Use of pulse oximetry as an investigative test for paediatric respiratory sleep disorders

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ABSTRACT
The article covers the following elements: practical and technological considerations for optimising data collection and output; reference ranges for oximetry parameters across the ages; things to consider when interpreting a pulse oximetry study (eg, sleep/wake times); the ability of pulse oximetry to predict obstructive sleep apnoea; using oximetry as a screening tool for sleep disordered breathing in children with Down syndrome; things to consider when setting up a home oximetry service; and a case of an infant being weaned from oxygen using pulse oximetry studies.

INTRODUCTION
Multiple diagnostic techniques are available for the assessment of suspected sleep disorders in children. Polysomnography (PSG) is commonly considered the gold standard diagnostic investigation in sleep medicine but is arguably limited by its complexity, cost and availability within the UK.1 Alternative diagnostic investigations for sleep disorders have therefore been considered.2

Pulse oximetry is a simple, cheap and readily available tool and provides a rapid, non-invasive estimate of the functional oxygen saturation (SpO₂) of haemoglobin (Hb) in arterial blood. Continuous nocturnal pulse oximetry (NPO) is increasingly used as a screening tool for obstructive sleep apnoea (OSA) in children at high risk, for example, those with Down syndrome (DS), to wean babies with chronic lung disease of prematurity (CLDP) out of oxygen, and alongside transcutaneous carbon dioxide (CO₂) monitoring to guide ventilation needs for children on home mechanical ventilation. Despite its lack of sensitivity, it has a high specificity for diagnosing OSA in typically developing (TD) children.

The use of NPO demands a basic understanding of sleep architecture, data analysis and an awareness of the test’s indications and limitations in order to appropriately interpret the findings.

PHYSIOLOGICAL BACKGROUND
Sleep is vital for health and well-being. It may be defined as a reversible state of reduced awareness and selective responsiveness to the environment, characterised by motor inhibition. Sleep disorders and associated fragmented sleep quality are associated with multiple adverse outcomes including poor growth and cardiovascular abnormalities.1 Furthermore, behavioural, cognitive and emotional difficulties are reported in TD children with OSA and those with complex medical conditions in association with multifactorial sleep disordered breathing (SDB).

For infants with CLDP, a structured approach to weaning home oxygen therapy (HOT) is associated with more rapid oxygen weaning in the community3 and minimises the potential for hypoxia and unwanted consequences including pulmonary hypertension, impaired growth and neurodevelopmental outcomes.

SLEEP ARCHITECTURE
Basic sleep neurophysiology recognises two discrete sleep states: rapid eye movement (REM), also termed active sleep, and non-rapid eye movement (non-REM) or quiet sleep, of which there are three types: N1, N2 and N3 (figure 1).

REM and non-REM sleep are defined by neurophysiology/PSG variables. When these are not available, replacement indicators such as heart-rate variability as visualised on pulse oximetry traces are used as surrogate markers to identify episodes of REM sleep (figure 2). In the absence of neurophysiology variables, this is described as active sleep.
Interpretations

Active/REM sleep occurs disproportionately throughout the night, and this is relevant for the timing and duration of sleep studies and the ability to capture events (figure 1). While quiet/non-REM sleep is associated with a regular breathing pattern and low and unchanging heart rate, active/REM sleep is characterised by irregular breathing, pauses in respiration and a higher and more variable heart rate. During active/REM sleep, there is skeletal muscle atonia, reduced pharyngeal dilator muscle activity and thus increased upper-airway resistance. SDB occurs in active sleep for almost all conditions in children. Infants demonstrate particular vulnerability due to the presence of central apnoea, lower functional residual capacity and increased chest wall compliance, which all predispose to desaturation events and respiratory instability. This wobble effect, or chest wall paradox, may be considered a normal phenomenon during active sleep until 3 years of age.

Sleep neurophysiology changes during infancy, and discrete sleep stages are generally present by 6 months of age. REM and non-REM sleep cycles are equally dispersed throughout the night in 30 min epochs. By adolescence, REM sleep reduces to 15%–20% sleep

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Figure 1  Hypnogram displaying typical sleep architecture and sleep stage distribution outside of infancy. In this figure, non-REM predominantly features in the first half of the night and REM sleep during the latter half of sleep. REM, rapid eye movement.

