NEW ZEALAND GUIDELINES FOR THE ASSESSMENT OF SLEEP-DISORDERED BREATHING IN CHILDHOOD

2014

Paediatric Society of New Zealand
STATEMENT OF INTENT

Clinical guidelines are produced to assist health professionals and consumers make decisions about health care in specific clinical circumstances. Research has shown that if properly developed, communicated and implemented, guidelines can improve care. While guidelines represent a statement of best practice based on the latest available evidence (at the time of publishing), they are not intended to replace the health professional's judgment in each individual case.

ACKNOWLEDGEMENTS

The guideline group thanks Kaleen Cooke for her support with formatting and distributing the Guideline document. Thanks also to XXXXX
The **Purpose** of the guideline is to provide an up to date evidence-based summary for the assessment of children with sleep disordered breathing.

This guideline seeks to provide the best evidence currently available to assist informed decision making by parents/caregivers and their health care providers to improve the health outcomes for children up to the age of 18 years with these conditions.

The **Scope** of the guideline addresses the best practice for the investigation and treatment of sleep-disordered breathing in childhood.

**Excluded:** the guideline does not address the management of sleep-disordered breathing in adults, the management of behavioural and non-respiratory disorders of sleep in children, nor does it intend to provide detailed management of respiratory disorders of neonates or infants.
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PURPOSE OF THE GUIDELINE
This guideline is intended for use by primary and secondary care practitioners involved in the care of children and young people with sleep disordered breathing.

The purpose of the guideline is to provide an evidence-based summary for the assessment of children with sleep disordered breathing. This guideline seeks to provide the best evidence currently available to assist informed decision making by parents/caregivers and their health care providers to improve the health outcomes for children with these conditions.

Copyright and Adaptation of the Guideline
The Paediatric Society of New Zealand encourages free exchange and sharing of evidence and guidelines, and the adaptation of the guidelines for local conditions. However, please note that guidelines are subject to copyright.

This guideline may be copied but acknowledgement must be given to the Paediatric Society of New Zealand: psnz@paradise.net.nz

Where guidelines are modified for local circumstances, significant departures from these national guidelines must be detailed with reasons for the departure. The Paediatric Society Guidelines group cannot be held responsible for such changes.

It is intended that this guideline be reviewed in 2020. Interim modifications will be made to the on-line version of the guideline as needed.

NOTE: The recommendations in these guidelines do not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.
### ABBREVIATIONS USED

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>ADHD</td>
<td>Attention deficit hyperactivity disorder</td>
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<tr>
<td>AHI</td>
<td>Apnoea hypopnea index</td>
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<tr>
<td>ALTE</td>
<td>Apparent life threatening event</td>
</tr>
<tr>
<td>AASM</td>
<td>American Academy of Sleep Medicine</td>
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<tr>
<td>AAP</td>
<td>American Academy of Pediatrics</td>
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<tr>
<td>ASA</td>
<td>Australasian Sleep Association</td>
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<td>ASTA</td>
<td>Australasian Sleep Technologist’s Association</td>
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<tr>
<td>AT</td>
<td>Adenotonsillectomy</td>
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<tr>
<td>BiPAP</td>
<td>Bilevel positive airway pressure</td>
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<td>BMI</td>
<td>Body mass index</td>
</tr>
<tr>
<td>CAI</td>
<td>Central apnoea index</td>
</tr>
<tr>
<td>CCHS</td>
<td>Congenital central hypoventilation syndrome</td>
</tr>
<tr>
<td>CF</td>
<td>Cystic fibrosis</td>
</tr>
<tr>
<td>CI</td>
<td>Confidence interval</td>
</tr>
<tr>
<td>CMDHB</td>
<td>Counties Manukau District Health Board</td>
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<tr>
<td>CPAP</td>
<td>Continuous positive airway pressure</td>
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<tr>
<td>CPF</td>
<td>Cough peak flow</td>
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<tr>
<td>CRP</td>
<td>C-Reactive protein</td>
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<tr>
<td>ECG</td>
<td>Electrocardiograph</td>
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<tr>
<td>EDS</td>
<td>Excessive daytime sleepiness</td>
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<tr>
<td>EEG</td>
<td>Electroencephalogram</td>
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<tr>
<td>EMG</td>
<td>Electromyography</td>
</tr>
<tr>
<td>ENT</td>
<td>Ear, nose and throat</td>
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<tr>
<td>EOG</td>
<td>Electro-oculogram</td>
</tr>
<tr>
<td>FEV₁</td>
<td>Forced expiratory volume in one second (“lung airflow measure”)</td>
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<tr>
<td>FRC</td>
<td>Functional residual capacity</td>
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<tr>
<td>FVC</td>
<td>Forced vital capacity</td>
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<td>HDL</td>
<td>High-density lipoprotein</td>
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<td>HDU</td>
<td>High dependency unit</td>
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<tr>
<td>HRCT</td>
<td>High resolution computed tomography “CT scan”</td>
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<tr>
<td>ICU</td>
<td>Intensive care unit</td>
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<tr>
<td>ITP</td>
<td>Idiopathic thrombocytopenia purpura</td>
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<tr>
<td>MOH</td>
<td>Ministry of Health</td>
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<tr>
<td>MSLT</td>
<td>Multiple sleep latency test</td>
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<tr>
<td>MWT</td>
<td>Maintenance of wakefulness test</td>
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<tr>
<td>NEPAP</td>
<td>Nasal expiratory positive airway pressure device</td>
</tr>
<tr>
<td>NIV</td>
<td>Non-invasive ventilation</td>
</tr>
<tr>
<td>NMD</td>
<td>Neuromuscular disease</td>
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<td>NPA</td>
<td>Nasopharyngeal airway</td>
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<tr>
<td>NPV</td>
<td>Negative predictive value</td>
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<tr>
<td>NREM</td>
<td>Non-REM sleep – slow wave sleep</td>
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<tr>
<td>OA</td>
<td>Oral appliance</td>
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<tr>
<td>OAHI</td>
<td>Obstructive apnoea hypopnea index</td>
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<td>ODI</td>
<td>Oxygen desaturation index</td>
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<tr>
<td>OSA</td>
<td>Obstructive sleep apnoea</td>
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<td>OSAS</td>
<td>Obstructive sleep apnoea syndrome</td>
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<td>OR</td>
<td>Odds ratio</td>
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<tr>
<td>PAP</td>
<td>Positive airway pressure</td>
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<td>PCD</td>
<td>Primary ciliary dyskinesia</td>
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<td>PLMs</td>
<td>Periodic limb movements</td>
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<tr>
<td>PPV</td>
<td>Positive predictive value</td>
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<td>PSG</td>
<td>Polysomnography</td>
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<tr>
<td>Abbreviation</td>
<td>Full Form</td>
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<tr>
<td>PWS</td>
<td>Prader-Willi syndrome</td>
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<tr>
<td>RDI</td>
<td>Respiratory disturbance index</td>
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<tr>
<td>REM</td>
<td>Rapid eye movement</td>
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<tr>
<td>RERA</td>
<td>Respiratory effort related arousal</td>
</tr>
<tr>
<td>RME</td>
<td>Rapid maxillary expansion</td>
</tr>
<tr>
<td>SDB</td>
<td>Sleep disordered breathing</td>
</tr>
<tr>
<td>SpO₂</td>
<td>Oxygen saturation measured by pulse oximetry</td>
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<tr>
<td>VC</td>
<td>Vital capacity</td>
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FOREWORD

In 2004 the New Zealand Guidelines group generated an evidence-based summary for the assessment of children with sleep disordered breathing (SDB). At that time the issues identified by the group were:

1. How should general practitioners and paediatricians approach the investigation of a child who snores?
2. Which children should be referred from around New Zealand to a sleep disorders centre for further evaluation?
3. What treatments are available for disorders of breathing during sleep in childhood and what are the known benefits of these treatments?

The document the American Academy of Paediatrics Clinical Practice Guideline: Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome (OSAS) (2002)\(^1\) with its accompanying technical report\(^2\) was selected as a base document for the New Zealand Guideline. This document found the following key points:

- The guideline was developed by a large group of professionals from a variety of specialties,
- The guideline is for use by primary care practitioners and the recommendations are for the diagnosis and treatment of obstructive sleep apnoea,
- The guideline is for children aged 2-18 years,
- The literature review was systematic and criteria for selection described, although there was no discussion on rejected papers, and
- The recommendations are clear and are related to the evidence.

The reviewers recommended the use of the guideline for the development of a New Zealand guideline, with adaptations for use in New Zealand particularly in relation to:

- ethnic and cultural differences
- the availability of polysomnography (PSG)
- an updated review of the literature including local and Australian papers to 2004
- how the diagnosis of OSAS should be made in New Zealand
- what tests are possible in regional centres as distinct from tertiary referral centres.

Since 2004 paediatric sleep medicine has evolved significantly internationally. The rationale for this revision of the document is to integrate relevant recommendations from evidence based literature published since 2004 and to incorporate new research up to 2014 and apply this to New Zealand resources and facilities. The aim was not to completely rewrite the guidelines but to include new areas of relevance and expand on growing areas specific to the assessment and management of SDB.

As in 2004, the revised American guidelines document the American Academy of Paediatrics Clinical Practice Guideline: Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome (2012)\(^3\) with its accompanying technical report\(^4\) has again been selected as a base document for the revision of the New Zealand guideline. Resources were more limited for the revision process. There was no technical support for the search and critical appraisal of the literature. As such the grading of evidence was not possible. Owing to lack of funding, the 2014 working group, which included many of the interested clinicians from the original group, was unable to meet face to face to review the guideline draft, so the document has been developed by multiple teleconferences and email correspondence.
What’s new in the 2015 Guidelines?

There are several changes and updates throughout this guideline. The major ones include:

- Removal of evidence based tables
- New sections:
  - 1.2 Indications for a sleep study
  - 1.3 Types of sleep studies
    - 1.3.1 Attended polysomnography (Type 1 sleep study)
    - 1.3.2 Unattended polysomnography (Type 2 sleep study)
    - 1.3.3 Cardiorespiratory / limited channel sleep study (Type 3 sleep study)
    - 1.3.4 Overnight Pulse Oximetry (Type 4 Sleep Study)
  - 2.6 Obesity
- New figure
  - Figure 2. Guide to scoring tonsillar size
- New information in appendices
  - Appendix 3 & 4 revised for AASM 2014 document and additional information to guide PSG based severity in paediatric sleep studies
- Additional information and significant revision to sections:
  - 2.2 Treatment with adenotonsillectomy without polysomnography
  - 2.4.1 CPAP and newer alternative treatments
  - 2.4.2 Other treatments – which includes anti-inflammatories, dental and oral appliances.
  - 2.5 Complex high risk children
  - Appendix 1. The algorithm / summary for investigation and management of suspected OSA in children undergone significant revision reflecting recommendations and updates incorporated throughout document.
INTRODUCTION

In 1889, Hill described snoring and restlessness at night as a cause of “backwardness and stupidity in children”\(^5,6\), but nearly 100 years passed before the first case series of children with obstructive sleep apnoea (OSA) was published\(^7\). Since that time, it has been recognised that OSA is one of the most common respiratory disorders of childhood, affecting an estimated 3-5% of normal children\(^3,8,9\) depending on the definition used and population studied. In 1982, Brouillette et al published a case series of 22 infants and children with severe complications of OSA such as cor pulmonale and failure to thrive\(^10\). In recent years, research in this condition has mushroomed, bringing increasing recognition that untreated OSA may result in significant adverse consequences even in milder cases of OSA\(^11-18\). In children, episodes of upper airway obstruction during sleep may lead to brief arousal from sleep without desaturation. These arousals can be associated with increases in heart rate and blood pressure which may have long term cardiovascular implications\(^19-21\), and may also be the mechanism by which OSA affects learning and behaviour during the day\(^14,15,22-25\).

In addition to the emerging research in OSA, there has been a considerable increase in publications and research on disorders of breathing during sleep in children with medical disorders such as neuromuscular disease (NMD), obesity, craniofacial syndromes and chronic lung diseases. In 2004, the original guideline on the assessment of SDB in childhood was published by the Paediatric Society of New Zealand Guidelines group. The current document provides a brief update and overview of the evidence in this emerging field particularly over the last 10 years, to provide up to date guidance for general practitioners and paediatricians caring for children with these conditions in New Zealand.

Terminology

The obstructive sleep apnoea syndrome (OSAS) in children was defined by the American Academy of Pediatrics as a disorder of breathing during sleep characterised by prolonged partial upper airway obstruction (obstructive hypopnoea) and/or intermittent complete obstruction (obstructive apnoea) that disrupts normal ventilation during sleep and normal sleep patterns\(^1\). In this document we use the term obstructive sleep apnoea, or OSA, to refer to this spectrum of severity of obstructive events during sleep and its consequences.

The term “sleep-disordered breathing” (SDB) is used as an umbrella term for all conditions where ventilation or sleep patterns are disturbed by abnormalities of breathing, including for example OSA or hypoventilation related to neuromuscular diseases or other disorders.

Habitual snoring is defined as snoring every night even when the child is otherwise well and is the cardinal feature of OSA.

Primary snoring is defined as habitual snoring that is not associated with hypoxaemia, hypercarbia, sleep disturbance or daytime symptoms.

Maori Health Issues

There remains limited published data on the prevalence of snoring, sleep disordered breathing or OSA in the New Zealand paediatric population. Two studies from adult sleep clinics in New Zealand undertaken 15-20 years ago, suggested that the prevalence of OSA is higher amongst Maori than non-Maori\(^26,27\). The 2004 guidelines cited two abstracts that looked at ethnic differences in prevalence of OSA symptoms in New Zealand adults\(^28,29\). The findings cannot necessarily be extrapolated to the paediatric population.

