Monitoring respiration during sleep

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Sleep-related breathing disorders

The sleep-related breathing disorders have been categorized in various ways. The most basic schema divides them into obstructive or central apneic events. An American Academy of Sleep Medicine (AASM) Task Force Report published in 1999 defined four separate syndromes associated with abnormal respiratory events during sleep among adults, namely, obstructive sleep apnea-hypopnea syndrome (OSAHS), central sleep apnea-hypopnea syndrome, Cheyne-Stokes breathing syndrome, and sleep hypventilation syndrome [1]. In this classification, the upper airway resistance syndrome was not regarded as a distinct syndrome; instead, respiratory event-related arousals (RERAs) were considered part of the syndrome of OSAHS.

Obstructive sleep apnea-hypopnea syndrome

OSAHS is characterized by repetitive reduction or cessation of airflow during sleep caused by partial or complete upper airway occlusion in the presence of respiratory efforts. Mixed apnea, in which an initial period of apnea caused by an absence of respiratory efforts precedes upper airway obstruction, is included in this syndrome. These events are typically accompanied by oxygen desaturation, arousals, and sleep disruption.

Apnea is characterized by the cessation of airflow for 10 seconds or longer. Although there is almost universal consensus regarding the definition of apnea in adults, the presence of hypopnea continues to be identified using various criteria, including (1) a 50% reduction in airflow accompanied by a 4% fall in oxygen saturation (SaO₂) or an arousal, (2) a 50% reduction in airflow accompanied by any fall in SaO₂, or (3) any reduction in airflow with or without oxygen desaturation or arousal [2].

The criteria used for scoring hypopneas influence the diagnosis of OSAHS and the rating of its severity. Different scoring criteria for hypopneas may result in varying apnea-hypopnea indices [3]. Interpretation of polysomnographic records ideally should include a description of the scoring method used to derive hypopneas.

The sum of apneas and hypopneas divided by the total sleep time is commonly referred to as the apnea-hypopnea index. The respiratory disturbance index (RDI) is the sum of apneas, hypopneas, and RERAs divided by the total sleep time.

Estimates of the severity of sleep-disordered breathing depend on the approach to measuring RDI. Redline et al examined the relationships among RDIs defined by different definitions of apneas and hypopneas in 5046 participants in the Sleep Heart Health Study who underwent overnight unattended 12-channel polysomnography. The correlation between RDIs based on various definitions ranged from 0.99 to 0.68, and the magnitude of the median RDI varied from 29.3 when it was based on events identified on the basis of flow or volume amplitude criteria alone to 2 for an RDI that required a 5% oxygen desaturation with events [4].
It is generally not necessary to distinguish apneas from hypopneas in routine clinical care, and often the two respiratory events are scored and reported together. The diagnostic criteria for apneas and hypopneas recommended by the AASM Task Force include a reduction (>50%) in the amplitude of breathing from baseline during sleep or a reduction (<50%) in the amplitude of breathing from baseline during sleep associated with either an oxygen desaturation (>3%) or an arousal plus an event duration of at least 10 seconds [1]. RERAs, which do not fulfill the criteria for either apnea or hypopnea, consist of increasing respiratory efforts that last 10 seconds or longer and culminate in an arousal or a progressively more negative esophageal pressure preceding a change in esophageal pressure to a less negative level.

The reference standard for measuring an obstructive apnea-hypopnea is a reduction in total oronasal airflow detected by a pneumotachometer placed in a well-fitted facemask [1]. Other methods used to identify obstructive apnea-hypopneas include measurement of nasal pressure, respiratory inductance plethysmography (RIP), piezo sensors, strain gauges, thoracic impedance, thermal sensors, and expired carbon dioxide (CO₂). Whereas measurement techniques that identify apneas also are able to detect hypopneas, methods that measure hypopneas may not necessarily be adequate in identifying apneic events. The reference standard for identifying a RERA is the measurement of esophageal pressure [1]. RERAs also can be detected using measurements of nasal pressure and surface diaphragmatic electromyography.

The demonstration of five or more obstructive apneas-hypopneas or RERAs per hour of sleep during an overnight study, plus excessive daytime sleepiness (that is not caused by other factors) or two or more of the following manifestations, including choking or gasping during sleep, recurrent awakenings from sleep, unrefreshing sleep, daytime fatigue, or impaired concentration, establishes the diagnosis of OSAHS [1].

