# Obstructive Sleep Apnoea

**8**

Daniel Y. T. Goh

## 8.1 Vignette of Typical Presentation/Real Life Example

A 7-year-old boy incidentally reports frequent snoring when he presents to the family doctor for a minor superficial injury. He is described to be a healthy child, physically active and not obese. He has no past medical history of note except for childhood asthma, allergic rhinitis and eczema. He has been attending preschool since 3 years of age and has just started primary school. He is described to be snoring regularly and on most nights; not loud, but consistent. This child is also described to be restless in bed; moves about a lot in bed, repositioning himself through the night. He sometimes sleeps with his neck hyperextended over the edge of the mattress. He is an average student and is sometimes described by teachers to be fidgety and hyperactive in class. He doesn't fall asleep in class or in the school-bus. Incidentally, his father also snores regularly.

Mom did raise her concerns about the snoring to the paediatrician previously but was told that it is normal to snore and it will spontaneously get better with time.

*Key points in history of significance:* Age, habitual snoring, atopic child, restlessness in sleep, abnormal sleeping positions, hyperactivity, no daytime hypersomnolence, positive family history.

*Assessment:* Tonsils and adenoids are enlarged. No other clinical abnormality noted. Overnight sleep study (polysomnography) shows moderately severe OSAS with an AHI of 6.8/h, no significant desaturation or hypercarbia (measured by end-tidal $CO_2$ measurement). Snoring is noted throughout the night. Events worst during REM sleep and in the supine position.

*Management:* Adenotonsillectomy done with significant improvement in snoring and resolution of the restlessness in sleep. The hyperactivity improved partially over time.

*Key learning notes:* Sleep and snoring need to be actively screened at all healthcare encounters/consultations; parents do not often voluntarily report sleep issues; awareness of snoring and OSA in childhood is still low in many countries in the region.

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## 8.2 Introduction

### 8.2.1 What Is OSAS

OSAS is a condition where there is obstruction in the upper airway during sleep resulting in apnoeas or hypopnoeas, which can be associated with gas-exchange abnormalities and sleep fragmentation and result in complications of poor sleep quality, and also developmental and learning impairment.

### 8.2.2 Spectrum of SDB: Primary Snoring to OSAS

OSAS is part of a group of conditions termed sleep-disordered breathing (SDB) which occurs in a spectrum of severity ranging from mild to severe upper airway obstruction.

On the milder end of the spectrum is Primary Snoring—where there is snoring but there is no apnoea or hypopnoea, no hypoxaemia, hypercarbia or significant arousals. The opposite end of the spectrum is obstructive sleep apnoea syndrome. In between is upper airway resistance syndrome (UARS) where there is evidence of increased respiratory effort and the presence of increased negative intrathoracic pressure during inspiration, associated with arousals and sleep fragmentation despite the absence of airflow or gas exchange abnormalities.

## 8.3 Epidemiology

This is a common condition in childhood that is often underestimated and under-diagnosed. It occurs in children of all ages but is most common in the pre-school age where it is reported to occur in 1–3% of children, corresponding to the peak age of adenoid and tonsillar hypertrophy. OSAS in childhood occurs equally in boys and in girls although some studies have suggested a higher prevalence in boys.

Apart from upper airway size, OSAS is probably determined by a complex myriad of factors including genetics, craniofacial morphology, neural control of the upper airway as well as respiratory control systems. Up to 40% of its variance can be attributed to genetic factors. There is often a family history of snoring or diagnosed OSAS. Inherited craniofacial structure abnormalities may explain in part the familial clustering of OSAS. Asian children may have a lower prevalence of OSAS compared to western populations, however the severity of OSAS may be greater.

