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Main Article

Accuracy of clinical assessment of paediatric obstructive sleep apnoea in two English centres

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Abstract

Objectives: To ascertain the sensitivity and specificity of clinical diagnosis of obstructive sleep apnoea in children, and to determine if a published clinical algorithm identifies those at high risk of post-adenotonsillectomy complications.

Method: Sixty-seven children aged three to eight years underwent clinical assessment and overnight polysomnography.

Results: Polysomnography detected a significant apnoea–hypopnoea index (i.e. ≥5, indicating significant obstructive sleep apnoea) in 13 (43 per cent) children with a clinical diagnosis of obstructive sleep apnoea and in six (19 per cent) children with no such diagnosis. The sensitivity of clinical assessment was 68.4 per cent and the specificity 59.5 per cent. The post-operative risk algorithm failed to identify any high risk children, although in actuality seven had severe obstructive sleep apnoea confirmed by polysomnography.

Conclusions: This study of two English centres confirms that the clinical diagnostic process for obstructive sleep apnoea is reasonably insensitive and has low specificity. The studied algorithm discriminated poorly between children with and without severe obstructive sleep apnoea. Realistic diagnostic screening guidelines for paediatric sleep apnoea are overdue in the UK, where access to polysomnography is limited.

Key words: Sleep Disordered Breathing; Obstructive Sleep Apnoea; Clinical Diagnosis; Sleep Disordered Breathing Questionnaire; Polysomnography; Respiratory Complications; Risk; Adenotonsillectomy

Introduction

Obstructive sleep apnoea (OSA) is characterised by repetitive partial or complete collapse of the pharyngeal airway (resulting in hypopnoea or apnoea, respectively) in the face of continued respiratory effort. Nocturnal hypopnoea and apnoea can result in episodic hypoxic hypercapnia as well as repeated arousals from sleep. Population studies suggest a prevalence of paediatric OSA of between 0.7 and 1.8 per cent.[1,2] The major cause in childhood is disproportionate adenotonsillar growth relative to airway calibre,[3] although body mass, neuromuscular control and craniofacial anatomy contribute in some children. Treatment with adenotonsillectomy gives polysomnographic resolution of OSA in 79–100 per cent of otherwise healthy children.[4,5] Severe, untreated OSA in the paediatric population can result in cor pulmonale, failure to thrive, permanent neurological damage and developmental delay.[6] New knowledge indicates that even mild obstruction in sleep can impair neurobehavioural and cerebrovascular function.[7] It is therefore important that the right children are diagnosed and treated. The diagnostic challenge is to distinguish children with OSA from those with primary snoring, which has a prevalence of 12 per cent.[2]

In the US, the agreed 'gold standard' for the diagnosis of paediatric OSA is polysomnography.[8] The Scottish Intercollegiate Guidelines Network suggests limited sleep studies as the minimum investigation to enable a diagnosis of adult OSA in the UK.[9] Limited sleep studies usually include some measurement of respiratory effort, often with an indirect measure of arousal such as video monitoring. A common combination of parameters comprises airflow, thoraco-abdominal movement, oximetry and heart rate measurement, sometimes supplemented with measures of snoring.

Currently, no UK guidelines exist for the diagnosis of paediatric OSA. Many otolaryngologists worldwide recognise that carrying out in-patient

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polysomnography on all children suspected of OSA is too time-consuming and costly.[10] Furthermore, there is a paucity of paediatric polysomnography facilities in the UK (CM Hill unpublished data).[11] Pragmatic approaches have been suggested in the UK, with the selective use of polysomnography in cases with a complex history or a high post-operative risk.[12]

Several studies have shown great variability when comparing paediatric OSA diagnoses made by history and clinical examination alone versus those made by polysomnography; some research has indicated detection of only one-third of cases using clinical method alone.[13–23] None of these studies was carried out in the UK. Pulse oximetry is thought to have a 97 per cent positive predictive value for OSA if periodic clusters of desaturations of less than 90 per cent are found. However, a negative overnight pulse oximetry recording does not rule out OSA, thus reducing the utility of pulse oximetry as a useful screening test.[24]

