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Investigation of the Child Who Snores

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Abstract

Snoring is common in childhood and can be a symptom of a serious sleep disorder with a myriad number of potentially serious consequences. It is important to take the symptom seriously. Performing a careful history and examination can help direct the clinician to appropriate investigations and treatment.

Introduction

In the last two decades there has been a growing awareness of the importance of sleep in health and disease. Particularly in children there is an increasing understanding that both sleep restriction and sleep fragmentation can have a deep and wide-ranging impact on infants and children. *Questions about sleep and snoring should form part of the routine health screening of all children.* Current research is focused on the relationship between sleep disorders and neurocognitive effects in children, including attention, concentration, learning and behaviour.

Even though obstructive sleep apnoea (OSA) has long been described in literature, it was not until the 1980's the reports started to appear of children with OSA. In 1981 the use of nasal mask continuous positive airway pressure (CPAP) support for OSA was described by Professor Colin Sullivan and others at the University of Sydney. The field took off from there but it was not until almost a decade later that full clinical sleep studies or polysomnography (PSG) were first performed on infants and children.

Statistics indicate that there has been a sharp increase in PSG's performed in both adults and children (age 12 years and under) in the last decade. About a third of children have reported sleep problems and double that number in children with developmental needs. Despite this Australian research indicates that parents seldom bring up the issue of sleep problems with their general practitioners. In turn GP's also seem reluctant to ask families about sleep problems.

Approach to the Snoring Child

History

Studies suggest that between 10 to 14% of children snore. The peak age of incidence tends to be between 2 and 4 years of age. This coincides with the relative large size of adenoids and tonsils in relation to the oro- and nasopharynx at that age. Snoring is a cardinal symptom of obstructive sleep apnoea (OSA). In adults snoring is often loud and when associated with daytime sleepiness and pauses in breathing in sleep, is strongly suggestive of OSA. However in children snoring is often softer and sometimes described as heavy breathing. It is different to stridor, which is high-pitched, more musical and worse in the day or when the child is upset..

Pauses in sleep breathing are often present but many parents might not be aware of the symptoms. The three most useful nocturnal symptoms of OSA in children are snoring on most nights, pauses in breathing and an increase in work of breathing. Where parents are unsure of symptoms it is worthwhile asking them to check again especially in the second half of the overnight sleep cycle. Other nocturnal symptoms include mouth breathing, sweatiness, restless sleep, unusual postures like neck hyperextension and bed-wetting especially when the child was previously dry overnight. Choking and vomiting are distressing symptoms and can be associated with apnoeas. Chronic nocturnal cough in children especially in the absence of wheeze can be the result of OSA. Parasomnias such as night terrors and sleep walking can be a result of partial arousal due to underlying OSA.

Daytime symptoms can also be non-specific and are thought to be the result of both sleep fragmentation as well as intermittent hypoxia. Excessive daytime sleepiness is uncommon in children, probably in 30% rather than the 90% or more in adults with OSA. In contrast, children with OSA often have hyperactive and difficult behaviour by day. They often have poor attention and concentration and academically underachieve. Mouth breathing in the day is a helpful symptom. Often there is a stertorous sound, like soft “awake snoring” likely due to enlarged adenoids. Poor appetite and sometimes dysphagia can be associated with childhood OSA, again due to large adenoids and tonsils. Headaches in the morning can result from fragmented sleep. A brief developmental history particularly in regard to language should be obtained.

A family history of snoring and OSA can be helpful. Good epidemiological research suggests that the younger the parents and the less obese they are when they snore, the higher the risk of OSA in the child. This is thought to be related to craniofacial characteristics that are inherited. Smoking in households seems to be a risk factor as well.

Physical Examination (PE)

It is important to get a baseline height and weight or even more helpfully, serial measurements on a growth chart. Unlike adults where obesity is a big contributing factor to OSA, many young children and infants with OSA have sub-optimal weight gain and linear growth. However the incidence of OSA is set to increase with the increasing incidence of childhood obesity.

