

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

2015



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.*

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## EXECUTIVE SUMMARY

About one third of children snore; about 10% snore most nights and 3-5% of children have obstructive sleep apnoea (OSA). As a symptom, snoring is often not considered unusual or to have health consequences. The potential for OSA to impact on health is often underappreciated by parents and families and not mentioned or raised as a health issue with General Practitioners.

The way OSA presents in children is different from adults. An awareness of the potential significance of poor quality sleep in children is very important. Even in mild cases and in young children, untreated OSA may result in significant adverse consequences including impacts on cardiovascular health (e.g. hypertension) and poor sleep quality, leading to impaired daytime functioning affecting development, behaviour and learning.

Symptoms of snoring and sleep disordered breathing (SDB) should be sought in any child with enlarged tonsils and/or disturbed or unrefreshing sleep, particularly if they have underlying comorbidities such as Down syndrome, obesity, previous cleft, craniofacial abnormalities or neuromuscular weakness, as the prevalence of underlying OSA and its subsequent consequences may be greater.

OSA can often be diagnosed without the need for elaborate tests and treated effectively with adenotonsillectomy. However when the diagnosis is in doubt, when there are underlying comorbidities or anaesthetic risk, or when ENT intervention fails to resolve symptoms, most experts recommend formal evaluation using sleep studies (polysomnography, PSG) and consideration of other treatments (e.g. nasal steroids; respiratory support).

The New Zealand Guideline for the Assessment of Sleep-Disordered Breathing in Childhood 2014 was produced by the Paediatric Sleep Medicine Clinical Network group. The purpose of the guideline is to provide an up to date evidence-based summary for the assessment of children with SDB.

This guideline is intended for use by primary and secondary care practitioners involved in the care of children and young people to consider the possibility of SDB. 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 SDB in childhood. The guideline **does not** address the management of SDB 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.

### Summary algorithm

Appendix 3 is a summary algorithm for the investigation and management of suspected OSA in children.

### Recommendations

The consensus recommendations from the guideline are as follows:

#### *Sleep medicine services for New Zealand children*

- There should be increased availability of adenotonsillectomy for the treatment of OSA for New Zealand children.
- There should be increased availability of paediatric diagnostic sleep medicine services, especially PSG, for New Zealand children.
- There should be consistent access to a nationally integrated paediatric sleep service for the investigation and management of SDB for New Zealand children.

### *Technical considerations for sleep studies*

- 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.
- 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.
- 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.
- 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.
- 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.

### *Assessment*

- As part of routine health maintenance visits ALL children should be regularly assessed for sleep problems using the BEARS mnemonic (Appendix 1), 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 paediatricians consider combining a validated paediatric sleep questionnaire (for example Appendix 2), physical examination (specifically ENT – see Figure 2 for tonsil size grading) and an overnight oximetry as this will increase the specificity and positive predictive value for OSA.
- 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 (including obesity, Downs syndrome, Mucopolysaccharoidoses, spina bifida, Achondroplasia, Prader-Willi syndrome, Cerebral palsy, Laryngomalacia, Pierre Robin Sequence and other craniofacial syndromes, previous palatal surgery, prematurity) should be referred to a paediatrician for more extensive evaluation (see section 2.5 and 2.6).

### *Adenotonsillectomy*

- If an otherwise healthy child is determined to have OSA based on history and examination, adenotonsillectomy remains the first line treatment.
- Children with habitual snoring plus other features suggestive of OSA on history or examination but who are otherwise healthy should be referred for consideration of adenotonsillectomy if the tonsils are moderate or large (grade 2 or more).
- Children under 3 years of age, failing to thrive, those with co-morbid conditions (as above) 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.
- Children who have symptoms of OSA but do not have enlarged tonsils or adenoids, or children whose symptoms of OSA persist after adenotonsillectomy should be referred to a paediatrician for more extensive evaluation.
- All children undergoing adenotonsillectomy for OSA should undergo clinical review 6-8 weeks post-adenotonsillectomy. If symptoms remain unresolved further evaluation is indicated by a centre with expertise in paediatric sleep medicine.

### *Referral for sleep studies*

- 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 surgical or anaesthetic risk should have the diagnosis of OSA made objectively (overnight oximetry ± formal sleep study) before surgery is contemplated.

### *Other treatments for OSA*

- Children with OSA 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.
- CPAP is an effective alternative treatment for OSA in children.
- 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.
- At present there is limited evidence for use of the alternative/new CPAP technologies in children – their role is still under evaluation.
- Nasal steroids may be considered treatment for mild OSA, particularly in children with allergic rhinitis, or when adenotonsillectomy is contraindicated, with appropriate follow-up to determine the effect of treatment.
- 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.
- No recommendation can be made regarding the use of oral appliances for children.

### *Complex high-risk children*

- Children with high risk underlying medical conditions (as above) should be routinely assessed for sleep problems at all clinical encounters, including sleep hygiene, sleep duration, snoring and other symptoms of SDB. A sleep questionnaire ± overnight oximetry may be considered as first line investigations.
- Children with complex medical disorders and suspected OSA should be discussed with a centre with expertise in paediatric sleep medicine for consideration of investigation and treatment.
- While children with high risk underlying medical conditions are at increased risk of residual OSA, adenotonsillectomy may well be the recommended first line management.
- Children with increased risk of central apnoea or hypoventilation should be referred to a centre with expertise in paediatric sleep medicine for further evaluation prior to adenotonsillectomy or any airway surgery.
- PSG is indicated in children with PWS prior to and six to ten weeks following the commencement of GH treatment and thereafter as clinically indicated.

### *Obesity*

- 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 may be considered as first line investigations.
- 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 measurement of blood pressure to look for hypertension (>95th percentile for gender, age, and height on 3 separate occasions) and blood tests for metabolic syndrome. Further investigation and appropriate referral may be required.

#### *Neuromuscular disease*

- Lung function (spirometry, cough peak flow 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 overnight oximetry and carbon dioxide monitoring or PSG should be considered in children with a forced vital capacity < 50% predicted, a daytime capillary pCO<sub>2</sub> 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 assisted 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 and future ventilation choices.
- NIV during sleep is indicated for children with chronic respiratory failure (daytime pCO<sub>2</sub> > 6.7 kPa/50 mmHg) ± symptomatic nocturnal hypoventilation.

#### *Disorders of breathing in infants*

- 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 (2-4 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.

#### *Cystic fibrosis, bronchiectasis, other chronic lung diseases*

- 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.
- Overnight oximetry ± carbon dioxide monitoring should be considered at least annually during a clinically stable period in children with CF and non-CF bronchiectasis who have an FEV<sub>1</sub> < 65% predicted.



- Supplemental oxygen should be considered for children with CF and non-CF bronchiectasis when overnight oximetry demonstrates a baseline oxygen saturation  $\leq$  93% and more than 10% of the night spent with oxygen saturations  $<$  90%.
- 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 NIV is being considered.

### **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.

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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

ADHD	Attention deficit hyperactivity disorder
AHI	Apnoea hypopnea index
ALTE	Apparent life threatening event
AASM	American Academy of Sleep Medicine
AAP	American Academy of Pediatrics
ASA	Australasian Sleep Association
ASTA	Australasian Sleep Technologist's Association
AT	Adenotonsillectomy
BiLevel	Bilevel positive airway pressure
BMI	Body mass index
CAI	Central apnoea index
CCHS	Congenital central hypoventilation syndrome
CF	Cystic fibrosis
CI	Confidence interval
CMDHB	Counties Manukau District Health Board
CPAP	Continuous positive airway pressure
CPF	Cough peak flow
CRP	C-Reactive protein
ECG	Electrocardiograph
EDS	Excessive daytime sleepiness
EEG	Electroencephalogram
EMG	Electromyography
ENT	Ear, nose and throat
EOG	Electro-oculogram
FEV <sub>1</sub>	Forced expiratory volume in one second ("lung airflow measure")
FRC	Functional residual capacity
FVC	Forced vital capacity
HDL	High-density lipoprotein
HDU	High dependency unit
HRCT	High resolution computed tomography "CT scan"
ICU	Intensive care unit
ITP	Idiopathic thrombocytopenia purpura
MOH	Ministry of Health
MSLT	Multiple sleep latency test
mSv	Millisievert
MWT	Maintenance of wakefulness test
NEPAP	Nasal expiratory positive airway pressure device
NIV	Non-invasive ventilation
NMD	Neuromuscular disease
NPA	Nasopharyngeal airway
NPV	Negative predictive value
NREM	Non-REM sleep – slow wave sleep
OA	Oral appliance
OAH	Obstructive apnoea hypopnea index
ODI	Oxygen desaturation index
OSA	Obstructive sleep apnoea
OSAS	Obstructive sleep apnoea syndrome
OR	Odds ratio
PAP	Positive airway pressure
PCD	Primary ciliary dyskinesia
PLMs	Periodic limb movements
PPV	Positive predictive value
PSG	Polysomnography
PWS	Prader-Willi syndrome

