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Sleep-disordered breathing

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Abstract. *Sleep-disordered breathing.* Sleep-disordered breathing (SDB) constitutes a spectrum of diseases, with primary snoring as the mildest form and obstructive sleep apnoea-hypopnoea syndrome (OSAHS) as the most severe form. These disorders are primarily caused by a partial or complete collapse of the upper airway during sleep. Risk factors, clinical features and physical findings are discussed in this review paper together with the diagnostic criteria. Polysomnographic studies remain the gold standard in the diagnosis and preoperative assessment of SDB. Surgical treatment for snoring most commonly includes some form of velopharyngeal surgery. The application of nasal continuous positive airway pressure (nCPAP) is the first-line treatment for moderately and severe OSAHS. Upper airway surgery is indicated for mild OSAHS and can be considered in moderately and severe OSAHS patients who are unable to comply with general measures/and or with medical treatment. Oral appliances are indicated for patients with mild SDB or nCPAP intolerance. The management of SDB requires a multidisciplinary approach. A thorough diagnostic work-up and proper patient selection are essential to guarantee a successful treatment outcome.

Introduction

The term sleep-disordered breathing (SDB) refers to sleep-induced conditions that alter the normal ventilatory pattern during sleep. Other conditions such as asthma or restrictive pulmonary disease may worsen during sleep or cause sleep disruption but their presence is not limited to sleep and therefore they are not classified as SDB.

The physiological spectrum ranges from partial upper airway (UA) collapse with an increase in UA resistance, manifested as snoring, flow limitation and hypopnoea, to complete UA collapse and apnoea.

Definitions

Snoring

Snoring is an inspiratory noise produced by the vibration of pharyngeal soft tissues.

Primary snoring is defined as snoring without sleep disruption and the absence of insomnia or excessive daytime sleepiness.

Habitual snoring is defined as snoring that occurs for >5 nights/week.

Upper airway resistance syndrome

This disorder was originally defined as the combination of a clinical complaint (excessive daytime sleepiness) with both flow limitation, increased respiratory effort, and arousal just after the peak in negative inspiratory oesophageal pressure. Current thinking is that this is not a distinct syndrome but rather a subtle form of the sleep apnoea syndrome.[1]

The American Academy of Sleep Medicine[1] formulated the definitions for **sleep apnoea (hypopnoea) syndrome (SAHS)** (Table 1).

Pathogenesis

Sleep predisposes to disordered breathing, even in otherwise healthy individuals, because the ventilatory control system is compromised during sleep. Several factors affecting both the upper airway and ventilatory drive occur during sleep in normal subjects and may contribute to SDB.

The state of wakefulness is characterised by the tonic excitation of the respiratory centres which has been named the ‘wakefulness stimulus’. This waking neural drive results in an overall drive to the respiratory muscles and is crucial to the generation of rhythmic breathing. At sleep onset, the ‘wakefulness stimulus’ disappears, which implies that ventilation becomes dependent on chemoreceptor control. This results in a reduction of minute ventilation and an increase in upper airway resistance.

Table 1

Definitions for sleep apnoea (hypopnoea) syndrome (SAHS) (1)
To meet the diagnostic criteria for **obstructive sleep apnoea (hypopnoea) syndrome** (OSAHS), the individual must fulfil criterion A or B, plus criterion C.
A. Excessive daytime sleepiness that is not better explained by other factors. B. Two or more of the following that are not better explained by other factors: – choking or gasping during sleep; – recurrent awakenings from sleep; – unrefreshing sleep; – daytime fatigue; – impaired concentration. C. Overnight polysomnography demonstrates five or more obstructive breathing events per hour during sleep. These events may include any combination of obstructive apnoeas, hypopnoeas or respiratory-effort-related arousals.
An obstructive apnoea/hypopnoea event is defined as a transient reduction or complete cessation of breathing. For clinical purposes, it is not deemed necessary to distinguish between apnoeas and hypopnoeas since both have the same pathophysiology. These events must fulfil criterion 1 or 2, plus criterion 3 as described below: 1. A >50% decrease from baseline in amplitude of a valid measure of breathing during sleep. 2. A <50% amplitude reduction of a valid measure of breathing during sleep associated with either an oxygen desaturation of >3% or an arousal. 3. The event lasts 10 seconds or longer.
RERA event (respiratory effort-related arousal) is characterised by increasingly negative oesophageal pressure, terminated by a sudden change in pressure to a less negative level and an arousal. This event lasts 10 seconds or longer.
An **arousal** is defined as any shift in EEG frequency to alpha or theta for at least 3 seconds but not longer than 15 seconds.
The **RDI** (respiratory disturbance index) is the total number of respiratory events (apnoeas and hypopnoeas)/hour of sleep
Severity criteria
Mild: 5 to 15 events/hour of sleep Moderate: 15 to 30 events/hour of sleep Severe: more than 30 events/hour of sleep
Idiopathic central sleep apnoea syndrome
This condition is characterised by recurrent apnoeic episodes during sleep in the absence of respiratory effort and upper airway obstruction during the episode. These events usually result in desaturation, recurrent arousal and daytime symptoms.

