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Review Article

Morbidity after adenotonsillectomy for paediatric obstructive sleep apnoea syndrome: waking up to a pragmatic approach

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Abstract

Introduction: Adenotonsillectomy is successful at eliminating airway obstruction in the majority of otherwise normal children with obstructive sleep apnoea syndrome. Children with this condition are at significantly higher risk of post-operative respiratory complications. Identifying children at risk of post-operative respiratory complications after adenotonsillectomy for obstructive sleep apnoea syndrome remains a challenge for clinicians, especially those at district general hospitals.

Aim: To review the evidence and to proffer a pragmatic approach to diagnosis and management, by classifying those at risk of post-operative respiratory complications into different risk subsets, with guidelines for management.

Conclusion: Patients in the high risk group should be operated upon at paediatric specialist centres with intensive care facilities. Those in the moderate risk group may undergo adenotonsillectomy at their district general hospital, provided facilities for administering continuous positive airway pressure are available on-site. Most children with obstructive sleep apnoea syndrome may be classified as low risk candidates and may safely be operated upon at their local district general hospital.

Key words: Obstructive Sleep Apnoea Syndrome, Paediatric; Post-operative Respiratory Complications; Adenotonsillectomy

Introduction

Sleep-disordered breathing exists as a continuum of severity, ranging from partial upper airway obstruction producing primary snoring (3–12 per cent), through intermediate conditions (i.e. upper airway resistance syndrome and obstructive hypoventilation), to continuous episodes of complete upper airway obstruction or obstructive sleep apnoea (0.7–3 per cent).[1] Adenotonsillectomy is successful in eliminating airway obstruction in 85–95 per cent of otherwise normal children with obstructive sleep apnoea syndrome (OSAS).[2–5] However, it is associated with significant post-operative respiratory morbidity; therefore, identification of children at risk is critical. Definitive pre-operative diagnosis is difficult, as symptoms in children may be highly varied and subtle to detect. Moreover, universally accepted diagnostic criteria for paediatric OSAS do not exist, rendering comparison of studies difficult and patient care protocols variable.[6] Anaesthetic management may also play an important role in influencing the post-operative outcome, with regard to opioid use for analgesia.

This review discusses the evidence surrounding these issues and suggests a pragmatic approach to management, with particular emphasis on everyday clinical practice in district general hospitals within the UK.

Definitions

Currently, there are no officially endorsed diagnostic criteria for childhood sleep-disordered breathing, unlike adult sleep-disordered breathing.[7] In children, OSAS involves episodes of hypopnoea and apnoea (Figure 1).[8] Obstructive apnoea may be defined as cessation of ventilation despite effort for 10 seconds or two breath cycles in older children, and for 6 seconds or 1.5 to two breaths in infants.[9] Obstructive sleep apnoea syndrome severity is graded according to the apnoea–hypopnoea index, which is the total number of apnoeic plus hypopnoeic events per hour of sleep. The apnoea–hypopnoea index would be 5–10 in cases of mild OSAS, 10–20 in moderate OSAS and >20 in severe OSAS. If the apnoea–hypopnoea index is less than five, upper airway resistance syndrome may be diagnosed. This is

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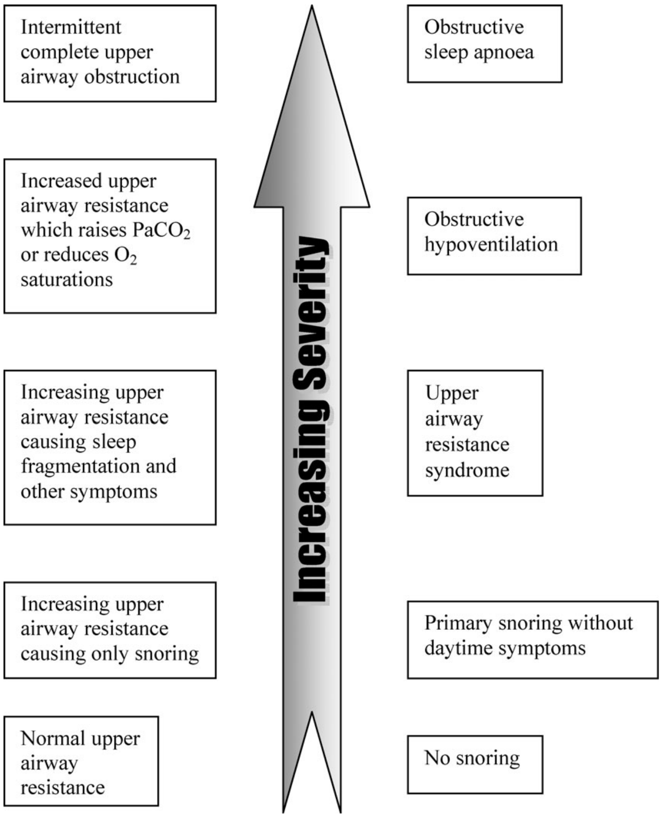