RDI	Respiratory disturbance index
REM	Rapid eye movement
RERA	Respiratory effort related arousal
RME	Rapid maxillary expansion
SDB	Sleep disordered breathing
SpO <sub>2</sub>	Oxygen saturation measured by pulse oximetry
VC	Vital capacity

## 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 (AAP) Clinical Practice Guideline: Diagnosis and Management of Childhood Obstructive Sleep Apnea Syndrome (OSAS) (2002)**<sup>1</sup> with its accompanying technical report<sup>2</sup> 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 AAP 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)**<sup>3</sup> with its accompanying technical report<sup>4</sup> 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 2014 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
- 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
- New figure
  - Figure 2. Guide to scoring tonsillar size
- New information and additional appendices
  - Appendix 1. Adaptation of the “BEARS” mnemonic
  - Appendix 2. Example of a structured paediatric OSA screening questionnaire
  - Appendix 3. The algorithm / summary for investigation and management of suspected OSA in children has undergone significant revision reflecting recommendations and updates incorporated throughout document
  - Appendix 4 & 5 revised for AASM 2014 document and additional information to guide PSG based severity in paediatric sleep studies.

## **INTRODUCTION**

In 1889, Hill described snoring and restlessness at night as a cause of “backwardness and stupidity in children”<sup>5,6</sup>, but nearly 100 years passed before the first case series of children with obstructive sleep apnoea (OSA) was published<sup>7</sup>. 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<sup>3,8,9</sup> 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<sup>10</sup>. 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<sup>11-18</sup>. 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<sup>19-21</sup>, and may also be the mechanism by which OSA affects learning and behaviour during the day<sup>14,15,22-25</sup>.

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 an 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 hypopnea) and/or intermittent complete obstruction (obstructive apnoea) that disrupts normal ventilation during sleep and normal sleep patterns<sup>1</sup>. 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.

### **Ethnicity Issues**

There remains limited published data on the prevalence of snoring, SDB 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 Māori than non-Māori<sup>26,27</sup>. The 2004 guidelines cited two abstracts that looked at ethnic differences in prevalence of OSA symptoms in New Zealand adults<sup>28,29</sup>. The findings cannot necessarily be extrapolated to the paediatric population.

Since 2004 this research, previously in abstract format, was combined and published<sup>30</sup>. This described the evidence from a New Zealand national survey in adults that Māori have a

higher prevalence of OSA than non-Māori<sup>30</sup>. This involved a population based mail-out survey (71% response rate) and was sent to 10,000 New Zealanders (5,500 of Māori descent and 4,500 non-Māori) 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 Māori had higher rates of SDB than non-Māori in that they were significantly more likely to 'always snore', have 'observed apnoea' and have daytime sleepiness than non-Māori. The higher risk among Māori reduced and became non-significant after adjusting for well-recognised risk factors such as increased body mass index (BMI) 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<sup>30</sup>.

The increased prevalence of OSA among American Indians and Hispanic adults, and increased severity among Pacific Islanders and Māori, 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)<sup>31</sup>. 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<sup>32</sup>. 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 OSAS (2012)<sup>3,4</sup>, 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 SDB 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 SDB. Direct referral by a General Practitioner to an otorhinolaryngologist (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 otorhinolaryngologists, 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 otorhinolaryngologists for adenotonsillectomy. A significant increase in ENT resources will 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 this test. 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 for the year prior to diagnosis, implying that earlier recognition, accurate diagnosis and treatment will result in cost savings<sup>33-35</sup>.
- **Decreased use of special educational services.** Behavioural problems and deficits in neurocognitive functioning have been well documented in children with OSA<sup>12,36-44</sup>. Treatment of OSA leads to improvements in quality of life<sup>22,45-48</sup>, and improvements in behaviour and mental functioning in children including suspected ADHD<sup>22,43,49-53</sup>. 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<sup>54-57</sup>.
- **Decreased use of support services.** NIV in children with chronic respiratory failure secondary to NMD is associated with improvements in quality of life<sup>55,58-62</sup>, and subsequent decreased use of support services such as psychology, psychiatry, pain specialist and potentially delay input from palliative care services.

**Closing the gaps**

A national network for sleep services will ensure the fair distribution of resources and allow equity of access to:

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

**Performance measures**

The following remain 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 by triage category (both hospital/sleep laboratory studies and home overnight oximetry)
- Total numbers and types of sleep studies undertaken nationally on children < 15 years
- Feedback from consumer groups about the availability of paediatric sleep medicine services

<b>Introduction: Gaps and Issues - Recommendations</b>
There should be increased availability of adenotonsillectomy for the treatment of OSA for New Zealand children.
There should be increased availability of paediatric diagnostic sleep medicine services, especially PSG, for New Zealand children.
There should be consistent access to a nationally integrated paediatric sleep service for the investigation and management of SDB for New Zealand children.

## **SECTION 1: SLEEP SERVICES & TECHNICAL CONSIDERATIONS**

### **1.1 Sleep services for children in New Zealand**

In 2014 sleep medicine services specifically for children are currently available in Auckland, Wellington Hospital, WellSleep (Otago University Wellington) and through the Sleep Well clinics (Dr Alex Bartle). Other peripheral centres (specifically Palmerston North, Christchurch and Dunedin) have paediatricians experienced in respiratory and/or sleep medicine but with limited or no access to full PSG. All services continue to be in a constant state of flux and are vulnerable in terms of maintenance of staffing and paediatric respiratory physiologist expertise. 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 Pattermore (Christchurch). 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.

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.

### **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). However, when PSG is used for assessing nocturnal seizures an extended EEG montage should be used and reviewed by appropriately trained personnel.

#### **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 of the treatment is maintained; or confirms whether or not 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, 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<sup>63</sup> to estimate sleep wake pattern from activity; multiple sleep latency test (MSLT) to establish objectively the level of daytime sleepiness; or maintenance of wakefulness test (MWT) to assess the efficacy of treatment for EDS.

### 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<sup>64</sup>. 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 routine sleep studies in most centres is long (4-6 months). 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<sup>3,65-68</sup>, 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)<sup>69</sup>. Given the potential for continuing adverse consequences of undiagnosed and untreated OSA<sup>48</sup>, 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 unsupervised 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 (Type 1) or unsupervised in hospital or at home (Type 2). 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. Type 3 and type 4 studies can be supervised or unsupervised.

An **attended** study refers to a sleep study continuously attended by medical, scientific/technical or nursing staff specifically trained in the performance of sleep studies. An **unattended** study is 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.

The duration of studies may vary:

- **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<sup>3,67,68,70</sup>. 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)<sup>399</sup> 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<sup>71-74</sup>.

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/hypopnea 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. PLM disorder, parasomnias) and may reveal other sleep disorders (e.g. a very short REM latency may be suggestive of narcolepsy but an MSLT is suggested to diagnose

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<sup>3,65,68</sup>. 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<sup>71,75</sup>. 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<sup>76</sup>.

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 4**)). 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<sup>75</sup>. 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<sup>75</sup>. Further research is required if unattended studies are to be used in children with medical comorbidities.

### **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) or 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, SDB 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 sleep and body movement arousals can be estimated visually<sup>76</sup>.