## 8.4 Risk Factors

Atopy is also described to be a risk factor for habitual snoring, and in turn, OSAS in childhood. Children with multiple allergies including asthma, allergic rhinitis and atopic eczema should be screened for snoring and possible OSAS (Fig. 8.1 and Table 8.1).
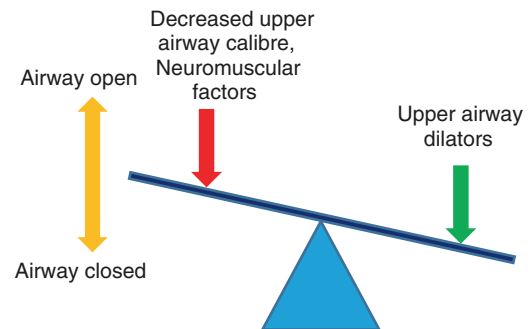


**Fig. 8.1** Counterbalancing forces that influence airway patency. (Adapted from Thach, B. Neuromuscular control of the upper airway. In Beckerman, R et al., Respiratory control disorders in infants and children, Baltimore, Williams & Wilkins, 1990, pg 47)

**Table 8.1**  Factors and mechanisms contributing to the development of OSAS in childhood

| | |
|---|---|
| *Reduced Upper airway calibre*—predominantly in the nasal, oropharyngeal, craniofacial areas | |
| Adenotonsillar hypertrophy | Note that the presence and severity of OSAS does not necessarily correlate with the tissue size |
| Micrognathia/Retrognathia | E.g. Pierre-Robin sequence |
| Macroglossia | E.g. Beckwith-Widemann syndrome |
| Midfacial hypoplasia | With or without craniofacial dysmorphic syndromes e.g., Craniosynostosis |
| Tissue infiltration | Adipose deposition in obesity, mucopolysaccharides in MPS |
| *Reduced upper airway tone* | |
| *Muscle weakness* | Muscular dystrophies, myopathies, Prader-Willi syndrome |
| Neurological disorders | Arnold-chiari malformation, hypotonic cerebral palsy |
| *Reduced central ventilatory drive* | |
| Brainstem lesions | Also contributing to reduced upper airway dilator muscle tone |
| *Combination of multiple factors* | |
| Down syndrome | Hypotonia, glossoptosis, obesity, midfacial hypoplasia, hypothyroidism |

**Table 8.2**  Symptoms of OSAS in childhood

| | |
|---|---|
| *Night symptoms during sleep* | |
| Habitual snoring | Snoring on all or most nights on a regular basis; typically defined as 3 or more nights of week. Note that the loudness of snoring does not necessarily correlate with the severity of OSAS |
| Pauses in breathing | Observed apnoea. These may also be described as episodes of snorting, gasping and choking during sleep |
| Paradoxical chest-abdominal movement | See-saw movement of chest and abdomen during breathing |
| Unusual sleeping positions | Usually hyperextension of neck |
| Restlessness in bed | Increased movement during sleep due to repeated change in position/posture of sleep |
| Diaphoresis | Sweatiness during sleep may be present, related to increased work of breathing and activity in bed |
| Nocturnal enuresis | Children with OSAS have higher risk for secondary enuresis which may resolve when OSAS is adequately treated |
| *Day symptoms* | |
| Mouth breathing and hyponasal speech, dry mouth | Due to adenoidal hypertrophy |
| Morning headaches | Due to carbon dioxide retention |
| Hyperactivity | This is a more common feature of poor sleep in children compared to hypersomnolence which is more common in adults |
| Behavioural changes | Mood swings, irritability, social withdrawal |
| Decreased school performance | Academic performance may deteriorate with untreated OSAS |

## 8.5   Presenting Features

The most common presenting symptom of OSAS in childhood is regular (Habitual) snoring. Some parents may however not report snoring in their child if they sleep in separate rooms (Table 8.2).