FIG. 1

Continuum of upper airway resistance severity in paediatric sleep-disordered breathing.

characterised by partial airway obstruction without measured gas exchange abnormalities, and the presence of sleep fragmentation or frequent microarousals.[10,11] A biphasic abdominal respiratory pattern, paradoxical breathing, ribcage movement and snoring are observed. Obstructive hypoventilation is defined as a 50 per cent reduction in airflow despite effort during the same time or breath cycles, with desaturation or arousal, leading to a peak end-tidal $PaCO_2$ of >55 mmHg or an end-tidal $PaCO_2$ of >45 mmHg for more than 60 per cent of the total sleep time.[9]

Pathophysiology

In otherwise normal children with OSAS, the current perspective is that adenotonsillar hypertrophy causes airway narrowing which, when superimposed on subtle abnormalities of upper airway motor control or neural drive, results in clinically significant dynamic airway obstruction during sleep.[12] Physiologically, sleep is associated with increased airway resistance and reduced muscle tone, which reaches a nadir in rapid eye movement (REM) sleep, rendering patients more vulnerable to airway obstruction during REM sleep. Normal infants and young children spend more time in REM sleep (40–80 per cent of the total sleep cycle) than older children and adults (10–30 per cent in comparison), and they react more seriously to increased anatomical or functional reduction of the upper airway, by suffering episodes of apnoea and desaturation.[13] Even brief apnoeas may lead to desaturation, as the oxygen consumption/supply ratio in children is higher and their functional residual capacity during sleep is lower.[14]

Most children with OSAS do not have significant apnoea whilst awake but suffer mixed central and obstructive apnoea when asleep. Normally, during sleep, chemoreceptors sense changing levels of CO_2 and O_2, leading to a compensatory increase in respiratory muscle activity. In OSAS, there is a defect in the central ventilatory drive response to hypercarbia, hypoxaemia and reduced airway muscle tone, possibly due to habituation or mechanical damage. This may explain the reported increased risk of post-operative respiratory complications in children with OSAS.[15] Once the causative obstruction is removed by adenotonsillectomy, this central, depressed response normalises.[12]

Pre-operative diagnosis of obstructive sleep apnoea syndrome in children

The goals of pre-operative diagnosis are to: (1) identify patients at risk of adverse outcomes if treatment is delayed; (2) avoid unnecessary intervention in patients not at risk; and (3) assess those at increased risk of complications resulting from adenotonsillectomy. Scientifically evaluated diagnostic methods include history and physical examination, polysomnography, overnight pulse oximetry, audio- and video-recording, cephalometry, and sleep nasoendoscopy.

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History and physical examination

The primary dilemma in paediatric OSAS is whether clinical history and examination, often the only available methods of assessing children for OSAS in a district general hospital, have predictive validity for diagnosis. Relying on history alone can sometimes be misleading. For example, children with OSAS experience obstruction mainly during REM sleep, which mostly occurs in the early morning hours when their parents are not observing them, leading to an underestimation of the degree of apnoea.[13] The loudness of snoring also does not necessarily correlate with apnoea severity.[16] Previous studies advocating polysomnography over clinical evaluation as the definitive diagnostic tool of childhood OSAS[16–22] were performed before upper airway resistance syndrome became widely recognised. In general, assessment of suspected OSAS should still begin with a detailed history of the child's sleep and daytime symptoms (Table I).[8]

