REVIEW ARTICLE
Obstructive sleep apnoea syndrome in children

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Summary
Obstructive sleep apnoea syndrome in children is a complex disorder characterised by repeated nocturnal episodes of increased upper airway resistive load. It is most commonly associated with adenotonsillar hypertrophy and more children are now presenting for adenotonsillectomy. These children may pose different anaesthetic problems to those having surgery for recurrent infection alone and anaesthetic morbidity and mortality has been reported. In addition, due to the varied symptomatology of the condition, children with unrecognised obstructive sleep apnoea syndrome may present for incidental surgery. This is of importance as patients with undiagnosed obstructive sleep apnoea syndrome may experience additional peri-operative morbidity when undergoing incidental surgery. This article aims to review the aetiology, pathophysiology, clinical presentation and anaesthetic management of children with obstructive sleep apnoea syndrome.

Keywords Age factors; children. Complications; sleep apnoea.

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Definitions and aetiology
Sleep apnoea has been defined as cessation of air flow at the mouth and nose for at least 10 s during sleep [1–3]. Sleep apnoea may be classified into obstructive, central or mixed.

Central sleep apnoea is present when no airflow at the nose and mouth occurs and there is no respiratory movement. It accounts for approximately 10% of cases of sleep apnoea and is a disorder of the respiratory centre [4].

Obstructive sleep apnoea (OSA) has been defined as the absence of airflow at the mouth and nose despite respiratory movement. It is the cause of 85% of cases of sleep apnoea [5].

Hypopnoea describes episodes of partial upper airway obstruction during sleep causing at least a 50% reduction in airflow with respiratory movement resulting in a fall in arterial oxygen saturation [6].

Mixed sleep apnoea is described as a period of central apnoea followed by an obstructive apnoeic episode and is seen in 5% of cases [2].

These definitions are controversial, as are the diagnostic criteria for sleep apnoea in children [7]. The International Classification of Sleep Disorders Diagnostic and Coding Manual, 1990 set standards predominately based on adults. However, these do not necessarily apply to children and some consider that OSA syndrome in children should be considered as a separate syndrome with specific symptoms, diagnostic criteria and therapeutic requirements [8, 9].

In children, the obstructive apnoeas are often of less than 10 s duration and hypopnoeas are frequently seen. Periods of arterial oxygen desaturation comparable with OSA can occur and may result in the same long-term complications [1, 10–12].

Obstructive sleep apnoea in children is a disorder of upper airway function, most commonly associated with adenotonsillar hypertrophy in children older than 6 weeks of age [3, 4]. However, the severity of OSA syndrome is not always proportional to the size of the tonsils and adenoids [13], suggesting that other risk factors must contribute. A common variable in most children with OSA syndrome is a structurally small pharyngeal air space. Craniofacial anomalies that result in maxillary or mandibular hypoplasia, or macroglossia can be associated with OSA syndrome [14–16]. OSA syndrome may be present in patients with neuromuscular disorders resulting in hypotonia or cerebral palsy. Some children may develop OSA syndrome following pharyngeal flap surgery [17].
Pathophysiology

Under normal circumstances during respiration, the genioglossus and geniohyoid muscles act as the main muscle groups in maintaining pharyngeal airway patency. During inspiration, tone in these muscles increases, counteracting the subatmospheric pharyngeal intraluminal pressure generated by the respiratory pump muscles [16, 18]. During sleep, inspiratory collapse of the upper airway can occur. This is due to the negative intraluminal pressure exceeding the force of the dilatory muscles of the pharynx, which in turn is due to either anatomical or pathophysiological factors [16, 19–21].

Obstructive sleep apnoea results from a narrowing of the airway and occurs as a consequence of an anatomical reduction in the upper airway or incoordination of upper airway dilatory muscle activity. Most commonly, it may be due to a combination of both of these factors [3, 16, 21–23]. In adults, obstructive apnoeas are most frequently observed during rapid eye movement (REM) sleep due to a reduction in muscular tone. In children, periods of complete or partial obstruction of the upper airway can occur during REM and non-REM sleep [24]. Sedative drugs, anaesthetic agents and opioid drugs can all exacerbate the mechanisms that cause apnoea by decreasing pharyngeal muscle tone and inhibiting the arousal and ventilatory response to hypoxia, hypocapnia and obstruction [23, 25, 26].