The decrease in ventilatory drive and the concomitant increase in upper airway resistance will result in a decrease in tidal volume, which in turn may cause hypopnoea or apnoea with partial or complete upper airway obstruction.

Several studies have demonstrated that the UA of patients with SDB is more susceptible to narrowing and collapse when upper airway dilator activity decreases during sleep.

Two hypotheses have been formulated to account for this increased collapsibility.[2]

The neural hypothesis states that upper airway dilator activity is more reduced during sleep in OSAHS patients as compared to normals. According to the anatomical hypothesis, the sleep-related reduction in OSAHS patients is normal but it occurs in a pharynx that is structurally less stable (or abnormal). The two hypotheses are not mutually exclusive but most studies today support the anatomical hypothesis.

In recent years, it has been recognised that the majority of OSAHS patients have multiple sites of upper airway obstruction that may vary according to the sleep stage and/or sleeping position.[3] This knowledge has important implications for treatment selection. In order to be successful, it will be necessary to combine several surgical procedures, each targeting a particular site of the UA, or to choose a procedure that alters the properties of the entire UA.

The arousal response that accompanies apnoea termination is likely to be determined by respiratory and non-respiratory stimuli. OSAHS patients have a higher arousal threshold compared to normals and this may further aggravate SDB.[4]

Prevalence of sleep-related breathing disorders

Janson *et al.*[5] administered questionnaires on sleep disturbances in a random population of 2202 subjects (aged 20-45 years) in four European cities (Reykjavik, Upsalla, Gothenburg and Antwerp). At all centres, 5% of the men and 2-3% of the women reported snoring every night. Snoring was positively correlated with age, male gender and body mass index in all areas.

The Wisconsin Sleep Cohort Study was a population-based prospective study using overnight polysomnography to investigate the epidemiological features of SDB. The results of this survey indicated that the estimated prevalence of SDB (defined as respiratory disturbance index (RDI) ≥5 events/hr) among employees between 30 and 60 years of age was 9 percent for women and 24 percent for men.[6] Two percent of women and 4 percent of men fulfil the minimum diagnostic criteria for sleep apnoea syndrome (RDI ≥5 events/hr and daytime hypersomnolence). In general population samples, the male/female ratio for SDB is 2-3/1, whereas a ratio of 10/1 is found in clinic-based studies.

There is however mounting evidence that 'sleep study OSAHS' as found in epidemiological studies is not the same as 'sleep clinical OSAHS', which presents because of significant symptoms. Symptomatic sleep apnoea in men meriting nCPAP treatment probably has a prevalence of 1-2%, depending on obesity prevalence.[7]

The prevalence of OSAS seems to increase with age, with peak values at the ages of about 50 to 60 years. There is, however, no consensus about whether OSAHS worsens over time in the absence of weight gain.

Clinical signs and symptoms

Snoring is the most constant symptom in patients with SDB. Heavy snoring is often a source of inconvenience and questioning the patient's bed partner can provide useful information about snoring and the nocturnal breathing pattern. In the mildest forms of SDB, snoring is an inspiratory noise associated with almost every breath. Snoring interrupted by quiet intervals representing apnoeas is a typical feature associated with OSAHS.