Whereas cardiorespiratory studies are more accessible and lower 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<sup>77</sup> 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 (level 1 AHI > 5) in a subset undergoing laboratory PSG, cardiorespiratory studies showed good sensitivity (88%) and specificity (98%). However, although this cohort study reported 'excellent agreement', no actual data were reported. Zucconi et al 2003<sup>78</sup> evaluated a cardiorespiratory device in the hospital ward (children 3-6 years, n=12) compared with laboratory PSG on another night. This showed good sensitivity (89%) but

very poor specificity. Poels 2003<sup>79</sup> 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<sup>76</sup> 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  $\geq$  70% and NPV > 90%). A recent study by Tan et al found that clinical decisions at the extremes of SDB (normal/primary snoring with OAHl<1, or severe OSA with OAHl>10/h) were unaffected by the choice of cardiorespiratory over PSG studies, whereas in the middle range of OAHl (1-10/h), a cardiorespiratory study may underestimate events and have significant impacts on treatment decision<sup>80</sup>. 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 in a ward setting may be useful in cases where a child is too unwell to have a formal study undertaken in a sleep laboratory, or as a relatively simple way of determining the efficacy of treatment in the early stages of initiation of non-invasive respiratory support.

#### **1.3.4 Overnight pulse oximetry (Type 4 sleep study)**

Type 4 studies incorporate 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 Health Guidelines <https://www.starship.org.nz/for-health-professionals/starship-clinical-guidelines/o/oximetry/> 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<sup>81</sup>. The development of the multiwave length oximetry and reduction in device size has made oximetry a ubiquitous and accurate marker of hypoxaemia.

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 (a fast averaging time i.e. 2-4 seconds) and storage sampling frequency (1Hz, or 2 second equivalent, depending on the device) 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 quantification of desaturation events 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 as the pattern of desaturation may be identical. Low baseline oxygen saturation will

reduce specificity further. Supplemental oxygen therapy may negate the utility of signal interpretation.

Despite these limitations, the appearance of the trends 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<sup>69</sup>. 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  $\geq 3$  clusters (a cluster =  $\geq 5$  desaturations within a 30 minute period) with  $\geq 3$  desaturation events to  $< 90\%$  had a positive predictive value (PPV) of 97% and could therefore reliably predict the need for adenotonsillectomy without the need for further testing<sup>81</sup>. 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<sup>82-84</sup>, a finding that mandates careful planning of surgical intervention and perioperative care<sup>82</sup>. Recently Horwood et al<sup>69</sup> 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<sup>85</sup>.

Overnight oximetry may also be evaluated using frequency of desaturation events (oxygen desaturation index, ODI). However Kirk et al 2003<sup>86</sup> (N=558 4-8 years) showed poor agreement between the ODI and AHI measured by PSG (sensitivity 67%, specificity 60%, PPV 60%, & NPV 67% for an AHI  $> 5$ /hour). Pena-Zarza 2012<sup>87</sup> 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 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.

<b>Sleep Services and Technical Considerations for Sleep Studies - Recommendations</b>
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.
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.
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.
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.
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.



## SECTION 2: SNORING AND OBSTRUCTIVE SLEEP APNOEA

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

While sleep disorders are common in children they are often under recognised. Approximately 25% of all children experience some type of sleep problem during childhood. While many are transient (e.g. bedtime resistance, night terrors) others can be chronic (e.g. OSA). Initial screening is important in recognising and identifying sleep issues in children. The “**BEARS**” mnemonic has been suggested as a good screening tool for reminding health professionals of important areas to cover when taking a history to screen for sleep issues at routine paediatric health visits.

An adaptation of the “**BEARS**” mnemonic<sup>88</sup> is (see also **APPENDIX 1**):

**B = Bedtime issues (trouble going to bed or trouble falling asleep)**

- Question “Does your child have any difficulty going to bed or falling asleep?”

**E = Excessive daytime sleepiness/excessive disruptive symptoms**

- Question “Is your child difficult to wake in the morning, act sleepy, or are they overactive, inattentive or easily frustrated?”

**A = Awakenings at night**

- Question “Does your child have trouble with waking up at night?”

**R = Regularity and duration of sleep (bedtime, wake time, average sleep duration)**

- Question “What time does your child go to bed and get up on schooldays? weekends?”

**S = Snoring/Sleep Disordered Breathing (SDB)**

- Question “Does your child have noisy breathing or snore on most nights?”

While there are > 60 different diagnosable sleep disorders, the most common is OSA. Several large epidemiologic studies in western societies have found that about 1 in 10 pre-schoolers snore<sup>89</sup> and 3-5% of children have OSA<sup>3,8,9</sup>. OSA is associated with significant morbidity, reduced quality of life<sup>90</sup>, failure to thrive, behavioural problems<sup>12,44</sup>, cognitive impairments, cor pulmonale, right ventricular hypertrophy and systemic hypertension<sup>91</sup>.

Habitual snoring is the cardinal clinical feature of OSA<sup>2,3,5-7,10,32-42</sup>. The diagnosis is unlikely in children who do not snore<sup>34</sup>. 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<sup>12,44</sup>. “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, but this needs to be qualified by checking the type of noise: snuffling, snoring, stridor or wheezing. 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<sup>4,10,32,34,41-46</sup>. However, certain symptoms in conjunction with tonsillar hypertrophy increase the likelihood of significant OSA in children who snore:

- witnessed obstructive apnoea (odds ratio (OR) 3.3<sup>32</sup>),
- frequent daytime mouth breathing (OR 3.7<sup>32</sup>),
- parent afraid / wakes child because of breathing (OR 4.4<sup>32</sup>),
- struggling to breathe while asleep (OR 5.54<sup>32</sup>),
- frequent waking from sleep in a child who has previously slept through<sup>47</sup>,

- secondary enuresis<sup>2,48,49</sup>,
- daytime behavioural problems<sup>8,10,11,13-16</sup>,
- failure to thrive or slowing of weight gain<sup>50-54</sup>.

Excessive daytime sleepiness (EDS) is not predictive of the presence of OSA in young children<sup>32</sup>, but is a cardinal feature of the OSA syndrome in adults, and thus this symptom should be sought, particularly in adolescents.

The use of a structured validated questionnaire (for example the Chervin questionnaire<sup>92</sup> 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<sup>35</sup>. Combining a questionnaire which is abnormal or "high risk", 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<sup>87</sup>. However, this is only if the oximetry is positive. If the oximetry is negative and the other scores positive, the authors recommended home polygraphy. If the questionnaire suggests low risk and examination does not reveal significant adenotonsillar hypertrophy, ongoing surveillance is recommended.

A lateral neck x-ray can be used to assess adenoid/tonsil size but its use is controversial and not universally accepted. While it is a simple technique, a number of methods exist for standardizing the procedure<sup>93</sup>, and the interpretation of its ability to predict OSA is variable<sup>94</sup>. There are significant methodological problems<sup>95</sup> that limit its clinical impact and the small amount of radiation (0.2mSv, variation reported in the literature 0.07-0.3mSv) is noted. In isolation the evidence for its use is weak and as such should not be used as a prerequisite for referral to ENT services. There is also no evidence to support its use in follow-up after treatment for OSA e.g. nasal steroids or previous adenoidectomy. The combination of the predictors of specific positive questions (e.g. observed apnoea, nocturnal enuresis), along with examination findings of mouth breathing and enlarged tonsils as well as radiological narrowing has a PPV 73% and NPV of 80% which is lower than using a validated questionnaire, examination and a positive overnight oximetry.

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<sup>10,40,42,44,46,55-61</sup>, 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<sup>96</sup> examined possible characteristics that may increase OSA severity in children (5-9 years) awaiting adenotonsillectomy. This was a cross-sectional screening study using data analysed 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.

<b>Screening by History and Examination - Recommendations (See Appendix 3 - Summary investigation and management of suspected OSA in children)</b>
As part of routine health maintenance visits <b>ALL</b> children should be regularly assessed for sleep problems using the <b>BEARS mnemonic (Appendix 1)</b> , 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 paediatricians consider combining a validated paediatric sleep questionnaire (for example <b>Appendix 2</b> ), physical examination (specifically ENT) and an overnight oximetry as this will increase the specificity and positive predictive value for OSA.
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.

## **2.2 Treatment with adenotonsillectomy without polysomnography**

Polysomnography remains the gold standard for the diagnosis and assessment of OSA<sup>3,67,68,70</sup>. 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<sup>3,65-68</sup>. In clinical practice this distinction may be considered less important, as both primary snoring and OSA have similar implications for behaviour, and learning<sup>11,68</sup>. Except that milder cases may resolve spontaneously (ref CHAT) or respond to medical treatments (see later). Adenotonsillar hypertrophy is the most common cause of upper airway obstruction in childhood, and adenotonsillectomy remains the treatment of choice<sup>3,97,98</sup>. If the associated factors mentioned in the previous section are present, consideration of adenotonsillectomy is warranted without sleep studies. However by using this pathway we accept that we will operate on some children who possibly could have been managed in other ways.