When OSA occurs, the site of the obstruction (Table 1) can be anywhere from the nasopharynx to the supraglottis, though four principal sites have been described [27]. The typical sequence of events in a patient with OSA consists of the onset of sleep, followed by upper airway occlusion due to decreased pharyngeal muscle activity resulting in cessation of airflow despite continuing respiratory effort. This leads to progressive hypoxaemia, hypocapnia, increased ventilatory effort and gradually more negative intra-airway pressure. Restoration of both upper airway patency and airflow is associated with arousal from sleep and may be initiated by any or all of these respiratory phenomena [28]. The patient then returns to sleep and the cycle repeats itself. In children, so-called microarousals at the termination of the apnoeic episode can also occur [8, 29].

Cardiac arrhythmias during the apnoeic episodes may occur but in children this is an inconstant finding. Sinus arrest, second degree atrioventricular block and paroxysmal tachycardia have been observed [6, 8, 23].

Children with long-standing OSA syndrome who develop chronic hypoventilation can become progressively hypoxaemic and hypercapnic, resulting in carbon dioxide insensitivity and a reliance on hypoxic respiratory drive [30]. Arteriolar hypoxaemic vasoconstriction and hypertension occur and may result in pulmonary hypertension [18]. This may lead to progressive right ventricular hypertrophy and dilation eventually progressing to right ventricular failure, the main cause of mortality in patients with OSA syndrome [23].

Clinical features

In contrast with adult OSA, in children there is an equal prevalence of affected boys and girls [31]. Children usually present between 3 and 7 years of age, but may be as young as 4 months or as old as 14 years. Questionnaire-based studies have estimated the prevalence of OSA syndrome in children to be in the range 1.6–3.4% [32, 33].

Due to the multiple symptomatology of OSA syndrome in children the diagnosis may be delayed; Frank et al. reported 32 children with OSA syndrome in whom symptoms had been present for longer than 12 months [34]. Not infrequently, even in its most severe form, the diagnosis of OSA syndrome may have been missed by physicians [2, 14, 35].

Careful questioning of the parents of these children is essential in making a diagnosis. The most commonly described symptoms in children with OSA syndrome are nocturnal snoring and noisy breathing, observed apnoeas and restlessness during sleep associated with frequent awakening [1–3, 34]. In children it may not be possible to differentiate primary snoring from OSA syndrome by clinical history alone [36]. Enuresis, nightmares and morning headache may occur. Obesity, a typical feature of adults with OSA, is uncommon in children [31] but, if observed, may be associated with reduced daytime activity [1, 34, 35]. More typically these children have poor weight gain and small stature [37]. In contrast with OSA in adults where daytime somnolence and poor concentration are characteristic features, these are not typical presenting complaints in children and, in fact, hyperactivity is common [31]. Systemic hypertension was found in 25% of cases in one series [3]. Hypoxic brain damage and sudden death during sleep have been reported [23, 35].

Whilst a child with long-standing OSA syndrome may

Table 1 Site of obstruction in obstructive sleep apnoea syndrome.

<table>
<thead>
<tr>
<th>Type</th>
<th>Site of obstruction</th>
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<tbody>
<tr>
<td>1</td>
<td>Anterioposterior displacement of the tongue against the posterior pharynx</td>
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<tr>
<td>2</td>
<td>Posterior displacement of the soft palate by the tongue against the posterior pharynx</td>
</tr>
<tr>
<td>3</td>
<td>Opposition of the lateral pharyngeal walls</td>
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<tr>
<td>4</td>
<td>Circular closure of the pharynx</td>
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appear normal, facial abnormalities, such as midfacial elongation and open bite deformity may have developed due to chronic upper airway obstruction [14, 19]. In others, primary craniofacial anomalies may exist. Rhinorhoea or excessive mouth breathing may be noted. Pharyngeal examination usually reveals adenotonsillar hypertrophy and pharyngeal mucosal thickening due to chronic mouth breathing. Examination of the chest may reveal inspiratory stridor and in severe cases there may be intercostal and sternal retraction, pectus excavatum and cyanosis [35, 38].