Excessive daytime sleepiness (EDS) is yet another major symptom of SDB. Excessive daytime sleepiness has been attributed to cerebral hypoxaemia and sleep fragmentation due to repeated arousals. Surprisingly, many patients will deny any significant daytime sleepiness. This may reflect tolerance to sleep fragmentation, personal tolerance to sleepiness as a negative trait, or the chronicity of the symptoms.

Habitual snoring and EDS are considered to represent the cardinal symptoms of SDB, although their absence does not exclude the disease. The relationship between these clinical complaints and disease expression is poorly understood. Snoring often occurs years before the onset of disease-related illness and many patients will deny any significant daytime somnolence.

In addition to these major symptoms, the patient may complain about morning headaches, nocturnal enuresis, altered state of consciousness, modifications of mood towards irritability and aggression, cognitive disturbances and a decrease of libido.

Consequences of SDB

Although no data from prospective case-control studies are available at present, SDB seems to be associated with increased morbidity and mortality that are attributed to the consequences and complications of the respiratory events occurring during sleep:
sleep fragmentation, intermittent hypoxaemia and hypercapnia, increased intrathoracic pressure swings, cardiac arrhythmias, polycythaemia, decreased cerebral blood flow, pulmonary hypertension and endocrinological disturbances.

A link between excessive daytime sleepiness resulting from SDB and an increased risk of work and road accidents has been documented. Findley *et al.*[8] demonstrated a sevenfold increase in the risk of road accidents in OSA patients (RDI >5 events/hr) compared to normals.

The results of epidemiological studies investigating the relationship between SDB and cardiovascular morbidity and mortality are often affected by the presence of confounding factors such as obesity, gender, diabetes, hypertension etc. in patients with SDB. Sleep apnoea is an independent risk factor for arterial hypertension and cardiovascular disease.[9]

Diagnosis of sleep-related breathing disorders

The diagnosis of SDB is essentially based on history, clinical signs and symptoms and full-night polysomnography (PSG).

History is taken by means of standard questionnaires for the evaluation of signs and symptoms, medical history, alcohol and tobacco consumption and use of medication.

Snoring can easily be documented using a visual analogue scale. The Epworth sleepiness score is widely employed to document the degree of excessive daytime sleepiness.[10]

The *general clinical examination* is usually quite normal. About 70% of the patients with SDB are found to be obese. In addition, increased neck circumference (>40 cm in men) is more often found in OSAHS.

An examination of the *ear, nose and throat* is recommended in each patient referred for SDB and may reveal anatomical abnormalities of the UA. Special attention should be paid to the pharyngeal dimensions and the pattern of upper airway narrowing should be documented using upper airway endoscopy with Müller's manoeuvre. In 2002, the Royal Belgian Society of ENT surgeons published a consensus paper on the details of the clinical ENT examination of patients with suspicion of sleep-disordered breathing and the reader is referred to this paper.[11]

A pulmonary function test is usually performed in patients admitted for PSG. Although lung function parameters are generally within normal limits, this examination is essential to exclude underlying lung disease.

Polysomnography

At present, we support the use of full PSG for the evaluation of subjects with complaints of socially disturbing snoring and/or excessive daytime sleepiness or other symptoms suggestive of SDB.[12] Nevertheless, we recognise that, in the future, the use of limited diagnostic procedures may be justified.

Treatment

The treatment of SDB disorders includes general measures, non-invasive devices, mandibular advancement devices, nasal continuous positive airway pressure and surgical procedures. In 2002, the Royal Belgian Society of ENT surgeons published a consensus paper on the surgical management of patients with SRBD and the reader is referred to this manuscript[13] (Figure 1).

After history and ENT clinical examination, a full PSG is required for patients with suspicion of SDB. When polysomnographic data are normal, a routine follow-up is proposed with general measures to achieve better sleep hygiene and to reduce risk factors. General measures include weight loss, alcohol and sedative avoidance, smoking cessation and sleep-position training for the patient, avoiding anteflexion of the head or lying on the back during sleep. Non-invasive treatments for SDB patients, including oral spray lubricant, nasal dilators (Nozovent®, Breathe Right®) to increase nasal patency, or a head-positioning pillow, did not prove beneficial when assessed using objective criteria in a polysomnographic study. However, as these devices are relatively inexpensive and have no side-effects, they can be used during a trial period, especially by the primary snoring patient.

