

British Snoring
&
Sleep Apnoea
Association

Snoring & Sleep Apnoea in Children

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**SNORING & SLEEP APNOEA
AND OTHER
SLEEP RELATED BREATHING DISORDERS**

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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

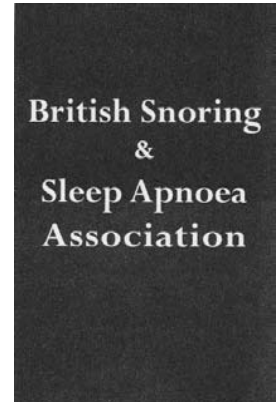
Physical examination

Audio recording

Video recording

Polysomnography (PGS)

Or a combination of the above



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History
usually from parental reports

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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

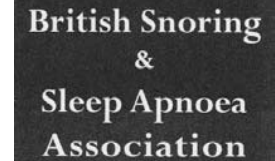
Microphone becomes displaced.

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?

History	How long have symptoms persisted Changes in symptoms/behaviour	Essential information but not reliable Parents may Over- under-estimate
Physical examination	Enlarged/infected tonsils & adenoids Facial morphology Underweight/thriving	Essential information Not always reliable as symptoms may be transient May not even be relevant to problem
Audio	Snoring/Apnoeas can be detected every night	But not severity One night observation not sufficient
Video	Can detect symptoms such as odd sleeping position	Essential information one night observation not sufficient

Polysomnography (PSG) – Gold Standard
Only method that quantifies ventilatory and
sleep abnormalities

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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?

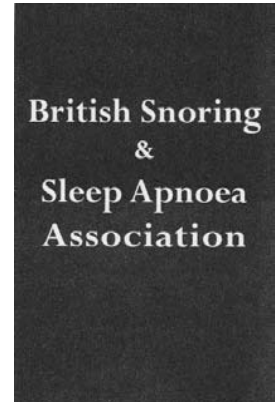
Example:

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.

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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

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

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?

Castranovo et al (2003)
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

Snoring	Facial morphology
Noisy breathing	Daytime fatigue
Restless sleep	EDS (not normal in children)
Mouth breathing	Abnormal/difficult behaviour
Enlarged/infected tonsils & adenoids	Impaired school performance
Frequent URTIs	Attentional problems
Ear infections	Developmental delay
Hearing & speech problems	Impaired growth
Morning headaches	

Sleep Disordered Breathing		
Clinical Features – Comparison between children and adults		
	Children	Adults
Population Characteristics		
Estimated Prevalence	2% OSA 5% PS	2-4% OSA 41% PS
Common age at presentation	2-6 years	30-60 years
Gender	M = F	M 2 x >F
Associated obesity	Minority of patients	Majority of patients
Underweight/failure to thrive	Frequent finding	Not seen
Enlarged tonsils & adenoids	Frequent finding	Not seen
Major cause	Adenotonsil	Obesity
Associated conditions	Craniofacial abnormalities	Menopause

Clinical Features		
Snoring	Continuous	With pauses
Main obstructive pattern	Hypoventilation	Apnoea
Arousals as apnoea stops	Not usually	Nearly always
Sleep pattern disruption	Normal	Nearly always: decreased delta & REM
EDS	Unusual	Very common, often severe
Daytime mouth breathing	Common	No
PSG findings		
Gas exchange abnormalities	Frequent	Usually
Length of OSA	Any duration abnormal	>10 sec abnormal
Abnormal AI	AI>1	AI>5
Sleep architecture	Normal	Usually altered
Movement/arousal	Abnormal sleeping position	Common

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Complications		
Neurobehavioural	Hyperactivity	Severe EDS
	Development delay	Cognitive impairment
	Behavioural problems often mistaken for ADHD	
Treatment		
Surgical	Adenotonsillectomy	UPPP, somnoplasty, septoplasty etc.
Non-surgical	CPAP, jaw repositioning devices, orthodontic treatment	Range of anti-snoring products and devices

DISCUSSION OF FEATURES COMMON TO CHILDREN

Adenotonsil hypertrophy

Mouth Breathing

Smoking

Craniofacial disorders

Behavioural problems

Quality of life

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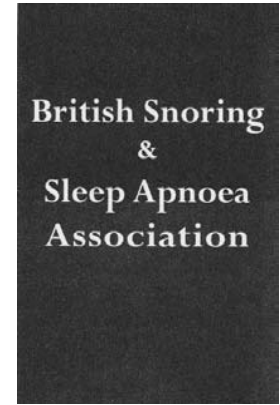
TONSILS & ADENOIDS

