Chapter 17: Sleep-related breathing disorders

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The last few years have seen a relative explosion in learned papers dealing with sleep-related breathing disorders. The knowledge that a number of significant physiological abnormalities may be attributed to sleep disorder is not, however, new. Recent interest has been stirred with understanding of the causes of these disorders and new approaches to treatment.

The sleep apnoea syndromes are a common cause of excessive daytime sleepiness and may lead to pulmonary hypertension, systemic hypertension, cardiac arrhythmias, mental dysfunction, heart failure and sudden death. The otolaryngologist is increasingly involved in the multidisciplinary approach to assessment and treatment of the obstructive and mixed forms of sleep apnoea; it is important for the modern otolaryngologist to be familiar with the diagnosis and treatment of sleep apnoea syndromes. Snoring is no longer to be considered a rather humorous, frivolous sign of no clinical significance (Robin, 1968), a status accorded for many years. Snoring is a common feature of sleep apnoea syndromes and recent literature has concentrated on it as a possible symptom or sign of a more sinister disorder (Guilleminault and Dement, 1978; Morton, 1986). Incidence figures for snoring state that 53% of an adult male population will snore intermittently and 31% will snore regularly; for women the figures given are 38% snoring intermittently and 19% regularly (Lugaresi, Coccagna and Cirignotta, 1978). Snoring increases with age and up to 60% of men and 40% of women between the ages of 60 and 65 will snore; in the 30-35 year age group the percentages are only 20% for men and 5% for women (Lugaresi et al, 1982). Most people in this large population will be subjectively undisturbed by their snoring yet current work at The Royal National Throat, Nose and Ear Hospital in London, and Stanford Medical Center, California, supports the view that patients with 'heroic' snoring (snoring that occurs in any body position and can be heard in the next room through a closed door), even without daytime sleepiness, may have an occult form of sleep apnoea syndrome and require treatment for medical as well as social reasons.

Before discussing sleep apnoea syndromes, some definitions should be given:

Apnoea - cessation of air flow at the nostrils and mouth for at least 10 seconds.
Apnoea subtypes - apnoea can be subdivided into:
  - central - no air flow or respiratory effort
  - obstructive - no air flow despite respiratory effort
  - mixed - a combination of central and obstructive apnoea.
Hypopnoea - a fall in the average tidal volume by more than 50%.
Apnoea index - the apnoea index equals apnoeas and hypopnoeas per hour of sleep.
Sleep apnoea syndrome - the diagnosis is made when the apnoea index equals or exceeds 5 episodes per hour or 30 over 7 hours. It must be classified as central, obstructive or mixed.

Since night-time sleep is interrupted by episodes of apnoea, the patient is sleepy the next day (excessive daytime somnolence); in fact this is so much so that he/she may fall
asleep while driving to work, or fall asleep at inappropriate moments during the working day. The obstructive form of sleep apnoea occurs almost exclusively in men (96%), and is regularly accompanied by loud snoring (100%) and abnormal movements during sleep (kicking of legs, slapping of arms, sitting up, falling out of bed and struggling for breath) (Guilleminault and Dement, 1978). Intellectual deterioration, with inattention and difficulty in concentrating, occurs in 78% while 48% have personality changes; 42% note impotence while 30% have intermittent nocturnal enuresis, and 36% morning headaches. Systemic hypertension (systolic pressure 150-210 mmHg, diastolic 95-120 mmHg) is present in 52%. Eighty per cent of the subjects are overweight. Interestingly enough, only 78% of this group with documented obstructive sleep apnoea have a chief complaint of excessive daytime somnolence - the remainder are referred primarily for loud snoring and apnoeic episodes during sleep, usually noted by the spouse and present for many years.

The diagnosis of sleep apnoea is confirmed by nocturnal polysomnogram. This study consists of simultaneous measurement during sleep of eye movement (electro-oculogram (EOG) or electronystagmogram (EEG)), electrocardiogram (ECG), chest/abdominal respiratory movements, nasal and oral air flow, oxygen saturation (using an ear-lobe or finger oximeter) and often a chin electromyogram (EMG). The study is ideally performed overnight in a sleep laboratory.