Important points in the history should include establishing where the child sleeps in relation to the caregiver being interviewed, and the extent to which the caregiver is aware of the child's sleep problems. The index of suspicion for OSAS should be lower in children with respiratory disease, obesity, Afro-Caribbean ancestry and a family history of OSAS.[23] Other symptoms suggestive of significant childhood sleep-disordered breathing include non-specific complaints such as snoring, disturbed sleep and enuresis.[24,25] Diurnal fatigue, the hallmark of adult OSAS, rarely occurs in paediatric OSAS.[25] Neurocognitive impairment, behavioural problems and failure to thrive may be associated with developmental delay, attention disorders and poor school performance. Untreated, OSAS may result in cor pulmonale with symptoms of heart failure.[25,26]

Physical examination whilst awake is important to evaluate airway structure and exacerbating factors, but findings are often normal, even in severe OSAS (Table II). There may be non-specific features related to adenotonsillar hypertrophy, such as mouth-breathing, nasal obstruction and adenoidal facies. Tonsillar size and OSAS severity have not been correlated.[3] Complications of OSAS may present as systemic hypertension, an increased component of the second heart sound (indicating pulmonary hypertension) and poor growth.

Polysomnography

Although clinical features of childhood OSAS are well documented, there is weak correlation with positive polysomnography. In numerous trials, the accuracy of clinical evaluation of paediatric OSAS in predicting positive polysomnograms was poor, ranging from 30–85 per cent.[16–22] At a sleep clinic,

TABLE I

CLINICAL HISTORY FOR THE CHILD WITH SUSPECTED SLEEP-DISORDERED BREATHING

Clinical aspect	Specific queries
Sleep history	Usual bedtime
	Usual sleep onset time
	Nocturnal awakenings
	Unusual sleep positions
	Movements during sleep
	Enuresis
	Problems with awakening in morning
Sleep environment	Usual sleeping location
	Bed-sharing or room-sharing
	Distracting factors in sleep environment (e.g. television)
Snoring & breathing history	Age at onset of snoring
	Frequency
	Proportion of night spent snoring
	Pitch & loudness
	Observed increased breathing effort or struggle whilst asleep
	Neck position (hyperextended)
	Parental interventions to improve breathing (e.g. waking child)
Daytime symptoms	Daytime mouth-breathing
	Nasal obstruction
	Poor eating
	Poor growth
	Behavioural (inattentive, hyperactive, poor school performance)
	Neurocognitive (poor school performance, developmental delay, poor memory)
Drug history	Current medications, & interference with sleep quality, upper airway tone or nasal resistance
Past medical & surgical history	Previous airway surgery (adenotonsillectomy)
	Previous intubation as neonate
	Previous cleft lip or palate repair
	Previous nasal surgery
	Thyroid or metabolic problems
	Recent weight gain
Family history	Snoring
	OSAS
	Obesity
	Family members on CPAP

OSAS = obstructive sleep apnoea syndrome; CPAP = continuous positive airway pressure

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TABLE II

PHYSICAL EXAMINATION OF THE CHILD WITH SNORING AND SUSPECTED SLEEP-DISORDERED BREATHING

Clinical aspect	Specific feature
General	Height
	Weight
	Growth curve
	Blood pressure
Body form	Obesity
	Neck anatomy (short neck)
Ear, nose & throat	Oropharyngeal size
	Tonsil size
	Adenoid enlargement
	Nasal patency
	Neck masses, including thyroid
Craniofacial	Facial shape & features
	Micrognathia
	Retrognathia
	Elongated soft palate
Cardiovascular	Signs of cor pulmonale
Features of rarer conditions e.g.:	Neuromuscular weakness
	Cerebral palsy

a clinical scoring system to assess breathing during sleep was able to discriminate between healthy and OSAS children, but failed to distinguish between those with mild and moderate OSAS[27] or between primary snoring and OSAS paediatric patients.[21] Prediction of OSAS severity based on parental observation is also unreliable.[1] Based on these studies, and the fact that only 20–30 per cent of snoring children have positive polysomnography, the American Thoracic Society recommended that polysomnography be obtained before adenotonsillectomy in order to distinguish primary snoring from OSAS.[9] In 2002, the American Academy of Pediatrics reiterated polysomnography as the gold standard for diagnostic evaluation of childhood OSAS.[28]