Central sleep apnea-hypopnea syndrome

This syndrome is characterized by repetitive episodes of sleep-related apnea unaccompanied by upper airway obstruction. Each respiratory event consists of reduced airflow, 10 seconds or longer in duration, associated with a reduction in esophageal pressure excursions from baseline levels and often with oxygen desaturation and arousals.

The diagnostic criteria for central sleep apnea-hypopnea syndrome consist of (1) excessive daytime sleepiness or frequent arousals/awakenings, and (2) at least five central apnea-hypopneas per hour of sleep during an overnight study, and (3) awake arterial carbon dioxide tension (PaCO₂) of less than 45 mm Hg [1].

Esophageal pressure monitoring is the reference standard measurement of central apnea-hypopneas [1]. Other methods, such as RIP, surface diaphragmatic electromyography, thermal sensors, expired CO₂, piezo sensors and strain gauges, are relatively insensitive in identifying these events.

Cheyne-Stokes breathing syndrome

In this syndrome, cyclical waxing and waning of respiration develops, with central apnea or hypopnea alternating with hyperpnea. Transient arousals that occur at the crest of hyperpnea may lead to sleep fragmentation and excessive somnolence.

The reference standards of measuring airflow and respiratory effort are pneumotachometry and esophageal pressure monitoring, respectively [1]. Other techniques for detecting Cheyne-Stokes breathing include RIP, surface diaphragmatic electromyography, oronasal airflow monitoring, and oximetry. Cheyne-Stokes breathing syndrome is diagnosed based on the following criteria: (1) presence of congestive heart failure or cerebral neurologic disorders, (2) three or more consecutive cycles of respiratory irregularity characterized by crescendo-decrescendo amplitude of breathing lasting at least 10 consecutive minutes, and (3) five or more central apnea-hypopneas per hour of sleep [1].

Sleep hypoventilation syndrome

Persons with sleep hypoventilation syndrome may have oxygen desaturation and hypercapnia during sleep unrelated to distinct periods of apnea-hypopnea. Periods of hypoventilation are more frequent and severe during rapid eye movement sleep than in non–rapid eye movement sleep. PaCO₂ monitoring is the reference standard measurement for identifying sleep hypoventilation [1]. Continuous oximetry (demonstrating a decline in SaO₂ without accompanying respiratory events), transcutaneous carbon dioxide (PtCO₂) monitoring, calibrated RIP (showing reduced tidal volume and minute ventilation), and end-tidal carbon dioxide (PetCO₂) measurements also have been used to monitor sleep hypoventilation. The diagnosis of sleep hypoventilation syndrome is based on the presence of cor pulmonale, pulmonary hyper-
tension, excessive somnolence not secondary to other factors, erythrocytosis or awake PaCO₂ of more than 45 mm Hg, and an increase in PaCO₂ during sleep by more than 10 mm Hg compared with levels during wakefulness or sleep-related oxygen desaturation not caused by apnea-hypopnea [1].

**Monitoring respiration during sleep**

Accurate monitoring of respiration during sleep, including measurements of airflow, respiratory effort, oxygenation, and ventilation, is indispensable in identifying sleep-disordered breathing.

**Measurement of respiratory effort**

Measurement of respiratory effort using either esophageal pressure monitoring or surface diaphragmatic electromyography is vital in distinguishing central from obstructive apneas.

**Esophageal pressure**

Changes in pleural pressure accompany respiratory effort. Esophageal pressure monitoring during polysomnography, using either esophageal balloons or newer catheter transducers, is considered the reference standard for detecting respiratory effort during sleep and is a direct measure of respiratory load [1]. This method requires a transnasal insertion of an esophageal catheter with a pressure transducer placed on its tip after topical anesthesia of the nares and pharynx. During episodes of RERAs in patients with upper airway resistance syndrome, esophageal pressures become increasingly more negative immediately preceding an arousal, followed by a rapid return to baseline levels [1]. Virkkula et al reported that esophageal pressure monitoring improved the diagnostic value of limited polygraphic recording of oxygen saturation, respiratory and leg movements, airflow, body position, and snoring in detecting sleep-disordered breathing [5].