In otherwise normal children, the most common cause of snoring and OSA is large tonsils and adenoids. Listen for nasal obstruction and stertor in wakefulness. An assessment or grading of tonsillar size in the oropharynx is helpful (Figure 1). If a tongue depressor is needed, try to leave this part of the examination last and avoid touching beyond the anterior one-third of the tongue to avoid a gag. It should be noted that relatively small tonsils *do not* in anyway exclude significant OSA. Adenoids are usually not visible through the oropharynx and can cause significant obstruction in sleep. Examine the anterior nasal cavity with an auriscope and note any swelling and pallor suggesting a degree of chronic rhinitis. Examine also the ear drums and note the presence of effusion as middle ear disease often accompanies childhood

OSA.

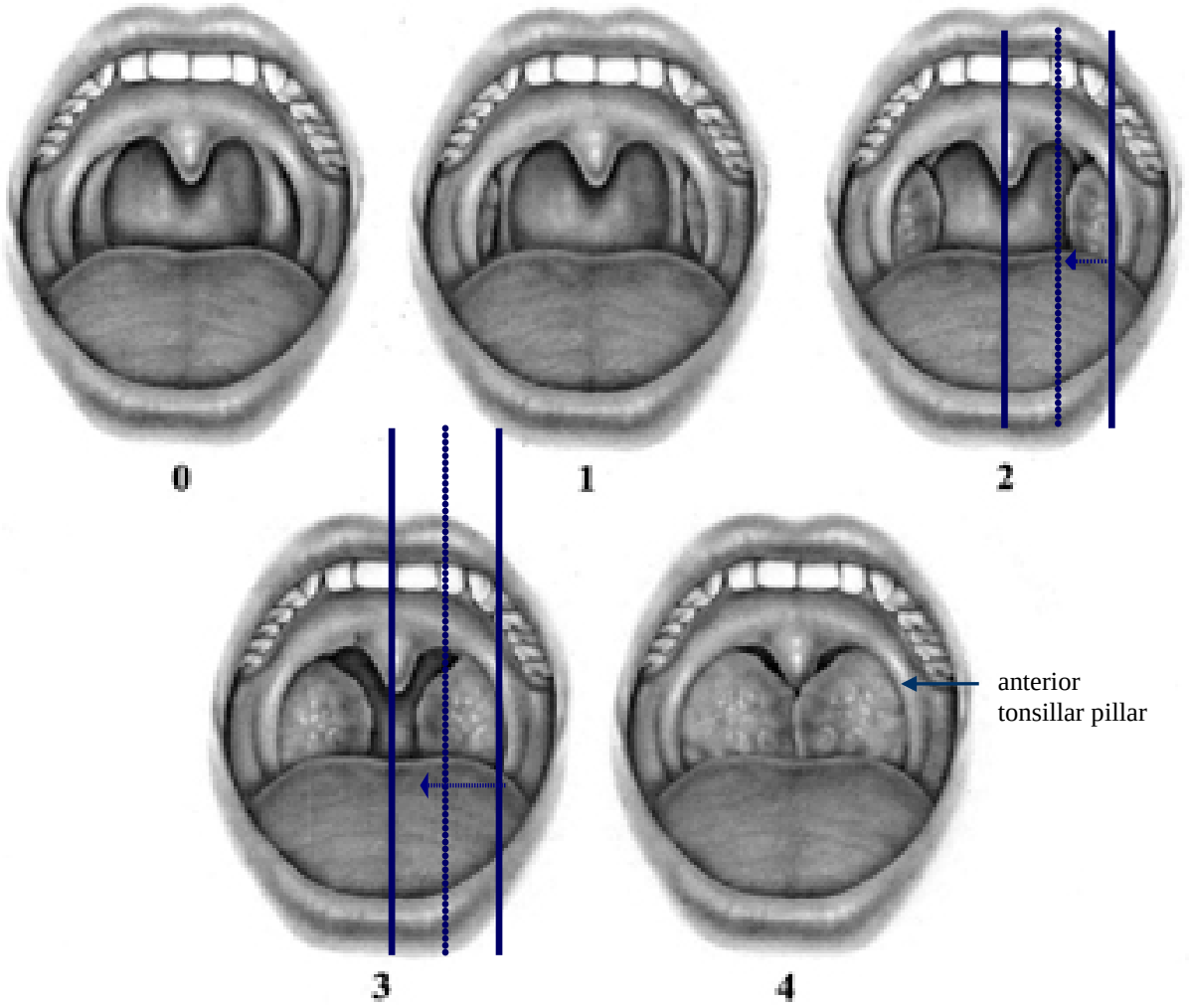

Figure 1. Grading tonsillar size (after Brodsky, *Pediatr Clin North Am* **1989;36**,1551-1569)

As an initial part of the PE it is important to note craniofacial characteristics that predispose to OSA. These include retrognathia and micrognathia, midfacial hypoplasia, a high arched palate and macroglossia. Where snoring might not be the initial presenting symptom, the astute clinician should always seek symptoms of OSA in children at risk.

These risk factors are set out in Table 1:

A. Craniofacial Syndromes:

- midfacial hypoplasia
  - Apert, Crouzon, Pfeiffer,
  - Treacher-Collins
- macroglossia/glossoptosis
  - Down Syndrome
  - Beckwith-Wiedeman
  - Robin sequence
- Other
  - Achondroplasia,Hallerrman-Streiff, Klipper-Feil, Goldenhar, Marfan

B. Neuromuscular Disorders

- cerebral palsy
- myasthenia gravis
- Möbius syndrome
- Muscular dystrophy

C. Postoperative disorders

- pharyngeal flap
- cleft lip repair

D. Miscellaneous disorders

- obesity
- Prader-Willi syndrome
- mucopolysaccharidosis
- sickle cell disease