Since 2004 the research, previously in abstract format, was combined and published as one paper\(^30\). This described the evidence from a New Zealand national survey in adults that Maori have a higher prevalence of OSA than non-Maori\(^30\). This involved a population based
mail-out survey (71% response rate) and was sent to 10,000 New Zealanders (5,500 of Maori descent and 4,500 non-Maori) aged 30-60 years selected at random from the electoral roll, and in a parallel study overnight home based cardio-respiratory sleep studies of a similarly aged stratified random sample (n=364). Results showed that symptoms of OSA are common in a New Zealand adult population and that Maori had higher rates of SDB than non-Maori in that they were significantly more likely to ‘always snore’, have ‘observed apnoea’ and have daytime sleepiness than non-Maori. The higher risk among Maori reduced and became non-significant after adjusting for well-recognised risk factors such as increased body mass index and large neck size. The prevalence of OSA was conservatively estimated to be 4.4% for Māori men, 4.1% for non-Māori men, 2.0% for Māori women, and 0.7% for non-Māori women.

The increased prevalence’s of OSA among American Indians and Hispanic adults, and increased severity among Pacific Islanders and Maori, appear to be mainly explained by increased obesity in these ethnicities. However, ethnic differences in cephalometry could, in theory, influence OSA. Most cephalometric studies have largely been conducted without specific regard to ethnicity and comparisons of findings across studies have been mainly limited by differences in sampling methods and the varying selection and definition of measured cephalometric variables.

There is one other study in adults which evaluated the influence of ethnicity on adherence with continuous positive airway pressure (CPAP). Participants underwent a 4-week supervised home trial of CPAP following pressure titration. Māori demonstrated significantly lower usage than non-Māori (median 5.11 versus 5.71, p = 0.05). Using multivariate regression modelling the disparity in CPAP adherence can be explained in part by lower education levels and socioeconomic status. While these findings cannot be extrapolated to the paediatric population it does support that research in this area is warranted.

To date there is only one published paediatric study to investigate the prevalence of SDB symptoms in a community sample of New Zealand 3-year olds. This was a cross sectional study in which parents of 823 children were recruited from the Dunedin community. Participants completed questionnaires designed to assess information relevant to their children's sleep, with a particular focus on snoring. Parents reported snoring at least once a week in 36.9% of children, and habitual snoring in 11.3% of children. Univariate analysis showed habitual snoring was more common amongst Māori (p = 0.04) and males (p = 0.05), and that habitual snorers came from more socio-economically deprived neighbourhoods (p<0.01). While this is a relatively small sample and cannot be extrapolated to the general New Zealand population, the prevalence of habitual snoring amongst New Zealand pre-schoolers was similar to that reported elsewhere. This study highlights the need for more research nationally to define and describe the occurrence of SDB and document ethnic and cultural differences.

GAPS BETWEEN EVIDENCE AND CURRENT PRACTICE

In revising these guidelines the working group continued to face the challenge of reconciling previously published guidelines with current New Zealand practice, and finding a way to bridge the gaps. Wholesale adoption in New Zealand of the American Academy of Pediatrics Guidelines for the diagnosis and management of childhood OSA syndrome (2012), (with or without recent updates) continues to be impractical, as the number of children who would require referral for PSG and other work-up would greatly exceed our current capacity. On the other hand, restricting guidelines to resources currently available in New Zealand would be misleading as it would ignore the real burden of ill health from sleep disordered breathing in children.
In 2004 the combined expertise of the team, following much iteration of clinical questions and resource implications, identified the following gaps to try and rationalise evidence based recommendations with the possibilities for improvement in clinical practice in New Zealand. Unfortunately the same gaps remain in 2014.

**What are the gaps?**
The principal gaps between evidence based recommendations and current practice in the field of SDB in childhood in New Zealand are:

- clinical recognition of at risk children
- availability of appropriate investigations, particularly PSG, and
- provision of timely treatment.

This revision continues to balance the international recommendations for "gold standard" investigation of SDB – PSG – with what is practically achievable within current clinical resources.

**How much effort will it take to close the gaps?**
The revised guidelines will help to rationalise the assessment and management of children with SDB, and to coordinate limited resources to identify those in need of investigation and treatment.

Primary care will continue to be the first point of contact for many children with suspected sleep-disordered breathing. Direct referral by a General Practitioner to an otolaryngologist (Ear, Nose and Throat (ENT) Surgeon) for consideration of adenotonsillectomy will be appropriate in many cases. Secondary care provided by general and regional paediatricians will be sought when there is doubt about the diagnosis, or in situations where a child has significant co-morbidity such as NMD, obesity, Down Syndrome or spina bifida. Paediatricians will also play a key role in the education of general practitioners and in promotion of child health in their regions. Tertiary care will provide diagnostic investigation and implementation of treatment for those children referred from secondary care services.

As with the original guideline, the revised guideline is intended for general practitioners and paediatricians, as well as otolaryngologists who treat children. Full implementation of the recommendations depends on widespread circulation of the guideline to general practitioners and paediatricians, which will be co-ordinated by the PSNZ and the paediatric sleep medicine clinical network group.

**Is there a reasonable likelihood that the recommended changes could be implemented?**
As in 2004 the key impacts of this revised guideline are likely to be changes in the investigation and management of children with suspected SDB. It is envisaged that implementing these changes will have the following potential impacts:

1. Increased recognition of symptoms of OSA, and the consequences of this condition if not treated, may lead to increased referral to otolaryngologists for adenotonsillectomy. Some increase in ENT resources may be required to meet this need.
2. The more widespread use of overnight oximetry as a tool for the assessment of a child with suspected SDB has already resulted in an increased demand for these tests. Since 2004 many regional (but not all) paediatric centres and some general practitioners have been undertaking this type of testing. There is ongoing need for access to and education on the limitations of oximetry and its interpretation, and this requires support.
3. Increased availability of comprehensive sleep studies (PSG) will be required to meet international standards for certain conditions. The provision of such services for children continues to lag considerably behind that provided to adults with similar conditions around New Zealand and has not progressed since 2004: in fact in many ways the gap...
has widened. Significant government investment is needed to address this critical deficiency in paediatric sleep services.

These potential costs should be balanced with evidence from the literature of benefits and cost savings in the following areas:

- **Decreased use of health services.** Children with OSA have high morbidity and use of health services from the first year of life before OSA is diagnosed, implying that earlier recognition, accurate diagnosis and treatment will result in cost savings.\(^{33-35}\)

- **Decreased use of special educational services.** Behavioural problems and deficits in neurocognitive functioning have been well documented in children with OSA. Treatment of OSA leads to improvements in quality of life, and improvements in behaviour and mental functioning in children including suspected ADHD. Thus, diagnosis of the condition and appropriate treatment both impact on learning and school performance.

- **Decreased use of hospital services.** Institution of non-invasive ventilation (NIV) in children with chronic respiratory failure secondary to NMD leads to decreased hospital admissions, reduced hospital days, and reduced intensive care days.\(^{54-57}\)

- **Decreased use of support services.** Non-invasive ventilation (NIV) in children with chronic respiratory failure secondary to NMD is associated with improvements in quality of life, and subsequent decreased use of support services such as psychology, psychiatry, pain specialist and potentially delay input from palliative care services.

**Closing the gaps**

Improved equity of access:

- to safe effective treatment for those children who are very likely to benefit (e.g. adenotonsillectomy)
- to limited investigation by secondary services for those children at increased risk of co-morbidity or of post-surgical complications, and
- to tertiary workup including PSG for those children likely to benefit (e.g. from non-invasive ventilation)

will improve the well-being of individual children, with fair distribution of resources.

**Performance measures**

The following remain the proposed as potential performance measures to evaluate the impact of the guideline:

- Waiting times for adenotonsillectomy
- Total numbers of adenotonsillectomy by indication
- Complications following routine ENT surgery
- Waiting time for sleep studies (both hospital/sleep laboratory studies and home overnight oximetry)
- Total numbers and types of sleep studies undertaken nationally on children aged < 14 years
- Feedback from consumer groups about the availability of paediatric sleep medicine services

**Recommendations**

<table>
<thead>
<tr>
<th>Recommendation</th>
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<tr>
<td>There should be increased availability of adenotonsillectomy for the treatment of OSA for New Zealand children.</td>
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<tr>
<td>There should be increased availability of paediatric diagnostic sleep medicine services, especially PSG, for New Zealand children.</td>
</tr>
<tr>
<td>There should be consistent access to a nationally integrated paediatric sleep service for the investigation and management of SDB for New Zealand children.</td>
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SECTION 1 – SLEEP SERVICES & TECHNICAL CONSIDERATIONS

1.1 Sleep services for children in New Zealand

In 2014 sleep services specifically for children are currently available in Dunedin, Christchurch, Palmerston North, Auckland, and Wellington Hospital and at WellSleep (Otago University Wellington). These services continue to be in evolution. It is therefore suggested that general practitioners and paediatricians familiarise themselves with their nearest centre’s resources, and contact those centres directly to discuss cases as needed:

- Auckland - Starship Children’s Health: Drs Jacob Twiss, Elizabeth Edwards, and David McNamara
- Palmerston North: Dr Jeff Brown
- Wellington and WellSleep: Professor Dawn Elder
- Christchurch: Associate Professor Phillip Pattemore
- Dunedin: Professor Barry Taylor

Some adult sleep services offer home or laboratory PSG for children in New Zealand (e.g. Waikato sleep services Hamilton; Edensleep; New Zealand Respiratory and Sleep Institute, Auckland) but without paediatric assessment and consistent use of paediatric standards. It is therefore recommended that before a full PSG is undertaken, a clinical opinion from a clinician with expertise in paediatric sleep medicine is obtained.

Currently, the paediatricians in New Zealand with formal qualifications in paediatric sleep medicine are Dr Elizabeth Edwards, Dr Jacob Twiss and Dr David McNamara at Starship Children’s Hospital and Professor Dawn Elder in Wellington. Paediatricians around the country who have an interest and clinical experience in sleep medicine include: Professor Barry Taylor (Dunedin), Dr Jeff Brown, (Palmerston North), and Associate Professor Philip Pattemore (Christchurch).

1.2 Indications for a sleep study.

There are three broad indications for performing a sleep study:

- **Diagnostic**: to assist in the diagnosis and evaluation of the severity of a sleep disorder.
- **Assessment of an intervention**: to initiate, confirm the adequacy of or titrate a treatment.
- **Follow-up / surveillance**: to re-evaluate a sleep disorder or the impact of a treatment over time as the child grows and develops.

**Diagnostic studies:**

**Polysomnography:**

- PSG can be used to:
  - determine the aetiology of SDB, including OSA, central apnea, hypoventilation and increased upper airway resistance;
  - investigate sleep-related causes of excessive daytime sleepiness (EDS) such as narcolepsy or sleep fragmentation due to frequent nocturnal arousals (e.g. periodic limb movements (PLMs));
  - define the aetiology of episodic nocturnal phenomena (e.g. parasomnias versus nocturnal seizures)

**Intervention studies:**

Intervention studies are undertaken to titrate or confirm the effectiveness of a new treatment. Therapies that require assessment of reversal or alleviation of SDB include CPAP, NIV and sometimes dental devices or surgical procedures.
Follow-up studies
Where treatment for a sleep-related breathing disorder has been successfully instituted it is important to ensure that with growth and development of the child the efficacy is maintained or that the need for the therapy persists at all such that the intervention can be withdrawn. Reassessment of the severity and presence of any underlying SDB is required if there is: significant weight change, persistence of symptoms following ENT surgery, or worsening of symptoms in cases previously assessed as having normal sleep, “normal”, primary snoring or mild OSA.

Investigation of EDS or persistence of daytime sleepiness despite correctly prescribed treatment, may require additional tests which are beyond the scope of this document. Tests that may be considered include actigraphy; multiple sleep latency test (MSLT); maintenance of wakefulness test (MWT) to estimate sleep wake pattern from activity, establish objectively the level of daytime sleepiness, and rule out alternative causes of daytime sleepiness.

1.3 Types of sleep studies
It is evident that the burden of disease associated with sleep disorders in children is great, given their high prevalence and significant associated morbidities. There is substantial pressure on the limited number of specialized facilities throughout New Zealand to meet growing demands for diagnosis and treatment, with the result that the waiting list for sleep studies in most centres is long. This high demand for investigation plus the relatively costly and labour intensive nature of attended PSG has led to the development of a number of simplified or “abbreviated” sleep studies. This is especially the case with OSA. These abbreviated studies have been classified according to the number of channels of physiological data recorded.

As objective testing is preferable to clinical evaluation alone, some centres may choose to carry out abbreviated studies, particularly to prioritise the waiting list for PSG and/or adenotonsillectomy (as the first line treatment for OSA in children). Given the potential for continuing adverse consequences of undiagnosed and untreated OSA, a pragmatic approach to diagnosis that expedites treatment for symptomatic children appears appropriate.

In choosing which test or tests are to be used, paediatricians should have a clear understanding of: (a) the question to be answered by undertaking the test i.e. indications for testing, (b) the sensitivity and specificity of the test(s) to diagnose a particular sleep disorder, (c) the overall utility of the test taking into consideration the prevalence of a given sleep disorder in the paediatric population, (d) the cost / benefit balance of the test in their particular clinical setting(s), (e) the technical limitations of the monitoring signals utilised in each particular study type and (f) comorbidities which may affect the reliability and interpretation of the result.

Ultimately the type of sleep study performed will be defined by local facilities, experience and expertise. If unattended and/or abbreviated sleep studies are all that is available, it is essential that those paediatricians reviewing the study reports remain objective and realistic about the limitations of such tests. In all sleep studies of any type, the raw data must be available for review by the sleep specialist interpreting or reporting on the studies, to give some estimate of quality/ ensure technical adequacy and accuracy of reporting.

Sleep studies may be divided into two broad categories (Amended from ASA document May 2014 “Guidelines for Sleep Studies in Adults” Ching Li Chai-Coetzer et al. http://www.sleep.org.au/documents/item/1112):
- Polysomnography (Type 1 & 2) or "comprehensive" study is considered the reference standard against which other diagnostic methods are evaluated. Twelve to thirteen
recording channels are routinely recorded in a facility where there is supervision by staff trained in the performance of PSG. The continuous recording of multiple physiological variables measures sleep architecture and cardio-respiratory function during sleep. A measure of carbon dioxide is recommended as well as sound and digital video recording.