Transnasal insertion of esophageal catheters in sleep studies may increase ipsilateral nasal resistance, as measured by anterior rhinomanometry, but does not affect combined nasal resistance [6]. Changes in nasal pressure and airflow during esophageal pressure monitoring may be particularly relevant in persons with already compromised nasal airflow. The amount of apneas and arousals has been shown to increase with nasal airflow obstruction. The use of nasoesophageal catheters is generally associated with only minimal changes in sleep architecture [5]. Patient compliance with esophageal catheter is generally good [5].

**Surface diaphragmatic electromyography**

Although the presence of respiratory efforts may be inferred by analysis of signal tracings derived from electrodes placed on the chest wall during polysomnography, surface diaphragmatic electromyography by itself is seldom helpful in detecting RERAs or central apnea-hypopneas [1].

**Measurement of airflow**

Airflow during sleep can be measured either directly or indirectly. The only method that measures airflow directly is pneumotachography. Thermal sensors and PetCO₂ monitors detect changes in the thermal and chemical characteristics of inspired ambient air and expired air originating from the airways; both methods provide only an indirect estimate of airflow [7].

Although indirect methods of measuring airflow can detect episodes of apnea reliably, they are less consistent in identifying hypopneas. Simultaneous measurement of lung volume or effort and thermal or PetCO₂ sensors is required to distinguish among central apneas, obstructive apneas, and a prolonged inspiration [7].

**Pneumotachometer**

A pneumotachometer, attached to a well-fitted facemask, can measure total oronasal airflow by detecting changes in pressure between inspiration and expiration and is the reference standard for measuring airflow [1]. Patient discomfort from a tightly fitting facemask may disturb sleep and limit its use in clinical sleep studies.

**Nasal pressure**

Nasal airflow can be measured quantitatively and directly with a pneumotachograph that detects changes in nasal pressure during respiration. Nasal airway pressure decreases during inspiration and increases during expiration. The fluctuations produced on the transducer signals are proportional to flow [8]. The device consists of a standard oxygen nasal cannula connected to a pressure transducer and placed in the nares.

The shape and amplitude of signals obtained from a nasal cannula are comparable to those from a
A plateau on the inspiratory flow signal is associated with increased upper airway resistance and airflow limitation. In one study, airway resistance was increased for breaths with flattened or intermediate inspiratory flow signal contours compared with breaths with normal flow contours [8].

Measurement of pressure by nasal prongs is superior to the use of thermistors in detecting respiratory events during sleep studies [9]. Nasal cannula/pressure sensors may recognize additional events characterized by flow limitation that are missed by thermistors [10]. Nasal pressure monitoring is not recommended for persons who are predominantly mouth breathers or who have nasal obstruction [7,11]. In persons with narrow nares or a deviated septum, nasal prongs used to assess nasal flow during sleep can increase nasal airflow resistance—as estimated by posterior rhinomanometry—and possibly alter the diagnosis of OSAHS and its severity [12]. Nasal prongs that partly occlude the nasal passages can cause sleep breathing disorders associated with brief arousals. Thurnheer et al observed that compared with facemask pneumotachography, nasal cannula pressure recordings provided accurate clinical assessment of ventilation during sleep even in patients who reported nasal obstruction [13].

Thermal sensors

Thermal sensors (thermistors or thermocouples) afford an indirect and semiquantitative measurement of airflow. These devices are placed over the nose and mouth and infer airflow by sensing differences in the temperature of the warmer expired air and the cooler inhaled ambient air. The flow signal generated is related directly to the sensor temperature and indirectly to airflow. Unfortunately, temperature changes of respiratory air often bear little correlation to airflow. The flow signal also is influenced by the pattern of airflow and the placement of the sensor in relation to the nostril. Even minor displacements of the thermal sensors or alternations in the proportion of nasal and oral breathing relative to the sensor position can lead to large changes in signal amplitude [14].

Although temperature-sensing receptors can detect apneas reliably, they are less accurate in identifying hypopnea [10]. Farre et al noted that thermal sensors were imprecise in monitoring airflow and, when a reduction in thermal sensor signal is used to quantify hypopneas, they tend to underestimate hypopneic events [15]. Thermistors do not allow the detection of inspiratory flow limitation, which is suggestive of upper airway narrowing.