- choanal atresia
- face and neck burns
- subglottic stenosis
- airway papillomatosis

Table 1. Risk factors for obstructive sleep apnoea in children

In Down Syndrome, for example, reports estimate that between 50 and 75% of children might have OSA. This is the result of a combination of risk factors like midfacial hypoplasia, microstomia and hypotonia.

The rest of the PE should assess possible complications of OSA. Look for chest deformities such as a pectus excavatum. Listen to the heart sounds especially for a loud second heart sound as pulmonary hypertension is a consequence of severe OSA. Where possible a blood pressure measurement should be attempted as systemic hypertension has been reported in childhood OSA and certainly is a major issue in adult OSA.

Investigations

What is a Sleep Study (PSG)?

A Sleep Study is an investigation that usually follows a consultative service with a qualified physician. This enables correct interpretation of the sleep study and the most appropriate treatment recommendations. As access to specialised paediatric laboratories might not be widely available, consultation also allows for prioritisation of the investigation. A careful sleep-related history including a sleep diary and careful examination can go a long way towards a diagnosis but the overnight PSG is considered the *gold standard* in the investigation of a wide spectrum of sleep disorders including sleep-disordered breathing (ranging from snoring to hypoventilation and apnoea), the titration of any non-invasive ventilatory or pressure support (including CPAP and bi-level pressure support), and the diagnosis of non-respiratory sleep disorders such as narcolepsy. It is an invaluable tool also for the diagnosis of parasomnias (such as night terrors and sleep-walking), sleep-related movement disorders such as periodic limb movement disorder (PLMD), and nocturnal seizure disorders.

