Snoring & Sleep Apnoea in Children

Marianne Davey MSc
SNORING & SLEEP APNOEA
AND OTHER
SLEEP RELATED BREATHING DISORDERS

Snoring (PS)

Upper Airway Resistance Syndrome (UARS)

Sleep Disordered Breathing (SDB)

Obstructive Sleep Apnoea (OSA)
Snoring:
Audible indicator that inspiratory airflow is restricted.
Common and generally benign condition.
May indicate presence of OSA or a lesser form of SDB.
Can be difficult to distinguish from OSA.

UARS:
Characterised by snoring
Partial upper airway obstruction which leads to –
repetitive episodes of increased respiratory effort that ends in arousals.
No evidence of apnoea, hypopnoea or reduction in oxygen saturation.
Symptoms do not meet clinical criteria for OSA but there is sleep fragmentation
and daytime behavioural problems which is often seen in children with OSA.

SDB:
Characterised by snoring and recurrent apnoea & hypopnoea episodes that
disrupt night time ventilation and sleep quality.

OSA:
Characterised by sleep related upper airway obstruction and reduction in
blood oxygen saturation.
DIAGNOSTIC METHODS

History
Physical examination
Audio recording
Video recording
Polysomnography (PGS)
Or a combination of the above
History
usually from parental reports

Loudness of snoring does not necessarily correlate with the severity of the problem

Frequency is a better indicator

Be mindful: parent reporting is not always accurate
Example
Children with OSA experience obstruction primarily during REM, which occurs predominantly in the early hours of the morning when parents are most likely not observing.

This might lead to underestimation of a potential problem.
Example

It is not always easy for parents to differentiate between what is just harmless noisy breathing and a more serious snoring noise.

Unlike the previous scenario parents may be prone to exaggeration of the symptoms in order to secure further treatment.
Physical examination

GP will examine the upper airway to determine adenotonsillar hypertrophy.

Not all tonsils/adenoids are diseased

Presence of large tonsils and adenoids is not necessarily an indicator of a sleep related breathing problem.

Facial morphology
(very often overlooked)
Milerad (2003)
Swedish Study

All children should be screened for snoring as part of routine healthcare and positive findings should be followed by a more detailed evaluation.
Audio-video-tape

Helpful if results are positive but poor predictive value if results are negative.

Example
Making a diagnosis (OSA) by observation does not allow for determination of severity of a problem.

Problems with audio/video
Microphone becomes displaced.
Child has a cold on chosen night giving inappropriate data.
Can we predict sleep related breathing disorders from history taking, physical examination, audio-video-recording?

<table>
<thead>
<tr>
<th></th>
<th>How long have symptoms persisted</th>
<th>Essential information but not reliable</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Changes in symptoms/behaviour</td>
<td>Parents may over- under-estimate</td>
</tr>
<tr>
<td></td>
<td></td>
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<tr>
<td>Physical examination</td>
<td>Enlarged/infected tonsils &amp; adenoids</td>
<td>Essential information not always reliable as symptoms may be transient</td>
</tr>
<tr>
<td></td>
<td>Facial morphology</td>
<td>May not even be relevant to problem</td>
</tr>
<tr>
<td></td>
<td>Underweight/thriving</td>
<td></td>
</tr>
<tr>
<td>Audio</td>
<td>Snoring/Apnoeas can be detected every night</td>
<td>But not severity One night observation not sufficient</td>
</tr>
<tr>
<td>Video</td>
<td>Can detect symptoms such as odd sleeping position</td>
<td>Essential information one night observation not sufficient</td>
</tr>
</tbody>
</table>
Polysomnography (PSG) – Gold Standard
Only method that quantifies ventilatory and
sleep abnormalities

Diagnosis based on threshold criteria of apnoea
index and degree of oxygen desaturation

Children present with different symptoms to those of adults

Children have continuous partial obstructive hypoventilation
with lower numbers of apnoeas, fewer arousals and less
sleep disruption than adults.
Other sleep related conditions have some of the symptoms of OSA but do not meet the clinical criteria. Can PSG distinguish between them?

Example:

Children with UARS suffer night time breathing related sleep disruption without OSA but suffer all the daytime symptoms associated with OSA.
According to Carroll (2003) PSG becomes dubious because of its heavy focus on breathing and minimal measures of sleep quality.

