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REVIEW ARTICLE/BRIEF REVIEW

Interpretation of sleep studies for patients with sleep-disordered breathing: What the anesthesiologist needs to know

Interprétation des études sur le sommeil pour les patients souffrant de troubles respiratoires du sommeil : ce que les anesthésiologistes doivent savoir

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Abstract *There is increased interest in the perioperative management of patients with sleep-disordered breathing (SDB). Anesthesiologists must distill information from clinical reports to make key decisions for optimizing perioperative care. A patient with SDB may present with a sleep study report at the time of surgery. Knowledge of the essential components of such a report can help the anesthesiologist evaluate the patient and optimize the perioperative management. In this narrative review, we describe how level I (i.e., laboratory-based) polysomnography (PSG) data are collected and scored using the recommended scoring guidelines, as well as the basic information and salient features of a typical PSG report relevant to the anesthesiologist. In addition, we briefly review the indications for sleep studies, including the types of laboratory-based studies, as well as the role and limitations of portable monitors (level II-IV studies) and examples of PSG reports in the clinical context.*

Résumé *La prise en charge périopératoire des patients souffrant de troubles respiratoires du sommeil (TRS) suscite un intérêt croissant. Les anesthésiologistes doivent extraire les informations issues de comptes rendus cliniques afin de prendre des décisions cruciales à l'optimisation des soins périopératoires. Il arrive qu'un patient atteint de TRS se présente avec un rapport d'étude du sommeil au moment de sa chirurgie. Une connaissance des composantes essentielles d'un tel rapport peut aider l'anesthésiologiste à évaluer le patient et optimiser sa prise en charge périopératoire. Dans ce compte rendu narratif, nous décrivons comment les données de polysomnographie (PSG) de niveau I (c.-à-d. réalisée en laboratoire) sont colligées et notées à l'aide des directives de notation recommandées, ainsi que les informations de base et les caractéristiques principales d'un rapport typique de PSG pertinentes à l'anesthésiologiste. En outre, nous passons brièvement en revue les indications des études sur le sommeil, notamment les types d'études de laboratoire, ainsi que le rôle et les limites des moniteurs portables (études de niveau II-IV) et présentons quelques exemples de rapports de PSG dans le contexte clinique.*

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Sleep-disordered breathing (SDB) comprises of a spectrum of disorders characterized by excessive abnormal breathing patterns during sleep. The mildest form is respiratory effort-related arousal, and the more severe forms range from apnea and hypopnea to gas-exchange abnormalities.[1] These disorders are prevalent in both the general and surgical populations.[2-5] Obstructive sleep apnea (OSA), the most common type of SDB, is associated with an increased

risk of perioperative morbidity and mortality.[6-12] Despite the evidence that OSA patients are at increased risk of complications, perioperative identification of suspected or diagnosed OSA remains suboptimal.[13]

A patient with SDB may present with habitual loud snoring, apneic episodes, and disturbed sleep, resulting in daytime hypersomnolence, fatigue, and reduced quality of life.[1] The gold standard for diagnosing SDB is the laboratory-based sleep study, or polysomnography (PSG).[14] A PSG report may also contain other useful information that allows further stratification of the disease severity and the risk it poses. Knowledge of essential components of a PSG report can help the anesthesiologist evaluate patients and optimize their perioperative care.

In this narrative review, we discuss the current literature and guidelines with the following objectives: (1) how a sleep study's raw data are collected and scored according to the American Academy of Sleep Medicine (AASM) Scoring Guidelines[15]; (2) collect the basic information and highlight the salient features of a typical sleep study report relevant to the anesthesiologist; (3) review the indications for sleep studies, differences between laboratory and home sleep studies, the roles and limitations of portable monitors, and examples of PSG reports in the clinical context.

Table 1 Clinical indications for polysomnography

Diagnostic	Evaluation of suspected: • Sleep disordered breathing (SDB) • Periodic limb movement disorder • Complicated parasomnia • Primary disorders of daytime hypersomnolence, such as narcolepsy
Therapeutic	• Evaluation of treatment effectiveness for SDB: positive airway pressure titration, dental appliances, upper airway surgery, positional and drug therapy • Split-night study (first half: diagnostic study to determine the nature and severity of SDB; second half: therapeutic trial)

Derived from *Kushida et al.*[14]

Clinical indications for sleep studies

A level I, or laboratory-based, PSG is commonly performed to diagnose SDB.[14] The International Classification of Sleep Disorder, 3rd Edition, subgrouped these conditions into OSA disorder, central sleep apnea (CSA) disorder, and sleep-related hypoventilation and hypoxemia disorders.[1] Table 1 summarizes the clinical indications for PSG.[14] We focused primarily on OSA, with a brief overview on CSA and obesity hypoventilation syndrome (OHS). Other indications for PSG that are not part of SDB are beyond the scope of this review.

Laboratory-based PSG, however, may be too resource- and time-intensive to perform.[16] To reduce the economic burden on health care systems and improve access to diagnostic testing, portable monitoring has been evaluated as an alternative to laboratory-based PSG.[16,17] Sleep apnea testing at home can be classified using the Sleep, Cardiovascular, Oximetry, Position, Effort, Respiratory parameters (SCOPER) classification, which incorporates new technologies available on the market.[17,18] This complex classification system, however, is beyond the scope of this review. A simpler, more traditional classification described by the AASM is still widely used. It categorizes unattended studies into types II-IV (Table 2).[17] Both the AASM and the Canadian Sleep Society recommend the use of type III monitors to diagnose moderate to severe OSA when there is a high index of clinical suspicion. The absence of significant medical comorbidities and other sleep disorders (i.e., CSA, periodic limb movement, insomnia, parasomnia, narcolepsy, and circadian rhythm disorder) are requisite criteria.[16,17] A negative study in the presence of a strong clinical suspicion for OSA cannot rule out the diagnosis, and a follow-up laboratory PSG is required.[17]

Table 2 Type of monitors for the evaluation of sleep-disordered breathing

Type I	In-laboratory, technologist attending, polysomnography (usual channels: EEG, EOG, chin EMG, ECG, airflow, respiratory effort and SpO_2; minimum of 7 channels as per AASM criteria)
Type II	Full, unattended polysomnography (minimum of 7 channels, as above)
Type III	Portable monitoring with three or more channels, including pulse oximetry and heart rate (minimum of 4 channels, including respiratory movement, airflow, heart rate, SpO_2 - per AASM)
Type IV	Portable monitoring with only one or two channels, including pulse oximetry

Derived from *Blackman et al.*[16] and *Kapur et al.*[17]

AASM = American Academy of Sleep Medicine; EEG = electroencephalography; ECG = electrocardiography; EMG = electromyography; EOG = electrooculography; PSG = polysomnography; SpO_2 = oxygen saturation

Monitoring channels and data acquisition

A level I study uses at least seven channels to record physiologic parameters during sleep (Table 3). A registered sleep technologist sets up, monitors, and verifies the accuracy of the recording throughout various sleep stages, including episodes of arousal.[15] Electrooculography (EOG) measures the resting potential between the cornea and retina. It is useful to identify slow eye movements associated with sleep onset and rapid eye movements (REMs) during sleep.[15] Electromyography (EMG) records electrical activity produced by skeletal muscles. Activity in the submentalis muscle (chin EMG) is monitored to assess upper airway muscle tone during sleep, which decreases with the onset of sleep and decreases even further during REM sleep. Activity in the anterior tibialis and brachioradialis muscles also provides additional information regarding limb movements.[15,19] Video monitoring or a body position sensor using actigraphy can be used to assess body position or any abnormal sleep behavior during the study. Electrocardiography continuously records heart activity during a sleep study.[15]