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)
Kawashima et al (2000)
Rosen (2000)
Rosen et al (2002)
Shintani et al (1997)
Sterni et al (2002)

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Have all written extensively on the presumed
role of adenotonsillar hypertrophy in
sleep related breathing disorders

Kawashina et al (2000)

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

Sterni et al (2003) & Rosen et al (2002)
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

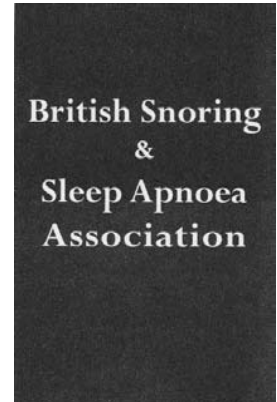
2 groups of children: 1 complete tonsillectomy the other tonsillar reduction

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

Uncommitted:
American Academy of Pediatrics (2002)

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

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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

Nieminen 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.

Chervin et al (2003) – symptom of childhood SDB but not adults.

Rebuttle:

Nieminen 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

Corbo et al (2001) – made further observations regarding demographic factors.

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

Craniofacial disorders:

Gonsalez et al (1996), Guilliminault et al (1981), Kawashima et al (2000), Nelson et al (2003) & Shintani et al (1997)

Gonsalez 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.

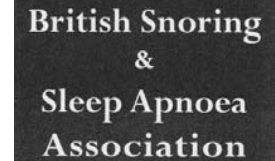
Kawashima et al (2000) noted differences in form and position of the mandible in children with OSA to those of controls.

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.



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Rebuttle:

Nelson et al (2003) – examined differences in craniofacial factors
between snorers and non snorers from childhood to adult.

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:

Guilleminault et al (1981) – adenotonsillectomy or maxillo-facial surgery with advancement of the mandible and orthodontic treatment.

Gonsalez et al (1996) – CPAP

compliance is often a problem with adults, but with children initiation is a major difficulty.

Sterni et al (2003) - surgery remains mainstay of treatment but craniofacial surgery common.

Jaw positioning appliances.

Radical surgical approaches currently not available in UK.

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

Seasonal hay fever

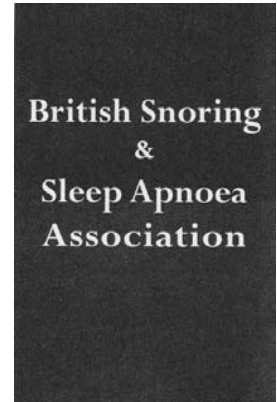
House dust mite

Feathers

Pet hair

Soaps

Detergents

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Behavioural problems and quality of life

Guilleminault et al (1981) – 50 children with OSA

- 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.

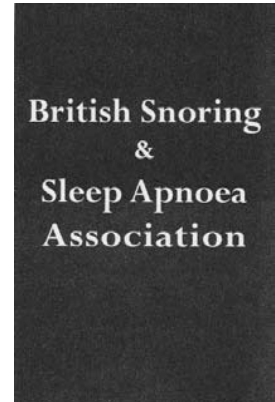
Behavioural problems cont/.....

Sterni et al (2003) – acknowledged importance of neurocognitive and behaviour consequences of SDB. Suggests problems resolve after treatment especially hyperactivity and aggressiveness.

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.

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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.

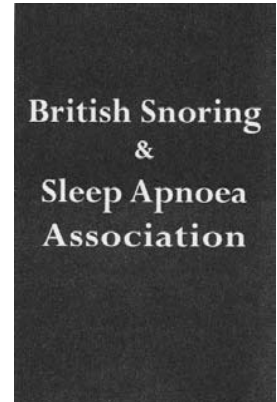
Diagnostic methods

Prevalence

Compared clinical features between adults & children

Discussed features common to children and not adults

Review of current research

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Conclusions:

Snoring in children usually benign but may be indicator of other SDB

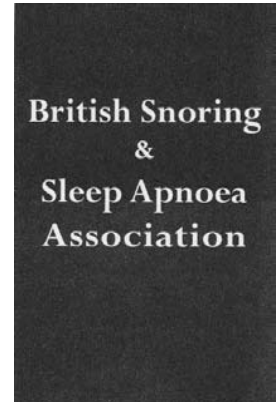
Look for unusual or changes in behaviour.

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)



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