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<sup>3,66,68,99,100</sup>. However, children with OSA are at an increased risk of peri-operative 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 a more common cause of adverse outcomes<sup>101</sup>.

The risk of perioperative respiratory events for adenotonsillectomy is increased from 1% to approximately 20% in children with OSA<sup>102</sup> and may be as high as 60% in severe OSA<sup>102</sup>. The overall incidence of major neurologic injury or death following adenotonsillectomy is approximately 1 in 27,000<sup>103</sup>. The vast majority of events are minor requiring only a period of supplemental oxygen or medication. However, medical intervention including CPAP and re-intubation may be required for these complications in children with OSA.<sup>104-107</sup> The risk of significant events requiring major intervention such as ICU admission is 6-8% in children with OSA<sup>108,109</sup> but may be as high as 20% if the OSA is severe<sup>82</sup>. 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<sup>82,101,110</sup> 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<sup>82,101,110,111</sup>.

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<sup>17,22,23,33,45-47,49,112-131</sup>. 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<sup>3,98</sup>. Other studies report variable improvement in neurocognitive outcomes following adenotonsillectomy (despite demonstrated improvement in respiratory parameters)<sup>132</sup>. Improvements are demonstrated in both children with primary snoring and those with OSA confirmed on PSG<sup>66</sup>.

One study has suggested that removal of either the tonsils or the adenoids alone carries a significant risk of persistence or recurrence of OSA<sup>120</sup>, and thus consideration should be given to removing both at the same surgery, unless contraindicated, to reduce the likelihood of persistent OSA.

All children undergoing adenotonsillectomy should be assessed for the presence of OSA<sup>67,103,133</sup>. 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<sup>134,135</sup> 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<sup>81</sup> (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<sup>136</sup>.

If a child undergoing adenotonsillectomy for recurrent tonsillitis 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<sup>103,133</sup>. Children with known severe OSA should have surgery performed preferentially in the morning rather than the afternoon<sup>137</sup>.

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<sup>103</sup> 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 a nasopharyngeal airway (NPA) may help prevent the need for post-operative ICU admission<sup>110</sup>. 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<sup>110</sup>. 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  $\leq$  3 years
2. Severe OSA\*
  - obstructive events (OAHl) or RDI\*\*  $\geq$  10 events per hour of sleep on PSG
  - or oxygen saturation nadir  $<$  80% on PSG or overnight oximetry
  - or McGill oximetry score  $\geq$ 3 on overnight oximetry (3 or more events  $<$  85%)
  - or elevated carbon dioxide on PSG or blood gas
3. Significant co-morbidities, especially:
  - Down syndrome
  - Craniofacial anomalies
  - Neuromuscular disease especially hypotonia
  - Cardiac problems or pulmonary hypertension
4. Obesity (weight  $>$  95th Centile, BMI  $>$ 30kg/m<sup>2</sup>)
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<sup>133</sup>.*

*\*\*The OAHl or number of obstructive apnoea and hypopnea 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
  - OAHl or RDI  $\geq$  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 (BMI  $>$ 35kg/m<sup>2</sup>)<sup>110</sup> 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.

**Post-operative and ongoing follow-up**

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<sup>3,97,100</sup>. 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<sup>100</sup>. 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<sup>138</sup>.

### **Treatment with adenotonsillectomy without polysomnography - Recommendations**

If an otherwise healthy child is determined to have OSA based on history and examination, adenotonsillectomy remains the first line treatment.

Children with habitual snoring plus other features suggestive of OSA on history or examination but who 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**).

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.

Children who have symptoms of OSA but do not have enlarged tonsils or adenoids, or children whose symptoms of OSA persist after adenotonsillectomy should be referred to a paediatrician for more extensive evaluation.

All children undergoing adenotonsillectomy for OSA should undergo clinical review 6-8 weeks post-adenotonsillectomy. If symptoms remain unresolved further evaluation is indicated by a centre with expertise in paediatric sleep medicine.

### **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:

- a. when definitive confirmation of the diagnosis is needed.
- b. 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)<sup>139</sup>

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 4 and 5** 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:

- a. Overnight oximetry. This can be useful if it shows a pattern of cyclic desaturation<sup>140</sup>. 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<sup>141</sup> and the limitations of the technology<sup>142</sup>, and it may not be as useful in children with co-morbidity<sup>1</sup>. A normal oximetry should not be used to exclude OSA, as it has a low negative predictive value<sup>105,140,143-145</sup>. 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>)
- b. Daytime (nap) PSG has a high PPV, but a low NPV<sup>146,147</sup>.
- c. Limited information is available to support the use of abbreviated sleep studies in children<sup>146,148-154</sup>. Validation studies in adults are not sufficient, due to the different detection requirements and scoring criteria necessary for interpretation of paediatric studies<sup>155</sup>.

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

### Referral for sleep studies before adenotonsillectomy - 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 surgical or anaesthetic 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 abnormality 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<sup>24,156-162</sup>. The exact threshold for the severity of OSA that should be treated with CPAP is not determined for children but in New Zealand an OAH1 > 5 i.e. moderate OSA in combination with significant symptoms 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)<sup>163</sup>. Few studies have analyzed objective adherence to CPAP in children<sup>163-167 168</sup>. Adherence rates are often sub-optimal – nightly rates of CPAP use range from 3 - 8 hours/night<sup>163-167 168</sup>. The pattern of CPAP use is often established very early in treatment-adherence may be optimised by getting the pressures 'right' early<sup>166</sup>. Previous research has described that children are more likely to be adherent if the baseline AHI is high/severe<sup>167</sup>; if there is a greater change in AHI on CPAP<sup>167</sup>; if they are younger (6-12 versus 13-18 years)<sup>164</sup>; if a full face mask is used instead of a nasal mask<sup>164</sup>; when subjects participated in an educational program<sup>169</sup>. 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<sup>168</sup>.

Even though 10-30% of children fail CPAP therapy<sup>164,165,167,170,171</sup> 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<sup>172,173</sup> While there may be role, these cannot be recommended based on current paediatric research evidence;
- **Pressure relief technology** (e.g. C-Flex) 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<sup>174</sup> that aims to evaluate the effects of Bi-Flex (similar to 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<sup>175</sup>. CPAP candidates (AHI > 5, 8-16 years) underwent PSGs using NEPAP and placebo. Nine responders (subjects with > 50% reduction in AHI from placebo to NEPAP night or AHI < 5 / hour on NEPAP night) were eligible for NEPAP for 30 days. Out of the 14 teenagers (age 13.4 ± 1.9 years), BMI z-score 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;
- **Nasal high flow air** (e.g. 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<sup>176</sup> by McGinley et al. Twelve children (age: 10 ± 1 years; BMI: 35 ± 14 kg/m<sup>2</sup>; AHI ranged from mild to severe (2-36 events/ hour), compared CPAP to high flow air at 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 event/hour but CPAP was titrated. Excluding 2 poor responders the effect of high flow was clinically similar to CPAP (1 vs 2 events/hour). In infants, Airvo it has been shown to have less nasal trauma than bubble CPAP<sup>171</sup>, however, there are case reports of pneumothorax/air leak but less than with CPAP<sup>177</sup>.

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<sup>178-180</sup>. 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<sup>181,182</sup>. 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 or improvement of sleep disordered breathing (e.g., significant weight gain or airway surgery respectively). 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).

<b>Treatment for OSA other than adenotonsillectomy - Recommendations</b>
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.
CPAP is an effective alternative treatment for OSA in children.
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.
At present there is limited evidence for use of the alternative/new CPAP technologies in children – their role is still under evaluation.

## **2.4.2 Alternative treatments**

### **2.4.2.1 Anti-inflammatory agents**

Nasal steroids (fluticasone, budesonide and mometasone) either alone<sup>183 184 185</sup> or in combination<sup>186</sup>, and leukotriene antagonists (Montelukast)<sup>187</sup> have been proposed as possible treatments for OSA particularly in children with allergic rhinitis<sup>188</sup>. A study using oral steroids failed to show a therapeutic effect on OSA<sup>189</sup>.