Children with long-standing OSA syndrome who become chronically hypoxaemic and hypercarbic may develop acute respiratory failure and right ventricular failure [8]. These children may present acutely, with life-threatening upper airway obstruction, following pharyngeal infection or due to excessive sedation following anaesthesia [39–41].

**Investigations**

The probable diagnosis of OSA syndrome is suggested from the history and examination. Investigations can be useful in confirming the diagnosis and in determining the severity of the condition.

The initial investigation of a child with suspected OSA syndrome should be aimed at identifying those with chronic hypoxaemia. A full blood count to determine the presence of polycythaemia, an electrocardiogram (ECG) and pulse oximetry should be performed. The ECG may indicate the presence of cor pulmonale [25, 38] with a large P wave in leads II and V1, a large R wave in V1 and a deep S wave in V6. Wilkinson et al. noted that 3% of children presenting for adenotonsillectomy had features of right ventricular strain on the ECG [42]. If features of right atrial or ventricular hypertrophy exist, a chest radiograph is useful in assessing the degree of cardiac enlargement and the possible presence of pulmonary oedema [29]. In children with features of right ventricular hypertrophy, echocardiography can be used to assess ventricular function. Cardiac catheterisation may show pulmonary hypertension to be present [19, 38] and incorrect diagnoses of cardiomyopathy or primary pulmonary hypertension have been reported [30, 43]. Arterial blood gas examination, although rarely performed, may show a degree of hypoxaemia, hypercapnia and elevation of serum bicarbonate [15, 23, 44]. Lateral radiographs of the neck during inspiration and expiration [45], direct flexible endoscopy [46], fluoroscopy [47], CT and MRI scanning [48] are all investigations which have been used to define the site of obstruction.

Polysomnography is the electrographic monitoring of physiological variables during sleep [49]. The aim of such studies is to document the number of episodes of increased upper airway resistive load and the presence of sleep disturbance. It has been used to clarify the diagnosis, particularly in borderline cases and to compare the effects of treatment on the measured variables [1, 50, 51]. Common measurements which may be included are the ECG, submental electromyogram (EMG), nasal/oral air flow, respiratory movement or effort, pulse oximetry ($S_{\text{O}_2}$), transcutaneous oxygen partial pressure, end-tidal carbon dioxide partial pressure ($P_{\text{E}}CO_2$), anterior tibialis EMG and video photography during natural or drug-induced sleep [6, 11]. Recording of submental EMG consists of electrode placement beneath the jaw to detect tonic activity in the upper airway muscles at the base of the tongue. The loss of such activity, together with the monitoring of ocular movements, heralds the onset of REM sleep. Interpretation of polysomnography is complex and at present there is no consensus on how the findings of sleep studies should be interpreted in children [6, 29]. Various scoring systems have been developed to aid diagnosis; some investigators use an apnoea/hypopnoea index (total number of obstructive episodes per hour of sleep) together with the degree of arterial desaturation and hypercarbia. Loughlin suggests that an apnoea/hypopnoea index greater than 5 and/or arterial desaturation greater than 4% and elevated $P_{\text{E}}CO_2$ greater than 6.3 kPa should be considered abnormal [29].

Whilst methods to determine the degree of severity of the condition have not been fully validated, morbidity and mortality of the OSA syndrome is greatest in those with chronic hypoxaemia and hypoventilation, and markers for these may indicate severity [52].

