstructive event. Thus, currently the term central apnea is used exclusively for those events characterized by cessation of respiratory drive to the inspiratory pump muscles.

5.3.1 Reference Standard Measurement: Esophageal pressure

The lack of respiratory effort can be determined by measurement of esophageal pressure[22,67,68] and demonstrating that it is absent during an apnea or reduced from baseline during a hypopnea.

5.3.2.1 Respiratory Inductance Plethysmography (RIP)

Recommendation - C
Evidence - 3

Complete absence of thoraco-abdominal movement documented by RIP, regardless of calibration, should reflect central apneas. However, available data are limited and suggest a high rate of misclassification by RIP or strain gauges.[67,68]

5.3.2.2 Diaphragm EMG (surface recording)

Recommendation - C
Evidence - 3

Measurement of a reliable diaphragm surface EMG signal is not a usual part of routine clinical polysomnography recording. It can be difficult to obtain stable recordings across the night of study. At present this is not recommended for routine clinical purposes although in some patients it may provide valuable information. Diaphragm EMG continues to have a role in research studies.

There are limited data regarding using diaphragm EMG to distinguish central from obstructive sleep apnea.[24] The task force consensus was that this technique would be acceptable if an adequate recording could be made prior to and maintained throughout a sleep study. In an individual patient this would require demonstration of an adequate diaphragm EMG recording at the conclusion of the sleep study.

5.3.2.3 Sensors detecting oro-nasal airflow

Recommendation - D
Evidence - 3

Thermal sensors or expired CO_2 qualitatively assess air-

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flow and may be able to detect apneas, but are incapable of distinguishing central from obstructive events. There are no published reports evaluating the use of these types of sensors for distinguishing central from obstructive apneas. It is unlikely that future research will validate these measurement techniques for this purpose.

5.3.2.4 Piezo Sensors and Strain Gauges

Recommendation - D
Evidence - 2a

Piezo sensors and strain gauges provide only qualitative information on changes in ribcage and abdominal movement, therefore they can not be used to reliably distinguish between central and obstructive events. In one study 37% of apneas scored as central based on strain gauge measurement were reclassified as obstructive or mixed based on esophageal pressure measurements.[68] This indicates a high misclassification rate.

5.3.3 Recommendations for Further Methodologic Research

More methodological studies are needed to determine the relative role of increased upper airway resistance and reduced activity of the respiratory pump muscles in respiratory events during sleep. This is particularly true for patients with congestive heart failure or stroke, where various degrees of obstructive sleep apnea and Cheyne-Stokes breathing may co-exist. If and when new therapeutic approaches are developed that require this distinction to be made, the need to establish the relative role of central and obstructive components may become more important. Studies are needed to determine the sensitivity and specificity of surface techniques such as RIP and diaphragm EMG to identify central apneas, because most previous studies have been conducted in patients suspected of having obstructive sleep apnea. In addition the reliability of these measurement techniques need to be established.

5.4 CHEYNE-STOKES BREATHING

The apneas and hypopneas which occur at the nadir of the cyclical breathing in CSB are central in origin and therefore the same comments regarding measurement for central apneas (section 5.3) apply. However, CSB is felt to be distinct from idiopathic central sleep apnea because of the unique pattern of crescendo and decrescendo breathing due to a fluctuation in breathing effort.

5.4.1 Reference Standard Measurements: Esophageal pressure; pneumotachometer

The reference standard method for documenting that the apneas or hypopneas in CSB are central in origin is measurement of an absence (apnea) or reduction (hypopnea) in esophageal pressure fluctuation. The reference standard method for documenting the crescendo-decrescendo change in ventilation is a measurement of airflow using a pneumotachometer. See comments under 5.1.1.

As described above (5.3) no other techniques for distinguishing central from obstructive apneas that have been systematically validated against esophageal pressure measurements. Likewise there are no studies which have systematically validated different methods for quantitating breathing (ventilation) and compared these to pneumotachometer measured flow.

5.4.2.1 Respiratory Inductance Plethysmography (RIP)

Recommendation - B
Evidence - 3

The characteristic crescendo-decrescendo pattern of breathing can be detected by RIP in patients with diseases that are known to be associated with CSB. It is recommended that a display of chest wall, ribcage, and the sum of these signals be displayed and used to detect the pattern of breathing and also to exclude paradoxical motion of the chest and abdomen which would could indicate a component of upper airway obstruction. If doubt exists whether obstructive sleep apnea is also present, further studies may be needed, including measurement of esophageal pressure.