Nasal continuous positive airway pressure (nCPAP)

Application of nCPAP is the first-line treatment for all OSAHS patients with moderate to severe disease.

The positive pressure acts as a pneumatic splint, opening up or preventing the collapse of the pharyngeal airway during sleep. Long-term CPAP treatment results in a decrease of UA oedema and an improvement of lung function parameters.[14] This treatment starts with clinical habituation and must be accepted by the patient. The effective mask pressure required to restore a normal ventilatory and sleep pattern is determined during a second PSG. A member of the sleep unit must evaluate long-term compliance with nCPAP therapy. The criteria for reimbursement of CPAP in Belgium are RDI >30/hr or RDI >20/hr and movement arousal index >30/hr.

Upper airway surgery and mandibular advancement devices (MADs)

Upper airway surgery or treatment by a mandibular advancement device might be considered for 1) patients with socially disturbing snoring (with or without daytime symptoms); 2) those with mild OSAHS or; 3) patients with moderate to severe disease in whom other non-invasive treatments have failed or have been refused.[15]

Mandibular advancement devices have gained increased attention and acceptance as a non-invasive alternative treatment for the management of SDB. The action of MADs, which are worn intra-orally at night to advance the lower jaw, is usually assumed to involve the enlargement of the retroglossal space by the anterior