Paediatric sleep studies are done across the entire age range from pre-term infants to teenagers. Cots or beds are used in specialised child-friendly sleep laboratories usually located within a major hospital. Access to after-hours medical and nursing staff apart from the sleep laboratory is of great advantage. The studies are often conducted by a combination of scientific and nursing staff with paediatric experience. Parents or other familiar carers are usually required to stay with the child at all times and accommodation usually in the same room is provided for the adult. Sometimes it takes an hour of more to

apply all the electrodes and sensors on the child and parents are actively recruited in the process. Toys and videos or other distractions are often employed to minimise any stress on the child. As far as possible most studies would try to adhere to the family's usual routine. Children are not sleep-deprived prior to the study and they are not sedated. Nap studies are not as reliable because some sleep stages, especially rapid eye movement or REM sleep, can be missed. Sleep-disordered breathing is much worse in REM sleep when there is a marked physiological decrease in airway dilator muscle tone, decreased or absent use of accessory muscles of respiration, decreased tidal volume and depressed chemoreceptor drive. Night to night variability is very low.

What is measured in a Sleep Study (PSG)?

A sleep study involves the continuous and simultaneous recording of multiple physiological parameters evaluating sleep and respiration. Sleep is assessed by the electroencephalogram (EEG), electrooculogram (EOG), and electromyogram (EMG). Respiration is assessed by measuring nasal and oral air flow, the former usually by pressure transducers with child- or infant-sized nasal prongs. These constitute probably the most uncomfortable part of the sleep study but are accepted as the best way to measure both the quantity as well as quality of the nasal flow. Oral airflow is usually measured by heat-sensitive sensors or thermistors. Respiratory effort is measured by movement transducers on the chest and abdomen, and/or respiratory muscle EMG, which are ECG type electrodes under the rib-cage and usually on the abdominal muscles. Pulse oximetry is used to measure oxy-haemoglobin saturation (SaO_2) through the nail bed. The averaging time of the oximeter should be short with artefact-minimising technology. Carbon dioxide (CO_2) is measured by transcutaneous and/or end

tidal CO_2. An electrocardiogram (ECG) is recorded with integrated heart rate. Other parameters that can be monitored are body position, leg movements (by anterior tibialis surface EMG) and snoring volume. The patient should be monitored by continuous infrared or low-light video, usually with remote control pan and tilt. In limited situations sometimes an arterial or capillary blood gas might be needed at the end of the study to confirm the accuracy of the non-invasively monitored parameters. This is particularly so when the study involves the titration of non-invasive ventilation settings.

What can a Sleep Study tell us?

The sleep study provides information about the sleep architecture through the standardised "scoring" or analysis of *sleep stages*. The report should contain the percentage of each stage of *REM sleep, light (non-REM) sleep* (stages 1 and 2) and *deep (non-REM) sleep* (stages 3 and 4). *Respiratory data* should include the baseline respiratory and heart rate, SaO_2, CO_2, including the lowest readings of the former and the range of the latter (should not rise by more than 13mmHg from wake to sleep). Respiratory events are scored when they are of more than one respiratory cycle in duration rather than of specific duration. These can either be central apnoea (absence or $\leq$ 20% of baseline respiratory effort and therefore oronasal airflow) usually with a drop of 3% of more of the SaO_2, or obstructive apnoea (continuing or increasing effort with absence or $\leq$ 20% airflow). Mixed apnoeas have a central as well as obstructive component. Respiratory events of both types can further be classified as central, obstructive of mixed hypopnoeas, where the airflow or effort is <50% but more than 20% of the preceding amplitude. The sleep report should express these events per hour of sleep such as an obstructive or mixed apnoea/hypopnoea index or a central

apnoea/hypopnoea index. *More than one obstructive apnoea per hour is considered abnormal in children and infants and adult criteria are not used.*

What are other investigations?

Lateral X-rays of the neck can provide useful information about the post-nasal space, size of the tonsils and adenoids. However as obstructive sleep apnoea is a dynamic process that occurs in sleep, the X-ray is unlikely to determine management. Patency of the vertical airways in wakefulness in no way excludes OSA. These X-rays might be helpful in determining the presence of remnant adenoidal tissue after previous adenoidectomy.

