Review Article

Anaesthetic management of patients with sleep apnoea syndrome

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Purpose: *Sleep apnoea syndrome (SAS) is a relatively common, potentially fatal, disorder. Patients with SAS exhibit repetitive, often prolonged episodes of apnoea during sleep, with serious nocturnal and diurnal physiologic derangements. Several anecdotal reports and clinical studies have documented anaesthetic-related occurrence of fatal and near-fatal respiratory complications in these patients. The purpose of this article is to outline the potential problems encountered in anaesthetic management of adult SAS patients, and to suggest a practical approach for anaesthesia both for incidental and specific procedures.*

Principal findings: *SASs have many implications for the anaesthetist. First, SAS patients are exquisitely sensitive to all central depressant drugs, with upper airway obstruction or respiratory arrest occurring even with minimal doses. Thus sedative and opioid premedication should be omitted as should the intra and postoperative use of opioids be limited or avoided. All anaesthetic drugs should be administered by titration to desired effect, preferably using short-acting drugs. When feasible, continuous regional anaesthesia using a catheter is the technique of choice. Where possible nonopioid analgesics or local anaesthetics should be used for postoperative analgesia. Perioperative monitoring for apnoea, desaturation, and dysrhythmias is essential. Secondly, SAS patients have a potentially difficult airway. Awake intubation is the safest approach to airway control. Extubation should only be tried in the fully conscious patient with intact upper airway function and under controlled situations. Thirdly, the cardiorespiratory complications of SAS and the presence of associated diseases can adversely influence anaesthetic management.*

Conclusion: *Perioperative risks attending SAS patients emphasize the importance of their detection, perioperative evaluation and planning.*

Objectif: *Le syndrome d'apnée du sommeil (SAS) est une affection relativement fréquente et potentiellement fatale. Ce syndrome est caractérisé par des périodes répétées et souvent prolongées d'apnée pendant le sommeil avec des dérèglements physiologiques nocturnes et diurnes graves. Plusieurs observations anecdotiques et cliniques ont rapporté, la survenue de complications fatales ou quasi fatales en rapport avec l'anesthésie de ces patients. Cet article vise à mettre en lumière les problèmes potentiels à envisager pour la gestion anesthésique des porteurs adultes du SAS et à suggérer une approche pratique pour l'anesthésie autant en vue d'une chirurgie en général que pour certaine interventions spécifiques.*

Constatations principales: *Le SAS a plusieurs répercussions sur l'anesthésie. D'abord, les patients souffrants de SAS sont extrêmement sensibles à tous les agents qui déprimment le SNC avec la conséquence que l'obstruction des voies respiratoires supérieures et l'arrêt respiratoire peuvent survenir même avec des doses infinitésimales. La prémédication aux sédatifs ou aux morphiniques doit être omise. Il faut aussi éviter ou au moins diminuer l'administration des morphiniques per- et postopératoires. Tous les anesthésiques doivent être administrés en titrant la posologie pour obtenir l'effet désiré préférablement avec des agents à courte durée d'action. Lorsque c'est possible, l'anesthésie régionale continue avec un cathéter est la technique de choix. De la même façon, des analgésiques non morphiniques devraient être utilisés pour l'analgésie postopératoire. Le monitorage de la respiration, de la saturation et du rythme cardiaque est essentiel. Deuxièmement, chez ces patients, l'accès aux voies aériennes peut être difficile. L'intubation à l'état vigile représente la meilleure méthode de contrôle des voies aériennes. L'extubation ne devrait être tentée que chez un patient com-*

Key words:
SLEEP: apnoea;
SLEEP APNOEA: surgery;
VENTILATION: sleep apnoea;
COMPLICATIONS: sleep apnoea, airway obstruction, postoperative hypoxaemia.

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plètement conscient dont le fonctionnement des voies aériennes est intact. Troisièmement, les complications cardio-respiratoires du SAS et les maladies associées peuvent influencer défavorablement la gestion de l'anesthésie.

Conclusion: *Les dangers périopératoires qui menacent le patient souffrant de SAS soulignent l'importance de leur détection, de l'évaluation périopératoire et de la planification.*

Over the past 25 yr, investigations of breathing during sleep have defined a group of patients, obese and nonobese, who develop a syndrome characterized by recurrent nocturnal apnoea and hypoxaemia during sleep.[1] The resulting apnoea-induced asphyxia engenders a host of pathophysiological consequences, primarily involving the cardiovascular and central nervous systems, which, in turn, yield a variety of well known clinical manifestations. These include pulmonary and systemic hypertension, right and left heart failure, polycythaemia, daytime hypersomnolence, and sometimes respiratory failure. This syndrome poses considerable problems to the anaesthetist and a rational plan for anaesthesia, based on understanding of its pathogenesis and pathophysiology, should be sought.

Normal sleep

In adults, a total night's sleep usually consists of 4 to 6 cycles. Each cycle includes both a quiet or non-rapid eye movement (NREM) sleep, and active or rapid eye movement (REM) sleep. There are four stages of NREM sleep which represent progressively deeper sleep, with progressive slowing of the electroencephalogram (EEG) waves. Stages 3 and 4 NREM are often combined, as they differ only in the proportion of slow waves, and are collectively called slow wave or deep sleep, the most restorative sleep period. REM sleep occurs after NREM sleep has been established. It is characterized by a generalized loss of muscle tone as evidenced by electromyography (EMG). However, the extraocular muscles are not paralyzed, and intermittent conjugate REMs can be detected by electrooculography (EOG). In the postoperative setting, this sleep architecture is disturbed. Deep sleep and REM sleep are often suppressed in early nights following surgery. However, a catching-up phase ensues four to five days later resulting in excessive deep sleep and REM sleep rebound.[2]

Upper airway muscle function and ventilatory control in wakefulness and sleep

Normally, during inspiration, the diaphragm contracts against the high resistance offered by the nose. This creates a subatmospheric intra-airway pressure which tends to narrow the upper airway (UAW) collapsible segments in the oro and hypopharynx. However, in the awake subject, there is phasic inspiratory activation of UAW dilator muscles slightly before diaphragmatic contraction, splinting UAW sufficiently to prevent its collapse.[3] These muscles include those which act on the soft palate (e.g., tensor palati), tongue (e.g., genioglossus) and hyoid (e.g., geniohyoid). During sleep, the rhythmic activity of these muscles wanes in NREM sleep and virtually disappears during REM sleep.[4] Moreover, upper airway resistance (UAR) increases and, during NREM sleep, it can be twice that in wakefulness.[5] In REM sleep, UAR is even higher because of the generalized loss of UAW muscle tone. Thus, the reduction of UAW muscle activity and generation of more subatmospheric inspiratory pressure secondary to the increase in UAR set the stage for upper airway obstruction (UAO) during sleep.

In addition, sleep causes respiratory depression in normal adults.[6] The normal increase in ventilation seen with hypoxia or hypercapnia during wakefulness is blunted during sleep, particularly during REM sleep. These sleep-related alterations in ventilatory control and UAW muscle function may be responsible for the brief non-repetitive apnoeas commonly found in normal asymptomatic subjects at sleep onset and during REM sleep.

