



Interpreting the mandibular jaw movement signal in pediatric obstructive sleep apnea diagnosis: A technical and practical review

Jean-Benoit Martinot^{a,b,*}, Nhat-Nam Le-Dong^c, Julie Cassibba^d, Didier Clause^a, Jean-Louis Pépin^{e,f,1}, David Gozal^{g,1}

^a Sleep Laboratory, CHU Université catholique de Louvain (UCL), Namur Site Sainte-Elisabeth, Namur, Belgium

^b Institute of Experimental and Clinical Research, UCL Bruxelles Woluwe, Brussels, Belgium

^c Sunrise, Namur, Belgium

^d Pediatric Department, Grenoble Alpes University Hospital, Grenoble, France

^e HP2 Laboratory, INSERM U1300, Grenoble Alpes University Hospital, Grenoble, France

^f EFCR Laboratory, Thorax and Vessels Division, Grenoble Alpes University Hospital, Grenoble, France

^g Department of Pediatrics, Joan C. Edwards School of Medicine, Marshall University, Huntington, WV, USA

ARTICLE INFO

Handling Editor: Monica Andersen

Keywords:

Obstructive sleep apnea
Pediatric
Home sleep apnea testing
Mandibular jaw movements

ABSTRACT

Pediatric obstructive sleep apnea (OSA) is a common yet often underdiagnosed condition, partly due to limited access to polysomnography. Mandibular jaw movement (MJM) analysis offers a promising alternative to conventional home sleep apnea testing in children, capturing the dynamic interactions between respiratory drive and upper airway musculature, enabling accurate identification of, and critical insights into, sleep-disordered breathing events.

This technical and practical review provides a structured framework for understanding and interpreting MJM signals during sleep in pediatric patients. It begins with the physiological basis and technical aspects of using a single-point contact device with integrated inertial sensors. It offers step-by-step instructions for interpreting MJM signals, from distinguishing sleep stages to identifying obstructive and central apneas, hypopneas, and respiratory effort-related arousals. The review is accompanied by an atlas of 30 annotated examples that illustrate key MJM signal patterns across scoring tasks.

Key findings from several clinical studies on the utility of MJM analysis in pediatric OSA are also summarized.

As the demand for accessible and accurate home-based diagnostic tools grows, MJM analysis stands out as an effective option for both the diagnosis and monitoring of pediatric OSA, with the potential to transform routine clinical practice and improve patient access to care.

Abbreviations

Terms	Definition
Arl	Arousal index
ECG	Electrocardiogram
EEG	Electroencephalogram
EMG	Electromyography
EOG	Electro-oculogram
FlowPr	Nasal airflow pressure
FlowTh	Oro-nasal thermal flow
AHI	Apnea-hypopnea index
HSAT	Home sleep apnea testing

(continued on next column)

(continued)

MJM	Mandibular jaw movement
MJM_Acc	MJM signal recorded by accelerometer
MJM_Gyr	MJM signal recorded by gyroscope
NREM	Non-rapid eye movement sleep
OSA	Obstructive sleep apnea
RE	Respiratory effort
REM	Rapid eye movement sleep
REMOV	Respiratory effort burden derived from MJM
RERA	Respiratory effort-related arousals
RDI	Respiratory disturbance index

(continued on next page)

* Corresponding author. Centre du Sommeil et de la Vigilance. CHU Université catholique de Louvain, Namur Site Sainte-Elisabeth. 15, Place Louise Godin. 5000 Namur, Belgium.

E-mail address: martinot.j@respisom.be (J.-B. Martinot).

¹ Co-senior authors.

<https://doi.org/10.1016/j.smr.2025.102159>

Received 9 May 2025; Received in revised form 30 July 2025; Accepted 21 August 2025

Available online 29 August 2025

1087-0792/© 2025 Elsevier Ltd. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

(continued)

RIP	Respiratory inductance plethysmography
RMMA	Rhythmic masticatory muscle activity
SOL	Sleep onset latency
SpO2	Peripheral capillary oxygen saturation
TMJ	Temporomandibular joints
TST	Total sleep time
WASO	Wake after sleep onset

1. Introduction

Obstructive sleep apnea (OSA) is not only prevalent in adults but also commonly affects children across all age groups, including infancy and early childhood. The prevalence of pediatric OSA is estimated to be as high as 5.7 % [1] and is increasingly recognized due to the rising incidence of obesity in young children [2].

Like in adults, untreated OSA in children is associated with cardiovascular and metabolic disorders [3,4]. In addition, pediatric OSA can lead to age-specific adverse outcomes such as neurobehavioral impairments that hinder academic performance [5–7], as well as a heightened risk of infections and asthma exacerbations [1,3].

Despite its impact, a significant proportion of children with OSA—up to one-third—remain untreated, revealing a critical gap in the comprehensive management of pediatric OSA [8]. A major contributing factor to this gap is underdiagnosis, largely due to the high cost and limited availability of in-laboratory polysomnography (PSG). These barriers often delay or prevent timely diagnosis, particularly in children with suspected OSA, thereby increasing the likelihood of untreated cases. This situation underscores the need for broader adoption of home sleep apnea testing (HSAT) as a more accessible and scalable diagnostic alternative for pediatric populations [9–11]. Although the validity and accuracy of HSAT in children remain under debate due to limited supporting evidence [12], recent advances in sleep monitoring technology have expanded its potential application in pediatric sleep medicine. Proposed alternatives include type III and IV devices [2], overnight oximetry [13], nasal cannula transducers [14], and advanced analytic technologies leveraging novel bio-signals [15–17]. However, compared to adult populations, research on wearable HSAT technologies in children [18,19] remains scarce, with limitations in study quantity, validation quality, and technical transparency.

In this context, mandibular jaw movement (MJM) analysis has recently emerged as a promising technology that offers a simple, convenient, yet reliable and effective approach for diagnosing OSA in children. MJM signals provide valuable insights into respiratory drive during sleep, supporting detailed scoring of sleep architecture, continuity, and the differentiation between central and obstructive respiratory events. The clinical utility of MJM analysis has been validated in pediatric populations [20–22]. However, current signal interpretation guidelines for MJM remain largely based on adult data [23,24], with no publications to date specifically addressing pediatric populations. As a result, MJM analysis is still emerging as a tool within pediatric sleep medicine and is not yet widely adopted in routine practice.

This technical and practical review seeks to address this gap by providing a comprehensive guide for interpreting MJM signals in the pediatric population. It aims to equip clinicians and sleep technicians with the knowledge and tools necessary to effectively apply MJM analysis in the diagnosis and monitoring of pediatric OSA.

2. Overview of the anatomical and physiological foundation of the MJM signal

The lower jaw, or mandible, as part of the stomatognathic system, is a mobile structure articulated at the bilateral temporomandibular joints (TMJs). This structure includes the mandibular condyle and its articular disc. MJMs rely on the coordinated masticatory muscles — innervated

by the mandibular branch of the trigeminal nerve and governed by the corticobulbar motor pathway — while the respiratory centers in the brainstem superimpose a phasic drive that synchronizes jaw muscle tone with the breathing cycle to maintain upper-airway patency. Among the involved muscles, the lateral pterygoid muscle plays a key role in mandibular motion, including elevation, depression, lateral excursion, and protrusion [25]. Other muscles are functionally grouped into mandibular elevators—such as the temporalis, masseter, and medial pterygoid—and mandibular depressors, including the mylohyoid, geniohyoid, and digastric muscles (see Fig. 1).