The EEG allows sleep to be staged and periods of rapid eye movement (REM) and non-REM sleep are identified by EOG. REM sleep usually occurs between 03:00 and 06:00 in normal individuals and is often accompanied by dreaming. Sleep apnoea is more common during REM sleep. The Holter monitor electrocardiogram shows the presence of bradycardia and tachycardia plus any other arrhythmia. The measurement of chest/abdominal movements and oral/nasal air flow is essential for documenting the length and number of apnoeas and hypopnoeas, as well as distinguishing central from obstructive apnoea. The oximeter documents any desaturation that may occur during sleep, while the chin EMG gives an index of the amount of mylohyoid muscle tone, so that the stage of sleep can be determined. Additional measurements can be made; body movements can be recorded with a vibration transducer and snoring documented with a tape recorder.

The nocturnal polysomnogram as outlined above is both time-consuming and expensive; full technical support is required and 'laboratory conditions' are necessary to standardize data collection. In a clinical sense full polysomnographic assessment is beyond the means of most departments of otolaryngology. The microprocessor has provided cheaper and more convenient devices which are becoming increasingly available for the purposes of sleep screening. The Vitalog, similar in use to the Holter monitor, can be taken home by the patient and worn overnight (Miles and Rule, 1986). The latest oximeters can similarly be worn in the home situation and are simple enough to be patient-operated - these store up to 8 hours of data, covering the sleep period, and can be used to display trends of oxygen desaturation by simple connection to a microcomputer. Devices such as these should put the diagnosis of sleep apnoea syndrome well within the reach of most departments and could be used to screen for the more 'at risk' patients who would benefit from further investigation and treatment.

Central sleep apnoea presents in a different way to obstructive sleep apnoea syndrome. Insomnia and depression were more frequent symptoms in one series (Guilleminault and
Dement, 1978) and excessive daytime somnolence may not be the most frequent complaint; 80% of the patients were male and the average age at presentation was 45 years.

Common causes of sleepiness must be evaluated. 'Expected sleepiness' may relate to lifestyle or the working of shifts; the taking of various drugs can lead to undue somnolence. Other causes include the narcolepsy - cataplexy syndrome (Mendelson, Gillin and Wyatt, 1977), which is commonly confused with sleep apnoea syndrome - the two conditions may coexist; periodic movements of sleep (nocturnal myoclonus) may be implicated. Central and obstructive sleep apnoea must be distinguished from these other possible causes of excessive daytime somnolence. Idiopathic hypersomnolence is a diagnosis of exclusion.

Narcolepsy is a sleep disorder characterized by attacks of sleep with sudden onset and short duration, around 15 minutes, that may occur at any time. The attacks are usually one to several hours apart and after an attack the patient feels refreshed. The onset of the disease is usually between 10 and 20 years of age; both sexes are equally affected. Narcolepsy is commonly associated with episodes of cataplexy - a sudden loss of tone in the major striated muscles producing total or partial collapse. These episodes may last from seconds up to 30 minutes. Sleep paralysis, a paralysis of striated muscles that occurs at the onset of sleep, and hypnogogic hallucinations (auditory, visual or tactile hallucinations that occur at sleep onset) are also seen in association with narcolepsy. Difficulties with sleep at night are seen. The diagnosis is confirmed by the presence of REM-onset sleep during a daytime sleep study, since daytime sleep does not normally begin with rapid eye movements.

Sleep apnoea syndromes may obviously coexist with conditions which contribute to the symptomatology. Chronic obstructive airways disease, pulmonary fibrosis, and heart disease with congestive cardiac failure may all mimic sleep apnoea syndromes and should be differentiated by appropriate history taking and relevant investigations. Cerebral tumours, hypoglycaemia, severe anaemia and hypothyroidism may all cause excessive daytime somnolence; depression is another common cause.

Pickwickian syndrome is a combination of excessive daytime somnolence, morbid obesity and right heart failure - alveolar hypoventilation produces an elevated $P_{CO_2}$, depressed $P_{O_2}$ and the patients show a marked tendency to oropharyngeal collapse during sleep. A polysomnogram is necessary to determine the role of obstructive sleep apnoea syndrome in these patients.