## 8.5.1   OSAS in Childhood vs. Adulthood

OSAS in childhood is not simply adult OSAS in a smaller scale. They are quite different in many ways; Adult criteria for OSAS when

**Table 8.3** Comparing childhood and adult OSAS

|  | Childhood OSAS | Adult OSAS |
|---|---|---|
| Age | Peak in preschool age | Increases with age |
| Gender | Generally equal in boys and girls. Some studies have described boys >girls | More in men and post-menopausal women |
| Body weight | Failure to thrive, normal or obese | Usually obese |
| Associated factors | Craniofacial abnormalities | Obesity |
| Daytime hypersomnolence | Usually absent | Present |
| Neurobehavioral | Hyperactivity and developmental delay | Impaired vigilance and cognitive impairment |
| Adenotonsillar hypertrophy | Often present | Rare |
| Airway obstruction | Persistent partial | Cyclical almost complete |
| Arousal | Usually absent. Microarousals may be present | Common, usually occurs at the termination of apnoea episodes |
| Sleep architecture | Usually normal | Decreased SWS |

applied to children would fail to identify serious childhood OSAS and underestimate its severity (Table 8.3).

## 8.6    Diagnosis

The evaluation of a child with suspected OSAS includes a detailed history, physical examination, and appropriate investigations. A prompt and accurate diagnosis is important to ensure timely treatment is instituted, to prevent or avoid complications.

The history would entail details of medical problems, sleep, development and behaviour, as well as family history. Snoring is an important symptom, in addition to the other presenting features in the table above. Habitual snoring (usually defined as three or more night of snoring per week, on a regular basis) should alert the doctor to proceed with a focused evaluation of OSAS.

Physical examination is often normal. Findings of adenotonsillar hypertrophy, systemic and/or pulmonary hypertension, poor growth (or obesity) and other features that may have causative association e.g. craniofacial dysmorphism, hypotonia, or neuromuscular disorders.

Many attempts have been made to use various combinations of symptoms and signs and clinical scores/criteria, but none has been shown to reliably differentiate primary snoring from OSAS.

Some of the more common modalities used in the diagnosis and assessment of suspected OSAS include (Refer to chapter on sleep diagnostics):

1. *Audio and video recordings*
   Studies using various combinations of audio, video and clinical findings have shown a wide range of sensitivity and specificity. Inconsistent findings in different studies from different centres suggest more work needs to be done before these modalities can be effectively used as diagnostic tools in the evaluation of OSAS in children.

2. *Overnight pulse oximetry recording*
   This is a simple and relatively low cost method to screen for desaturation but does not determine the presence of OSAS. Not all respiratory events are associated with or result in desaturation. It also does not detect events that result in arousals before a desaturation occurs. This by itself would be of limited value in diagnosing OSAS.

3. *Home studies (abbreviated polysomnography)*
   Various modalities have been studied to evaluate their utility in the diagnosis of OSAS; these include pulse transit time (PTT), heart rate variability, inductance plethysmography, in various combinations, with or without pulse oximetry, have not been able to demonstrate good correlation with gold standard

overnight laboratory polysomnography. Some of these may be useful as a screening test to determine who needs to proceed with a full overnight polysomnography.

4. *Overnight polysomnography (Sleep Study)*

This is the gold standard for the evaluation of OSAS in children. It is the only diagnostic technique that can quantitate the severity of OSA and related gas exchange abnormalities and sleep disturbances. It can also determine the risk of postoperative complications and also enables comparison for post-intervention evaluations should symptoms persist or recur after treatment. It is therefore important that the polysomnography be performed before any intervention is instituted in a child with OSAS. The polysomnography is also useful for the titration of CPAP in children already diagnosed to have OSAS. Data is however still lacking on identifying which polysomnographic parameter is associated with or predicts morbidity in childhood OSAS. The sleep study in children requires appropriate equipment and trained staff. The results also need to be scored and interpreted using age-appropriate criteria. The availability of such facilities especially in the Asian region is still fairly limited, especially facilities and expertise in the evaluation of paediatric OSAS. The role of nap studies and home studies with current conventional equipment is still limited in the assessment of childhood OSAS.