Polysomnography

Patients with a suspected breathing sleep disorder can be evaluated by polysomnography (PSG), an overnight study of both sleep and respiration in a sleep laboratory. This involves continuous polygraphic monitoring of sleep stages, nasal-oral airflow, respiratory effort, oxygen saturation, and continuous electrocardiography (ECG). Recording of airflow at the nose and mouth can be accomplished by thermistors mounted in or near the nose and in front of the teeth. Chest and abdominal wall movements are usually detected noninvasively by use of a strain gauge or by respiratory inductance plethysmography. Alternatively, intrathoracic pressure swings in response to diaphragmatic respiratory efforts can be detected and measured by an intraoesophageal balloon. Arterial oxygen saturation (SaO_2) can be estimated by the use of ear or pulse oximetry. Cardiac dysrhythmias during apnoeic episodes are monitored by ECG. All these recordings are then related to phase of sleep by continuous EEG, EOG, and EMG using surface electrodes taped around the scalp, on either side of the eyes and under the chin, respectively.

Based on PSG information the following definition of terms is applied:

Apnoea

A cessation of nasal-oral airflow lasting 10 sec or more,

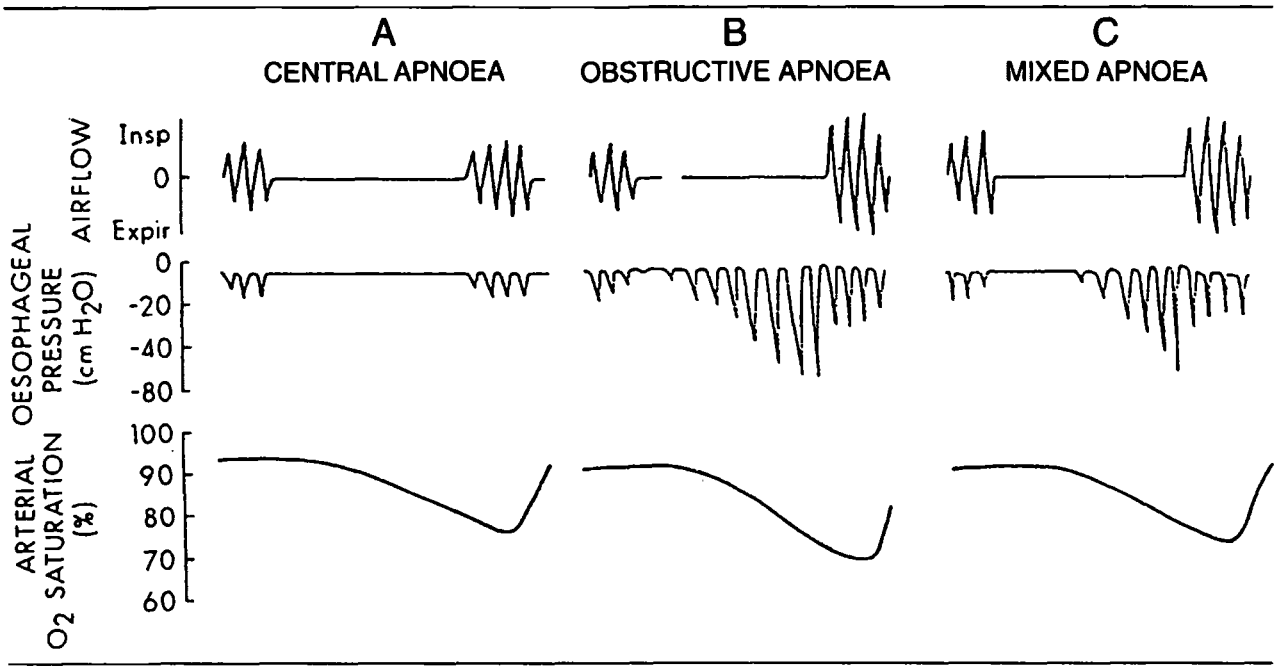

FIGURE 1 Diagrammatic representation of the 3 patterns of apnoea. Reproduced from: *Orr WC.* Utilization of polysomnography in the assessment of sleep disorders. Med Clin North Am 1985; 69: 1153–67, with permission.

accompanied by oxygen desaturation of at least 4% from the baseline value. Apnoea may be central, obstructive, or mixed (Figure 1): *Central* airflow cessation is accompanied by cessation of respiratory efforts; *Obstructive* airflow ceases despite continuation of respiratory efforts; *Mixed*, an initial central apnoea component is followed by an obstructive event.

Hypopnoea
An incomplete cessation of airflow lasting 10 sec or more associated with a 50% or more reduction in tidal volume.

Apnoea hypopnoea index (AHI)
The number of abnormal respiratory events (apnoea + hypopnoea) per hour of sleep.

Lowest saturation (LSAT)
The lowest oxyhaemoglobin saturation associated with an abnormal respiratory event.

Sleep apnoea syndrome (SAS)
The presence of an AHI ≥ 5 or AHI ≥ 10 (the normal upper limit of apnoea and/or hypopnoea is controversial) plus symptoms or signs of impairment from sleep apnoea e.g., daytime hypersomnolence, cor pulmonale or polycythaemia.

Some patients may exhibit periodic hypopnoea instead of apnoea; the so-called *sleep hypopnoea syndrome*[7] which is indistinguishable from SAS. In both syndromes loud snoring has generally been regarded as evidence of reduced airflow. However, snoring is also common in asymptomatic persons who do not exhibit sleep apnoea or hypopnoea. Therefore, asymptomatic snoring may represent a mild degree of reduced airflow, whereas hypopnoea and apnoea represent greater degrees of reduced airflow. The term sleep-disordered breathing (SDB) may be used to indicate any degree of sleep-induced airflow limitation i.e., snoring, hypopnoea or apnoea.

Epidemiology of SAS

Sleep apnoea syndrome is a common disorder; the estimated prevalence in middle-aged adult population is in the order of 1–4%.[8–10] Males predominate among patients with SAS, and severe disease would be expected in 0.3–0.5% of men.[9,11,12] Recent data, however, indicate that the prevalence of SAS in women is much higher than previously suspected.[10,13] Most affected women are postmenopausal or have an aggravating factor such as morbid obesity or structural UAW abnormality. Obesity, particularly if affecting the neck, is a risk factor for SDB and SAS.[11] The syndrome is extremely frequent in the elderly, affecting at least 18%,[14] but its clinical importance is not known. Moreover, several clinical and epidemiological studies have shown that SDB and SAS occur frequently in patients with essential hypertension,[15] congestive heart failure (CHF)[16] and diabetes

TABLE I Conditions associated with sleep apnoea*

Obstructive sleep apnoea	*Central sleep apnoea*[33]
Altered upper airway anatomy – Due to localized pathology: e.g., nasal obstruction,[19] tonsillar hypertrophy,[20] nasopharyngeal carcinoma,[21] mandibular hypoplasia,[22] laryngeal abnormalities[23] – As a part of a systemic disorder: e.g., obesity,[24,25] hypothyroidism,[26] acromegaly[27]	*Hypercapnic* – Neurologic disorders: e.g., brain stem tumours or infarcts, bulbar poliomyelitis, bilateral cervical cordotomy, encephalitis – Respiratory muscular disorders: e.g., myasthenia gravis, myotonic dystrophy, diaphragmatic paralysis
Altered upper airway function e.g., alcohol ingestion,[28] benzodiazepines,[29] diabetes mellitus,[17] chronic renal failure[30]	*Nonhypercapnic* Conditions associated with Cheyne-Stokes respiration, e.g., congestive heart failure, chronic renal failure, brain lesions
Others e.g., systemic hypertension,[13,15,31] chronic obstructive pulmonary disease (COPD)[32]	

*OSA is commonly seen in association with anatomical or functional abnormalities of the upper airway. CSA is often seen in disorders manifesting either hypoventilation (hypercapnic CSA) or hyperventilation as a component of periodic breathing (nonhypercapnic CSA).

mellitus.[17] Sleep apnoea syndrome may have strong familial tendency[18] that may be caused by peculiarities in structure of facial skeleton or UAW soft tissues.