Nasal steroids have been shown to improve OSA in the short term ( $\leq 8$  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  $\geq 8$  weeks. Two studies showed shrinkage of adenoids<sup>185,190</sup> 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 OSA as an alternative prior to T & A, 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.

Currently in New Zealand Montelukast is not funded for treatment of OSA. However, there is some evidence that Montelukast may be helpful in the treatment of mild OSA either alone<sup>187</sup> or in combination with nasal steroids<sup>186</sup>. The findings of these trials are preliminary and need to be re-examined in larger randomised controlled trials. However, the findings may be relevant to individual children with mild OSA ( $< 7$  years, allergic rhinitis, enlarged adenoids and non-obese) who are able to fund treatment.

#### **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<sup>191,192</sup>, Rapid maxillary expansion (RME) has also been proven effective<sup>193,194</sup>. The combination of surgical treatment and OA treatment has been shown to be effective if the OA treatment is undertaken first<sup>195</sup>. 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<sup>196</sup>. Another study<sup>197</sup> 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 apnoea'<sup>198</sup>, 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<sup>199,200</sup>. 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<sup>201-205</sup>. Again, further research is needed, but this treatment may be considered in individual cases.

<b>Alternative Treatments for OSA - Recommendations</b>
Nasal steroids may be considered treatment for mild OSA, particularly in children with allergic rhinitis, or when adenotonsillectomy is contraindicated, with appropriate follow-up to determine the effect of treatment.
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.
No recommendation can be made regarding the use of oral appliances for children.

## 2.5 Complex high-risk children

Children with high risk underlying medical conditions (**see list below**) should be routinely assessed for sleep problems at all clinical encounters, including sleep hygiene, sleep duration, snoring and other symptoms of SDB. Obstructive sleep apnoea should be particularly considered due to the high rates of OSA and its consequences. A sleep questionnaire and/or overnight oximetry if available may be considered as first line investigations.

### List of important high risk conditions:

- Down Syndrome<sup>206-222</sup>
- Mucopolysaccharoidoses<sup>223-227</sup>
- Spina bifida<sup>228-230</sup>
- Achondroplasia<sup>231-237</sup>
- Prader-Willi syndrome (PWS)<sup>238-242</sup>
- Cerebral Palsy<sup>243-249</sup>
- Laryngomalacia<sup>250-253</sup>
- Pierre Robin Sequence<sup>254-259</sup> and other craniofacial syndromes<sup>260-265</sup>
- Previous palatal surgery<sup>266-270</sup>
- Prematurity<sup>271-273,274,275,276,277</sup>
- Obesity (**see section 2.6**)

Children with complex medical disorders and suspected OSA should be discussed with a centre with expertise in paediatric sleep medicine for consideration of investigation and treatment. Adenotonsillectomy may well be the recommended first line management. However, 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<sup>228,278</sup>, may not be indicated at all, or may even be contra-indicated. International guidelines advise PSG prior to adenotonsillectomy for these children in order to make an accurate diagnosis due to increased perioperative risk<sup>67</sup> and decreased accuracy of parental symptom reporting<sup>279</sup>. 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 may not be necessary to make the diagnosis. For example, studies have shown that in cases of Down syndrome where parents suspect OSA the incidence is close to 100%<sup>213,217</sup>. Nevertheless, an assessment of perioperative risk is required in order to guide post-operative monitoring. At a minimum, overnight oximetry should be performed<sup>218,280</sup> and PSG may be required in some circumstances. 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<sup>67,139,281,282</sup>.

As incomplete resolution of OSA is more common in children with a high preoperative AHI<sup>283-285</sup>, obesity<sup>48,283-285</sup>, craniofacial anomalies that obstruct the upper airway<sup>286</sup>, neurologic disorders such as myelomeningocele<sup>287</sup> and Down syndrome<sup>288</sup>, 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 adenotonsillectomy 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 SDB 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.

Obstructive and central sleep apnoea and respiratory abnormalities are common in children with PWS. Children with PWS have been reported to be at risk of sudden death during sleep and this risk appears to be elevated by OSA, including during growth hormone therapy<sup>238,241</sup>. Children with PWS are now often treated with growth hormone, from ~ 6 months of age, which has been shown to have a multitude of beneficial effects. Growth hormone supplementation may increase the volume of lymphoid tissue in the upper airway. This has raised the issue of whether routine monitoring for physiological abnormalities during sleep should be undertaken. Limited studies<sup>240</sup> 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 outside of growth hormone treatment and additional studies with larger numbers of subjects and longitudinal data are needed<sup>239</sup>. It is recommended that children with PWS be assessed for symptoms of SDB at all clinical encounters. If there is clinical concern about SDB, a referral for consideration of investigation and treatment together with an overnight oximetry (if available) should be made to a centre with expertise in paediatric sleep medicine. It is acknowledged that due to the risk of both obstructive and central respiratory events, oximetry may be more difficult to interpret, especially < 2 year olds. If GH supplementation is to be considered it is recommended that a PSG be undertaken prior and 6-10 weeks following commencement of growth hormone and as clinically indicated thereafter<sup>238</sup>. If children with PWS on GH develop (or have an increase in) symptoms of OSA and/or an overnight oximetry is diagnostic of OSA then GH will be discontinued pending definitive review especially in older/obese children. If suspicion is low and/or oximetry is equivocal, GH may be continued until definitive review.

Other high risk conditions listed above may also be at risk of a combination of obstructive and central sleep apnoea and/or hypoventilation. Children with disorders associated with Arnold-Chiari malformation (e.g. achondroplasia) may be screened with oximetry combined with transcutaneous carbon dioxide, but a PSG may be needed to diagnose the type of SDB (e.g. central vs OSA) particularly in the first few months of life as a guide for neurosurgery<sup>235</sup>.

<b>Complex High-Risk Children - Recommendations</b>
Children with high risk underlying medical conditions ( <b>see list</b> ) should be routinely assessed for sleep problems at all clinical encounters, including sleep hygiene, sleep duration, snoring and other symptoms of SDB. A sleep questionnaire ± overnight oximetry may be considered as first line investigations.
Children with complex medical disorders and suspected OSA should be discussed with a centre with expertise in paediatric sleep medicine for consideration of investigation and treatment.
While children with high risk underlying medical conditions are at increased risk of residual OSA, adenotonsillectomy may well be the recommended first line management.
Children with increased risk of central apnoea or hypoventilation should be referred to a centre with expertise in paediatric sleep medicine for further evaluation prior to adenotonsillectomy or any airway surgery.
PSG is indicated in children with PWS prior to and six to ten weeks following the commencement of GH treatment and thereafter as clinically indicated.

## 2.6 Obesity

### An increasing problem

Obesity is defined as a BMI >95<sup>th</sup> percentile for gender and age<sup>289</sup>. 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<sup>290-293</sup>. An increasing proportion of children being referred for evaluation of SDB fulfils the criteria for

either overweight or obese<sup>294</sup>. The New Zealand 2012/2013 health survey found 11% of 2-14 year olds to be obese (defined as BMI >30kg/m<sup>2</sup>) with a further 22% being overweight (defined as BMI 25-29kg/m<sup>2</sup>). 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>). The incidence of obesity was found to be greatest in Māori (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 Māori 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<sup>295-297</sup>. 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<sup>298,299</sup>. In the only New Zealand survey, which involved 3 year old children, 11% snored and 15% (N=93) were obese<sup>32</sup>. 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<sup>4</sup>. The prevalence of OSA in obese children is estimated to vary between 13-59%<sup>300-302</sup>.