5. *Drug-induced sleep endoscopy (DISE)*

This is a diagnostic tool to assess the upper airway in conditions that mimic natural sleep. This evaluates the exact area or areas of upper airway collapse during sleep that enables better selection of patients for appropriate surgical intervention. It is performed under sedation to simulate the dynamic state of the upper airway during sleep. Guidelines are being developed on who, where and how to perform DISE, including the modalities for monitoring. It is however important to note that sedation may not induce REM sleep where obstructive events tend to occur, and hence may not be entirely representative of what goes on during a normal night's sleep. As such this modality is unable to provide information for the entire night's sleep. The level of sedation and the technical skill of the endoscopist are essential factors for the success and accuracy of this procedure.

## 8.7   Severity Grading and AHI Classification

The most commonly used criteria for assessment of severity of OSAS is the Apnoea-Hypopnoea Index (AHI) (Table 8.4).

Unlike in adults, obstructive apnoea episodes are not normally common in children and hence the presence of an AHI of 1 or greater is considered abnormal. In older children above 12 years of age, an AHI of >5 may be taken as the threshold at the discretion of the attending doctor, taking into consideration the risk factors for OSAS, symptom presentation, complications as well as the overall condition of the child. It is also noteworthy that limiting our diagnostic assessment to

**Table 8.4**  Classification of severity of OSA in childhood

| OSAS | Normal | Mild | Moderate | Severe |
|---|---|---|---|---|
| AHI (/h) | <1 | 1–5 | >5–10 | >10 |
| SaO$_2$ nadir | ≥92% | 86–91% | 76–85% | ≤75% |
| Peak CO$_2$ (mmHg) | <53 | 55–59 | 60–64 | ≥65 |
| Hypoventilation (EtCO$_2$ > 50) as %TST | <10% | 10–24% | 25–49% | ≥50% |

[a]If SaO$_2$<90% for >10% TST, place in next category
Ref: Carole LM et al., Am Rev. Resp Dis '92;146:1235

a single AHI cut-off would obviously be an over-simplification of this complex condition. It is perhaps necessary to develop a score that depicts the OSAS severity and prognosis, and would likely need to include multiple other parameters and associated comorbidities.

## 8.8    Complications

Childhood OSAS can result in behavioural and cognitive problems, attention deficit hyperactivity disorder, failure to thrive, enuresis, and even systemic hypertension, pulmonary hypertension and cor pulmonale.

Poor growth and failure to thrive is hypothesized to be related to increased work of breathing with increased baseline caloric expenditure. There is also suggestion that decreased production of growth hormone may be related to sleep fragmentation and also contribute to poor growth. Growth velocity increases after treatment of OSAS.

Enuresis may be a result of increased urine production from hormonal dysregulation associated with increased levels of catecholamines and frequent arousals. Enuresis often resolves with appropriate treatment of OSAS.

Behavioural and cognitive deficits has been described in children with OSAS. Snoring is also described to be associated with poorer academic performance. Sleep deprived children often exhibit hyperactivity and restlessness, more so than daytime hypersomnolence. Intermittent nocturnal hypoxia together with frequent arousals result in sleep fragmentation and these may lead to the development of neurobehavioral consequences. Reports have suggested that these deficits do improve with successful treatment of OSAS, although some may not fully resolve. More recent reports have demonstrated no improvement in cognitive function, through structured testing, in pre-school and primary school aged children after adenotonsillectomy, There is therefore concern that long term residual cognitive deficits may be a consequence of delayed diagnosis and treatment of childhood OSAS.

Cardiovascular complications of systemic hypertension and even pulmonary hypertension and cor pulmonale may result as a consequence of OSAS in childhood. Primary snoring itself has been described to be associated with elevation in systemic hypertension even in the absence of significant OSAS based on current polysomnographic criteria. These complications are fortunately less frequently seen with more awareness and earlier diagnosis and treatment of OSAS.