PSG cannot measure:
- EDS
- Impaired neuro-cognitive function
- Behavioural abnormalities
- Other adverse outcomes related to SDB in children

One test alone is not sufficient to give crucial information
A combination of all tests will give the best diagnosis
Prevalence figures for PS & OSA  
Review of questionnaire based studies

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>Age (yrs)</th>
<th>Study number</th>
<th>Prevalence %</th>
<th>PS OSA</th>
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<tbody>
<tr>
<td>Mitchell</td>
<td>2003</td>
<td>NZ</td>
<td>0-1</td>
<td>1800</td>
<td>50</td>
<td>PS</td>
</tr>
<tr>
<td>Carroll</td>
<td>2003</td>
<td>USA</td>
<td>not specified</td>
<td>NS</td>
<td>20</td>
<td>PS</td>
</tr>
<tr>
<td>Milerad</td>
<td>2003</td>
<td>Sweden</td>
<td>Infancy</td>
<td>NS</td>
<td>16</td>
<td>PS</td>
</tr>
<tr>
<td>Sterni</td>
<td>2003</td>
<td>USA</td>
<td>not specified</td>
<td>N/A</td>
<td>10</td>
<td>PS</td>
</tr>
<tr>
<td>Corbo</td>
<td>1989</td>
<td>Italy</td>
<td>6-13</td>
<td>1615</td>
<td>7.3</td>
<td>PS</td>
</tr>
<tr>
<td>Corbo</td>
<td>2001</td>
<td>Italy</td>
<td>10-15</td>
<td>2209</td>
<td>5.6</td>
<td>PS</td>
</tr>
<tr>
<td>Nieminen</td>
<td>1997</td>
<td>Finland</td>
<td>&gt;5</td>
<td>78</td>
<td>33</td>
<td>OSA</td>
</tr>
<tr>
<td>Milerad</td>
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<td>Infancy</td>
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<td>2</td>
<td>OSA</td>
</tr>
<tr>
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<td>USA</td>
<td>Not specified</td>
<td>NS</td>
<td>2</td>
<td>OSA</td>
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</table>

Have these figures taken into account the transient seasonal effect of URTIs?  
Would they be different if questionnaire was administered in June as opposed to December?
Questionnaire and PSG based study

Parents reports snoring on questionnaire
PSG results show no objectively detected snoring

Parents tend to combine snoring with other noises
insignificant noises such as gasps/snorts

Degree of overestimation by parents
Review of questionnaire based studies

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If 50% of newborns to 1 years snore and by age 15 only 5.6% snore – can we assume that snoring decreases with age?
Symptoms/causes/conditions that might lead us to suspect PS, UARS, SDB or OSA

<table>
<thead>
<tr>
<th>Snoring</th>
<th>Facial morphology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noisy breathing</td>
<td>Daytime fatigue</td>
</tr>
<tr>
<td>Restless sleep</td>
<td>EDS</td>
</tr>
<tr>
<td></td>
<td>(not normal in children)</td>
</tr>
<tr>
<td>Mouth breathing</td>
<td>Abnormal/difficult behaviour</td>
</tr>
<tr>
<td>Enlarged/infected tonsils &amp; adenoids</td>
<td>Impaired school performance</td>
</tr>
<tr>
<td>Frequent URTIs</td>
<td>Attentional problems</td>
</tr>
<tr>
<td>Ear infections</td>
<td>Developmental delay</td>
</tr>
<tr>
<td>Hearing &amp; speech problems</td>
<td>Impaired growth</td>
</tr>
<tr>
<td>Morning headaches</td>
<td></td>
</tr>
</tbody>
</table>
### Sleep Disordered Breathing
Clinical Features – Comparison between children and adults