A minimum of three respiratory parameters are monitored during the study: airflow, thoracoabdominal effort, arterial oxygen saturation.[15] They are utilized to detect apnea, hypopnea, and respiratory effort-related arousal (RERA). Oronasal thermal flow sensors and nasal pressure transducers are used to measure airflow.[15] The former detects changes in temperature of the inspired (cooler) and expired (warmer) air. It is recommended for detecting apnea because of its main advantage of being able to detect both nasal and oral airflow. The latter is an acceptable alternative sensor for detecting apnea if the thermal signal is inadequate or lost due to poor placement. The nasal pressure transducer generates a signal that is proportional to the square of the airflow. Because of the nonlinear characteristic of the nasal pressure signal, low flow rates can be underestimated. A reduced amplitude on

Table 3 Summary of monitoring channels

Parameter	Sensors	Purpose
EEG	Minimum 3 channels: frontal, central, occipital with a reference electrode on mastoid process	Sleep staging
EOG	Electrodes are placed above and below the eyes at the level of the outer canthi with a mastoid reference site	Identify slow and rapid eye movements
EMG	Submentalis muscle (required)	Monitor upper airway muscle tone
	Anterior tibialis muscle	Detection of periodic limb movements
	or	Detection of suspected REM behaviour disorder
	Brachioradialis muscle	
Airflow	Oronasal thermal airflow	Recommended sensor for the detection of apnea and alternative sensor for hypopnea
Diagnostic	and	
study	Nasal pressure transducer	Recommended sensor for detection of hypopnea and RERAs and alternative sensor for apneas
Positive airway pressure (PAP) titration	PAP device flow signal	Detection of apnea and hypopnea
Respiratory effort	Esophageal manometry (gold standard)	Measures changes in intrathoracic pressure; not used frequently as it is invasive
	Respiratory inductance plethysmography (RIP) belt	Measures chest and abdominal excursion for differentiation of obstructive and central events
Arterial oxygen saturation	Pulse oximetry	Detection of oxygen desaturation
ECG	Modified lead II electrode	Heart rate and rhythm monitoring
Snoring	Microphone, piezoelectric sensor	Recording of snoring
(optional)		
Body position	Video recording or position sensors	Detection of supine-related apnea
(optional)		
Adequacy of ventilation (optional)	Transcutaneous/end-tidal CO_2	Detection of hypoventilation

Derived from *Berry et al.*[15]

EEG = electroencephalography; ECG = electrocardiography; EMG = electromyography; EOG = electrooculography

the pressure signal waveform seen during mouth breathing may result in misclassifying hypopneic events as apnea. The nasal pressure transducer, however, can detect subtle changes in airflow and is therefore recommended for the detecting hypopnea and RERAs in combination with the EEG signal to define arousal. As already stated, the oronasal thermal sensor can be used as an alternative.[20] During a positive airway pressure (PAP) titration study, a PAP device flow signal may be used to detect changes in airflow.[15]

Various methods for monitoring respiratory effort are available, but most sleep laboratory facilities utilize respiratory inductance plethysmography to monitor thoracoabdominal excursions.[15] It uses wire coils to generate and measure changes in the electromagnetic field associated with changes in the cross-sectional area of the chest/abdomen.[20] Effort is assumed in the presence of thoracoabdominal movements, allowing differentiation of central from obstructive apnea and hypopnea. Oxygenation is measured continuously using pulse oximetry. A signal average time of ≤ three seconds is required to document desaturation events accurately and quantify the severity of sleep-related breathing disorders.[15] Snoring can be recorded using an optional acoustic sensor (e.g., a snoring microphone or a piezoelectric sensor) placed on the patient's neck to detect vibration.[15] Adequacy of ventilation can be measured by capnography via transcutaneous or end-tidal carbon dioxide monitoring, although this step is considered optional.[15]

Portable monitors to evaluate SDB

Portable monitors are increasingly being used as alternatives to diagnostic tests for OSA and in research. Their use in a selected patient population following comprehensive assessment by a sleep specialist physician can be as accurate as laboratory-based PSG.[17] There are some limitations to the clinical utility of portable monitors, however. Level II-IV studies are unattended and are therefore prone to technical failure—e.g., via improper sensor application or sensor dislodgment—resulting in poor-quality data.[17] In addition, numerous portable monitors are available on the market, with considerable heterogeneity in terms of the parameters monitored and how it is accomplished. Surrogates to the accepted standard laboratory PSG may be used. For example, the apnea-hypopnea index (AHI) may be substituted for the oxygen desaturation index (ODI). Furthermore, sleep staging is not monitored with level III-IV studies, resulting in an inability to detect hypopneas that are associated only with cortical arousals and RERAs,

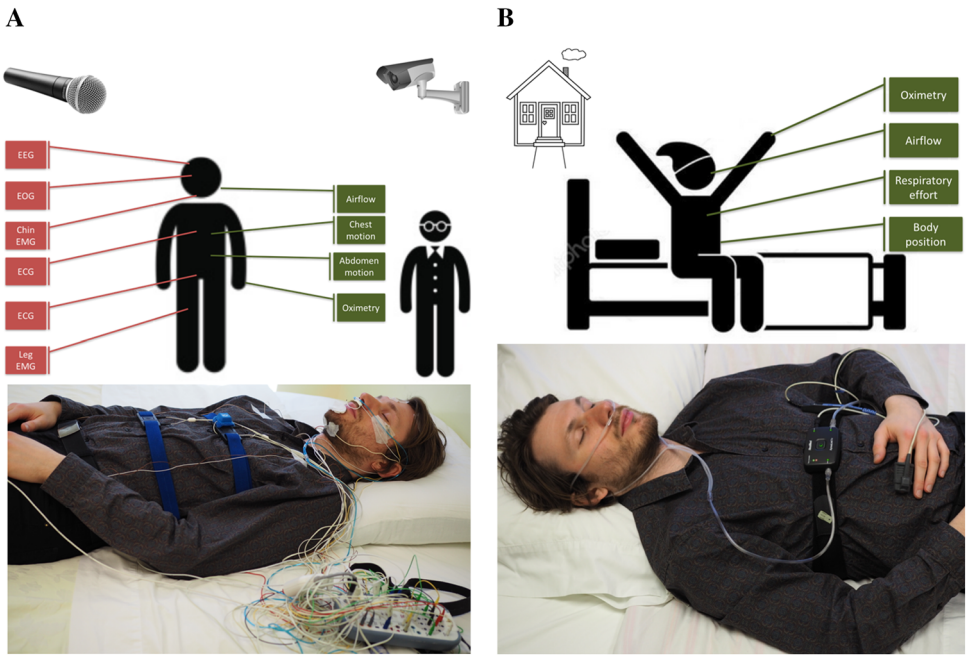


Fig. 1 A Laboratory-based (type I) polysomnography. Various electrodes are applied to monitor EEG, EOG, chin EMG, ECG, airflow, respiratory effort, and oxygen saturation. A video camera is present in the room to record body position and parasomnia behaviour. A microphone is used to record snoring. A registered sleep technologist is present to set up, monitor, and verify accuracy of the recording. EEG = electroencephalography, EOG = electrooculography, EMG = electromyography, ECG = electrocardiography. B Home sleep study with a type III monitor. The study is carried out in the comfort of the patient's own home. A minimum of four channels are used to record respiratory effort, airflow, heart rate, and oxygen saturation