Sleep is essential to the body's reparative process and maintenance of overall health; untreated OSAS may be associated with poor sleep quality and resultant metabolic sequelae, including glucose metabolism disorders and the risk of developing diabetes. This together with cardiovascular risks may result in higher risks of strokes and heart attacks later in life, contributing to a reduced lifespan in adulthood. There is also demonstrable increase in healthcare utilisation in children with OSAS; particularly so in those younger than 5 years old. The severity of the OSAS also correlates directly with the total annual costs and independent to age. OSAS in childhood has also been associated with decreased health-related quality of life, which improves after adenotonsillectomy.

## 8.9    Treatment

### 8.9.1    Adenotonsillectomy (T&A)

Adenotonsillectomy is usually the first-line treatment in childhood OSAS in the presence of adenoid and tonsillar hypertrophy.

In otherwise well and healthy children, OSAS usually resolves in up to 75% to 100% after T&A. Persistent OSAS after T&A has however been reported to occur in up to 13 to 29% of children defined as low-risk patients, while it may persist in up to 75% in higher-risk patients such as obese children.

In the Childhood Adenotonsillectomy Trial (CHAT) study, children with mild to moderate OSAS (AHI ≤5) who underwent early T&A showed normalisation of polysomnography in 79% compared to 46% in the group with watchful waiting with supportive care. There was also significant reduction in symptoms and improvement in behaviour and quality of life in the treatment group.

Adenotonsillectomy for OSAS may present complications in some children; these include pulmonary oedema, hypoxaemia and bronchospasm. These are fortunately uncommon but those who may be at higher risk should undergo close monitoring peri- and post-operatively. The risk factors include:

1. Young age—especially below 3 years old
2. Severe OSA confirmed on polysomnography
3. Presence of cardiac complications e.g. Right ventricular hypertrophy
4. Failure to thrive
5. Significant obesity
6. Presence of craniofacial anomalies
7. Neuromuscular disorders

Source: American Academy of Pediatrics, Clinical Practice Guidelines. Diagnosis and management of childhood OSAS.

It is generally recommended that removal of both the adenoids and tonsils should be performed to avoid recurrence of symptoms even if one appears to be predominant. In addition, tonsillotomy (or partial tonsillectomy) has been shown to be have a much higher risk (more than three-fold) of OSAS recurrence compared to tonsillectomy.

In patients with concomitant enlarged nasal turbinates, the addition of radiofrequency reduction of the inferior turbinates during adenotonsillectomy has been shown to improve the AHI. However, the duration of effectiveness is variable and the therapy may need to be repeated if the turbinate hypertrophy recurs.

Patients with additional risk factors of OSAS such as obesity may still benefit from T&A but the relative risk and benefit should be evaluated by the attending doctor. There may be residual OSAS even after surgery.

OSAS symptoms may take up to 4 to 6 weeks to resolve after a T&A as there may be postoperative oedema. If there are persistent or residual symptoms, especially in the presence of additional risk factors, a follow-up sleep study is indicated, at least 6 weeks post T&A.

### 8.9.2 Positive Airway Pressure Therapy (CPAP/BiPAP)

Positive airway pressure serves to stent the airway open and overcome dynamic upper airway obstruction. Studies have shown that it improves polysomnographic findings in OSAS and also significantly improves symptoms and neurobehavioral function.

This may be considered for patients who are not surgical candidates for T&A or those with persistent OSAS post-T&A. It may also be a transitory management modality for those with severe OSAS while awaiting surgery, or for stabilisation of condition prior to T&A, with the aim to reduce the post-surgical complication risks.

Two modalities of positive airway pressure therapy can be used. There is no evidence that one is superior to the other and the practice and preference varies between different institutions. The settings should be set through a polysomnographic titration and long-term follow-up is necessary as the pressure settings may change over time as the child grows.

One of the main challenges with positive airway pressure in children is the interface—the size, shape and fit significantly impacts on the comfort, acceptance and hence compliance to use. Most masks are developed in western countries and not primarily designed to fit the Asian facial contours. Also, the range of masks available in some parts of Asia may be limited, contributed by the small market and few distributors as well as relative high cost.

