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REVIEW

Reframing Sleep Diagnostics: A Structured Clinical Guide to Polysomnography Evaluation

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ABSTRACT

Polysomnography (PSG) remains the gold standard for evaluating sleep-disordered breathing (SDB) and related conditions, yet its clinical potential is often limited by overreliance on the apnea–hypopnea index (AHI) and similar summary metrics. This narrative review offers a structured, physiology-informed approach to PSG interpretation that integrates sleep architecture, arousal burden, respiratory event morphology, oxygenation patterns, and CO_2 trends. Recognizing phenotypic patterns—such as REM-related and positional OSA—through sleep stage and positional stratification is essential for directing targeted therapy. We outline common interpretive pitfalls, including automated scoring errors, overlooked signal artifacts, and the first night effect, emphasizing the need for careful manual review and clinical correlation. In pediatric and syndromic populations, age-adjusted interpretation is critical for detecting subtle respiratory disturbances that can impact neurodevelopment or behavior. A practical stepwise framework is provided to guide interpretation, streamline clinical workflows, and reduce diagnostic error. PSG findings are also contextualized within broader systemic outcomes, such as their links to hypertension, dyslipidemia, insulin resistance, and cognitive decline. Ultimately, PSG should be viewed not as a static diagnostic report, but as a cornerstone of personalized sleep medicine—informing mechanism-based, outcome-oriented interventions tailored to individual patients.

Keywords: Polysomnography, Sleep-disordered breathing, Sleep architecture, REM-related OSA, Narrative review

1. Introduction

Sleep is a vital physiological process closely intertwined with cardiovascular, metabolic, and neurocognitive health. Chronic sleep deprivation and untreated sleep disorders elevate the risk of hypertension, insulin resistance, arrhythmias, cognitive impairment, mood disturbances, and early mortality [1, 2]. Despite these widespread systemic effects, clinical sleep assessment remains underutilized and often oversimplified to summary indices that fail to capture the complexity of sleep physiology.

Polysomnography (PSG) is the gold standard for diagnosing sleep disorders, particularly sleep-disordered breathing (SDB). As a comprehensive physiological test, PSG records neuroelectrical activity, respiratory patterns, oxygen saturation, and motor function throughout the sleep cycle. However, in routine practice, interpretation is frequently reduced to the apnea–hypopnea index (AHI)—a convenient yet narrow metric that may overlook meaningful pathophysiology [3]. This reductionist approach limits PSG's potential to identify actionable disease subtypes and guide targeted therapy.

SDB encompasses a spectrum of disorders, including obstructive sleep apnea (OSA), central sleep apnea (CSA), and mixed variants. While these may present with overlapping symptoms such as excessive daytime sleepiness or snoring, their underlying mechanisms differ substantially. OSA involves recurrent upper airway collapse with preserved respiratory drive, whereas CSA reflects ventilatory instability

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with transient reductions or absence of central respiratory output, often linked to heart failure, high-altitude exposure, or opioid use [3]. Recognizing these distinctions is essential for directing appropriate interventions.

Recent advances have identified four key traits critical to understanding SDB: upper airway anatomical collapsibility, loop gain (ventilatory control instability), arousal threshold, and upper airway muscle responsiveness. Summarized in the PALM framework, these traits underpin phenotype-driven therapy [5]. Event morphology, respiratory effort patterns, REM versus NREM distribution, and positional dependency—all extractable from PSG—serve as non-invasive surrogates for these traits, enabling clinicians to move beyond severity-based classifications toward mechanism-informed care [6].

Age significantly modifies sleep physiology and PSG interpretation. With aging, there is a natural decline in total sleep time and efficiency, accompanied by increased arousals, elevated AHI, and reduced REM and slow-wave sleep [7]. An AHI of 10 events per hour, clearly pathological in children, may be within normal limits in older adults. Sex differences also influence SDB phenotypes; women more often exhibit REM-predominant OSA, lower AHI, and higher arousal burdens for equivalent respiratory disturbances, contributing to underdiagnosis [8]. Body habitus and comorbidities such as neuromuscular disease, craniofacial syndromes, or heart failure further shape PSG profiles. Pediatric PSG demands age-specific scoring and careful interpretation, as even mild disturbances can have neurodevelopmental or behavioral consequences, necessitating scrutiny beyond the AHI alone. In contrast, PSG findings in older adults often mirror cumulative physiologic aging, requiring correlation with functional status to avoid overdiagnosis and overtreatment.

Beyond respiratory disorders, PSG aids in diagnosing periodic limb movement disorder, REM behavior disorder (RBD), narcolepsy, and nocturnal seizures—each with distinctive electrodiagnostic features. Accurate interpretation demands waveform-level analysis across sleep stages, as reliance on summary reports may obscure critical findings. Extended EEG montages, synchronized audiovisual monitoring, and CO_2 measurements are often essential in complex cases.