<table>
<thead>
<tr>
<th>Population Characteristics</th>
<th>Children</th>
<th>Adults</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estimated Prevalence</td>
<td>2% OSA 5% PS</td>
<td>2-4% OSA 41% PS</td>
</tr>
<tr>
<td>Common age at presentation</td>
<td>2-6 years</td>
<td>30-60 years</td>
</tr>
<tr>
<td>Gender</td>
<td>M = F</td>
<td>M 2 x &gt;F</td>
</tr>
<tr>
<td>Associated obesity</td>
<td>Minority of patients</td>
<td>Majority of patients</td>
</tr>
<tr>
<td>Underweight/failure to thrive</td>
<td>Frequent finding</td>
<td>Not seen</td>
</tr>
<tr>
<td>Enlarged tonsils &amp; adenoids</td>
<td>Frequent finding</td>
<td>Not seen</td>
</tr>
<tr>
<td>Major cause</td>
<td>Adenotonsil</td>
<td>Obesity</td>
</tr>
<tr>
<td>Associated conditions</td>
<td>Craniofacial abnormalities</td>
<td>Menopause</td>
</tr>
<tr>
<td><strong>Clinical Features</strong></td>
<td></td>
<td></td>
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<tr>
<td>-----------------------</td>
<td>----------------</td>
<td>----------------</td>
</tr>
<tr>
<td>Snoring</td>
<td>Continuous</td>
<td>With pauses</td>
</tr>
<tr>
<td>Main obstructive pattern</td>
<td>Hypoventilation</td>
<td>Apnoea</td>
</tr>
<tr>
<td>Arousals as apnoea stops</td>
<td>Not usually</td>
<td>Nearly always</td>
</tr>
<tr>
<td>Sleep pattern disruption</td>
<td>Normal</td>
<td>Nearly always: decreased delta &amp; REM</td>
</tr>
<tr>
<td>EDS</td>
<td>Unusual</td>
<td>Very common, often severe</td>
</tr>
<tr>
<td>Daytime mouth breathing</td>
<td>Common</td>
<td>No</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>PSG findings</strong></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Gas exchange abnormalities</td>
<td>Frequent</td>
<td>Usually</td>
</tr>
<tr>
<td>Length of OSA</td>
<td>Any duration abnormal</td>
<td>&gt;10 sec abnormal</td>
</tr>
<tr>
<td>Abnormal AI</td>
<td>AI&gt;1</td>
<td>AI&gt;5</td>
</tr>
<tr>
<td>Sleep architecture</td>
<td>Normal</td>
<td>Usually altered</td>
</tr>
<tr>
<td>Movement/arousal</td>
<td>Abnormal sleeping position</td>
<td>Common</td>
</tr>
<tr>
<td>Complications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------------------------</td>
<td>-------------------------</td>
<td></td>
</tr>
<tr>
<td>Neurobehavioural</td>
<td>Hyperactivity</td>
<td>Severe EDS</td>
</tr>
<tr>
<td></td>
<td>Development delay</td>
<td>Cognitive impairment</td>
</tr>
<tr>
<td></td>
<td>Behavioural problems</td>
<td></td>
</tr>
<tr>
<td></td>
<td>often mistaken for ADHD</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgical</td>
<td>Adenotonsillectomy</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-surgical</td>
<td>CPAP, jaw repositioning</td>
</tr>
<tr>
<td></td>
<td>devices, orthodontic</td>
</tr>
<tr>
<td></td>
<td>treatment</td>
</tr>
</tbody>
</table>
DISCUSSION OF FEATURES COMMON TO CHILDREN

- Adenotonsil hypertrophy
- Mouth Breathing
- Smoking
- Craniofacial disorders
- Behavioural problems
- Quality of life
TONSILS & ADENOIDS

Snoring always indicates some degree of partial obstruction.

It is presumed that hypertrophy of the tonsils and adenoids is a major cause of sleep related breathing problems in children.

Consensus among all authors that adenoidal hypertrophy peaks around 5-6 years.
Densert et al (2001)  
Rosen (2000)  
Shintani et al (1997)  

Have all written extensively on the presumed role of adenotonsillar hypertrophy in sleep related breathing disorders
Found dentofacial morphology to be a crucial factor in the development of OSA

Shintani et al (1997)
Suggested enlarged tonsils and adenoids give rise to condition ‘adenoid facies’
Characterised by:
Long narrow face
Short upper lip
Prominent upper incisor teeth
High arched palate
Lips apart posture
Retrognathic mandible
Shintani et al (1997) also suggesting this facial structural narrowing may be a genetic factor.

Children with small airway due to genetic factors are easily influenced by adenotonsillar hypertrophy and vice versa…

Professor Douglas believes in some cases adult OSA results from facial structural narrowing in the airway – a condition since childhood.
Rosen (2000) – SDB is commonly associated with adenotonsillar hypertrophy and most children will have symptomatic resolution following adenotonsillectomy.

Similar views but Rosen (2002) does acknowledge craniofacial abnormalities can be a major cause.
Controversy: Densert et al (2001) suggest: not to remove diseased tissue but to reduce it in size.

Rationale: evaluate simplified and less traumatic surgical procedure

2 groups of children: 1 complete tonsillectomy the other tonsillar reduction

2 years after treatment both groups satisfied with outcome and no statistically significant differences in clinical symptoms between groups
Uncommitted:

Most common in pre-school children
Age when tonsils/adenoids are largest in relation to the underlying airway size.

Combination of adenotonsillar hypertrophy and neuromuscular tone of the upper airway during sleep may be responsible for SDB
Rebuttle: Nieminen et al (1997) adenoidal tissue does not play a crucial role in the development of snoring and sleep apnoea

Studied 78 children
34 had previously undergone adenotonsillectomy

80% of those who had undergone surgery had a higher AHI than those who had not.