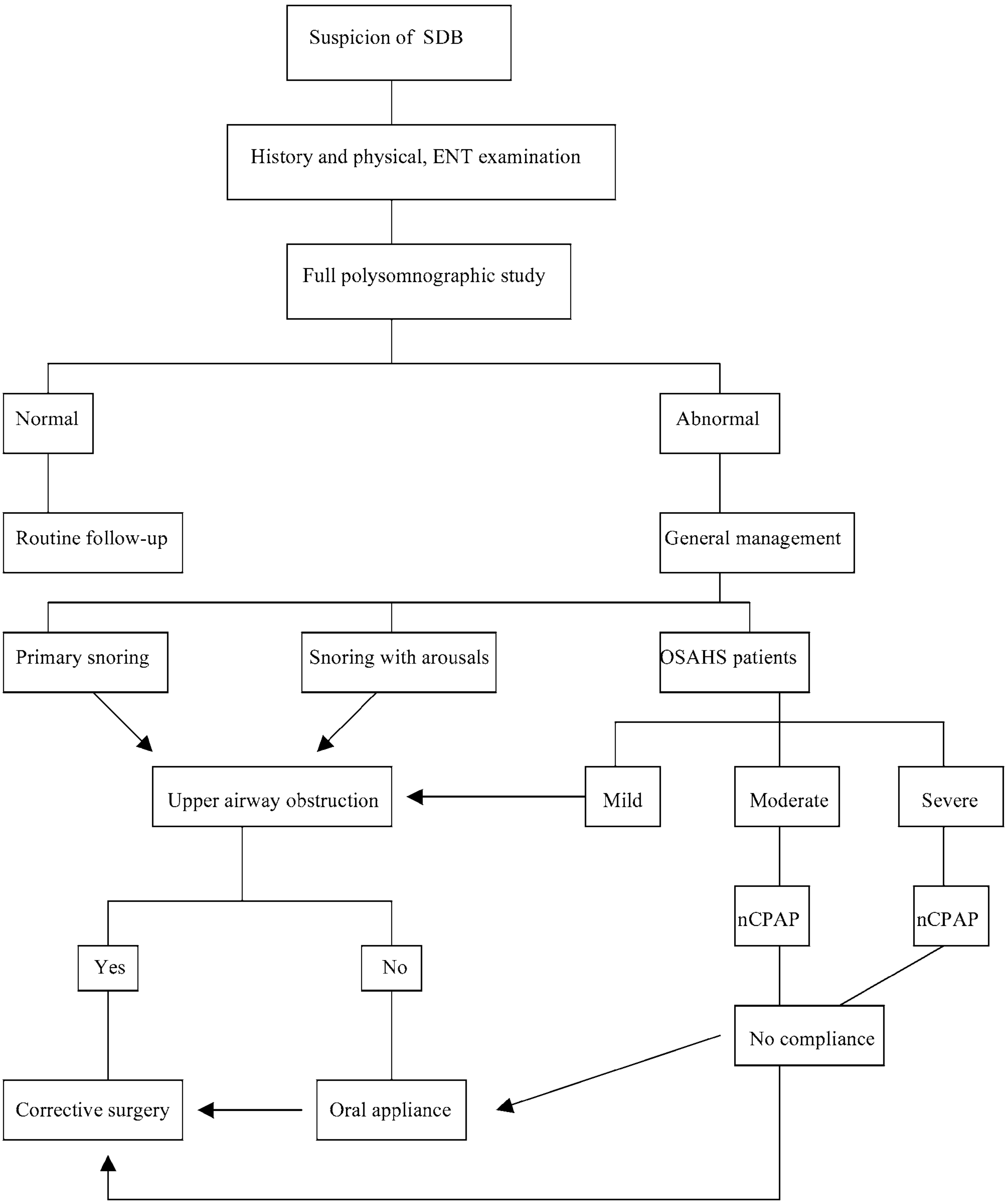

Figure 1
Management of patients suspected of sleep-disordered breathing. (Adapted from reference 13)

displacement of the tongue, the major muscle of which, the genioglossus muscle, inserts at the lingual surface of the anterior mandibular arch. By means of these anatomical changes, MADs can diminish collapsibility and thus reduce the severity of SBD by widening the cross-sectional dimension of the upper airway. A task force of the American Academy of Sleep Medicine recently published recommendations for the use of MADs in the treatment of SDB.[15]

Treatment with an MAD may be indicated in habitual snorers with or without associated daytime sleepiness, in snorers with arousals or in mild OSAS patients when no evidence of upper airway obstruction is observed or when there is retrognathia. Although MADs are not as effective as nasal continuous positive airway pressure (nCPAP) therapy, adaptation of an MAD may be indicated in subjects with moderately severe obstructive sleep apnoea (OSAHS) who do not tolerate or comply with their nCPAP device, or as a temporary alternative. MADs should also be considered as rescue treatment in patients with persistent SDB after uvulopalatopharyngoplasty (UPPP).

Corrective surgery for upper airway obstruction includes nasal, adenoid, velopharyngeal and retrobasilingual surgery. A detailed description of the various surgical techniques and their results can be found elsewhere.[16] At present, no data from randomised controlled trials are available comparing any surgical intervention for OSAHS with another surgical or non-surgical treatment modality.[17] Partner opinion is the best subjective measure of success in the treatment of snoring. Successful treatment of OSAHS is frequently defined as a postoperative reduction in AHI >50% and a postoperative AHI ≤20/hr or a postoperative apnoea index (AI) <10/hr. As there is often a discrepancy between objective and subjective results, it is mandatory to document the results of any surgical procedure for OSAHS or snorers with arousals by a postoperative PSG 3-6 months after the procedure.

Nasal surgery

Nasal surgery, including septo(rhino)plasty, inferior turbinate volume reduction (radiofrequency, microdebrider, turbinoplasty,...) and endoscopic sinus surgery, may be proposed to patients with SDB and increased nasal resistance. Although there is no good correlation between waking nasal resistance and the severity of SDB, the relationship between nasal disorders, especially nasal obstruction, and SDB is intimate.[18] Nasal obstruction may trigger sleep disorders in normal subjects and exacerbate them in snorers or OSAHS patients. Subjective analysis with questionnaires about snoring, daytime fatigue or excessive daytime sleepiness has revealed that the treatment of nasal disorders in SDB patients may be beneficial. However, objective data with pre- and post-therapy polysomnographic studies are far less encouraging. The success rate of isolated nasal surgery, for instance, seems to be less than 20%, although normalisation of nasal resistance is achieved in most cases.[19] Rhinological procedures may also be of interest for patients with poor compliance with nCPAP therapy due to nasal problems. An improvement in mask tolerance can be expected after medical or surgical treatment of nasal obstruction in these patients.