Conditions producing obstructive sleep apnoea syndrome must be looked for and treatment possibilities evaluated. Any condition which narrows the oropharynx, hypopharynx or supraglottic larynx can produce obstructive sleep apnoea syndrome. This includes hypognathic mandible, hypertrophic tonsillar tissue, long soft palate and uvula, large tongue (as in acromegaly and hypothyroidism) and tumours of the pharynx, supraglottic larynx, tongue base and neck. Less common as a sole cause, but possibly contributing to obstructive sleep apnoea syndrome, is nasal or nasopharyngeal obstruction due to nasal septal deviation, large adenoids, allergy or tumour.

Drugs which may contribute to or cause sleep apnoea syndromes include alcohol, sleeping pills, tranquilizers, sedatives, anti-epileptic drugs and antihistamines. Withdrawal of the drug should be effected when evaluating the patient.
The evaluation of obstructive sleep apnoea syndrome requires, therefore, careful history taking and examination - nose, nasopharynx, oral cavity, oropharynx, hypopharynx, larynx and neck must be inspected to rule out abnormal enlargements and narrowing as well as obstructing tumours. Further investigation will now commonly include haemoglobin (anaemia), haematocrit (polycythaemia in chronic oxygen deprivation), high kilovoltage neck imaging in normal respiration and phonation (palatal and faucial relations), fibreoptic nasoendoscopy and rhinomanometry (assessment of obstruction to nasal air flow).

The severity of sleep apnoea syndromes can be gauged by polysomnography. The higher the apnoea index, the worse the condition. Oxygen desaturation greater than 50%, or long runs of either bradycardia or tachycardia, suggest a severe state, as do ventricular arrhythmias. Pulmonary hypertension or right heart failure as a result of sleep apnoea syndrome obviously denotes a severe form of the disease. Patients with coronary artery disease, chronic obstructive, fibrotic pulmonary disease, or morbid obesity may start with a low oxygen saturation, elevated carbon dioxide, pulmonary hypertension and a tendency for cardiac arrhythmias, so that what would normally be a mild case of sleep apnoea syndrome may be severe in these individuals. Sudden death in sleep apnoeics is not rare and has been noted in both infant and adult populations (Guilleminault et al, 1984).

No firm classification is accepted at the time of writing. Each case must be judged individually for severity; there are no hard and fast rules for guidance - a situation not unfamiliar to the practising clinician in most areas of surgery. Severity and type of sleep apnoea syndrome determine treatment possibilities and it is necessary to seek some classification. Surgical management options for obstructive sleep apnoea syndrome are discussed below and indications for the various types of surgical management available are listed.

**Investigations for obstructive sleep apnoea syndrome**

*Fibreoptic endoscopy*

The flexible nasoendoscope is routinely used as an outpatient facility in many departments of otolaryngology. Nasoendoscopy during sleep can provide valuable information as to the site of obstruction in obstructive sleep apnoea syndrome (Guilleminault et al, 1978; Rojewski et al, 1982). Nocturnal nasoendoscopy is chiefly a research tool used to site the level of obstruction visually; the Müller manoeuvre, performed on the awake patient, provides similar information. The nasoendoscope is passed to the postnatal space and the patient is instructed to attempt a snore with the mouth closed - the principal site of obstruction can be seen directly (Sher et al, 1985).

*Imaging*

A lateral cephalometric X-ray of the head and neck, performed by a competent technician, may be useful in determining the site of obstruction (Riley et al, 1983). A lateral high kilovoltage neck radiograph, taken in phonation, is also useful in demonstrating velopharyngeal sphincter action and the relevant dimensions of this important region. A hypopharyngeal site of obstruction is suggested by a dimension of less than 10 mm, measured from the base of the tongue to the posterior pharyngeal wall.
Computerized tomographic (CT) scanning at the level of the oro- and hypopharynx can be useful, particularly if the patient falls asleep during the examination (not so unlikely as it may sound when the patient is suffering true excessive daytime somnolence) (Haponik et al, 1982).

In some centres fluoroscopy may also be of use and its proponents make strong claims for its use in establishing the exact site of obstruction - a most necessary preliminary to successful management, particularly of the surgical type (Smith et al, 1978; Suratt et al, 1983).