Mouth breathing typical in both groups
Odd sleeping position common to both groups

Conclusion: adenoidectomy may be a risk factor for OSA rather than conclusive treatment
(commonly known for adults who undergo surgery to run similar risks)
Outcomes of surgery:

Rosen (2002) – OSA is the indication for surgery in approximately 80% of their paediatric cases

But……less than 10% undergo any diagnostic procedure prior to surgery

Nieminien et al (1997) of those children who had previous surgery, 80% of them had an AHI>5
Significantly higher than non-surgery children

Conclusion: it would be wise to consider all other options before undertaking surgery.

Similarly, adults take note – surgery is appropriate for a very small number of snorers
Mouth breathing
Castronovo et al (2003) – persistent mouth breathing may lead to impaired development of facial structures, especially mandible.
Reason: 60% of adult face is formed by 4 yrs.


Rebuttle:
Nieminne et al (1997) – reported on mouth breathing equally typical in children already undergone surgery as those who had not.
Smoking – provokes mucosal oedema and inflammation resulting in narrowing of the pharynx.

Corbo et al (1989) – respiratory symptoms closely related to passive parental smoking
Dose effect relationship

Boys more likely than girls to snore
Increased levels of testosterone in boys after puberty have an apnoea-promoting effect which may be responsible for increased prevalence in boys.

Children whose parents smoked were twice as likely to snore than those whose parents did not smoke.
Mitchell & Thompson (2003) study of snoring infants 0-12 months

Exposure to environmental tobacco smoke found same symptoms as those described by Corbo et al

Regards to males being more likely to snore than females – Testosterone in 15 years olds - but in infants?

In adults females are protected somewhat due to a more favourable airway anatomy

Gonzalez et al (1996) – from respiratory point of view patients with craniofacial abnormalities have any number of features that might be expected to affect upper airway function.

Retrognathia, deviated septum, depression of the nasal bridge, abnormalities of the maxillary dental arch or short hard palate.

Mandible retrognathia is a common cause of sleep disordered breathing due to the retracted chin.

Root of tongue – large or incorrectly positioned tongue will impact on the size of the airway space.

Common reason for snoring in adults known as ‘tongue base’ snoring.
Shintani et al (1997) – ‘adenoidal face’
Maxillary growth impaired by obstruction of the tonsils and adenoids.

Rebuttle:

Craniofacial factors are associated with SDB, but airway and palate size may not be significant factors.

Conclusion:
All other authors have suggested airway and palate size is significant in the development of SDB especially when associated with enlarged tonsils and adenoids.
Treatment options:

Gonzalez et al (1996) – CPAP compliance is often a problem with adults, but with children initiation is a major difficulty.

Let us not forget to investigate the simple causes first – allergies

Seasonal hay fever
House dust mite
Feathers
Pet hair
Soaps
Detergents
Behavioural problems and quality of life


42% reported to be hyperactive but more often ‘asocial’ behaviour (fighting with peers, aggressiveness, crying easily)

76% switching quickly from hyperactivity to excessive somnolence and withdrawn. EDS however is not a common symptom in children with SDB.

20% personality changes
16% delayed language acquisition and decreased school performance
Rosen et al (2002) – used HRQL questionnaire
298 participants aged 8-14
Mild SDB may be associated with daytime
neurocognitive and behavioural dysfunction,
with problems increasing with increasing severity of SDB.

Chervin et al (2000) used Pediatric Sleep Questionnaire
(PSQ)
EDS less prominent than other daytime behaviours.
Confirmed previous reports of daytime symptoms not
seen in adults.
Mainly: mouth breathing, delayed growth, inattention,
hyperactivity.
Behavioural problems cont/…..


Rebuttle: Owens et al (2000) – most data anecdotal and tends to be focused on severe end of SDB scale.

Small study of 18 children: Failed to demonstrate more behaviour problems with increased severity of symptoms, and suggested relationship in opposite direction – children with mild SA reported more problems than those with severe OSA.
Behaviour problems cont/…

Franco et al (2000)
Quality of life poorly understood
Anecdotal reports
Objective measures difficult

Small study using HRQL for 61 children aged 1-12 years
Poor correlation with objective external measures in emotional distress and daytime function domains.

Conclusion: despite some negative reports it is generally accepted that unusual behaviours need further investigation.
Diagnostic methods

Prevalence

Compared clinical features between adults & children

Discussed features common to children and not adults

Review of current research
Conclusions:
Snoring in children usually benign but may be indicator of other SDB

Look for unusual or changes in behaviour.

Making a diagnosis: don’t rely on one method alone

Adenotonsillar hypertrophy is not always an indicator of snoring and/or OSA.

Craniofacial abnormalities: take a look

If in doubt – refer to paediatric sleep specialist
(few and far between – London, Edinburgh, Paris, Geneva …….)