Figure 2  Pulse oximetry study (Visi-Download software). The trace demonstrates infant sleep-wave cycling with periods of active and quiet sleep characterised by heart-rate variability. Episodes of desaturation occur during presumed active/REM sleep. Reproduced from Everitt et al.26 SpO₂, oxygen saturation.
and occurs in the latter part of the night. The tendency for abnormalities to occur in REM sleep means that age-specific variables require consideration.

SDB may be broadly categorised into obstructive or central events. Obstructive apnoea and hypopnoea present as a reduction in airflow in association with ongoing respiratory effort. They occur secondary to complete or incomplete airway obstruction, which may be associated with oxygen desaturation and followed by arousal. OSA is the most common form of SDB in TD children.

Central sleep apnoea (CSA) and hypopnoea are characterised by the absence or reduction in airflow with concomitant absence or reduction of respiratory effort. Central events are the consequence of disruption in respiratory balance and may arise from dysfunctional central drive, impaired respiratory effort (muscle disorders) and disorders of increased respiratory load (obstructive airway disease, lung disease and restriction from obesity). SDB in children with complex medical needs is commonly multifactorial in nature, and OSA may coexist with CSA and hypopnoea.

### Types of sleep study

There are several different types of sleep study of varying complexity, and their associated features and limitations are outlined in **table 1**. PSG is a complex, multichannel investigation and commonly considered the gold standard diagnostic sleep investigation. Due to the complexity and cost of PSG, simpler diagnostic tools for the investigation of paediatric sleep disorders have been explored.

### PULSE OXIMETRY

Pulse oximetry is a single parameter tool that provides a rapid, non-invasive estimate of the functional SpO2 of Hb in arterial blood. NPO provides quantitative data on the frequency and severity of desaturation events.

### Technological background

The first portable oximetry device was developed in the early 1940s with recognition that SpO2 could be computed from differences in the transmission of red

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**Table 1  Different types of sleep studies and their features, strengths and limitations**

<table>
<thead>
<tr>
<th>Sleep study</th>
<th>Location</th>
<th>Features</th>
<th>Strengths</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polysomnography</td>
<td>Hospital</td>
<td>Sleep staging, Sleep duration, Arousal frequency, SpO2, OAHI and CAHI, ±CO2, measures</td>
<td>Widely accepted gold standard.</td>
<td>Expensive, labour intensive, time-consuming analysis Requires inpatient stay.</td>
</tr>
<tr>
<td>Cardiorespiratory Sleep Study/polygraphy</td>
<td>Home or hospital</td>
<td>Sleep duration, Arousal frequency, SpO2, OAHI and CAHI, ±CO2, measures (no EEG, EOG or EMG)</td>
<td>Discriminatory between obstructive and central sleep events.</td>
<td>Specific sleep stages are inferred. Transcutaneous CO2 probes are expensive and fragile.</td>
</tr>
<tr>
<td>Oxycapnography</td>
<td>Home or hospital</td>
<td>Transcutaneous CO2 alongside assessment of pulse and estimated SpO2, HR and ±CO2</td>
<td>Provides information regarding gas exchange and assessment of ventilation</td>
<td>Does not discriminate between obstructive and central events. Transcutaneous CO2 probes are expensive and fragile.</td>
</tr>
<tr>
<td>Pulse oximetry</td>
<td>Home or hospital</td>
<td>Assessment of pulse and estimated SpO2 and HR</td>
<td>Simple, inexpensive and accessible</td>
<td>Does not discriminate between obstructive and central events</td>
</tr>
</tbody>
</table>

AHI, apnoea hypopnoea index; CAHI, central apnoea hypopnoea index; CO2, carbon dioxide; EEG, electroencephalogram; EMG, electromyography; EOG, electrooculography; HR, heart rate; OAHI, obstructive apnoea hypopnoea index; SpO2, peripheral oxygen saturation.
Interpretations