Table 4 Outline of a polysomnography report

Feature	Parameters to look for	Implications for the anesthesiologist
Type of sleep study	Determine type of monitors used: I-IV Determine if it is a diagnostic or a therapeutic sleep study	A diagnostic study helps estimate the type and severity of sleep-disordered breathing A therapeutic study indicates the optimal treatment setting, such as the optimal CPAP or BPAP to abolish OSA, and the mask used by the patient
Sleep quality	Determine data reliability by looking at total sleep time, how long the patient slept, how much time spent in supine or REM sleep	Obstructive events and oxygen desaturation tend to be more severe during supine and REM sleep OSA severity may be underestimated if minimal time is spent in either supine position or REM sleep during the study
Respiratory events	a. Overall apnea-hypopnea index (AHI) b. Determine the nature of the respiratory events: obstructive, central, or mixed c. REM and non-REM AHIs; supine and non-supine AHIs	a. Indicates disease severity based on AASM criteria b. Obstructive events are amenable to treatment with CPAP, positional therapy, and oxygen therapy Central events will likely require close monitoring. Be aware of increased susceptibility for respiratory arrest with opioid use c. Define REM-related and supine-related OSA
Oxygenation	Determine additional parameters of OSA severity: ODI, mean SpO_2, O_2 nadir, T90	It is important to look at the nature of hypoxemia to evaluate recurrent desaturations (using ODI), or prolonged hypoxemia (using T90 and mean SpO_2), as well as the severity (hypoxemia of long duration > 20% of total sleep time, or sustained drop during REM sleep indicating hypoventilation)
Hypoventilation	Look for sustained hypoxemia and scooped-out pattern on the graphic summary (Fig. 4B)	a. Consider OHS in obese patients with severe OSA and significant hypoxemia b. Check serum bicarbonate level in the high-risk group. Positive result should be confirmed using an arterial blood gas analysis
Graphic summary	Look for different patterns based on body position, REM and non-REM sleep, and during a therapeutic study (CPAP *vs* no CPAP)	Can help determine key elements (e.g., sleep quality, nocturnal hypoxemia with hypoventilation, REM- or supine-related OSA

AASM = American Academy of Sleep Medicine; AHI = apnea-hypopnea index; BPAP = bilevel positive airway pressure; CPAP = continuous positive airway pressure; ODI = oxygen desaturation index; OHS = obesity hypoventilation syndrome OSA = obstructive sleep apnea; PSG = polysomnography; REM = rapid eye movement; T90 = percentage of total sleep time spent with $SpO_2 < 90\%$

and potentially underestimating OSA severity.[17] A level IV study using pulse oximetry has significant limitations in differentiating OSA from CSA. Another limitation is the use of time in bed, rather than actual sleep time, to calculate the respiratory disturbance during sleep and the accuracy of the device in detecting arterial oxyhemoglobin saturation < 70%, which can further undermine the determination of disease severity.[17] Figures 1A and 1B show the basic differences between a laboratory-based sleep study and one that is home-based.

Laboratory-based PSG studies

Polysomnography studies can be broadly categorized as diagnostic, interventional, or split-night studies.[14] A *diagnostic study* is used to identify the presence of SDB and determine its severity. An *interventional study* is performed on a separate occasion to implement and evaluate the effectiveness of a proposed treatment. Such treatment may include the use of oral appliances,[21] upper airway surgery,[22] PAP,[46] sleep posture modification, and/or drug therapy.[23] A *split-night study* can establish the SDB diagnosis, with treatment titration is instituted the same night.[14] This protocol is potentially well suited for preoperative assessment when patients have limited time prior to their surgery.

The following section describes the commonly used channels and physiological variables measured during a sleep study. Table 4 summarizes information gained from the sleep studies that is relevant for the anesthesiologist.

Sleep quality parameters and architecture

Sleep quality and architecture are discussed in the first section of any PSG report. Sleep architecture is described via quantitative data regarding the sleep stage distribution throughout the night. It usually includes a hypnogram (a graphic summary of sleep stages *vs* time). Sleep stage is scored by analyzing each 30-sec window of the EEG recording, or "epoch." Important sleep parameters, the

Table 5 Sleep parameter terminology

Sleep parameter	Definition
Lights-out time	Time at the start of recording
Lights-on time	Time at the end of recording
Total recording time (TRT)	Time from lights-out to lights-on (in minutes)
Total sleep time (TST)	Time spent in stage N1-3 and REM sleep
Sleep latency (SL)	Time from lights-out to first epoch of any sleep (in minutes)
Stage R latency (RL)	Time from sleep onset to first epoch of stage R (in minutes)
Wake after sleep onset (WASO)	Stage wakefulness after sleep onset until the end of recording
Sleep efficiency	TST/TRT × 100
Arousal Index (AI)	Number of arousals per hour of sleep (number of arousals × 60/TST)
Apnea-Hypopnea Index (AHI)	Average number of apneic and hypopneic events per hour of sleep: mild 5-15 events·hr^{-1}, moderate 15-30 events·hr^{-1}, severe > 30 events·hr^{-1}
Respiratory Disturbance Index	Average number of apneas, hypopneas and respiratory effort related arousals (RERA) per hour of sleep
(RDI)	
Oxygen Desaturation Index (ODI)	Average number of desaturation episodes (≥ 4% decrease in saturation from the average saturation in the preceding 120 sec, lasting > 10 sec) per hour of sleep
Oxygen nadir	Lowest arterial oxygen saturation during total sleep time
T90	Percentage of total sleep time spent with SpO_2 < 90%

Derived from *Berry et al.*[15]

terminology, and their definitions are summarized in Table 5.[15]

Sleep can be subcategorized into non-rapid eye movement (NREM) and REM sleep, based on the characteristic EEG, EOG, and EMG patterns (Table 6).[15] Normal sleep typically occurs as three to five cycles of alternating NREM and REM sleep during the night. NREM sleep can be further subdivided into three distinct EEG stages—N1, N2, and N3[15]—where stage N1 is the transition from wakefulness to sleep onset. Frequent brief arousals that prevent sleep from being physiologically consolidated—otherwise known as sleep fragmentation—occur during SDB and may result in more-than-average time spent in N1 sleep.[24] Sleep fragmentation has been shown to increase daytime somnolence, reduce various psychomotor performances, and worsen upper airway collapsibility.[24] Stage R, or REM, sleep is characterized by rapid eye movements; low-amplitude, mixed-frequency EEG activity; and skeletal muscle hypotonia (observed mainly on chin EMG). Dreaming, irregular breathing, respiratory events (e.g., apnea, hypopnea, severe hypoxemia), and increased sympathetic system stimulation are known to occur more frequently during this stage.[15,19]

Arousals

Cortical arousals are considered normal phenomena and can be induced by several stimuli, including noise, periodic limb movements, and respiratory events.[25] Scoring of arousal mandates a sudden shift in EEG frequency, which includes alpha, theta, and/or frequencies > 16 Hz, lasting > three seconds and a stable sleep rhythm for ten seconds prior to the change. During REM sleep, arousal needs to be accompanied by an increase in submental EMG recording.[15] Quantifying arousal using an arousal index (average number of arousals per hour of sleep) remains a controversial area because there is a lack of consensus as to what constitutes an abnormal value.[24,26] A normal arousal index varies with age: 10.0 ± 4.6 per hour in young adults; 16.0 ± 5.6 per hour during middle age, and 21.0 ± 6.8 per hour in the elderly.[27] Arousals occur more frequently with SDB, resulting in a high arousal index and sleep fragmentation (Figs 2A and 2B).[25]

Respiratory events

A description of the respiratory events during sleep constitutes an important element of the PSG report. *Apnea* is defined as a reduction in airflow of at least 90%, detected by oronasal thermal flow, and lasting ≥ ten seconds (Table 7).[15] A reduction in oxyhemoglobin saturation may occur but is not required for identification of apnea. Apnea can be classified as obstructive, central, or mixed based on the presence or absence of inspiratory effort. An obstructive event is marked by the presence of continuous respiratory effort throughout the duration of