Alternative abbreviated diagnostic screening tools for OSA are available, and include respiratory inductance plethysmography, oxygen saturation, electrocardiography (ECG) and video monitoring. One study assessed the use of abbreviated polysomnography in a small number of children, and found a sensitivity and specificity of 100 per cent for the detection of an apnoea–hypopnoea index (AHI) of >3.[25] In the same study, an AHI of >1 was detected with a sensitivity of 92 per cent and a specificity of 100 per cent, compared with full polysomnography.[25] Another study found that an experienced observer assessing home video and audio recordings could detect paediatric OSA with a sensitivity of 94 per cent but with a poor specificity (68 per cent).[26] Audiotaping does not give consistent results,[27] and nap studies are not sensitive but have a high positive predictive value.[28,29]

In the absence of widely available respiratory diagnostic facilities, UK clinicians usually rely on clinical examination and pulse oximetry to aid diagnosis. Recently published clinical algorithms have also used these readily available parameters to select children likely to be at high risk of post-adenotonsillectomy complications.[10] There is a recognised risk of post-adenotonsillectomy respiratory complications (e.g. prolonged desaturation and hypercapnia) in children with an AHI of >10, especially in patients younger than three years.[30,31]

This study was conducted as part of a prospective investigation of neurocognitive function in children with sleep-disordered breathing. This provided an opportunity to examine the quality of clinical assessment of paediatric OSA in the context of limited diagnostic resources in a UK setting.

Specifically, we aimed: (1) to determine the effectiveness of clinical assessment, alone or in concert with a screening questionnaire plus overnight pulse oximetry, to identify paediatric OSA, compared with that of polysomnography; and (2) to test a recently published clinical algorithm by Leong and Davis, designed to predict post-operative risk in such children (see Appendix 1).[12]

Materials and methods

Subjects and clinical evaluation

Children were enrolled in two consecutive studies from 2005 to 2007. They were recruited from the waiting lists of two hospitals on the south coast of England, Southampton University Hospitals NHS Trust and Queen Alexandra Hospital, Portsmouth. Children were aged three to eight years and had no evidence of craniofacial abnormalities, neuromuscular disorders, moderate or severe learning disabilities, chronic respiratory or cardiac conditions, or allergic rhinitis. All children had a history of snoring and were either: (1) listed for adenotonsillectomy to treat OSA; (2) listed for adenotonsillectomy or tonsillectomy for other indications; or (3) listed for adenoidectomy with or without grommet insertion, with no history of OSA.

These children had been evaluated and a decision regarding surgery had been made in normal clinical conditions, reflecting current practice. The main indication for surgery and presenting symptoms were obtained from detailed evaluation of the clinical records.

All parents consented to their child's enrolment in the studies, after careful explanation of the method and aims.

Pediatric Sleep Questionnaire

The Pediatric Sleep Questionnaire was completed by children's parents.

This validated questionnaire has 22 items (see Appendix 2) documenting the presence or absence of common symptoms such as snoring, observed apnoeas, breathing difficulty during sleep, daytime sleepiness, and inattentive or hyperactive behaviour. Positive responses are scored as 1 and negative responses as 0. The over all score is divided by 22 to give an end value. The Pediatric Sleep Questionnaire has been reported to have a sensitivity of between 0.81 and 0.85 and a specificity of 0.87 in detecting OSA, compared with polysomnographic diagnosis.[32] A cut-off value of $>$ seven positive responses is thought to be most effective in identifying OSA.[25] Subscales within the Pediatric Sleep Questionnaire include a four-item sleepiness scale, a four-item snoring scale and a six-item inattention–hyperactivity scale.

The Pediatric Sleep Questionnaire was only available to researchers in this study, not clinicians, and therefore did not influence clinical assessment.