- **Limited channel sleep studies (Type 3 & 4):** A smaller number of variables are recorded (see below), usually a combination of respiratory variables including arterial oxygen saturation, respiratory effort and airflow/nasal pressure. Sleep staging is usually not possible from limited channel sleep studies that do not include EEG, EOG and EMG. A Type 3 study is one that incorporates at least 4 monitored channels. A Type 4 study only incorporates one or two variables.

In turn these studies may be either:

- **Supervised:** a study continuously attended by medical, scientific/technical or nursing staff specifically trained in the performance of sleep studies, or
- **Unsupervised:** a study where staff are absent during the recording period. These studies are usually undertaken using specific portable equipment and often undertaken in the home or in the hospital ward.

Their duration may be:

- **Full night:** a study conducted over the entire normal sleep period, beginning at the usual bedtime and usually lasting more than 6 hours.
- **Split-night study:** an overnight sleep study "split" into two parts. It has a diagnostic period followed by a therapeutic intervention (most commonly, identification of moderate or severe OSA followed by CPAP titration).
- **Limited or restricted duration:** a study where the planned length of study is less than 6 hours. Such studies would include "nap" studies conducted during the hours of daylight e.g. to study the aetiology of SDB in neonates.

**Types of Sleep Study**

1.3.1 **Attended polysomnography (Type 1 sleep study)**

A type 1 sleep study is considered the reference standard against which other diagnostic methods are evaluated.\(^3\, ^{87}\, ^{68}\, ^{70}\). A type 1 study refers to a sleep study carried out throughout the night in a sleep laboratory in the presence of trained technical staff and under the supervision of a qualified sleep specialist. It involves the continuous recording of multiple physiological variables so that detailed measures of sleep and breathing can be made. Recommended signals include: electroencephalogram (EEG) signals, bilateral electro-oculograms (EOGs), submental electromyography (EMG), electrocardiography (ECG), pulse oximetry, respiratory thoraco-abdominal movements, nasal pressure and oronasal airflow, sound, a measure of carbon dioxide (transcutaneous or end tidal), position, digital video recording and leg EMG for periodic limb movements. The reader is referred to the AASM (2014)\(^399\) for details of the variables that should be measured and technical recording specifications during PSG. Whereas it is recognised that some children may sleep poorly the first night they are in a sleep laboratory (because of anxiety, unfamiliar environment and attached sensors), research evaluating this so-called first night effect suggests few children are misclassified on the basis of a single night PSG\(^71\, ^{74}\).

PSG enables measurement of sleep architecture (the amount and distribution of the various stages of sleep) and accurate quantification of respiratory events (against time spent asleep). Rapid eye movement (REM) sleep is frequently associated with exacerbation of a SDB abnormality and, in some cases; sleep apnoea/hypopnoea may be confined entirely to REM sleep. PSG distinguishes obstructive from central events, determines the effects of body position on sleep disordered breathing, allows the recognition of alternative diagnoses (e.g. epilepsy, PLM disorder, parasomnias) and may reveal other sleep disorders (e.g. narcolepsy). PSG provides information on sleep fragmentation and arousals and their
aetiology, which are likely important in the genesis of daytime symptoms arising from abnormal sleep-related respiratory events. Body and head position can be confirmed with synchronised video monitoring and electronic position sensors.

Overall there is a large body of evidence supporting the reliability and accuracy of attended PSG studies\(^4,65,68\). However attended PSG is expensive and a very limited resource in New Zealand when compared to the numbers of children at risk of SDB.

### 1.3.2 Unattended polysomnography (Type 2 sleep study)

An unattended PSG is usually undertaken at home, but may also be carried out on a hospital ward. A higher success rate in terms of good quality signals may be achieved when the study is set up by experienced personnel rather than when set up by parents or untrained health care professionals\(^71,75\). While unattended and attended PSGs may involve identical channels, an unattended PSG or ambulatory study may record a minimum of seven channels including EEG, EOG, chin EMG, ECG or heart rate, airflow, respiratory effort and oxygen saturation. The important distinction from cardiorespiratory and overnight oximetry studies is that in a PSG sleep itself is staged and quantified as well as respiratory recordings. Audio and video monitoring improves the reliability of unattended studies\(^76\).

This type of testing allows for sleep staging, and therefore calculation of total sleep time and respiratory indices (e.g. obstructive apnoea hypopnea index (OAHI – see Appendix 3)). One of the major limitations is the potential for data loss overnight, particularly airflow signals (thermistor or nasal pressure). However, the potential concern regarding signal loss has not been borne out by recent studies showing high success rate (91%) in children\(^75\). However, studies to date have included uncomplicated normal children of varying ages, and excluded children with complex medical problems, neurodevelopment delay and significant comorbidities – for example in a recent study by Marcus et al only 3% n=6 had cerebral palsy/developmental delay\(^75\). Further research is required to compare the accuracy of unattended with attended PSGs in children with medical comorbidities who often require paediatric sleep evaluation.

### 1.3.3 Cardiorespiratory / limited channel sleep study (Type 3 sleep study)

Limited channel cardiorespiratory sleep studies measure a more restricted panel of variables. Usually cardiorespiratory studies monitor at least four parameters: oximetry plus respiratory effort (chest, abdominal, or both), airflow (nasal or oral by pressure or thermistor), head or body position, ECG, tonometry (a marker of autonomic control), actigraphy (vibration detection) and sound/video recording. These studies may be attended or unattended and can be performed in the sleep laboratory, hospital or at home.

The diagnostic accuracy of cardiorespiratory studies is limited by the lack of estimate of total sleep time. Total recording time is used as the denominator, resulting in an underestimate of the severity of SDB. Also SBD leading to arousal and secondary sleep disruption rather than desaturation may be missed by limited channel studies (cardiorespiratory or overnight oximetry) when sleep is not assessed. Video recording may reduce this error as body movement arousals can be scored visually\(^76\).

Whereas cardiorespiratory studies are more accessible and low cost, they have a variable failure rate due to data loss, particularly when the setup is performed by caregivers rather than trained staff. Rosen et al 2003\(^77\) using a cardiorespiratory device at home on children aged 8 to 11 years, demonstrated that 94% of studies were technically adequate and when compared to PSG, in a subset undergoing laboratory PSG, cardiorespiratory studies showed good sensitivity and specificity. However, although this cohort study reported ‘excellent agreement’ no actual data were reported. Zuconi et al 2003\(^78\) evaluated a cardiorespiratory device in the hospital ward (children 3-6 year, N=12) compared with laboratory PSG on an alternative night. This showed good sensitivity (89%) but very poor specificity. Poels 2003\(^79\)
used a home caregiver set-up device in 2-7 year old children (n=24) prior to adenotonsillectomy and due a high failure rate (25-30%) concluded cardiorespiratory studies were of limited use. Conversely, Jacob et al 1995\textsuperscript{76} reported that limited channel home testing plus video was an adequate (83%) and practical option in the evaluation of routine OSA in children (n=21, 2-12 years) with adenotonsillar hypertrophy. This study involved specialist staff setting up the unit at home (PPV ≥ 70% and NPV > 90%). A recent study by Tan et al found that clinical decisions at the extremes of SDB (normal/primary snoring with OAHI<1, or severe OSA with OAHI>10/h) were unaffected by the choice of cardiorespiratory over PSG studies, whereas in the middle range of OAHI (1-10/h), a cardiorespiratory study may underestimate events and have significant impacts on treatment decision\textsuperscript{80}. Overall the literature suggests that cardiorespiratory sleep studies typically have a high positive predictive value for the presence of OSA, but a significant false negative rate (low negative predictive value).

In the setting of limited availability of full PSG, cardiorespiratory studies may be useful in cases where a child is too unwell to have a formal study undertaken in a sleep laboratory, or as a way of simply determining the efficacy of treatment in the early stages of initiation of non-invasive respiratory support.

1.3.4 \textit{Overnight pulse oximetry (Type 4 sleep study)}

Overnight oximetry incorporates only one or two measured parameters – for example oxygen saturation, heart rate, transcutaneous carbon dioxide or airflow. Such studies are usually unattended and conducted in the patient’s home or on a hospital ward.

Oximetry guidelines are available on the Starship Children’s Heath Guidelines (http://www.adhb.govt.nz/StarShipClinicalGuidelines/Oximetry.htm) to assist clinicians with the performance, reporting and interpretation of paediatric overnight pulse oximetry studies. This is an excellent resource and the working group recommend that health professionals at any centre undertaking oximetry should be familiar with and utilise these guidelines for education of other health professionals and paediatric trainees.

Oximetry is an accurate, quantifiable, reliable and informative signal\textsuperscript{81}. The development of the multiwave length oximetry and reduction in device size has made oximetry a ubiquitous and accurate marker of hypoxemia.

As is the case for cardiorespiratory studies however, a focus on desaturation events will miss children with OSA who have significant sleep disruption without desaturation (high positive predictive value but low negative predictive value). As with all tests, it is critical for the requesting health professional to understand the indications for pulse oximetry, what the instrument measures and reports, its limitations and how to interpret results.

Technical factors such as adequate signal acquisition, averaging time and storage sampling frequency are crucial to the reliability of oximetry. Careful choices of equipment and software are needed for services undertaking oximetry for diagnostic purposes, and results should be interpreted by trained practitioners. Errors in interpretation may lead to inappropriate treatment decisions being made. Further, there is a lack of positional data, and the calculation of the level of respiratory disturbance is based on time in bed rather than actual sleep time.

Additional limitations of overnight oximetry studies (including oximetry with heart rate) apply to cardiac, pulmonary and neurological patients. In these children overnight oximetry is an unsuitable tool to make the important differentiation between obstructive and central sleep apnoea. Low baseline awake oxygen saturation will reduce specificity further. Supplemental oxygen therapy may negate the utility of signal interpretation.
Despite these limitations, the appearance of the trend in pulse oximetry and heart rate, and the analysis of desaturation events measured on overnight oximetry may be helpful as a quick, inexpensive alternative to a PSG for the diagnosis of OSA. In the context of a sleep laboratory with a high (60%) pre-test probability of OSA in uncomplicated referred patients, a positive oximetry trend graph (see Figure 1) (defined as ≥ 3 clusters (a cluster = ≥ 5 desaturations within a 30 minute period) with ≥ 3 desaturation to< 90%) had a positive predictive values (PPV) of 97% and could therefore reliably predict the need for adenotonsillectomy without the need for further testing. On the other hand a patient with a negative oximetry had a post-test probability of OSA of 47% (negative predictive value, NPV) and therefore required further testing to exclude or confirm the diagnosis of OSA. Children with frequent oxygen desaturation events < 80% are at increased risk of requiring major airway intervention such as re-intubation in the post-operative period, a finding that mandates careful planning of surgical intervention and perioperative care. Recently Horwood et al. demonstrated that oximetry studies evaluated with the McGill oximetry score expedite diagnosis and treatment of children with adenotonsillar hypertrophy referred for suspected OSA. Pavone et al demonstrated good reproducibility of oximetry testing.

Overnight oximetry may also be evaluated using frequency of desaturation events (oxygen desaturation index, ODI). However Kirk et al 2003 showed poor agreement between the ODI and AHI measured by PSG (AHI > 5/hour sensitivity 67% % specificity 60% PPV 60% & NPV 67%). Pena-Zarza 2012 reported on the use of oximetry along with clinical evaluation and paediatric sleep questionnaire. No one tool was reliable when compared to laboratory PSG but used in conjunction the threefold assessment had a high specificity (98%) and PPV (94%).

In the hospital environment overnight oximetry studies, particularly in conjunction with transcutaneous carbon dioxide measurement, may be useful for the assessment of children and young people with NMD at risk of development of hypoventilation, or as an easy method of checking adequacy of non-invasive respiratory support. For other medically fragile children for whom hypoventilation may be a component of their respiratory status (e.g. achondroplasia, spina bifida, severe obesity etc.) assessment of the child's gas exchange may be helpful in prioritising further evaluation or planning for definitive PSG at a future date.

<table>
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<tr>
<th>Recommendations</th>
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<tbody>
<tr>
<td>Regardless of the availability or not of sleep studies, it is recommended that children presenting with sleep problems be assessed with a comprehensive history and clinical assessment.</td>
</tr>
<tr>
<td>As overnight oximetry is commonly used in New Zealand to screen for OSA and to support a referral for ENT intervention, it is recommended that all health professionals are educated on the limitations of oximetry and how to interpret the results.</td>
</tr>
<tr>
<td>It is recommended that limited channel diagnostic devices are only used in developmentally normal children with a high clinical suspicion of OSA following specialist review.</td>
</tr>
<tr>
<td>While home unattended studies have been shown to be feasible, as they require attention to safety, it is recommended that where possible, trained staff should set up the monitors, and adequate parental instruction be given to ensure technically reliable information.</td>
</tr>
<tr>
<td>Despite the availability of limited channel sleep studies in New Zealand, it remains vital that there is access to attended PSG studies for the comprehensive and definitive assessment of SDB, particularly for those children with complex health needs.</td>
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SECTION 2: SNORING AND OBSTRUCTIVE SLEEP APNOEA

2.1 Screening by history and examination - “The question of snoring”

Obstructive sleep apnoea is a common condition in childhood: several large epidemiologic studies in western societies have found that about 1 in 10 pre-schoolers snore and 3-5% of children have OSA. OSA is associated with significant morbidity, reduced quality of life, failure to thrive, behavioural problems, cognitive impairments, cor pulmonale, right ventricular hypertrophy and systemic hypertension.

Habitual snoring is the cardinal clinical feature of OSA. The diagnosis is unlikely in children who do not snore. Snoring should not be assumed to be normal in childhood and may indicate OSA requiring treatment. Indeed, children with primary snoring have also been shown to have more behavioural problems than non-snoring children.