Despite its diagnostic breadth, PSG remains underutilized as a tool for physiologic phenotyping. Routine reports frequently emphasize AHI, oxygen desaturation index, and sleep efficiency, while overlooking REM distribution, positional patterns, or event morphology—factors with direct therapeutic implications. As sleep medicine advances toward personalized, physiology-guided care, PSG must be repositioned not just as a confirmatory test, but as a cornerstone for therapeutic stratification. Future directions should align PSG interpretation with precision medicine principles, leveraging its full capacity to tailor interventions to individual pathophysiological profiles [4].

2. Methods

This article was designed as a narrative review with the aim of providing a structured, physiology-informed framework for interpreting PSG in clinical practice. We performed a targeted literature search using PubMed and Scopus databases to identify key studies published between 2000 and 2024. The main search terms included combinations of "polysomnography," "sleep architecture," "obstructive sleep apnea," "REM-related OSA," "positional OSA," "phenotyping," "pediatric sleep," "arousal index," and "CO_2 monitoring." Additional references were identified by reviewing the bibliographies of relevant articles. Priority was given to guidelines and consensus statements from the American Academy of Sleep Medicine (AASM), as well as high-quality reviews and original research articles that offered insight into PSG scoring, interpretation pitfalls, and phenotype-driven management.

No formal inclusion or exclusion criteria were applied given the narrative scope; instead, studies were selected based on their relevance to the clinical interpretation of PSG across adult and pediatric populations, with attention to coverage of diverse pathophysiological phenotypes and interpretive considerations. The content of this review reflects a synthesis of the literature combined with our clinical experience in managing patients with sleep-disordered breathing and related conditions.

3. Overview of polysomnography

3.1. Definition and purpose

PSG is a comprehensive, multi-channel diagnostic tool that measures and synchronizes a range of physiological signals during sleep. It is designed to evaluate sleep architecture, detect pathological disruptions in respiration and movement, and characterize arousal-related events that may contribute to sleep fragmentation or daytime dysfunction [9]. Standard PSG captures electroencephalographic (EEG) activity for sleep staging, electrooculographic (EOG) and electromyographic (EMG) signals for REM identification and muscle tone, respiratory effort via

thoracoabdominal belts, airflow using nasal pressure transducers and oronasal thermistors, peripheral oxygen saturation (SpO_2), electrocardiography (ECG), body position, and snore vibration [10]. Together, these channels allow for an integrated assessment of sleep continuity, sleep stage transitions, and cardiorespiratory stability throughout the night [9, 10].

Each component of the PSG provides specific insights into sleep physiology. EEG, EOG, and chin EMG form the basis for sleep staging, enabling classification into NREM (N1–N3) and REM stages and detection of microarousals [11]. Leg EMG allows identification of periodic limb movements, while ECG enables detection of arrhythmias or autonomic fluctuations associated with respiratory events [10]. Airflow is assessed via nasal pressure (for hypopneas) and thermistors (for apneas), while respiratory effort is measured by thoracic and abdominal movement belts to differentiate obstructive from central events [9]. SpO_2 provides a noninvasive measure of oxygenation and desaturation burden, and in some protocols, end-tidal or transcutaneous CO_2 monitoring is added to detect nocturnal hypoventilation [10]. Snore microphones and body position sensors further aid in phenotyping positional OSA or detecting REM-related breathing disturbances. The temporal alignment of these variables allows for precise correlation between respiratory events, arousals, and desaturations, which is critical for accurate diagnosis [9].

PSG is not merely a diagnostic confirmation tool but a physiologic investigation that must be interpreted within the context of clinical history. Its utility extends beyond the detection of OSA or CSA; it also plays a role in characterizing less overt disorders such as REM behavior disorder, periodic limb movement disorder, or sleep-related hypoventilation. As illustrated in pediatric populations with comorbid conditions—such as epilepsy, neuromuscular disease, or vagus nerve stimulation—PSG findings may reflect interacting pathologies rather than isolated sleep pathology [12, 13]. Accordingly, the results must inform treatment selection, whether through positive airway pressure (PAP) titration, pharmacologic adjustments, or surgical decision-making. When interpreted comprehensively, PSG functions not only as a diagnostic anchor but as a dynamic platform for precision sleep medicine.

3.2. Indication of PSG

PSG is indicated when clinical suspicion arises for sleep disorders that require objective physiological monitoring to establish a diagnosis, assess severity, or guide management. The most robust and widely accepted indication is the evaluation of SDB, particularly OSA. PSG is essential in patients presenting with habitual snoring, witnessed apneas, gasping or choking during sleep, excessive daytime sleepiness, refractory hypertension, or unexplained cardiovascular or metabolic comorbidities [2, 4, 8]. While home-based sleep studies may be considered for high-probability cases in uncomplicated adults, full-night in-laboratory PSG remains the gold standard, particularly when diagnostic uncertainty exists or when therapeutic interventions such as positive airway pressure (PAP) titration or upper airway surgery are being considered [5, 10]. The choice of PSG level—ranging from full in-laboratory to home-based testing—should be individualized based on clinical urgency, patient complexity, and the need for real-time titration or extended physiological monitoring [9].