However, significant limitations exist within these guidelines. The quoted studies relied on adult criteria for interpretation of polysomnography, now acknowledged as inappropriate for children.[17–19,21,22] Little data are available regarding normal respiratory parameters during sleep in healthy children.[29] Performance and interpretation of polysomnogram results have not yet been standardised or evaluated for different age groups.[26] Moreover, formal studies have not yet been performed to evaluate the correlation between polysomnographic parameters and adverse outcomes in children.[28] Unsurprisingly, the number of patients diagnosed with OSAS by polysomnography may vary depending on the criteria used for interpretation.[30] Despite the presence of negative polysomnographic findings (the gold standard), the benefits of adenotonsillectomy are still observed in children with symptomatic airway obstruction, including improvement in behaviour and quality of life.[5,18,20] The guidelines do not explicitly acknowledge the existence of upper airway resistance syndrome and obstructive hypoventilation, diagnoses which require oesophageal pressure monitoring, an investigation not routine in most centres, let alone in district general hospitals. Hence, these studies may have underestimated the number of children with significant sleep-disordered breathing.[31] In the absence of guidelines, some paediatric sleep laboratories have developed their own diagnostic standards for childhood upper airway resistance syndrome, based on arousal index, sleep efficiency, severity of snoring and technician observations of increased respiratory effort.[8] Such variability emphasises the need to establish unambiguous definitions and validated guidelines to facilitate diagnosis of childhood upper airway resistance syndrome.

Should all children be recommended to undergo polysomnography before adenotonsillectomy? The problem is cost and availability, given the financial constraints of the UK National Health Service. The price of polysomnography is quoted as US$600–2800 (£320–1490).[31] Although avoiding unnecessary surgery for children with primary snoring is laudable, insisting that all children with obstructive symptoms undergo pre-operative polysomnography may delay treatment for affected children and deny treatment to those without access to paediatric sleep laboratories. Distorted results may also arise from the 'first night' effect of sleeping in a strange environment; recordings taken on two consecutive nights may be more reliable, but this raises further financial and practical issues.[26] Nap polysomnography can be performed in the daytime and is more convenient for patients and laboratory staff, but it has far lower predictive value than overnight polysomnography, due to the reduced amount of REM sleep and total sleep time.[28] Unattended home polysomnography in children has been studied in very few centres; findings suggest that apparently similar results to overnight laboratory studies can be obtained, but sophisticated equipment and the cooperation of caregivers are required.[32]

In reality, only 12 per cent of school-aged children who undergo adenotonsillectomy for OSAS have pre-operative polysomnography.[33] Most children with snoring and daytime OSAS symptoms never see a sleep specialist; they are either referred to an otolaryngologist or remain unidentified.[8] For the snoring child with daytime symptoms and enlarged tonsils or adenoids, it is argued that the likely diagnosis is upper airway resistance syndrome or OSAS, and that polysomnography is unnecessary.[31] Most paediatric otolaryngologists surveyed nationwide relied on clinical diagnosis to recommend adenotonsillectomy for OSAS in children, reserving polysomnography only for doubtful diagnoses.[34]

Ultimately, polysomnography appears to be most useful to confirm the diagnosis of OSAS when history and physical examination fail to concur, and to document the severity of OSAS in: children younger than three years; high risk patients for whom surgery is contraindicated; cases of craniofacial anomalies, morbid obesity and cerebral palsy; and in children who remain symptomatic after adenotonsillectomy. When surgery is contraindicated or has failed to alleviate symptoms, polysomnography is also a prerequisite to treatment with nasal continuous positive airway pressure (CPAP).[31]

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Overnight pulse oximetry

Overnight pulse oximetry may be used to screen for OSAS. Detecting intermittent episodes of hypoxaemia in a snoring child may suggest OSAS, particularly with O_2 saturation nadirs of <80 per cent.[26] Overnight pulse oximetry may estimate the severity of OSAS and predict those at risk of post-adenotonsillectomy respiratory compromise.[35,36] However, its low negative predictive value means that children with normal test results may still have significant OSAS. Another pitfall is that lower respiratory tract infections, asthma and pulmonary hypertension (which may all coexist with OSAS) may concurrently cause desaturation during sleep.[37]