**Pulmonary function testing**

Hypoventilation must be fully evaluated when assessing an obstructive sleep apnoea sufferer. The measurement of flow-volume loops has been suggested as a test for obstructive sleep apnoea during the day (Haponik et al, 1981; Sanders et al, 1981); however, results obtained on awake patients do not regularly diagnose obstructive sleep apnoea syndrome during sleep, even when the tests are performed with the patient supine (Riley et al, 1983; Tammelin et al, 1983). The tests can be helpful if positive for upper airway obstruction. Cardiopulmonary disorders with upset of ventilatory function can be assessed with blood gas studies. As a general rule, pulmonary function and blood gas studies obtained while awake do not correlate with studies obtained during sleep (Garay et al, 1981). In many centres an in-dwelling catheter is placed prior to sleep, so that arterial oxygen saturation (SaO₂) levels as indicated by ear-lobe oximetry can be matched with blood gas values; this should become unnecessary with reliable oximetry and is obviously unacceptable as a routine clinical tool.

**Sleep latency test**

This test measures the time taken by a subject to fall asleep and is monitored by an EEG. It gives information on the degree of excessive daytime somnolence experienced by a subject and can be used for assessment purposes both before and after therapy - it does offer an objective method for measuring response to therapy (Orr and Moran, 1985). Sleep apnoea syndrome patients have a short sleep latency time, usually less than 4 minutes; normal values fall in the 6-15 minute range. Multiple sleep latency tests consist of evaluations over a 48-hour period, with a polysomnogram each night and six sleep latency tests per day at 2-hour intervals. Tests of mental function are performed throughout. Patients with obstructive sleep apnoea syndrome tend to have shorter sleep latency tests earlier in the day and longer ones later in the day. Normal subjects tend to have longer sleep latency tests at the beginning and end of the day with the shortest times in the middle of the day.

**Sleep apnoea in children and infants** (see Volume 6, Chapter 23)

Obstruction of the upper airway may arise from multiple factors, both congenital and acquired. Infants with large tonsils and adenoids, Pierre Robin syndrome, retrognathia or Crouzon's disease may present with obstructive sleep apnoea syndrome. Cleft palate repair with a pharyngeal flap may also initiate the syndrome. Sleep apnoea syndrome has been linked with sudden infant death syndrome (Guilleminault et al, 1984). Central and obstructive factors must be fully evaluated in the management of these children and preoperative assessment must include chest radiology, ECG and full blood count - also be alert to the risks
from secondary effects of prolonged sleep apnoea syndrome (polycythaemia, cardiomegaly, pulmonary hypertension, etc).

**Treatment**

The main emphasis in this section will be on surgical treatment, now usually performed by the otolaryngologist/head and neck surgeon. As in all areas of surgical management, treatment chosen will depend upon the cause and severity of the condition.

**Non-surgical treatment**

Medical means are used for the treatment of central sleep apnoea and most of the drugs used can improve obstructive sleep apnoea as well. Weight loss for the obese is often helpful (Harman, Wynne and Block, 1982) and the use of a tricyclic antidepressant, such as protriptyline, has also produced some improvements (Clark et al, 1979). It is necessary to be aware of the increased potential for cardiac arrhythmias with tricyclic agents; there is an increased incidence of arrhythmia in the sleep apnoea syndrome sufferer. Monitoring is required and protriptyline may be given in one 20 mg dose at night and increased to 30 mg if symptoms persist - the side-effects of tachycardia, urinary retention and dry mouth may occur (a result of the anticholinergic action).

L-Tryptophan has been successfully used in patients with mild central sleep apnoea syndrome (Schmidt and Jackson, 1982). The usual dose is between 3 g and 5 g taken orally 30 minutes before bedtime.

Patients with hypoventilation and decreased ventilatory drive may benefit from medroxy-progesterone acetate, a respiratory stimulant. Sleep apnoea syndrome sufferers, including those with obesity, have found this preparation of use (Strohl et al, 1981); it is of most use in mild cases with an apnoea index below 50 and oxygen saturation above 60%. An average daily dose by mouth is between 60 mg and 120 mg.