Table 6 Sleep stages: scoring criteria

Sleep Stage	EEG Pattern	Eye Movements	Chin EMG	% of TST
W	Beta (>13 Hz) and alpha (8-13 Hz) waves >50% of an epoch, over the occipital area	Eye blinks (0.5-2.0 Hz), reading or rapid eye movements	Normal to high	—
N1	Alpha rhythm is attenuated. Low-amplitude, mixed-frequency activity (4-7 Hz) is present for > 50% of the epoch	Slow eye movements	Lower than stage W	5%
	In the absence of alpha rhythm, N1 is scored if any of the following is present:			
	- Theta waves (4-8 Hz)			
	- Vertex sharp waves (negative sharp waves, followed by a positive component, lasting < 0.5 sec), central region			
	- Slow eye movements			
N2	One or more K-complexes (well-defined, negative, sharp wave followed by a positive slow wave, lasting at least 0.5 sec)	No eye movements but slow eye movements may persist occasionally	Variable; lower than stage W, may be as low as stage R	50%
	and/or			
	Sleep spindles (distinct waves with frequency of 11-16 Hz lasting ≥ 0.5 sec)			
	Seen in first half of that epoch or last half of previous epoch			
N3	Delta (0-4 Hz), slow-wave activity (0.5-2.0 Hz, with 75 μV) ≥ 20% of an epoch	No eye movements	Variable; usually lower than stage N2	20%
	Sleep spindles may be present			
REM	Low-amplitude, mixed-frequency EEG activity, absent K-complexes and sleep spindles	REMs	Low	25%
	Cycles at 90-120 min, with increases in duration during the latter part of the night			

Derived from *Berry et al.*[15]

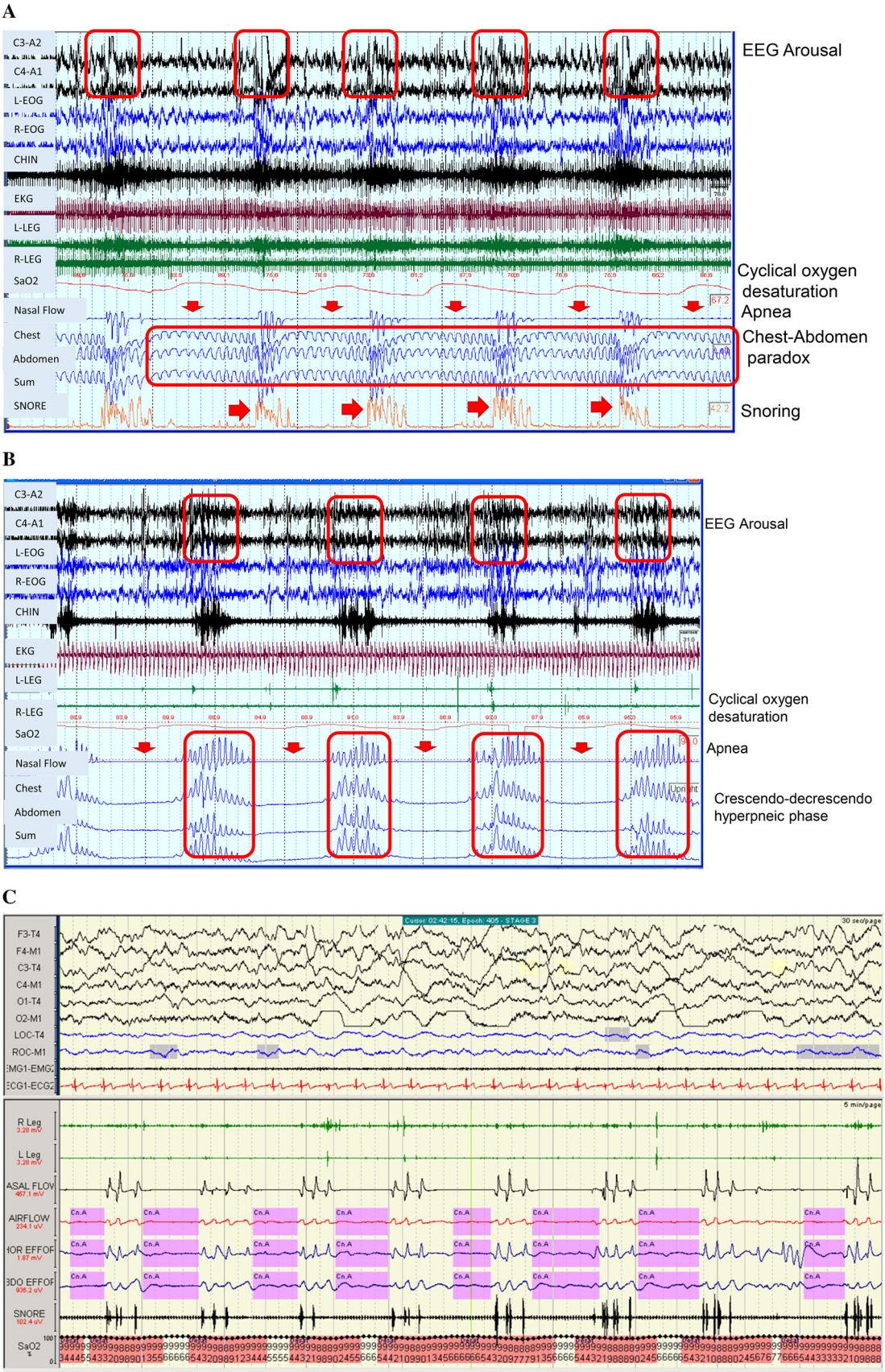
EEG =electroencephalography; EMG = electromyography; REM = rapid eye movement; TST = total sleep time

absent airflow (Fig. 2A). A central event is marked by a lack of respiratory effort (Fig. 2B). Mixed apnea begins as a central event, but there is resumption of inspiratory effort during the latter part of the event in the presence of upper airway collapse, hence converting to an obstructive pattern.[15]

Hypopnea is defined as a reduction in airflow of at least 30% for ≥ ten seconds with a concomitant 3-4% drop in oxygen saturation or EEG arousal (Table 7).[15] Differentiating between obstructive and central hypopnea can be difficult, and not all laboratories do so. Hypopnea is classified as obstructive if there is snoring, airflow limitation (as evidenced by inspiratory flattening of the nasal pressure signal), and/or thoracoabdominal paradox. Hypopnea is classified as central if none of these features is present.[15]

Respiratory events can be scored and evaluated using portable devices. Figure 3 provides an example: a portable monitor called the Apnea Link Air™ (ResMed, San Diego, CA, USA). It is a type III sleep apnea monitoring device consisting of a nasal cannula attached to a small case that houses a pressure transducer. It measures airflow through a nasal pressure transducer, providing an AHI based on the recording time. The device has a proprietary pneumatic effort sensor that measures respiratory effort, allowing differentiation of apneas into central and obstructive events. It has been validated for its diagnostic properties against the gold standard PSG.[28-30] The data are downloaded from the device and are analyzed automatically, with provision for manual scoring.