Aetiology and pathogenesis of SAS

Patients with SAS may have an underlying disorder that is partially or completely responsible for their apnoea (Table I). In most patients, however, none of these abnormalities exist, and the aetiology of SAS is unknown. In such patients several studies provide important insights into the mechanism of their apnoea during sleep. Obstructive and mixed apnoeas predominate among patients with SAS.[1] However, it is common to record the three types of apnoea in the same patient, implying some commonality in pathogenesis of the various types of apnoea.

Obstructive sleep apnoea (OSA)

In patients with OSA syndrome (OSAS), both functional and anatomical abnormalities may be implicated in the pathogenesis of their disorder.

FUNCTIONAL FACTORS

In patients with OSAS, the sleep related reduction in tonic and phasic UAW muscle activity may be excessive even during NREM sleep. As the compliance of UAW tissues depends on *tonic* UAW muscle activity, the pharynx of patients with OSAS becomes excessively floppy and easily collapsible during sleep.[34] The intrinsic compliance of the pharynx is also increased as demonstrated during wakefulness by measurement of the change in pharyngeal area for a given change in pressure,[35] or with changes in lung volume.[36] In this latter study,[36] lung volume has been shown to influence pharyngeal calibre, the so-called pharyngeal/lung volume dependence (PLVD). This PLVD is exaggerated in OSAS patients who showed a greater decrease in pharyngeal cross-sectional area during an active expiration from functional residual capacity to residual volume than control subjects. On the other hand, as contraction of the diaphragm changes little during sleep, the decrease in *phasic* inspiratory activity of UAW muscles shifts the net balance of forces which determine the pharyngeal patency so that the inward narrowing force predominates, favouring UAO.[37] In addition, the increase that occurs in UAR in patients with OSAS is three- to fourfold that seen in normal subjects. Such high UAR is also demonstrable during wakefulness[38] and would be expected to result in the generation of more subatmospheric pharyngeal pressures during inspiration.

ANATOMICAL FACTORS

Only a minority of OSAS patients exhibit visible anatomical narrowing of the UAW that is evident on examination of the pharynx, nose, or larynx (Table I). However, several investigators using special techniques have documented the presence of subtle UAW abnormalities in patients with typical OSAS who present no specific lesions on routine examination. The most frequent abnormality is oropharyngeal narrowing which has been demonstrated by standard computerized tomographic (CT) scanning of the UAW[39] and by acoustic reflection measurements of pharyngeal area.[36,40] Likewise, ultrafast CT scanning, which permits detection of real time changes in the UAW cross-sectional area, demonstrated smaller oropharyngeal and nasopharyngeal airways in patients with OSAS compared with control subjects.[41] Cephalometric *x*-ray analyses[40,42] have also shown that the posterior airway space is abnormally small in many OSAS patients. The mandible is frequently small and/or retropositioned, with the hyoid bone in a lower position than normal.

Central sleep apnoea (CSA)

Central sleep apnoea arises as a result of transient withdrawal or inhibition of central drive to the respiratory muscles.[33] It may have no identifiable cause (primary central alveolar hypoventilation and idiopathic CSA), or be attributable to an underlying condition (Table I).