This method may be acceptable for detecting the central apneas and hypopneas at the nadir of breathing during CSB. However, the reservations with this method outlined in 5.3.2.1 apply. RIP is currently the most common method for documenting CSB,[35,69-72] but there have been no formal validation studies to demonstrate the reliability and accuracy of this method in these patients.

5.4.2.2 Diaphragm EMG (surface recording)

Recommendation - C
Evidence - 3

See comments under 5.3.2.2.

5.4.2.3 Sensors detecting oro-nasal airflow

Recommendation - D
Evidence - 3

These sensors should be able to detect apneas and may demonstrate the typical CSB breathing pattern but alone can not distinguish central from obstructive apneas and hypopneas. See comments under 5.3.2.3.

5.4.2.4 Oximetry

Recommendation - D
Evidence - 3

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Currently there are no data or systematic studies on the accuracy and reliability of oximetry alone to detect CSB. Since there are many related disorders that can lead to cyclical fluctuation of O_2 saturation the task force consensus was that it was unlikely this method would have sufficient sensitivity or specificity to be clinically useful.

5.4.3 Recommendations for Further Methodologic Research

The same recommendations for methodologic research that were described for measurement of central apnea (5.3.3) also pertain to measurement of CSB. In addition, there is a need for further, carefully performed and controlled studies to examine the prevalence of CSB during sleep in patients with neurological disease, particularly stroke. There is a definite need for studies that include measurements of respiration that can reasonably distinguish CSB from other forms of disordered breathing during sleep. Outcome studies are also needed to determine whether it is important to distinguish patients with CSB from those who have obstructive sleep apnea and hypopnea in association with heart failure or stroke.

5.5 SLEEP HYPOVENTILATION

Sleep hypoventilation specifically refers to alveolar hypoventilation. It is commonly accepted that arterial carbon dioxide ($PaCO_2$) is directly correlated with and usually within a few torr of alveolar CO_2 (P_ACO_2) and therefore is the reference standard measurement. In normal adults the $PaCO_2$ normally rises between 2 and 7 torr during sleep.[73-75] Sleep hypoventilation may occur in subjects who are obese or have an underlying disorder such as COPD, kyphoscoliosis, or neuromuscular disease who do not yet have daytime hypercapnia.[76] Wakefulness and vigilance are important respiratory stimulants even in those with no chemosensitivity.[77] In normal subjects there is usually little or no difference in the level of $PaCO_2$ between NREM and REM sleep.[78-80] Although it is possible to monitor arterial blood gases during a sleep study it is rarely practical or feasible to do so. Awake $PaCO_2$ is usually controlled within narrow limits; chronic hypercapnia is commonly defined as a sustained increase in PaCO2 above 45 torr[81] and almost always indicates significant respiratory system pathology.

5.5.1 Reference Standard Measurement: $PaCO_2$

Sleep hypoventilation is best identified by an increase in the level of $PaCO_2$ ≥10 torr during sleep compared with an awake supine value. Currently there are no methods with demonstrated accuracy and precision which can be recommended as a substitute for the measurement of $PaCO_2$.

5.5.2.1 Unexplained Oxygen Desaturation.

Recommendation - B
Evidence - 3

Periods of a substantial decline (> 10%) in oxygen saturation, without evidence of upper airway obstruction, may indicate a period of hypoventilation. This is a nonspecific finding because a change in lung volume and/or closing volume may alter ventilation/perfusion relationships and cause hypoxemia without a change in alveloar ventilation. Additionally any patient using supplemental oxygen could have sleep hypoventilation that was not associated with marked desaturation. There are currently no data available that have assessed the accuracy of nocturnal oxygen desaturation as an indicator of sleep hypoventilation.

5.5.2.2 Transcutaneous CO_2 ($TcCO_2$)

Recommendation - C
Evidence - 1

There are two methods of measuring $TcCO_2$.[82] The first is based on a silver chloride electrode which measures CO_2 that has diffused from the skin through a gas permeable membrane into solution. The second utilizes an infrared capnometer that analyzes CO_2 in a gas phase. The response time of the first method is quicker (< 1 minute) than the second (> 2 minutes).[82]