An RERA is characterized by a reduction in airflow lasting ≥ ten seconds that does not meet the criteria for apnea or hypopnea. The inspiratory nasal pressure waveform is flattened, accompanied by increasing respiratory effort and arousal from sleep. The significance of RERAs is a matter of some debate, and the AASM considers scoring of RERAs to be optional.[15] RERAs are frequently observed in patients with upper airway resistance syndrome (UARS), a condition characterized by airflow limitation and increased respiratory effort due to high airway resistance during sleep.[31] The UARS is often misdiagnosed as simple snoring or idiopathic hypersomnia during diagnostic sleep studies because consistent diagnostic criteria have not been established, and whether it is a distinct syndrome or a mild form of OSA is still controversial.[32] The AASM considers UARS as part of the OSA syndrome.[1]


A
C3-A2
C4-A1
L-EOG
R-EOG
CHIN
EKG
L-LEG
R-LEG
SaO2
Nasal Flow
Chest
Abdomen
Sum
SNORE
EEG Arousal
Cyclical oxygen desaturation
Apnea
Chest-Abdomen paradox
Snoring
B
C3-A2
C4-A1
L-EOG
R-EOG
CHIN
EKG
L-LEG
R-LEG
SaO2
Nasal Flow
Chest
Abdomen
Sum
EEG Arousal
Cyclical oxygen desaturation
Apnea
Crescendo-decrescendo hyperpneic phase
C
F3-T4
F4-M1
C3-T4
C4-M1
O1-T4
O2-M1
LOC-T4
ROC-M1
R Leg
L Leg
SNORE
SaO2

◀**Fig. 2** A A 120-sec epoch from a sleep study shows multiple episodes of obstructive apneas, accompanied by snoring, desaturation, and continued respiratory effort as evidenced by chest-abdomen paradoxical movements. Monitoring channels from top to bottom: C3-A2 and C4-A1 = 2 EEG channels; L-EOG and R-EOG = left and right electro-oculogram; Chin = chin electromyogram, ECG = electrocardiography; R-Leg and L-Leg = right and left leg electromyography; SaO_2 = oxygen saturation; nasal flow = nasal airflow waveform; Chest and abdomen = thoracoabdominal excursions using RIP belt; sum = sum of chest and abdominal waveform; snoring = snoring recorded with a microphone. EEG = electroencephalography; RIP = respiratory inductance plethysmography. B A 300-sec epoch from a sleep study shows central sleep apnea with the Cheyne-Stokes breathing pattern. The arrows indicate central apneic events with absent respiratory effort (i.e. no chest or abdominal movement and lack of snoring). Cyclical breathing pattern of central apnea is followed by a crescendo-decrescendo hyperpneic phase, highlighted within the boxes. The time from the start of a central apnea to the start of the next event (cycle length) is more than 60 sec, consistent with central sleep apnea with the Cheyne-Stokes breathing pattern. Monitoring channels from top to bottom: Upper panel (30-sec epoch): F3-T4, F4-M1, C3-T4, C4-M1, O1-T4, and O2-M1 = 6 EEG channels; LOC-T4 and ROC-M1 = left and right EOG: EMG1-EMG2 = chin electromyogram; ECG = electrocardiogram. Lower panel (300-sec epoch): R Leg and L Leg = anterior tibialis EMG; SaO2% = oxygen saturation; nasal flow and airflow = nasal transducer and nasal thermistor waveforms; THOR EFFORT and ABDO EFFORT = thoraco-abdominal excursions using respiratory inductance plethysmography; Snore = snoring. CSA = central sleep apnea; CSB = Cheyne-Stokes breathing; EEG = electroencephalography; RIP = respiratory inductance plethysmography. C - A 300-sec sleep study epoch (lower panel) in a patient taking opioids for chronic non-cancer pain shows central sleep apnea with ataxic breathing or Biot's respiration pattern. Arrows indicate central apneic events with absent respiratory effort: i.e., no pressure transducer airflow signals (nasal transducer and thermistor), chest or abdominal movement, or snoring. The airflow patterns occur in an ataxic or irregular breathing pattern associated with cyclical oxygen desaturation. The events are terminated by appearance of airflow and snoring, where a classically described "cluster breathing" pattern is observed. Monitoring channels from top to bottom of figures: F3-T4, F4-M1, C3-T4, C4-M1, O1-T4, and O2-M1 = 6 EEG channels; LOC-T4 and ROC-M1 = left and right EOG; EMG1-EMG2 = chin electromyography, ECG1-ECG2 = electrocardiography; R-Leg and L-Leg = right & left leg electromyography; NASAL FLOW = nasal airflow waveform; AIRFLOW = nasal thermistor; THOR EFFORT and ABDO EFFORT = thoracoabdominal excursions using respiratory inductance plethysmography; SNORE = snoring channel; SaO_2 = oxygen saturation

Obstructive sleep apnea

The hallmark of OSA is repetitive partial or complete upper airway collapse during sleep, resulting in multiple episodes of obstructive apnea and/or hypopnea throughout the night.[1] The degree, duration, and frequency of airway obstruction determine the severity of associated oxygen desaturation. Brief arousal from sleep usually follows, terminating the abnormal breathing pattern and restoring oxygenation. The AHI—the average number of apneic and hypopneic events per hour of sleep—is used to diagnose OSA and its severity. The clinical diagnosis of OSA syndrome mandates either an AHI of $\geq$ 15 events·hr^{-1} or an AHI of $\geq$ 5 events·hr^{-1} with the classic symptoms of OSA. The condition is classified as mild when the AHI is 5-15 events·hr^{-1}, moderate at 15-30 events·hr^{-1}, and severe at $>$ 30 events·hr.$^{-1}$ [1] The severity of OSA may play an important role in its morbidity and mortality rates. The AHI has been found to correlate with adverse cardiovascular health outcomes,[33,34] including mortality, in the general population.[35-38] In the perioperative setting, there is a limited but growing body of evidence confirming the long-held belief among perioperative physicians that OSA severity, as defined by the preoperative AHI, is predictive of increased adverse outcomes.[39,40] Mutter *et al.* found that patients with severe OSA (AHI $>$ 30) or undiagnosed OSA was associated with significantly increased risk of cardiorespiratory complications.[39] Their study was the first to show a positive association between severity of OSA and postoperative risks.

The respiratory disturbance index (RDI) is another mechanism for describing the severity of SDB. In addition to the average number of apneic and hypopneic events per hour of sleep, the RDI incorporates RERAs. Occasionally, patients present with a normal AHI but have frequent RERAs and symptoms of OSA, an abnormality that would be reflected in an elevated RDI.[41] In a prospective observational study of surgical patients without an OSA diagnosis (AHI $<$ 5 events·hr^{-1}) preoperatively, the RDI was a significant, independent predictor of postoperative moderate to severe SDB (AHI $>$ 15 events·hr^{-1}).[41] Preoperative RERAs may indicate a predisposition to more severe upper airway obstruction and/or respiratory depression that may be aggravated by opioids and sedatives administered during the perioperative period.[41] To date, however, there is a paucity of data available to assess the association between RERAs or SDB that develop during the postoperative period and adverse outcomes. This effect needs to be explored in future clinical studies. In addition, it is important to recognize that the AHI and RDI may exhibit night-to-night variability, with the possibility of missing a diagnosis of mild OSA.[41]

Additional metrics of OSA severity—ODI, mean arterial oxygen saturation, O_2 nadir, and total overnight duration of oxygen saturation $<$ 90% (T90)—can be useful to the anesthesiologist.[42] Recording the ODI—revealing the hourly desaturation events during sleep—can be based on either $\geq$ 3% or 4% desaturation for at least ten seconds, although it should be clearly stated on the report which cutoff value is being used. The current AASM Manual for the Scoring of Sleep and Associated Events recommends the use of a 3% cutoff.[15] Postoperative hypoxemia is best predicted by preoperative hypoxemia and apneic events.[43] A prospective observational study involving more than 500 surgical patients examined parameters from preoperative overnight oximetry

Table 7 American Academy of Sleep Medicine criteria for respiratory events

Score of respiratory event as apnea when BOTH of the following criteria are met:
a) There is a drop in the peak signal excursion by ≥ 90% of pre-event baseline using an oronasal thermal sensor (diagnostic study), PAP device flow (titration study), or an alternative apnea sensor (diagnostic study).
b) The duration of the ≥ 90% drop in sensor signal is ≥10 sec.
Score a respiratory event as hypopnea when ALL of the following criteria are met:
a) The peak signal excursions drop by ≥ 30% of pre-event baseline using nasal pressure (diagnostic study), PAP device flow (titration study), or an alternative hypopnea sensor.
b) The duration of ≥30% drop in signal excursion is ≥ 10 sec.
c) There is ≥ 3% oxygen desaturation from the pre-event baseline or the event is associated with arousal (recommended) OR there is ≥ 4% oxygen desaturation from the pre-event baseline (optional).
Score a respiratory event as a respiratory effort-related arousal (RERA) if:
There is a sequence of breaths lasting ≥10 sec characterized by increasing respiratory effort or by flattening of the inspiratory portion of the nasal pressure/PAP device flow waveform, leading to arousal from sleep when the sequence of breaths does not meet the criteria for apnea or hypopnea.
Scoring Cheyne-Stokes breathing requires both criteria to be met:
a) At least three consecutive central apneas and/or hypopneas separated by a crescendo-decrescendo change in breathing amplitude with a cycle length of ≥ 40 sec.
b) At least five central apneic and/or hypopneic events per hour of sleep associated with the crescendo-decrescendo breathing pattern recorded over ≥ 2 hr of monitoring.
Score hypoventilation during sleep if either of the conditions below occur:
a) An increase in arterial PCO_2 (or surrogate) to a value > 55 mmHg for ≥ 10 min.
b) A ≥ 10 mmHg increase in arterial PCO_2 (or surrogate) during sleep (compared with an awake supine value) to > 50 mmHg for ≥ 10 min.