Table 2  Reference ranges for oximetry parameters across the age spectrum

<table>
<thead>
<tr>
<th>Author</th>
<th>Gestation (weeks) (n)</th>
<th>Oximetry timing (postconceptual age, weeks)</th>
<th>Mean saturations (%)</th>
<th>Min saturation (%)</th>
<th>ODI3</th>
<th>ODI4</th>
<th>% time &lt;90%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Williams et al</td>
<td>35 (34–36) (n=43)</td>
<td>35 (34–36)</td>
<td>97.8 (97.1–98.3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Williams et al</td>
<td>35 (34–36) (n=43)</td>
<td>40</td>
<td>98.8 (98.4–99.4)</td>
<td>32.8 (25.9–41.4)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Williams et al</td>
<td>40 (39–42) (n=42)</td>
<td>40 (39–42)</td>
<td>97.9 (96.7–98.9)</td>
<td>29.3 (23.5–36.6)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evans et al</td>
<td>39 (37–42) (n=45)</td>
<td>44 (43–44)</td>
<td>97.1 (13.7–18.6)</td>
<td>80.4 (78.8–82.0)</td>
<td>25.4 (22.0–28.8)</td>
<td>16.2 (13.7–18.6)</td>
<td>0.39 (0.26–0.55)</td>
</tr>
<tr>
<td>Evans et al</td>
<td>39 (37–42) (n=38)</td>
<td>56 (54–57)</td>
<td>97.7 (97.2–98.1)</td>
<td>84.7 (83.3–86.1)</td>
<td>13.9 (11.4–16.5)</td>
<td>8.12 (6.46–9.77)</td>
<td>0.11 (0.06–0.20)</td>
</tr>
<tr>
<td>Vézina et al</td>
<td>(n=562)</td>
<td>1.1 year</td>
<td>97.1 (95.5–98.0)</td>
<td>6.7 (1.4–15.8)</td>
<td>0.1 (0.0–0.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ong et al</td>
<td>(n=66)</td>
<td>6 months–12 years (median 7 years 10 months)</td>
<td>97.57 (97.38–97.76)</td>
<td>91.09 (90.3–91.9)</td>
<td>2.58 (95th centile 6.43)</td>
<td>1.14 (0.93–1.34)</td>
<td>0.00 (0.05)</td>
</tr>
</tbody>
</table>

ODI3, Oxygen Desaturation Index at 3%; ODI4, Oxygen Desaturation Index at 4%.

Figure 4  Influence of averaging time on the number of desaturations for an 80% SpO2 alarm threshold. An averaging time of 3 s (green) leads to six desaturations; an averaging time of 10 s (red) leads to three desaturations; and an averaging time of 16 s (blue) results in one desaturation. Figure reproduced from Vagedes et al. SpO2, oxygen saturation.
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diastole and the differential absorption of light by oxyhaemoglobin (HbO₂) and deoxygenated Hb. HbO₂ more readily absorbs infrared light and less readily absorbs red light due to allosteric changes that occur when Hb is bound by oxygen. These changes in chemical bonds increase the scattering of red light, so less is received by the photodiode. The relative amount of red and infrared lights received quantifies the proportion of Hb bound to oxygen.

An SpO₂ probe is placed around thin tissue with high vascular density such as the toe, finger or ear lobe. Under normal conditions, the sensitivity and specificity of modern oximeters with artefact rejection algorithms is 98%–99%. However, conditions that affect perfusion such as hypothermia may reduce SpO₂ accuracy, as well as conditions that affect Hb saturation, including sickle cell disease, cyanotic congenital heart disease, haemoglobinopathies and altitude. These factors impact the HbO₂ dissociation curve, and NPO alone should be used with caution to diagnose SDB due to absence of specific reference ranges.

Practical considerations
A range of pulse oximeters are now available, and the past decade has seen significant technological advancements. These include the incorporation of artefact rejection algorithms and the ability to apply a range of averaging times. Papers providing age-specific reference values (table 2) use these technologies, so device specification and settings are important for data analysis and interpretation.