Oronasal thermistors are typically located at the upper lip; in this location, thermistors may be unable to differentiate between high and low rates of airflow and detect hypopneas. Akre et al introduced the use of internal thermistors to measure airflow in the pharynx. They reported that this method was more sensitive than external thermistors in detecting minor changes in airflow and hypopneas [16,17]. In awake, normal subjects, the reliability of internal thermistors in diagnosing hypopneas is comparable to that of pneumotachography [18].

In summary, signals obtained from thermocouples and thermistors provide only qualitative data regarding airflow, and as a rule, thermal sensors are unable to identify reliably the presence of hypopnea and cannot distinguish central from obstructive apnea-hypopneas [1].

Expired carbon dioxide

Ambient air contains negligible amounts of CO2 compared with expired air from the lungs, which has a higher concentration of CO2. A qualitative measure of airflow can be obtained using infrared analyzers of expired CO2 placed in front of the nose and mouth. An advantage of PetCO2 monitoring over thermal sensing techniques is its ability to infer the occurrence of hypoventilation by a rising PetCO2 level. Minute fluctuations in lung volume that accompany each heart beat also may be transmitted to the sensor via a patent upper airway during central apneas [7]. These fluctuations may appear as cardiac oscillations in the CO2 tracings, further corroborating the diagnosis of central apneas.

Tracheal sound recording

Tracheal sound recordings, made by using a stethoscope head taped over the manubrium sternum and air-coupled to a microphone, have been proposed as a method of detecting and monitoring airflow. This method is limited by interference from environmental noise [19].

Strain gauges

Rib cage and abdominal excursions can be measured by placing length-sensitive strain gauges below the axilla and at the level of the umbilicus, respectively [20]. Respiratory movements can be detected by a single uncalibrated abdominal or chest gauge. Calibration of the rib cage and abdominal gauges against another volume-measuring device is required to measure volume changes quantitatively. The
summed rib cage-abdominal volume signals do not distinguish central events (no net volume change caused by absence of respiratory effort) from obstructive sleep apnea (no net volume change caused by rib cage-abdominal paradox). Loss of tone of the diaphragm or the accessory respiratory muscles also can lead to paradoxical motion of the rib cage and abdomen [7]. Esophageal pressure monitoring may be needed to verify respiratory efforts whenever most apneas detected by strain gauges appear central in origin [20].

Displacement of the strain gauges during the monitoring period because of changes in sleep position or body movements influences signal quality [20]. Accuracy of measurements is affected by overstretching or understretching of the gauges and alterations in muscle tone during sleep [7].

Respiratory inductance plethysmography

Respiratory inductance plethysmography (RIP) can be used to measure changes semi-quantitatively in chest and abdominal volume during respiration. Transducers are placed around the chest and abdomen to monitor changes in the cross-sectional area of the respective body compartments as reflected by changes in inductance (resistance to change in flow of current) of the transducers [7]. RIP is based on the principle of a two-compartment model of thoracoabdominal wall movement during respiration [21]. With a closed glottis, the sum of chest and abdominal volume is fixed, and any increase or loss of volume of the rib cage is accompanied by a simultaneous, equal but opposite change in volume of the abdomen [22]. The sum of the signals from calibrated chest and abdominal sensors can estimate tidal volume and respiratory pattern during sleep but cannot provide data regarding airflow [11].

Thoracoabdominal asynchrony during breathing is currently most commonly identified by visual analysis of records. Brown et al described a novel automated analysis approach using a recursive linear regression to identify synchrony or asynchrony between ribcage and abdominal movements during breathing in 15 infants [23]. Paradoxical ribcage motion also can be assessed by measuring thoracic delay based on the degree to which peaks in ribcage and abdominal signals are synchronized in time [23].

Hypopneas could be scored reproducibly using RIP to monitor thoracoabdominal movement with or without a simultaneous flow sensor signal [24]. Hypopnea is scored if there is a at least a 50% reduction of RIP sum from baseline of either calibrated or uncalibrated signals; at least a 50% reduction from baseline accompanied by either an arousal or an oxygen desaturation (≥ 3%) in either chest or abdominal signal (single channel) [1].