Derived from *Berry et al.*[15]

PAP = positive airway pressure

to determine predictors of postoperative complications. Univariate logistic regression analysis revealed that a mean preoperative overnight $SpO_2 < 93\%$, ODI > 29 events·hr^{-1}, and 90% oxygen saturation (T90) > 7% were significant predictors for adverse events.[40] Moreover, after adjusting for age, sex, body mass index, and American Society of Anesthesiologists' physical status, an ODI > 28.5 events·hr^{-1} was found to predict a twofold risk of postoperative adverse events.[40]

To illustrate this point, we prepared graphic summaries of two patients with severe OSA (Figs. 4A and 4B). Both have severe OSA as defined by the AHI but may have different risk profiles based on the severity of their oxygen desaturation. The first patient has an AHI of 30 events·hr^{-1} due to obstructive hypopnea that occur predominantly during REM sleep and in the supine position (Fig. 4A). Despite the frequent respiratory events, few desaturation events occurred, and a nominal amount of time was spent with SpO_2 at < 90%. The second patient has persistent, rather than cyclical, hypoxemia. He shows excessively high AHI with similar episodes of apnea and hypopnea (Fig. 4B). The T90 was 95% of total sleep time with an overall mean SpO_2 of 82% and oxygen nadir of 64% for three minutes. The second patient's findings likely portend a higher risk of postoperative complications. These examples shows that OSA severity based on AHI may be supplemented by adding preoperative hypoxemia parameters. Future clinical studies should be designed to evaluate the difference in predictive abilities of preoperative hypoxemia versus AHI values.

It is important to identify and optimize patients with suspected or previously diagnosed OSA at the time of surgery. Positive airway pressure therapy is one of the main treatment options for patients diagnosed with moderate to severe OSA. There is a growing body of evidence to support the use of PAP to reduce adverse outcomes during the postoperative period.[6,39,44] A recent meta-analysis also showed a trend towards a shorter hospital stay when PAP was used.[45] The optimal selected pressure recommended by the AASM should reduce the RDI to fewer than five events per hour and maintain oxygen saturation at > 90%.[46] Patients with UARS may also respond to therapy for OSA (e.g., nasal continuous positive airway pressure, or CPAP), although compliance is often poor, and good quality evidence is still lacking.[32]

Effect of body position and sleep stages on respiratory events

The severity of SDB may vary depending on the sleep position or sleep stage, with SDB typically being worse in the supine position and during REM sleep. Supine position-related OSA is a dominant phenotype of the OSA syndrome, wherein obstructive events occur twice as

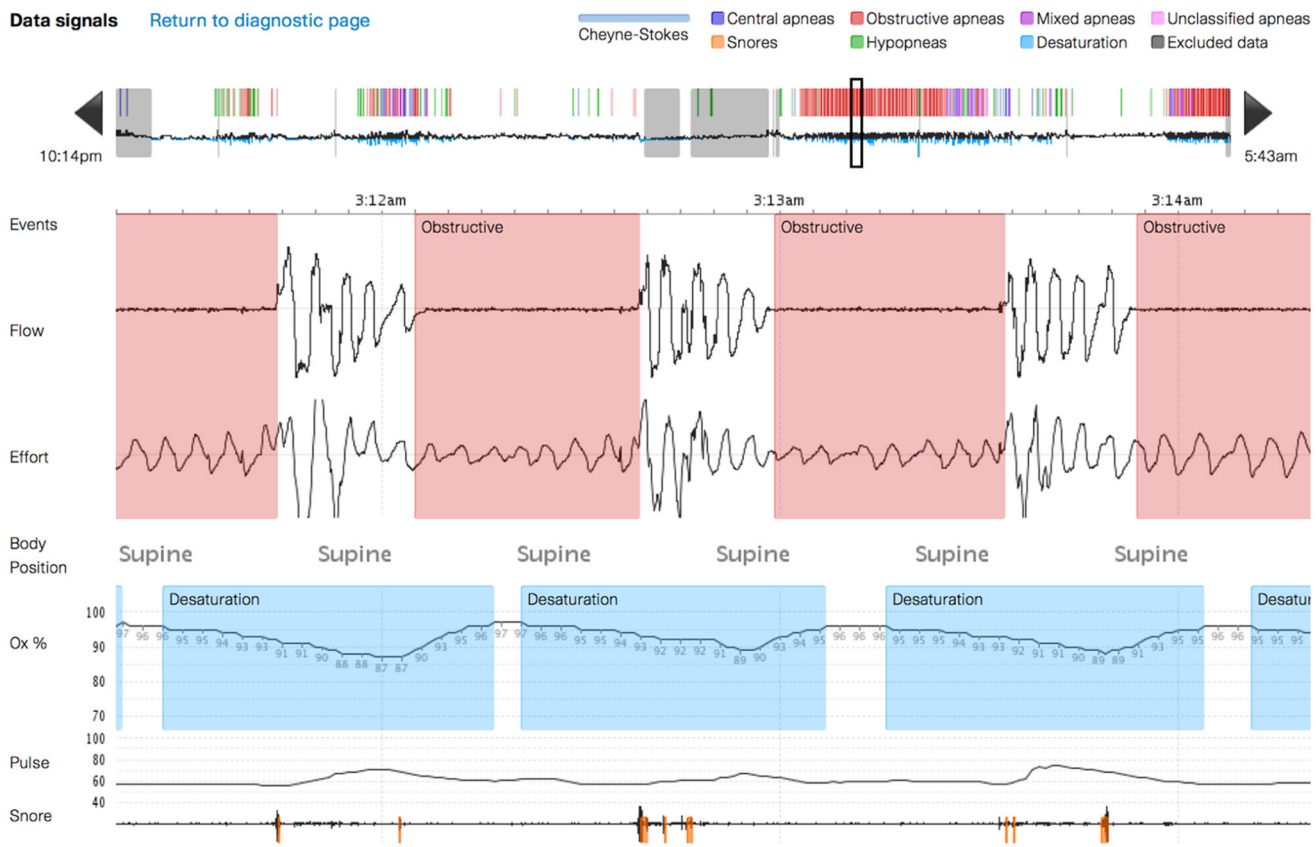


Fig. 3 Recording of obstructive apneas from a type III portable sleep study monitor: Apnea Link Air™ (ResMed, San Diego, CA, USA). Frequent obstructive apneic events are observed (highlighted in red) while the patient is supine. The events are noted towards the early hours of the morning, possibly during the REM sleep, but this cannot be confirmed as EEG data are not recorded by type III studies. Snoring and chest abdominal paradoxical movements are observed during the apneic episodes followed by desaturation towards the end of each apneic events. EEG = electroencephalography; REM = rapid eye movement

frequently in the supine position.[47,48] The diagnosis of supine-predominant OSA requires an overall AHI of > 5 events·hr^{-1}, with the supine AHI more than two times the non-supine AHI. Supine isolated OSA requires an additional criterion of non-supine AHI showing < 5 events·hr^{-1}.[47] During the postoperative period, an observational study that examined factors associated with exacerbation of SDB showed that both OSA and non-OSA patients spent a significantly higher percentage of sleep in the supine position on nights 1 and 3 after surgery.[49] The postoperative AHI was higher in the supine position compared with the AHI in a non-supine position.[49] The impact of the worsening AHI is unclear. It may suggest that patients with supine-predominant or isolated OSA are at increased risk of adverse events. Data on clinically meaningful outcomes, however, are scarce. Avoidance of the supine position during sleep appears to be effective in reducing the AHI, but there is a lack of good quality evidence in the literature.[23,40] Furthermore, depending on the nature of the surgery, avoidance of the supine position may be impractical in the perioperative setting.