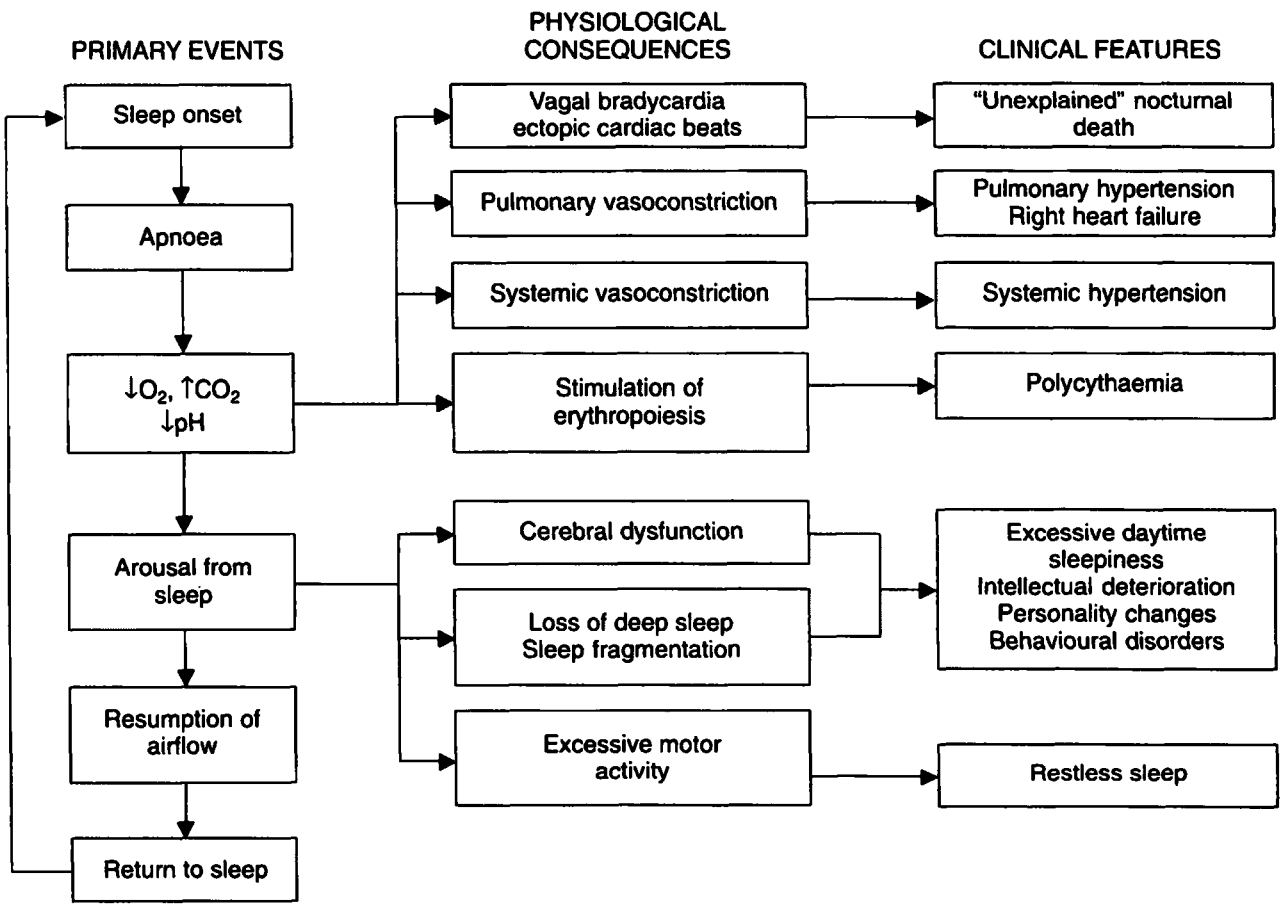

FIGURE 2 The primary sequence of events resulting in obstructive sleep apnoeas, the resulting physiologic responses and clinical features. Reproduced from: *Bradley TD, Phillipson EA.* Pathogenesis and pathophysiology of the obstructive sleep apnea syndrome. Med Clin North Am 1985; 69: 1169–85, with permission.

Pathophysiological consequences

The systemic effects of sleep apnoea, whether of central or obstructive aetiology, are essentially similar. Figure 2 illustrates the physiological responses to the recurrent asphyxial episodes and recurrent arousals during sleep in OSAS patients. During apnoea, especially if prolonged, the SaO_2 decreases dramatically and this is accompanied by changes in heart rate and rhythm, and by rapid increase in pulmonary and systemic arterial pressures. Bradycardia during obstructive apnoea followed by heart rate acceleration upon resumption of ventilation is characteristic. In addition, prolonged sinus pauses, second degree heart block and ventricular dysrhythmias have been documented in association with the apnoeic events.[43] The severity of bradycardia and increase in ventricular ectopy have been observed when SaO_2 decreases below 60%.[44] The elevations in pulmonary and systemic pressures are also related to the severity of the arterial hypoxaemia, maximal elevations being observed with the nadir of SaO_2 during REM sleep.[45] Pulmonary hypertension may be sustained in daytime with eventual development of right ventricular (RV) failure that may be accompanied by polycythaemia. Daytime hypoxaemia attributable to the combined effects of associated obesity and COPD appears to be a necessary prerequisite to the development of such complications.[46] Obesity and COPD are also implicated in the pathogenesis of hypercapnic respiratory failure in certain patients with severe OSAS.[47] These obese hypercapnic OSAS patients should be distinguished from patients with obesity hypoventilation syndrome who have abnormal ventilatory responses with awake hypercapnia being secondary to chronic alveolar hypoventilation, independent from apnoea or COPD.[48] The term "Pickwickian" is currently often used to describe both forms of obesity-associated hypercap-

TABLE II Clinical features of sleep apnoea syndromes

Symptoms	*Signs*
Nocturnal events	Reduced sleep latency by EEG
Snoring	Obesity
Restless sleep with frequent movements	Hypertension
Sudden arousals with choking or shortness of breath	Cardiac dysrhythmias
Nocturnal awakenings and insomnia	Upper airway abnormalities
Nocturnal enuresis or frequent nocturia	Pulmonary hypertension, RV hypertrophy on ECG, cardiomegaly
Nocturnal sweating	Peripheral oedema
Diurnal history	Polycythaemia
Excessive daytime sleepiness	
Personality changes	
Morning headaches	
Morning dry throat	
Sexual dysfunction	

nic respiratory insufficiency. Pickwickian patients are major anaesthetic hazards as they often combine extreme obesity with both cardiac and respiratory failure, with high perioperative mortality.[25]

More commonly, systemic hypertension persists during wakefulness, accounting for high (50%) incidence of hypertension among OSAS patients.[49] Recent evidence also implicates OSA in the pathogenesis of left ventricular (LV) failure,[50] possibly through increased LV afterload related to exaggerated negative intrathoracic pressure swings and intermittent hypoxia during OSA.

Diagnosis of SAS

The diagnosis of SAS can be suggested from patient's history and physical examination (Table II and Figure 2) and confirmed by PSG. However, because of PSG's inherent logistical complexity, expense and relative inaccessibility, clinical evaluation may assume a major role in the diagnosis in many instances.[51] Snoring is the hallmark of SAS and is noted in all patients. Although not all snorers have sleep apnoea, heavy snorers are more likely to be hypertensive[52] and to develop angina pectoris and stroke[53] compared with nonsnorers. The pharyngeal compliance of snorers is higher than in normal subjects[34,35] and they may have common craniofacial characteristics as SAS patients.[54] Further, asymptomatic snorers may even develop apnoea after weight gain[55] or alcohol ingestion.[28] Taken together, the preceding observations emphasize the concept that snoring represents an intermediate stage between complete UAW normality and evident UAO, and that even a mild degree of SDB can have adverse health effects.

Excessive daytime sleepiness (EDS) is the commonest presenting complaint in OSAS patients. Patients with mild sleepiness may have morning tiredness as their only complaint, but frequently they fall asleep whenever sedentary. Patients with more severe EDS are always sleepy, but can remain awake with effort. Extreme EDS may be expressed as "sleep attacks" in entirely inappropriate situations such as while driving or talking.

For anaesthetists, a history of EDS coupled with reports of heavy snoring and anecdotal respiratory pauses from the sleeping partner is strongly suggestive of SAS. It should be the custom to enquire specifically about these features during the preanaesthetic visit,[56] particularly among patients with risk factors, cardiovascular disease or diabetes. A simplified diagnostic investigation, combining overnight oximetry and monitoring of respiratory movement, seems to give sufficient information to confirm or negate a diagnosis of OSAS in the majority of patients with clinical symptoms.[57,58] Formal PSG is required in those patients in whom the diagnosis remains in doubt.

Treatment of SAS

All SAS patients must avoid alcohol, sedatives, sleep deprivation and supine sleep posture, which may induce or worsen the symptoms. Weight reduction is extremely important in obese patients and can lead to complete resolution of the symptoms.[53] Any underlying treatable disorder should be controlled e.g., medical treatment of CHF, or surgical excision or correction of UAW obstructive lesions (Table III).

