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075

a0005 # Obstructive sleep apnoea in childhood

HELEN M CAULFIELD

s0005 ## SEARCH STRATEGY

p0005 The content of this chapter is supported by a Medline search using the key words obstructive sleep apnoea, sleep-disordered breathing, child, paediatric, snoring, sleep disorders, heart failure, pulmonary hypertension, polysomnography and focusing on definitions, symptoms, sleep studies and outcomes of intervention.

s0010 ## DEFINITIONS AND EPIDEMIOLOGY

p0010 Obstructive sleep apnoea (OSA) is characterized by episodic partial or complete obstruction of the upper airway during sleep. This causes apnoea or cessation of breathing. Intermittent episodes of brief cessation of breathing may be physiological. An 'apnoea' is defined in adults as cessation of breathing for ten seconds or more. Six seconds or less may be pathological in children. The differences in sleep physiology between adults and children have bedevilled attempts to define OSA in children with the same precision as in adult medicine.[1, 2] Upper airway obstructions during sleep affect pulmonary ventilation and can lead to a drop in peripheral oxygen saturation (hypoxaemia) and retention of carbon dioxide (hypercarbia). Upper airway obstruction disrupts normal sleeping patterns by causing arousals, presumably induced by the effect of hypoxaemia and hypercarbia on the respiratory centre. It is now recognized that sleep-disordered breathing is a spectrum of airway obstruction, and the term encompasses simple snoring, upper airway resistance syndrome (UARS) and the more severe OSA.

Simple snoring

s0015

p0015 Simple snoring is defined as snoring without obstructive apnoeas, frequent arousals or gas exchange abnormalities. It is generally considered benign although there is growing evidence that it may not be as innocuous a condition as has been believed. In addition to evidence that the secretion of growth hormone is reduced in children with simple snoring,[3] cohort studies of children with habitual snoring suggest they have a higher incidence of neurocognitive disorders, with demonstrable adverse effects on quality of life. Furthermore, these effects are reversible by adenotonsillectomy, and it may be that we

need to revise our criteria for intervention in this group of children.[4]

s0020 ## Upper airways resistance syndrome

p0020 Upper airways resistance syndrome is a more subtle form of sleep-disordered breathing than OSA. It is characterized by partial upper airway obstruction. The frequency and severity of apnoeas is insufficient to warrant a diagnosis of OSA. UARS can lead to significant clinical symptoms as a result of night-time arousals and pulmonary hypoventilation.[5] This disorder is more common than OSA but is often underdiagnosed.[6] [****/***]

s0025 ## Obstructive sleep apnoea

p0025 OSA causes loud persistent snoring interrupted by gasping or choking episodes and silent periods which are apnoeas. It causes significant sleep disruption. This can lead to daytime neurobehavioural problems such as an increase in total sleep time, hyperactivity, irritability, bed-wetting and morning headaches.[7] OSA may significantly affect a child and their family's quality of life.[8] [***]

p0030 Untreated OSA can result in significant morbidity such as failure to thrive, pulmonary hypertension and right heart failure.[9] Failure to thrive is not common in these times but children with OSA tend to have a growth spurt following adenotonsillectomy.[3] Although overt right heart failure occurs less frequently, asymptomatic degrees of pulmonary hypertension may be common.[10]

p0035 Difficulties concerning definition mean that the exact prevalence of sleep-disordered breathing in children is unknown but may be as high as 11 percent,[11] and it affects between 26 and 65 percent of all children attending otolaryngology clinics in the UK.[12] OSA has a prevalence of approximately 2 percent in the paediatric population and is more common in African children.[13, 14] Sleep-disordered breathing can affect children of all ages but its peak incidence is between the ages of three and seven, when the adenoid and tonsillar lymphoid tissue is disproportionately large relative to the pharyngeal airway.[15] There is an equal incidence in boys and girls but it presents earlier in boys.[13]

p0040 Factors that increase the risk of OSA in otherwise healthy children include adenotonsillar hypertrophy, family predisposition, obesity and mild craniofacial disproportion.[11, 14] OSA is also associated with a number of craniofacial syndromes and systemic diseases, in which it has a much higher incidence than in otherwise healthy children (**Table 075.1**). This is a result of craniofacial disproportion and the presence of coexisting upper airway hypotonia. The site of upper airway obstruction is not confined to the adenotonsillar area in these children, and requires investigation.[16] [***/**]

Table 075.1 Children with increased risk of OSA. t0005

Craniofacial syndromes	Systemic illness
Pierre Robin sequence	Cerebral palsy
Crouzon's syndrome	Myotonic dystrophies
Goldenhar syndrome	Obesity
Treacher Collins syndrome	Sickle cell disease
Apert's syndrome	Glycogen storage disorder
Down's syndrome	Achondroplasia
Hunter's syndrome	
Velocardiofacial syndrome	
Beckwith–Wiedemann syndrome	

s0030 ## MECHANISM OF SLEEP-DISORDERED BREATHING AND PHYSIOLOGICAL AFFECTS

p0045 Breathing against a partially or completely obstructed upper airway causes the clinical signs of sleep-disordered breathing, which include loud snoring, increased respiratory effort with flaring of the nostrils, suprasternal and intercostal recession. Complete obstruction of the pharyngeal airway, as in OSA, leads to silent periods followed by choking or gasping as the child rouses from sleep to re-establish their airway.