Overnight oximetry can sometimes help prioritise a PSG or even treatment, but must be used with caution as so-called normal pulse oximetry does not exclude significant sleep-disordered breathing. The morbidity associated with OSA is caused by not only the change in SaO_2 but also the concomitant sleep fragmentation and possibly high work of breathing itself. It is also important to be familiar with the averaging time and other limitations of the technology. A downloadable oximetry recording with characteristic cyclical desaturation might be more helpful. Unlike the PSG no information is available about sleep stages, work of breathing and other sleep disorders such as PLMD, which can impact on daytime clinical presentation.

Do all snoring children need a Sleep Study?

Adenotonsillectomy (TA) remains the treatment of choice for most children with OSA with a “cure rate” of over 80%. Rarely do children need CPAP, which is the treatment of choice for adults with OSA. Access to paediatric sleep laboratories can be limited

especially in regional areas. Children at both ends of the spectrum of snoring and sleep apnoea might need a PSG. On the mild end, where TA is not otherwise indicated, such as for recurrent tonsillitis (5 or 6 episodes per year), the PSG is helpful to help decide the need for the operation. At the severe end of the spectrum the PSG helps prioritise the timing as well as confirm the indication. More importantly post-operative complications are more common in severe OSA and close post-operative monitoring is mandatory. This is particular so for those very young children (less than 2 years) and those with risk factors (as in Table 1) or who have the effects of chronic OSA (such as pulmonary hypertension). It is recommended that such children be referred to paediatric sleep services as they are also the same group of patients who might not be cured by TA and might need further treatment for OSA. This usually takes the form of CPAP or more complex surgery. Careful follow-up is important. Rarely is tracheostomy needed but in the very severe cases and where there is obvious obstruction when awake, this is a consideration usually of last resort.

Medical Treatment for Snoring

A trial of nasal steroids such as mometasone can help the child while waiting for a PSG or for surgery. There is no place for long or medium- term oral steroids and there is no evidence that low dose antibiotics help. More recently the oral leukotriene receptor antagonist montelukast has been shown to have some benefit in PSG-documented OSA in small numbers of children. At this stage it is unclear how long the effects last for. Children do improve with growth and with age and the natural involution of the lymphoid tissues. However if OSA is confirmed on PSG treatment should not be delayed.

Children who have failed TA might need CPAP. Careful education of the family and fitting of the mask and strap with practice sessions at home optimise acceptance and compliance. Initially the child should be observed on CPAP in hospital first before a subsequent sleep study to titrate the correct pressures on CPAP. Some centres might start CPAP for a brief period of time prior to surgery if the PSG shows very severe OSA. This serves to decrease swelling in the upper airways and optimises chemoreceptor sensitivity.

Common Myths about Snoring and OSA in Children

1. *Snoring is normal*

   Snoring and OSA can run in families and therefore accepted as part of normal sleep. However 90% of children do not snore. Recent research suggests that snoring itself might actually cause adverse neurocognitive and behavioural effects during the day even when blood gas parameters are entirely normal.

2. *The child is too young to have adenotonsillectomy or that they will grow out of it.*

   Research suggests that the younger the child is diagnosed and treated the less the long term neurocognitive deficits. Other longer term effects of untreated OSA in early childhood include suboptimal physical growth and orthodontic issues and possibly metabolic and cardiovascular effects. Obviously the youngest and most at risk children should attend tertiary centres where there is more experience with paediatric peri-operative care.

3. *Children do not sleep in sleep labs*

   With careful pre-admission acclimatisation and in experienced units, infants and children do sleep very well. Our data show that children have an average sleep efficiency of over 83%.

4. *Children will get more infections after adenotonsillectomy*

   Parents are reassured that cervical lymph glands continue to provide a ring of protection against infection. Lymphoid cells rise to pre-operative levels within days following TA. The child will no longer suffer from adenoiditis and tonsillitis.