The distribution of body fat may be more important in predicting the severity of OSA than BMI alone<sup>8</sup>. However, Verhulst have found that waist circumference is associated with an increased risk of central apnoeas in obese children rather than obstructive apnoeas<sup>303</sup>. 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<sup>304</sup>. For every increment in BMI by 1 kg/m<sup>2</sup> 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<sup>305</sup>. 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<sup>306</sup>. 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.<sup>307</sup> Arens et al showed that obese children have enlarged adenoidal tissues, potentially the result of systemic low-grade inflammatory processes that accompany obesity.<sup>308</sup> Obese children with OSA have also been shown to have a significantly greater adenoidal size compared to obese children with primary snoring<sup>309</sup> and need relatively less adenotonsillar hypertrophy to cause OSA<sup>310</sup>. Increased fat around the viscera, chest wall and abdomen leads to increased respiratory load, decreased lung volumes and functional residual capacity, particularly when supine<sup>311</sup>. 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<sup>312-314</sup>. While some work suggests that society's trend towards shortened sleep duration and increased irregularity of sleep leads to obesity<sup>315-320</sup>, the presence of OSA may promote weight gain and obesity via sleep fragmentation and associated daytime sleepiness<sup>321-324</sup>. 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 grehlin levels<sup>325</sup>. 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<sup>18,20,326,327</sup>, insulin resistance and impaired glucose tolerance<sup>328-330</sup>, increased inflammatory markers (CRP, white cell count)<sup>331-333</sup>, elevated triglycerides and low HDL<sup>330</sup>, and arterial wall stiffness and endothelial dysfunction<sup>334,335</sup>. Childhood obesity also confers an increased risk of future obesity and insulin resistance<sup>336</sup>, further weight gain over expected growth, and risk for development of cardiovascular risk factors as young adults<sup>337</sup>. 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<sup>338</sup>. 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<sup>339</sup>.

### **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<sup>303</sup>. Evidence suggests that obese children with tonsils that occupy more than 50% of the pharyngeal diameter have a high prevalence of OSA<sup>340</sup>. Adenotonsillectomy can significantly reduce the AHI in this high risk group and remains the first line treatment. However, Costa et al in a meta-analysis found that adenotonsillectomy did not resolve OSA in the majority of obese children (postoperatively only 49% had an AHI<5/h, 25% <2/h, and 12% <1/h)<sup>341</sup>. As factors other than adenotonsillar hypertrophy may contribute to OSA in obese children, these children are at increased risk of residual OSA following adenotonsillectomy<sup>283</sup>. Bhattacharjee<sup>342</sup> found the highest risk of residual disease was in obese children >7 years<sup>100</sup>. 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<sup>343</sup>.

Obesity also places a child at higher risk of perioperative complications such as laryngospasm<sup>344</sup> 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 judicious 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 then CPAP is indicated.

There is evidence that weight loss and lifestyle interventions in adolescents can improve symptoms of SDB<sup>303,345</sup> but results are variable and studies in obese children are sparse. Regular exercise<sup>346</sup> and a weight management programme is recommended as an essential part of the treatment plan<sup>3</sup>.

As there there may be persistence after treatment, and compliance with CPAP can be poor, bariatric surgery may be considered for adolescents<sup>347,348</sup>. 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 goals and importantly have decisional capacity to be able to provide

informed consent. The procedure itself - usually sleeve gastrectomy or roux-en-Y gastric bypass - is not without complications, with major complications requiring repeat laparotomy ~7% and minor issues 3% (Surgeons CMDHB personal communication). While short term studies in adolescents are promising in terms of improved weight, blood pressure, quality of life and improved OSA<sup>300,349,350</sup>, longer term studies in adults demonstrate persistent OSA and relapse<sup>351,352</sup>.

Like obesity, OSA is viewed by some as a low grade systemic inflammatory disease and is considered a risk factor for the development of metabolic syndrome<sup>353-356</sup>. 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<sup>357</sup>. Further background evidence to the link between obesity and insulin resistance, dyslipidemia and other metabolic abnormalities are reviewed in the AAP technical document<sup>4</sup>. 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.

<b>Obesity - Recommendations</b>
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 may be considered as first line investigations.
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 measurement of 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.

## **2.7 Neuromuscular disease**

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<sup>56,61</sup>. Non-invasive ventilation probably improves survival in NMD<sup>288,358-361</sup>.

The literature would suggest that NIV is frequently started for the first time for respiratory failure due to acute pneumonia<sup>57</sup>. 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, to date this practice has not been supported in New Zealand for progressive NMD due to the cost and staffing requirements to support this in the community necessitating in hospital care and negative effects on quality of life for both the child and family. 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 inspiratory and expiratory 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.

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<sup>362,363</sup>.

**The following children should undergo regular annual sleep studies (PSG or overnight oximetry and carbon dioxide monitoring (transcutaneous or end-tidal) if PSG is unavailable) and consideration of NIV:**

1. Children with recurrent admissions with lower respiratory tract infection. These children may have reduced ventilation during acute illness and may benefit from NIV even with normal PSG<sup>54,55</sup>.
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  $\geq 4$ mmol/L<sup>364-366</sup>.

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<sup>367-369</sup>. 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<sup>363,370</sup>. A CPF <270 L/min in adults or adolescents (or the equivalent in younger children) indicates the need for routine assistive airway secretion clearance<sup>367</sup>. 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<sup>371</sup>.

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<sup>370</sup>. Note that patients with FVC < 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.

Respiratory emergency action plans are recommended for all children with NMD who are managed on NIV. Brief rest periods on NIV (e.g. 45-90 minutes) may be helpful during or recovering from an acute respiratory tract infection. NIV when awake in the daytime may be

initiated for persistent hypercapnia or low baseline SaO<sub>2</sub> (<92%). However, note that increasing daytime hours of use of NIV is a predictor of impending diurnal respiratory failure.

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<sup>363,370</sup>.

<b>Neuromuscular Disease - Recommendations</b>
Lung function (spirometry, cough peak flow 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 overnight oximetry and carbon dioxide monitoring or PSG should be considered in children with a forced vital capacity < 50% predicted, a daytime capillary pCO <sub>2</sub> 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 assisted 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 and future ventilation choices.
NIV during sleep is indicated for children with chronic respiratory failure (daytime pCO <sub>2</sub> > 6.7 kPa/50 mmHg) ± symptomatic nocturnal hypoventilation.

## **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<sup>372</sup>. If OSA is clinically suspected<sup>373</sup>, 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<sup>372</sup>. There is no evidence that home respiratory ± cardio-monitoring prevents sudden infant death<sup>374</sup>. 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 risk factor 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<sup>274</sup> is perhaps the largest cohort study (N=4145 (1.0% population) - mean age 44.2 mo (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 those born at 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 for gestational age. Two other papers published since 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.<sup>275, 276</sup>



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<sup>375-377</sup>. 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<sup>378</sup>. 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<sup>372</sup>.

<b>Disorders of Breathing in Infants - Recommendations</b>
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 (2-4 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<sup>379</sup>. 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<sup>379</sup>.

### **2.9.1 Asthma**

Studies have found persistent wheezing in children to be a risk factor for SDB<sup>304</sup>. Ross et al<sup>380</sup> 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 having SDB in those with severe asthma compared to other asthmatics 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<sup>381</sup> 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 only indicated in children with asthma when there is clinical suspicion of OSA, morning headaches or cor pulmonale<sup>372</sup>.

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

While bronchiectasis is prevalent in New Zealand particularly in Māori and Pacific Island children<sup>382</sup> 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<sup>383</sup>. 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<sup>384</sup>, 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 (Type 1). Upper airway symptoms and abnormal signs were frequent:  $n=8$  allergic rhinitis;  $n=6$  nasal septal deviation;  $n=2$  polyps;  $n=6$  tonsils  $\geq$  size 2. 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 population rates. Cigarette smoke exposure is an important risk factor for OSA in PCD patients.

Finally Santamaria et al<sup>385</sup> evaluated SDB in 16 stable PCD patients (aged 4 – 17 years) in comparison with 42 controls by undertaking an overnight cardiorespiratory sleep study (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 indices 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  $\pm$  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<sup>386</sup>. Forty children (aged 0.5-11 years) with CF and 18 controls were assessed with PSG (Type 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<sup>387-391</sup>. Two studies suggest that simple gas exchange studies may be useful in CF to monitor hypoxaemia and hypercapnia<sup>392,393</sup>. Sub-clinical pulmonary hypertension develops in a significant proportion of patients with CF and is strongly correlated with hypoxaemia, independent of pulmonary function<sup>394</sup>.

Daytime measurements of lung function have poor specificity for nocturnal hypoventilation in CF<sup>387,390,395</sup>. A continuous overnight oximetry recording to identify children who might benefit from nocturnal supplemental oxygen has been suggested for those with: a resting oxygen saturation awake in room air of <95%<sup>372</sup> or <93%<sup>390</sup>, or a FEV<sub>1</sub><65% predicted<sup>390</sup>, during a clinically stable period.