p0050 Partial (hypopnoea) or complete upper airway obstruction (apnoea) during sleep can lead to hypoxia and hypercarbia. The degree of hypoxia is influenced by the duration of the apnoeic event, the condition of the cardiopulmonary system and whether a coexisting neuromuscular disorder is present. Hypoxia leads to a rise in sympathetic output causing peripheral vasoconstriction, which results in tachycardia and a rise in blood pressure. Changes in the pulse and blood pressure reflect increased sympathetic activity and are markers of subcortical arousal. They correlate directly with the severity of the sleep-disordered breathing and can be used to quantify it. The diastolic blood pressure during rapid eye movement (REM) sleep shows a significant correlation with the number of apnoeas and hypopnoeas.[17] The pulse transit time is a noninvasive marker of blood pressure. It is the interval between the R wave on an electrocardiogram (ECG) and the arrival of the pulse at the finger. The pulse transit time is increased in the presence of increased respiratory effort and decreased in the presence of tachycardia associated with arousal. It is a more sensitive measure of obstructive events than visible electroencephalogram (EEG) arousals found on full polysomnography recording.[18]

p0055 Upper airway obstructive events are terminated by these subcortical arousals. The child is unaware of these but they may occur several hundred times a night. Repeated arousals greatly disturb a child's sleep pattern which can lead to changes in behaviour and concentration ability as well as having general effects on a child's quality of life.[4, 5] The rise in pulmonary blood pressure as a result

of increased sympathetic activity leads to transient pulmonary hypertension. Long-standing sleep apnoea can result in irreversible pulmonary hypertension and if sustained this will lead to right heart failure and cor pulmonale.[9]

p0060 A gradual, improved understanding of these physiological changes that occur during sleep has helped clinicians identify the parameters that need to be measured to ascertain the severity of the sleep-disordered breathing. Full polysomnography is the 'gold standard' investigation for sleep disorders. It can differentiate sleep-disordered breathing from other types of sleep disorder, such as periodic leg movement and narcolepsy. However, if sleep-disordered breathing is suspected, full polysomnography can be substituted by less elaborate sleep studies which measure and analyse pulse rate and oxygen saturation complemented with video and sound recordings of the sleeping child, for example the Visilab^{TM} analysis. [***]

s0035 CLINICAL SYMPTOMS

p0065 The cardinal symptom of sleep-disordered breathing in children is snoring. Severe sleep apnoea with loud, persistent snoring is more likely in younger children. Children with significant obstruction sweat during sleep, particularly in the nuchal area, and have a tracheal tug and intercostal recession with loud stertorous breathing. Children who have repeated apnoeas leading to arousals are very restless at night, often adopting unusual sleeping positions in an attempt to relieve their upper airway obstruction, and are irritable on waking. They may also complain of very dry mouth and morning headaches. The high intrathoracic pressures generated in a child with OSA may cause oesophageal reflux leading to unexplained regurgitation or vomiting during sleep.

p0070 A further commonly witnessed symptom is choking episodes. This is best enquired about by imitating a guttural noise, as parents are often not aware of why the noise is being created. Parents often describe witnessed apnoeas as momentary breath holding. Most parents, if asked directly, will deny that their child stops breathing at night. This can lead to an underestimation of the presence of sleep apnoea if the clinical history is not taken with enough attention to detail.

p0075 True daytime somnolence as described in adults is unusual in children. Hyperactivity is more common and affected children are often irritable on waking.[7]

p0080 It is also important to identify whether the affected child has associated rhinosinusitis as this can exacerbate the sleep-disordered breathing. Rhinosinusitis may be allergic, infective or structural in origin. Medical treatment of this will improve the child's sleep-disordered breathing. Similarly, pulmonary disease may also exacerbate sleep apnoea by causing increased work in breathing requiring higher intrathoracic pressures. The child with severe OSA has chronic hypoventilation at night, which may lead to pulmonary atelectasia. These children develop a cough with most viral upper respiratory tract infections. A history of previous hospital admission with acute airway compromise heralds an increased risk of complications following surgical intervention.

CLINICAL EXAMINATION s0040

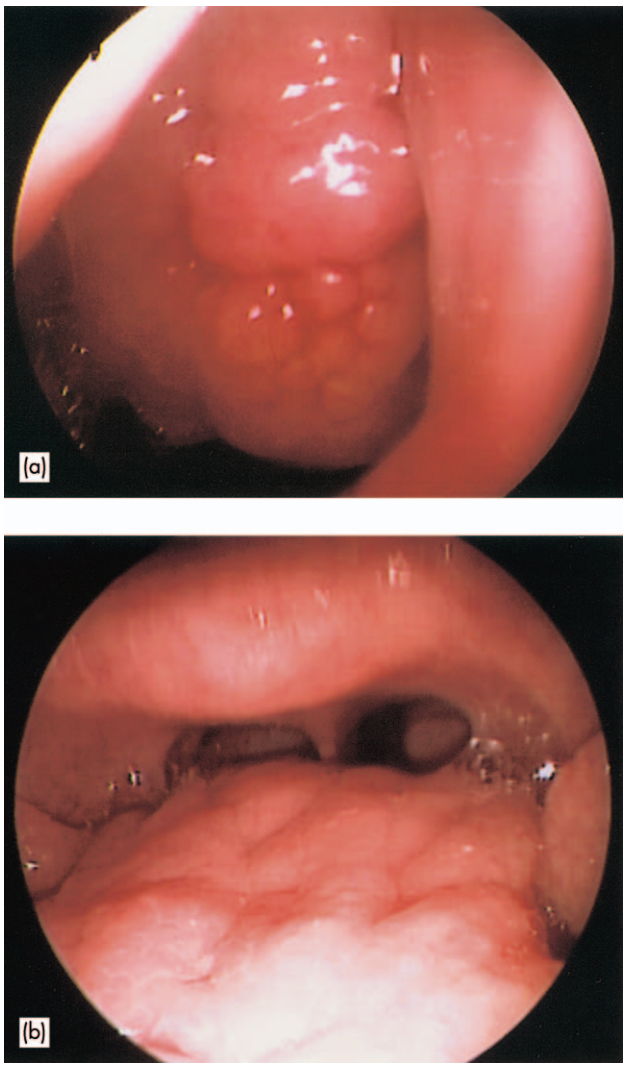
p0085 Clinical examination should include a full ENT examination, height and weight measurements and cardiopulmonary examination. Sleep-disordered breathing is more common in the obese child. Conversely, sleep-disordered breathing in a child can cause failure to thrive or a decrease in growth rate.