Polysomnography

All children underwent one night of attended polysomnography in a purpose-built sleep laboratory using computerised systems (Embla system and Somnologica Studio software, Medcare Flaga, Reykjavik, Iceland, and Alice 5 system, Respironics, Chichester, UK), according to American Thoracic Society standards.[33] A standard montage was recorded, including: encephalography (10/20 electroencephalography lead placement C3/A2, O1/A2, C4/A1, O2/A1); right and left electro-oculogram (EOG); submental electromyography (EMG); diaphragmatic EMG; thoracic

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and abdominal excursions; nasal airflow (Protech, Mukilteo, WA); finger pulse oximetry (Nonin and Masimo technologies; Nonin Medical Inc, Plymouth Minesota, USA and Masimo Inc, Irvine, CA, USA), ECG; and synchronous video-recording. Obstructive apnoea was defined as the presence of chest or abdominal wall movement with no airflow, or airflow decreased by more than 80 per cent compared with the previous breath, for two or more breaths. Hypopnoeic episodes were classified as for apnoeic episodes, but with a flow reduction of 50–80 per cent compared with the previous breath, with an associated oxygen desaturation or respiratory arousal. Oxygen desaturation was classified as a 3 per cent or more decrease in oxygen saturation from the baseline. The AHI was defined as the number of episodes of obstructive apnoea, hypopnoea and mixed apnoea per hour of total sleep time. An AHI of >5 was defined as significant OSA.[34] The 2008 revised International Classification of Sleep Disorders recommended that paediatric OSA be diagnosed in the presence of an AHI of >1; therefore, data were also analysed using this criteria.[35]

Post-operative risk

Using clinical information extracted from case files along with oxygen saturation values from overnight polysomnography studies, children were placed in a post-operative risk category of mild, moderate or severe, as directed by a recently published algorithm (Appendix 1). Any post-operative problems were identified by a retrospective review of the hospital notes, including anaesthetic charts.

Statistical methods

Data were analysed using the Statistical Package for the Social Sciences version 15 software. Continuous data were explored for normality using the Shapiro Wilk test. Parametric data were described using means and standard deviations (SDs), and non-parametric data using medians and ranges. Sensitivity (i.e. the proportion of true positives correctly identified) was assessed using the formula positives/true positives. Specificity (i.e. the proportion of true negatives correctly identified) was assessed using the formula negatives/true negatives.

Results and analysis

Sixty-seven children aged three to eight years old participated in the study (mean age 4.3 years, SD 1.2 years; 40 males, 27 females). Six children where excluded, five as their sleep study failed to record adequate data, and one whose hospital notes were unavailable for review.

Sensitivity and specificity of clinical diagnosis

Of the 61 children included, the majority ($n = 48$) underwent adenotonsillectomy with or without grommets insertion. Of these 48 children, the main clinical indication for surgery was OSA in 27 and recurrent tonsillitis in 21. A further three children underwent tonsillectomy alone for recurrent tonsillitis. Eight children underwent adenoidectomy and grommet insertion for hearing loss, and one child had this procedure for recurrent ear infections. One child underwent adenoidectomy for nasal obstruction.

TABLE I

CLINICAL *VS* POLYSOMNOGRAPHIC OSA DIAGNOSIS

Clinical diagnosis of OSA?	PSG AHI result			Total
	<1	1–4.9	≥5	
Yes	4	13	13	30
No	8	17	6	31
Total	12	30	19	61

Data represent number of patients. OSA = obstructive sleep apnoea; PSG AHI = polysomnographic apnoea–hypopnoea index

Of those children with a clinical diagnosis of OSA, 13 (43 per cent) had an AHI of ≥5, 13 (43 per cent) had an AHI of 1–4.9 and four had a normal AHI. Conversely, six children with no clinical suspicion of OSA had a significant AHI (i.e. ≥5), and a further 17 had an AHI of 1–4.9 (see Table I). The median AHI of those with a clinical diagnosis of OSA was 4.6 ($n = 30$, range 0.4–41.7), compared with an AHI of 2.1 ($n = 31$, range 0–10.9) in those with no clinical diagnosis of OSA. Thus, a clinical diagnosis of OSA using AHI diagnostic thresholds of ≥5 and ≥1 (used in isolation) yielded sensitivities of 68.4 and 53.1 per cent and specificities of 59.5 and 66.6 per cent, respectively, compared with polysomnographic findings.