MJMs comprise two primary actions of the condyle: (1) rotational (hinge-like) motion between the disc and condylar head, and (2) translational (gliding) motion between the disc and the temporal surface of the joint [26]. These movements are not only essential for mastication during wakefulness but also play a critical role in maintaining upper airway patency during sleep [27]. As muscle tone decreases during sleep, the mandible tends to drop and shift posteriorly, which can influence upper airway patency. The upper airway musculature, including muscles linked to the mandible, coordinates a range of vital functions—breathing, swallowing, speech, and mastication—with the mandible serving as a central structural element [28,29].

During wakefulness, MJMs are typically rapid, variable in amplitude and frequency, and unpredictable. In contrast, MJMs during sleep reflect a more regulated pattern relying on a delicate dynamic balance. Specifically, the mandible acts as a mechanical lever, helping to stiffen and increase the rigidity of the pharyngeal walls just a few tenths of milliseconds before diaphragmatic contraction. The MJM signal thus captures the neuromuscular engagement of jaw muscles involved in ventilatory drive, providing key insights into the biomechanics of airflow through the upper airway during sleep [30–34].

Under normal respiratory conditions, MJM signals closely mirror airflow patterns [24]. These movements, regulated by the brainstem’s respiratory centers during sleep, occur at frequencies corresponding to the respiratory rate (0.15–0.60 Hz), indicating a predominantly brainstem-driven process. The motor nucleus of the trigeminal nerve’s motor branch—located in the mid-pons—innervates both the agonist and antagonist muscles of the mandible. The rhythmic MJMs observed during non-rapid eye movement (NREM) are driven by interactions between the motor trigeminal nerves and respiratory centers in the brainstem. A similar neuroanatomical relationship exists between the hypoglossal nucleus, which controls tongue movement during sleep, and the spinal respiratory motor neurons associated with diaphragmatic and accessory muscle activity [35,36].

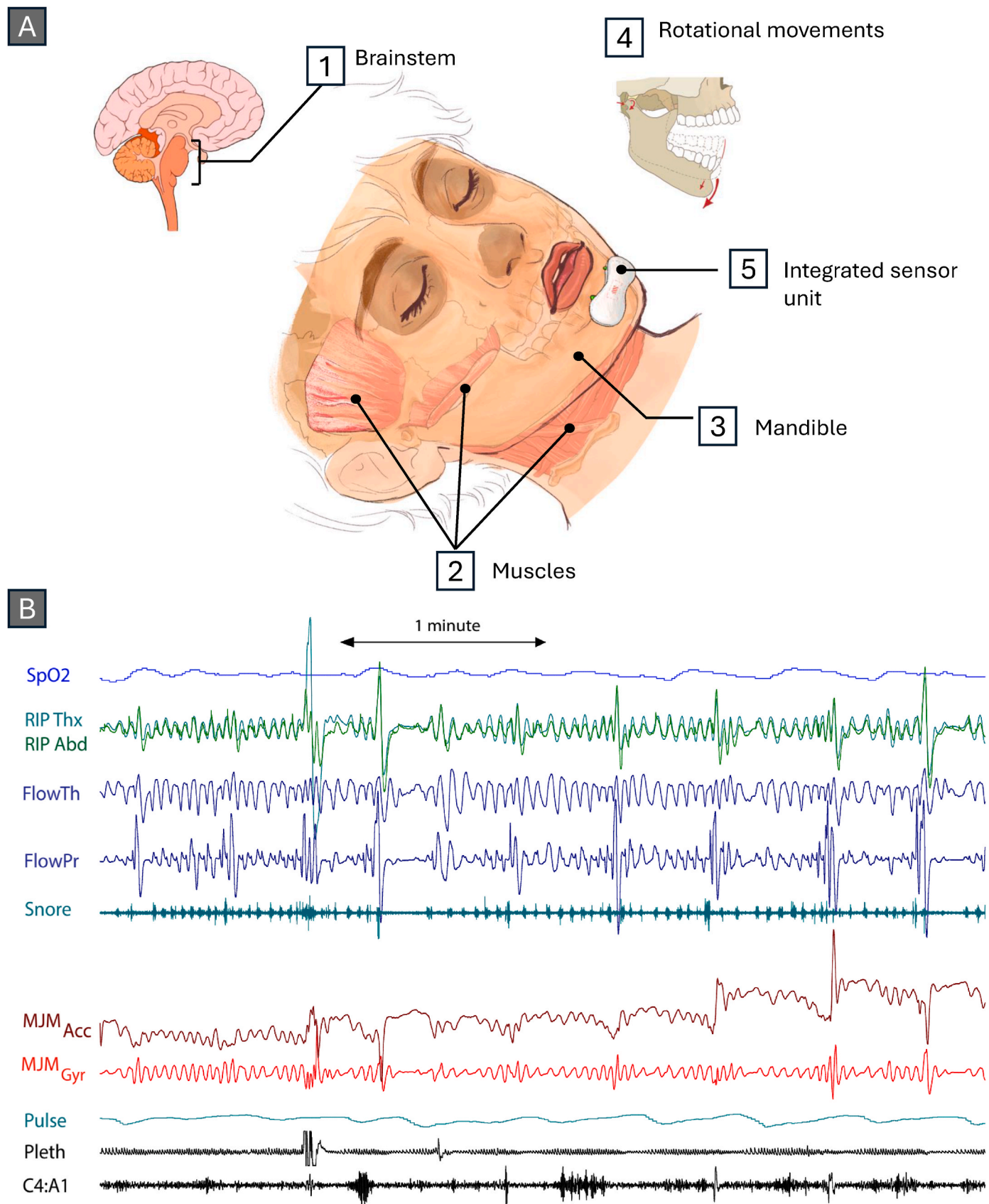
The stiffening of the pharynx prior to diaphragmatic contraction is crucial to prevent dynamic airway collapse caused by negative intrathoracic pressure. This negative pressure reflex likely involves recruitment of trigeminal motoneurons, resulting in MJMs. The central coordination of this activity, involving direct and indirect connections between the mandible, hyoid bone, and upper airway muscles, underpins the role of MJMs as a functional indicator of pharyngeal stability and respiratory drive during sleep.

3. Technical aspects of MJM signal recording

3.1. Overview of MJM monitoring technologies

MJMs can be studied using distinct technologies: from the complex historical magnetometry to optical jaw movement tracking device [20, 23,37–39], and more recently, with a single-point sensor incorporating both accelerometer and gyroscope components, which allows for less invasive recording during sleep [21,22]. While these approaches aim to capture the same jaw movements, they differ significantly in terms of sensor design and physical properties, measured quantities and the physiological interpretation of the signals.

This review focuses on the interpretation of sleep-related MJM signals acquired through the recent technology using a single sensor



(caption on next page)

Fig. 1. Anatomical and physiological basis of MJM monitoring

Legend: A) Schematic illustration of the neural and muscular mechanisms regulating the mandibular jaw movements (MJMs) during sleep. The brainstem (1) coordinates the activation of the elevator and depressor muscles (2), generating rotational motion at the temporomandibular joint (4). The mandible (3) acts as a mechanical lever to stiffen pharyngeal muscles, thereby maintaining upper airway patency. An integrated inertial sensor unit affixed to the chin (5) captures the acceleration and rotational speed of the mandible. B) A 4-minute fragment of polysomnography (PSG) recording with integrated MJM signals from a child, illustrating specific patterns of MJM signals associated with obstructive and central hypopneas during N2 sleep. Abbreviations: MJM_Acc/MJM_Gyr: MJM signals recorded via the accelerometer or gyroscope embedded in the sensor; FlowPr: nasal airflow pressure; FlowTh: oro-nasal thermal flow; Snore: snoring sound recorded via microphone; Pleth: plethysmography; C4:A1: electroencephalogram; RIP Abd & RIP Thx: abdominal and thoracic respiratory inductive plethysmography; SpO2: pulse derived oxyhemoglobin saturation.

equipped with a gyroscope (MJM_Gyr) and an accelerometer (MJM_Acc). However, to provide context, we briefly describe the technical principles of magnetometric recordings and highlight key differences between the two approaches.