Many studies have reported the results of $TcCO_2$ measurements in patient populations known to hypoventilate. Increases in $TcCO_2$, especially in REM sleep, are commonly reported, as are reductions in $TcCO_2$ with treatment.[83-86] These data suggest that $TcCO_2$ might be a useful alternative measure of $PaCO_2$. It has been validated in awake patients[87-89] and in at least one report it was shown to be very accurate when compared to the reference standard.[90] However, there is only one study which reports the accuracy of $TcCO_2$ during sleep in patients with sleep hypoventilation.[91] There was a large discrepancy in many instances between $PaCO_2$ and $TcCO_2$ measured simultaneously. It is not clear at this time whether in the future $TcCO_2$ can be shown to be accurate during sleep in some circumstances. Thus, although one study reported level 1 evidence indicating poor accuracy, $TcCO_2$ appears to be valid in other clinical settings and has responded as expected in small case series of patients. The task force consensus was that further investigations were required to determine its accuracy and precision under different study conditions during sleep. At this time, largely based on the findings of one study, the task force consensus was that $TcCO_2$ should not be used routinely to diagnose sleep hypoventilation but may have some utility in the future pending results of further research.

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5.5.2.3 Calibrated Respiratory Inductance Plethysmography

Recommendation - D
Evidence - 2a

Properly calibrated RIP has been shown to be an accurate method of quantifying tidal volume and therefore, minute ventilation.[92] It may be possible to assess hypoventilation with this technique.

Calibrated RIP has been studied in different patient populations with variable results. The only study that compared the accuracy of CRIP to pneumotachograph expired volume in unrestrained, sleeping subjects found relatively poor correlations, especially with body position changes.[93] Clinical experience indicates that it can be difficult to properly calibrate respiratory inductance bands in obese patients. In addition minute ventilation is an indirect measure of alveolar ventilation and the relation between the two may change from wakefulness to sleep. Thus it is doubtful that this technology can measure ventilation accurately and reliably enough to diagnose sleep hypoventilation.

5.5.2.4 Expired End tidal CO_2 ($ETCO_2$)

Recommendation - D
Evidence - 1

Continuous measurement of CO_2 in expired air shows a gradual increase until near the end of a complete expiration when there is a plateau in the CO_2 level. This $ETCO_2$ measurement reflects P_ACO_2.

$ETCO_2$ has been used in several clinical scenarios as a measurement of P_ACO_2 including patients weaning from mechanical ventilation,[94-96] anesthesia,[97] exercise[98] and sleep.[99] There are several potential limitations to the practical measurement of $ETCO_2$ including the inability to measure $ETCO_2$ during CPAP or bilevel pressure therapy to assess response to treatment.[82] The only study that has compared $ETCO_2$ to the reference standard found poor accuracy in sleeping patients under conditions of spontaneous breathing, nasal prong oxygen, and positive pressure therapy.[91] Patients with underlying lung disease are unlikely to have uniform ventilation-perfusion ratios, which will further affect the accuracy of $ETCO_2$.[82] Furthermore, as tidal volume decreases, the ability to identify an alveolar plateau and thus infer alveolar gas concentration becomes increasingly difficult. For all these reasons it is unlikely that this technology will be useful for non-invasive measurement of $PaCO_2$ during sleep.

5.5.3 Recommendations for further methodologic research

Further assessment of $TcCO_2$ during sleep should be compared to $PaCO_2$ with particular assessment of accuracy and reliability under different study conditions (e.g., NREM vs REM). There should be less emphasis on the comparison of absolute values and more emphasis on the change in $TcCO_2$ vs the change in $PaCO_2$. In addition, future studies should determine the accuracy and reliability of oxygen desaturation as a marker for hypoventilation periods during sleep.

5.6 EXCESSIVE DAYTIME SLEEPINESS

The existing operational definition of excessive daytime sleepiness (EDS) in the *International Classification of Sleep Disorders*[100] is based on the behavior of falling asleep, e.g. difficulty maintaining the alert, awake state and unintentional falling asleep. The complaint of sleepiness, however, is broadly interpreted to include drowsiness, low vitality and tiredness in addition to the occurrence of uncontrollable sleepiness.

5.6.1 Reference Standard

Although measurement of the physiologic propensity to fall asleep in the daytime (Multiple Sleep Latency Test, see below) is considered the reference standard for the objective definition of sleepiness, there is no reference standard for subjective measures of sleepiness. Until the components of daytime sleepiness are better understood, sleepiness identified by any method, subjective or objective, should be viewed as clinically important. At this time a rating of the acceptability of existing techniques is not possible.