p0090 Subtle craniofacial disproportion should be looked for. Children with a triangular chin, steep mandibular angle, retrognathia, narrow high-arched palate and long soft palate are likely to have sleep-disordered breathing. Mouth breathing indicates nasal obstruction, and examination within the nose should look at the structure of the septum and the quality of the nasal lining for the presence of rhinosinusitis. Many children with sleep-disordered breathing are daytime mouth breathers and therefore the presence of chronic mouth breathing should raise the index of clinical suspicion.

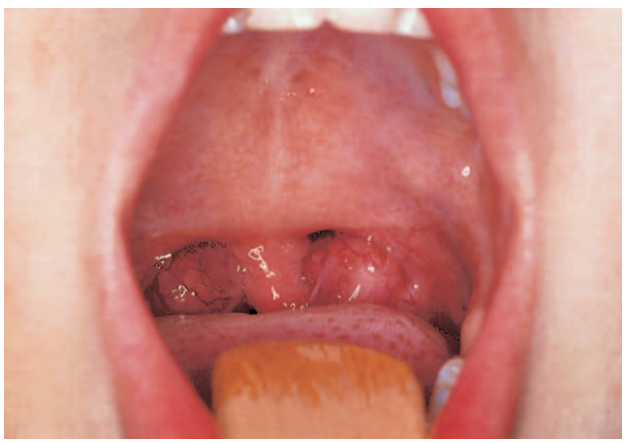
p0095 Hyporesonant voice points to enlarged adenoids. In some children it may be possible to perform nasendoscopy with a fibre optic endoscope to ascertain the size of the adenoid pad and extent of choanal obstruction (**Figure 075.1**). Examination within the oral cavity should exclude a submucous cleft and be used to document the size of the tonsils (**Figure 075.2**). Although most children with OSA have large tonsils, studies show that there is little correlation between the size of the tonsils and the severity of sleep-disordered breathing.[19] Small tonsils can cause upper airway obstruction if there is excessive medialization of the lateral pharyngeal walls during sleep. This can be the result of poor neuromuscular control of the pharyngeal airway, as in cerebral palsy, and previous cleft palate repair. Alternatively, a very large adenoid pad can cause a marked reduction in the calibre of the airway even in the presence of relatively small tonsils. Also, the site of upper airway obstruction is not necessarily adenotonsillar, particularly in syndromic children.[16]

p0100 Daytime stertorous breathing is indicative of severe sleep apnoea, as is growth retardation, which has been shown to be due to a reduction in growth hormone secretion.[3, 20] The presence of broken veins on the face indicates chronic nocturnal hypercarbia. Pectus excavatum can result from long-standing intercostal recession during sleep. The presence of noisy breathing, other than stertor, should alert the clinician to the presence of possible distal airway pathology.

p0105 Clinical history and examination will confirm the presence of sleep-disordered breathing but cannot reliably distinguish between children with simple snoring and



f0005 **Figure 075.1** **(a)** A nasendoscopic view of the adenoids in a child showing >50 percent choanal obstruction. **(b)** An endoscopic view of the same adenoids using a 90° rigid fibreoptic telescope introduced through the mouth.



f0010 **Figure 075.2** An intraoral photograph of a seven-year-old child showing large obstructive tonsils.

OSA. On the whole, the clinical impression is that mild sleep apnoea is overdiagnosed and severe OSA is underdiagnosed. There is a number of reasons why the history can be misleading. Children with OSA experience most of their obstruction during rapid eye movement sleep (REM sleep), which occurs primarily in the early hours of the morning when parents are not observing them.[21] Children with UARS have a pattern of partial upper airway obstruction which leads to gas exchange abnormalities rather than true apnoeas and may be underreported.[6]

p0110 The history and clinical examination will enable the clinician to decide which child needs further investigation but it cannot always determine the need for intervention. An accurate diagnosis is, therefore, needed to ensure appropriate treatment, avoid unnecessary treatment and identify children at risk of developing complications of treatment. [***/**]

INVESTIGATIONS

s0045

p0115 Investigations are needed in paediatric sleep-disordered breathing to:

- p0120 identify children who are at increased risk of the complications associated with sleep-disordered breathing;
- p0125 avoid unnecessary intervention in children with simple snoring;
- p0130 identify children who are at increased risk of complications following surgical intervention;
- p0135 identify the site of upper airway obstruction in a child with sleep-disordered breathing.

Sleep studies

s0050

p0140 The clinical history and examination will identify most children with sleep-disordered breathing but are not sensitive enough to define severity or differentiate between simple snoring and more severe forms of sleep-disordered breathing. Symptom questionnaires designed to complement the clinical history are good at identifying more severe cases.[22] However, more objective evidence of the severity of sleep-disordered breathing may be sought.

p0145 Pulse oxymetry is a screening tool. It relies on indirect measurement of the arterial oxygen saturation using a probe, usually applied to the finger.[23] It is minimally invasive, and can be undertaken in a child's home if a recording device is available. Readings above 97 percent exclude serious hypoxia or hypercarbia.[24] Readings below 87 percent may suggest coexistent cardiac or broncho-pulmonary disease. Between these two extremes it gives little information as to the severity of the sleep apnoea and will miss apnoeic episodes not associated with oxygen desaturation.[25] However, despite these shortcomings, pulse oxymetry does provide some information regarding

t0010 **Table 075.2** The apnoea/hypopnoea index.