Treatment of OSAS

For those patients without a correctable associated disease, several therapeutic options have been developed over the past 15 yr. In many instances, however, these therapeutic choices are limited by the local medical environment. Currently, the application of continuous positive airway pressure through the nose (nCPAP) is

TABLE III Surgical procedures for obstructive upper airway pathology in patients with OSAS

Location of obstruction	*Surgical procedure*
Nose	– Nasal polypectomy
	– Septoplasty
	– Tumour excision
Nasopharynx	– Tumour excision
Oropharynx	– Tonsillectomy
Hypopharynx	– Lingual tonsillectomy
	– Vallecular cyst excision
	– Partial epiglottectomy
	– Mandibular advancement

TABLE IV Currently available site-specific upper airway procedures for idiopathic OSAS

Site of obstruction	*Proposed procedure*
Oropharynx	– Uvulopalatopharyngoplasty (UPPP),[62] resection of the uvula, free edge of the soft palate and the tonsils, in addition to stretching of posterior pharyngeal wall mucosa to remove excessive folds.
Hypopharynx	– Lingual reduction surgery, either midline glossectomy,[63] in which resection involves only a midline portion of the tongue base; or lingualplasty,[64] which involves more extensive posterior and lateral excision of the tongue base. Redundant hypopharyngeal tissues are also resected.
	– Mandibular advancement surgery, either total advancement[65] using a bilateral bony cuts posterior to the last molars; or a more isolated geniotubercle advancement[66] through an inferior sagittal osteotomy, combined with hyoid myotomy-suspension.
	– Maxillo-mandibular advancement (MMA),[67] advancement of the maxilla (using Le Fort I osteotomy), the mandible (using sagittal split osteotomy), and hyoid with its muscular attachments (hyoid myotomy-suspension).

the treatment of choice for most patients with idiopathic OSAS. By acting as a pneumatic splint preventing airway collapse, nCPAP markedly improves or abolishes OSA and its complications,[59] with a long-term acceptance rate of 60–80%. Blowers capable of generating pressures from 2.5 to 20 cm H_2O through a snugly fitting plastic nasal mask are commercially available for home use. Pressures of 5–10 cm H_2O are adequate in most patients, although obese patients may require higher pressures in the range of 15–20 cm H_2O.

Drug therapy of OSAS is of limited value. It may be indicated in some patients who satisfy specific criteria. For example, obese patients with hypercapnic respiratory failure, RV failure and polycythaemia (i.e., Pickwickians) benefit more from progesterone therapy than do the more common obese OSAS patients without awake hypercapnia.[60] Protriptyline,[61] a nonsedating tricyclic antidepressant may be effective in some patients with mild to moderate OSAS in whom hypersomnolence is troublesome and nCPAP is not an option. The mechanism of improvement may be related to its suppressant effect on REM sleep and an augmentation of UAW muscle activity. Anticholinergic side effects are frequent and may limit the usefulness of this agent in many patients.

Several surgical procedures are currently performed in patients with idiopathic OSAS (Table IV), particularly when nCPAP is ineffective, not available or poorly tolerated. As the oropharynx is the major site of airway compromise in most OSAS patients, UPPP is currently the most commonly used procedure. Procedures which address hypopharyngeal obstruction are often pursued by centres committed to total surgical management of OSAS. Patients with obstruction involving both oro- and hypopharynx will often need staging procedures to address the obstruction at these levels.

Tracheostomy is still probably the most effective surgical procedure, with both immediate and sustained improvement.[68] However, because of its inherent invasiveness, this procedure is reserved for patients in whom other approaches have failed or as an emergency measure in those patients with life-threatening cardiac dysrhythmias, persistent hypertension or cardiac failure.

Treatment of CSA syndrome (CSAS)

In hypercapnic patients, respiratory stimulants such as theophylline, acetazolamide and progesterone may be tried. However, most patients with severe disease often require mechanical intermittent positive pressure ventilation during sleep through a tight-fitting nasal mask[69] or tracheostomy.[70] Patients with idiopathic CSA have been effectively treated by nCPAP.[71]

Effect of general anaesthesia on the upper airway muscles of healthy patients

As already stated, during wakefulness, the UAW muscles have tonic activity with phasic bursts preceding inspiration which serve to overcome the tendency of inspiratory subatmospheric pressure to collapse the pharynx. Recently, it has become appreciated that anaesthetic agents tend to reduce the UAW muscle tone to a greater degree than diaphragmatic tone. This produces an unfavourable balance for maintaining a patent pharyngeal airway and offers an explanation for the obstructive apnoea-promoting properties of these drugs commonly observed perioperatively in healthy patients. This tendency to UAO induced by anaesthetic drugs

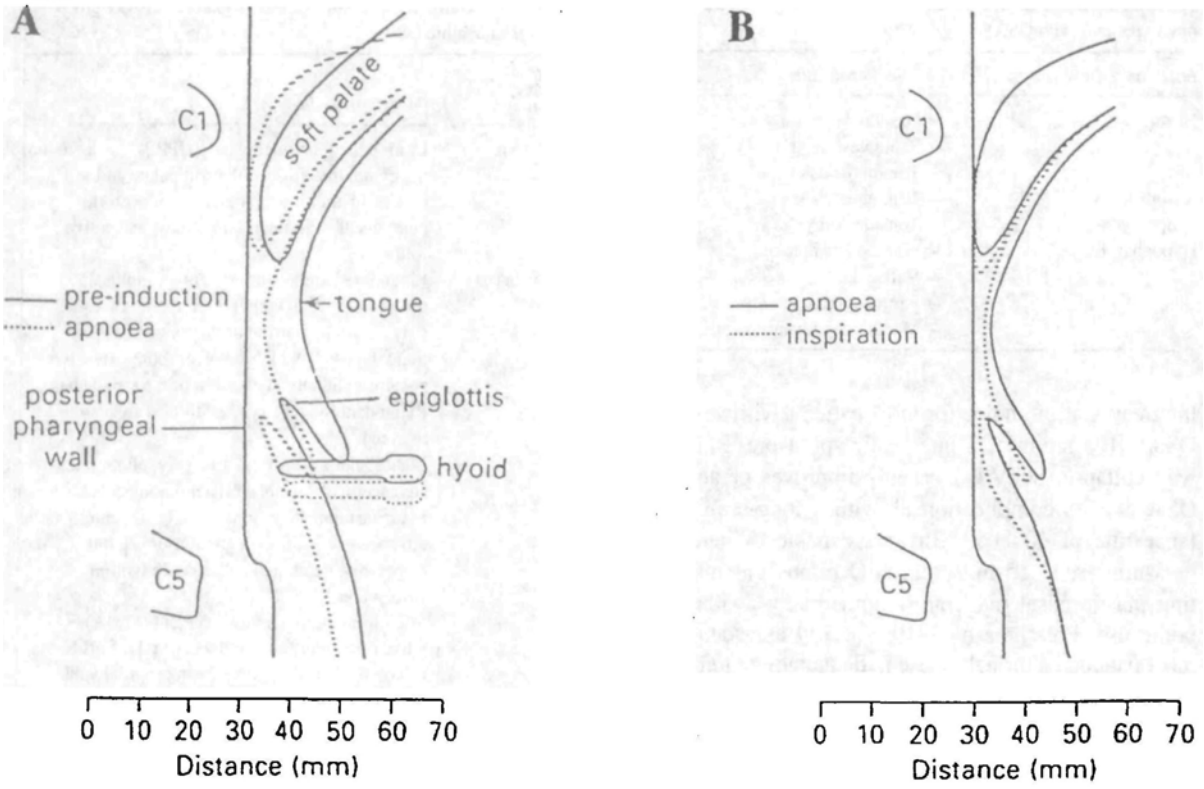

FIGURE 3 Changes in pharyngeal outline in the sagittal plane. (A) during the apnoeic pause immediately following thiopentone induction. There are highly significant approximations to the posterior pharyngeal wall of the soft palate, tongue base and epiglottis, (B) after re-establishment of spontaneous breathing under N_2O/O_2-enflurane anaesthesia. There is extensive collapse of the pharynx with significant reduction in most of its dimensions. Reproduced from reference [79], with permission.

would be expected to be enhanced in OSAS patients who already have narrower and readily collapsible UAW.