“Noisy breathing during sleep” may be a better screening question, as children often do not have the vibratory type of snoring seen in adults especially if they are predominately mouth breathing. All children should be screened for a history of OSA at clinical visits by asking about a history of snoring or noisy breathing during sleep.

Within the group of children who snore, the literature would suggest that other aspects of the history, and features on clinical examination, have poor specificity for the presence of OSA. However, certain symptoms increase likelihood of significant OSA in children who snore:

- witnessed obstructive apnoea (odds ratio (OR) 3.3)
- frequent daytime mouth breathing (OR 3.7)
- parent afraid / wakes child because of breathing (OR 4.4)
- struggling to breathe while asleep (OR 5.5)
- frequent waking from sleep in a child who has previously slept through
- secondary enuresis
- daytime behavioural problems
- failure to thrive or slowing of weight gain

The use of a structured validated questionnaire (for example the Chervin questionnaire or link to Starship Children’s Health, Clinical guidelines on OSA Appendix 2 https://www.starship.org.nz/for-health-professionals/starship-clinical-guidelines/o/obstructive-sleep-apnoea/#Questionnaire) improves the specificity of the history for OSA by combining many of these symptoms into a total score. Combining a questionnaire with physical examination (adenoidal facies, nasal obstruction and tonsillar hypertrophy) and overnight home oximetry results improves specificity to 98% with a PPV of 94% in moderate to severe cases.

Excessive daytime sleepiness (EDS) is not predictive of the presence of OSA in young children, but is a cardinal feature of the OSA syndrome in adults, and thus this symptom should be sought, particularly in adolescents.

It should be noted that the size of the tonsils or adenoids on clinical examination is not linearly correlated with the presence or severity of OSA, so small tonsils should not exclude the diagnosis if other features are present. Other examination features such as obesity, NMD, cranio-facial abnormalities and atopy increase the likelihood of OSA but their absence does not exclude it (see section 2.5 complex high risk children).

Weinstock et al. examined possible characteristics that may increase OSA severity in children (5-9 years) awaiting adenotonsillectomy. This was a cross-sectional screening study using data analyzed from the Childhood Adenotonsillectomy Trial, a randomized, controlled,
multicentre study evaluating adenotonsillectomy versus medical management. Regression analysis assessed the relationship between the AHI and risk factors obtained by direct measurement or questionnaire. Of the 1,244 children undergoing screening PSG, 464 (37%) were eligible (in the AHI range 2-30 or OAI 1-20 and without severe oxygen desaturation) and randomized. Univariate analyses showed significant associations of AHI with race, BMI z-score, environmental tobacco smoke, family income, and referral source. After adjusting for potential confounders, African American race ($p = 0.003$) and environmental tobacco smoke ($p = 0.026$) were each associated with an approximately 20% increase in AHI. After adjusting for these factors, obesity and other factors were not significant. Level of AHI was significantly associated with race and environmental tobacco smoke, highlighting the potential effect of environmental factors, and possibly genetic factors, on paediatric OSAS severity. Efforts to reduce environmental tobacco smoke exposure may help reduce OSAS severity. There should be routine enquiry at assessment for tobacco smoking at the home address (any smoking is more important than whether smoking is indoors or outdoors) and willingness to quit.

\[ \text{Recommendations (See Appendix 1 - Summary investigation and management of suspected OSA in children)} \]

As part of routine health maintenance visits ALL children should be assessed for sleep problems, including snoring and sleep disordered breathing.

If children have snoring, a detailed history of associated symptoms (e.g. habitual snoring, with laboured breathing, observed apnoea, restless sleep & daytime neurobehavioural abnormalities or sleepiness) and a clinical examination should be undertaken to determine whether further evaluation is needed.

It is recommended that clinicians consider combining a validated paediatric sleep questionnaire (for example Appendix 2), physical examination (specifically ENT) and an overnight oximetry as will increase the specificity and positive predictive value for OSAS.

Whatever the tonsillar size children with habitual snoring ± other historical factors suggestive of OSA should be referred to an ENT specialist or a paediatrician for more extensive evaluation.

Children with symptoms of OSA and significant co-morbidity such as morbid obesity, NMD, or craniofacial abnormalities should be referred to a paediatrician for more extensive evaluation.

\[ \text{2.2 Treatment with adenotonsillectomy without polysomnography} \]

Polysomnography remains the gold standard for the diagnosis and assessment of OSA\textsuperscript{3,67,68,70}. However, current resource constraints in New Zealand make it unfeasible to use PSG routinely for the large group of children with suspected OSA. Clinical assessments and questionnaires are poor at differentiating between children with OSA and primary snoring\textsuperscript{3,65-68}. In clinical practice this distinction is less important, as both primary snoring and OSA have similar implications for behaviour, and learning\textsuperscript{11,68}. Adenotonsillar hypertrophy is the most common cause of upper airway obstruction in childhood, and adenotonsillectomy is the treatment of choice\textsuperscript{3,93,94}. If the associated factors mentioned in the previous section are present, consideration of adenotonsillectomy is warranted without sleep studies.

Adenotonsillectomy is a common operation which is generally well-tolerated and safe, often being performed as a day stay procedure, with a low rate of complications. The procedure is effective in treating the majority of children with SDB without co-morbidities\textsuperscript{3,66,68,95,96}. However, children with OSA are at an increased risk of adverse events. While in the past haemorrhage has been the main cause of adverse outcomes, with the increasing performance of adenotonsillectomy for the indication of OSA, respiratory events are now more commonly a cause of adverse outcomes\textsuperscript{67}. 
The risk of perioperative respiratory events for adenotonsillectomy is increased from 1% to approximately 20% in children with OSA and may be as high as 60% in severe OSA. The overall incidence of major neurologic injury or death following adenotonsillectomy is approximately 1 in 27000. The vast majority of events are minor requiring only a period of oxygen requirement or medication. However, medical intervention including supplemental oxygen, CPAP and re-intubation may be required for these complications in children with OSA. The risk of significant events requiring major intervention such as ICU admission is 6-8% in children with OSA but may be as high as 20% if the OSA is severe. Most children with risk factors (see section 2.5) have no adverse events and a significant proportion of children with adverse events have no prior risk factor which makes planning cost-effective perioperative management difficult. However, almost all children experiencing major respiratory events requiring reintubation or ICU admission described in systematic studies had more than one risk factor.

In most cases of OSA in childhood, adenotonsillectomy is a highly effective treatment, leading to resolution of abnormal respiration during sleep, and improvements in growth, restless sleep and daytime behaviour. The majority of studies involving children with either primary snoring or OSA show improvement in aspects of general cognition, memory, attention, executive function, behaviour, quality of life, hyperactivity, and ADHD following treatment with adenotonsillectomy. Other studies report variable improvement in neurocognitive outcomes following adenotonsillectomy (despite demonstrated improvement in respiratory parameters). Improvements are demonstrated in both children with primary snoring and those with OSA confirmed on PSG.

One study has suggested that removal of either the tonsils or the adenoids alone carries a significant risk of persistence or recurrence of OSA, and thus consideration should be given to removing both at the same surgery, unless contraindicated, to reduce the likelihood of recurrence of OSA.

All children undergoing adenotonsillectomy should be assessed for the presence of OSA. The evaluation of children with possible OSA should be directed at assessing the likelihood of OSA and the risk of perioperative events. Whereas the correlation between clinical assessment of OSA and objective measurement with PSG is poor, resource constraints mean that most children will proceed to adenotonsillectomy on the basis of clinical assessment, questionnaire and oximetry. Where PSG is not performed the risk of perioperative events can be assessed on the basis of age, the presence of co-morbidities and oximetry findings (see below). Oximetry should be scored according to the McGill oximetry score (see also Starship Clinical Guidelines for Oximetry - http://www.adhb.govt.nz/StarShipClinicalGuidelines/Oximetry.htm) and also the oxygen nadir should be reported. Children without risk factors and with a normal or "indeterminate" oximetry are very unlikely to experience a clinically significant event.

If a child undergoing adenotonsillectomy for sore throat is incidentally found at the time of anaesthetic to have a history of significant OSA symptoms they should be managed as if they have moderate to severe OSA. Children with known severe OSA should have surgery performed preferentially in the morning rather than the afternoon. Children who are at increased risk should be monitored in an environment with, at minimum, continuous oximetry with alarms. There should be access to airway management equipment if necessary, although this may be via a "code" or resuscitation team. Note that alarms must be clearly audible to staff to be effective. In addition, mortality has occurred in the recovery room, so monitoring should be instituted in theatre and be continuous from the recovery room to the ward. It is likely that the patient is most at risk when unconscious or asleep so a stable clinical condition when awake may be falsely reassuring.
Some children should at minimum be monitored on a paediatric ward overnight, others should be monitored in a facility with direct access to an Intensive Care Unit (ICU). Use of nasopharyngeal airways (NPA) may help prevent the need for post-operative ICU admission. As almost all hospitals do not have easy elective access to an ICU a policy of preventive post-operative ICU admission is generally not efficient or necessary. Children who are the most extreme risk should still have a planned post-operative ICU admission.

**Children with ANY of the following risk factors should be admitted to hospital overnight for monitoring post-adenotonsillectomy:**

1. Age <= 3 years
2. Severe OSA*
   - obstructive events (OAH) or RDI** >= 10 events per hour of sleep on PSG
   - or oxygen saturation nadir < 80% on PSG or overnight oximetry
   - or McGill oximetry score 3 on overnight oximetry (3 or more events < 85%)
   - or elevated carbon dioxide on PSG or blood gas
3. Significant co-morbidities, especially:
   - Down's syndrome
   - Craniofacial anomalies
   - Neurological problems especially hypotonia
   - Cardiac problems or pulmonary hypertension
4. Obesity (weight > 95th Centile)
5. Failure to thrive (weight < 5th centile)
6. Ex-premature infants
7. Current increased work of breathing due to lung problems

*In the absence of oximetry or PSG consider children with a significant history of snoring and apnoea to have severe OSA.*

** The OAHI or number of obstructive apnoea and hypopnoea per hour of sleep is the preferred index. If this is not reported use the Respiratory Disturbance Index (RDI)

**Children in either of the following risk categories should be monitored post-operatively in a hospital with an onsite ICU and should be considered for closer monitoring such as an HDU and/or a post-operative NPA.**

1. Children with 2 or more of the above risk factors
   - e.g. age < 3 years plus a co-morbidity
2. Children with very severe OSA
   - OAH or RDI >= 30 events per hour of sleep on PSG
   - or oxygen saturation nadir < 70% on PSG or overnight oximetry
   - or McGill oximetry score 4 on overnight oximetry (3 or more events < 80%)

**Children with very severe OSA and morbid obesity should have a planned post-operative ICU admission.**

Children may be discharged when there are no concerns with their noise of breathing, respiratory effort or oxygenation during sleep and other criteria for discharge are met.

All children undergoing adenotonsillectomy for OSA should undergo clinical review of symptoms 6-8 weeks post-adenotonsillectomy to determine whether further evaluation and treatment are indicated. SDB persists after surgery in a proportion of children, variably reported as between 13 and 73%, depending on the criteria used and the population studied. The majority of these children had significant improvement in neurocognitive parameters, symptom scores, and respiratory parameters, but not complete resolution. Persistence or post-operative recurrence of SDB is associated with obesity, preoperative severity of SDB, and underlying NMD or craniofacial abnormalities. Children in whom
symptoms persist postoperatively, with severe OSA, obesity or significant co-morbidities (excluding age) should undergo follow-up PSG. The ideal timing for PSG is 2-3 months post-operatively but may vary according resource constraints. Children who have had adenotonsillectomy remain at increased risk of OSA in later life especially children who are obese or who have a family history of OSA 134.

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<tr>
<td>If an otherwise healthy child is determined to have OSAS based on history and examination, adenotonsillectomy remains the first line treatment.</td>
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<tr>
<td>Children with habitual snoring plus other features suggestive of OSA on history or examination, but are otherwise healthy, should be referred for consideration of adenotonsillectomy if the tonsils are moderate or large (grade 2 or more – see Figure 2 for tonsil size grading).</td>
</tr>
<tr>
<td>Children under 3 years of age, those with co-morbid conditions including obesity, and those with more severe OSA* should remain in overnight after adenotonsillectomy, in a unit with personnel skilled in paediatric airway management and with continuous oximetry monitoring.</td>
</tr>
<tr>
<td>Children whose symptoms of OSA persist after adenotonsillectomy, or who do not have enlarged tonsils or adenoids, should be referred to a paediatrician for more extensive evaluation.</td>
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2.3 Referral for sleep studies before adenotonsillectomy

In ideal circumstances OSA would be confirmed by PSG prior to surgery but in New Zealand this test is not widely available. In most cases confirmation of the diagnosis is not critical. However, PSG is indicated:

- when confirmation of the diagnosis is needed;
- when the severity of OSA needs to be assessed in order to weigh against the risks of adenotonsillectomy. This applies if there is significant risk of post-surgical complications (e.g., age < 3 years, medical conditions such as haemophilia or idiopathic thrombocytopenia purpura (ITP)) or anaesthetic complications (including malignant hyperthermia, pulmonary hypertension) 135.

The technical considerations of PSG and other types of sleep study to confirm the diagnosis of SDB, particularly OSA, have been discussed in more detail in Section 1. Appendix 3 and 4 outline the criteria for defining sleep disordered breathing and the diagnostic criteria and PSG based severity in paediatric sleep studies. In brief, possible alternatives to PSG for the diagnosis of OSA prior to adenotonsillectomy include:

- Overnight oximetry. This can be useful if it shows a pattern of cyclic desaturation 136. An example of this pattern is shown in Figure 1. However interpretation of this test involves knowledge of the performance characteristics of the oximeter being used 137 and the limitations of the technology 138, and it may not be useful in children with co-morbidity 139. A normal oximetry should not be used to exclude OSA, as it has a low negative predictive value 140-142. Oximetry guidelines are available on the Starship Children’s Health Guidelines to assist clinicians with the performance, reporting and interpretation of paediatric overnight pulse oximetry studies. (http://www.adhb.govt.nz/StarShipClinicalGuidelines/Oximetry.htm)

- Daytime (nap) PSG has a high PPV, but a low NPV 143, 144.