**Management**

In most cases the definitive treatment of children with OSA syndrome is surgical by adenotonsillectomy. The recognition of the association between adenotonsillar hypertrophy and OSA syndrome has led to an increasing number of children presenting for adenotonsillectomy with this syndrome in the USA and Europe [20]. A retrospective survey of adenotonsillectomies at the Mt Sinai Medical Center found that in 1978 all 282 operations were for recurrent infection. By 1986, 81% of the 135 cases were for this indication while 19% were for OSA [53]. At the Children’s Hospital in Philadelphia, most adenotonsillectomies are now performed on patients with OSA [54]. Other surgical procedures such as uvulopalatopharyngoplasty, glossectomy, maxillary advancement and tracheostomy may be required to relieve the upper airway obstruction [1, 14, 23, 34].

The use of nasal continuous positive airway pressure (CPAP), which is now well established in the management of adult OSA, has only recently been described in children.
So far, the technique has been reserved for severe cases with craniofacial abnormalities or with residual problems after surgery [55–57].

**Anaesthetic management**

Anaesthetists may encounter children with OSA syndrome in one of three ways. They may present acutely, with severe upper airway obstruction and right ventricular failure, for adenotonsillectomy or for incidental surgery. In each case there may or may not be a pre-existing diagnosis of OSA.

Management of children presenting with decompensated acute OSA syndrome consists of relieving the upper airway obstruction and treatment of the cardiac failure prior to surgical correction. The airway obstruction may be relieved either using an oropharyngeal or nasopharyngeal airway or, if necessary, tracheal intubation. Metered oxygen should be administered with care as high inspired oxygen concentrations may cause a rise in pulmonary artery pressure resulting in respiratory depression [38]. Cardiac failure should be treated with digoxin and diuretics. If an upper respiratory tract infection exists, antibiotics should be given. Once the child is stabilised and the cardiac failure has resolved then surgical relief of the upper airway obstruction is indicated [38].

The anaesthetic management of all children with OSA syndrome is centred around the preservation and control of the upper airway. Whichever anaesthetic technique is chosen, particular attention should be paid to airway management and the risk of cardiovascular collapse. Close communication between all medical staff involved in their care is fundamental to the well-being of these children, to allow an adequate period of assessment and planning prior to surgery [58]. The recovery from anaesthesia and surgery is a critical period as upper airway obstruction and apnoea with hypoxia may continue for several days postoperatively [5, 23, 40, 59, 60]. In addition, postoperative cardiac failure with pulmonary oedema can develop [61]. Identification of children with OSA syndrome who are at the greatest risk of these complications in the peri-operative period is a major concern of the anaesthetist. Both the type of anaesthetic employed and the severity of the condition may contribute to the risks.

**Pre-operative preparation**

Awareness, by the anaesthetist, of the clinical features of OSA syndrome in children is important as the diagnosis may previously have been missed. The anaesthetist should enquire specifically about sleeping habit and snoring in all children presenting for anaesthesia. If clinical features of OSA syndrome are present a full pre-operative assessment should include baseline investigations of haematocrit, ECG and pulse oximetry. Any suggestion of chronic hypoxaemia or right ventricular hypertrophy warrants further investigation as described previously. The identification of children who have the greatest risk of peri-operative complications is critical and in a retrospective study looking at respiratory complications in 37 children with OSA syndrome presenting for adenotonsillectomy, Rosen et al. [57] suggest a series of criteria (Table 2).

If a child presents for incidental surgery and is thought to have OSA syndrome, serious consideration needs to be given as to the relative merits of proceeding with the incidental surgery or undertaking corrective otolaryngological surgery first.

Sedative drugs, anaesthetic agents and opioid analgesics can all exacerbate the mechanisms that cause apnoea by decreasing pharyngeal muscle tone and inhibiting the arousal and ventilatory response to hypoxia, hypercapnia and obstruction [23, 25, 26]. This may lead to severe hypoxia in the peri-operative period which can persist postoperatively [40]. Most authors state that pre-operative sedative drugs are contraindicated in children with obstructive sleep apnoea [23, 40, 42]. The use of anticholinergic drugs such as atropine or glycopyrrolate which act as antialagogues may reduce the risk of laryngospasm. In addition, they provide some cardiovascular protection against the bradycardic effects of anaesthetic agents which are particularly detrimental in individuals with OSA syndrome and cardiac dysfunction [41].