PSG is also central to the workup of hypersomnia disorders, particularly narcolepsy and idiopathic hypersomnia. In such cases, PSG is typically performed the night prior to a Multiple Sleep Latency Test (MSLT), which measures sleep onset latency and the occurrence of sleep-onset REM periods. This tandem testing is essential to differentiate narcolepsy from other causes of excessive daytime sleepiness (EDS) and to exclude insufficient sleep or comorbid OSA as confounders [1]. The role of PSG in narcolepsy is particularly crucial in pediatric populations, where symptom expression may be atypical and behavioral manifestations may mimic psychiatric or neurodevelopmental conditions [12–15].

In pediatric populations, PSG is indicated for a broader range of presentations compared to adults. Suspected OSA—especially in children with adenotonsillar hypertrophy, craniofacial anomalies, Down syndrome, neuromuscular conditions, or unexplained growth delays—warrants PSG evaluation [16]. Pediatric-specific diagnostic criteria apply, with an AHI $\geq$ 1 considered abnormal [9, 13]. Beyond OSA, PSG is useful in children with suspected central hypoventilation, congenital airway malformations, or when symptoms such as nocturnal enuresis (which has been linked to elevated arousals, impaired bladder signaling, and atrial natriuretic peptide release during apneic episodes), behavioral problems, or academic decline raise suspicion of sleep fragmentation or undiagnosed SDB [12, 17].

PSG is also indicated in the evaluation of parasomnias, nocturnal seizures, and REM sleep behavior disorder (RBD), particularly when episodes are atypical, potentially injurious, or diagnostically unclear. In such cases, PSG with extended EEG montages and synchronized video monitoring is necessary to differentiate parasomnias from epileptic events and to confirm REM without atonia in RBD [13, 15]. Similarly, in patients with restless legs syndrome (RLS)

or periodic limb movement disorder (PLMD), PSG may be useful when diagnosis is uncertain or when the severity and impact of periodic limb movements on sleep architecture require objective quantification [13].

Other indications include suspected sleep-related hypoventilation or hypoxemia, such as in patients with advanced neuromuscular disease, severe obesity, or restrictive thoracic disorders (e.g., kyphoscoliosis), where PSG can help characterize ventilatory patterns, assess oxygen desaturation burden, and guide decisions about non-invasive ventilation [6, 18]. Preoperative PSG may be appropriate in high-risk surgical candidates—such as those undergoing bariatric surgery—particularly when there is high suspicion of undiagnosed SDB that could complicate anesthesia or postoperative outcomes [10]. PSG is also used to evaluate treatment failure or residual symptoms in patients previously diagnosed with OSA or CSA, especially when subjective improvement does not align with therapeutic expectations [3, 5].

Conversely, PSG is not routinely indicated for the evaluation of chronic insomnia, sleep-related bruxism, or circadian rhythm sleep-wake disorders unless there is evidence of comorbid sleep pathology—such as sleep apnea or periodic limb movements—that may contribute to the clinical presentation. In such cases, PSG may help rule out alternative diagnoses but is not required for primary diagnosis [10, 15].

3.3. PSG levels (AASM classification)

The American Academy of Sleep Medicine (AASM) classifies polysomnographic studies into four diagnostic levels based on the extent of physiological monitoring, the presence or absence of technical supervision, and the study environment. This stratification—into Level 1 through Level 4 PSG—allows clinicians to match diagnostic intensity with clinical complexity, while balancing resource allocation and accessibility.

Level 1 PSG, also known as full attended in-laboratory polysomnography, is the reference standard for diagnosing sleep disorders. It is performed overnight in a sleep center under continuous monitoring by a trained sleep technologist. Standard sensors used in Level 1 include electroencephalography (EEG), electrooculography (EOG), electromyography (EMG) from the chin and anterior tibialis, nasal airflow (via pressure transducer and thermistor), thoracoabdominal respiratory effort belts, electrocardiography (ECG), SpO2, body position sensors, snore microphones, and—where indicated—end-tidal or transcutaneous CO2 monitoring. This setting allows real-time identification of technical artifacts, direct observation of sleep behavior, and immediate intervention for therapeutic titration, such as PAP adjustment. Level 1 PSG is the preferred modality in complex cases, including patients with suspected comorbid sleep disorders, cardiorespiratory instability, epilepsy, parasomnias, or neuromuscular disease.

Level 2 PSG mirrors Level 1 in terms of the physiological parameters monitored but is performed in an unattended setting, typically the patient's home. It includes the same full montage of EEG, EOG, EMG, airflow, respiratory effort, and oxygenation channels. While offering the advantage of recording in a natural sleep environment and reducing cost, the absence of a technologist increases the risk of undetected signal loss, patient noncompliance with sensor placement, and reduced data integrity. Level 2 studies may be appropriate for relatively stable patients in remote areas or those with logistical barriers to in-lab testing, but they are not suitable when seizure monitoring or real-time intervention is needed.