Apnoea/Hypopnoea index (per hour)	Level of OSA
5–20	Mild
20–40	Moderate
>40	Severe

the severity of OSA, as it will identify severe cases with significant desaturations. It has a high positive predictive value of approximately 97 percent. It is not so helpful in assessing mild-to-moderate OSA, with a low negative predictive value of approximately 47 percent.[26]

p0150 One step up from simple pulse oxymetry is a 'mini-sleep' study system. These systems combine pulse oxymetry with video footage and sound recording. Computer software measures movement from the video, snoring from the microphone and analyses the pulse oxymeter output. The best known is the 'Visilab'. Increased work of breathing (hypopnoeas) and apnoeic episodes without significant oxygen desaturation can be detected using this form of sleep study. A rise in pulse rate associated with a desaturation signifies an arousal and the number of desaturations corresponds to the number of apnoeas and hypopnoeas. These data are more useful in adult sleep medicine and age-appropriate criteria for children are not yet standardized.[2]

p0155 The American Sleep Apnoea Association currently grades sleep apnoea in the form of the apnoea/hypopnoea index. This is the total number of apnoeas and hypopnoeas divided by the total sleep time, which gives an index per hour (**Table 075.2**).

p0160 The gold standard investigation for sleep disorders is full polysomnography. This monitors EEG activity, chest and abdominal movement, oxygen saturation, nasal or oral airflow, end tidal carbon dioxide and continuous ECG recordings. Video monitoring during polysomnography is also helpful but is not standard. Full polysomnography identifies EEG arousal and provides an apnoea/hypopnoea index. It also differentiates between different types of sleep disorder and identifies central apnoeas.

p0165 Although ideal, full polysomnography is detailed and expensive and cannot be provided for every child suspected of suffering with sleep-disordered breathing. There is, therefore, the need for rationalization of sleep study investigations depending on the clinical needs of the child (**Table 075.3**). [Grade B]

s0055 Sleep nasendoscopy

p0170 Although polysomnography is invaluable and essential in documenting the severity of OSA it provides no information concerning the site of upper airway obstruction. Ascertaining the site of upper airway obstruction is essential in children with obesity, small tonsils or craniofacial disproportion and in those with syndromes and neuromuscular disease. The reason for this is that the site of upper airway obstruction is not necessarily adenotonsillar and may be anywhere from the palate to the supraglottis.[16] The site of upper airway obstruction varies not only between children with different syndromes but also between children with the same syndrome.

t0015 **Table 075.3** Recommendations for investigation.

Which child?	Type of investigation
Healthy child >3 years	No investigations necessary
Healthy child >3 years with increased risk of post-operative respiratory complications	Chest x-ray and ECG Visilab study
Healthy child <3 years	Chest x-ray and ECG Overnight pulse oxymetry
Healthy children with: obesity; small tonsils; mild cranofacial disproportion	Visilab study Sleep nasendoscopy Microlaryngobronchoscopy Post-intervention sleep study
Neuromuscular disease	Full polysomonography Sleep nasendoscopy Post-intervention sleep study

p0175 Direct visualization of the upper airway using a flexible fibre-optic endoscope in children with obstructive 'awake' apnoea has proved to be very accurate in diagnosing the site of airway obstruction.[27] It has also become the first-line investigation in the management of children with stridor. The disadvantage with 'awake' flexible endoscopy is that it provides little information regarding the site of upper airway collapse that occurs when the child is asleep.

p0180 Sleep endoscopy using a flexible fibre-optic endoscope (sleep nasendoscopy) was first described by Croft and Pringle,[28] and is routinely used to assess the site of snoring and airway obstruction in adults. In children, sleep nasendoscopy can be performed with the child breathing spontaneously a mixture of halothane and oxygen. This is thought to provide a relaxation of the upper airway musculature that mimics natural sleep.[16]

p0185 Sleep nasendoscopy should be performed in the operating theatre with a skilled paediatric anaesthetist and full cardiopulmonary monitoring. The sites of upper airway obstruction can be documented using a four-level classification system (**Figure 075.3**). The site or level at which the upper airway obstruction is occurring will define the intervention that is needed to correct it. To classify the site of obstruction the upper aerodigestive