Pediatric Sleep Questionnaire sensitivity and specificity

Fifty-eight out of the 61 children had a completed Pediatric Sleep Questionnaire. Eighty-four per cent of these children ($n = 49$) had a significant score on this questionnaire, but only 30.6 per cent of those significant ($n = 15$) had OSA confirmed by polysomnography using an AHI threshold of ≥5 (see Table II). The Pediatric Sleep Questionnaire scale had sensitivities of 88.2 and 84.8 per cent and specificities of 17.1 and 16.6 per cent, using polysomnographic AHI thresholds of ≥5 and ≥1, respectively. The snoring and sleepiness subscales, when examined separately, did not improve the specificity of the questionnaire.

The use of either a clinical diagnosis or a positive Pediatric Sleep Questionnaire resulted in even more children being allocated to the likely OSA

TABLE II

PEDIATRIC SLEEP QUESTIONNAIRE *VS* POLYSOMNOGRAPHY

PSQ result	PSG AHI result			Total
	<1	1–4.9	≥5	
Missing	1	0	2	3
Not significant	2	5	2	9
Significant	10	24	15	49
Total	13	29	19	61

Data represent number of patients. PSQ = Pediatric Sleep Questionnaire; PSG AHI = polysomnographic apnoea–hypopnoea index

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group (85 per cent), based on an AHI of ≥5. This gave an increased sensitivity of 89.4 per cent but an unchanged, high false positive rate (17.1 per cent).

Predictive value of nocturnal pulse oximetry

Mean and minimum overnight oxygen saturation data were available for all children as part of their polysomnography study. Verhulst *et al.* reported a mean overnight oxygen saturation value of 97.0 per cent (SD 0.6, range 96.0–98.0) and a mean minimum value of 91.8 per cent (SD 2.7, range 82.0–96.0) in 60 European children aged six to 16 years with no polysomnographic evidence of sleep-disordered breathing.[36] Traeger *et al.* reported similar values in 66 North American children aged two to nine years: mean oxygen saturation 97.1 per cent (SD 1.0, range 95–98) and minimum oxygen saturation 92.0 per cent (SD 3.0, range 81–95).[37] These studies were used to determine an acceptable mean overnight oxygen saturation threshold of ≤95 per cent and a minimum oxygen saturation threshold of <81 per cent. Using these criteria, 12 children screened positive on the basis of oximetry. However, importantly, these oxygen saturation parameters failed to identify seven children with a clinical diagnosis of OSA confirmed by polysomnography (using an AHI of ≥5). Within our clinical population, the mean overnight oxygen saturation was 97.0 per cent (SD 1.2, range 93–99) and the mean minimum oxygen saturation was 87.6 per cent (SD 8.6, range 40–96).

Identification of post-operative risk

Using the algorithm recommended by Leong *et al.*, none of the children in our study fell into the high post-operative risk category, despite seven having AHIs of >20.[12] Nine fell into the moderate risk category, six children due to concomitant asthma. One child had failure to thrive, one had a recent respiratory infection and one had desaturations of 40 per cent on overnight oximetry. Furthermore, five children categorised as mild risk had AHIs of >10 (see Table III). Therefore, the algorithm failed to identify those children determined by polysomnographic criteria to be at high risk.

Five children had post-operative complications: one had a primary haemorrhage, two secondary haemorrhages and two post-operative infections. However, no child suffered respiratory complications post-operatively. All of the children who experienced post-operative complications were in the mild risk category as assigned by the algorithm.

TABLE III

ALGORITHM *VS* POLYSOMNOGRAPHY

Algorithm risk category	PSG AHI result		Total
	<10	>10	
Mild	47	5	52
Moderate	3	6	9
Total	50	11	61

Data represent number of patients. PSG AHI = polysomnographic apnoea–hypopnoea index

Discussion

To the authors' knowledge, this is the first published study to examine contemporary UK practice regarding clinical diagnosis of paediatric OSA. Our sampling method allowed a naturalistic observation of practice across two large hospitals, and the results indicate that the current reliance on readily available standard tools to establish a paediatric OSA diagnosis has limitations. Only 43 per cent of those predicted to have OSA had an AHI of ≥5, a threshold that most UK centres would deem appropriate for surgical management, and a value that is below chance. The sensitivity of clinical diagnosis alone was 68.4 per cent, with a specificity of 59.5 per cent. Adjusting the diagnostic threshold to an AHI of ≥1 reduced the sensitivity of clinical diagnosis, although the specificity was somewhat improved. These results are comparable with those of previous studies assessing paediatric populations in the US, India and Finland, which demonstrated a diagnostic clinical accuracy of 30–73 per cent.[13]