Level 3 studies, often referred to as cardiorespiratory polygraphy or home sleep apnea tests (HSAT), involve limited-channel recordings and exclude EEG. Typically, Level 3 includes at least four signals: airflow (via nasal cannula or thermistor), thoracoabdominal effort belts, SpO_2, and heart rate or ECG. Without EEG, sleep staging and arousal detection are not possible, limiting diagnostic precision, especially in patients with insomnia, suspected central sleep apnea, or sleep fragmentation of unclear etiology. Level 3 testing is generally reserved for patients with a high pretest probability of moderate to severe OSA without significant comorbidities. It should not be used to evaluate complex sleep disorders or to exclude mild SDB.

Level 4 studies represent the most basic form of sleep testing and typically involve one or two channels, usually pulse oximetry alone or combined with airflow or actigraphy. While not diagnostic on their own, Level 4 studies may be useful for screening purposes, especially in resource-limited settings, or as preliminary assessments in high-risk populations. However, their inability to distinguish between obstructive and central events, or to detect sleep fragmentation, makes them inadequate as stand-alone diagnostic tools.

In clinical practice, the choice of PSG level should be individualized based on diagnostic complexity, patient comorbidity, clinical urgency, and the need for therapeutic titration. While cost and accessibility are valid considerations, they should not compromise diagnostic accuracy or delay appropriate care. A careful understanding of the limitations and strengths of each PSG level is essential for optimal application in sleep medicine.

4. PSG parameters and interpretation framework

4.1. Sleep architecture, quantity and efficiency

PSG allows for detailed characterization of sleep architecture by quantifying transitions between rapid eye movement (REM) and non-REM (NREM) sleep, the latter subdivided into stages N1, N2, and N3. Stage N1 represents light sleep and the transition from wakefulness, while N2 is marked by the presence of sleep spindles and K-complexes. Stage N3, or slow-wave sleep, is characterized by high-amplitude delta waves and is associated with physiologic restoration and memory consolidation. REM sleep, defined by low-voltage mixed-frequency EEG, phasic eye movements, and muscle atonia, plays a critical role in emotional regulation and cognitive integration. Normal sleep proceeds in ultradian cycles alternating between NREM and REM approximately every 90–120 minutes, with REM periods increasing in duration as the night progresses. This progressive elongation of REM in later cycles has direct clinical implications—REM-predominant OSA may be easily missed without stage-specific analysis. With aging, there is a physiological shift marked by reductions in REM and N3 proportions, and a concomitant increase in N1 sleep and arousals, often leading to misinterpretation of normal aging as pathology [7].

Beyond staging, PSG evaluates core metrics of sleep continuity that provide clinically meaningful insight. Total sleep time (TST) refers to the cumulative duration of all sleep epochs and serves as a quantitative marker of sleep sufficiency. Sleep efficiency (SE), defined as TST divided by time in bed and expressed as a percentage, reflects global sleep consolidation, with values below 85% generally considered suboptimal. Sleep latency (SL), the interval between lights off and the onset of sleep, provides an index of sleep initiation, and latencies over 30 minutes are often suggestive of insomnia [9]. Wake after sleep onset (WASO), representing periods of wakefulness after initial sleep initiation, is a robust marker of sleep maintenance fragmentation. These metrics are essential not only for diagnosing insomnia but also for understanding the impact of coexisting sleep disorders such as OSA, periodic limb movement disorder, or circadian misalignment.

In clinical interpretation, it is crucial to not only quantify sleep stages but also assess the distribution of N1, N2, N3, and REM as percentages of total sleep time. For example, a REM proportion below 20% or slow-wave sleep below 15% may indicate disrupted architecture in adults, particularly when associated with arousals or abnormal event clustering. The quality of sleep—as reflected by continuity metrics, arousal index, and fragmentation—is as critical as quantity.

Recent studies highlight that these continuity metrics may predict long-term cognitive outcomes more robustly than traditional staging variables. In the Sleep and Dementia Consortium, higher sleep maintenance efficiency and shorter WASO were independently associated with superior global cognition over five years, whereas REM and N3 proportions were not predictive [14]. These findings argue for a shift in interpretive emphasis from staging percentages toward sleep stability, particularly in aging populations and those at risk for neurodegeneration.

Importantly, age-related changes in sleep continuity metrics must be interpreted within an individualized and age-adjusted framework. TST tends to decrease by approximately 10 minutes per decade, accompanied by reductions in sleep efficiency and slow-wave sleep, and increases in WASO and SL [7]. For instance, a sleep efficiency of 80% may be entirely appropriate in a healthy adult aged 70, but pathological in a 30-year-old. These normative shifts are further modulated by sex, body mass index, and comorbidities such as chronic pain or neuropsychiatric disorders [1, 13].