3.2. Magnetometric-based recording: principles and limitations

The magnetometric sensor (Nomics, Liège, Belgium) was commonly used in early MJM research (2008–2019) [23,37–42]. This technique relies on two coupled resonant circuits: a transmitter and a receiver positioned on the forehead and the chin, respectively. This technique measures vertical displacement by detecting changes in the distance between the two circuits, enabling the determination of the jaw's position. The electronic module of the sensor converts the measured distance into a voltage signal, typically sampled at a rate of approximately 10 Hz. The output voltage is directly proportional to the vertical separation between the transmitter and receiver, meaning that movements such as mouth opening generate measurable changes in signal amplitude. These changes can be expressed either in millimeters of displacement or as a normalized percentage based on an individual calibration range.

Despite its initial contribution to establishing the physiological basis of MJM in sleep medicine, magnetometric technology presents several limitations compared to integrated inertial sensor systems. Key drawbacks include a loss of precision due to potential misalignment of the magnetometers and discomfort related to the size and configuration of the sensors—issues particularly problematic in pediatric populations. These factors significantly limit the feasibility of magnetometric systems in home-based sleep studies. Additionally, magnetometric emitters may interfere with pulse oximetry, disrupting concurrent physiological measurements.

Magnetometry also captures only a single dimension of mandibular movement—vertical displacement—while lateralized or complex motions, such as those associated with sleep-related bruxism, remain undetectable. Moreover, this technology measures only the distance between two fixed points rather than providing a comprehensive analysis of the mandible's motion dynamics. Consequently, while magnetometry offers valuable information on mouth opening, it does not capture the full range of mandibular movements, and thus limits its effectiveness in certain clinical contexts.

3.3. Inertial sensor-based MJM monitoring: a modern approach

Introduced in 2019, the latest MJM monitoring technology (Sunrise, Namur, Belgium) marked a significant advancement in sleep research and OSA diagnosis [20–22,24,43]. This technology employs a single-point contact sensor attached to the chin, integrating a tri-axial accelerometer and a tri-axial gyroscope (see Fig. 1). The accelerometer measures both gravitational acceleration and acceleration of the mandible (in mm/s²), providing information on mandibular position, while the gyroscope captures rotational velocity (in °/s), quantifying the angular displacement of the mandible around the temporomandibular joint. Together, these two components generate six independent channels (3 MJM_Acc channels and 3 MJM_Gyr channels), enabling the three-dimensional characterization of MJMs during sleep. MJM signals are sampled at 25 Hz and filtered within the 0.15–0.60 Hz range, aligning with respiratory cycle frequencies and ensuring a faithful

representation of sleep-related ventilatory activity.

While tri-axial accelerometers have been widely used in actigraphy-based sleep monitoring [44], the application of this technology to the lower mandible produces fundamentally different signals. This divergence stems not only from anatomical and physiological differences but also from the interpretation of MJM signal. Furthermore, MJM_Acc differs from intraoral accelerometers embedded in oral appliances [45], which are designed primarily for monitoring adherence to oral appliance therapy. These intraoral systems lack the specificity and resolution required to accurately assess respiratory activities or support sleep staging and sleep-disordered breathing (SDB) event detection.

The integration of accelerometer and gyroscope data provides critical advantages in terms of dimensionality, signal resolution, and interpretability. Unlike magnetometric measurements, the amplitude of MJM_Acc and MJM_Gyr signals is independent of mandibular length, making them more robust to anatomical variability across age groups [46]. The MJM signal captures the interplay between the central respiratory drive (mediated by the trigeminal motor nucleus in the pons), brainstem respiratory centers, and modulatory influences from higher brain centers involved in sleep-stage regulation. It also reflects the activity of muscles anchored to the mandible that maintain pharyngeal patency during sleep.

Importantly, MJM_Acc and MJM_Gyr signals complement one another. Information on mandibular positioning from MJM_Acc enhances the interpretation of amplitude variations in MJM_Gyr, particularly under specific physiological conditions. Conversely, MJM_Gyr provides necessary information to MJM_Acc signal, supporting full sleep staging, something not achievable with MJM_Acc alone.

3.4. Technical reliability of inertial sensor-based MJM recording

The high technical failure rates of pediatric sleep tests using type-3 and type-4 devices [2] remain a major challenge to implementing this approach in children. In a previous clinical study assessing the reliability of MJM analysis via inertial sensors for diagnosing pediatric OSA, the failure rate was only 3.4 % (5/145 recordings), primarily due to wireless connection issues [21]. Notably, recent clinical studies involving both adults [47,48] and children [22] have not reported technical failures with the latest generation of the MJM sensor.

3.5. Integrity of MJM signal across adverse conditions

As MJMs represent a physiologically essential and robust activity involved in maintaining upper airway patency during both wakefulness and sleep, their acquisition is minimally affected by adverse conditions, including systemic pathology, pharmacological treatment, or positional changes. MJM-based monitoring can be reliably performed in patients under antihypertensive treatment [49]. Head position, including side-lying with facial pressure against a pillow, has not been shown to compromise the integrity of the MJM signal. Mandibular motion is supported by the activity of the masticatory muscles, which are among the strongest muscles in the human body [50]. Secure placement of the sensor on the jaw ensures reliable signal acquisition across all head positions. The only clinical conditions that may affect MJM signal acquisition involve structural abnormalities of the temporomandibular joint (e.g., condylar malformations in Pierre Robin sequence). In rare

neuromuscular disorders such as Steinert's disease [51], MJM amplitude may be reduced due to impaired muscle function. While validation data in such populations remain limited, clinical experience indicates that MJM signals remain observable and clinically interpretable. Empirical support for this observation comes from recordings in REM-related muscle atonia, where the MJM signal has been shown to be detectable and clinically meaningful [52].

3.6. Interpretability of MJM signal

MJM signals offer clear and straightforward interpretation rules for detecting OSA-related events. Physiological data show that MJM oscillations closely mirror esophageal pressure (OeP) with high synchrony [43]. During cortical arousals, while esophageal pressure trends toward barometric levels, MJM signals indicate mouth closure, marking the

moment of flow restoration. The MJM signal remains unaffected by thoracic volume shifts and directly reflects respiratory trigeminal drive, resulting in clear and interpretable signal patterns. Notably, MJM retains high predictability even during elevated respiratory effort.

Evidence from magnetometric MJM recordings confirmed this robustness in adults, with a mean inter-scorer agreement of 90.7 % for detecting apneas, hypopneas and RERAs [23]. However, equivalent data in pediatric OSA are lacking, reflecting the ongoing integration of MJM analysis in pediatrics and the absence of standardized scoring protocols. This highlights the need for future studies in pediatric patients to evaluate inter-scorer agreement, for which the guidelines proposed here may serve as a foundation.