Table 3  Saturation targets for weaning oxygen in ex-preterm infants using motion-resistant oximeters with short averaging times

<table>
<thead>
<tr>
<th>Corrected age post term</th>
<th>Minimum mean saturations (%)</th>
<th>Time less than 90% (%)</th>
<th>OD13</th>
</tr>
</thead>
<tbody>
<tr>
<td>37–40 weeks</td>
<td>&gt;93</td>
<td>&lt;3</td>
<td>&lt;35</td>
</tr>
<tr>
<td>40–44 weeks</td>
<td>&gt;93</td>
<td>&lt;3</td>
<td>&lt;30</td>
</tr>
<tr>
<td>44–56 weeks (1–4 months)</td>
<td>&gt;93</td>
<td>&lt;3</td>
<td>&lt;15</td>
</tr>
<tr>
<td>Over 56 weeks (over 4 months)</td>
<td>&gt;93</td>
<td>&lt;3</td>
<td>&lt;7</td>
</tr>
</tbody>
</table>

Reproduced from Everitt et al.²⁵

OD13, Oxygen Desaturation Index at 3%.

Figure 5  Nocturnal pulse oximetry (Visi-Download software) demonstrating periods of wake (grey) and sleep. Periods of wake are intimated by a sudden change and rise in heart rate. Artefact was removed prior to data analysis.

How does motion impact oximetry results?
Artefact may be caused by movement, reduced signal and poor probe adhesion. Artefact removal technology is particularly relevant to restless infants and mobile young children.

What is the impact of oximeter averaging time on results?
Averaging time refers to the specific window of time over which an SpO₂ value is estimated. Shorter averaging times allow detection of transient desaturations and prevent smoothing out of
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Figure 6  (6A) Cardiorespiratory sleep study demonstrating an obstructive event. (6B) Cardiorespiratory sleep study demonstrating a central event. RIP, respiratory inductance plethysmography. SpO₂, oxygen saturation.
events (figure 4).\cite{5} Within a preterm cohort, longer averaging times were associated with reduced detection of brief events and underestimation of desaturations of greatest severity\cite{6} with potential implications for neurodevelopmental outcomes and growth. Accordingly, international guidance when undertaking sleep studies recommends using a short averaging time of 2–3 s for accuracy\cite{7} for children and adults.

Is a single night of oximetry enough?
Recent British Thoracic Society consensus recommends a single night of pulse oximetry monitoring to diagnose severe OSA in TD children.\cite{2} Children with complex medical conditions demonstrate night-to-night variability in saturation indices, and multiple nights of monitoring requires consideration.\cite{8,9} Future research is necessary to determine the most consistent saturation indices for diagnostic reporting.

What is the minimum acceptable duration of overnight monitoring?
SDB is most likely to occur during active sleep and beyond infancy this is disproportionately dispersed throughout the night. The duration of total sleep should therefore ensure that periods of vulnerability are reflected which can be achieved by acquiring data for a complete night. The minimum acceptable study duration requires further research, however current recommendations propose artefact-free recording time (AFRT) of greater than 6 hours of continuous sleep duration.\cite{2} Four hours of AFRT including at least two active sleep cycles may be sufficient, particularly for infants where active sleep is more evenly distributed throughout the night.\cite{10}

Does supplementary transcutaneous CO2 monitoring improve the diagnostic accuracy of NPO?
Oxycapnography refers to the concomitant monitoring of end-tidal or transcutaneous CO2 and oximetry. It can be useful for discriminating between OSA and hypoventilation in children with comorbidities such as DS, although it should be noted that children with very severe OSA can retain CO2 secondary to airway obstruction. In these instances, the clinical history is helpful to discriminate between airway obstruction and hypoventilation. There should be a low threshold for undertaking more detailed cardiorespiratory sleep study (CRSS) if findings are not consistent with the clinical picture.

Optimising data interpretation accuracy for NPO performed in the home setting
NPO is commonly performed in the home setting, minimising potential disruption for infants, children
and young people and their families while avoiding an inpatient bed and associated attended costs. Experience from home sleep services recognises the importance of clear instruction for carers regarding equipment use and troubleshooting advice. Instructional videos and pictorial leaflets offer simplistic visual aids for carers, and qualitative feedback suggests these tools are informative. Data analysis requires appropriate staff training including the importance of artefact removal during periods of wake/disrupted sleep (figure 5). There may be value in shared education and training to support home pulse oximetry implementation and data analysis across a regional network.

Sleep audiovisual recordings are a typical feature of multichannel studies and may be recorded in NPO. Video information regarding obstructed breathing, effort of breathing, and the presence of apnoea and associated arousals may be useful to aid diagnosis; however, further research in this area is required. In the absence of electroencephalogram monitoring or concomitant video, comprehensive carer documentation regarding sleep onset, wakeful periods, feeding, alarms and respiratory events, including snoring and effort of breathing, is vital for data analysis and clinical interpretation.