Sleep continuity disturbances can significantly impair daytime function even in the absence of respiratory pathology. A patient with a normal AHI may experience severe fatigue or cognitive impairment if WASO is high or sleep is fragmented by periodic limb movements or frequent arousals. Indeed, individuals with upper airway resistance syndrome (UARS) may present with significant daytime symptoms despite a normal AHI, and their diagnosis rests on recognizing disrupted sleep continuity. As Edwards et al have argued, PSG should not be reduced to a single-number interpretation; instead, its full diagnostic potential emerges when continuity metrics are integrated alongside architecture, arousals, and event morphology [3].

4.2. Respiratory events, oxygenation, and positional trends

Respiratory event analysis in PSG centers around the AHI, which quantifies the frequency of obstructive apneas (cessation of airflow with respiratory effort), central apneas (cessation without effort), and hypopneas (partial reductions in airflow accompanied by desaturation or arousal). Although AHI remains the most widely reported metric, it provides only a partial picture. It fails to account for the duration, morphology, or physiologic impact of individual events. The respiratory disturbance index

(RDI) expands upon AHI by including respiratory effort–related arousals (RERAs), which are particularly relevant in patients with UARS who may exhibit non-apneic flow limitation yet experience significant daytime sleepiness [5].

Oxygenation indices provide essential context for interpreting respiratory disturbance severity. Commonly reported parameters include the lowest oxygen saturation (LSAT), the percentage of total sleep time spent with SpO_2 below 90%, and cumulative desaturation indices. The oxygen desaturation index (ODI), typically defined as the number of ≥3% or ≥4% desaturations per hour of sleep, offers additional granularity—particularly in patients with mild AHI but heavy desaturation burden. Duration and depth of desaturations are increasingly recognized as more robust markers of hypoxic stress than event count alone [4].

In particular, prolonged and deeper desaturations—often from sustained apneas—have been associated with increased cardiovascular risk, systemic inflammation, and mortality, supporting the notion that desaturation burden may serve as a more robust prognostic marker than event count alone [2, 5].

Body position also has a significant influence on respiratory event expression. Supine positioning increases upper airway collapsibility, exacerbating obstruction in susceptible individuals. Positional OSA, typically defined by a supine AHI at least twice that of non-supine AHI, is a common phenotype and may be effectively addressed with positional therapy. Evidence shows that airway mechanics improve in lateral decubitus, reducing collapsibility even in the absence of changes in arousal threshold or ventilatory control parameters [5].

A comprehensive interpretation of PSG must go beyond numerical indices like AHI and incorporate event timing and pattern. Clustering of respiratory events during REM sleep may indicate REM-related OSA, while exclusive or exacerbated events during supine sleep point toward positional OSA—both requiring phenotype-specific management strategies.

A physiologically grounded interpretation of PSG should synthesize these elements—AHI, RDI, oxygen saturation trends, and body position effects—within the broader context of sleep architecture and clinical presentation. Reliance on summary indices without waveform review or sleep-stage stratification risks under-recognition of meaningful pathophysiology. REM-related OSA, for instance, often reflects the vulnerability of the atonic airway during REM sleep and may differ fundamentally from NREM-predominant OSA, which is more commonly associated with high loop gain or arousal threshold disturbances [3]. Integrating such insights allows clinicians to move beyond diagnostic labels toward individualized, mechanism-informed management strategies.

5. PSG interpretation in special population

Pediatric PSG requires a tailored interpretive framework due to age-specific differences in sleep physiology, respiratory control, and anatomical development. Sleep architecture in children is distinct from adults, with newborns spending 50–75% of their day asleep, and REM accounting for up to 50% of total sleep time. Circadian rhythm emerges around 6 months of age, and NREM-REM cycles are shorter—approximately 50 minutes in infants—gradually maturing into 90-minute cycles by adolescence. Preschool children may still nap once or twice per day, while school-aged children generally sleep 9–11 hours at night with no naps and demonstrate increased slow-wave sleep. These developmental features influence sleep efficiency and respiratory patterns, and interpretation of pediatric PSG must consider these shifting baselines to avoid misclassification of normal developmental variants as pathological [16, 17].

Respiratory event scoring in children differs fundamentally from adults, and stricter thresholds are used to define pathology. Apneas are scored when airflow reduction of ≥90% lasts for at least two missed breaths, while hypopneas require ≥50% reduction in nasal pressure, associated with either an arousal or ≥3% oxygen desaturation [9]. RERAs are defined by flattening of the nasal pressure waveform with increasing respiratory effort, often accompanied by snoring or rising CO_2. Central apneas are scored when there is complete absence of respiratory effort for two or more missed breaths, with or without associated arousal or desaturation. According to pediatric criteria, an AHI or RDI ≥ 1 event/hour is abnormal. Severity classification follows established cutoffs: mild (AHI/RDI 1–5), moderate (5–15), and severe (>15) [15]. Due to shorter sleep durations in children, as few as seven respiratory events during a full-night PSG may suffice for diagnosis, making precision in scoring essential [19].