Excessive leaks around the mask can compromise the effectiveness of positive airway pressure and also contribute to patient-ventilator dyssynchrony. Mouth breathing can be alleviated by the use of a chin strap. Dryness in the airway can be reduced with the use of a heated humidifier. Long term adverse effects of the mask include mid-facial hypoplasia and local skin irritation as well as pressure sores. Careful selection and training of caregivers in the appropriate application of the masks are vital in ensuring good outcomes which in turn positively reinforces compliance.

High-flow nasal cannula (HFNC) has been shown to reduce respiratory events, improve oxygenation, reduce heart rate, and may be effective for CPAP-intolerant children with moderate to severe OSA.

### 8.9.3 Weight Management

Weight loss can be significantly therapeutic in children with obesity and OSAS, in addition to its general health promoting effects. Many studies have demonstrated significant improvement in OSAS symptoms and polysomnographic criteria with weight loss; decreases in BMI z scores are associated with significant corresponding improvements in AHI values. Weight loss should be recommended as part of the management plan if the child is overweight or obese.

Adherence to weight loss programmes is always the biggest challenge in the young; multimodality interventions are often required, including dietary restrictions, physical exercise and psychological support.

### 8.9.4 Medical Treatment

- Topical intranasal corticosteroids
  Corticosteroids can reduce adenotonsillar tissue inflammation and proliferation in invitro models. They may be helpful in mild OSAS in lieu of T&A or in patients with residual mild OSA after T&A. Various topical ste-

roids have been used for 4 to 6 weeks, showing a sustainable effect for up to 8 weeks or even several months. The optimal dosage and duration of treatment are however still unclear.

- Leukotriene receptor antagonists
  Treatment over 16 weeks has been shown to be able to reduce the AHI by a small margin of between 1 to 5/h.

  Combination anti-inflammatory medications with topical nasal steroids and Montelukast has been shown to improve AHI by a margin of over 3.6/h over a 12-week period in post-adenotonsillectomy children with residual mild OSAS.