Other drugs used include acetazolamide (White et al, 1982) and naloxone (Atkinson, Surath and Wilhoit, 1983), without conclusive results. All drug therapy plans should exclude central nervous system depressants (Guilleminault et al, 1982), alcohol being a major culprit.

High altitudes may exacerbate the condition of a sleep apnoea syndrome patient. Low flow oxygen 1-3 L/min) can be helpful at night - particularly where hypoxia is implicated in the aetiology of the apnoea (Martin et al, 1982). Oxygen therapy is contraindicated if hypoxia is helping to initiate the breathing response.

Continuous positive airway pressure (CPAP) has proved most effective in the management of obstructive sleep apnoea syndrome (Sanders, Moore and Eveslage, 1983). Obese individuals, with a mixed apnoeic picture, often benefit from this therapy - surgery has an increased risk in this group since the problem is not entirely obstructive (Rapoport et al, 1982). The equipment for nocturnal use is now commercially available and it provides a tightly-fitting nasal mask, connected by tubing to a quiet pump; the mask is worn during sleep and the patient quickly comes to rely on the improved quality of sleep achieved.
Similar improvements can be achieved by the use of prosthetic devices; many have been patented. They may be indicated in selected cases where tongue retention in a forward position seems particularly desirable and the patient can tolerate the device (Cartwright and Samelson, 1982). It is estimated that over 300 anti-snoring devices have received patents in the USA during this century (Garfield, 1983).

**Surgical treatment**

**General anaesthesia for obstructive sleep apnoea syndrome patients**

These patients require special attention from the anaesthetist since they may pose several problems. They are often obese and possess the short, thick neck for which anaesthetic intubation is often difficult. Mask ventilation can be inadequate since hypopharyngeal structures behave under anaesthetic as they do under sleep conditions, collapsing inwards; pharyngeal tubing and mandibular elevation may help but rapid intubation under these adverse conditions may be required. Due to the common combination of central sleep apnoea, obstructive sleep apnoea and chronic obstructive airways disease, the use of high concentration oxygen plus positive pressure ventilation, which blows off carbon dioxide, may depress the ventilatory drive reducing or abolishing spontaneous respiration, even in a relatively light anaesthetic state. Paralysing agents routinely used for intubation may aggravate this tendency.

Immediate postoperative problems may occur in the recovery area since the ventilatory drive may remain depressed, due to the high PO\textsubscript{2} and low PCO\textsubscript{2} following anaesthesia. Mask ventilation may present similar problems to those encountered during the induction period, with the added problems of postoperative oedema and bleeding. Severe hypoxia is a risk since these patients are often used to a relatively low PO\textsubscript{2} under normal circumstances, due to accompanying pulmonary or cardiac disorders, and they need just a short time to reach a dangerously hypoxic level. Increase in pulmonary compliance is often seen, adding to the difficulty.

The following rules of thumb are suggested for general anaesthesia in the obstructive sleep apnoea syndrome:

1. Intubations in sleep apnoea syndrome patients may be difficult and conventional intubation techniques may not apply; fibreoptic 'rail-roading' of the endotracheal tube should be considered, or even awake nasal intubation;

2. Preoperative sedation is considered undesirable - despite some suggestions from recent research that REM-sleep blocking premedication is safe in children (B. Donaghue, 1987, personal communication);

3. Paralysing agents should be avoided during intubation;

4. Nasopharyngeal tubes should be checked for length (nares to epiglottic tip) and inserted for airway maintenance during induction; simple oral airway maintenance during induction; simple oral airways may not give sufficient anterior tongue displacement for an adequate airway; the same is true for mandible elevation or dislocation;
(5) ear-lobe oximetry is useful as a monitor of haemoglobin $\text{SaO}_2$ and is simple to use during induction of anaesthesia, extubation and in the postoperative period;

(6) facilities should be available for tracheostomy;

(7) extubation should not be performed until the patient is fully awake;

(8) narcotics and hypnotics should be used with extra caution in these patients, both before and after surgical procedures; respiration must be carefully observed;

(9) careful postoperative monitoring is essential, the nasopharyngeal tube being left in place where adequacy of the airway is in doubt;

(10) selected cases may benefit from the preoperative administration of steroids and 48-hour, postoperative humidification of inspired air is helpful; steroids may be given in reducing dosage over 5 days.