5.6.2 Techniques for Assessment of Daytime Sleepiness

Current techniques for assessing daytime sleepiness include physiologic tests to measure the propensity to fall asleep or stay awake and self-rated instruments to measure perceived feelings of sleepiness and sleepy behavior. Some instruments assess the presence of current sleepiness; others attempt to measure "usual" sleepiness, or that which is sustained over longer periods of time. These various techniques measure different aspects of sleepiness, and the lack of correlation between different measures has not led to conclusions that any individual test has poor validity. Several published articles include thoughtful critiques of a wide range of objective and subjective sleepiness measures.[101-103]

5.6.3 Objective Measures

5.6.3.1 Multiple Sleep Latency Test (MSLT)

The MSLT measures the speed of falling asleep under conditions that favor sleep, in a series of 20 minute trials over the day.[104] It is the only measure with clearly defined standard procedures, with clinical practice guidelines, and with established cutpoints for severity.[105] The MSLT is

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often used as the gold standard for evaluation of other sleepiness measures. The 1992 AASM guidelines cite the MSLT as the means of establishing a diagnosis of specific sleep disorders or to determine the severity of sleepiness. The ICSD[100] defines severe sleepiness as a MSLT score of 5 or less (average sleep latency of the nap trials = 5 minutes or less) and moderate sleepiness as a score between 5 and 10. The MSLT has been shown to reliably detect sleepiness under experimental and clinical conditions.[105] The guidelines also state that normative data, sensitivity and specificity of the MSLT are needed, and further studies are required to define the MSLT's role in diagnostic considerations of idiopathic hypersomnolence and sleep-related breathing disorders.

5.6.3.2 Maintenance of Wakefulness Test (MWT)

The MWT was developed as a measure of the ability to stay awake when desired.[106] Compared with that for the MSLT, validation literature is less extensive. One study suggests that the MSLT and the MWT measure different dimensions of sleepiness,[107] but both measures produced similar findings in a recent multicenter trial of a wake-promoting drug.[108] A major limitation of the MWT at the present time is the lack of standardization and limited data for normative values.[109]

5.6.4 Subjective Measures

Sleep symptom histories and inventories typically contain many questions that solicit perceived sleepiness. Many inventories pose questions to be answered "yes" or "no"; others solicit a graded response on frequency or severity, and still others are posed as how one usually feels, rather than the feeling at that moment. Two questions common to most symptom inventories are the frequency of: 1) excessive daytime sleepiness, periods when it is "difficult to fight off sleep" or "uncontrollable sleepiness"; and 2) awakening unrefreshed - assessing the presence of a sleep drive that is not satisfied by sleep time, whereby there is sleepiness regardless of the number of hours of sleep. There has been no validation of these single-question measures and they have not been significant components of predictive models for OSA in clinic or general population samples.[16,110,111]

5.6.4.1 Stanford Sleepiness Scale (SSS)

The SSS elicits the immediate state of perceived sleepiness on a scale of 1-7 (fully alert to struggling to remain awake) and thus can sample sleepiness at different time points (e.g., times of the day or over a period of days).[112] The strength of the scale is in monitoring changes in subjective sleepiness over short time periods and assessing patterns.[112]

5.6.4.2 Sleep-Wake Activity Inventory (SWAI)

The SWAI is a 59 item scale of 6 factors. The EDS factor comprises 12 items and can be used alone.[113] The validity and the psychometric properties of the SWAI have been carefully investigated.[114] The EDS subscale of SWAI correlates with MSLT scores and, in sleep apnea patients, the EDS subscale score has been shown to decrease with 6 weeks of CPAP treatment.

5.6.4.3 Epworth Sleepiness Scale (ESS)

The ESS elicits the likelihood of falling asleep in 8 different situations.[115] Validation studies have shown that the ESS discriminates severe OSA patients and narcoleptics from normal controls and, based on a sample of 87 medical students, was reliable over a 5 month period.[115-117] Although the scale contains both very passive situations (lying down to rest, watching TV) and very active situations (conversation), factor analysis has revealed only a single factor.[116]

5.6.5 Recommendations for Reporting Severity of Daytime Sleepiness

Current practice parameters[108] stress the need to obtain a subjective assessment of EDS in the clinical evaluation; however no specific tool can be recommended at this time. Ascertainment of daytime sleepiness by any method should be considered clinically important. Self reporting should incorporate descriptions of sleep behavior in active versus passive situations, and the degree to which sleepiness impairs social or occupational function. Although any of these measures discussed above would seem useful to classify a patient with at least a minimum criteria of daytime sleepiness, none of the current methods can be recommended to place individuals into different severity categories, as recommended in 4.1.3

5.6.6 Recommendations for further methodologic research

Standardization of sleepiness measures and further validation and refinement of current tests and questionnaires is needed before severity criteria (4.1.3) can be evidence-based. Undoubtedly there will be gender, age, and ethnicity differences in many aspects of the currently used measures, especially in the meaning of "sleepiness", in familiarity of the active and passive situations in which sleepiness is assessed (e.g., meetings, theater, driving), and in the degree to which sleepiness would interfere with life (lack of employment versus demanding job).