The accuracy of RIP in monitoring the volume and duration of respiration depends on its initial calibration and the constancy of calibration with body movements and changes in lung volumes [25]. Various procedures, such as the simultaneous equation method, isovolume maneuver method, and least squares regression method, can be used to calibrate RIP [25,26]. Displacements of the transducer bands or alterations in posture during sleep can lead to inaccuracies in measurements. Bands should be taped firmly to the skin to avoid slippage during overnight monitoring. Sleep-related thoracoabdominal distortion or movement asynchrony also can affect accuracy of RIP measurements during sleep [26,27].

Thoracic impedance

Thoracic impedance can be used to measure airflow qualitatively. Impedance varies with the relative amount of conductive materials (body fluids and tissue) and nonconductive air between a pair of electrodes placed at opposite sides of the thoracic cage. It decreases as the volume of conductive material increases in proportion to air and vice versa. The volume of air contained within the thoracic cage during the different phases of respiration can be estimated based on changes in recorded impedance [7].

Measurement of snoring intensity

Another method that has been used to measure airflow is measurement of snoring intensity. One study demonstrated a linear correlation, albeit weak, between snoring intensity and respiratory effort and flow limitation during sleep [28].

Piezo sensors

Piezo sensors can monitor changes in airflow qualitatively but cannot distinguish central apnea-hypopneas from obstructive respiratory events [1].

Magnetometers

Respiratory magnetometer recordings of chest and abdominal motion have been shown to be able to distinguish between obstructive and central apneic
events by differences in patterns of motion (i.e., paradoxical motion of the rib cage and abdomen with obstructive events) [29]. The recordings also can be used to monitor changes in body position during the sleep study.

**Canopy with a neck seal**

The use of a canopy ventilation monitor to measure ventilation quantitatively during sleep has been described [30]. The device directly measures gas flow using a pneumotachograph and consists of a rigid canopy fitted over the head. It is sealed at the neck, which creates an airtight enclosure through which a continuous flow of air or oxygen is provided. Inflow of gas is kept equal to outflow. Airflow is measured as respiration alters the flow in and out of the canopy. Canopy ventilation monitoring has a reported accuracy of approximately 92% in measuring tidal volume [30].

**Flow-volume loop analysis**

The presence of airway obstruction during wakefulness and sleep can be inferred by analyzing abnormalities of the flow-volume loop. Flow limitation and an elevated upper airway resistance are suggested by the presence of a plateau (normally rounded) on the contour of the inspiratory flow tracing obtained during continuous positive pressure (CPAP) therapy for OSAHS. In one study, breath-by-breath analysis of the flow-volume curve of a tidal breath was accurate in identifying inspiratory flow limitation during sleep in persons with OSAHS on CPAP therapy [31]. Inspiratory flow limitation was defined by the presence of an inspiratory plateau or reduction in inspiratory flow independent of any increase in inspiratory efforts.

**Cardiac oscillometry**

Small oscillations at cardiac frequency may be appreciated in the airflow signal tracing during episodes of central apnea. These cardiogenic oscillations are believed to be related to persistence of airway patency possibly coupled with relaxation of the thoracic muscles during central apneas [32].

**Air mattress**

Chow et al described the use of an air mattress system that consists of multiple air compartments to monitor noninvasively thoracic and abdominal movements separately. The sensitivity and accuracy rates of the air mattress for detecting hypopnoeas were above 90% compared with respiratory inductive plethysmography [33].

**Measurement of oxygenation and ventilation**

Oxygenation and ventilation change rapidly during sleep in patients with sleep-disordered breathing. To be accurate and reliable, methods to assess oxygenation and ventilation must be capable of rapid and repetitive measurements. Direct measurements of arterial oxygen tension (PaO₂), arterial carbon dioxide tension (PaCO₂), and SaO₂ via arterial blood sampling are more accurate than estimates derived from noninvasive methods such as pulse oximetry, transcutaneous oxygen tension (PtcO₂) measurement, transcutaneous carbon dioxide tension (PtcCO₂) measurement, or airway CO₂ (PetCO₂) monitoring. Arterial blood gas sampling provides only a static measure of oxygenation and ventilation rather than a continuous monitoring, however. Repetitive sampling of arterial blood during sleep studies is painful, time consuming, inconvenient, expensive, and intrusive of sleep and is associated with more complications than noninvasive assessments.