**Nasal surgery**

Selected patients from a population of obstructive sleep apnoea syndrome sufferers may benefit from nasal surgery of one form or another; nasal obstruction is rarely the primary cause however. Correction of a deviated nasal septum may give both subjective improvement and a diminution in the number and duration of obstructive sleep apnoea episodes (Simmons and Hill, 1974; Heimer et al, 1983).

Correction of the deviated nasal septum could help by:

(1) improving reflex mechanisms: upper airway obstruction could lead to apnoea via disturbed reflex mechanisms (trigeminally or vagally mediated), which normally act to preserve airway patency in the presence of negative pressure in the upper airway (Mathew, Abn-Osba and Thach, 1982);

(2) direct mechanical effects: obstruction of the upper airway causes an increase in the pressure gradient for air flows and the pressures within the upper airway become more negative relative to atmospheric pressure - leading to secondary collapse in the hypopharyngeal area, snoring and, possibly, sleep apnoea syndrome.

When nasal obstruction is present and symptomatic it should be treated. If surgical treatment is required for a deviated nasal septum, nasal polyps, hypertrophic turbinates or enlarged adenoids, then it should be performed to attain improvement of the symptomatic nasal obstruction - and as an additional benefit it may improve, to an unpredictable degree, the symptoms of obstructive sleep apnoea syndrome, including snoring.

**Uvulopalatopharyngoplasty**

This operation involves resection of about 1.5 cm of the free, posterior border of the soft palate and includes the uvula, posterior tonsillar pillars and part of the posterior pharyngeal wall medial to the pillars each side - the amount of tissue taken varies with the
amount of 'redundant', loose, mucous membrane present (Simmons, Guilleminault and Silvestri, 1983). The operation is performed with a tonsillectomy if tonsils are present. It has been about 50% successful in the correction of obstructive sleep apnoea syndrome (attenuated apnoeas tend to persist) and 95% successful in relieving snoring. Subjective improvement is usually marked and believed to be related to the less frequent/shorter duration apnoeas induced in most subjects; there are also beneficial secondary social results for the sufferer.

Fujita et al (1981) described the surgical technique in the USA, which is a modification of a snoring operation reported by Ikematsu (1964). Variations of the surgical technique have been reported by Simmons (Simmons, Guilleminault and Silvestri, 1983) and Hernandez (1982).

Despite continued experience with this procedure and well-documented series (Fujita et al, 1985; Katsantonis et al, 1985), the criteria for 'success' are still vague and the parameters for prediction of surgical outcome uncertain. The authors feel that a 'successful operation' should reduce the apnoea index to less than 20 and maintain a haemoglobin SaO₂ greater than 80%. With these two demands in mind, the present authors' indications for the operation are:

1. oxygen saturation which drops below 80%;
2. apnoea index worse than 20;
3. significant daytime sleepiness;
4. heroic snoring - producing marital or social problems;
5. significant cardiac arrhythmias during sleep.

Two or more of any of these indications is a reason to consider this operation.

The history is an important consideration. Excessive daytime somnolence leading to loss of job, or accidents with machinery, is clearly significant. The authors consider it justifiable to perform the operation for 'heroic' snoring alone, but recommend a preoperative sleep screening/Vitalog study to rule out concomitant obstructive sleep apnoea syndrome (with full polysomnography in suspects). If obstructive sleep apnoea syndrome is diagnosed then close follow-up must be ensured; postoperative polysomnography will demonstrate the degree of control achieved and indicate the need for any further therapy.