Audio- and video-recording

Ideally, any child suspected of having OSAS should be observed while asleep. Asking parents to provide an audio-visual tape of the child sleeping may be an efficient means of observing the child asleep, but whether such recordings are representative or worst case data should be ascertained. Sleep audio- and videotape analysis has been used to screen for OSAS, with sounds of struggle appearing to be more predictive of OSAS than pauses in breathing.[38] However, distinguishing obstructive from central events is difficult, and discrepancies in results from different centres indicate the need for further study.[38,39]

Cephalometry

Cephalometry provides data about mandibular and craniofacial dimensions, including the upper airway.[40] Although one study correlated craniofacial differences to the apnoea–hypopnoea index severity in children with OSAS,[40] a clear relationship has not been established.[41] Cephalometry may be more helpful in diagnosing OSAS in children with existing craniofacial abnormalities, such as craniosynostosis.[41] Lateral neck radiographs may be useful to evaluate adenoid size, but they do not predict the presence or severity of obstruction when the child is sleeping and supine.[42]

Sleep nasoendoscopy

Sleep nasoendoscopy has been used to determine the level of upper airway obstruction in children, providing a dynamic picture of the upper airway in all phases of respiration. A classification system dividing airway obstruction into four anatomical levels has previously been suggested.[43] The major criticism of this technique is that anaesthetically-induced sleep does not truly mimic natural sleep, potentially leading to spurious results. The success of sleep nasoendoscopy also depends on considerable skill and patience on the part of the anaesthetist, in order to achieve the narrow window of anaesthesia depth which will induce a child to snore.

Anaesthetic management

Optimal anaesthetic management for children undergoing adenotonsillectomy for OSAS is controversial. Pre-operative sedation is used cautiously, and avoided if possible in children with suspected OSAS. During anaesthetic induction, paediatric patients with OSAS are at high risk of airway obstruction, desaturation and laryngospasm.[44] Trace volatile anaesthetic agents may further depress pre-existing abnormal ventilatory drive and potentiate airway obstruction due to reduced function of the airway muscles.[44] Benzodiazepines and opioids may have prolonged effects on respiratory function and may significantly exacerbate the situation, but there is little specific evidence for or against the peri-operative use of opioids or sedatives in children with OSAS.[45] Although inhalational anaesthesia and fentanyl resulted in respiratory depression in one study,[46] anaesthetic techniques including opioid use did not lead to increased post-operative respiratory complications in others.[35,45,47,48] The use of pethidine, compared with nalbuphine (an opioid with a ceiling effect for respiratory depression), did not result in greater post-operative oxygen desaturation in these children.[49] Nonetheless, many anaesthetists prefer not to administer opioids until the patient has fully emerged from the anaesthetic, regained spontaneous ventilation, has been extubated, and can maintain a spontaneous airway and gas exchange.[44,50] Opioids in reduced amounts may subsequently be titrated carefully to the patient's pain threshold and respiratory status.

Post-operative respiratory morbidity

After adenotonsillectomy, even when extubated fully 'awake', children with OSAS have a 16–27 per cent risk of post-operative respiratory complications, compared with 0–1.3 per cent in the general paediatric population.[35,47] They are more vulnerable to respiratory compromise post-operatively, due to upper airway oedema and increased oropharyngeal secretions.[14] Respiratory depression secondary to analgesic and anaesthetic agents may persist well after awakening, as these patients have an abnormal ventilatory response to CO_2.[15] Moreover, OSAS patients are at risk of post-operative pulmonary oedema.[51] Long-standing partial airway obstruction from enlarged tonsils and adenoids acts as a natural form of positive end-expiratory pressure (PEEP). With sudden relief of PEEP, transudation of fluid into interstitial and alveolar spaces occurs, resulting in pulmonary oedema, which may require reintubation.[52]

Identifying those at risk: moderate and high risk patients

To date, the most important predictors of post-adenotonsillectomy respiratory morbidity are OSAS severity and the presence of coexisting medical conditions.[37] We have classified the different risk factors into moderate and high risk subsets (Figure 2). Patients in the high risk group should

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undergo surgery at paediatric specialist centres with intensive care facilities, whilst those in the moderate risk group may undergo adenotonsillectomy at their local hospital, provided facilities for administering CPAP are available on-site. Close post-operative overnight observation, with apnoea monitoring and pulse oximetry, is mandatory for both moderate and high risk subgroups so that, if necessary, nasal CPAP or bilevel positive airway pressure may be administered to avoid reintubation.[53,54] For district general hospitals without such on-site facilities, it may be more appropriate to refer high risk patients pre-operatively to tertiary referral centres with the facilities to administer CPAP or bilevel positive airway pressure in the event of post-operative respiratory compromise (Figure 2). Pre-operative referral is intended to avert the possibility of emergency reintubation and subsequent delayed transfer to a paediatric unit due to unavailable intensive care beds.