Of particular interest is the finding that 19 per cent of children not clinically identified as having OSA had an AHI of ≥5. Some of these children were listed for adenotonsillectomy or tonsillectomy for other indications, and hence would have been fortuitously treated for their sleep-disordered breathing. However, 20 per cent of children listed for grommet insertion with adenoidectomy were found to have OSA on polysomnographic investigation, and these two children may also have benefited from tonsillectomy had their diagnosis been established pre-operatively.

An encouraging aspect of our findings was that the children with the most severe OSA (i.e. AHI of >20) were all positively identified by clinical assessment alone. However, research has identified cardiovascular and neurocognitive changes in children with even mild sleep-disordered breathing.[7,38] In the light of such challenges to current treatment thresholds, it is important that children with mild and moderate OSA are also positively identified.

The Pediatric Sleep Questionnaire has been previously reported to have a sensitivity of 81 per cent and a specificity of 87 per cent in predicting OSA in children aged two to 18 years.[24] In this study, 84 per cent of subjects scored in the clinical range, generating a comparable sensitivity (88.2 per cent) but very poor specificity (17.1 per cent). The difference in specificity in our data may reflect sampling bias. All children recruited to these studies were selected as known snorers. As the Pediatric Sleep Questionnaire contains three questions about snoring, this may explain its high false positive rate. Nonetheless, this is a concern, as it is these very children who would potentially benefit from a screening questionnaire. Combining the Pediatric Sleep Questionnaire and clinical diagnosis increased the sensitivity of diagnosis (to 89.4 per cent) but did not change the overall specificity of diagnosis – that is, the false positive rate remained unacceptably high. This shows the validity of the questionnaire as an initial screening tool for children at risk of OSA, but indicates that it has limited additional value beyond standard clinical assessment.

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An important limitation of our analysis is the fact that only mean and minimum overnight oxygen saturation values were available. Where oximetry is used alone for assessment of OSA, multiple parameter assessment is recommended – specifically, visual assessment of pulse and oxygen saturation traces for desaturation episodes (which in experienced hands can have a positive predictive value of 97 per cent)[24] along with measures of baseline oxygen saturation variability.[39]

However, even where state-of-the-art, motion-resistant pulse oximeters are used and studies are analysed by experienced practitioners, OSA can be missed.[40] There are a number of reasons why pulse oximetry may provide false reassurance. Firstly, if children are assessed in a hospital environment they may sleep poorly. Children are most likely to obstruct their airways during later rapid eye movement sleep cycles, which usually occur towards the end of the night. These cycles may be absent if the child has a very short sleep period. Furthermore, children may respond to apnoeic episodes by brief arousals from sleep rather than significant oxygen desaturation. The abrupt change from sleep to a waking state reverses muscular hypotonia and hence airway obstruction. These repeated arousals are clearly observed during polysomnography but are missed by oximetry alone. The lack of oxygen desaturation in this situation cannot necessarily be interpreted as reassuring, as repeated arousals cause fragmentation of sleep with deleterious neurocognitive consequences.[41] This was in part supported by our data: the majority of children with significant OSA determined by AHI had mean oxygen saturation values in the normal range. Thus, children with clinically suspected OSA and negative overnight pulse oximetry should undergo further respiratory studies.

Previous work has suggested that the most significant risk factors for respiratory compromise after surgery are an age below three years and an AHI of >10.[9] Abnormal ECG or echocardiogram results, a weight less than the fifth percentile, and craniofacial abnormalities are also related to a higher risk. Post-operative respiratory complication rates of between 16 and 27 per cent have been reported.[11]

The algorithm suggested by Leong *et al.*[12] allocates children to risk categories in order to guide decisions about post-operative care in a UK setting, where polysomnography is not readily available. The algorithm takes account of factors associated with a higher risk of respiratory compromise, such as failure to thrive, an age of less than two years and craniofacial abnormalities. It was notable that, in the current study population, a significant number of children allocated to the low risk category had an AHI of >10, a value associated with post-operative risk. This is best illustrated by one child who was classified by the algorithm as being low risk on the basis of available clinical data, who in actuality had an AHI of 39.4 and required post-operative intensive therapy unit observation. Thus, high risk children may be missed by the recommended algorithm in institutions where polysomnography is not available.

