

- Infection around the tube, and otorrhoea secondary to middle ear infection (could be viewed as a side-effect of grommets).

The most common negative consequence associated with the insertion of grommets is permanent scarring of the tympanic membrane (tympanosclerosis), which occurs in 30 - 50% of patients. However, it almost never causes significant hearing loss and is therefore of no clinical consequence. Permanent perforations occur in as few as 3% of patients and are often seen in conditions requiring long-term ventilation tubes.

The side-effects associated with grommets are often more troublesome. The most frequently encountered side-effect is secondary infection — in up to 30% of patients. This is associated with otorrhoea, which, apart from being socially unacceptable, may also cause blocking of the tube. This, in turn, could promote earlier extrusion of the tube, and/or be associated with a permanent small perforation. Aural toilet in combination with antibiotic steroid drops is helpful for this condition. Small perforations after insertion of grommets need to be dealt with on an individual basis, as they may either be left or repaired later by tympanoplasty.

Summary

- Most cases of OME spontaneously resolve over a period of time (from 3 to 6 months).
- When indicated, the insertion of grommets remains a very useful intervention for the following reasons:
 - it is a minor procedure
 - the associated side-effects/complications are minimal
 - it is of great value in normalising hearing thresholds
 - it therefore assists with speech development in these children
 - it may allow a child to 'out-grow' Eustachian tube dysfunction, and thus avoid anatomical changes to the tympanic membrane such as retraction/atrophy.

Conclusion

In private practice OME remains one of the most commonly seen conditions and grommets have become one of our most effective ways to manage this condition.⁴

References available on request.

SNORING — MORE THAN A SOCIAL NUISANCE!

ISMAIL A PATEL, MB BCH, FCORL (SA) **Consultant Specialist, ENT Department, University of the Witwatersrand and Chris Hani Baragwanath Hospital, Johannesburg**

SYLVESTER D MASEGE, MB BCH, FCORL (SA), DCH **ENT Specialist: Anglogold Health Service, Carletonville**

Snoring is an undesirable sound originating from the soft tissues of the upper airway during sleep. It has more often than not been part of a social dogma that has plagued society through the sands of time. However, it is a manifestation of a large group of sleep disorders loosely termed sleep-disordered breathing (SDB). SDB is a broad term encompassing obstructive sleep apnoea (OSA), obstructive sleep hypopnoea (OSH), excessive daytime somnolence and upper airway resistance syndrome (UARS).

There are numerous sleep disorders that are organised in the International Classification of Sleep Disorders by the American Sleep Disorders Association. The predominant problems that may warrant a patient seeking assessment and possible surgical intervention is snoring (adults) and obstructive sleep apnoea (adults and children). A brief approach to understanding these conditions is discussed with special attention being paid to clearly defining certain sleep disorders.

Definitions

- **Snoring:** noisy breathing arising from upper respiratory tract during sleep.
- **Hypopnoea:** nasal airflow falling by 50 - 75% for longer than 10 seconds.
- **Apnoea:** nasal airflow falling by > 75% for longer than 10 seconds.
- **Obstructive sleep apnoea (OSA):** cessation of airflow at the nose or mouth for at least 10 seconds.
- **Apnoea/hypopnoea index (AI):** number of apnoea/hypopnoea episodes in 1 hour.

OSA is a sleep disorder in which there are repeated reductions or cessations in airflow at the nostrils or mouth and can be either central, obstructive or mixed. In obstructive apnoea there is absence of airflow with continued inspiratory effort and in central apnoea there is no inspiratory effort — the latter is not amenable to surgical correction. The mixed type presents with symptoms of both obstructive and central apnoea.

The apnoea index (AI) is defined as the number of episodes of apnoea per hour. Reports usually define OSA as an apnoea index of 5 or more and a respiratory disturbance index (RDI) of at least 10 on polysomnograph readings. A typical apnoeic episode in a patient with OSA usually lasts 20 - 30 seconds and seldom exceeds 100 seconds. It generally has an AI greater than 20. The severity is markedly variable, and it is often associated with other physiological sequelae (e.g. systemic hypertension, cor pulmonale, cardiac arrhythmias, etc.).

Grading of sleep apnoea syndrome (SAS) as per American Sleep Association:

- mild: 5 - 20 apnoeic episodes per hour
- moderate: 21 - 40 per hour
- severe: > 40 per hour.