General anaesthetics and sedatives

Several animal studies indicated that anaesthetic agents, both intravenous[72] and inhalational,[72,73] and benzodiazepines[72] cause greater depression of the activity of the UAW muscles than of the diaphragm. In normal human subjects, diazepam causes a selective decrease in genioglossal activity relative to that of the diaphragm favouring inspiratory UAO.[74] Another study[75] in healthy subjects demonstrated that midazolam in sedative doses markedly increased supraglottic UAR and induced central followed by obstructive apnoeic events. The authors[75] suggested that the observed increase in UAR resulted from diminished UAW muscle tone. Recently, central and obstructive apnoea has also been reported in normal subjects breathing N_2O,[76] although the effect of this agent on UAW muscles has not been explored.

The site of UAO in anaesthetized normal humans has been investigated. Using a flexible bronchoscope, Boidin[77] explored the UAW in spontaneously breathing patients under deep halothane anaesthesia. He demonstrated that UAO was associated most consistently with contact between the epiglottis and the posterior pharyngeal wall, and that the motion of the epiglottis and hyoid are closely associated, and controlled by the neck strap muscles. Drummond[78] made similar observations, using surface EMGs of tongue and neck muscles. Upper airway obstruction during thiopentone induction was associated with loss of tonic activity in the neck strap muscles with consequent dorsal displacement of the hyoid. A more recent radiographic study,[79] however, implicated the soft palate as the major site of UAO in anaesthetized patients (Figure 3A). Secondary collapse of the pharynx with multiple sites of obstruction occurred when the patients attempted inspiration (Figure 3B), similar to what has been observed in OSAS.

Opioid analgesics

There are no direct studies of the effect of opioid analgesics on UAW muscles. However, the study by Catley *et al.*[80] demonstrated that *iv* morphine infusion in healthy patients can result in episodic obstructive apnoeas with pronounced oxygen desaturation. They suggested that opioids preferentially depress UAW muscle activity in a way similar to the effect of sleep. However, it appears that both dose and route of administration of opioids are important for alteration of UAW muscle function. Robinson *et al.*[81] demonstrated a lack of selective depression of UAW muscle function by small doses of oral opioids administered to healthy adults.

Neuromuscular blockers

Several studies have shown that UAW muscles are much more sensitive to non-depolarizing neuromuscular blocking drugs (NMBDs) than are those of ventilation. It has been shown that small doses of non-depolarizing NMBDs, as might be given for defasciculation[82] or priming[83] may produce UAO. Profound UAW muscle weakness has also been demonstrated when recovering from d-tubocurarine induced paralysis, despite adequate ventilation.[84] The potentially serious development of undetected postoperative UAO has prompted several recent editorials,[85–87] emphasizing the need for reliable criteria for assessing the adequacy of UAW neuromuscular function postoperatively.

Anaesthetic management of SAS patients

A rational approach to anaesthetic management of patients with SAS requires a clear understanding of the pathogenesis and pathophysiological consequences of the syndrome. Of primary importance is the extreme vulnerability of these patients to UAO and/or respiratory depression following administration of even minimal doses of any central depressant drug. This coupled with the well-known difficulty in managing the airway, can easily account for catastrophic perioperative complications. It can be seen that maintenance of adequate oxygenation and prevention of hypoxaemia and its consequences are the major concerns perioperatively.

The preoperative phase

In patients with known SAS, the preoperative evaluation should address the following aspects:

ASSOCIATED MEDICAL DISEASES

The anaesthetist needs to be aware of their presence as they may influence the anaesthetist's choice of drugs and techniques. Specific investigations and treatments should be ordered as appropriate to optimize patient's condition before surgery.

CARDIORESPIRATORY COMPLICATIONS

A thorough respiratory and cardiovascular assessment is essential to identify such complications and assume adequate preoperative control. Relevant objective studies should be ordered. The ECG may show evidence of RV hypertrophy, LV hypertrophy or associated ischaemic heart disease. Chest radiography may reveal cardiomegaly, enlargement of pulmonary arteries or evidence of associated COPD. Echocardiography may further confirm RV hypertrophy[88] and other cardiac abnormalities.

Pulmonary function tests are often normal in SAS patients, but may show a restrictive pattern in morbidly obese patients or obstructive pattern in patients with COPD. Arterial blood gases (ABGs) are normal in most SAS patients, but a degree of hypoxaemia is common in many obese patients. By noting $PaCO_2$ and bicarbonate values the subset of SAS patients with chronic hypercapnia (Pickwickians, hypercapnic CSAS) can be identified. These patients have diminished sensitivity to CO_2 and are likely to need mechanical ventilation when their hypoxic drive to breathing is abolished by subanaesthetic concentrations of inhalational anaesthetics postoperatively.[89]

Before operation, patients with persistent uncontrollable hypertension and/or cardiorespiratory failure should be treated by nCPAP. Minitracheostomy has been successfully used preoperatively as alternative to tracheostomy for such purpose.[90] Polycythaemic patients may require preoperative phlebotomy if the haematocrit exceeds 55%, because of the hazard of thromboembolic phenomena.

THE SEVERITY OF SAS

The anaesthetist needs to have some clue as to the severity of SAS, as this may influence the perioperative management. Severe SAS may be indicated by such clinical findings as RV failure or incapacitating hypersomnolence and/or by PSG findings e.g., severe nocturnal hypoxaemia or serious nocturnal dysrhythmias. In one retrospective study,[91] the severity of OSAS as measured by either the AHI or the LSAT has been shown to be the most significant risk factor predictive for development of a perioperative airway complication (failed intubation, postextubation UAO). Using relative operating characteristic plots (Figure 4) to determine accuracy of these variables in predicting airway risk indicated that neither parameter is highly accurate predictor. However, this type of analysis provided useful information: "caution limits" were proposed at an AHI ≥ 70 and LSAT $<80\%$ as indicators for significant airway risk.