Another limitation of this study was the fact that the research polysomnography results of some children were available to their surgeons pre-operatively, and may have expedited surgery and influenced peri- and post-operative care. This may explain the lack of post-operative respiratory compromise in our study group. Alternatively, respiratory compromise may be less prevalent than previously supposed, due to more careful anaesthetic techniques.

- **Facilities for overnight paediatric polysomnography are sparse in the UK**
- **Studies in other countries have shown clinical diagnosis of paediatric obstructive sleep apnoea to be unreliable**
- **In the UK, no guidelines exist for the diagnosis of paediatric obstructive sleep apnoea**
- **In this study of two English centres, clinical diagnosis of paediatric obstructive sleep apnoea was not reliable**
- **Use of the Pediatric Sleep Questionnaire and mean overnight pulse oximetry do not improve the accuracy of diagnosis**
- **A diagnostic algorithm adopting a pragmatic approach to risk identification missed children at high risk (as defined by polysomnographic respiratory parameters)**

Finally, it is possible that selection bias operated in this research and that the sample was unrepresentative of all children on the waiting list. Nonetheless, these data had inherent validity, as clinical diagnoses had been documented and surgical plans determined prior to the offer of polysomnographic study. It is equally feasible that there are many more children with undiagnosed OSA either under out-patient review or indeed in primary care. Research has shown that 40 per cent of children with proven OSA have to self-refer, despite general practitioners being aware of the relevant symptoms.[42] In one study of general paediatric clinics, sleep problems were only detected in 15 per cent of children in whom they were present.[43]

Conclusion

Our study findings were consistent with those of international studies in demonstrating a low specificity of clinical diagnosis, with or without screening questionnaires, in detecting OSA in children, when compared with polysomnography. Mean oxygen saturation values alone did not add to the sensitivity or specificity of assessment. Our study showed that the clinical diagnosis of paediatric OSA in two English paediatric centres using commonly available tools was inaccurate. Current American Academy of Pediatrics guidelines recommend polysomnography as the only method which quantifies ventilatory and sleep abnormalities, and as the diagnostic test of choice in all children. In the UK, facilities for full polysomnography are limited. Realistic assessment guidelines, which recognise current resource limitations within the UK and the merits of alternative respiratory diagnostic techniques, are overdue.

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Appendix 1. Suggested approach to diagnosis and management of childhood obstructive sleep apnoea syndrome