Rapid eye movement-related OSA is another phenotype of the OSA syndrome wherein obstructive events occur twice as frequently during REM *vs* NREM sleep.[48] The condition can be further categorized into REM-predominant OSA (REM AHI > two times the non-REM AHI) and REM-isolated OSA (when an additional criterion of NREM is fulfilled—i.e., AHI < 5 events·hr^{-1}).[50] Although there is limited understanding of the worsening of apnea and hypopnea during REM sleep,[51] it has been postulated to be due to hypotonia of the upper airway muscles and decreased response of the genioglossus to negative intrapharyngeal pressure, resulting in an increased propensity for upper airway collapse.[52,53] Conventional treatment modalities are usually used for this phenotype, although treatment targeting the genioglossus muscle tone (e.g., transnasal insufflation) has been described.[54] Conditions or medications that affect the time spent in REM or supine sleep may have an impact on the overall AHI and postoperative OSA severity. However, long-term and postoperative outcome data on patient with supine- or REM-related OSA *vs* "typical" OSA are currently lacking.

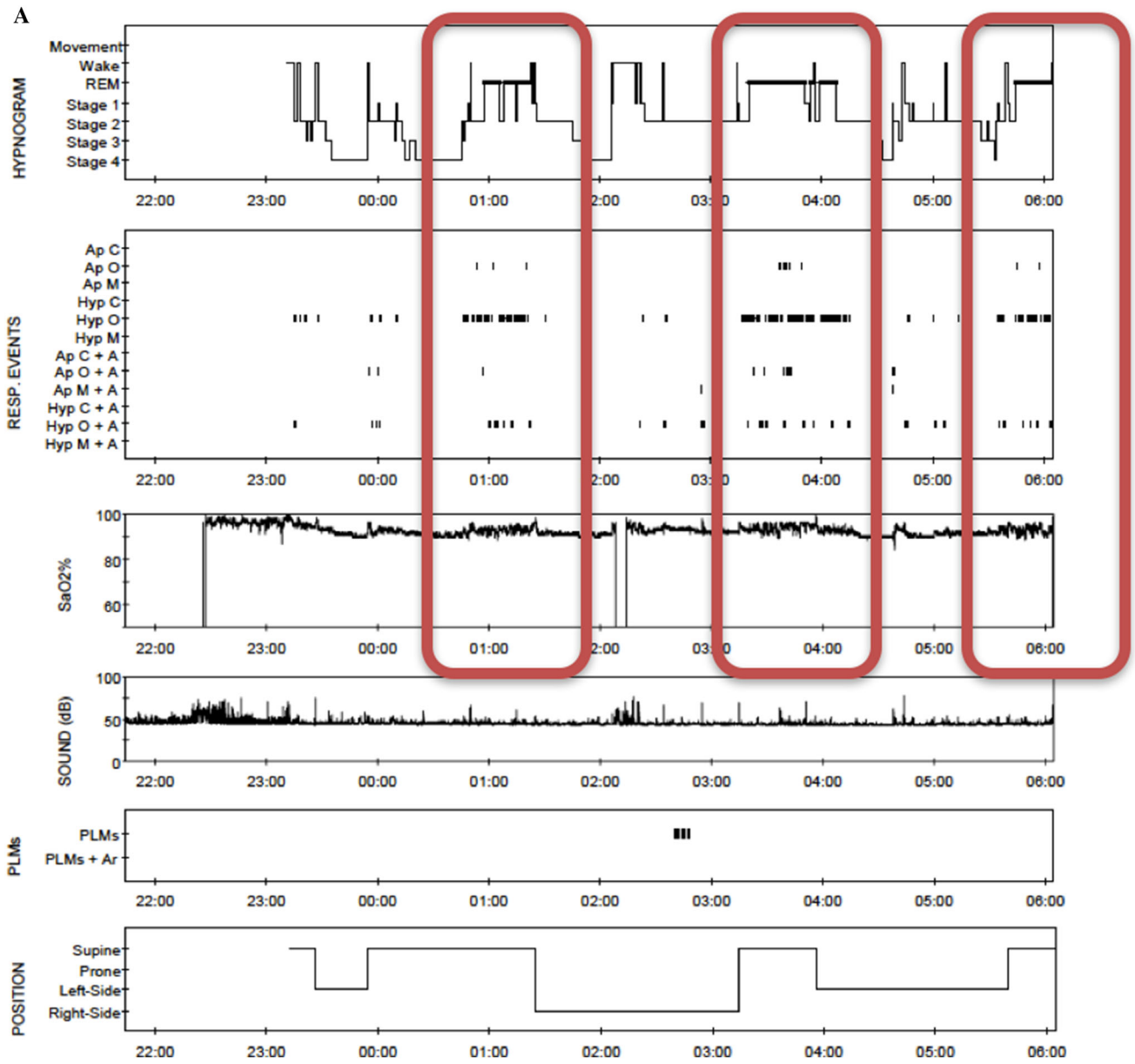


Fig. 4 A Graphic summary of a patient with severe supine- and REM-predominant OSA with mild hypoxemia. AHI = 30/hr (25 apneic and 167 hypopneic evemts); AHI (REM) 81/hr *vs* 15.5/hr (non-REM); AHI (supine) 51 *vs* 13.6/ hr (non-supine); mean oxygen saturation = 92%; oxygen nadir = 84%; T90 = 3 min (0.83% TST). AHI = apnea-hypopnea index; TST = total sleep time; T90 = percentage of total sleep time spent with $SpO_2 < 90\%$; OSA = obstructive sleep apnea; REM = rapid eye movement. B Graphic summary of a patient with severe OSA with significant hypoxemia. No comments can be made about supine/REM-predominant OSA. The data are suspicious for a diagnosis of OHS. AHI = 130/ hr; supine/ REM AHI = 0/hr as patient did not spend time in either supine or REM sleep; mean oxygen saturation = 82.8%; oxygen nadir = 64.5%; T90 = 292 min (95% TST); mean transcutaneous CO_2 = 55 mmHg. AHI = apnea-hypopnea index; OHS = obesity hypoventilation syndrome; OSA = obstructive sleep apnea; REM= rapid eye movement; TST = total sleep time; T90 = percentage of total sleep time spent with $SpO_2 < 90\%$

Hypoventilation

In morbidly obese patients with severe OSA, clinicians should consider OHS, which is associated with increased overall morbidity and mortality.[55-59] Although the evidence on perioperative complications is limited, non-cardiac surgical patients with unrecognized OHS in a recent study were found to be at significantly higher risk of respiratory failure (odds ratio [OR], 10.9), heart failure (OR, 5.4), prolonged intubation (OR, 3.1), intensive care unit admission (OR, 10.9), and longer hospital stay when compared to OSA patients without hypoventilation.[60] The