Severe obstructive sleep apnoea syndrome still requires tracheostomy for immediate management; these patients show haemoglobin SaO₂ below 50% on a repeated basis and/or significant, sleep-induced, cardiac arrhythmias (ventricular tachycardia, long runs of ventricular extrasystoles or bradycardia below 30 beats/minute). Uvulopalatopharyngoplasty is now often combined with tracheostomy in the hope that the obstructive sleep apnoea syndrome will be corrected by it, thus enabling early reversal of the tracheostomy. This combined procedure minimizes worry over further airway deterioration subsequent to uvulopalatopharyngoplasty alone (postoperative oedema or bleeding) and can be converted to permanent tracheostomy if decannulation fails. Most patients with obstructive sleep apnoea
syndrome undergoing tracheostomy and uvulopalatopharyngoplasty have a contributing disorder which produces daytime hypoventilation; they have abnormal SaO₂ levels while awake.

The relative contribution of the various components of uvulopalatopharyngoplasty in a successful outcome is not yet known. It would seem that the palate resection is the part of the operation which does the most good in the majority of patients without tonsils, particularly for the relief of troublesome snoring. Scarring and contracture may well play a part in preventing collapse at this level, the main obstructive component. More experience with the technique, under properly controlled conditions, will be necessary before an answer to this question is found. Identification of the sleep apnoea syndrome patient most likely to benefit from the procedure is still fraught with difficulty and there are, as yet, no clear guidelines. The uvulopalatopharyngoplasty is not effective for obstruction at the level of the hypopharynx, and these patients should be identified from the physical examination and subsequent investigations.

The presence of retrognathia, Angle class II malocclusion or macroglossia, producing difficulty in visualization of the larynx on indirect mirror examination, should raise the suspicion that the level of obstruction is in the hypopharynx. Obese patients, with short, fat necks should also be examined with hypopharyngeal obstruction in mind - the large amount of fat in the neck tends to sag inwards during sleep, as tonus in the pharyngeal musculature diminishes. A depressed, malformed hyoid may also contribute to the obstruction.

There are many subjects with obstructive sleep apnoea syndrome, where the site(s) of obstruction cannot be determined by simple physical examination; a multidisciplinary approach must be used and the otolaryngologist can contribute significantly with the manoeuvres and investigations previously described.

### Tracheostomy

This ancient operation in the repertoire of the otolaryngologist still provides the standard by which all surgical procedures for obstructive sleep apnoea syndrome must be judged. It is invariably successful but has the distinct disadvantage of a hole in the neck. The tracheostomy tube is valved during the working day, to enable normal speech, and left open at night. Variations on the theme have been tried, but removal of the tracheostomy generally leads to a return of obstructive sleep apnoea syndrome; some cases tried in conjunction with uvulopalatopharyngoplasty give cause for optimism.

General anaesthesia is preferred for this procedure and a cuffed tracheostomy tube is inserted in adults. The type of tube chosen for permanent use is at the discretion of the individual surgeon. Fenestration of the tube is desirable and the patient is taught self-management at an early stage. The Montgomery (1980) tracheostomy tube requires no ties and has several advantages over conventional tracheostomy tubes, in patients with necks of normal diameter. It does not have a cuff, a disadvantage in the early postoperative period in case of bleeding or need for positive pressure ventilation. Permanence of the tracheostomy will be facilitated by skin flaps, such as those turned inward to line the tract as described by Fee and Ward (1977).
An extra-long tracheostomy tube may be required in particularly obese patients; it is wise to have this tube available at the start of the procedure. Positioning of the tube tip after insertion must be carefully checked, since there is a real risk of insertion into the right main bronchus. Chest X-ray or fibreoptic bronchoscopy will confirm the correct location. It is necessary to assess the tube daily for a while to avert the problem of it slipping into a lower position. Obese patients with thick, fat necks heal slowly after tracheostomy. Antibiotic prophylaxis is recommended in these patients.

**Other surgical procedures for obstructive sleep apnoea syndrome**

**Mandibular advancement**

Malocclusion and retrognathia are surgically correctable; sagittal split osteotomies will correct the abnormality and pull the tongue forward, eliminating obstructive sleep apnoea syndrome due to these deformities (Bear and Priest, 1980).