6.0 NOTES

The sections that refer to these notes are listed in square parentheses.

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Table 4. Recommendations and Quality of Evidence for Methods of Measuring Breathing Disorders During Sleep

Event/Condition	Method of Measurement	Recommendation Grade*	Quality of Evidence†
Obstructive Hypopnea/ Apnea	1. Pneumotachometer	A	1
	2. Nasal Pressure	B	2a; 2b
A hyponea/apnea is assumed	3. RIP — sum channel 50% decrease from baseline	B	2a; 2b
to be obstructive unless criteria	4. RIP — dual channel 50% decrease from baseline	C	3
for a central event are met	5. RIP — single channel	C	3
	6. Piezo sensors, Strain gauges, Thoracic impedance	C	3
	7. Thermal sensors	D	3
	8. Expired CO_2	D	3
	9. Measurement of breathing <50% (but clear) decrease from baseline with 3% O_2 desaturation or arousal	B	2a
Respiratory Effort	1. Esophageal pressure	A	1
Related Arousal	2. Nasal pressure	C	3
	3. Supraglottic pressure	C	3
	4. Diaphragm EMG	D	3
Central Hypopnea/ Apnea	1. Esophageal pressure/Pneumotachometer	A	1
	2. RIP	C	3
To distinguish from an	3. Diaphragm EMG	C	3
obstructive apnea	4. Oronasal airflow (Thermal, Expired CO_2) sensors	D	3
	5. Piezo sensors and Strain gauges	D	2a
Cheyne-Stokes Breathing	1. Esophageal pressure/ Pneumotachometer	A	1
	2. RIP	B	3
	3. Diaphragm EMG	C	3
	4. Oronasal airflow (Thermal, Expired CO2) sensors	D	3
	5. Oximetry	D	3
Sleep Hypoventilation	1. $PaCO_2$	A	1
	2. O_2 desaturation (unexplained)	B	3
	3. $TcCO_2$	C	1
	4. Calibrated RIP	D	2a
	5. Expired end tidal CO_2	D	1

* See Table 2
† See Table 3

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1. For the present the task force felt there was insufficient clinical, pathophysiological or outcome data to justify classification of respiratory effort related arousals as a discrete syndrome, such as the Upper Airway Resistance Syndrome. Therefore the task force proposes to include RERAs as part of the definition of the OSAHS syndrome. This is suggested on the basis that RERAs and hypopneas share a similar pathophysiologic mechanism and it would therefore be reasonable to use the same frequency of these events as apneas and hypopneas in defining the OSAH syndrome. Given that few laboratories make routine measurements of esophageal pressure, it is unlikely that many patients will be diagnosed with OSAHS on the basis of RERAs. [Sections 3.0, 4.1.2]

2. The 10 second criterion is used by convention. The task force recognized that although there was no absolute justification for this cutoff, it is a standard that has been used since the first description of sleep apnea, it is what all current research and clinical studies use, and there is no data available indicating that a different criterion is superior. [Sections 4.1.2.1, 4.1.2.2, 4.2.2.1]

3. This is best quantified by a validated quality of life instrument such as the SF-36.[118] Although several instruments have been studied in sleep apnea patients the SF-36 has been the instrument most widely used and it has shown consistent findings of low scores especially in the Vitality/Energy, Role-Emotional, Mental Health, and Social Functioning domains.[119,120] These domains have also been shown to improve with CPAP treatment which suggests their validity for assessing quality of life in OSAHS.[120] [Sections 4.1.4, 4.4.4]

4. There is no research data which would justify an arbitrary cutoff of 5 apneas and hypopneas per hour of sleep. It was selected to reflect the cutoff chosen for the OSAHS [Sections 4.1.2, 4.2.2]