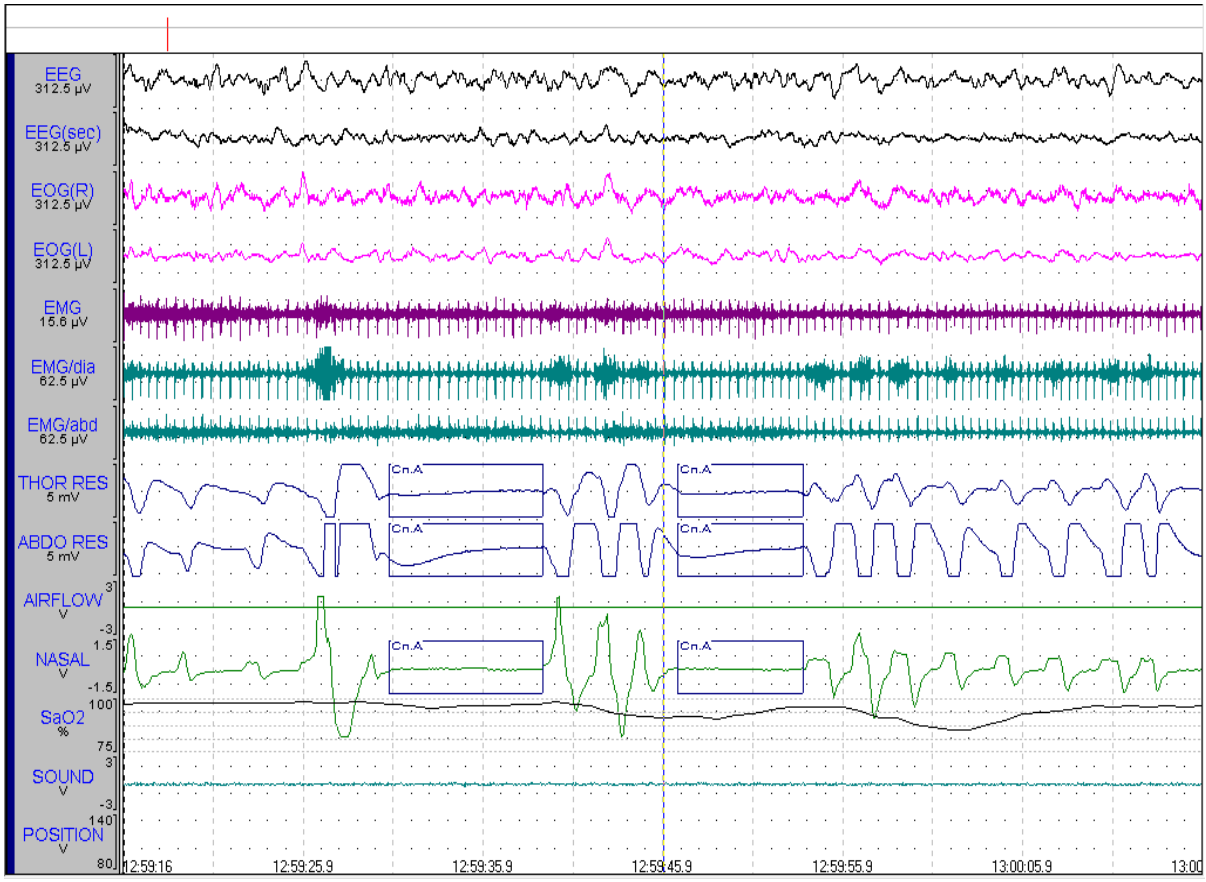

Figure 2. One-minute "epoch" or page from a sleep study. Two *central apnoeas* (CnA) where there is absent effort and nasal airflow with resultant drop in oxy-haemoglobin saturation

(SaO_2) are noted. EEG shows Stage 4 Sleep. Frontal brain waves are picked up in the electrooculogram (EOG) as well.