Role of pulse oximetry as a screening tool for OSA in children with DS
Children with DS are vulnerable to SDB with reported prevalence of 75%. Generalised hypotonia and craniofacial anatomy contribute to upper-airway obstruction. NPO may be used as a screening tool in DS for moderate OSA, where early diagnosis is important to preclude the adverse consequences of OSA on limited cognitive reserve. Delta Index 12s (DI12s) is a measure of SpO2 variability and in children with DS, DI12s >0.555 and 3% oxygen desaturation index (ODI3) >6.15 are demonstrated to have high sensitivity to predict moderate and severe OSA. Night-to-night variability is recognised in DS and a multiple night study may provide a more representative indication of the impact of the airway on SpO2. There should be a low threshold for proceeding to more detailed CRSS if any abnormalities are identified on a screened oximetry trace or if findings are not consistent with the clinical picture. These studies should include capnography.

Role of pulse oximetry for oxygen weaning infants with CLDP
CLDP accounts for 68% of children in the UK requiring HOT. NPO is widely used to aid the oxygen weaning process, and a structured weaning approach is associated with improved outcomes and more rapid community weaning. Guidelines suggest performing an NPO study prehospital discharge to establish baseline requirement followed by monthly interval home NPO. A two-night oximetry study featuring night 1 at baseline and, where appropriate, night 2 weaned by 0.1 L/min is proposed. Weaning then takes place in accordance with age-specific reference ranges (table 3). Failure to wean HOT, growth or clinical concern at greater than 1 year of corrected age requires further investigation and consideration of referral for specialist respiratory review.

Role of pulse oximetry to predict OSA in TD children
OSA in TD children with adenotonsillar hypertrophy is typically a clinical diagnosis, and routine preoperative sleep monitoring, either NPO or a more detailed study is not universally recommended. Whilst NPO demonstrates high sensitivity for severe OSA for TD children over the age of 2 years and may provide information to guide the timing of adenotonsillectomy and inform perioperative care, for children with complex medical conditions (including obesity, DS, cerebral palsy and neuromuscular disease), and those under 2 years of age, NPO sensitivity is reduced, and a more detailed sleep study, such as CRSS, should be considered to confirm or refute the diagnosis. Furthermore, in children with comorbidities, a more detailed sleep study preadenotonsillectomy may help predict those at increased risk of perioperative complications and thus aid planning, reducing unscheduled admissions to paediatric high dependency and intensive care.

Analysis
Pulse oximetry is used to report a range of indices which include mean saturations (SAT50), the...
with maturity in young infants and so are helpful when weaning infants from oxygen. However, the calculation and presentation of these vary according to the software package employed. Interpreting data relative to published reference ranges involves consideration of both the hardware and software used to undertake the test.

Limitations

► Importantly, pulse oximetry is a single-channel investigation that does not discriminate the aetiology of oxygen desaturation events which can only be accurately determined using CRSS (figure 6A,B) or PSG.

► Sleep periods may only be estimated from heart-rate variability combined with carer report and interpretation.

► NPO may underestimate mild OSA, and if there are ongoing clinical concerns despite reassuring NPO, consideration should be given to more detailed study such as CRSS.

► Since pulse oximetry cannot discriminate between central and obstructive events, it has low diagnostic accuracy for children with complex medical conditions at risk of multifactorial SDB, and a more detailed, specific diagnostic study such as CRSS would be preferable.

► Pulse oximetry alone cannot accurately diagnose hypventilation, and addition of capnography may be helpful.

AREAS FOR FURTHER RESEARCH

► Future studies may seek to enhance our understanding of night-to-night variability and the diagnostic accuracy of NPO in medically complex children.

► Research may further explore the impact of differing target saturations on neurodevelopmental outcomes for infants with CLDP weaning HOT.

► Studies may further identify and define optimal targets for desaturation indices.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Not applicable.

Ethics approval Not applicable.

Provenance and peer review Commissioned; externally peer reviewed.

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REFERENCES


Interpretations


Answers to the multiple choice questions

1. A and E.
2. B, C and D.
3. A, B, C and D.
4. B and E.