The risk of post-operative respiratory complications is increased in very young children, due to their relatively large tongue, smaller mandible and neuromotor immaturity.[14,47,48,55,56] Maturational differences in the respiratory system of the toddler, including a compliant chest wall and asymptomatic pulmonary hypertension, may exacerbate desaturation in obstructive apnoea.[12] Most studies have found that children younger than three years are at increased risk of post-operative respiratory

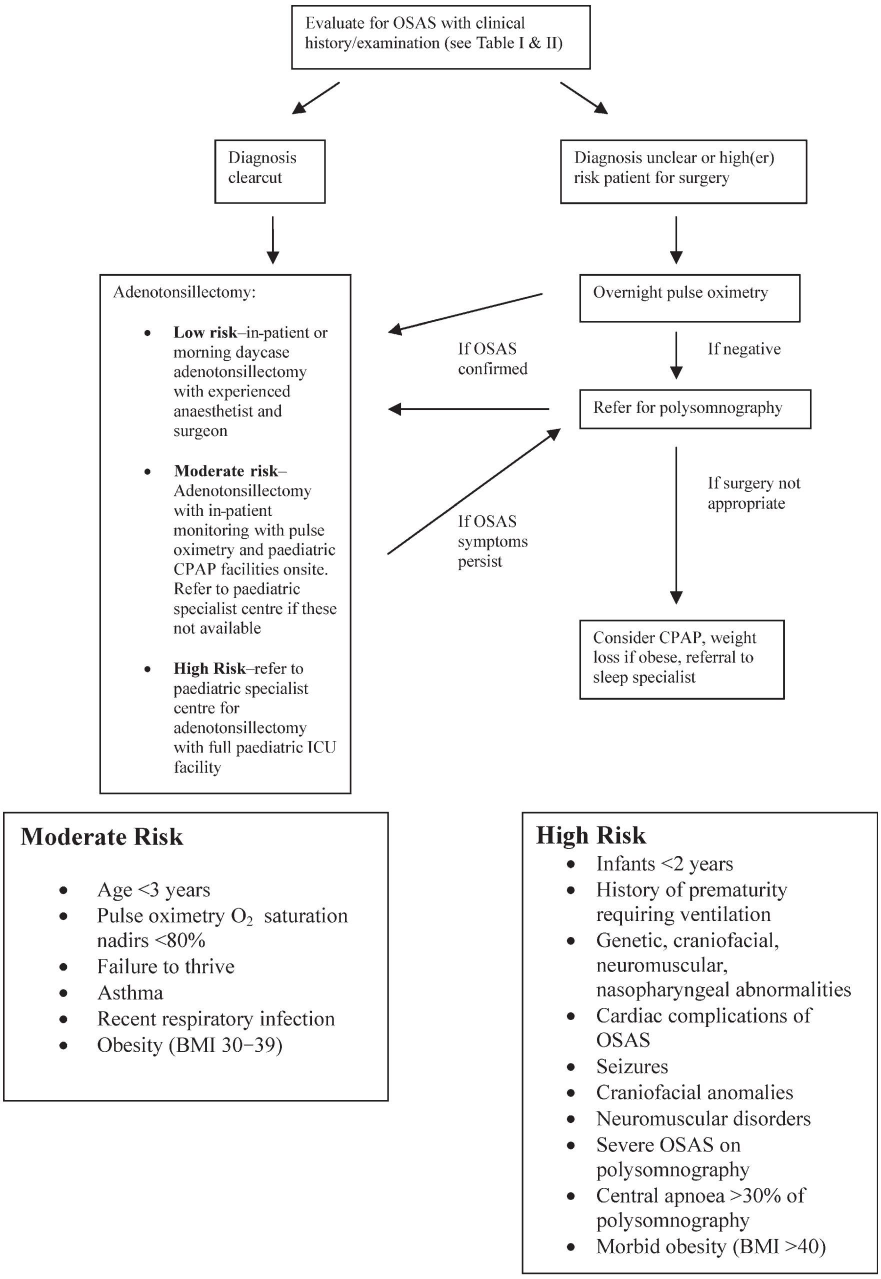

FIG. 2

Suggested approach to diagnosis and management of childhood obstructive sleep apnoea syndrome (OSAS). CPAP = continuous positive airway pressure; ICU = intensive care unit; BMI = body mass index compromise;[14,47,55–58] others have quoted the age group at risk as two years and younger.[35,48] Those with pulmonary hypertension or other cardiac abnormalities, craniofacial syndromes, neuromuscular disorders (e.g. cerebral palsy), seizures, failure to thrive, and morbid obesity (body mass index >40) are at high risk of post-operative respiratory morbidity (Table III).[35,47,48,55,59] As a result, routine in-patient admission for overnight observation, with apnoea monitoring and pulse oximetry, has been recommended for these risk groups. Obesity (body mass index 30–39) is only weakly correlated with OSAS severity, hence its inclusion in the moderate risk subset (Figure 2).[60] A history of premature neonatal ventilatory support is associated with a higher risk of post-adenotonsillectomy complications, possibly due to the increased incidence of craniofacial abnormalities and chronic lung disease in premature infants.[61] A pre-existing history of asthma is associated with an increased risk of post-operative respiratory complications in children, but the precise relationship between different severities of asthma and OSAS remains unclear.[62]

Pre-operative polysomnography may help to identify those at risk if a severe apnoea–hypopnoea index ($\geq$20) is detected.[30] Elective admission to paediatric intensive care facilities has been suggested for children with OSAS associated with central apnoea accounting for >30 per cent of total respiratory events detected on polysomnography.[14] Although polysomnography is almost 100 per cent sensitive, relying on polysomnographic severity alone may ultimately only predict 25 per cent of high risk patients.[35]

The low risk majority