**Pulse oximetry**

With pulse oximetry, a pulsating vascular bed (e.g., earlobe or fingertip) is placed between a two-wavelength light source and a sensor. This arrangement is designed to eliminate any artifact that might originate from absorption of light by venous blood or tissue [34].

Pulse oximeters are used routinely during overnight polysomnography to monitor SaO₂. They are easy to use, portable, relatively inexpensive, readily available, noninvasive, respond rapidly to changes in SaO₂, and allow continuous monitoring of SaO₂ [7].

Several factors influence the accuracy and reliability of pulse oximetry. Pulse oximetry response time can be affected by changes in heart rate and circulation time. Altering the pulse oximeter response time influences the accuracy of pulse oximeters in measuring changes in SaO₂. For instance, SaO₂ recordings may be inaccurate if the oximeter response time approximates the duration of oxygen desaturation events. In one study that involved subjects with severe OSAHS, increasing the pulse oximeter averaging time from 3 seconds to 12 and 21 seconds resulted in significant differences in the measured SaO₂, with underestimation of oxygen desaturation by up to 60% [35].
SaO2 measurement and response characteristics using pulse oximetry also vary with sensor location (e.g., earlobe or fingertip) and type [36]. Finally, sensitivity of pulse oximetry is greater with shorter sampling intervals, and the least filtering to achieve the most rapid response is recommended [7,37].

Several factors limit the use of oximetry in the evaluation of persons with sleep-disordered breathing. Oximetry alone is inadequate in persons without oxygen desaturation [37]. The presence of dyshemoglobin species, such as carboxyhemoglobin or methemoglobin, produces errors in measurement because of its reliance on only two light wavelengths [7]. Reduced skin perfusion caused by hypothermia, hypotension, or vasoconstriction and by poor sensor attachment may alter signal amplitude [14]. Finally, oximetry readings may overestimate low oxygen saturation values [38].

As a screening test for OSAHS, nocturnal pulse oximetry has a reported sensitivity rate of 69% and a specificity rate of 97%. Accuracy was decreased in persons who had higher awake baseline SaO2, were less overweight, and had milder disease [39]. Yamashiro and Kryger noted that nocturnal oximetry may not be able to detect breathing disorders during sleep with sufficient sensitivity and specificity and is ineffective in identifying other disorders of sleep [40]. In another study that compared clinical assessment, unsupervised home oximetry, and formal polysomnography in the diagnosis of OSAHS, clinical assessment was superior to home oximetry analyzed by counting the number of recorded arterial oxygen desaturations [41].

Epstein et al compared polysomnography to two patterns of oxyhemoglobin desaturation used as a method of screening for OSAHS: (1) a “deep” pattern that consisted of more than 4% fall in SaO2 to less than or equal to 90% and (2) a “fluctuating” pattern that consisted of repetitive, brief drops in SaO2 [42]. As screening tools for sleep-disordered breathing, the “deep” pattern had greater specificity and positive predictive value and the “fluctuating” pattern had a greater sensitivity and negative predictive value. For mild disease, screening nocturnal oximetry using the “fluctuating” pattern is less sensitive compared with polysomnography, with 61% of patients with abnormal polysomnographic studies having normal oximetry results [42].

Transcutaneous oxygen monitoring

Oxygen tension at the skin surface (PtcO2), which is measured using a modified Clark electrode, is influenced by cutaneous perfusion, temperature, and metabolism. The application of PtcO2 monitoring during adult polysomnography is limited by the variable relationship between PaO2 and PtcO2 and its slow response time that fails to mirror rapid changes in PaO2. It requires meticulous skin preparation. Blood flow to the skin can be increased by local application of heat, with periodic site changes every 4 to 6 hours to prevent cutaneous thermal injury [37]. A delay in recording in the warm-up period after site changes is expected [37].