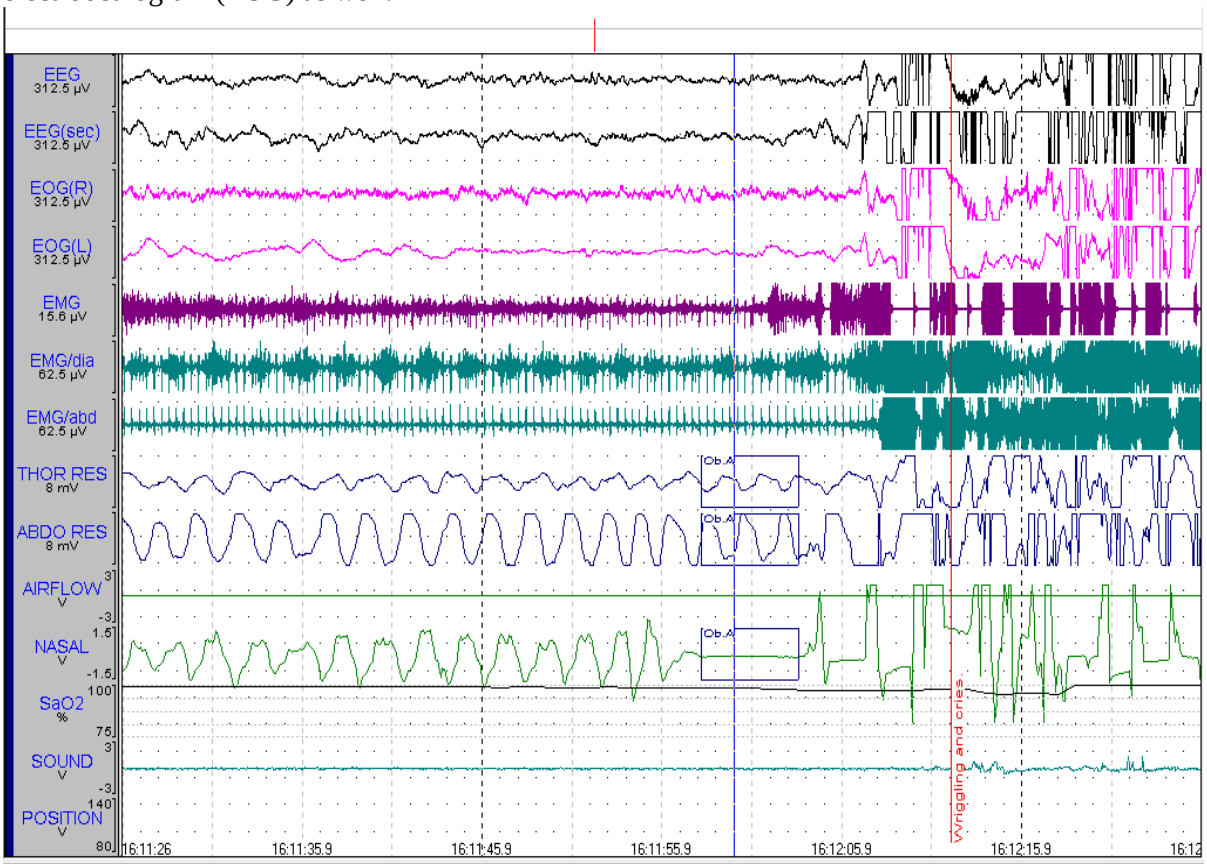

*Figure 3.*One-minute epoch showing an *obstructive apnoea* (ObA) with arousal from light sleep. Note the gross movement following the event. The nasal airflow shows evidence of flow limitation. The diaphragmatic electromyogram (EMG/dia) suggests an increase in work of breathing with a crescendo at the end of the ObA. The thoracic and abdominal bands (THOR RES/ ABDO RES) show paradoxical or opposite movements in the ObA event.