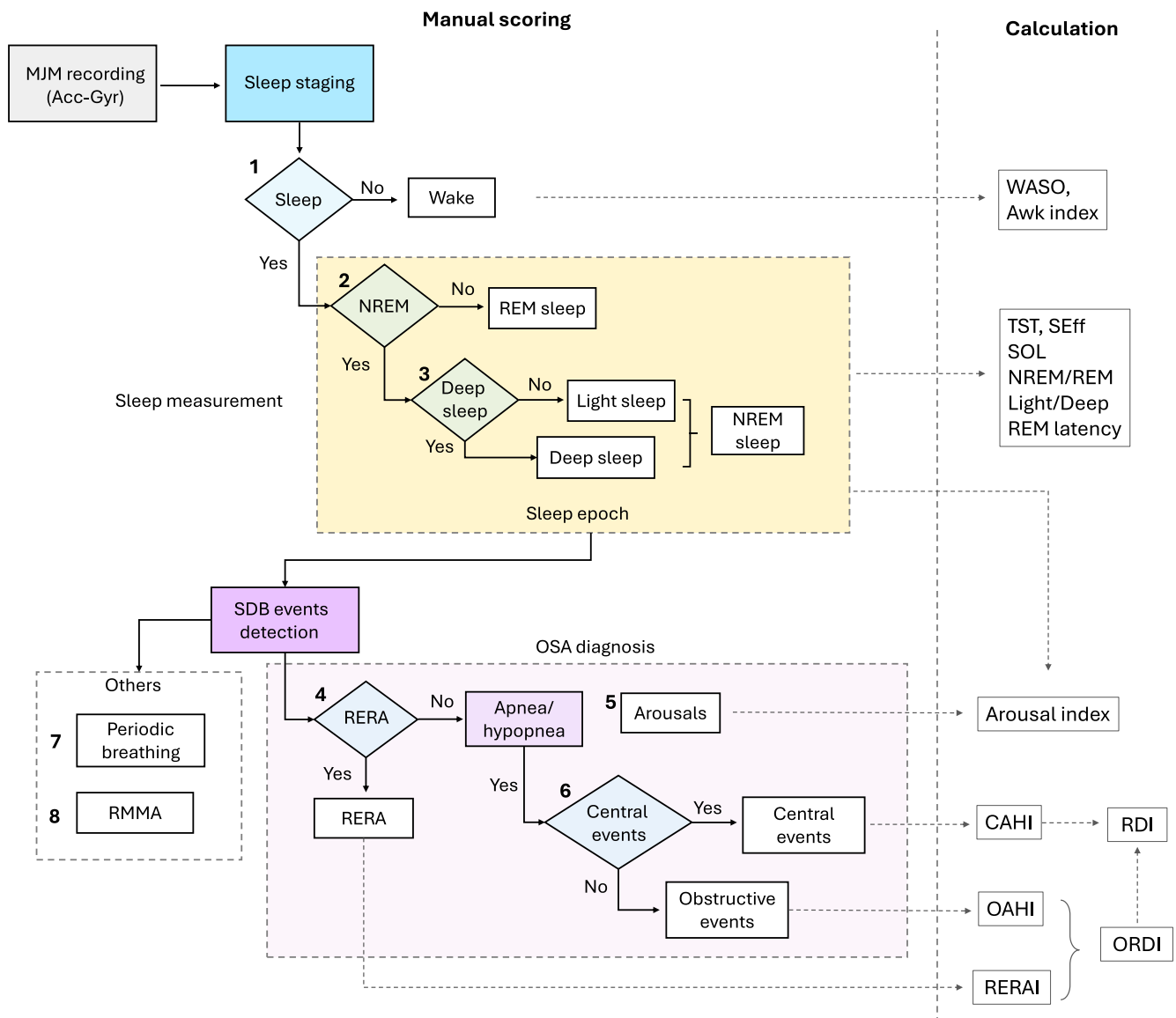


Fig. 2. Algorithm for manual interpretation of MJM signal

Legend: Acc: accelerometer; Gyr: gyroscope; WASO: wake after sleep onset; Awk: awakening; TST: total sleep time; SOL: sleep onset latency; NREM: non-REM; REM: rapid-eye movement; RERA(I): respiratory effort-related arousal (index); SDB: sleep-disordered breathing; SEff: sleep efficiency; OAH I/CAHI: obstructive/central apnea-hypopnea index; (O)RDI: (obstructive) respiratory disturbance index; RMMA: rhythmic masticatory muscle activity.

4. Principles of MJM signal interpretation

MJM signal recordings can be performed either independently or in combination with standard sleep studies, such as conventional PSG or multichannel polygraphy (PG), using type III or IV devices in accordance with established protocols, in sleep laboratories or in-home settings. In clinical practice, MJM signals can be interpreted in several ways: through manual scoring [20,23,39], automated analysis using artificial intelligence-based algorithms [21,22], or as an additional channel integrated into the manual PSG scoring framework [20,39].

Fig. 2 presents a hierarchical algorithm designed to guide MJM signal interpretation in pediatric sleep studies. Fundamentally, the interpretation of MJM signals follows principles similar to those used for conventional physiological signals. The interpretation workflow is structured to address multiple clinical objectives, including the evaluation of sleep architecture and the identification of respiratory events—such as apneas, hypopneas, and respiratory effort-related arousals (RERAs)—as well as distinguishing between obstructive and central events.

In this guide, we demonstrate how each of these tasks can be performed entirely through the visual analysis of MJM_Gyr and/or MJM_Acc signals. The main principle involves analyzing the behavior of MJM signals and applying predefined interpretation rules to recognize specific patterns associated with a targeted event, such as distinct sleep stages, arousals, obstructive or central respiratory events. By integrating the outcomes of all scoring tasks, conventional clinical indices can be generated from MJM recordings. These include total sleep time (TST), sleep onset latency (SOL), wake after sleep onset (WASO), apnea-hypopnea index (AHI), respiratory disturbance index (RDI), or arousal index (Ari) (see Fig. 2).

Beyond conventional hourly indices, the cumulative duration of obstructive respiratory events (also known as REMOV, expressed as % of TST) can be a valuable metric for assessing the burden of respiratory effort and its impact on sleep continuity. The clinical relevance of REMOV has been demonstrated in both adult and pediatric populations [39,48,53].

4.1. A visual guide to interpreting MJM signals

The companion atlas included with this review provides a comprehensive visual reference for interpreting MJM signal patterns, with 30 representative examples covering various scoring tasks (Supplementary Document, Figures S1–S30). In each example, MJM_Acc and MJM_Gyr signals are displayed in synchrony with conventional physiological signals—including abdominal/thoracic respiratory inductance plethysmography (RIP), airflow, SpO₂, electroencephalography (EEG), and electrooculography (EOG)—to support comparative analysis and enhance interpretability. Figures S1A, S1B, and S2 first illustrate the fundamental principle of MJM signal recording, clearly demonstrating the dynamic interaction between mandibular movement and respiratory drive during a prolonged period of respiratory effort.

4.2. Interpreting sleep structure

Accurate differentiation between wakefulness and various sleep stages is critical for evaluating sleep architecture and duration. In Figure S3, as a child transitions from wakefulness to sleep, MJM patterns become more stable, contrasting with the larger, more erratic displacements seen during wakefulness.

Figure S4 illustrates how MJM signals change across NREM stages. There is a clear incremental rise in the angular speed of the mandible as the child progresses into deeper sleep. During N1, ventilatory control is more unstable, and subtle alterations in jaw position often coincide with variable EEG patterns. During N3, increased respiratory drive is reflected in larger jaw rotations, likely in response to heightened upper airway resistance.

Figure S5 captures the transition from N2 to REM sleep. During REM sleep, MJM remains active, unlike the reduced electromyographic activity of the genioglossus muscle, suggesting that the mandible plays a key role in maintaining airflow during this stage. Mandibular jaw behavior in REM exhibits stochastic variations in both frequency and amplitude, features that aid in visually identifying REM sleep (Figures S5, S6 and S7). Conversely, Figure S8 depicts the transition from REM to N2, where MJM amplitude and frequency stabilize, aligning with the typical characteristics of N2 and a reduction in rapid eye movements.