- Limited information is available to support the use of abbreviated sleep studies in children 144-150. Validation studies in adults are not sufficient, due to the different detection requirements and scoring criteria necessary for interpretation of paediatric studies 151.

Where doubt exists, individual cases should be discussed with a centre with expertise in paediatric sleep medicine.
**Recommendations**

A characteristic abnormal overnight oximetry recording (see Figure 1) in an otherwise healthy child with a clinical history suggestive of OSA is sufficient to confirm the diagnosis of OSA, but a normal oximetry does not exclude OSA.

As abbreviated sleep studies are not well validated in children, the need for formal sleep studies in children with SDB should be discussed with a centre with expertise in paediatric sleep medicine.

Children whom an ENT surgeon or anaesthetist consider to be at high risk should have the diagnosis of OSA made objectively (overnight oximetry ± formal sleep study) before surgery is contemplated.

### 2.4 Treatment for OSA other than adenotonsillectomy

#### 2.4.1 CPAP and newer alternatives

Continuous positive airway pressure (CPAP) is the recommended treatment option in children with specific surgical contraindications, minimal adenotonsillar tissue, persistent OSA after adenotonsillectomy, or OSA due to obesity or craniofacial or NMD. CPAP is effective and well tolerated, and improvements in mask interfaces in recent years have meant CPAP can be successful even in very young children. The exact threshold for the severity of OSA that should be treated with CPAP is not determined for children but in New Zealand an OAI > 5 i.e. moderate OSA is usual (see Appendix 4).

Identifying barriers to adherence (perceived comfort, mask leak, nasal congestion, optimal pressure, parental acceptance/commitment to therapy) is important for individual children and teenagers particularly (ABCQ - Adherence Barriers to CPAP Questionnaire for children). Few studies have analyzed objective adherence to CPAP in children. Adherence rates are often sub-optimal – nightly rates of CPAP use range from 3 - 8 hours/night. The pattern of CPAP use is often established very early in treatment, adherence may be optimised by getting the pressures ‘right’ early. Previous research has described that children are more likely to be adherent if the baseline AHI is high/severe; if there is a greater change in AHI on CPAP; if they are younger (6-12 versus 13-18 years); if a full face is used instead of a nasal mask; when subjects participated in an educational program. In contrast, a recent study by Faroux et al reported that CPAP adherence was not affected by age, type of interface, duration of CPAP or the efficacy of treatment.

Even though 10-30% of children fail CPAP therapy there remains limited evidence for a role in paediatrics for the newer generation of alternative CPAP technologies which include:

- **Auto-CPAP devices** - automated or auto-adjusting CPAP from different manufacturers are commercially available for adults. These different devices may have different algorithms and sensitivities to detect abnormal breathing episodes e.g. some devices use vibration and some use airflow to determine the pressure. Not all devices have comparable ability to detect and correct events in children. While there may be role these cannot be recommended based on current paediatric research evidence;

- **C-Flex technology** – or pressure relief technology is designed to make CPAP more comfortable by reducing pressure at the beginning of exhalation and returning to therapeutic pressure just before inhalation. The level of pressure relief varies based on the patient’s expiratory flow. There is one study by Marcus et al that aims to evaluate the effects of Bi-Flex (similar C-Flex) on adherence and efficacy in children compared to standard CPAP. Fifty six children (2-16 years) with OSA were randomized to CPAP or Bi-Flex. They were evaluated with baseline diagnostic PSG, a titration PSG & then on PAP at 3 months. Both CPAP and Bi-Flex were efficacious in treating OSA and...
there were no significant improvement in adherence (both were sub-optimal) or efficacy using bilevel pressure relief technology compared to CPAP;  

- **Nasal expiratory positive airway pressure (NEPAP) devices** (e.g. Provent) – the NEPAP device acts as a one way valve and creates pressure during expiration only. While there is research on its use in adults, there is only a small pilot study by Marcus et al in children. CPAP candidates (AHI > 5, 8-16 years), underwent PSGs after NEPAP and placebo. Nine responders (subjects with > 50% reduction in AHI from placebo to NEPAP night or AHI < 5 / hour on NEPAP night) eligible for NEPAP for 30 days. Out of the 14 teenagers (age 13.4 ± 1.9 years), BMI z-scores 2.2 ± 1), 3 showed no improvement, 2 worsened, and there was better response with increasing age and less hypercapnia. Eight used NEPAP at home with adherence of 83% and showed improved sleepiness and quality of life. Due to variability in individual responses, efficacy of NEPAP should be evaluated with PSG;  

- **Airvo** - there is a growing interest in use of nasal high flow air as an alternative to CPAP for OSA. There is one case series to date by McGinley et al. Twelve children (age: 10 ± 1 years; BMI: 35 ± 14 kg/m²; AHI ranged from mild to severe (2-36 events/hour), compared CPAP to high flow 20 L/min. The high flow was not titrated but AHI improved from a mean of 11/hour to a mean of 5/hour. On CPAP patients had a mean AHI of 1/hour but CPAP was titrated. Excluding 2 poor responders the effect of high flow was clinically similar to CPAP (1 vs 2/hour). In infants Airvo it has been shown to have less nasal trauma than bubble CPAP, however, there are case reports of pneumothorax/air leak.

Intervention studies are performed to implement and titrate, or confirm the effectiveness of respiratory support (CPAP or NIV). PSG is useful to determine optimal PAP settings in children and infants. While these are ideally undertaken as attended level 1 studies, in some cases split night studies or abbreviated studies may be undertaken to titrate respiratory support. Regular follow-up PSG in children on PAP support is indicated to determine whether pressure requirements have changed as a result of the child’s growth and development, whether symptoms recur while on PAP, or if additional or alternate treatment has been instituted. The frequency of PSG follow up should be based on the child’s growth rate, clinical stability or disease progression or other factors that may precipitate a worsening of sleep disordered breathing (e.g., significant weight gain). It is recommended that children receiving PAP should have at least an annual PSG however younger child may need review more frequently 4-6 monthly.

<table>
<thead>
<tr>
<th>Recommendations</th>
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<tbody>
<tr>
<td>Children with OSAS who do not have adenotonsillar hypertrophy or have symptoms that persist after adenotonsillectomy, or in whom adenotonsillectomy is contraindicated, should be discussed with a centre with expertise in paediatric sleep medicine.</td>
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<tr>
<td>CPAP is an effective alternative treatment for OSA in children.</td>
</tr>
<tr>
<td>Formal sleep studies are indicated for children with OSA treated with CPAP or NIV to titrate the support required and to periodically re-evaluate the appropriateness of settings.</td>
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<tr>
<td>At present there is limited evidence for added benefit from the alternative/new CPAP technologies in children – their role is still under evaluation.</td>
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### 2.4.2 Other treatments

#### 2.4.2.1 Anti-inflammatories

Nasal steroids (fluticasone or budesonide) either alone or in combination, and leukotriene antagonists (Montelukast) have been proposed as possible treatments for OSA particularly in children with allergic rhinitis. A study using oral steroids failed to show a therapeutic effect on OSA. At this stage further research is needed on Montelukast treatment and OSA before a specific recommendation can be made.
Nasal steroids have been shown to improve OSA in the short term (≤ 8 weeks), however the duration of treatment is variable (2-6 weeks), as is the treatment response (mild to moderate at best) and it is uncertain whether the effect is sustained ≥ 8 weeks. Two studies showed shrinkage of adenoids \(^{181,186}\) but studies did not specifically direct therapy to children with atopy or allergic rhinitis so the outcome in these groups is unknown.

It is suggested that nasal steroids be trialled in children with mild residual OSA (defined as AHI < 5/hour) post adenotonsillectomy or when adenotonsillectomy is contraindicated. Intranasal steroids are not recommended as the first line treatment for moderate or severe OSA. It is advised that response to treatment be measured after a 6 week course. Because the long term effect of therapy is unknown, children should be followed to monitor for side effects and change in clinical status. If there are ongoing nasal symptoms post-surgery and significant allergic rhinitis exists there may be a role for desensitisation for some children.

2.4.2.2 Dental therapy and oral appliances

There has been increasing interest in the use of oral appliances (OA) in the specific paediatric circumstances of dental malocclusion and maxillary constriction. In addition to the mandibular advancement splints \(^{187,188}\). Rapid maxillary expansion (RME) has also been proven effective \(^{189,190}\). The combination of surgical treatment and OA treatment has been shown to be effective if the OA treatment is undertaken first \(^{191}\). A systemic review subsequently evaluated 15 articles on the longer term benefit of RME, and found stability of airway dimensions and breathing at an average of 11 months \(^{192}\). Another study \(^{193}\) showed an overall improvement in dental malocclusion following long term follow up of RME in teenagers, but a significant amount of relapse after an initial excellent improvement.

A recent article on the ‘Role of oral health professional in pediatric obstructive sleep apnea’ \(^{194}\), concluded that “the dentist can play an important role in identifying and treating those cases with OA’s, who refuse the surgery, or those with structural abnormality in which myofunctional appliances are beneficial”.

Oral appliances that fit inside the mouth and usually serve to hold the mandible forward have been suggested to treat OSA in children \(^{195,196}\). Whereas there is some evidence suggesting that oral appliances improve OSA in adults there is currently insufficient evidence to recommend this treatment for children.

Mandibular distraction may be an efficacious treatment for OSA in children with craniofacial malformations \(^{197-201}\). Again, further research is needed, but this treatment may be considered in individual cases.

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<th>Recommendations</th>
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<tr>
<td>Nasal steroids may be considered treatment for mild OSAS, particularly in children with allergic rhinitis, or when adenotonsillectomy is contraindicated, with appropriate follow-up to determine the effect of treatment.</td>
</tr>
<tr>
<td>Mandibular distraction or other types of surgery may be considered as treatment for OSA in children with craniofacial disorders, in consultation with a paediatric sleep service and surgical specialists.</td>
</tr>
<tr>
<td>No recommendation can be made regarding the use of oral appliances for children.</td>
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2.5 Complex high-risk children
Obstructive sleep apnoea should be particularly considered in the following conditions due to the high rates of OSA and its consequences.

List of important conditions:
- Down Syndrome
- Mucopolysaccharoidoses
- Spina bifida
- Achondroplasia
- Prader-Willi syndrome (PWS)
- Cerebral Palsy
- Laryngomalacia
- Pierre Robin Sequence and other craniofacial syndromes
- Previous palatal surgery
- Prematurity
- Obesity (see section 2.6)

Children with these disorders potentially have pathophysiology in addition to adenotonsillar hypertrophy that may contribute to OSA, and thus adenotonsillectomy may not lead to complete resolution of the problem, may not be indicated at all, or may even be contraindicated. International guidelines advise PSG prior to adenotonsillectomy for these children in order to make an accurate diagnosis due to increased perioperative risk and decreased accuracy of parental symptom reporting. However, in children with clear symptoms of SDB who have underlying conditions which have a high pre-test probability of OSA, such as Down syndrome or craniofacial syndromes, PSG is unlikely to add to the likelihood of diagnosis. For example, studies have shown that in cases of Down’s syndrome where parents suspect OSA the incidence is close to 100%. Nevertheless, an assessment of perioperative risk is required in order to guide post-operative monitoring. At a minimum, overnight oximetry should be performed and PSG may be required in some circumstances (Reference - RCPCH Working Party on Sleep Physiology and Respiratory Control Disorders in Childhood Standards for Services for Children with Disorders of Sleep Physiology Report September 2009). While the American Academy of Pediatrics guideline suggest that all children with Down syndrome should have PSG even if not snoring, this is not realistic or feasible based on current New Zealand resources.

Children with PWS have been reported to be at risk of sudden death during sleep and this risk appears to be elevated during growth hormone therapy. Limited studies provide insufficient support for the routine use of PSG to predict risk of death or to monitor for development of significant cardiorespiratory abnormalities in this population and additional studies with larger numbers of subjects and longitudinal data are needed. Children with PWS should be assessed at least clinically and with oximetry prior to and following the commencement of growth hormone and as clinically indicated thereafter. PSG should be considered (and in Australia is required) prior to the prescription of human growth hormone for children with PWS and 6-10 weeks post initiation.

As incomplete resolution of OSA is more common in children with a high preoperative AHI, obesity, craniofacial anomalies that obstruct the upper airway, neurologic disorders such as myelomeningocele and Down syndrome, it is recommended that cases are clinically reviewed 6-8 weeks following adenotonsillectomy with consideration to PSG if symptomatic, particularly in children with preoperative evidence for moderate to severe OSAS (i.e. those with abnormal pre-operative oximetry or on PSG).

Children with comorbid conditions at high risk of OSA who are being considered for alternative treatments to adenotonsillectomy (e.g. CPAP, tracheostomy, upper airway
surgery or other treatments) or with evidence of incomplete resolution of OSA post adentonsillectomy may benefit from a PSG prior to intervention to define the aetiology and severity of underlying SDB. The indications for such treatments will vary, but hypoxaemia or hypercapnia during sleep (risk factors for pulmonary hypertension), hypertension, or significant daytime impairments due to sleep-disordered breathing would be the main indications. Where there is significant awake airway obstruction and/or awake blood gas abnormalities then PSG is unnecessary and only likely to delay appropriate intervention.

Children with some of the high risk conditions listed above are also at risk of central sleep apnoea or hypoventilation, which is indistinguishable from OSA either clinically or using overnight oximetry. Therefore a more detailed sleep study may be needed to diagnose the type of SDB (e.g. central vs OSA) and thus guide treatment. Some disorders associated with Arnold-Chiari malformation are associated with severe central apnoea and/or hypoventilation. These children may require screening with oximetry combined with transcutaneous carbon dioxide and/or PSG in the first few months of life as a guide for neurosurgery.