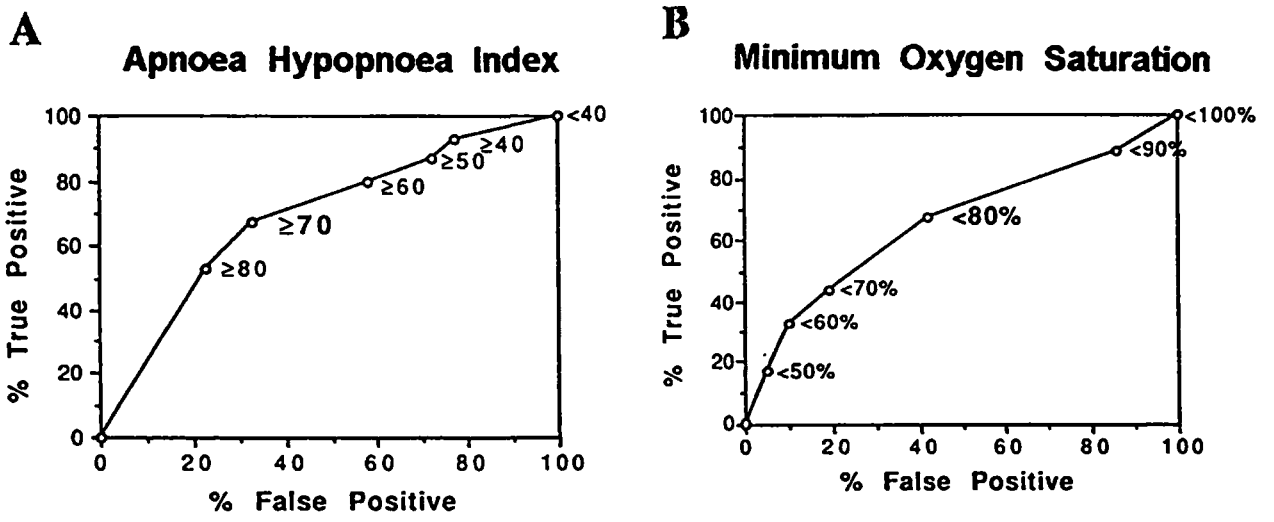

FIGURE 4 Relative operating characteristic curves for AHI (A) and LSAT (B). Caution limits are proposed at AHI ≥ 70 and LSAT <80% respectively, levels which yield maximum combined sensitivity and specificity of the stated measure. Changing the caution limits to AHI ≥ 40 and LSAT <90% increases the true positive rate (i.e., increases sensitivity) but at the obvious cost of increasing the false positive rate. Conversely changing these limits to AHI ≥ 80 or LSAT <60% will result in low false positive rate but is not adequately sensitive. Reproduced from reference [91], with permission.

EVALUATION OF THE UPPER AIRWAY

The pathological, functional, and/or anatomical factors implicated in the pathogenesis of OSA can lead to difficulty with airway management.[22,56,91,92] The UAW should, therefore, be systematically examined using simple bedside tests.[93] The patient should be questioned regarding previous anaesthetic-related UAO or intubation difficulty; and any previous anaesthetic records should be examined. In OSAS patients who have cephalometric studies performed as part of their surgical workup, it may be useful to examine the radiographs looking for such predictors of difficult intubation as maxillary-mandibular discrepancy or a long mandibulohyoid distance.[94]

EVALUATION OF OBESITY AND ITS DISTRIBUTION

The presence and extent of obesity should be determined as this may necessitate special transport and operating room preparations.[25] The distribution of excess adipose tissue at certain locations may impose several physiologic derangements and technical difficulties in anaesthetic management, and this should also be examined.

PHARMACOLOGICAL CONSIDERATIONS

(a) *The current drug therapy.* OSAS patients may be taking tricyclic antidepressants, their chronic use may deplete noradrenaline from nerve endings which become hypersensitive to catecholamines and other directly acting sympathomimetics.[95] This can present perioperatively as blood pressure liability and/or dysrhythmias. The anticholinergic activity of tricyclics may summate with other atropine-like drugs and lead to tachycardia or atrial ectopic beats.

(b) *Preoperative medication.* Patients with SAS may be very sensitive to all central depressants. Respiratory arrest,[96] UAO,[24,97] coma,[97] and even death[98] have been reported after administration of sedatives or opioids in such patients. Therefore, sedative and opioid premedication should be avoided[56,57,99] particularly in patients with severe SAS. A well-conducted preoperative visit, with psychological support and careful explanation, is of utmost importance.[93,99] An anticholinergic agent should be added if awake intubation is planned. Steroids (dexamethazone) may be indicated to diminish UAW oedema following UPPP[100] or multiple attempts at intubation. Histamine (H_2) blockers and antacids may be considered in patients with high aspiration risk[99]: the morbidly obese, anticipated difficult intubation and patients with gastro-oesophageal reflux.[101]

The intraoperative phase

Given the detrimental effects of general anaesthesia (GA) on the UAW and the central role of depressed consciousness in the pathogenesis of sleep apnoea, the accentuation of sleep apnoea by GA is understandable. Thus, preservation of consciousness is considered often to be an advantage in SAS patients. Indeed, regional anaesthesia (RA) has been recommended as the best technique where possible.[96,99,102,103] However, GA may

be inevitable for many procedures. General anaesthesia may also be combined with RA either for surgery itself or to provide postoperative analgesia.

REGIONAL ANAESTHESIA

In SAS patients it is best to rely entirely on RA, with the patient retaining his conscious control over respiratory system and UAW. The use of a continuous catheter technique is preferable to single injection. This allows for slow careful titration of the local anaesthetic to desired effect so that any major complication produced by the block can be avoided, a particular concern in a patient with a known difficult airway. The use of a catheter also allows for "top-ups" intraoperatively whenever surgery is prolonged, and postoperatively for analgesia. The classic example of continuous catheter technique is the continuous epidural, but catheters have also been used to provide continuous subarachnoid block[104] and plexus blockade of upper[105] and lower[106] limb surgery. Sedative supplementation should only be met sparingly, with all equipment of airway management readily available. Sedation with the often supine position with regional techniques might induce or worsen apnoeas in SAS patients. These patients, particularly those with known difficult airway or cardiorespiratory complications, should, therefore, be monitored during RA in the same intensive and vigilant fashion as if receiving GA. Alternatively, the anaesthetist may elect to use a combined technique as indicated in the next section.

COMBINED RA AND GA

The addition of a sequence of light general anaesthesia (with elective tracheal intubation and controlled ventilation) to regional block (often a continuous epidural) may confer several advantages in SAS patients. These include guaranteed control of the airway, decreased requirements for GA (opioids, inhalational agents), excellent muscle relaxation, and, not least, prolonged postoperative pain relief without central depression or UAW compromise.

GENERAL ANAESTHESIA

In OSAS patients, the unique potential combination of extreme vulnerability to UAO following even minimal sedation, difficulty with tracheal intubation and high incidence of gastro-oesophageal reflux,[101] presents the anaesthetist with a truly difficult airway for management.[107] Therefore, if GA is mandatory, a technique that ensures airway maintenance is necessary.

Induction and airway management

If the preoperative airway assessment does not indicate a difficult problem, a rapid induction-intubation sequence may be feasible. Induction is most appropriately accomplished by the *iv* route, using a short acting barbiturate or propofol.[57,99] Being the most rapid in action, succinylcholine remains the NMBD of choice to facilitate tracheal intubation. In the event of failed intubation, its short action permits early return of spontaneous ventilation. Adequate preoxygenation and cricoid pressure are vital. If UAO develops during induction, attempts should be made to relieve the obstruction by head-jaw positioning[79,108] and/or use of oropharyngeal and/or nasopharyngeal airways.[22] Combining these manoeuvres with the application of CPAP using a suitable anaesthetic breathing system relieves obstruction in most cases.[57,99] Moreover this manoeuvre may entail oxygen insufflation in the pharynx which consequently delays the onset of arterial oxygen desaturation[109] and allows for further intubation attempts. However, it is not possible to predict reliably which patient will develop UAO or whose trachea will be difficult to intubate. Therefore, awake intubation may be the safest approach to gaining airway control in OSAS patients, even if airway assessment is apparently benign. Topical anaesthesia of the UAW with or without supplementation by UAW nerve blocks is crucial to the success of awake intubation.[93] However, as nasal[110] and pharyngeal[111] local anaesthesia may cause UAO, these drugs should be used with caution.