Figures S9A and S9B further illustrate the mandibular twitch—a brief, phasic burst of muscle activity typical of REM sleep—and show how these twitches are temporally aligned with corresponding changes in EEG and EOG signals.

4.3. Identifying sleep-disordered breathing events during sleep

Once sleep periods are accurately identified, the next interpretation involves classifying respiratory events, such as apneas, hypopneas, flow limitations, and RERAs.

Figure S10 presents a period of quiet, stable breathing with low mandibular rotational velocity during sleep, serving as a reference baseline against which variations observed in SDB events can be compared.

The angular velocity and displacement of the mandible serve as sensitive and reliable markers of respiratory efforts (RE), as shown in Figures S11, S12A, S12B and S13. Periods with increased RE are indicated by elevated peak-to-peak angular velocities (MJM_Gyr) typically exceeding the 75th percentile ($2.57^\circ/\text{s}$), and closely synchronized with the respiratory rate. The higher mandibular angular velocity reflects greater upper airway muscle engagement and often corresponds with escalating airflow as the ventilatory drive intensifies from normal or reduced levels to more active conditions near the arousal threshold. This phenomenon, known as drive-dependent OSA, demonstrates compensatory increases in airflow as a function of upper airway resistance. These episodes, typically lasting at least 10 s and rarely exceeding 1 min, are often accompanied by airflow reductions classified as hypopneas or apneas.

Several examples show differentiation between obstructive (Figure S14) and central events (Figures S15 and S16) based on the presence or absence of respiratory drive captured by MJM_Gyr, respectively. Obstructive events display a characteristic pattern of increasing MJM_Gyr amplitude (Figure S12), reflecting sustained respiratory effort against a blocked airway. In contrast, central events are marked by minimal or absent respiratory drive, often with angular velocities falling below the 25th percentile ($0.63^\circ/\text{s}$), potentially occurring in cyclic patterns with intermittent periods of respiratory command. For example, Figure S15 highlights successive central hypopneas, each lasting at least 20 s and accompanied with nearly zero mandibular rotation speed. Mouth opening, while also associated with RE, may occur in both obstructive and central hypopneas, as shown in Figure S16.

Some hypopneas may display both central and obstructive origins and are thus classified as mixed events. Mixed apneas or hypopneas typically begin with reduced MJM velocity, followed by a resurgence of centrally driven velocity before arousal, possibly indicating a phase of elevated loop gain, as shown in Figure S17.

Beyond apneas and hypopneas, MJM signals are sensitive enough to capture mild elevations in RE during RERAs and flow limitations. Figures S12A and S12B highlight a prolonged period of increased respiratory effort terminating in an arousal (RERA). Similarly, Figure S18 illustrates a crescendo of respiratory effort, also ending with arousal, emphasizing how MJM signals track these effort escalations. Figure S19 depicts how MJM signals capture subtle yet persistent RE increases during a long episode of flow limitation. Figure S20 shows how a brief awakening may destabilize ventilatory control, potentially inducing a

sequence of central events due to CO₂ washout and elevated loop gain. In [Figure S21](#), flow-limited breaths occur at low levels of drive, as demonstrated by reduced mandibular jaw rotations and less rounded pressure-flow curves.

Several examples underscore the utility of MJM analysis in monitoring respiratory instability during sleep, especially after arousals or following transitions between sleep stages. For example, [Figure S22](#) outlines a sequence involving RERA, central apnea, and obstructive episodes, illustrating how one event may precipitate another under conditions of heightened loop gain. Likewise, [Figure S23](#) captures central apneas after an arousal in a cyclic pattern, suggesting that CO₂ washout can quickly swing the respiratory control system into periodic breathing. The cyclical breathing patterns are also captured in [Figures S24A and 24B](#) through both MJM_{Acc} and MJM_{Gyr} signals.

Furthermore, during rhythmic masticatory muscle activities (RMMA) or sleep bruxism, the mandible moves at a higher frequency (~1 Hz) and greater amplitude compared to baseline respiratory movements ([Figures S25A & S25B](#)). These episodes, typically occurring after arousals, are easily identifiable due to their distinct frequency and amplitude characteristics. Such events often lead to forced ventilation and may contribute to CO₂ washout, resulting in subsequent reductions in respiratory and trigeminal drive.

4.4. Identifying cortical and subcortical arousals

Throughout the visual companion guide, MJM signals demonstrate effectiveness in detecting arousal events ([Figures S3, S8-10, S15, S18-21, S23, S25-30](#)). Arousals are typically marked by prominent MJM peaks, characterized by angular, non-gravitational acceleration at the chin exceeding the 75th percentile threshold (0.14 m/s²), clearly distinguishing them from baseline sleep periods. Mandibular displacement becomes more pronounced when mouth closure is accompanied by head movements, and the MJM signal is often amplified in the presence of scorable cortical arousals, likely due to cortico-bulbar reflex activation.

Importantly, MJM signals also allow for sensitive detection of subtle arousals and differentiation between cortical and subcortical arousals. For instance, [Figure S20](#) shows how a cortical arousal triggers an increase in airflow along with a noticeable shift in mandibular position. In contrast, subcortical arousals manifest as distinct jaw movements without corresponding prominent changes in scalp EEG.

[Figures S28 and S29](#) depict scenarios where jaw movements and sympathetic activations (evidenced by limb movements) mark arousals that remain below the conventional EEG cortical scoring threshold. These subcortical arousals may either terminate disordered breathing episodes or occur spontaneously, reflecting intrinsic brainstem mechanisms. In [Figure S28](#), a second subcortical arousal even precedes a brief central apnea, linking the transition from deeper subcortical drive to temporary cessation of respiratory effort.

Finally, [Figure S30](#) offers a visual comparison between a high-amplitude jaw displacement (associated with a cortical arousal) and smaller-amplitude jaw movements (subcortical arousals).

5. Clinical applications of MJM analysis in pediatric OSA

In 2015, a clinical study was conducted on 33 children (median age: 5.0 years; range: 2.0–15.0) with snoring and OSA symptoms [20]. The aim was to determine whether MJM signals alone, recorded via magnetometry and integrated into PSG as the jaw activity channel, could assess RE in pediatric OSA. While prior investigations [37,38] explored MJM's potential in sleep/awake and apnea/hypopnea event detection, this study was the first to clinically apply MJM analysis in children.

Building on empirical observations, the authors identified specific MJM patterns indicative of RE during sleep, including large, singular, and prolonged mouth openings. These MJM patterns correlated strongly with airflow and other standard PSG signals, highlighting their

relevance in evaluating OSA severity. The study emphasized the interpretability of MJM data, making it well-suited for both manual scoring and the development of transparent, physiology-based algorithms—offering an alternative to 'black box' approaches, prioritizing interpretability and physiological relevance.

In 2017, a second study investigated MJM's utility in tracking residual RE after adenotonsillectomy (AT) in children with OSA [39]. Using the same manual scoring process described by Martinot et al. (2015) [20], mandibular movement signal was compared with conventional PSG signals in 25 children (mean age: 5.9 years; range: 1.7–13.0). The study highlighted two pivotal insights. First, it confirmed MJM's capability to quantify clinical response to treatment, underlining its potential in managing pediatric OSA over time—from OSA screening and diagnosis to post-treatment follow-up. Second, the MJM signal provides information beyond PSG conventional indices, more accurately reflecting overall respiratory burden during sleep, especially for events not included in AHI like RERAs. Notably, these pediatric findings aligned with those observed in adult OSA populations.