Velopharyngeal surgery

Velopharyngeal surgery is the cornerstone of the surgical management of SDB patients and essentially includes three different approaches: uvulopalatopharyngoplasty (UPPP), laser-assisted uvuloplasty (LAUP) and radiofrequency tissue volume reduction (RFTVR). The best candidates for velopharyngeal surgery are patients with a BMI of less than 28 kg/m^2 with a low (modified) Mallampati score, a low tonsil score, and without upper airway obstruction revealed at the retrobasilingual level. If a high tonsil score is observed during anterior pharyngoscopy, a tonsillectomy procedure must be performed during the velopharyngeal surgery. Velopharyngeal procedures are proposed to reduce soft palate vibration and with the aim of enlarging the velopharyngeal isthmus. They can be performed under general anaesthesia (UPPP) or under local anaesthesia (LAUP, RFTVR) and during single-stage sessions (UPPP) or multiple-stage sessions (LAUP, RFTVR).

Reductions in snoring to an acceptable level after surgery seem to be achieved in 60 to 80% of patients.[20] Cure rates tend to fall with time after surgery. These results are obtained with the three procedures, but there are major differences in the postoperative period since RFTVR is the least painful procedure[21] (Table 2).

Palatal implants have been introduced recently for the management of the snoring patient. These Pillar or palatal implants are inserted into the soft palate in order to increase the rigidity of the

Table 2
Post-operative pain and side-effects of velopharyngeal surgery

	UPPP	LAUP	RFTVR
Post-operative pain	severe (+/- 2 weeks)	severe (+/- 2 weeks)	moderate (+/- 1 week)
Narcotic drugs required	frequent	frequent	rare
Wound infection	frequent	rare	rare
Wound dehiscence	frequent	rare	rare
Bleeding	rare	rare	rare
Velopharyngeal fistula	rare	rare	rare
Posterior pillar narrowing	frequent	frequent	rare
Velopharyngeal stenosis	rare	rare	–
Problem with smell, taste	frequent	rare	rare
Pharyngeal dryness	frequent	rare	rare
Globus sensation	frequent	rare	rare
Voice change	frequent	frequent	rare
Pharyngonasal reflux	frequent	rare	rare

UPPP: uvulopalatopharyngoplasty, LAUP: laser-assisted uvulopalatoplasty, RFTVR: radio-frequency tissue volume reduction. (adapted from ref 21).

soft palate and subsequently to reduce snoring. Although clinical experience has been reported in only a limited number of patients, long-term results (up to 1 year) are promising in primary snorers, in whom there was a significant improvement in snoring and day-time sleepiness.[22]

The success rate for velopharyngeal surgery in unselected OSAHS patients is less than 40%.[23] Surgical success can be improved by proper patient selection based upon the pre-operative identification of the site(s) of UA obstruction.

Retrobasilingual surgery

Retrobasilingual surgery is also proposed for patients with SDB. One might consider procedures such as soft-tissue volume reduction using laser or radio frequency, or hard-tissue corrective management when mandibular osteotomy, genioglossus advancement or hyoid suspension are performed. These latter procedures are indicated for OSAS patients when compliance with nCPAP therapy is poor and when upper airway obstruction is demonstrated at the retrobasilingual level.

As many patients with SRBD have different levels of obstruction in their upper airway it seems logical to propose a multilevel surgical strategy. Combined UPPP and RFTVR of the tongue base, multilevel RFTVR in inferior turbinates, the velopharynx and the tongue base or combined nasal and velopharyngeal surgery are also accepted methods for the treatment of the SDBD patients. The risk of combining these procedures in one surgical setting must be taken into account, especially in OSAHS patients. The combination of nasal surgery (with postoperative nasal packing) and velopharyngeal or tongue base surgery in one setting might result in postoperative airway compromise.