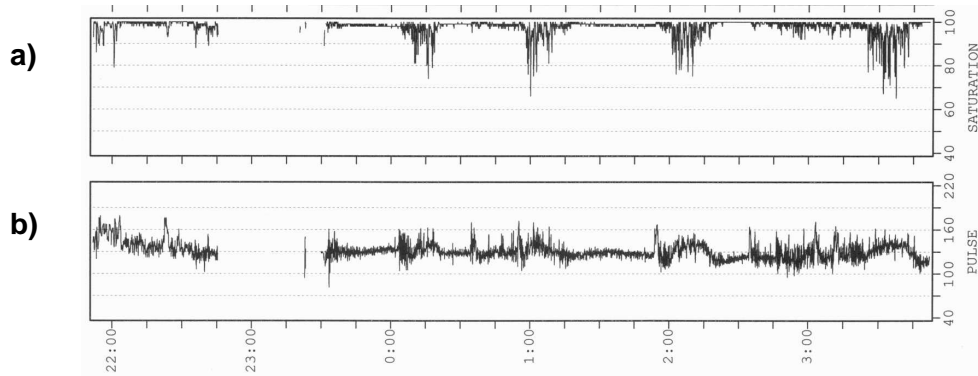
Since the 2004 guidelines were completed two Cochrane reviews have been published evaluating oxygen therapy for CF<sup>396</sup> and NIV in CF<sup>397</sup>. Oxygen therapy at home may be considered when the oxygen saturation is below 90% for more than 10% of the night, in a clinically stable period<sup>398</sup>. 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<sup>399</sup>, and thus undertaking carbon dioxide monitoring 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<sup>391,399-401</sup>. 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<sup>402</sup>. 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<sup>397</sup>.

<b>Cystic Fibrosis, Bronchiectasis, Other Chronic Lung Diseases - Recommendations</b>
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.
Overnight oximetry ± carbon dioxide monitoring should be considered at least annually during a clinically stable period in children with CF and non-CF bronchiectasis who have an FEV <sub>1</sub> < 65% predicted.
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 < 90%.
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 NIV is being considered.

**FIGURE 1. Example of an abnormal overnight oximetry consistent with OSA**

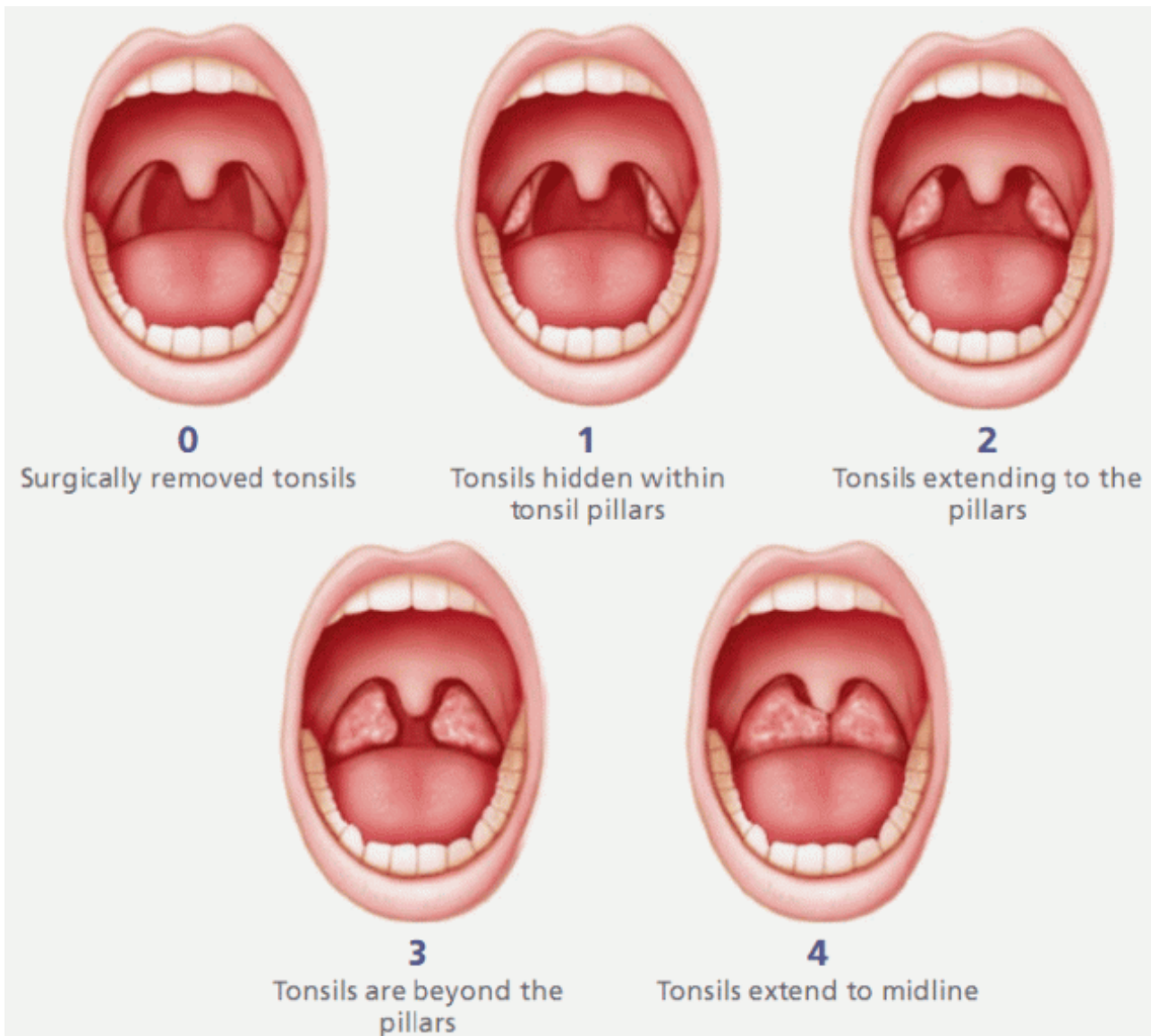
A section of an overnight a) oxygen saturation and b) heart rate recording demonstrating clusters of desaturation with  $\geq 3$  dips in oxygen saturation below 90%. This pattern has a high positive predictive value for OSA in an otherwise normal child with a history suggestive of OSA<sup>140</sup>.



**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



### **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 list

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

Members	Paediatric Society of New Zealand
Jeff Brown	Paediatric and Child Health Division of RACP
Gillian Nixon	Chair 2004 Guidelines Group
Richard Aickin	Chair Clinical Network Group, PSNZ
Pat Tuohy	Chief Child Health Advisor, MoH
Katie Burgess, Secretary	Australasian Sleep Technologists Association
Dr Mike Hlavac, President	New Zealand Branch Australasian Sleep Association
Secretary	NZ Society of Otolaryngology & Head & Neck Surgery
Secretary	Australasian Society of Paediatric Otorhinolaryngology
Chief Executive Officer	Royal NZ College of General Practitioners
Chairperson	NZ Rural General Practice Network
Bernadette Drummond	Paediatric Dentistry Association
Secretary	College of Anaesthetists

## Dissemination list (in addition to consultation list):

Scott Stevenson	ORL Surgical Network
Chairperson	Royal Australian & NZ College Radiologists (NZ branch)
Jenny Prince	Royal New Zealand Plunket Society
Chief Executive Officer	New Zealand Nurses Organisation
Jane Ayling	New Zealand College of Primary Health Care Nurses
The Director	Nurses Society of New Zealand
Executive Director	Aotearoa (NZ) College of Nurses Inc.
National Co-ordinator	Independent Nurse Practitioners Association
Executive Officer	Parent to Parent
Executive Officer	Muscular Dystrophy Association of New Zealand Inc.
Chairperson	New Zealand Association of Plastic surgeons
President	Cleft New Zealand Inc.
Executive Officer	New Zealand Downs Syndrome Society
Chief Executive Officer	Prader-Willi Syndrome Association (NZ) Inc.
Chairperson	Sleep Apnoea Association of NZ Inc.
Dr Rhys Jones	Senior Lecturer & Director of Teaching at Te Kupenga Hauora Māori (TKHM), University of Auckland

## APPENDIX 1. Adaptation of the “BEARS” mnemonic<sup>88</sup>

**B = Bedtime issues (trouble going to bed or trouble falling asleep)**

- Question “Does your child have any difficulty going to bed or falling asleep?”