Transcutaneous carbon dioxide

Transcutaneous carbon dioxide (PtcCO2) refers to the CO2 tension at the epidermal surface. It can be monitored noninvasively and continuously during sleep using a silver chloride electrode or an infrared capnometer. PtcCO2 monitoring may provide useful information during pediatric polysomnography because pediatric OSAHS is associated with partial airway obstruction, alveolar hypoventilation, and hypercarbia. PtcCO2 monitoring is most commonly used in neonates. It requires meticulous skin preparation and arterial blood gas sampling for calibration [37].

Among adults, PtcCO2 often differs significantly from a simultaneously obtained PaCO2 [1,43]. Routine PtcCO2 monitoring has minimal clinical use during adult polysomnography. Its slow response time makes it unsuitable for monitoring blood gas tensions during sleep, in which rapid and short-lasting changes can occur [7]. PtcCO2 monitoring may be of some use in adults with waking hypercapnia or suspected sleep-related alveolar hypoventilation.

Expired end tidal carbon dioxide

Airway carbon dioxide (PetCO2) measured at the end of a complete expiration is related to PaCO2. PetCO2 can be monitored continuously during polysomnography using infrared spectrophotometers or respiratory mass spectrometers. PetCO2 measurements are affected by conditions that alter the relationships among ventilation, perfusion, and PaCO2 [38]. PetCO2 may underestimate PaCO2 when dead space to tidal volume ratio is increased during sleep because of a reduction in tidal volume. PetCO2 measurements using facemasks or nasal cannula or during nasal CPAP ventilation may not reflect PaCO2 reliably because of gas dilution with room air or continuous gas leakage via the CPAP mask, respectively. Hypoventilation, mouth breathing, or concomitant use of supplemental oxygen therapy also can give rise to inaccuracies in measurement [37,43].
In one study, neither PetCO$_2$ nor PtcCO$_2$ accurately or consistently reflected simultaneously recorded PaCO$_2$ values during polysomnography in persons who were spontaneously breathing room air, receiving supplemental oxygen given via nasal cannula, or receiving nocturnal positive pressure ventilatory assistance [43].

Newer approaches

Pulse transit time analysis

Blood pressure fluctuates during sleep in persons with OSA. Blood pressure transiently increases during arousals from sleep and falls during inspiration. Davies et al reported that the degree of inspiratory fall in blood pressure progressively increased from normal sleep, through snoring, to obstructive respiratory events. The frequency of arousal-related increases in blood pressure also rose during obstructive apnea and during snoring accompanied by arousals [44].

Pulse transit time (PTT) is the transmission time for the arterial pulse pressure wave to travel from the aortic valve to the periphery. It is measured using electrocardiography as the interval between the R-wave and the subsequent pulse shock wave detected at the finger. PTT is typically approximately 250 milliseconds. The speed of the shock wave is affected by the stiffness of the arterial walls and blood pressure. PTT is inversely related to blood pressure: as blood pressure rises, PTT falls because of increases in arterial wall stiffness and pulse wave speed. PTT increases during inspiratory falls in blood pressure and decreases during arousal-induced increases in blood pressure [45].

With esophageal pressure as a reference, PTT has been reported to have high sensitivity and specificity rates in distinguishing between central and obstructive apnea-hypopnea [46]. Among persons with OSAHS, PTT studies also have been demonstrated to differentiate reliably between persons who require nasal CPAP and persons who do not [47].

Forced oscillation technique

Forced oscillation technique has been proposed as a method for detecting upper airway obstruction during sleep and titrating CPAP therapy [48–51]. This technique is a noninvasive measure of input impedance of the respiratory system that uses high-frequency pressure oscillation to the upper airway [49]. Forced oscillation techniques are able to partition reliably the airway component of respiratory impedance from that of lung tissue [50]. This technique does not require patient cooperation and may prove useful for assessing uncooperative patients.

Contrary to earlier concerns, Badia et al observed that the use of forced oscillation technique does not alter upper airway muscle tone or affect electroencephalographic variables [49]. This novel approach requires further standardization before it can be used in clinical sleep studies [50].

Steltner et al evaluated the performance of a new algorithm for automated detection and classification of apneas and hypopneas based on time series analysis of nasal mask pressure and a forced oscillation signal related to respiratory input impedance [52]. They noted no significant difference in the variability and discrepancy between automated analysis and visual analysis of standard polysomnographic signals.

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