Clinical indications for PSG in children extend beyond snoring or observed apneas. Children with adenotonsillar hypertrophy remain the most frequent referrals, but PSG is also essential for evaluating sleep in children with dysmorphic features, neuromuscular disease, growth failure, unexplained behavioral disturbances, and genetic syndromes. In these populations, symptoms such as hyperactivity, academic decline, or failure to thrive may be the only clues to significant SDB. PSG should be performed before initiating interventions, including surgery,

pharmacotherapy, or ventilatory support. In the case of Prader–Willi syndrome, achondroplasia, Crouzon syndrome, or spinal muscular atrophy, early PSG is strongly recommended due to the high prevalence of OSA and central hypoventilation in these groups [17].

The value of PSG in such populations lies not only in detecting apneas and hypopneas but also in assessing arousal burden, sleep efficiency, REM distribution, and CO_2 retention. In Oros et al.'s cohort of 108 children with complex disorders, 80.5% had OSA on PSG, with some subgroups—such as those with achondroplasia or Crouzon syndrome—showing near-universal prevalence [17]. Nocturnal hypoventilation was identified in 15% of patients, underscoring the need for end-tidal or transcutaneous CO_2 monitoring. Multidisciplinary management strategies included adenotonsillectomy, CPAP, BiPAP, and neurosurgical interventions, with treatment guided directly by PSG findings and titration studies.

Thus, PSG in pediatric and syndromic populations should not be viewed as a simplified adult test, but as a comprehensive neurorespiratory evaluation tailored to developmental physiology. Accurate diagnosis depends on meticulous scoring, age-adjusted interpretation, and synthesis of respiratory, sleep stage, and arousal data. In children with comorbid conditions, PSG is indispensable not only for diagnosis but also for establishing severity, guiding intervention, and tracking treatment response.

6. Clinical decision-making based on PSG

Interpreting a PSG study begins with confirming technical adequacy. This involves ensuring all essential signals—EEG, airflow, respiratory effort, oxygen saturation, and CO_2 monitoring—are present and interpretable throughout the night. Artifacts, particularly in airflow or effort channels, can obscure respiratory events and reduce diagnostic confidence. For a study to be valid, sufficient total sleep time (typically $\geq$6 hours) is needed to assess sleep efficiency, stage distribution, and event frequency. Incomplete recordings or those with inadequate sleep duration may require repetition, especially when discordant with clinical suspicion [20].

Once technical validity is established, PSG findings must be interpreted in the context of clinical symptoms. Objective indices such as AHI, RDI, RERAs, and LSAT are important but should not stand alone. For instance, a child with significant daytime sleepiness, hyperactivity, or growth concerns and a modest AHI may still have clinically relevant SDB if frequent arousals or desaturation clusters are present. Conversely, a high AHI in a minimally symptomatic patient may not necessitate immediate intervention if sleep efficiency is intact and desaturation burden is low. This disconnect between numerical severity and clinical impact has been well documented in both pediatric and adult populations and underscores the need to correlate PSG metrics with functional status [20]. PSG results should also be viewed alongside known systemic effects of SDB, including links to metabolic dysregulation, cognitive impairment, hypertension, and dyslipidemia.

PSG serves not only as a diagnostic test but as a cornerstone of precision sleep medicine. Identifying SDB phenotypes is essential, as these often represent "milder" forms that may respond well to conservative, non-invasive treatments. For example, positional therapy can reduce supine AHI by more than 50% in selected patients, with pooled analyses showing significant improvements in overall AHI and durable benefits in adherent individuals [21, 22]. Likewise, mandibular advancement devices lower AHI by about 50% in mild-to-moderate OSA, with slightly greater efficacy in non-obese phenotypes and those with REM-predominant disease [23, 24]. Incorporating phenotype assessment helps align therapy with underlying pathophysiology, improving both efficacy and patient acceptance. CPAP remains first-line for most patients with moderate-to-severe OSA, especially when excessive daytime sleepiness or cardiovascular risk factors are present. In children, adenotonsillectomy is preferred for OSA related to lymphoid hypertrophy. Positional therapy is useful in supine-predominant OSA, while weight loss is recommended in obesity-related cases, often alongside CPAP or surgical options. Importantly, PSG findings should be interpreted with data from drug-induced sleep endoscopy (DISE) when available, as DISE offers direct insight into airway dynamics during sleep and aids in site-specific planning. In cases where OSA persists despite CPAP or surgery, or when collapse patterns are unclear, combining PSG and DISE enhances treatment precision by correlating functional impairment with structural obstruction.