Following its validation in adult OSA [54], the inertial sensor-based technology was also evaluated in a pediatric population in 2021 [21]. In this study, 140 children (median age: 6.9 years; range: 3.0–17.0) underwent in-lab PSG with simultaneous MJM recording via a single-point tri-axial accelerometer/gyroscope sensor. Automated scoring algorithms generated an MJM-derived RDI that was compared to PSG-derived RDI. MJM-RDI showed strong correlation with PSG-RDI (Pearson's $r = 0.84$; ICC = 0.79), indicating that MJM-based indices track respiratory events consistently across a wide range of OSA severities. Bland-Altman analysis confirmed good agreement (median bias: +1.57 events/hour; no proportional bias). When compared with standard PSG-AHI thresholds of 1, 5, and 10 events/hour, MJM-RDI demonstrated good diagnostic performance, with area-under-the-curve (AUC) values of 0.75, 0.90, and 0.98, respectively. These findings validated the accuracy and clinical relevance of MJM analysis for pediatric OSA detection and supported its introduction into home-based testing. It not only compares favorably to other HSATs (pooled AUC of 0.89), as reported by Gao et al. (2021) [18], but also matches the most recent meta-analytic estimates for portable devices (pooled AUC of 0.93) by Touhuti et al. (2023) [19]. However, due to the limited number of available clinical studies and the significant heterogeneity in device architectures, scoring algorithms, and reference thresholds, direct comparative evaluation remains premature.

More recently, Cassibba et al. (2022) evaluated the reliability of automated MJM analysis in children undergoing noninvasive ventilation (NIV) or continuous positive airway pressure (CPAP) therapy [22]. The study included 15 children (median age: 12.0 years; range: 5.0–18.0) with severe residual OSA, eight of whom had congenital upper airway syndromes. Simultaneous in-lab PSG and MJM monitoring demonstrated strong agreement for residual AHI measurement, with a median bias of −0.25 events/hour (95% CI: −3.40 to +2.04), indicating near-parallel results. After three months of treatment, follow-up MJM recordings at home revealed significant reductions in residual AHI. These findings underscored MJM's potential for both clinical and home settings, offering a versatile tool for evaluating therapeutic effectiveness in real-world environments.

In summary, MJM analysis has emerged as a practical, accurate, and physiologically grounded tool for pediatric OSA assessment. Its strong agreement with PSG-derived indices supports its clinical integration for screening, diagnosis, treatment evaluation, and follow-up in children with sleep-disordered breathing.

6. Discussion

This review has outlined the technical, practical, and clinical relevance of the MJM signal, along with a comprehensive guide for visual operator-based interpretation in the context of pediatric OSA diagnosis.

Interpreting MJM signals allows for the identification of key

physiological mechanisms underlying respiratory disturbances in OSA. For example, the role of mandibular-related muscles in stabilizing the pharynx under inspiratory load—much like how the rib cage supports the lungs during diaphragmatic contraction—highlights the pivotal function of mandibular dynamics in maintaining upper airway patency during sleep. Simultaneous monitoring of jaw movements and muscle activation across various sleep stages enables the assessment of both the respiratory drive responsible for keeping the upper airway open and the neuromuscular compensation mechanisms that modulate this process.

Mandibular dynamics and trigeminal motor engagement are best quantified via parameters such as displacement velocity and acceleration, rather than mere positional changes. Variations in respiratory drive—reflected by shifts in airflow amplitude and nasal pressure waveforms—are closely associated with changes in trigeminal motor control during SDB. This relationship is evident in mandibular behavior during episodes of airflow disturbance and subsequent recovery phases, which are influenced by regulatory inputs from higher brain centers that vary by sleep stage. Consequently, MJM signals serve as critical indicators of airway stability and the presence of sleep-related breathing disorders.

Beyond their utility for manual scoring, the identification of specific MJM signal patterns and standardized interpretation rules forms the foundation for developing and validating artificial intelligence-based algorithms, including explainable approaches. Clinical studies have already demonstrated the feasibility, reliability, and accuracy of automated MJM analysis for pediatric OSA detection and monitoring.

The implementation of a portable, single-point MJM sensor presents several practical advantages over conventional sleep studies, particularly in pediatric populations. These include improved access to home sleep apnea testing (HSAT), reduced costs, increased comfort, lower technical failure rates, and shorter time to diagnosis and intervention. Notably, the MJM signal's sensitivity to subtle, RE-related disturbances—often missed by conventional signals—has important implications for both therapeutic evaluation and a more accurate assessment of OSA burden.

While the clinical utility and physiological relevance of MJM analysis in pediatric OSA have been increasingly recognized, the evidence base is still modest and originates primarily from early-adopter research centers. Future research should focus on large-scale, multi-center validation studies to confirm the robustness and generalizability of MJM-based monitoring across broader pediatric populations.

7. Conclusion

In conclusion, MJM recordings and analysis provide a technically simple, physiologically robust, and clinically meaningful approach for detecting and monitoring pediatric OSA. As sleep medicine increasingly embraces patient-centered, cost-effective solutions, MJM technology stands out as a highly promising tool in the management of pediatric sleep-disordered breathing.

Practical points

- 1 Mandibular jaw movement (MJM) analysis is a reliable, practical alternative to polysomnography for diagnosing pediatric OSA, capturing respiratory drive and upper airway muscle activity.
- 2 A structured atlas supports MJM interpretation, illustrating typical MJM patterns in respiratory events, sleep bruxism, arousals, and sleep stages.

Research agenda

- 1 Further pediatric-specific validation should investigate MJM analysis in diverse cohorts across ages, phenotypes, and comorbidities.
- 2 Prospective studies should assess how MJM-based diagnosis and monitoring impact long-term outcomes in pediatric OSA management.

Author contributions

JBM contributed to the conception and design, data acquisition, analysis and interpretation, he drafted and critically revised the manuscript.

NNLD contributed to the conception and design, data acquisition, analysis and interpretation, he drafted and critically revised the manuscript.

JC contributed to the conception, data interpretation, she drafted and critically revised the manuscript.

DC contributed to data acquisition and interpretation, and critically revised the manuscript.

JLP contributed to the conception, data interpretation, he drafted and critically revised the manuscript.

DG contributed to the conception, data interpretation, he drafted and critically revised the manuscript.

All authors gave their final approval and agreed to be accountable for all aspects of the work.

Funding Acknowledgments

This literature review did not receive any specific grant from funding agencies in the public, institutional, commercial, or not-for-profit sectors.

Conflicts of interest

JBM is a scientific advisor to Sunrise and has been an investigator in pharmaceutical trials for Jazz Pharmaceuticals and Takeda.

NNLD is an employee of Sunrise.

JC does not have any conflicts of interest to disclose.

DG does not have any conflicts of interest to disclose.

DC does not have any conflicts of interest to disclose.

JLP is a scientific advisor to Sunrise, has received grants and/or personal fees from ResMed, Philips, Fisher & Paykel, Sefam, AstraZeneca, AGIR à dom, Elevie, VitalAire, Boehringer Ingelheim, Jazz Pharmaceuticals and Itamar Medical Ltd, and has received research support for clinical studies from Mutualia and Air Liquide Foundation.

JLP is supported by the French National Research Agency (ANR) in the framework of the "FRANCE 2030" program, the "e-health and integrated care" chair of Grenoble Alpes University Foundation and "Sleep Health-AI chair" in "MIAI Cluster" of artificial intelligence (ANR-23-IAEL-0006).

Acknowledgments

The authors wish to thank Mr. Stéphane Denison and Ms. Hari Gomez for their assistance with the illustrations.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.smrv.2025.102159>.