It is therefore of paramount importance to realise that everyone with SDB snores, but everyone who snores

does not necessarily have SDB. Snoring in the absence of SDB is termed primary or simple snoring. There is some evidence, however, that snoring may be one end of a clinical continuum that extends to severe OSA on the other end. Although our opening statement suggests that snoring is just a social nuisance, there may be some health problems associated even with primary snoring.

Upper airway resistance syndrome (UARS) is characterised by snoring with increased resistance in the upper airway, resulting in multiple transient arousals during sleep. This results in the patient being unable to achieve a restful sleep pattern, to the point of causing daytime somnolence. There are no distinct diagnostic criteria for this entity. No major change is seen in arterial blood saturation and the RDI usually remains low (< 5). Patients with UARS can be treated with nasal continuous positive airway pressure (n-CPAP).

Prevalence

- An Italian series showed that 24% of males and 14 % of females were habitual snorers.
- Ten per cent of males under 30 years snored compared with 60% of males over 60 years.
- If a person is over 15% overweight, then the likelihood of snoring is over 50%.

Pathophysiology

The pathophysiology of snoring is set out in Fig. 1.

Approach to diagnosis

History

The mainstay of management is a detailed history and a thorough physical examination. Usually in adults, it is the exasperated spouse who brings in their partner unwillingly for an evaluation, or a disconcerted parent who has witnessed an apnoeic episode in their child.

The following signs need to be assessed when taking a history:

- loud snoring

- witnessed breathing cessation, gasping, choking
- frequent arousals from sleep
- morning headaches
- excessive daytime somnolence
- poor concentration/ADHD
- night-time alcohol/sedative use.

The Epworth sleepiness scale

The Epworth sleepiness scale assesses the likelihood of a patient dozing off in certain situations (0 — would never doze off; 3 — high chance of dozing off). The score ranges from 0 - 24, with a score above 16 indicating major sleepiness. The predictive parameters of OSA are set out in Table I.

Table I. **Predictive parameters of OSA on examination**

- Age > 30 years
- Male
- High body mass index (> 27)
- Neck circumference > 42 cm
- Epworth sleepiness study > 11
- Witnessed apnoeic episodes