**Recommendations**

<table>
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<tr>
<td>Children with complex medical disorders and suspected OSAS should be discussed</td>
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<td>with a centre with expertise in paediatric sleep medicine for consideration of</td>
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<td>investigation and treatment.</td>
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<tr>
<td>Children with increased risk of central apnoea or hypoventilation should be</td>
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<tr>
<td>referred to a centre with expertise in paediatric sleep medicine for further</td>
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<tr>
<td>evaluation prior to adentonsillectomy or any airway surgery.</td>
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### 2.6 Obesity

#### An increasing problem

Obesity is defined as a BMI >95th percentile for gender and age. Over the last 10 years epidemiological studies have conclusively demonstrated that both the prevalence and severity of overweight and obese children and adolescents has increased worldwide. An increasing proportion of children being referred for evaluation of SDB fulfils the criteria for either overweight or obese. The New Zealand 2012/2013 health survey found 11% of 2-14 year olds to be obese with a further 22% being overweight. This was an increase from 8% in 2006/2007 (The NZ Health Survey 2012/2013, MOH [http://www.health.govt.nz/nz-health-statistics/national-collections-and-surveys/surveys/current-recent-surveys/new-zealand-health-survey](http://www.health.govt.nz/nz-health-statistics/national-collections-and-surveys/surveys/current-recent-surveys/new-zealand-health-survey)). The incidence of obesity was found to be greatest in Maori (19%) and Pacific (27%) children. Therefore as New Zealand children show similar trends for obesity and thus high risk for significant OSA, special consideration is given in the revision of the guidelines to raise awareness and recognition, particularly for Maori and Pacific Island children, so that general practitioners and paediatricians consider and regularly screen for OSA in this high risk group.

#### Relationship to snoring and OSA

Internationally several studies have shown that being overweight and obese is associated with an increased prevalence of snoring. The incidence of habitual snoring in children and adolescents is in the range of 5-7% but in those who are obese the risk is up to two times higher than this. In the only New Zealand survey involving 3 year old children 11% snored and 15% (N=93) were obese. The 2012 AAP technical report summarised a collection of 11 studies looking at the relationship between obesity and SDB and found the risk of snoring increased up to 4 fold when obese. The prevalence of OSA in obese children is estimated to vary between 13-59%.
The distribution of body fat may be more important in predicting the severity of OSA than BMI alone. However, Verhulst have found that waist circumference is associated with an increased risk of central apnoeas in obese children rather than obstructive apnoeas. A case control study by Redline et al in 2-18 year old children found that the risk of actual OSA among obese children was increased 4-5 fold. For every increment in BMI by 1 kg/m² beyond the mean BMI for age and gender, the risk of OSA increased by 12%. In the adult Wisconsin cohort study a 10% weight gain increased the risk of OSA 6 fold. Longitudinal data from the adult Cleveland Family Study showed that an increase in bodyweight over time increased the risk for and accelerated the progression of OSA, suggesting that the incidence of sleep apnoea will rise as the incidence of obesity continues to increase. However, it is uncertain if this relationship holds true for children and young people. It can be concluded that although the incidence of OSA in obese children varies between studies (according to the method of assessment for the SDB e.g. oximetry, PSG or questionnaire), it is a consistent finding that the risk of habitual snoring in obese children is significantly increased.

**Potential mechanisms and complications**

Increased body fat around the upper airway structures and tongue leads to airway narrowing and increased pharyngeal collapsibility. Arens et al showed that obese children have enlarged adenoidal tissues, potentially the result of systemic low-grade inflammatory processes that accompany obesity. Obese children with OSA have also been shown to have a significantly greater adenoidal size compared to obese children with primary snoring and need relatively less adenotonsillar hypertrophy to cause OSA. Increased fat around the viscera, chest wall and abdomen leads to increased respiratory load, decreased lung volumes and functional residual capacity, particularly when supine. Consequently obesity leads to decreased lung volumes and oxygen reserve, and will add to the work of breathing during sleep.

Sleep architecture can be altered in obesity and assessment of sleep hygiene and sleep duration is important in overweight and obese children particularly teenagers when delayed sleep phase disorder may compound sleep symptoms. While some work suggests that society’s trend towards shortened sleep duration and increased irregularity of sleep leads to obesity, the presence of OSA may promote weight gain and obesity via sleep fragmentation and associated daytime sleepiness. Possible mechanisms for this are that poor sleep is associated with decreased leptin (the “satiety” hormone, regulates fat storage and metabolism) & increased ghrelin levels (a hormone associated with drive for higher glycaemic foods) which results in hunger and increased caloric intake with a preference for high glycaemic foods. Spruyt showed that the combination of OSA and obesity in 5-9 year olds, who were 2 times more likely to eat fast food, was positively associated with raised ghrelin levels. Poor sleep may also result in decreased motivation, exercise, and calorie expenditure again compounding the risk of obesity.

Obesity and the associated SDB have been associated with cardiovascular and metabolic complications - elevations in blood pressure, insulin resistance and impaired glucose tolerance, increased inflammatory markers (CRP, white cell count), elevated triglycerides and low HDL, and arterial wall stiffness and endothelial dysfunction. Childhood obesity also confers an increased risk of future obesity and insulin resistance, further weight gain over expected growth, and risk for development of cardiovascular risk factors as young adults. Gozal et al demonstrated an improvement in insulin resistance with adenotonsillectomy in the context of obesity, but not in non-obese subjects, which supports interactions between OSA and obesity. Unfortunately, the potential advantages conferred by treating OSA might be limited by parallel evidence suggesting that the treatment of OSA (e.g. adenotonsillectomy) is associated with weight gain.
Response to treatments and considerations

Although obesity appears to be an independent risk factor for OSA it is important to note that adenotonsillar hypertrophy remains a significant risk factor in these children. Evidence suggests that obese children with tonsils that occupy more than 50% of the pharyngeal diameter have a high prevalence of OSA. Adenotonsillectomy can significantly reduce the AHI in this high risk group and remains the first line treatment. However, Costa et al in a metanalysis found that adenotonsillectomy did not resolve OSA in the majority of obese children (postoperatively only 49% had an AHI<5, 25% <2, and 12% <1). As factors other than adenotonsillar hypertrophy may contribute to OSA in obese children, these children are at increased risk of residual OSA following adenotonsillectomy. Bhattacharjee found the highest risk of residual disease was in obese children >7 years. Mitchell found that post adenotonsillectomy the respiratory disturbance index improved and also quality of life, but there was no resultant change in BMI, and OSA did not actually resolve in the majority of children.

Obesity also places a child at higher risk of perioperative complications such as laryngospasm and increased risk of hypoventilation and post-operative respiratory compromise. It is recommended that this high risk group be observed closely overnight in HDU or PICU after adenotonsillectomy with judicial use of oxygen and narcotics. Six to eight weeks following treatment, clinical reassessment is recommended to determine if symptoms of OSA have resolved. Children with symptoms that persist following adenotonsillectomy should be considered for formal PSG and if moderate OSA persists CPAP is indicated.

There is evidence that weight loss and lifestyle interventions can improve symptoms of SDB but results are variable and studies in obese children are sparse. Regular exercise and a weight management programme is recommended as an essential part of the treatment plan.

As there is a significant relapse rate, and compliance with CPAP can be poor, bariatric surgery may be considered for adolescents. However, adolescents being considered for bariatric surgery in New Zealand must meet stringent criteria: BMI > 40 with comorbidities (e.g. OSA, diabetes); obese for > 5 years; failed non-surgical attempts at weight loss; understanding of and motivated for surgery; accepting of and compliant with long term follow up (e.g. controlled co-morbidities); be of acceptable operative risk; motivated; have reasonable expectations; able to establish regular exercise; able to meet specific weight loss goal and importantly have decisional capacity to be able to provide informed consent. The procedure itself is not without complications - usually sleeve gastrectomy or roux-en-Y gastric bypass - major complications requiring repeat laparotomy ~7% and minor issues 3% (Surgeons CMDHB personnel communication). While short term studies in adolescents are promising in terms of improved weight, blood pressure, quality of life and improved OSA, longer term studies in adults demonstrate persistent OSA and relapse.

Like obesity, OSA nowadays is viewed by some as a low grade systemic inflammatory disease and is considered a risk factor for the development of metabolic syndrome. Redline et al found in adolescents that those with SDB had approximately a 6 fold risk of metabolic syndrome and in that study 70% of those with SDB (AHI>5) were overweight. Further background evidence to the link between obesity and insulin resistance, dyslipidemia and other metabolic abnormalities are reviewed in the AAP technical document. The summary recommendation is that screening of obese and overweight children for markers of metabolic syndrome should be considered as part of their management of SDB.
### Recommendations

All overweight and obese children and adolescents should be routinely assessed for sleep problems, including sleep hygiene, sleep duration, snoring and other symptoms of SDB. A sleep questionnaire ± overnight oximetry (if available) may be considered as first line. While overweight and obese children are at increased risk of residual OSA, adenotonsillectomy remains the recommended first line management. Overweight and obese children and adolescents are at increased anaesthetic and perioperative risk. A careful preoperative assessment is recommended. Due to the risk of hypoventilation, oxygen and narcotics should be used judiciously. Consideration should be given to performing surgery only where specialised paediatric anaesthetic staff and paediatric HDU/ICU facilities are available. Clinical review is recommended 6-8 weeks post adenotonsillectomy and if symptoms persist a PSG is indicated with consideration to ongoing pressure support until weight loss can be achieved. A weight management plan in addition to other treatments is essential for children and adolescents who have OSA and are overweight or obese. It is recommended that health care professionals be aware of the risk of cardiovascular complications and metabolic syndrome in any overweight or obese child or adolescent who presents with snoring. Investigation of these complications should be considered including a blood pressure to look for hypertension (>95<sup>th</sup> percentile for gender, age, and height on 3 separate occasions) and blood tests for metabolic syndrome. Further investigation and appropriate referral may be required.

<table>
<thead>
<tr>
<th>2.7 Neuromuscular disease</th>
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<tr>
<td>Children with NMD are at risk of central apnoea, obstructive apnoea, and hypoventilation during sleep owing to the reduction in respiratory drive, respiratory muscle activity (including the upper airway), and ventilation that occurs during normal sleep. Non-invasive ventilation can be used successfully in young children, and there is increasing evidence that it can reverse respiratory insufficiency due to NMD, improve quality of life, and reduce hospitalisations&lt;sup&gt;56,61&lt;/sup&gt;. Non-invasive ventilation probably improves survival in NMD&lt;sup&gt;284,354-357&lt;/sup&gt;. The literature would suggest that NIV is frequently started for the first time for respiratory failure due to acute pneumonia&lt;sup&gt;57&lt;/sup&gt;. The possibility of such support should be raised with families before such an acute situation arises, such as during routine outpatient visits. This may allow enough time for evaluation and consideration of NIV in an elective situation. While long-term invasive tracheal ventilation is becoming more common internationally, this practice is not supported in New Zealand for progressive NMD. Due to the burden of care of NIV, discussion should be held at an early stage about what level of respiratory support is available so that family expectations can be managed and a more palliative approach planned if appropriate. Sleep-disordered breathing and other respiratory complications may be predicted by awake respiratory function testing. Respiratory assessment needs to be conducted regularly to allow for timely intervention. All children, when old enough, should undergo annual lung function testing which needs to be increased to 6-monthly in those judged high risk for respiratory complications. Standard routine lung function assessment should include spirometry, peak cough flow and spot-check measurement of oxygen saturation. Measurement of maximal mouth pressures is an alternative if peak cough flow measurement is not available but both tests are not required. While supine spirometry may be 20% lower than sitting spirometry, addition of supine testing is unlikely to change management when routine overnight testing is available.</td>
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Daytime pulmonary function and awake blood gases can serve as guides for children most likely to have SDB. Children with NMD should also be screened for symptoms of nocturnal hypoventilation\textsuperscript{358,359}.

The following children should undergo regular annual sleep studies (PSG ± overnight oximetry and capnography if PSG is unavailable) ± institution of treatment if NIV is being considered:

1. Children with recurrent admissions with lower respiratory tract infection. These children may have reduced ventilation during acute illness and may require NIV even with normal PSG\textsuperscript{54,55}.
2. Children with symptoms of nocturnal hypoventilation: morning headaches, EDS, unrefreshing sleep, failure to thrive or developmental delay disproportionate to their underlying disease.
3. Children with forced vital capacity (FVC) < 50% predicted or a daytime capillary carbon dioxide above 45 mmHg (6.0kPa), especially if the base excess is ≥4mmol/L\textsuperscript{360-362}.

Just as crucial as the measurement of SDB or respiratory failure is the need to assess and assist the child’s ability to clear airway secretions and effectiveness of cough. Interventions to improve airway clearance can prevent hospitalisation and reduce the incidence of pneumonia\textsuperscript{363-365}. Lung volume recruitment and airway clearance assistance strategies should be employed before and alongside NIV. Cough peak flow (CPF) should be routinely measured in all children > 12 years\textsuperscript{359,366}. A CPF <270 L/min in adults or adolescents (or the equivalent in younger children) indicates the need for routine assistive airway secretion clearance\textsuperscript{363}. Manual cough-assist and breath-stacking are effective and should be employed before more advanced techniques such as mechanical insufflation/exsufflation ("cough-assist") devices. A CPF <160 L/min indicates ineffective cough-clearance and suggests more advanced assistance such as a cough-assist device may be required\textsuperscript{367}.

During respiratory infections children who are normally above these CPF thresholds may fall below them when unwell. For this reason, all hospitals that admit children with NMD should have cough-assist or similar devices and trained staff available for children admitted with pneumonia and/or atelectasis\textsuperscript{366}. Note that patients with VC < 50% or peak cough flow < 270 L/min should also have a capillary blood gas measured when admitted acutely unwell. Suction machines and/or bag-mask devices may be needed at home or in hospital in case of mobilisation of large mucus plugs which may obstruct the airway. An oximeter in the home may help caregivers identify the need to intensify airway secretion clearance or the need for hospital admission.