5. May be associated with awake hypercapnia due to underlying lung disease. In the absence of $PaCO_2$ measurement during sleep an additional component of sleep hypoventilation cannot be assumed. [Section 4.4.5]

6. There was an error in the reported methods used in this study regarding calibration of the RIP. An uncalibrated addition of chest and abdominal signal was used in the final (manual) analysis as the sum signal rather than the calibrated, computer held data (NJ Douglas, personal communication, 1997). [Section 5.1.2.2]

7. In the original description of the Wisconsin Sleep Cohort[19] the criteria for scoring hypopnea events was mistakenly stated as a decrease in respiratory airflow (sum of respiratory and abdominal movement is correct) in combination with a ≥4% drop in O_2 saturation. The Sleep Cohort Study has consistently used the more rigorous definition of a clear decrease in the amplitude of calibrated RIP associated with a ≥4% drop in O_2 saturation. [Section 5.1.2.8]

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APPENDIX 1. RECOMMENDATIONS OF METHODS FOR DISTINGUISHING BETWEEN APNEAS AND HYPOPNEAS

In routine clinical practice it is not necessary to differentiate apneas from hypopneas (see 5.1), and methods used to identify hypopneas are considered fully adequate for identifying obstructive apnea events as well. However, for some applications such as research studies that aim to differentiate risk profiles or elucidate pathogenesis, it may be useful to score and report apneas and hypopneas separately. This section focuses on methodology for distinguishing between obstructive apneas and hypopneas in such situations.

A.1 Reference Standard Measurement: Absence of air flow as detected by pneumotachometer

A pneumotachometer used with a tight fitting face mask can accurately measure total oronasal airflow. Reductions in airflow (hypopnea) can then be distinguished from an absence of airflow (apnea).

A.2.1 Thermal Sensor

Recommendation - A
Evidence - 2a

These sensors function by detecting changes in the temperature of expired air. The sensor should be positioned such that air expired nasally and through the mouth can be sampled.

There are limited published data that document the accuracy and precision of these devices. In laboratory models that have compared thermocouples and thermistors to a pneumotachograph, the signals from the thermal sensors have been shown to be non-linearly related to actual airflow, while generally resulting in overestimation of ventilation.[49] The task force felt consensus was that although these sensors are unacceptable for detecting hypopneas they would likely have relatively low false negative and false positive rates for apnea detection if they are properly positioned.

A.2.2 Expired CO_2

Recommendation - A
Evidence - 3

These sensors function by detecting CO_2 in expired air. They are not as widely used as thermal sensors. Like thermal sensors, CO_2 sensors should be positioned to sample both nasal and mouth breathing. As with thermal sensors there are no published papers documenting the accuracy and precision of these devices in the detection of apneas.

A.2.3 Uncalibrated Respiratory Inductance Plethysmography (RIP)

Recommendation - D
Evidence - 2a

Calibrated RIP may be able to distinguish apneas from hypopneas when the sum signal is flat, however this has not been systematically investigated. In clinical practice it is uncommon to use calibrated RIP and it is unlikely that uncalibrated plethysmography would accurately distinguish apneas from hypopneas.[49]

A.2.4 Nasal Pressure

Recommendation - D
Evidence - 2a

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Recently, nasal prongs attached to a pneumotachograph have been used to measure airflow semi-quantitatively. Characterization of airflow is based on detecting pressure changes associated with turbulence at the nostrils.[43] This technology may be useful for identifying hypopneas (see 5.1) but appears inappropriate for distinguishing apneas from hypopneas.

This technology may be subject to false positives as a result of nasal obstruction or mouth breathing. The signals obtained using this technology provide non-linear estimates of airflow, resulting in a net under-estimation of ventilation.[43,121,122] This may result in classification of as many as 25% of hypopneas as apneas.[122] Improved estimation of airflow may be obtained by applying mathematical adjustments to nasal prong measurements.[43] However, for circumstances where distinguishing apneas from hypopneas are important, use of nasal prong data may provide misleading information.

A.2.5 Piezo Sensors and Strain Gauges.

Recommendation - D
Evidence - 3

There are no published reports that address the ability of either piezo sensors or strain gauges to distinguish apneas from hypopneas. The task force consensus was that these sensors did not provide data that would be linearly related to flow and as such would have no utility.

A.3 Recommendations for Further Methodologic Research

Further research into technologies that distinguish between apneas and hypopneas is probably not warranted. Available data have not demonstrated the importance of distinguishing between these two events for predicting short or long term outcomes.

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