**E = Excessive daytime sleepiness/excessive disruptive symptoms**

- Question “Is your child difficult to wake in the morning, act sleepy, or are they overactive, inattentive or easily frustrated?”

**A = Awakenings at night**

- Question “Does your child have trouble with waking up at night?”

**R = Regularity and duration of sleep (bedtime, wake time, average sleep duration)**

- Question “What time does your child go to bed and get up on schooldays? weekends?”

**S = Snoring/Sleep Disordered Breathing (SDB)**

- Question “Does your child have noisy breathing or snore on most nights?”



**APPENDIX 2. Example of a structured paediatric OSA screening questionnaire**



SURNAME _____ NHI _____
FIRST NAME: _____
DOB: _____ SEX _____
<small>Please ensure patient details are completed here</small>

Caregiver: \_\_\_\_\_ Date: \_\_\_\_\_  
(print)

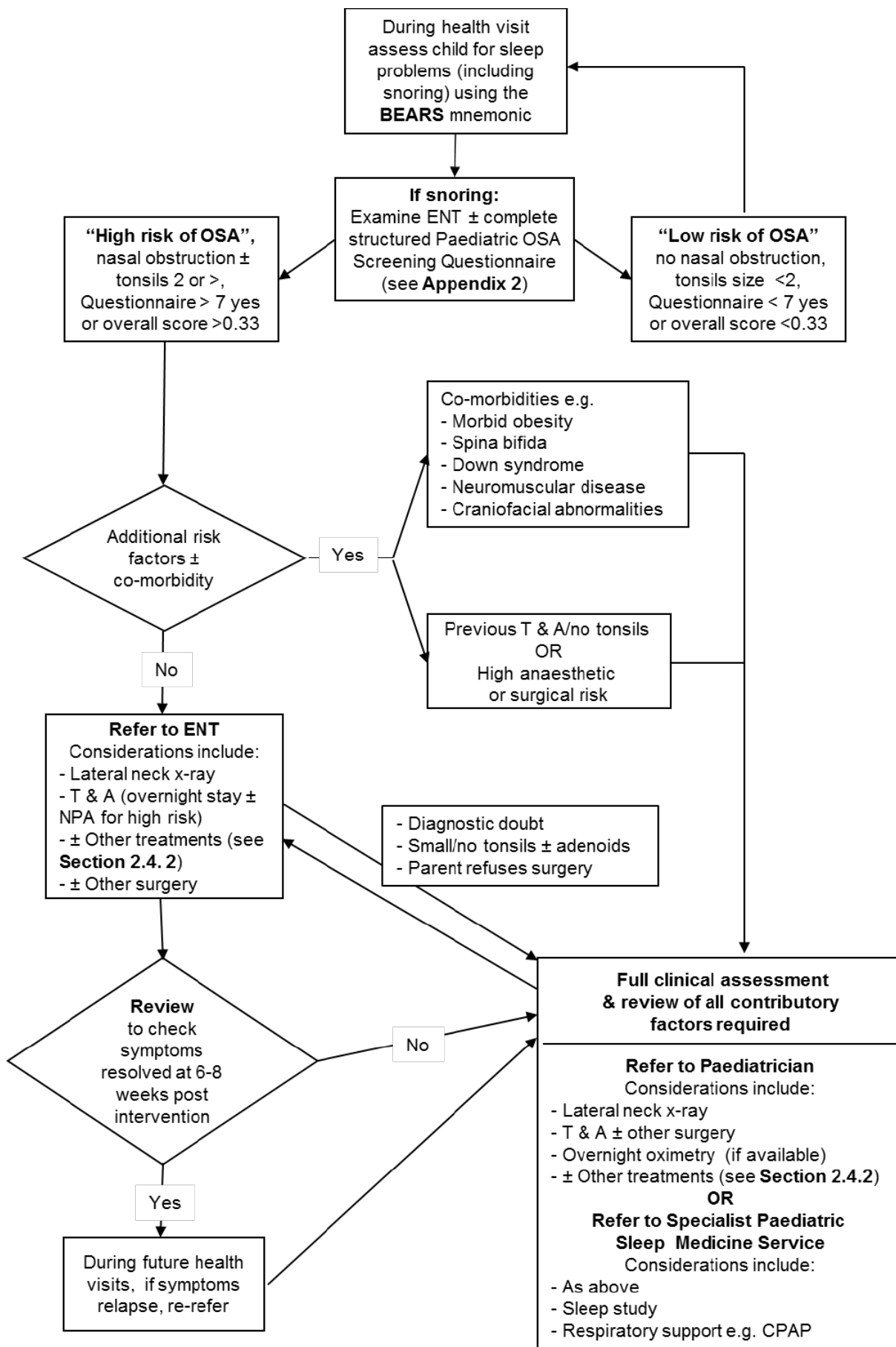
**Please answer on behalf of your child for the past month. If you don't know, circle “?”**

Does your child have any of the following:	Please circle eg	Yes/ No / ?
• A constant blocked or runny nose? .....		Yes/ No / ?
• Recurrent chest infections/pneumonia? .....		Yes / No / ?
• An airway narrowing or abnormality? .....		Yes / No / ?
• A syndrome or other major health problem? .....		Yes / No / ?
Type .....		
• Does your child have large tonsils? .....		Yes / No / ?
• Has your child had their adenoids or tonsils removed? .....		Yes / No / ?

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 often . . .		
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).<sup>92</sup>

**APPENDIX 3. Summary investigation and management of suspected OSA in children**



## APPENDIX 4. Overview of criteria for defining sleep disordered breathing in paediatric sleep studies

The AASM 2014 document<sup>403</sup> 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**.<sup>403,404</sup>

Score a respiratory event as an **obstructive apnoea** if it meets **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 **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 **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 **hypopnoea** if it meets **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 sub-classify **hypopneas**, score a hypopnea as **obstructive** if **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 sub-classify **hypopneas**, score a hypopnea as **central** if **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  $\geq 2$  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  $p\text{CO}_2$  leading to arousal from sleep when the sequence of breaths does not meet criteria for an apnoea or hypopnea.

## APPENDIX 5. 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 OAHl 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 (OAHl <1.2 respiratory event/hour), oxygen desaturation or formally defined respiratory arousals<sup>12,404</sup>. Essentially a diagnosis of primary snoring is given where a child snores but does not reach the threshold for a diagnosis of OSAS.

**Obstructive Sleep Apnoea is defined by an OAHl >1.2/hr** <sup>405</sup>.

#### ***Severity assignment:***

Optimal severity classification remains unclear and may vary according to the outcome measure used. In general OAHl 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.

	<b>Normal</b>	<b>Mild</b>	<b>Moderate</b>	<b>Severe</b>	<b>Extreme</b>
PSG severity (OAHl)	< 1.2/hr	≥1.2-<5/hr	≥5-<10/hr	≥10-30/hr	≥30

#### ***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<sup>406,407,408</sup>.

#### ***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<sup>379,409</sup>

Age 2-15 years -

- PSG – Central Apnoea Index > 5 / hr

#### ***Severity assignment:***

Severity assigned on basis of highest index

	<b>Mild</b>	<b>Moderate</b>	<b>Severe</b>	<b>Very Severe</b>
CAI < 2 years	10-15	15-20	20-30	>30
CAI > 2 years	5-10	10-15	15-20	>20

### **Hypoventilation**

Sometimes baseline transcutaneous carbon dioxide data is unreliable. Confirmation of good quality signals is important.

#### ***Diagnosis:***

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

#### ***Severity assignment:***

Scores severity according to highest index

	<b>Mild</b>	<b>Moderate</b>	<b>Severe</b>
Mean CO <sub>2</sub> > 6.7kPa	<7kPa	7-8kPa	>8kPa

	<b>Borderline</b>	<b>Mild</b>	<b>Moderate</b>	<b>Severe</b>
tcCO <sub>2</sub> >6.7 kPa	Hypoventilation in REM only	>= 25%		
Mean CO <sub>2</sub> > 6.7kPa	<7 kPa	<7kPa	7-8kPa	>8kPa
pH	Normal	Normal	Normal	<7.3
Elevated arousal index	No	No	No	Yes

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