Children with NMD are also at risk of bulbar weakness causing swallowing difficulties and aspiration. A feeding history should be taken annually and referral to a speech language therapist made and/or videofluoroscopy swallow study performed if appropriate\textsuperscript{359,366}.
Recommendations

Lung function (spirometry, CPF and lung volumes if able) should be monitored at least on an annual basis in children with NMD, with the aim of anticipating the onset of SDB, and the risk of respiratory failure during respiratory illnesses.

Annual PSG ± overnight oximetry and capnography should be considered in children with a forced vital capacity < 50% predicted, a daytime capillary pCO₂ above 45 mmHg (6.0kPa), symptoms of nocturnal hypoventilation or recurrent admissions with respiratory tract infections.

Children who have difficulty clearing airway secretions, have an ineffective cough or low peak cough flow (< 160 L/min) should be managed with assistive airway clearance techniques.

All hospitals that admit children with NMD should have trained staff (e.g. physiotherapists) and equipment (e.g. NIV) available for managing children whose respiratory function deteriorates with acute respiratory infections.

NIV should only be initiated after discussion with parents and patients about long-term prognosis.

NIV during sleep is indicated for children with chronic respiratory failure (daytime pCO₂ > 6.7 kPa/50 mmHg) ± symptomatic nocturnal hypoventilation. NIV when awake in the daytime may be initiated for persistent hypercapnia or low baseline SaO₂ (<92%).

2.8 Disorders of breathing in infants

2.8.1 Apparent life-threatening events (ALTE)

A detailed discussion about the investigation of an infant who presents after an apparent life-threatening event (ALTE) is beyond the scope of this document. A careful and considered history and examination should establish a cause in the majority of cases. Polysomnography is not indicated for routine evaluation in infants with ALTE. If OSA is clinically suspected, or if bradycardia is demonstrated on cardiac monitoring in the absence of central apnoea, or if an underlying disorder of control of breathing (e.g. central alveolar hypoventilation syndrome) is suspected, a referral should be discussed with a centre with expertise in paediatric sleep medicine. There is no evidence that home respiratory-monitoring prevents sudden infant death. Home apnoea monitoring should not therefore be recommended after discharge in the absence of evidence on history that recurrent apnoea with or without oxygen desaturation was the primary reason for presentation.

2.8.2 Chronic Neonatal Lung Disease, Bronchopulmonary Dysplasia & Home Oxygen

Prematurity has already been highlighted as a group at high risk for OSA but until recently the clinical characteristics of OSA in very young children (<2 years particularly premature infants) was not well defined. The Australian paper by Raynes-Greenow et al is perhaps the largest cohort study (N=4145 (1.0% population) - mean age 44.2 mth (SD 13.9) to date using coded record linked population health data to examine the relationship between gestational age and weight for gestational age and sleep apnoea up to 6 years of age. Adenoidectomy, tonsillectomy or both were common (85.6%). The results suggested that children born <32 weeks compared to term were over 2.7 times (OR 95% CI 2.16, 3.49) more likely to be diagnosed with sleep apnoea, and that the diagnosis of SDB is more prevalent in children born preterm, but not those who are small. Two other papers published since the 2004, while small sample sizes, are worthy of mention as they emphasise that a high level of clinical awareness is required to identify OSA in the formerly preterm infant.

Infants with chronic neonatal lung disease on home oxygen should have continuous overnight recording of oximetry for at least 6-8 hours on a regular basis to guide reduction of oxygen prescription. PSG is not required to titrate supplemental oxygen for chronic lung disease. Guidelines for weaning oxygen are available from regional level III neonatal units.
The British Thoracic Society Guidelines for home oxygen in children published in 2009 also provide guidance\textsuperscript{374}. This is a comprehensive formal guideline. Oximeters used for overnight monitoring in infants and children should have high movement resistance (ability to detect the true oxygen saturation during motion) and a low averaging time (2-3 seconds) in order to detect brief desaturation events.

Level 1 PSG should be considered in this group if OSA is suspected clinically, if there are clusters of desaturation on overnight oximetry (suggesting more than just CNLD), or an underlying disorder of control of breathing (e.g. central alveolar hypoventilation syndrome) is suspected\textsuperscript{368}.

**Recommendations**

| It is recommended that infants who present with an ALTE and have symptoms of SDB or infants with chronic lung disease when an underlying disorder of breathing is clinically suspected (e.g. OSA or central alveolar hypoventilation syndrome) should be discussed with a centre with expertise in paediatric sleep medicine. |
| Oximeters used for overnight monitoring in infants should have high movement resistance (ability to detect the true oxygen saturation during motion) and a low averaging time (2-3 seconds) in order to detect brief oxygen desaturation events. |
| Decisions about oxygen prescription for infants on home oxygen can be guided by overnight recordings of oxygen saturation, usually without the need for more detailed studies of breathing during sleep. |

**2.9 Cystic Fibrosis, Bronchiectasis, Other Chronic Lung Diseases**

Children with underlying lung disease (e.g., particularly cystic fibrosis (CF), bronchiectasis including primary ciliary dyskinesia (PCD), and asthma) may be at increased risk of developing sleep-disordered breathing.

Patients with low FRC have reduced functional reserve and may become hypoxic during sleep, particularly in REM sleep when accessory respiratory muscle hypotonia occurs\textsuperscript{375}. Other factors that may exacerbate SDB in children with chronic lung disease include increased bronchoconstriction during sleep, reduced mucociliary clearing or cough and increased arousals or sleep fragmentation\textsuperscript{375}.

**2.9.1 Asthma**

Studies have found persistent wheezing in children to be a risk factor for SDB\textsuperscript{300}. Ross et al\textsuperscript{376} prospectively followed 108 children (aged 4-18 years) over 1 year in a specialist asthma clinic. Asthma severity was evaluated by assessment of a combination of symptom burden, controller therapy and exacerbations. The study findings are weakened by the fact that SDB was assessed by history of habitual snoring and overnight oximetry alone, and there was no pulmonary function testing as 25% were too young. However, after adjustment for race, sex and obesity the OR of severe asthma with SDB was 3.6 (1.3-10.4, 95% CI). This study supports current guidelines recommending consideration of SDB as a contributing factor in poorly controlled asthma and identifies SDB as an independent predictor of asthma severity.

Brockmann et al\textsuperscript{377} recently published a systematic review to examine the association between asthma and SDB in children <18 years of age. Seventeen studies were selected, which included 155 children (mean age 8.6 ± 2.5 years). All included studies defined asthma and SDB based on questionnaires, and only two performed a sleep study for diagnosing OSA. SDB was significantly more frequent in children with asthma compared with non-asthmatics (23.9% vs 16.7% respectively, p < 0.0001). Children with asthma had a significantly higher risk for SDB (OR 1.9 [1.7; 2.2]).
In summary asthma seems to be a significant risk factor for developing SDB. However, only a minority of the studies based the diagnosis of SDB on PSG. It is recommended that overnight oximetry and PSG are rarely indicated in children with asthma, unless there is clinical suspicion of OSA, morning headaches or cor pulmonale.  

2.9.2 Non-cystic fibrosis (CF) Bronchiectasis and Primary Ciliary Dyskinesia (PCD)

While bronchiectasis is prevalent in New Zealand particularly in Maori and Pacific Island children, there is no local research evaluating SDB in this population. The only study to date is from Turkey, where a high prevalence of non-CF bronchiectasis has also been reported. This study evaluated the effect of respiratory symptoms at night and disease severity on subjective sleep quality in children with non-CF bronchiectasis. Twenty-two percent of the children with non-CF bronchiectasis had SDB in comparison with 9% of controls (p = 0.003). Bronchiectasis patients who snored had poorer sleep quality (p < 0.001) and patients with wheezing had a significantly higher rate of snoring (p = 0.04). Children with worse HRCT scores also had worse sleep quality (r = 0.28, p = 0.04). This study suggests that children with non-CF bronchiectasis should be asked about sleep problems, especially snoring at outpatient reviews especially if they have multilobar/ widespread disease.

The same centre in Turkey also evaluated SDB in 29 children with PCD, and whether this was related to upper respiratory system manifestations and severity of the underlying lung disease. All patients completed a sleep questionnaire and underwent an overnight PSG (level 1). Out of the children upper airway symptoms were notable - n=8 allergic rhinitis; n=6 nasal septal deviation; n=2 polyps; n=6 tonsils >= size 2 deviation. The number of children with HRCT chest scan evidence of bronchiectasis was not stated. Sixty-five percent of PCD patients had habitual snoring. Fifty-two percent of the PCD patients had OSA on PSG. Habitual snoring and OSA were more common in PCD patients who had cigarette smoke exposure in their homes (p<0.001 and p=0.02, respectively). This study supports that children with PCD have a higher rate of SDB compared to controls and a higher rate of OSA compared to population rates. Cigarette smoke exposure is an important risk factor for OSA in PCD patients.

Finally Santamaria et al evaluated SDB in 16 stable PCD patients (aged 4 – 17 years) in comparison with 42 controls by undertaking an overnight PSG (level 3 device, no EEG). Half of the subjects (n = 8) had HRCT evidence of bronchiectasis, all had chronic rhinosinusitis and 50% adenoidal hypertrophy. Subjects with PCD had more respiratory events and higher oxygen desaturation indexes than controls. As nocturnal desaturation is associated with lung function and structure abnormalities, SDB may significantly contribute to pulmonary morbidity. This study in children with PCD ± bronchiectasis supports that, in addition to clinical history, oximetry may be a useful screening tool.

2.9.3 Cystic fibrosis (CF)

A recent case-control study by Spicuzza et al has documented the occurrence of OSA in children with CF with normal or mildly impaired lung function. Forty children (aged 0.5-11 years) with CF and 18 controls were assessed with PSG (level 1). The AHI was significantly higher in the CF group compared with controls (mean [SE] 7.3 [1.3] vs 0.5 [0.4], respectively, p < 0.001), particularly in preschool-aged children and in children with upper airway abnormalities. Seventy percent (n=28) of the children with CF had mild to moderate OSA. Children with CF compared to controls also had reduced sleep efficiency, REM sleep duration and more fragmented sleep based on increased number of arousals per hour. This study supports that children with CF regardless of severity of lung disease should be asked about sleep problems, especially snoring at outpatient reviews.

Progressive deterioration of lung function in CF patients may lead to significant hypoxaemia and hypercapnia, especially during sleep. Two studies suggest that simple gas exchange studies may be useful in CF to monitor hypoxaemia and hypercapnia. Sub-
clinical pulmonary hypertension develops in a significant proportion of patients with CF and is strongly correlated with hypoxaemia, independent of pulmonary function\textsuperscript{390}.

Daytime measurements of lung function have poor specificity for nocturnal hypoventilation in CF\textsuperscript{383,386,391}. A continuous overnight oximetry recording to identify children who might benefit from nocturnal supplemental oxygen has been suggested for those with: a resting oxygen saturations awake in room air of <95\%\textsuperscript{388} or <93\%\textsuperscript{386}, or a FEV\textsubscript{1}<65\% predicted\textsuperscript{386}, during a clinically stable period.

Since the 2004 guidelines were completed two Cochrane reviews have been published evaluating oxygen therapy for CF\textsuperscript{392} and NIV in CF\textsuperscript{393}. Oxygen therapy at home may be considered when the oxygen saturation is below 90\% for more than 10\% of the night, in a clinically stable period\textsuperscript{394}. All the studies in the Cochrane review on oxygen therapy compared low-flow oxygen to room air. Ten studies were short-term. Four studies looked at supplemental oxygen at night. There was more regular attendance at school in those receiving long-term oxygen. There is little evidence to support or oppose the long-term use of oxygen therapy in those with advanced CF lung disease. Supplemental oxygen therapy may lead to an increase in nocturnal carbon dioxide\textsuperscript{395}, and thus undertaking capnography in conjunction with oximetry is recommended if oxygen therapy is to be considered.

Finally studies have shown that in moderate to severe CF lung disease when NIV is used in addition to oxygen, this may improve gas exchange during sleep to a greater extent than oxygen therapy alone\textsuperscript{387,395-397}. These benefits of NIV have largely been demonstrated in single treatment sessions with small numbers of participants. The impact of this therapy on pulmonary exacerbations and disease progression remains unclear. In CF patients in hypercapnic respiratory failure, NIV may improve sleep quality and health status which may be advantageous for those awaiting transplant\textsuperscript{398}. In summary however, while NIV may be a useful adjunct to other airway clearance techniques for some patients with severe disease, to date there is no definitive evidence regarding the efficacy, safety and acceptability of NIV in CF\textsuperscript{393}.