References

- [1] Marcus C, Brooks LJ, Draper KA, Gozal D, Halbower AC, Jones J, Schechter MS, Sheldon SH, Spruyt K, Lehmann C, Shiffman RN. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2012;130:576–84. <https://doi.org/10.1542/peds.2012-1671>.
- [2] Riha RL, Celmina M, Cooper B, Hamutcu-Ersu R, Kaditis A, Morley A, et al. ERS technical standards for using type III devices (limited channel studies) in the diagnosis of sleep disordered breathing in adults and children. *Eur Respir J* 2023; 61(1):2200422. <https://doi.org/10.1183/13993003.00422-2022>.
- [3] Jennum P, Ibsen R, Kjellberg J. Morbidity and mortality in children with obstructive sleep apnoea: a controlled national study. *Thorax* 2013;68(10):949–54.
- [4] DelRosso LM, Mogavero MP, Ferri R. Effect of sleep disorders on blood pressure and hypertension in children. *Curr Hypertens Rep* 2020;22(11):88. <https://doi.org/10.1007/s11906-020-01100-x>.

- [5] Marcus CL, Moore RH, Rosen CL, Giordani B, Garetz SL, Taylor HG, et al. A randomized trial of adenotonsillectomy for childhood sleep apnea. *N Engl J Med* 2013;368:2366–76. <https://doi.org/10.1056/NEJMoa1215881>.
- [6] Taylor HG, Bowen SR, Beebe DW, Hodges E, Amin R, Arens R, et al. Cognitive effects of adenotonsillectomy for obstructive sleep apnea. *Pediatrics* 2016;138(2):e20154458. <https://doi.org/10.1542/peds.2015-4458>.
- [7] Brockmann PE, Gozal D. Neurocognitive consequences in children with sleep disordered breathing: who is at risk? *Children* 2022;9(9):1278.
- [8] Min JW, Zhang XM, Griffis HM, Cielo CM, Tapia IE, Williamson AA. Sociodemographic disparities and healthcare utilization in pediatric obstructive sleep apnea management. *Sleep Med* 2023;109:211–8. <https://doi.org/10.1016/j.sleep.2023.07.009>.
- [9] Ross KR, Redline S. Is it time to head home for the night? Home sleep testing in young children. *Ann Am Thorac Soc* 2020;17(10):1207–9. <https://doi.org/10.1513/AnnalsATS.202008-970ED>.
- [10] Tan HL, Kaditis AG. PRO: home sleep studies in children - are we there yet? *SLEEP* 2023;zsad040.
- [11] Gutiérrez-Tobal GC, Álvarez D, Kheirandish-Gozal L, del Campo F, Gozal D, Hornero R. Reliability of machine learning to diagnose pediatric obstructive sleep apnea: systematic review and meta-analysis. *Pediatr Pulmonol* 2022;57(8):1931–43.
- [12] Kirk V, Baughn J, D'Andrea L, Friedman N, Galion A, Garetz S, et al. American academy of sleep medicine position paper for the use of a home sleep apnea test for the diagnosis of OSA in children. *J Clin Sleep Med* 2017;13(10):1199–203. <https://doi.org/10.5664/jcsm.6772>.
- [13] Selby A, Buchan E, Davies M. Role of overnight oximetry in assessing the severity of obstructive sleep apnoea in typically developing children: a multicentre study. *Arch Dis Child* 2024;109(4):308–13. <https://doi.org/10.1136/archdischild-2023-326191>.
- [14] Jurando MJ, Sampol G, Quintana M, Romero O, Cambrodí R, Ferré A, Sampol J. Nasal cannula use during polysomnography in children aged under three with suspected sleep apnea. *Sleep Med* 2022;99:41–8. <https://doi.org/10.1016/j.sleep.2022.07.009>.
- [15] Tanphaichitr A, Thianboonsong A, Banhiran W, Vathanophas V, Ungkanont K. Watch peripheral arterial tonometry in the diagnosis of pediatric obstructive sleep apnea. *Otolaryngol Head Neck Surg* 2018;159(1):166–72. <https://doi.org/10.1177/0194599818768215>.
- [16] Choi JH, Lee B, Lee JY, Kim HJ. Validating the watch-PAT for diagnosing obstructive sleep apnea in adolescents. *J Clin Sleep Med* 2018;14(10):1741–7. <https://doi.org/10.5664/jcsm.7386>.
- [17] Hilmlsson H, Berman S, Magnusdottir S. Sleep apnea diagnosis in children using software-generated apnea-hypopnea index (AHI) derived from data recorded with a single photoplethysmogram sensor (PPG): results from the Childhood Adenotonsillectomy Study (CHAT) based on cardiopulmonary coupling analysis. *Sleep Breath* 2020;24(4):1739–49. <https://doi.org/10.1007/s11325-020-02049-6>.
- [18] Gao X, Li Y, Xu W, Han D. Diagnostic accuracy of level IV portable sleep monitors versus polysomnography for pediatric obstructive sleep apnea: a systematic review and meta-analysis. *Sleep Med* 2021;87:127–37. <https://doi.org/10.1016/j.sleep.2021.08.029>.
- [19] Tuohuti A, Lin Z, Cai J, Chen X. Can portable sleep monitors replace polysomnography for diagnosis of pediatric OSA: a systematic review and meta-analysis. *Eur Arch Otorhinolaryngol* 2023;280(10):4351–9. <https://doi.org/10.1007/s00405-023-08095-6>.
- [20] Martinot JB, Senny F, Denison S, Cuthbert V, Gueulette E, Guénard H, Pépin JL. Mandibular movements identify respiratory effort in pediatric obstructive sleep apnea. *J Clin Sleep Med* 2015;11(5):567–74. <https://doi.org/10.5664/jcsm.4706>.
- [21] Martinot JB, Cuthbert V, Le-Dong NN, Coumans N, De Marneffe D, Letesson C, et al. Clinical validation of a mandibular movement signal based system for the diagnosis of pediatric sleep apnea. *Pediatr Pulmonol* 2022;57(8):1904–13. <https://doi.org/10.1002/ppul.25320>.
- [22] Cassibba J, Aubertin G, Martinot JB, Le-Dong NN, Hullo E, Beydon N, et al. Analysis of mandibular jaw movements to assess ventilatory support management of children with obstructive sleep apnea syndrome treated with positive airway pressure therapies. *Pediatr Pulmonol* 2024;59(7):1905–11. <https://doi.org/10.1002/ppul.27005>.
- [23] Maury G, Frédéric Senny F, Cambron F, Albert A, Seidel L, Poirrier R. Mandible behaviour interpretation during wakefulness, sleep and sleep-disordered breathing. *J Sleep Res* 2014;23(6):709–16. <https://doi.org/10.1111/jsr.12180>.
- [24] Malhotra A, Martinot JB, Pépin JL. Insights on mandibular jaw movements during polysomnography in obstructive sleep apnea. *J Clin Sleep Med* 2023. <https://doi.org/10.5664/jcsm.10830>.
- [25] McFarland DH, Lund JP. Modification of mastication and respiration during swallowing in the adult human. *J Neurophysiol* 1995;74(4):1509–17. <https://doi.org/10.1152/jn.1995.74.4.1509>.
- [26] Brown T. Mandibular movements. *Monogr Oral Sci* 1975;4:126–50. <https://doi.org/10.1159/000397870>.
- [27] Miyamoto K, Ozbek M, Sjöholm TT, Love LL, Fleetham JA, Ryan CF. Mandibular posture during sleep in patients with obstructive sleep apnoea. *Arch Oral Biol* 1999;44(8):657–64. [https://doi.org/10.1016/s0003-9969\(99\)00057-6](https://doi.org/10.1016/s0003-9969(99)00057-6).