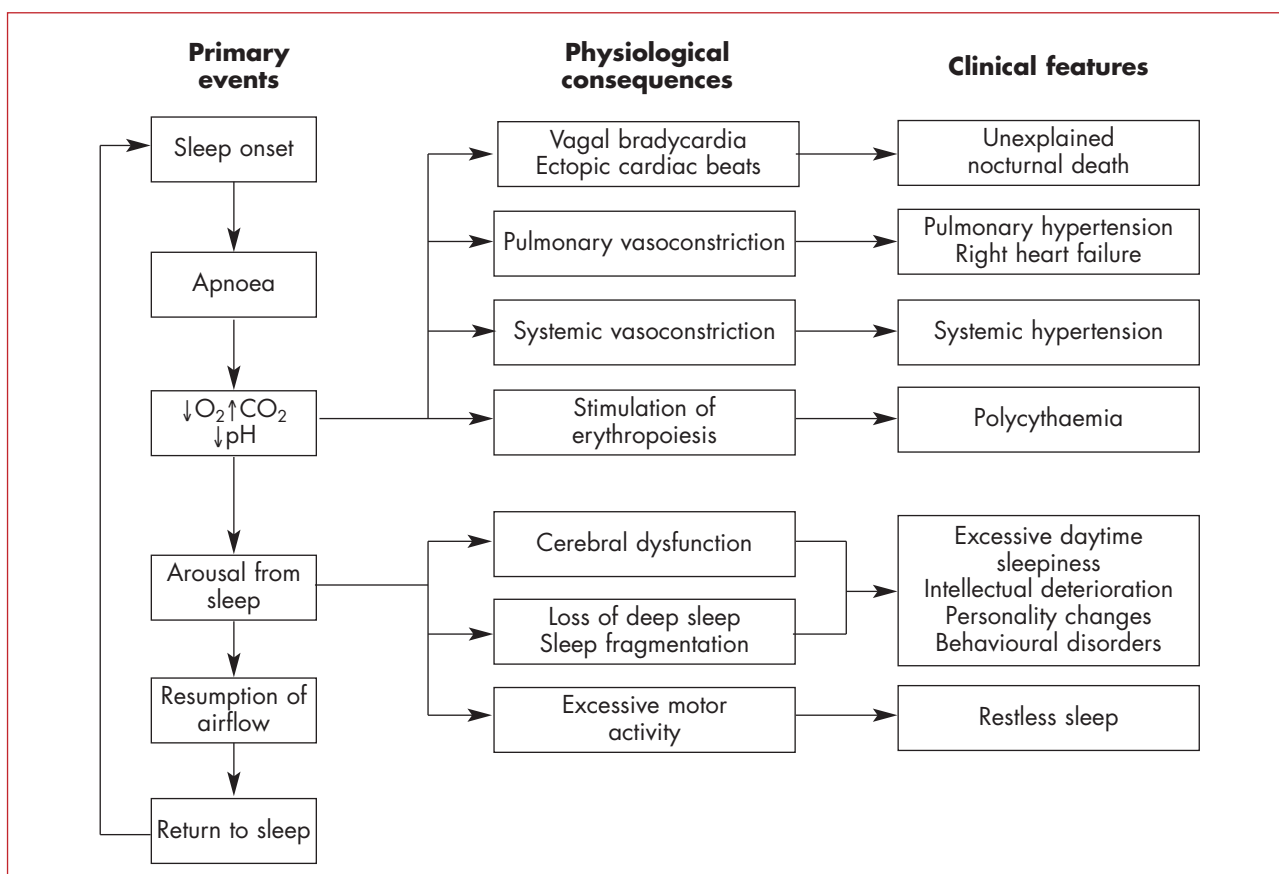


Fig. 1. The pathophysiology of snoring.

Physical findings

The examination of a patient with SDB is concentrated on the upper airway. However it is important to assess patients in a systematic way paying particular attention to physical characteristics (most are obese and/or may have short necks). A comprehensive cardiopulmonary evaluation is essential. The upper airway begins at the entrance of the nose and continues to the hypopharynx. Any part of this tract can cause obstruction, and sometimes obstruction is found in several areas in varying degrees.

Areas of special concern

Nose: nasal valve, septum and choana.

Nasopharynx: Particularly important in children because adenoids, the most common cause of OSA in children, are commonly hypertrophied, producing obstruction.

Oropharynx: The soft palate, tonsils, palatoglossal and palatopharyngeal arches, and the tongue are structures of concern. The cross-sectional diameter of the pharynx can be reduced in many patients, playing an important role in the pathogenesis of OSA.

Hypopharynx: The base of the tongue is the most influential structure in this area.

Diagnostic studies

These studies are directed by clinical findings.

- The polysomnogram is the gold standard for diagnosis. It is performed overnight in a sleep laboratory and the patient's sleep is monitored.
- The multiple sleep latency test measures the length of time required for a patient to fall asleep — normally it is 10 - 15 minutes.
- Other tests include cephalometric radiographs, which are commonly used. They are easy to perform and cheap. CT scan or MRI are used as clinically indicated. Fluoroscopy is a dynamic study that can be of tremendous value, if available.

Management

The management is tailored to the individual clinical picture. Usually conservative techniques are tried, with CPAP being a favoured modality of treatment. Surgery is usually indicated if CPAP is not suitable or if the patient does not tolerate CPAP.

Medical care

- Weight loss in obese patient
- Eliminate alcohol and sedatives
- Sleeping on the side/on the abdomen/propped up 60°
- Drugs that decrease REM sleep, e.g. acetazolamide, protriptiline
- High concentration oxygen (controversial)
- Nasal CPAP
- Obturator devices.

Surgical treatment

Phase I:

- Nasal reconstruction — e.g. septoplasty, functional endoscopic sinus surgery (FESS)
- Adenotonsillectomy
- Laser-assisted uvulopalatopharyngoplasty (LAUP)
- Genioglossal advancement.

Phase II:

- Midline glossectomy
- Bimaxillary advancement
- Maxillo-mandibular osteotomy and advancement
- Tracheotomy.

References available on request.

SINGLE SUTURE

TRANSFUSIONS AND vCJD

In the first report of possible transmission of variant Creutzfeldt-Jakob disease (vCJD) through blood, the UK Health Minister, John Reid, told parliament in December last year that a patient in the UK had died from vCJD after a blood transfusion. The disease was confirmed at postmortem examination. The patient had received a blood transfusion in 1996 from a donor, who was, at the time, free of signs of vCJD, but who developed the condition in 1999 and later died. However, it is still possible that the development of the disease was coincidental, with both patients eating contaminated meat. But this case may well alter the UK's policy of allowing people who have received a blood transfusion from giving blood. Fifteen patients in the UK are known to have received blood donated by people who later developed vCJD. They are being contacted and offered counselling.

Pincock S. *Lancet* 2004; **363**: 43.