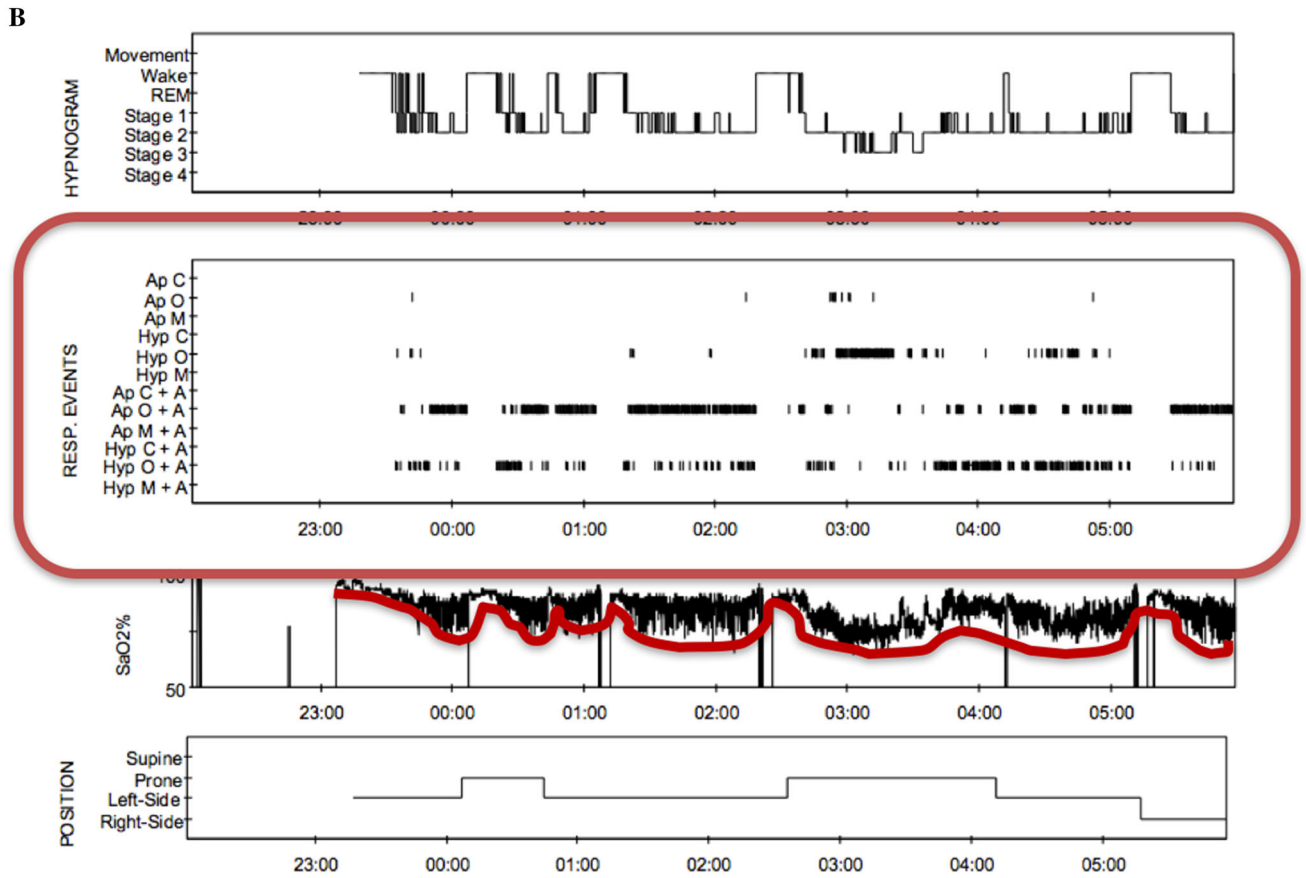


Fig. 4 continued

diagnosis of OHS requires documented awake hypoventilation ($PaCO_2 > 45$ mmHg) in obese patients with body mass index > 30 kg·m^{-2}. An important component of the diagnosis is the exclusion of other causes of alveolar hypoventilation, such as conditions affecting the respiratory muscles, chest wall, or lungs as well as a variety of neurological conditions.[61] Predictors of daytime hypercapnia in obese patients with OSA have included body mass index > 39 kg·m^{-2}, severe OSA with AHI > 64 events·hr^{-1}, and restrictive chest wall mechanics.[57]

Preoperative screening for hypercapnia should be performed in patients deemed to be at high risk. The second patient in Fig. 2B falls into this category and should be investigated for OHS. Elevated serum bicarbonate can be used as a sensitive marker of metabolic compensation for long-standing respiratory acidosis. A serum bicarbonate level > 27 mEq·L^{-1} has been shown to have 92% sensitivity for predicting hypercapnia in patients with OSA.[62] A positive result should be confirmed using an arterial blood gas assay.

Cardiac events

Both benign and lethal nocturnal arrhythmias—e.g., frequent ventricular premature contractions, atrial fibrillation, non-sustained ventricular tachycardia, sinus arrest, second degree heart block—are commonly observed in patients with OSA.[33,63,64] In a non-surgical OSA population, nocturnal hypoxemia has been identified as an independent predictor of sudden cardiac death. Parameters such as mean nocturnal oxygen saturation $< 93\%$ and oxygen nadir $< 78\%$ have been reported with hazard ratios of 2.9 and 2.6, respectively.[37] Moreover, there is a strong correlation between OSA severity and the risk of nocturnal sudden cardiac death. Patients with severe OSA (AHI ≥ 40) had a 40% rise in the relative risk for sudden cardiac death compared to those with mild to moderate OSA (AHI 5-39).[38] Because OSA has the potential to increase cardiovascular complications in the perioperative setting, anesthesiologists should maintain high vigilance regarding cardiac arrhythmias in patients with severe OSA.[65]

Central sleep apnea

Central sleep apnea encompasses a variety of conditions, including Cheyne-Stokes breathing (CSB), primary CSA, CSA due to medical conditions, medication-induced CSA, and high-altitude periodic breathing.[1] A diagnosis of CSA disorder requires at least five apneic and hypopneic events per hour of sleep, with central events being the predominant type (> 50% of the total apneic/hypopneic events).[15] There is currently no severity marker for CSA in the literature[66] and no studies looking at the effects of various CSA subtypes on postoperative outcomes.

Central sleep apnea with CSB is characterized by the presence of the cyclical breathing pattern of central apnea or hypopnea followed by a crescendo-decrescendo hyperpneic phase (Fig. 2B), where the cycle length is usually > 60 sec.[15] Table 7 contains AASM scoring criteria for CSB.[15] This abnormal breathing pattern is commonly seen in patients with congestive cardiac failure, with a prevalence of 25-40%.[1] Risk factors for CSB in congestive cardiac failure patients include male sex, age > 60 yr, atrial fibrillation, and the presence of hypocapnia (≤ 38 mmHg). Some patients with heart failure have mixed obstructive and central events, with the latter being more common towards the morning and after implementation of PAP therapy. The CSB pattern may be seen following an acute stroke and possibly in patients with renal failure.[1,67] In *CSA due to medical conditions* without CSB, congenital or acquired brain stem lesions are usually responsible.[1] In *CSA due to medications/substance use*, potent, long-acting opioids are the most common offending drugs.[68] The estimated prevalence of CSA is 24% among chronic opioid users. A morphine equivalent daily dose of > 200 mg has been shown to be associated with an increased risk of CSA.[68] Slow, irregular breathing patterns (ataxic or cluster breathing),[69] hypoventilation, and OSA may also be seen with this condition (Fig. 2C). A diagnosis of *primary CSA* can be made only after other medical conditions and substance abuse have been excluded.[1]

Conclusion

This review has highlighted important sleep study parameters relevant for anesthesiologists in the perioperative setting. Having learned the basic tenets of a typical PSG report, the anesthesiologist can make informed decisions, such as requesting appropriate tests, optimizing treatment, and monitoring patients at risk of cardiorespiratory events.

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