**Intra-operative management**

Life-threatening upper airway obstruction and difficulty with intubation can occur following induction of anaesthesia, especially in children with associated craniofacial anomalies [62]. If right ventricular strain is present, the child can develop cardiac decompensation during induction, resulting in pulmonary oedema [23, 61, 63].

**Table 2** Risk factors associated with postoperative respiratory complications. Presence of any of the following features indicates high risk.

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<thead>
<tr>
<th>Clinical features</th>
<th>Surgical features</th>
<th>Polysomnographic features</th>
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<td>Age &lt; 2 years</td>
<td>Uvulopalato-</td>
<td>Apnoea/hypopnoea</td>
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<td></td>
<td>pharyngoplasty</td>
<td>index &gt; 40</td>
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<tr>
<td>Craniofacial</td>
<td></td>
<td>Arterial oxygen</td>
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<tr>
<td>abnormalities</td>
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<td>saturation &lt; 70%</td>
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<td>Failure to</td>
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<td>thrive</td>
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<td>Hypotonia</td>
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<td>Morbid obesity</td>
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<td>Previous airway</td>
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<tr>
<td>trauma</td>
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<td>Cor pulmonale</td>
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There are insufficient published data on the best methods for induction and maintenance of anaesthesia in these children to produce definitive recommendations. Yates described the management of six children with signs of cor pulmonale undergoing adenotonsillectomy [44] in whom anaesthesia was induced with halothane and oxygen, with or without nitrous oxide. The tracheas of five were intubated under deep halothane anaesthesia while breathing spontaneously and one child required succinylcholine to facilitate intubation due to cyanosis during induction. The patients were allowed to breathe spontaneously throughout surgery. Two patients had some degree of upper airway obstruction during induction and the author suggests that use of CPAP was beneficial in helping to maintain the upper airway. Helfaer et al. studied 15 children with mild OSA syndrome presenting for adenotonsillectomy and compared a halothane-based anaesthetic with a fentanyl-based anaesthetic [50]. Intravenous induction of anaesthesia was employed with subsequent use of a long-acting muscle relaxant to allow intubation and controlled ventilation. Difficulty in airway maintenance or intubation was not reported.

Although pre-oxygenation may have an unpredictable effect on pulmonary artery pressure and respiratory drive, it is probably wise to provide a period of pre-oxygenation in these children if difficult intubation is anticipated [43]. An inhalational induction produces a smooth transition from spontaneous to assisted ventilation which may ease the management of a difficult airway [25, 40]. The effects of anaesthesia are reversible if difficulties in maintaining an airway occur. The use of inhalational agents in patients with pulmonary hypertension has been reported to lower pulmonary artery pressure [43]. However, this method of induction can be unpleasant for the child and result in coughing and laryngospasm, especially if an anticholinergic premedication has not been given. In addition, most inhalational agents have a myocardial depressant effect in the high concentrations required for induction. Halothane is less likely than isoflurane or enflurane to cause irritation or laryngospasm. Sevoflurane has smoother and more rapid inhalational induction characteristics with faster emergence compared with halothane, which may be advantageous [64]. Nitrous oxide has been reported to produce a rise in pulmonary artery pressure [41].

The intravenous route for induction of anaesthesia is rapid, does not produce an excitation phase and reduces the likelihood of vomiting and laryngospasm. However, following intravenous induction, there may be complete loss of the upper airway with an inability to ventilate the lungs. Achieving intravenous access may be distressing to the child although this is less likely with the use of topical local anaesthetic creams [23, 25]. If the upper airway becomes obstructed during induction this can usually be relieved by jaw manipulation, the use of an oral or nasal pharyngeal airway or the use of CPAP [25, 40, 43].