Preoperative information about the site(s) of UA obstruction can be obtained with static methods such as cephalometry, computed tomography or magnetic resonance imaging, but dynamic studies are far superior. Sleep endoscopy during drug-induced sleep (propofol or midazolam) is a non-invasive method for identifying the site of UA collapse. When combined with polysomnographic and other clinical data, sleep endoscopy was found to be very helpful for providing individually tailored treatment advice.[24] Figure 2 sets out an approach for surgical treatment that takes the site of UA obstruction into account.

Conclusions

Sleep-disordered breathing and sleep medicine integrate many specialities such as neurology, pulmonology, oral surgery and otorhinolaryngology.

The otorhinolaryngologist performs airway evaluation before

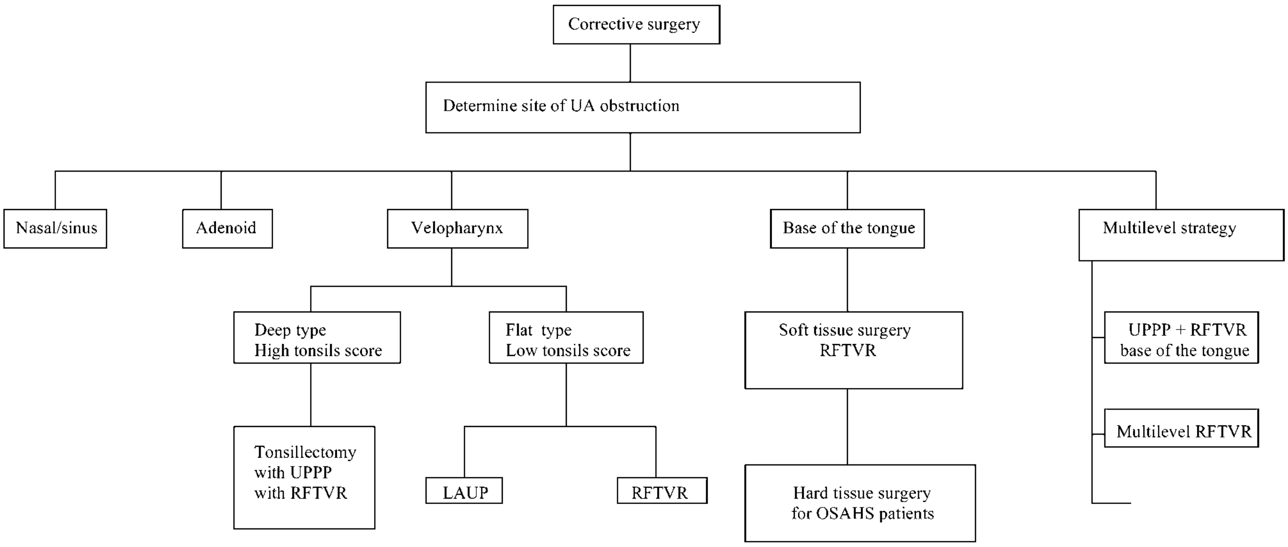

Figure 2
Options for UA surgery according to site of UA obstruction and clinical features

surgical treatment and the surgical treatment itself when indicated. We must also emphasise that the otorhinolaryngologist must have a working knowledge of the physiology of sleep and respiration and the health consequences of the sleep-related breathing disorders.

Finally, surgical modalities and tools are evolving that reduce postoperative discomfort without jeopardising outcomes.

Patient information

Sleep-disordered breathing encompasses different clinical situations: from primary snoring to obstructive sleep apnoea-hypopnoea syndrome. Snoring is caused by diffuse vibrations of the pharyngeal walls and suggests increased upper airway resistance during sleep. In about 5% to 10% of snorers, pharyngeal narrowing leads to complete occlusion and apnoea. Snoring is therefore a marker of obstructive sleep apnoea-hypopnoea syndrome (OSAHS), which is considered to be a risk factor for hypertension, cardiovascular disease or cerebrovascular disease. A sleep evaluation approach known as a ‘polysomnographic study’ is mandatory for patients with a clinical suspicion of apnoea or when a surgical correction of an upper airway obstruction is indicated for patients with sleep-related breathing disorders.