- [28] Salazar DHR, Honnlee S, Liu ZJ. Tongue, palatal, hyoid and pharyngeal muscle activity during chewing, swallowing, and respiration. *Arch Oral Biol* 2024;157:105845. <https://doi.org/10.1016/j.archoralbio.2023.105845>.
- [29] Hao N, Sasa A, Kulvanich S, Nakajima Y, Nagoya K, Magara J, et al. Coordination of respiration, swallowing, and chewing in healthy young adults. *Front Physiol* 2021;12:696071. <https://doi.org/10.3389/fphys.2021.696071>. eCollection 2021.
- [30] Brouillette RT, Thach BT. A neuromuscular mechanism maintaining extrathoracic airway patency. *J Appl Physiol Respir Environ Exerc Physiol* 1979;46(4):772–9. <https://doi.org/10.1152/jappl.1979.46.4.772>.
- [31] Roberts JL, Reed WR, Thach BT. Pharyngeal airway-stabilizing function of sternohyoid and sternothyroid muscles in the rabbit. *J Appl Physiol Respir Environ Exerc Physiol* 1984;57(6):1790–5. <https://doi.org/10.1152/jappl.1984.57.6.1790>.
- [32] Van de Graaff WB, Gottfried SB, Mitra J, van Lunteren E, Cherniack NS, Strohl KP. Respiratory function of hyoid muscles and hyoid arch. *J Appl Physiol Respir Environ Exerc Physiol* 1984;57(1):197–204. <https://doi.org/10.1152/jappl.1984.57.1.197>.
- [33] van Lunteren E, Haxhiu MA, Cherniack NS. Relation between upper airway volume and hyoid muscle length. *J Appl Physiol* 1987;63(4):1443–9. <https://doi.org/10.1152/jappl.1987.63.4.1443>.
- [34] Suratt PM, McTier RF, Wilhoit SC. Collapsibility of the nasopharyngeal airway in obstructive sleep apnea. *Am Rev Respir Dis* 1985;132(5):967–71. <https://doi.org/10.1164/arrd.1985.132.5.967>.
- [35] Kubin L. Neural control of the upper airway: respiratory and state-dependent mechanisms. *Compr Physiol* 2016;6:1801–50.
- [36] Travers JB, Norgren R. Afferent projections to the oral motor nuclei in the rat. *J Comp Neurol* 1983;220:280–98.
- [37] Senny F, Destiné J, Poirrier R. Midsagittal jaw movement analysis for the scoring of sleep apneas and hypopneas. *IEEE Trans Biomed Eng* 2008;55(1):87–95. <https://doi.org/10.1109/TBME.2007.899351>.
- [38] Senny F, Maury G, Cambron L, Leroux A, Destiné J, Poirrier R. The sleep/wake state scoring from mandible movement signal. *Sleep Breath* 2012;16(2):535–42.
- [39] Martinot JB, Le-Dong NN, Denison S, Guénard H, Borel JC, Silkoff PE, et al. Persistent respiratory effort after adenotonsillectomy in children with sleep-disordered breathing. *Laryngoscope* 2018;128(5):1230–7. <https://doi.org/10.1002/lary.26830>.
- [40] Martinot JB, Le-Dong NN, Cuthbert V, Denison S, Silkoff PE, Guénard H, Gozal D, Pépin JL, Borel JC. Mandibular movements as accurate reporters of respiratory effort during sleep: validation against diaphragmatic electromyography. *Front Neurol* 2017;8:353. <https://doi.org/10.3389/fneur.2017.00353>.
- [41] Martinot JB, Le-Dong NN, Crespeigne E, Silkoff PE, Cuthbert V, Denison S, Borel JC, Pépin JL. Mandibular movement analysis to assess efficacy of oral appliance therapy in OSA. *Chest* 2018;154(6):1340–7. <https://doi.org/10.1016/j.chest.2018.08.1027>.
- [42] Martinot JB, Le-Dong NN, Cuthbert V, Denison S, Silkoff P, Borel JC, Pépin JL. The key role of the mandible in modulating airflow amplitude during sleep. *Respir Physiol Neurobiol* 2020;279:103447. <https://doi.org/10.1016/j.resp.2020.103447>.
- [43] Pépin JL, Le-Dong NN, Cuthbert V, Coumans N, Tamisier R, Malhotra A, Martinot JB. Mandibular movements are a reliable noninvasive alternative to esophageal pressure for measuring respiratory effort in patients with sleep apnea syndrome. *Nat Sci Sleep* 2022;14:635–44. <https://doi.org/10.2147/NSS.S346229>.
- [44] Smith MT, McCrae CS, Cheung J, Martin JL, Harrod CG, Head JL, Carden KA. Use of actigraphy for the evaluation of sleep disorders and circadian rhythm sleep-wake disorders: an American academy of sleep medicine systematic review, meta-analysis, and GRADE assessment. *J Clin Sleep Med* 2018;14(7):1209–30. <https://doi.org/10.5664/jcsm.7228>.
- [45] Yang J, Rosenmöller B, van Riet T, Tan ML, Jamaludin FS, Ho JP, de Lange J. Smart mandibular advancement devices for obstructive sleep apnea: a systematic literature review. *Sleep Breath* 2024;28(5):1879–87. <https://doi.org/10.1007/s11325-024-03068-3>.
- [46] Ingervall B. Variation of the range of movement of the mandible in relation of facial morphology in children. *Scand J Dent Res* 1970;78(6):533–43.
- [47] Pépin JL, Cistulli PA, Crespeigne E, Tamisier R, Bailly S, et al. Mandibular jaw movement automated analysis for oral appliance monitoring in obstructive sleep apnea: a prospective cohort study. *Ann Am Thorac Soc* 2024 May;21(5):814–22. <https://doi.org/10.1513/AnnalsATS.202312-1077OC>.
- [48] Pépin JL, Martinot JB, Le-Dong NN, Leroy S, Clause D, Malhotra A, Lavigne G, Cistulli PA. Exploring the dose-response relationship between mandibular protrusion and respiratory effort burden in oral appliance therapy for OSA. *Ann Am Thorac Soc* 2025. <https://doi.org/10.1513/AnnalsATS.202408-889OC>. Online ahead of print.
- [49] Martinot JB, Le-Dong NN, Malhotra A, Pépin JL. Respiratory effort during sleep and prevalent hypertension in obstructive sleep apnoea. *Eur Respir J* 2023;61(3):2201486. <https://doi.org/10.1183/13993003.01486-2022>.
- [50] Martha E Rich. Masseter muscle bite force in first bicuspid and collapsed occlusion cases. *Int J Orthod Milwaukee* 2012;23(2):29–33. PMID: 22873021.
- [51] Baptista H, Cardoso IL. Steinert syndrome and repercussions in dental medicine. *Arch Oral Biol* 2017;75:37–47. <https://doi.org/10.1016/j.archoralbio.2016.12.008>.
- [52] Martinot JB, Pépin JL. Mandibular jaw movements as a non-invasive measure of respiratory effort during sleep: application in clinical practice. *Front Sleep* 2023;2:1145620. <https://doi.org/10.3389/frsle.2023.1145620>.
- [53] Pépin JL, Tamisier R, Borel JC, Baguet Lévy P. A critical review of peripheral arterial tone and pulse transit time as indirect diagnostic methods for detecting sleep disordered breathing and characterizing sleep structure. *Curr Opin Pulm Med* 2009;15(6):550–8. <https://doi.org/10.1097/MCP.0b013e3283318585>.
- [54] Pépin JL, Letesson C, Le-Dong NN, Dedave A, Denison S, Cuthbert V, Martinot JB, Gozal D. Assessment of mandibular movement monitoring with machine learning analysis for the diagnosis of obstructive sleep apnea. *JAMA Netw Open* 2020;3(1):e1919657. <https://doi.org/10.1001/jamanetworkopen.2019.19657>.