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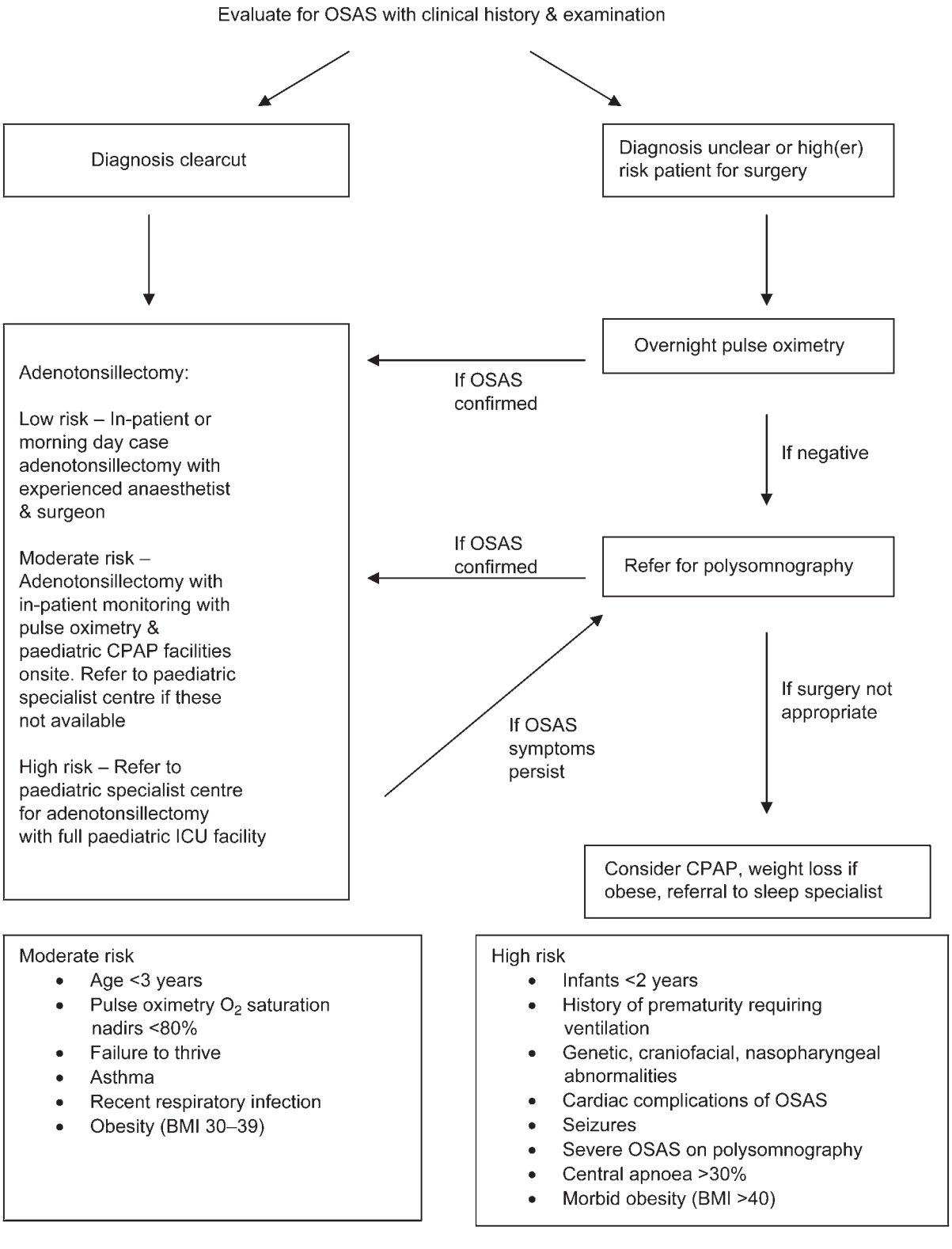

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Appendix 2. Pediatric Sleep Related Breathing Disorder Questionnaire

Child's name_____Date completed_____

While sleeping does your child ... (please delete incorrect answer)

(1) Snore more than half the time? Yes/No
(2) Always snore? Y/N
(3) Snore loudly? Y/N
(4) Have 'heavy' or loud breathing? Y/N
(5) Have trouble breathing, or struggle to breathe? Y/N
(6) Have you ever seen your child stop breathing during the night? Y/N

Does your child...

(7) Tend to breathe through the mouth during the day? Y/N
(8) Have a dry mouth on waking up in the morning? Y/N
(9) Occasionally wet the bed? Y/N
(10) Wake up feeling unrefreshed in the morning? Y/N
(11) Have a problem with sleepiness during the day? Y/N
(12) Has a teacher or other supervisor commented that your child appears sleepy during the day? Y/N
(13) Is it hard to wake your child up in the morning? Y/N
(14) Does your child wake up with headaches in the morning? Y/N
(15) Did your child stop growing at a normal rate at any time since birth? Y/N
(16) Is your child overweight? Y/N

My child often...

(17) Does not seem to listen when spoken to directly Y/N
(18) Has difficulty organising tasks and activities Y/N
(19) Is easily distracted by extraneous stimuli Y/N
(20) Fidgets with hands or feet or squirms in seat Y/N
(21) Is 'on the go' or often acts as if 'driven by a motor' Y/N
(22) Interrupts or intrudes on others (e.g. butts into conversation or games) Y/N

Subscales: questions 1–4 = snoring; 10–13 = sleepiness; 17–22 = behavioural).

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