Treatment of non-apnoeic snoring begins with the identification and correction of risk factors (weight loss, smoking habit, avoidance of hypnotic medication,...). Velopharyngeal surgery and/or a rhinological procedure and/or a retrobasilingual procedure are proposed to reduce snoring and to enhance the sleep quality of both the patient and the bed partner. Velopharyngeal surgery is performed with different tools and instruments: uvulopalatopharyngoplasty (UPPP), laser-assisted uvulopalatoplasty (LAUP) or radio-frequency tissue volume reduction (RFTVR). Radio frequency is associated with less postoperative discomfort than other techniques. After these procedures, there is less snoring in 60% to 80% of cases.

For apnoeic patients, initial treatment includes nasal mask with continuous positive airway pressure (nCPAP). This treatment is associated with a high cure rate for sleep apnoea-hypopnoea syndrome patients and with a decreased risk of OSAHS-related comorbidity. Upper airway surgery or the use of a mandibular advancement device might be considered in those patients for whom CPAP treatment fails or is unacceptable after a therapeutic trial.

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CME Questions

1. What is incorrect in the following definition of sleep apnoea-hypopnoea syndrome?

   A – Five or more obstructive events per hour of sleep demonstrated with polysomnography
   B – And lasting more than 10 seconds
   C – With a > 50% decrease from baseline in amplitude of a valid measure of breathing during sleep
   D – Or a < 50% amplitude reduction of a valid measure of breathing during sleep associated with either an oxygen desaturation of > 3% or an arousal
   E – With an arousal defined as any shift in EEG to alpha or theta rhythm for at least 15 seconds

2. The prevalence in men of OSAHS is usually estimated to be approximately:

   A – 0.1%
   B – 2%
   C – 5%
   D – 10%
   E – 15%

3. What is incorrect regarding the following statement: Excessive daytime sleepiness is:

   A – A major symptom of SDB
   B – Attributed to cerebral hypoxaemia
   C – Attributed to sleep fragmentation due to repetitive arousals
   D – Related to the Epworth Sleepiness Scale
   E – Secondary to central apnoea in SDB patients

4. Respiratory events during sleep involve all the consequences and complications below, with one exception.

   A – Cardiac arrhythmias
   B – Intermittent hypocapnia
   C – Pulmonary hypertension
   D – Intermittent hypoxaemia
   E – Sleep fragmentation

5. Moderate to severe forms of OSAHS must be treated with

   A – nCPAP
   B – Nasal dilators
   C – Oxygenotherapy for every patient
   D – Hypnotic medications
   E – Oral corticoid

6. Which of the following does not apply to nasal surgery for SDB patients?

   A – Indicated for patients with nasal obstruction and poor nCPAP compliance
   B – Successful for > 80% of OSAHS patients
   C – Included in a multilevel strategy of upper airway obstruction treatment
   D – Indicated for patients with increased nasal resistance
   E – Sometimes associated with a worsening of OSAHS

7. Velopharyngeal surgery for SDB patients involves all these different approaches, with one exception:

   A – RFTVR
   B – UPPP
   C – LAUP
   D – Pillar implant
   E – Velopharyngeal stenosis

8. A second polysomnography after a treatment is mandatory in all these circumstances, with one exception:

   A – Primary snoring patient
   B – For moderate OSAHS
   C – For severe OSAHS
   D – To adapt nCPAP mask for OSAHS patients
   E – After surgical failure

9. Which examination is essential before a surgical procedure for SDB?

   A – MRI
   B – CT Scan
   C – Polysomnographic study
   D – Cephalometry
   E – Rhinomanometry

10. What is incorrect regarding the following statement: excessive daytime sleepiness …:

   A – RFTVR seems to be less painful for the patient than UPPP
   B – Velopharyngeal surgery is successful in 60-80% of primary snoring patients
   C – The best candidates for velopharyngeal surgery are patients with a BMI > 28 kg/m^2
   D – Tonsillectomy may help in the management of SDB patients
   E – Snoring cure rates tend to drop with time after surgery

Answers: 1E; 2C; 3E; 4B; 5A; 6B; 7E; 8A; 9C; 10C