Tracheal intubation is the preferred option for adenotonsillectomy and in children with associated craniofacial abnormalities this can prove particularly difficult. Intubation can be facilitated by using high concentrations of inhalational agents in the spontaneously breathing child [40, 62]. This technique ensures that spontaneous ventilation occurs at all times, but it may take a considerable period of time to achieve an adequate depth of anaesthesia in the partially obstructed child. Alternatively, a muscle relaxant can be used to facilitate intubation. If the airway is already compromised, complete obstruction may follow the administration of succinylcholine and in patients with cardiac dysfunction it can induce cardiac arrhythmias. Nondepolarising muscle relaxants have a longer onset of action than succinylcholine and in the partially obstructed child prolonged positive pressure ventilation via a face mask may result in gastric inflation, diaphragmatic splitting and regurgitation. If complete airway obstruction occurs reversal of these agents is more difficult. Fortunately, tracheal intubation is rarely difficult in a child with isolated adenotonsillar hypertrophy and a rapid airway maintenance technique using suxamethonium is probably the safest. Advocates for awake intubation exist, but this may be difficult in an uncooperative child [62]. Blind nasal intubation is an alternative technique in skilled hands.

The laryngeal mask airway (LMA) has been used successfully in many cases of failed intubation and its elective use in ENT surgery is becoming more widespread [65]. Adequate surgical access can be achieved with no aspiration of blood. The LMA can be left in situ until the child’s protective reflexes return. One study found recovery to be significantly better, with less airway obstruction, when compared with the tracheal tube [66]. Extra vigilance on the part of the anaesthetist is required when using the LMA because of the danger of it becoming dislodged.

Standard peri-operative monitoring using $S_{\text{O}_2}$, arterial blood pressure, ECG and $P_{\text{aCO}_2}$ should be undertaken [10, 25]. In patients with severe upper airway obstruction and right ventricular dysfunction, arterial cannulation to measure invasive arterial blood pressure and blood gases can be useful.

Whether spontaneous or controlled ventilation should be used for adenotonsillectomy in the general population is controversial [42, 58, 67]. In the USA, most anaesthetists advocate controlled ventilation, while in the UK spontaneous ventilation is most commonly used. A spontaneous ventilation technique requires the use of a sufficient end-tidal concentration of inhalational agent to prevent coughing. In patients with chronic hypoxia and hypercapnia, this is likely to result in respiratory and myocardial depression. Intermittent positive pressure ventilation reduces the
requirement for inhalational agents and their undesirable cardiovascular and respiratory effects. In addition, normocapnia is more readily maintained [67].

The use of opioids during anaesthesia may result in respiratory depression, especially in the postoperative period. However, Helfaer et al. did not find an increase in postoperative respiratory complications in children who received fentanyl 2 µg.kg⁻¹ intra-operatively [50]. It has been suggested that codeine phosphate produces less respiratory depression than morphine and may be a useful alternative [68]. It should be given intramuscularly as intravenous administration can produce arrhythmias [69].

Postoperative management

Extubation before the return of laryngeal and pharyngeal reflexes may result in upper airway obstruction. Some anaesthetists have suggested that extubation during deep, spontaneously breathing anaesthesia results in smoother recovery conditions with less coughing and retching, reducing the risk of re-bleeding [58]. If this is planned, the insertion of an oral or nasopharyngeal airway at the end of the procedure may be helpful [40]. Awake extubation requires full reversal of muscle relaxation and return of upper airway reflexes. The use of short-acting non-depolarising muscle relaxants allows a rapid full reversal [5, 67]. Regardless of the technique used, direct inspection of the pharynx is important following adenotonsillectomy to ensure a bloodless field before extubation. Equipment and drugs for re-intubation should be at hand as urgent re-intubation may be necessary [25]. Extubation in the lateral position, with a slight head down tilt, will ensure safe drainage of blood if re-bleeding should occur. Careful observation of the breathing pattern and for pharyngeal bleeding in the immediate postextubation period is essential.