7. Pitfalls and pearls in PSG interpretation

Despite its diagnostic utility, PSG remains vulnerable to misinterpretation when reduced to summary metrics or viewed outside its clinical and physiological context. One of the earliest challenges is the "first night effect," where unfamiliarity with the sleep lab environment alters sleep architecture—typically reducing REM and slow-wave sleep—and may lead to underestimation of disease severity or missed detection of sleep-stage-dependent events. This highlights the need for caution when interpreting borderline results or unusually low sleep efficiency.

Technical artifacts present another source of error, especially when relying heavily on automated scoring without waveform validation. Displaced nasal cannulas, faulty effort belts, ECG noise contaminating EEG channels, or inconsistent oximetry signals can all compromise data quality. Over-reliance on auto-scoring risks misclassification of sleep stages and respiratory events. Manual review by experienced technologists and sleep physicians is critical to differentiate true physiological events from artifacts, particularly when findings are borderline or discordant with symptoms.

Inter-scorer variability—especially in sleep staging and scoring of events without clear desaturation or arousal—can also significantly affect indices like AHI and arousal burden, underscoring the need for rigorous scorer training and periodic concordance checks [25, 26]. Emerging machine learning tools show promise in improving scoring consistency, event detection, and phenotype classification, potentially reducing some of these human variability challenges [27].

Although the AHI remains a widely used and convenient metric for classifying sleep-disordered breathing, it does not fully capture the physiological burden of the disorder. Important features such as event duration, depth of desaturation, arousal frequency, and REM- or position-specific vulnerabilities may be overlooked when relying on AHI alone. While Edwards et al. [3] highlighted these limitations, more recent work by Malhotra et al. [28] and Stepnowsky et al. [27] underscores how integrating measures like ODI and hypoxic burden can further refine risk stratification. Incorporating adjunct metrics—including ODI, LSAT, arousal index, and stratified AHI—thus provides a more complete picture of disease impact. As Malhotra et al. emphasize, these complementary parameters more closely link sleep-disordered breathing to adverse outcomes, while Ruehland et al. demonstrated that even variations in hypopnea scoring significantly affect AHI and OSA prevalence, reinforcing the need for multiparametric interpretation to guide individualized care [28, 29]. This ensures that clinical decisions reflect not just numerical thresholds but also patient symptoms, comorbidities, and functional impact.

8. Conclusion

Effective interpretation of PSG relies on more than accurate scoring—it requires clinical judgment, attention to physiological context, and individualized application. While indices such as AHI, RDI, and sleep efficiency provide quantitative anchors, their significance must be weighed alongside symptom burden, comorbidities, and demographic factors such as age and sleep stage distribution. Recognizing patterns such as REM-related or positional OSA, integrating arousal indices, and identifying artifacts are essential steps in transforming PSG data into actionable clinical insight. Ultimately, PSG interpretation is not a numerical exercise but a clinical decision-making process that bridges physiology with patient-centered care. Importantly, although such comprehensive approaches may be more resource-intensive initially, they hold promise for improving long-term cost-effectiveness by guiding precise, mechanism-based interventions that can reduce morbidity and healthcare utilization.

Conflict of interest

The authors declare no conflict of interest.

Ethical approval

Ethical review and approval were waived for this study, due to the fact that no human trial was conducted.

Data availability

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Author contributions

MY: Conceptualization; Writing — Original Draft; Writing Review & Editing. **EZ:** Conceptualization; Supervision; Writing Review & Editing.

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Appendix 1. Step-by-Step approach to interpreting a PSG report

This stepwise approach is proposed as a practical framework reflecting expert consensus and best practices; it is not a formally validated protocol and should be applied in conjunction with individual clinical judgment and patient-specific considerations.

	Focus Area	Action Item
Step 1	Confirm Technical Adequacy	Ensure ≥6 hours of total sleep time (TST) Verify signal quality across EEG, airflow, respiratory belts, SpO_2, ECG Identify and annotate artifacts; re-score affected epochs if needed
Step 2	Review General and Anthropometric Data	Record age, sex, BMI Note clinical indication and relevant comorbidities (e.g., obesity, HF)
Step 3	Assess Sleep Architecture and Continuity	Evaluate TST, sleep efficiency, sleep latency, and WASO Assess stage distribution (N1, N2, N3, REM) Determine arousal index and assess for sleep fragmentation
Step 4	Examine Respiratory Parameters	Analyze AHI and RDI Differentiate obstructive, central, mixed apneas, and RERAs Compare REM vs. NREM AHI Assess positional AHI (supine vs. non-supine)
Step 5	Evaluate Oxygenation and CO_2 Trends	Note lowest SpO_2 (LSAT) and % time <90% saturation Review oxygen desaturation index (ODI) Evaluate CO_2 trends (if available); flag CO_2 >50 mmHg >25% of TST
Step 6	Identify Other Physiologic or Behavioral Findings	Check PLMI if PLMD suspected Review video/audio for parasomnias, choking, seizure-like events Recommend extended EEG if epileptiform activity suspected
Step 7	Correlate PSG with Clinical Symptoms	Match findings with symptoms (e.g., EDS, insomnia) Consider UARS in symptomatic patients with normal AHI Integrate age, BMI, comorbidities into interpretation
Step 8	Formulate Diagnosis and Management Plan	Assign SDB severity (mild, moderate, severe) Tailor treatment: CPAP, positional therapy, MADs, DISE, surgery Decide if follow-up PSG is needed (e.g., post-therapy reassessment)