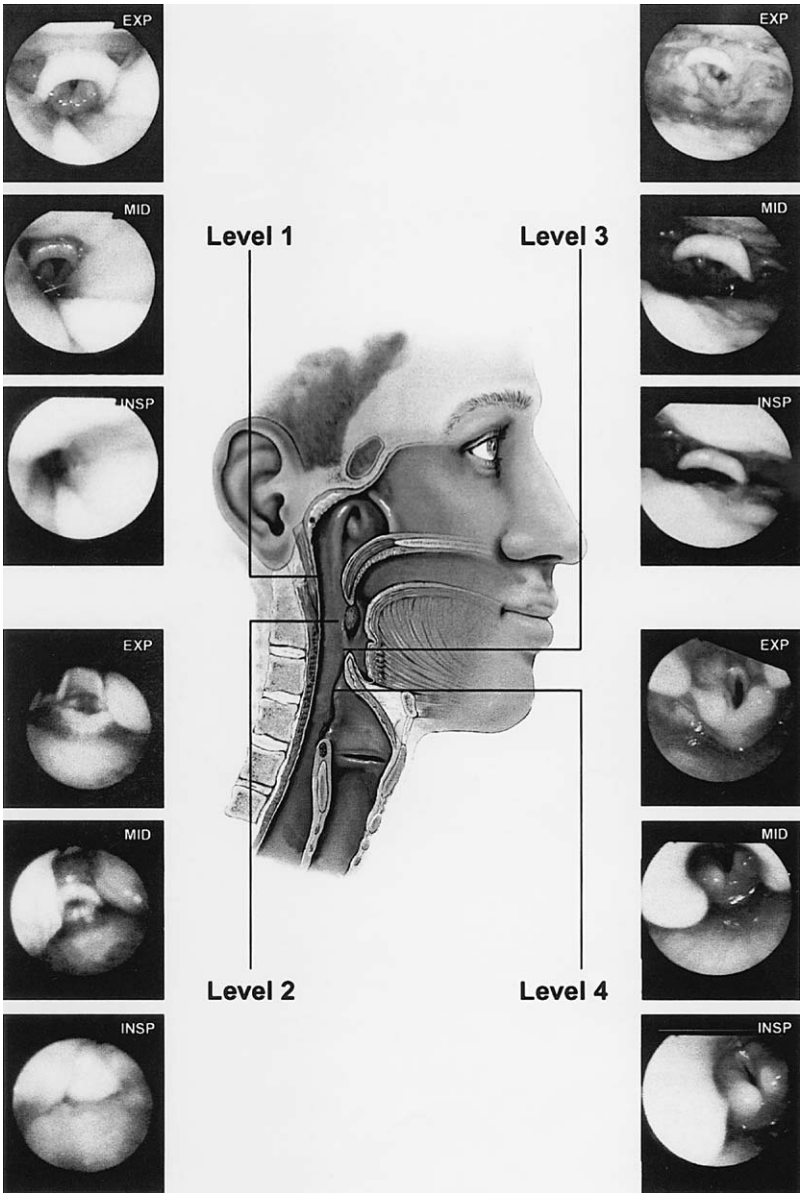


Figure 075.3 A classification system for documenting the findings at sleep nasendoscopy. The photographs show the endoscopic view of the obstruction at each level from 1 to 4. f0015

tract can be divided into the following four functional levels:

p0190 • level 1 or adenoid pad and velopharyngeal obstruction;
p0195 • level 2 or tonsillar obstruction;
p0200 • level 3 or tongue base obstruction;
p0205 • level 4 or supraglottic obstruction. [Grade B]

s0060 ## Rigid laryngobronchoscopy

p0210 Formal rigid laryngobronchoscopy should be performed in the assessment of syndromic children with complex obstructive breathing and in children who have a history of prematurity and prolonged inhalation on a neonatal intensive care unit.[16] It is important to exclude the presence of pathology distal to the glottis that may be exacerbating the upper airway symptoms, for example subglottic stenosis, tracheomalacia, innominate artery compression, bronchomalacia or vascular rings. The incidence of distal airway pathology is higher in Down's syndrome patients than in the general population. In one study, approximately 28 percent of children with Down's syndrome and sleep-disordered breathing were found to have laryngeal or tracheal abnormalities on rigid laryngobronchoscopy.[29] The incidence of distal airway pathology in other syndromes is unknown but laryngeal abnormalities have been described in Goldenhar syndrome,[30] and major aortic arch abnormalities in velocardiofacial syndrome can cause tracheal compression.[31] [Grade C]

s0065

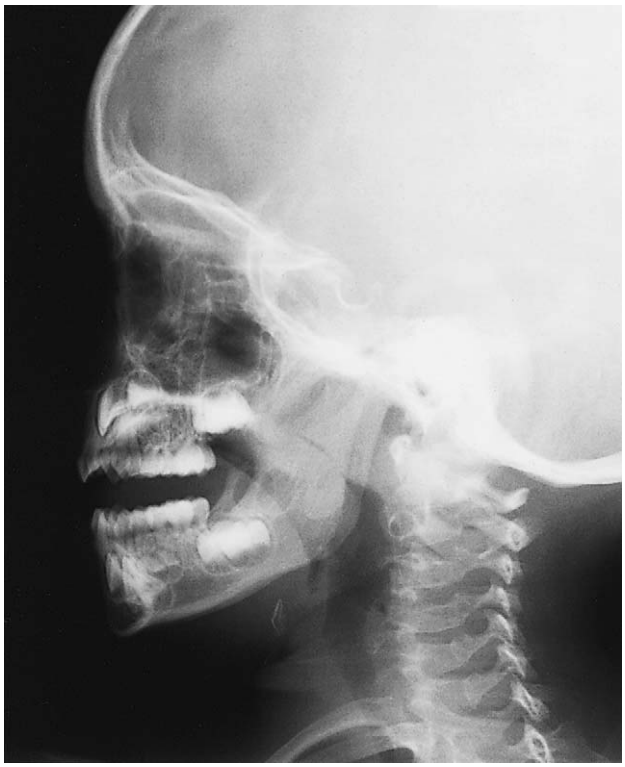
Imaging

p0215 A chest x-ray is mandatory in all children with moderate-to-severe OSA. It will identify pulmonary hypertension and right ventricular hypertrophy causing cardiomegaly. A chest x-ray also detects atelectasia caused by chronic hyperventilation. A lateral x-ray of the post-nasal space is useful in ascertaining adenoid size in children in whom flexible nasendoscopy has not been possible (**Figure 075.4**). Cardiac echocardiography is needed to exclude pulmonary hypertension or right-sided heart failure if EEG or chest x-ray are suggestive. Cardiac catheterization may then be necessary to detect pulmonary venous pressures in severe cases. Cephalometry, fluoroscopy and dynamic magnetic resonance imaging have been investigated in the assessment of OSA in children but have a limited role.[32] [Grade B]

s0070

RECOMMENDATIONS FOR INVESTIGATIONS

p0220 In otherwise healthy children over three years old with a history compatible with OSA and large tonsils and adenoids on examination, further investigation of their sleep-disordered breathing is not essential prior to adenotonsillectomy unless they are suspected of having severe OSA. In such cases, pulse oxymetry or a Visilab study should be undertaken. A chest x-ray should be performed to look for atelectasia and an ECG to look for signs of right ventricular hypertrophy or pulmonary hypertension.