There has been considerable uncertainty and controversy regarding the postoperative management of children with OSA syndrome [52]. Concern about the continued risk of apnoeas and upper airway obstruction during the immediate postoperative period due to excessive sedation or pharyngeal oedema has been shown to be unfounded in children with mild OSA syndrome. Helfaer et al. compared pre-operative and immediate postoperative polysomnography in 15 children with mild OSA syndrome who underwent adenotonsillectomy [52]. Mild OSA syndrome was defined as an apnoea/hypopnoea index less than 15 and no associated risk factors as defined by Rosen et al. [57]. A significant improvement in polysomnography findings was observed during the first postoperative night, which occurred regardless of the use of intra-operative fentanyl 2 µg.kg⁻¹. They concluded that, in this group of children, intensive postoperative monitoring is unnecessary.

Children with severe OSA syndrome can develop life-threatening respiratory complications in the immediate postoperative period [57] and these children should be observed intensively for at least 24 h [40, 50, 57]. Nursing should be on a one-to-one basis with monitoring of the breathing pattern and respiratory rate, pulse oximetry and ECG [23]. Oxygen therapy in these children should be metered, with consideration given to regular monitoring of arterial partial pressure of carbon dioxide in order to detect its retention [60]. A balance between oversedation, resulting in obtunded reflexes, and too little, causing crying and straining which may lead to further bleeding and oedema, can prove a considerable challenge.

Various analgesics have been advocated following adenotonsillectomy. Nonsteroidal anti-inflammatory drugs do not produce sedation but may, in theory, aggravate bleeding. Alternatively, regular paracetamol 15–20 mg.kg⁻¹ 6 hourly either orally or rectally may suffice [42]. If these measures are inadequate, the use of small intravenous doses of morphine, with close monitoring of its effects, can be used although there is a risk of airway compromise. It has been suggested that the use of a local anaesthetic spray on the pharyngeal mucosa is a safe technique, although the loss of sensation within the pharynx may increase the risk of upper airway obstruction [23, 27].

The long-term outcome following adenotonsillectomy is good and most children improve over several days [34, 59, 70]. Polysomnographic studies have been undertaken to follow up these children several weeks after surgery [51]. Right ventricular ejection fraction has been shown to increase significantly after tonsillectomy [71]. However, following relief of upper airway obstruction, cardiac failure with pulmonary oedema has been reported [61]. This may be due to an abrupt fall in airway pressure and a subsequent increase in systemic venous return resulting in an increased pulmonary hydrostatic pressure and pulmonary oedema [14, 25]. There are no reliable criteria to predict which patients are at risk of cardiac failure postoperatively.

There are only anecdotal reports regarding the incidence of postoperative complications of children with OSA syndrome who have had incidental surgery [41]. Inferred data from children presenting for adenotonsillectomy do suggest this group of children may be at particular risk during the postoperative period. Great caution is warranted as significant upper airway obstruction will persist in the absence of corrective surgery.

Conclusion

With the increased recognition of adenotonsillar hypertrophy as a cause of OSA syndrome it is likely that more
children will present for adenotonsillectomy with this condition. A careful history of sleep habit is essential and appropriate investigations should be performed when the diagnosis is suspected. A clear understanding of the pathophysiology of severe OSA syndrome is helpful in anticipating the risks involved in surgery and anaesthesia. These children are at significant risk of respiratory and cardiovascular complications and whichever anaesthetic technique is chosen the anaesthetist should be aware of the consequences. Postoperatively children with severe OSA syndrome should be managed in an intensive care environment.

The majority of children with OSA syndrome who have mild disease probably have little increased anaesthetic morbidity when presenting for adenotonsillectomy. However, in the absence of a large prospective study investigating the patient risk factors and anaesthetic techniques, an awareness of the potential complications is essential when the anaesthetist is presented with any child with suspected OSA syndrome.

As with many otolaryngological challenges in children, good communication and understanding between paediatricians, anaesthetists and surgeons is essential for the successful management of this condition.

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