### Recommendations

<table>
<thead>
<tr>
<th>Recommendations</th>
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<tbody>
<tr>
<td>All children with chronic lung diseases (e.g. asthma, CF and non-CF bronchiectasis) should be asked about environmental tobacco smoke exposure and sleep problems, especially snoring, at outpatient reviews particularly if they have extensive or severe disease.</td>
</tr>
<tr>
<td>Overnight oximetry ± capnography should be considered at least annually during a clinically stable period in children with CF and non-CF bronchiectasis who have an FEV\textsubscript{1} &lt; 65% predicted.</td>
</tr>
<tr>
<td>Supplemental oxygen should be considered for children with CF and non-CF bronchiectasis when overnight oximetry demonstrates a baseline oxygen saturation ≤ 93% and more than 10% of the night spent with oxygen saturations &lt; 90%.</td>
</tr>
<tr>
<td>Children with CF and non-CF bronchiectasis who have symptoms of SDB or symptoms of sleep-related hypoventilation should be referred to centres with expertise in paediatric sleep medicine for consideration of PSG if non-invasive ventilation is being considered.</td>
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</tbody>
</table>
FIGURE 1. Example of an abnormal overnight oximetry consistent with OSA

A section of an overnight a) oxygen saturation and b) heart rate recording demonstrating clusters of desaturation with ≥3 dips in oxygen saturation below 90%. This pattern has a high positive predictive value for OSA in an otherwise normal child with a history suggestive of OSA\textsuperscript{136}.
FIGURE 2. Guide to scoring tonsillar size
(Reference - http://sleepmedicineboardreview.wordpress.com)
- Grade 0: Tonsils absent
- Grade 1: hidden behind tonsillar pillars
- Grade 2: Extend to pillars
- Grade 3: Visible beyond pillars
- Grade 4: Enlarged to midline

0
Surgically removed tonsils

1
Tonsils hidden within tonsil pillars

2
Tonsils extending to the pillars

3
Tonsils are beyond the pillars

4
Tonsils extend to midline
Guideline team

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Declaration of Competing Interests

All working party members provide sleep services for children but, with the exception of Professor Dawn Elder and Dr Alex Bartle, no members derive income from sleep studies for children. No other declarations of competing interest have been made by members, and thus the guidelines reflect the unbiased views of the guideline team.
**Consultation**

As part of the Peer Review Process the following organisations and individuals were invited to comment during the consultation phase:

<table>
<thead>
<tr>
<th>Members</th>
<th>Paediatric Society of New Zealand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jeff Brown</td>
<td>Paediatric and Child Health Division of RACP</td>
</tr>
<tr>
<td>Gillian Nixon</td>
<td>Chair 2004 Guidelines Group</td>
</tr>
<tr>
<td>Richard Aickin</td>
<td>Chair Clinical Network Group, PSNZ</td>
</tr>
<tr>
<td>Pat Tuohy</td>
<td>Chief Child Health Advisor, MoH</td>
</tr>
<tr>
<td>Katie Burgess, Secretary</td>
<td>Australasian Sleep Technologists Association</td>
</tr>
<tr>
<td>Dr Mike Hlavac, President</td>
<td>New Zealand Branch Australasian Sleep Association</td>
</tr>
<tr>
<td>Secretary</td>
<td>NZ Society of Otolaryngology &amp; Head &amp; Neck Surgery</td>
</tr>
<tr>
<td>Secretary</td>
<td>Australasian Society of Paediatric Otorhinolaryngology</td>
</tr>
<tr>
<td>Chief Executive Officer</td>
<td>Royal NZ College of General Practitioners</td>
</tr>
<tr>
<td>Chairperson</td>
<td>NZ Rural General Practice Network</td>
</tr>
<tr>
<td>Bernadette Drummond</td>
<td>Paediatric Dentistry Association</td>
</tr>
<tr>
<td>Secretary</td>
<td>College of Anaesthetists</td>
</tr>
</tbody>
</table>
APPENDIX 1. Summary investigation and management of suspected OSA in children

During health visit assess child for sleep problems including snoring

If symptoms suggestive of OSA, examine ENT ± complete structured paediatric OSA screening questionnaire (see example APPENDIX 2)

Yes

Co-morbidities e.g.
- Morbid obesity
- Spina bifida
- Down syndrome
- Neuromuscular disease
- Craniofacial abnormalities

No

Previous T & A/no tonsils
OR
High anaesthetic or surgical risk

Previous T & A/no tonsils ± adnomids
- Diagnostic doubt
- Parent refuses surgery

Refer to ENT
Considerations include:
- T & A (overnight stay ± NPA for high risk)
- ± Other treatments (see Section 2.4.2)
- ± Other surgery

Review to check symptoms resolved at 6-8 weeks post intervention

Yes

During future health visits if symptoms relapse re-refer

No

Full clinical assessment & review of all contributory factors required

Refer to Paediatrician
Considerations include:
- T & A ± other surgery
- Overnight oximetry (if available)
- ± Other treatments (see Section 2.4.2)
OR
Referral to Specialist Paediatric Sleep Medicine Service
Considerations include:
- As above
- Sleep study
- Respiratory support e.g. CPAP
APPENDIX 2. Example of a structured paediatric OSA screening questionnaire

<table>
<thead>
<tr>
<th>Surname</th>
<th>NHI</th>
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</thead>
<tbody>
<tr>
<td>First Name</td>
<td></td>
</tr>
<tr>
<td>DOB</td>
<td>SEX</td>
</tr>
</tbody>
</table>

Please answer on behalf of your child for the past month. If you don’t know, circle “?”

<table>
<thead>
<tr>
<th>Question</th>
<th>Yes</th>
<th>No</th>
<th>?</th>
</tr>
</thead>
<tbody>
<tr>
<td>A constant blocked or runny nose?</td>
<td></td>
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<td></td>
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<tr>
<td>Recurrent chest infections/pneumonia?</td>
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<tr>
<td>An airway narrowing or abnormality?</td>
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<tr>
<td>A syndrome or other major health problem?</td>
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<tr>
<td>Type</td>
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<tr>
<td>Does your child have large tonsils?</td>
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<td></td>
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<tr>
<td>Has your child had their adenoids or tonsils removed?</td>
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</table>

While sleeping, does your child . . .

1. Snore more than half the time?                                         Yes / No / ?
2. Always snore?                                                          Yes / No / ?
3. Snore loudly?                                                          Yes / No / ?
4. Have trouble breathing, or struggle to breathe?                       Yes / No / ?
5. Have “heavy” or loud breathing?                                        Yes / No / ?
6. Have you ever seen your child stop breathing during the night?         Yes / No / ?

Does your child . . .

7. Tend to breathe through the mouth during the day?                      Yes / No / ?
8. Have a dry mouth on waking up in the morning?                          Yes / No / ?
9. Occasionally wet the bed?                                               Yes / No / ?
10. Wake up feeling unrefreshed in the morning?                           Yes / No / ?
11. Have a problem with sleepiness during the day?                        Yes / No / ?
12. Has a teacher commented that your child appears sleepy during the day? Yes / No / ?
13. Is it hard to wake your child up in the morning?                       Yes / No / ?
14. Does your child wake up with headaches in the morning?                Yes / No / ?
15. Did your child stop growing at a normal rate at any time since birth? Yes / No / ?
16. Is your child overweight?                                             Yes / No / ?

My child . . .

17. Does not seem to listen when spoken to directly.                      Yes / No / ?
18. Has difficulty organizing task and activities.                       Yes / No / ?
19. Is easily distracted by extraneous stimuli.                          Yes / No / ?
20. Fidgets with hands or feet or squirms in seat.                        Yes / No / ?
21. Is ‘on the go’ or often acts as if ‘driven by a motor’.              Yes / No / ?
22. Interrupts or intrudes on others (e.g. butts into conversations or games). Yes / No / ?

Scoring: Calculate the average score for non-missing items (Yes=1; No=0; Don’t know=missing). An average score of 0.33 (or > 7 “Yes” if no missing items) has a 78% sensitivity and a 72% specificity for detecting PSG diagnosed OSA (AHI >5).
APPENDIX 3. Overview of criteria for defining sleep disordered breathing in paediatric sleep studies

The AASM 2014 document\textsuperscript{399} gives detailed guidelines on the methodology and definitions of standardised event and scoring of paediatric sleep studies. Details of the technical aspects of setting up sleep studies and subsequent scoring is beyond the scope of this document. What follows is a summary of the criteria for respiratory events and thresholds for defining severity of SDB in sleep studies. For more details the reader is referred to the AASM manual for the scoring of sleep and associated events 2.1 2014.\textsuperscript{399,400}

Score a respiratory event as an \textbf{obstructive apnoea} if it meets \textbf{ALL} the following criteria:
1. The event lasts for $\geq 2$ breaths
2. The event is associated with a $\geq 90\%$ drop in the signal amplitude of the nasal pressure, PAP device flow (titration study) or alternative signal (e.g. thermistor) from pre-event baseline for $\geq 90\%$ of the entire respiratory event
3. The event is associated with continued or increased respiratory effort throughout the entire period of decreased airflow
4. The duration of the apnoea is measured from the end of the last normal breath to the beginning of the first breath that achieves the pre-event baseline inspiratory excursion.

Score a respiratory event as a \textbf{mixed apnoea} if it meets both 1. and 2. above and is associated with absent respiratory effort in the initial portion of the event followed by resumption of respiratory effort before the end of the event.

Score a respiratory event as a \textbf{central apnoea} if it is associated with absent respiratory effort throughout the entire duration of the event and one of the following is met:
1. The event lasts $\geq 20$ seconds
2. The event lasts $\geq 2$ breaths and is associated with an arousal or a $\geq 3\%$ oxygen desaturation.

Scoring hypopneas as central or obstructive events is optional. Score a respiratory event as a \textbf{hypopnoea} if it meets \textbf{ALL} of the following criteria:
1. The event is associated with a $\geq 30\%$ fall in the amplitude of the nasal pressure, PAP device flow (titration study) or alternative signal (e.g. thermistor) compared to the pre-event baseline excursion
2. The duration of the $\geq 30\%$ drop in signal excursion lasts $\geq 2$ breaths
3. There is an associated $\geq 3\%$ oxygen desaturation from pre-event baseline or the event is associated with an arousal.

If electing to score \textbf{obstructive hypopneas}, score a hypopnea as obstructive if \textbf{ANY} of the following criteria are met:
1. Snoring during the event
2. Increased inspiratory flattening of the nasal pressure or PAP device flow signal compared to baseline breathing
3. Associated thoracoabdominal paradox occurs during the event but not during pre-event breathing.

If electing to score \textbf{central hypopneas}, score a hypopnea as central if \textbf{NONE} of the following criteria are met:
1. Snoring during the event
2. Increased inspiratory flattening of the nasal pressure or PAP device flow signal compared to baseline breathing
3. Associated thoracoabdominal paradox occurs during the event but not during pre-event breathing.
Scoring a **respiratory effort related arousal (RERA)** event is optional. If electing to score RERAs, score a RERA if there is a sequence of breaths lasting ≥ 2 breaths (or the duration of two breaths during baseline breathing) when the breathing sequence is characterized by increasing respiratory effort, flattening of the inspiratory portion of the nasal pressure (diagnostic study) or PAP device flow (titration study) waveform, snoring, or an elevation in the end-tidal PCO2 leading to arousal from sleep when the sequence of breaths does not meet criteria for an apnea or hypopnea.
APPENDIX 4. Diagnostic criteria and PSG based severity in paediatric sleep studies
The following aims to provide background on how obstructive and central sleep events and hypoventilation are classified according to severity and reported in paediatric sleep studies.

Obstructive Sleep Apnoea

Definition:
The OAHI has been used in clinical trials and epidemiological studies to classify patients as either having OSA or primary snoring as well as to classify the severity of OSA.

Primary snoring is characterised by habitual snoring, but with few obstructive respiratory events (OAHI <1.2 respiratory event/hour), oxygen desaturation or formally defined respiratory arousals\textsuperscript{12,404}. Essentially primary snoring is a child who snores but does not reach the threshold for a diagnosis of OSAS.

Obstructive Sleep Apnoea Syndrome is defined by an OAHI >1.2/hr \textsuperscript{401}.

Severity assignment:
Optimal severity classification remains unclear and may vary according to the outcome measure used. In general OAHI is utilised to broadly group severity though final categorisation may depend on modifiers as below. If in doubt it is usually more helpful to describe / summarise the findings rather than make an arbitrary severity classification.

<table>
<thead>
<tr>
<th>PSG severity (OAHI)</th>
<th>Normal</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Extreme</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1.2/hr</td>
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<td>≥1.2-&lt;5/hr</td>
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<td>≥5-&lt;10/hr</td>
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<td>≥10-30/hr</td>
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<td>≥30</td>
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Potential ‘modifiers’ from PSG:
- Magnitude and nadir of obstructive event desaturations
- Degree of obstructive work of breathing
- Presence of obstruction related hypoventilation (hypercarbia)
- Impact on sleep quality (overall architecture, fragmentation, total arousal index, respiratory arousal index)

Both the utility and definition of an upper limit for the arousal index remains uncertain. Beyond infancy, an upper limit of 14/hr TST for total arousals and/or 1/hr TST for respiratory arousals may be seen as abnormal\textsuperscript{402,403,404}.

Additional ‘modifiers’:
- Due to the limitations of PSG, severity classification may sometimes be modified based on presence or lack of daytime impact (e.g. extreme somnolence) though, if doing so, this ought to be made clear.

Central Sleep Apnoea Syndrome

Diagnostic test:
Age < 2 years -
- PSG – Central Apnoea Index > 10 / hr

Age 2-15 years -
- PSG – Central Apnoea Index > 5 / hr

Severity assignment:
Severity assigned on basis of highest index

<table>
<thead>
<tr>
<th>CAI &lt; 2 years</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Very Severe</th>
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<tbody>
<tr>
<td>10-15</td>
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<td>&gt;30</td>
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<table>
<thead>
<tr>
<th>CAI &gt; 2 years</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Very Severe</th>
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<td>5-10</td>
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Hypoventilation

Diagnosis:
(1) Hypoventilation – CO2 >6.7 kPa for >25% sleep
(2) Sleep hypoventilation usually as above with a clear distinction in CO2 between awake and asleep
(3) REM hypoventilation – average rise in CO2 during REM sleep of greater than 5mmHg (0.7kPa)

Severity assignment:
Scores severity according to highest index

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean CO2 &gt; 6.7kPa</td>
<td>&lt;7kPa</td>
<td>7-8kPa</td>
<td>&gt;8kPa</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>tCO2&gt;6.7 kPa</th>
<th>Borderline</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoventilation in REM only</td>
<td>&gt;= 25%</td>
<td>&lt;7kPa</td>
<td>7-8kPa</td>
<td>&gt;8kPa</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mean CO2 &gt; 6.7kPa</th>
<th>Borderline</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>&lt;7.3</td>
<td>&lt;7.3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Elevated arousal index</th>
<th>Borderline</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>
REFERENCES


99. Cote CJ, Posner KL, Domino KB. Death or neurologic injury after tonsillectomy in children with a focus on obstructive sleep apnea: houston, we have a problem! Anesthesia and Analgesia 2014; 118:1276-1283.


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