### 8.9.5 Others

- Oral appliances
  Mandibular advancing devices and rapid maxillary expansion devices may be useful in selected patients. These are generally used in adults and there have only been limited studies in childhood OSAS. Further studies are needed to determine the clinical indications and patient selection for optimal intervention with these and other oral appliances in childhood OSAS.
- Myofunctional therapy
  Myofunctional therapy aims to train patients to improve labial seal, lip tone and the use of nasal breathing as well as to promote favourable positioning of the tongue within the oral cavity. It involves a structured re-education programme of specific oropharyngeal exercises performed daily. Data suggests some improvement in AHI although there is much heterogeneity in interventions, and the results are marginal. This treatment itself has few complications and is certainly non-invasive, but does require cooperation from the child and compliance to the exercise routine over extended periods of time. Further studies are warranted before a formal recommendation can be made on its use as treatment modality in childhood OSAS (Fig. 8.2).
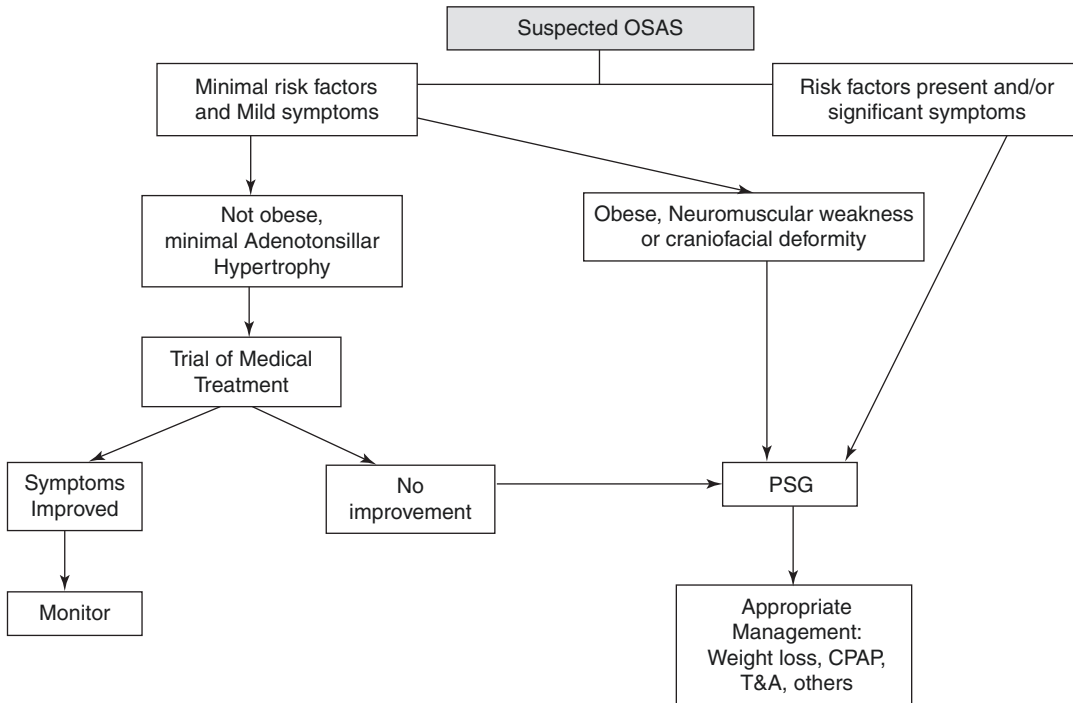
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                        │   Suspected OSAS    │
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        ┌──────────────────────────┐      ┌──────────────────────────┐
        │   Minimal risk factors   │      │  Risk factors present and/or │
        │    and Mild symptoms     │      │   significant symptoms    │
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        │       Not obese,         │      │ Obese, Neuromuscular weakness │
        │   minimal Adenotonsillar │      │    or craniofacial deformity  │
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   │  Symptoms  │        │    No      │            │   PSG    │
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   │  Monitor   │                             │    Appropriate     │
   └────────────┘                             │    Management:     │
                                              │ Weight loss, CPAP, │
                                              │    T&A, others     │
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**Fig. 8.2**  Management Algorithm for Childhood OSA

## 8.10   Clinical Course and Prognosis

Most children with snoring do improve beyond the peak age of adenotonsillar hypertrophy, after 4 to 8 years old. The persistent of symptoms and that of OSAS may be determined by the chronicity and severity of OSAS as well as the presence of risk factors, such as obesity.

Children with OSAS that is treated, may recur with OSAS later in adulthood if there is development of additional risk factors such as obesity.

Complications of childhood OSAS such as learning and behavioural problems do often improve with treatment, but the course may be determined by the severity and also delay in treatment of the OSAS; some studies have suggested that some of the sequelae may not be totally reversible if treatment is delayed.

OSAS in childhood, especially if severe and untreated, may be a risk factor for the development of chronic diseases later in adulthood; these can include hypertension, diabetes, coronary heart disease and even strokes.

## 8.11   Some Potential Research Areas

• Development of diagnostic and screening tools for childhood OSAS

The availability of low-cost, high-sensitivity and high specificity modalities for the diagnosis and assessment of OSAS would be useful especially in developing countries where the current gold-standard overnight polysomnography is not easily available, and where wait-times for a sleep study can be very long. These could also be used as screening

tools to determine who needs to be referred for a formal polysomnography.

- Evaluation of newer imaging techniques in guiding treatment choices, pre- and post-surgery assessment and management.
- Identification of potential biomarkers as correlates of disease severity and morbidity to guide management and follow-up.
- Studies on the prevalence and sequelae of OSAS in different parts of the world, using a common protocol to better compare data across different sites.
- Long term clinical course of childhood OSAS and its relationship with OSAS in adulthood, as well as the chronic diseases in adulthood, such as hypertension, diabetes, coronary heart disease and stroke.

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