f0020 **Figure 075.4** A lateral soft tissue x-ray of the head and neck of a three-year-old child with significant obstruction of the nasopharyngeal airway by enlarged adenoids.

p0225 Otherwise healthy children under three years of age and other 'at risk' children (listed in **Table 075.1**) are likely to have severe OSA and should have a chest x-ray, ECG and a Visilab study.

p0230 Children who are clinically obese, have small tonsils and adenoids on examination or have mild craniofacial disproportion need further investigation to ascertain the severity of the sleep-disordered breathing and the site of upper airway obstruction. This is because adenotonsillectomy may not be curative, and an outcome measure is needed. A Visilab study would provide this. Sleep nasendoscopy is needed to confirm the site of upper airway obstruction prior to adenotonsillectomy so that upper airway obstruction at other sites can be identified and appropriate intervention can be planned.

p0235 Syndromic children and those with neuromuscular disease need investigation with full polysomnography as they are likely to have complex upper airway obstruction, may have associated central apnoeas and ongoing problems throughout their childhood despite intervention. Sleep nasendoscopy is needed to confirm the site of upper airway obstruction in these children if site-specific treatment is sought. [Grade B]

p0240 See also **Table 075.3**.

s0075

TREATMENT

s0080

Medical treatment

p0245 Children with OSA who present with mucopurulent nasal discharge should have this treated, as it will improve their nocturnal symptoms. Treatment should consist of a four- to six-week course of systemic antibiotics combined with topical intranasal or systemic steroids. These children should be tested for sensitivity to airborne allergens that may be irritating the mucosal lining of the nose and paranasal sinuses.

p0250 Children with severe OSA, who have a persistent cough or signs of pulmonary atelectasia on chest x-ray, should be given systemic antibiotics and systemic steroids prior to a general anesthetic and have preoperative chest physiotherapy. This will help clear bronchial secretions and improve pulmonary function, thereby reducing the risk of post-operative respiratory failure. [Grade D]

s0085

CONTINUOUS POSITIVE AIRWAY PRESSURE

p0255 Continuous positive airway pressure (CPAP) provides continuous insufflation of the nasopharyngeal airway during sleep, thereby splinting the airway and maintaining its patency. It is usually delivered via a nasal mask and has to be used every night on an indefinite basis. It is a

long-term therapy and requires frequent assessment of compliance and efficacy by a sleep physician. The CPAP pressures are titrated using polysomnography in the sleep laboratory and are periodically adjusted thereafter. Recent work with autotitrated CPAP in children has shown promise.[1] Patients such as the obese child, syndromic children and children with cerebral palsy may not be considered surgical candidates and may benefit from long-term CPAP.[33] CPAP can be started from birth and is well tolerated in older children. CPAP can also be used prior to planned surgical intervention in cases of severe OSA to minimize post-operative risk by improving pulmonary ventilation. [Grade B]

s0090 ### NASOPHARYNGEAL AIRWAY

p0260 Pharyngeal airway obstruction may be dramatically relieved with the use of a nasopharyngeal airway. This is of particular use in the newborn and in the first few months of life. It has been traditionally used in children with Pierre Robin sequence but also has a role in children with Apert's and Crouzon's syndrome. The length of the nasopharyngeal airway can be tailored according to the site of airway obstruction. In Pierre Robin sequence the tip of the nasopharyngeal airway needs to lie just above the epiglottis and in Apert's syndrome to the free edge of the soft palate. Many children with Pierre Robin sequence improve in the first few months of life, and the nasopharyngeal airway may be sufficient treatment of their upper airway obstruction. A sleep study can be performed with the tube *in situ* to assess its effectiveness, and another study performed on planned removal of the airway a few months later to assess progress. [Grade C]

s0095 ### MANDIBULAR ADVANCEMENT SPLINTS

p0265 Mandibular advancement splints have been used in some paediatric patients with OSA who cannot tolerate CPAP and are not suitable for surgical intervention. However, a recent study in adults has shown that the symptom relief and improvement in sleep parameters was better with CPAP than with splints.[34] [Grade B]

s0100 ## Surgical treatment

p0270 Adenotonsillectomy is the treatment of choice for otherwise healthy children suffering with sleep-disordered breathing. The results of surgery are an improvement in nocturnal hypoxia, behaviour, quality of life and growth rate.[1, 2, 3, 4, 5, 7] [****/***] Polysomnography performed before and after adenotonsillectomy in children with sleep-disordered breathing has demonstrated a universal improvement in their apnoea/hypopnoea index and an abolition of OSA in approximately 90 percent of cases.[35] However, this means that 10 percent of apparently healthy children continue to have OSA after adenotonsillectomy. This group consists of the clinically obese, those with mild craniofacial disproportion and those with small tonsils on examination. Adenotonsillectomy should still be considered in these children as it can have a satisfactory outcome in some cases.[26] Performing sleep nasendoscopy at the induction of anesthesia will identify the site of the upper airway obstruction, which may be at more than one level.[16]

p0275 The results of adenotonsillectomy in children with Down's syndrome, cerebral palsy and craniofacial abnormalities are not so encouraging. Recent studies show that between 54 and 100 percent of children with Down's syndrome have OSA on polysomnography and the results of adenotonsillectomy are disappointing.[37] Other surgical treatments for OSA in Down's syndrome include tonsillar plication after tonsillectomy, midface advancement, uvulopalatopharyngoplasty (UVPP), anterior tongue reduction and hyoid suspension. Even with these forms of surgery an improvement in the child's symptoms is seen in only approximately 70 percent of cases, and deaths from upper airway obstruction are still not uncommon.[38]