SUGGESTED FLOW CHART FOR SLEEP STUDY REFERRAL

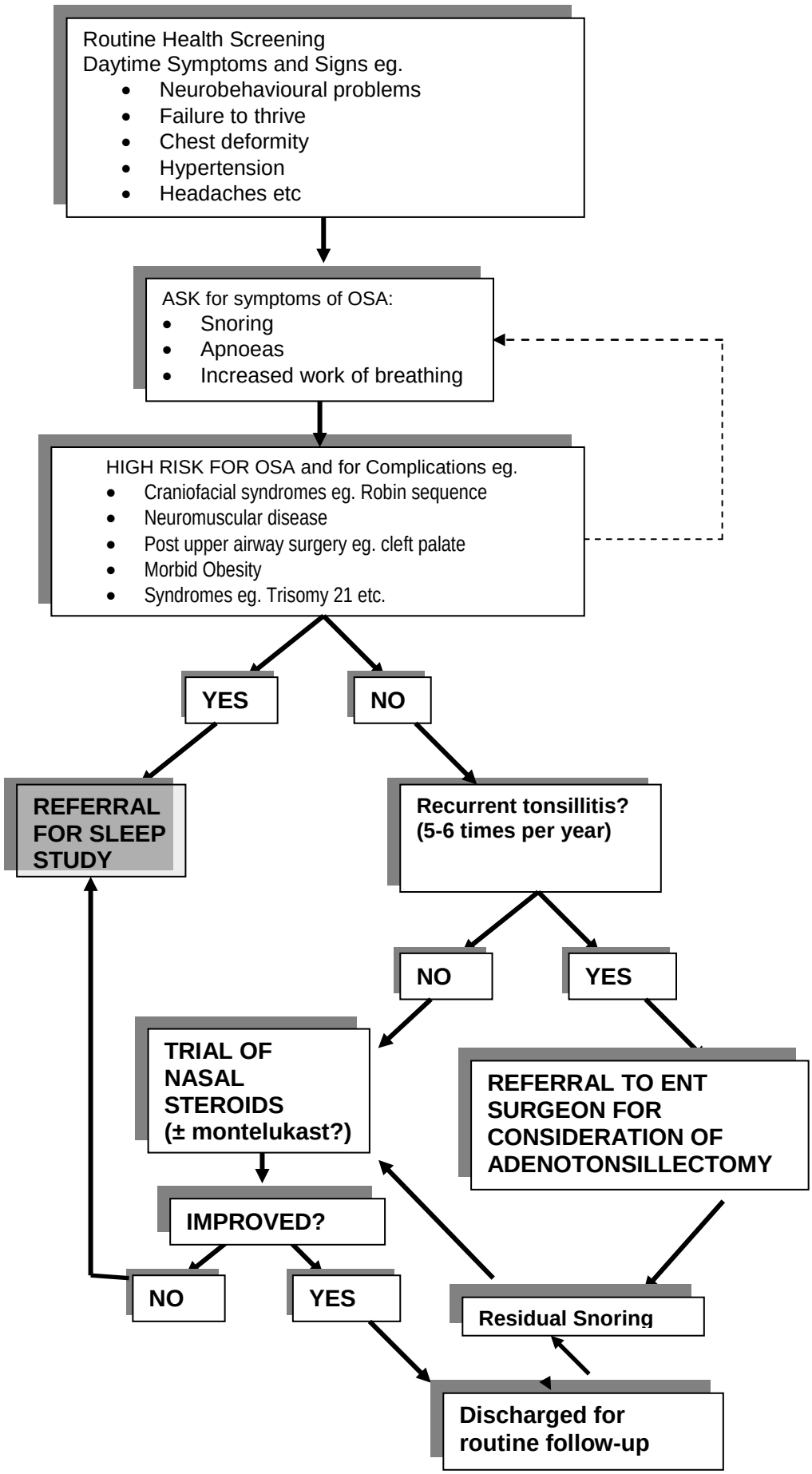

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Figure 4 Suggested Flow Chart for Sleep Study Referral

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