Step 1: Confirm Technical Adequacy

1. Open the raw PSG data or summary report.
2. Check the total sleep time (TST); ensure at least 6 hours of recorded sleep to allow representative staging and respiratory analysis.
3. Review signal quality: Scroll through key channels (EEG, EOG, EMG, airflow, respiratory effort belts, SpO_2, ECG). Identify any signal dropout or artifact periods.
4. Annotate artifacts manually or verify the automated artifact log. Re-score affected epochs if necessary.
5. Confirm that all channels—especially respiratory and oxygenation signals—are interpretable across the full study duration.

Step 2: Review General and Anthropometric Data

1. Locate the patient demographics section.
2. Record age, sex, and BMI, as these influence respiratory thresholds and staging norms.
3. Confirm the clinical indication (e.g., suspected OSA, post-operative monitoring, titration), which guides interpretation scope.

4. Document comorbidities such as cardiovascular disease, cognitive impairment, or metabolic syndrome, as PSG metrics may correlate with systemic burden.

Step 3: Assess Sleep Architecture and Continuity

1. Review the sleep stage histogram or hypnogram to assess distribution of N1, N2, N3, and REM.
2. Extract:
   - TST: Total minutes spent asleep.
   - Sleep efficiency: % of time asleep divided by total time in bed.
   - Sleep latency: Time from "lights out" to first sleep epoch.
   - WASO: Time awake after initial sleep onset.
3. Find the arousal index (number of arousals per hour of sleep). Note whether arousals are spontaneous or respiratory-related.
4. Evaluate fragmentation: Are arousals clustered in a specific sleep stage or position? Does the hypnogram show sleep instability or failed REM transitions?

Step 4: Examine Respiratory Parameters

1. Locate the respiratory event summary.
2. Extract:
   - AHI: Total apneas + hypopneas per hour of sleep.
   - RDI: Includes RERAs along with apneas and hypopneas.
   - Event types: Count and differentiate obstructive, central, mixed, and RERA events.
3. Stratify AHI and RDI by sleep stage (REM vs. NREM) to identify REM-related OSA.
4. Stratify by sleep position (supine vs. non-supine) to detect positional OSA.
5. Highlight patients with supine-predominant or REM-predominant patterns—these phenotypes may respond to non-CPAP interventions.

Step 5: Evaluate Oxygenation and CO_2 Trends

1. Open the oximetry summary:
   - Note LSAT (lowest oxygen saturation during sleep).
   - Record % of sleep spent with $SpO_2 < 90\%$.
   - Review the oxygen desaturation index (ODI), especially in patients with modest AHI but significant desaturation burden.
2. If available, review CO_2 trend data:
   - Extract transcutaneous or end-tidal CO_2 values.
   - Note if $CO_2 > 50$ mmHg for more than 25% of TST—this may indicate nocturnal hypoventilation, particularly in neuromuscular or obesity-hypoventilation syndromes.

Step 6: Identify Other Physiologic or Behavioral Findings

1. Review leg EMG channels to calculate the periodic limb movement index (PLMI).
2. If PLMS are present but not the main clinical concern, document only their presence—full scoring is not necessary unless PLMD is suspected.
3. Evaluate video and audio:
   - Check for snoring, choking, or abnormal motor events.
   - Document suspected parasomnias or seizure-like activity.
4. Refer for extended EEG if epileptiform activity is suspected.

Step 7: Correlate PSG with Clinical Symptoms

1. Compare PSG metrics with the patient's presenting symptoms (e.g., EDS, insomnia, behavioral problems).
2. If symptoms are disproportionate to AHI, investigate for UARS or non-respiratory sleep disruption.
3. Integrate PSG findings with clinical context, including age, BMI, comorbidities, and functional impairments.
4. View PSG not only as a diagnostic tool but as a guide for targeted therapy and long-term disease monitoring.

Step 8: Formulate Diagnosis and Management Plan

1. Use AHI/RDI to classify severity (mild, moderate, severe).
2. Tailor recommendations:
   - CPAP for moderate-to-severe OSA or REM-predominant OSA.
   - Positional therapy for positional OSA.
   - Weight loss or surgery as indicated.
   - Consider mandibular advancement devices in mild or positional cases with anatomical suitability.
   - DISE if airway collapse pattern remains unclear or if surgical treatment is planned.
3. Determine whether follow-up PSG is needed—e.g., after PAP titration, surgery, or significant weight change.