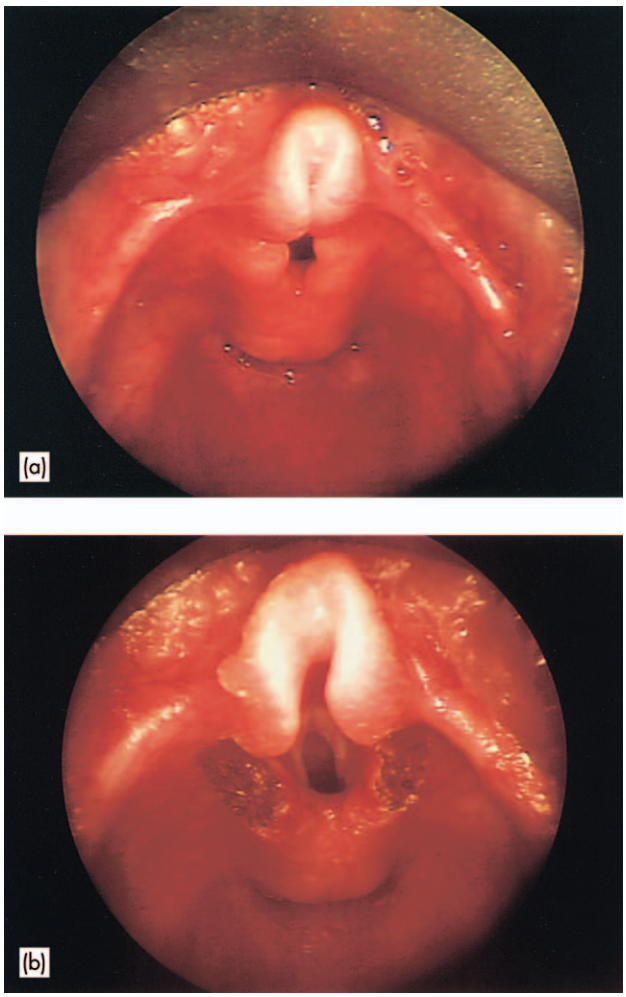
p0280 Children with cerebral palsy also have much a higher incidence of OSA and sleep-disrupted breathing than age-matched controls when investigated with polysomnography.[40] Adenotonsillectomy in this group is rarely curative and UVPP has been advocated as an alternative to tracheostomy.[41] The difficulty with predicting outcome with UVPP is the multifactorial nature of the airway obstruction, notably the presence of central apnoeas, the general hypotonia of the pharynx, the poor gag reflex, salivary pooling and the multisegment collapse of the upper airway. These factors contribute to a progression from OSA to obstructive awake apnoea and noisy daytime breathing. Laser supraglottoplasty has been used to treat selected cases, with resolution of the noisy breathing (**Figure 075.5**). Other treatments tried as an alternative to tracheostomy include hyoid suspension, mandibular advancement and tongue reduction.

p0285 Children with craniofacial syndromes can be treated with a nasopharyngeal airway or tracheostomy until formal midface advancement is performed at about five years of age. Distraction osteogenesis has shown some early promising results and can be performed at a much earlier age.[42] [Grade B/C]

s0105 ### TREATMENT OPTIONS ACCORDING TO THE LEVEL OF OBSTRUCTION SEEN AT SLEEP NASENDOSCOPY

p0290 Depending on the level of obstruction seen at sleep nasendoscopy the following are treatment options.

- p0295 Level 1 obstruction (velopharyngeal obstruction) is relieved by adenoidectomy or UVPP.
- p0300 Level 2 obstruction (tonsillar) is relieved by tonsillectomy.



f0025 **Figure 075.5** **(a)** An endoscopic view of the crowded supraglottis in a child with cerebral palsy. **(b)** The endoscopic view after dividing the aryepiglottic folds and vaporizing the redundant soft tissue overlying the arytenoid cartilages with a carbon dioxide laser.

p0305 • Level 3 obstruction (tongue base) can be relieved with the use of nasopharyngeal airways, glossopexy, mandibular advancement splints or CPAP.

p0310 • Level 4 obstruction (supraglottic) is due to collapse of the supraglottic tissues and manifests in its commonest form as laryngomalacia. It is a common finding in children with cerebral palsy who have upper airway obstruction. The treatment of obstruction at this level can be site specific in the form of laser supraglottoplasty or anterior epiglottopexy. A tracheostomy will be curative if the more localized procedures are insufficient. [Grade B]

s0110 POST-OPERATIVE COMPLICATIONS AND REDUCTION OF PERIOPERATIVE RISK

p0315 The potential surgical complications following adenotonsillectomy include pain, which causes poor oral intake, and haemorrhage. More worrying is that up to 20 percent of children who undergo adenotonsillectomy for OSA develop post-operative respiratory failure.[43] For this reason, otherwise healthy children undergoing adenotonsillectomy for OSA should not be booked as day cases.