Children without the risk factors previously discussed do not necessarily require intensive monitoring.[45] These children make up the majority of those who undergo adenotonsillectomy for OSAS. Operations should be undertaken in the morning, and may, in the absence of other risk factors, be performed as daycase procedures.[59,63] Children who undergo adenotonsillectomy in the afternoon are more likely to desaturate post-operatively than those undergoing surgery in the morning, possibly due to the shorter recovery time from the effects of anaesthesia before their first post-operative nocturnal REM sleep.[64] Safe practice therefore recommends that patients undergoing surgery within afternoon theatre lists should stay in hospital for overnight observation.

TABLE III

REPORTED RISK FACTORS FOR POST-OPERATIVE RESPIRATORY COMPLICATIONS IN CHILDREN WITH OSAS UNDERGOING ADENOTONSILLECTOMY

Age <3 years
History of prematurity requiring ventilation
Failure to thrive
Obesity
Asthma
Recent respiratory infection
Pulse oximetry O_2 saturation nadirs <80 per cent
Severe OSAS on polysomnography
Cardiac complications of OSAS
Seizures
Craniofacial anomalies
Neuromuscular disorders (e.g. cerebral palsy)

OSAS = obstructive sleep apnoea syndrome

Conclusion

Identifying children at risk of post-operative respiratory complications after adenotonsillectomy for OSAS remains challenging, especially at district general hospitals, where access to sleep laboratories and paediatric intensive care facilities are often limited. Based on available evidence, we propose a pragmatic approach to diagnosis and management, by classifying those at risk of post-operative respiratory complications into different risk subsets, based on the most important predictors – coexisting medical conditions and OSAS severity. Candidates classified as high risk can then undergo adenotonsillectomy with appropriate monitoring and back-up facilities. Controversy still surrounds the issue of optimal anaesthetic management for these children; there is little specific evidence for or against the peri-operative use of opioids or sedatives in children with OSAS. The impact of aggressive pre-operative preparation (including administration of antibiotics, bronchodilators, and oral or nasal steroids) as a risk reduction strategy to decrease respiratory morbidity certainly merits future study. Fortunately, the large majority of children with OSAS are low risk candidates who may safely undergo adenotonsillectomy at their local district general hospital.

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