**Genial tubercle advancement**

The genioglossus muscle attaches to the genial tubercle on the inner aspect of the anterior mandible; advancement of this structure should pull the tongue forward, even in the absence of retrognathia (Riley et al, 1986). The lower one-third of the anterior mandible, with attached genioglossus and digastric muscles, is wedged out between the mental foramina below the roots of the incisor teeth. Exposure is obtained via an incision in the gingivolabial sulcus. The wedge of bone is tapered so that the back edge is larger than the front. The bony wedge is pulled anteriorly so that the back of the tapered bone wedge lies on the anterior edge of the mandible, advancing the genial tubercle and attached muscles by the thickness of the mandibular body. The tapered resection prevents the bone wedge from slipping backwards; it is held in the new position with stainless steel wires. The operation can be combined with section of the hyoid depressor muscles (sternohyoid, thyrohyoid and omohyoid) on the lower aspect of the hyoid bone; hyoid suspension minimizes relapse.

**Hyoid expansion**

In some patients with obstructive sleep apnoea syndrome, inspiratory collapse of the hypopharyngeal lumen is observed; this is the likely mechanism of airway obstruction. Expansion hyoidoplasty was developed as an alternative to tracheostomy, the original work being carried out in dogs and with human cadaver studies (Patton, Ogura and Thawley, 1984). The hyoid is sectioned to give three portions, cutting the bone at the lesser cornu on each side. The three pieces are then attached to a curved piece of stainless steel, so that the side portions are pushed outwards, expanding the hypopharynx. The operation may prove useful in patients with symptomatic superior hypopharyngeal-base-of-tongue inspiratory collapse resistant to medical therapy.

**Hyoid suspension**

There are various materials available for suspension of the hyoid bone. Freeze-dried fascia has been used (R.G.) after cutting the hyoid on both sides at the lesser cornu and detaching the infrahyoid muscles. Two holes were drilled in the lower aspect of the anterior
mandible and the hyoid pulled up to within 1.5 cm of the lower border of the mandible using the fascial strips. Uvulopalatopharyngoplasty and tracheostomy were performed at the same time, the latter being reversed soon afterwards. The procedure is similar to laryngeal suspension performed as part of the reconstruction after base-of-tongue and supraglottic laryngeal resection (Goode, 1976). It may be combined with advancement of the genial tubercle (Riley et al, 1986).

**Base of tongue resection**

In many cases of obstructive sleep apnoea syndrome, a large tongue seems to be the cause of obstruction; limited tongue base resection has been developed in response to this observation; simultaneous uvulopalatopharyngoplasty and tracheostomy may be advocated. A tracheostomy may be reversed later if adequate clearance of the obstruction is obtained. There are two routes.

**Transoral**

This is the route of preference since it avoids an external incision and the problems that go with it; however, the very size of the tongue base itself may prevent adequate access. The portion for removal is usually between the foramen caecum and the valleculae, particularly in the midline. Horizontal wedge resection is currently under evaluation (R.G.) and this involves wedge removal from the middle portion of the tongue base. The wedge measures 2-3 cm in each plane and the wound is closed with 2-0 chromic catgut sutures. Care is taken not to transect the lingual vessels, or hypoglossal nerves which lie on the lateral aspect of the tongue base. In some cases, a vertical wedge excision may be preferable, but the vertical excision tends to narrow the hypopharynx at this level.

**External**

Obstruction to the transoral route indicates an external approach. A horizontal incision is made at the level of the hyoid and extended inwards, over the top of the hyoid bone, to reach the valleculae - similar to the approach used for laryngectomy.

A tracheostomy with cuffed tracheostomy tube is routinely performed with tongue-based resections, to avoid postoperative airway obstruction secondary to oedema and/or bleeding. Selected cases of obstructive sleep apnoea syndrome have demonstrable improvement following this procedure - but the indications for this operation are still not clear.

Further experience is needed with all of these listed procedures to enable a valid assessment of their place, if any, in the management of obstructive sleep apnoea syndrome. Effective surgical management, based on the site of obstruction, would seem to be a realistic goal. The authors have not yet achieved the diagnostic expertise that will allow the knowledge of whether nasal surgery, uvulopalatopharyngoplasty, tracheostomy, genial tubercle advancement, hyoid suspension, resection of the tongue base or some combination of these operations is the management of choice in idiopathic obstructive sleep apnoea syndrome. It is certain, however, that the modern otolaryngologist will continue to play a major role in both the assessment and treatment of patients with sleep-related breathing disorders.