p0320 Identified risk factors for the development of post-operative complications in otherwise healthy children are:

p0325 • severe OSA;
p0330 • a history of acute airway compromise;
p0335 • young age (less than three years);
p0340 • cardiac complications;
p0345 • failure to thrive;
p0350 • obesity;
p0355 • prematurity.

p0360 Children with craniofacial syndromes, neuromuscular diseases and systemic illnesses are also at increased risk of post-operative respiratory complications (**Table 075.1**).

p0365 These 'at risk' children should be admitted 24 hours prior to an anesthetic for the administration of systemic steroid to reduce pulmonary atelectasia. They should also undergo pulse oxymetry the night before a general anesthetic and for one or two nights post-operatively to document the severity of oxygen desaturations, even though a previous sleep study will have been carried out. Post-operative admission to an intensive care unit should be considered for all high-risk children, especially those who will have persistent OSA despite the planned surgical intervention. Systemic steroids and antibiotics, nebulized bronchodilators and chest physiotherapy are needed in the perioperative period if the child has audible crepitations on chest examination. [Grade D]

p0370 Children with OSA are very sensitive to narcotic analgesia, which has a significant detrimental effect on their respiratory drive.[44] Narcotics should be completely avoided in the perioperative period in any 'at risk' child. An experienced paediatric anesthetist will be needed to provide the general anesthetic. Following surgery, the endotrachial tube should not be removed until the patent has fully recovered from the anaesthetic, so as to minimize the risk of upper airway obstruction.

p0375 Children with Down's syndrome or cerebral palsy may need to be intubated in intensive care for the first 24 hours post-operatively until surgical oedema has settled. Alternatively, a nasopharyngeal prong can be used to stent the soft palate following UVPP or adenotonsillectomy in these children. Systemic steroids should be continued post-operatively for the first 24 hours to help with surgical oedema and pulmonary ateletasia. They have also been shown to reduce post-operative pain following tonsillectomy.[45] Patients who are on CPAP preoperatively can restart this immediately post-operatively and then can be weaned from it if the surgery is deemed successful. [Grade B]

s0115 ## LONG-TERM POST-OPERATIVE FOLLOW-UP

p0380 All patients should have clinical follow-up following initial treatment to document improvement or resolution of their symptoms. Children diagnosed as having mild or moderate sleep apnoea who have complete resolution of their symptoms do not require post-intervention sleep studies.

p0385 Children who continue to have symptoms, had severe OSA on presentation or are in the 'high-risk' group should have a post-intervention sleep study (**Table 075.3**). The sleep study should be performed approximately six weeks after surgery to ensure that the upper airway remodelling is complete. [Grade B]

s0120 ## CONCLUSION

p0390 Before paediatric OSA was recognized, children affected would present with severe failure to thrive and growth retardation, right-sided heart failure, cor pulmonale, permanent neurological damage, behavioural disturbance, hypersomnolence and developmental delay. Since the advent of paediatric sleep laboratories in the 1970s, OSA has been a widely recognized problem commonly encountered by paediatricians, otolaryngologists, anaesthetists and neurologists.

p0395 Adenotonsillectomy provides a cure in the great majority of children with OSA but in syndromic children and those with neuromuscular diseases it can present a real therapeutic challenge. Such children need treatment decisions to be made in collaboration with paediatric, sleep physician and anaesthetic colleagues and the parents or carers, so as to arrive at the most acceptable treatment option.

s0125 ## KEY POINTS

p0400 • Prevalence of sleep-disordered breathing is approximately 10 percent of the paediatric population.

p0405 • Adenotonsillectomy cures or improves sleep apnoea in 90 percent of children.

p0410 • Sleep-disordered breathing can cause neurobehavioural and cardiac complications.

p0415 • Children at increased risk of OSA have been identified.

p0420 • Sleep studies provide objective measure of the severity of sleep-disordered breathing.

p0425 • Sleep nasendoscopy can identify the site of upper airway obstruction.

p0430 • Children at increased risk of respiratory failure following surgical intervention have been identified.

p0435 • Children with persistent symptoms after intervention need follow-up sleep studies.

s0130 ## Best clinical practice

p0440 ✓ Snoring children should be screened for the presence of sleep-disordered breathing.

p0445 ✓ 'At risk' children need preoperative sleep studies and careful perioperative management to minimize the possibility of post-operative respiratory failure.

p0450 ✓ Syndromic children and those with neuromuscular disease pose a significant management challenge and need a multidisciplinary approach.

p0455 ✓ The site of upper airway obstruction should be investigated with sleep nasendoscopy if there is any doubt that it may not be adenotonsillar.

p0460 ✓ Persistent symptoms of OSA after intervention need follow-up with a post-operative sleep study.

s0135 ## Deficiencies in current knowledge and areas for future research

p0465 ➤ Understanding of the natural history of sleep-disordered breathing. There is concern that children with OSA may go on to develop adult sleep-related breathing disorders. This tendency may be reversed by early adenotonsillectomy.

p0470 ➤ Understanding of the clinical relevance of simple snoring. It may be that simple snoring is a marker for more sinister airway obstruction.

p0475 ➤ Accurate prevalence data for sleep-disordered breathing and age-appropriate diagnostic criteria.

p0480 ➤ Understanding of the mechanism of arousal and how it leads to daytime symptoms.

p0485 ➤ Development of standardized perioperative care designed to reduce post-operative respiratory failure.

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List of Abbreviations:

CPAP	Continuous positive airway pressure
ECG	electrocardiogram
EEG	electroencephalogram
OSA	Obstructive sleep apnoea
REM	rapid eye movement
UARS	upper airway resistance syndrome
UVPP	uvulopalatopharyngoplasty

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