Selection of GA technique

Based on the recovery pattern, techniques that ensure prompt recovery of consciousness and UAW integrity at the end of anaesthesia should be sought. In this respect, a relaxant-based technique with tracheal intubation and controlled ventilation is most satisfactory, particularly for prolonged procedures. Controlled ventilation is often mandatory in patients with morbid obesity, awake hypercapnia or CSAS who cannot maintain satisfactory spontaneous breathing under anaesthesia. In all SAS patients, this technique permits surgery to be done under light planes of anaesthesia with excellent muscular relaxation, maintenance of satisfactory levels of ventilation and ABG tensions and prompt recovery after reversal of residual action of NMBDs. However, further clinical investigations will be necessary to support the validity of these assumptions in SAS patients.

NMBDs

The competitive NMBDs of intermediate duration e.g., atracurium and vecuronium are preferable to longer acting relaxants. Both atracurium and vecuronium show excellent tendency to spontaneous recovery of neuromuscular function, thus their reversal is predictable. For

lengthy surgery, these drugs can be administered either by incremental doses or by infusion. An anticholinesterase should always be used to antagonize the block, as small degrees of residual weakness may precipitate UAO.[86]

OPIOID ANALGESICS

The use of intraoperative opioid supplementation should be limited and short-acting opioids used if they are deemed necessary. One study[91] noted that the amount of opioid administered intraoperatively correlated with extubation problems. Postextubation UAO occurred at a fentanyl dose of 2.9 $\mu g \cdot kg^{-1}$ compared with 1.7 $\mu g \cdot kg^{-1}$ in the group who did not develop complications. This study also indicated that the intraoperative use of NMBDs and inhalational agents is not a risk factor for developing airway complications in OSAS patients.

INHALATIONAL AGENTS

The use of nitrous oxide and volatile agents may be beneficial to decrease the amount of opioids required to maintain anaesthesia.[56] However, when pulmonary hypertension is present, nitrous oxide may cause a marked increase in pulmonary vascular resistance and pulmonary artery pressure,[112] and its use may be inadvisable particularly when RV dysfunction is present. This also implies the use of a high oxygen concentration (FiO_2) which is helpful to decrease pulmonary artery pressure.

VENTILATION

Controlled ventilation using minute volumes to produce normocapnia should be used. In Pickwickian patients, the preoperative $PaCO_2$ values should be maintained.[113] Hypocapnia has been implicated in the pathogenesis of both sleep apnoea[114] and postoperative hypoxaemia,[115] thus the use of hyperventilation with hypocapnia should be avoided.

INTRAOPERATIVE MONITORING

This should include routine monitoring of ECG, blood pressure, pulse oximetry, end-tidal CO_2 and neuromuscular blockade. In Pickwickian patients arterial cannulation for direct blood pressure monitoring and ABG analysis is often necessary. These patients also benefit from central venous and pulmonary artery pressure monitoring to guide fluid balance and optimize cardiac function both intra and postoperatively.[116]

Extubation

This should be performed only in the fully awake alert patient. There should be evidence of complete return of normal neuromuscular function, including both twitch and clinical criteria. In particular, a maximum inspiratory pressure of ≥45 cm H_2O and sustained head raising correlate with the ability to maintain UAW patency.[84] If UAW oedema is suspected, it must be ascertained that this will be unlikely to compromise airway patency. The presence of a "cuff leak" when the cuff is deflated and the tube occluded is a very sensitive predictor of successful extubation in patients with possible UAO.[117] However, this test is not specific and in many patients who have no leak the trachea can be successfully extubated. Respiratory monitoring is imperative after extubation; and all equipment and personnel necessary for airway management must be immediately available.

The postoperative phase

The postoperative period is a particularly critical time for SAS patients recovering from GA. They are at high risk of hypoxaemia, and its possible consequences of cardiac and cerebral dysfunction. Episodic hypoxaemia has been shown to occur both early (first day)[80] and later (second to fifth days)[118] in non-apnoea surgical patients postoperatively. These episodic events occurred during sleep and were often associated with obstructive or central apnoeic episodes. Early episodic desaturations are thought to result from the combined effects of opioids (given for pain relief) and sleep on activity of the UAW musculature and central respiratory drive. Late episodic desaturations may be secondary to REM sleep rebound particularly during the fourth and fifth nights,[2] with its associated increase in frequency and severity of apnoeic episodes.

Patients with SAS are no less vulnerable to the detrimental effects of opioid analgesics and altered sleep pattern[119] in the postoperative period than normal non-apnoeic patients. Moreover, given the deleterious effects of residual anaesthetics, accentuation of pre-existing sleep apnoea is expected during the immediate post-anaesthetic period. Several case reports have documented such severe respiratory compromise (UAO, respiratory arrest, or oxygen desaturation) as to necessitate immediate intervention in patients with OSAS,[24,96,120–122] CSAS,[102] or alveolar hypoventilation syndrome.[103,123] Acute pulmonary oedema[22,91] secondary to frequent and/or prolonged OSA episodes may further aggravate the hypoxaemia.

ICU ADMISSION?

SASs can be mild, moderate or severe in nature, the distinction among groups being based on symptom severity and/or PSG criteria. Often, patients with severe SAS will need to be managed in the Intensive Care Unit (ICU) postoperatively.[57] However, it is not known which patient with mild or moderate disease will worsen

so as to justify ICU admission on a routine basis. As deterioration is expected most consistently in the immediate postoperative period, it may be prudent to monitor these patients on a high-dependency ward with pulse oximetry during this period. Further action will be dictated by the patient's course during such monitoring i.e., discharge, further hospitalization or ICU admission for more comprehensive monitoring (ECG and apnoea monitors, in addition to oximetry) and therapy. However, there are no rigid rules and this approach must be individualized for each patient, depending on the pathophysiology, type of surgery, anaesthetic technique used, analgesic requirements and the perioperative course.

Postoperative management

POSITIONING

Adoption of a sitting posture may be useful postoperatively in OSAS patients. This manoeuvre has been shown to reduce OSAs and improve SaO_2 particularly in morbidly obese patients.[124]

OXYGEN THERAPY

The routine use of supplemental oxygen is a matter of debate. Early episodic desaturation due to sleep apnoea could be prevented by administering 28% oxygen by facemask.[125] However, there is concern that oxygen may increase the apnoea duration by delaying the arousal caused by hypoxaemia, thereby leading to CO_2 retention and respiratory acidosis.[126] Therefore, it may be preferable to provide oxygen supplementation only when desaturation is indicated by oximetry. It must be noted that extremely low SaO_2 may be acceptable in the subset of SAS patients with awake hypercapnia as in Pickwickians[25] and patients with central alveolar hypoventilation.[103] These patients may respond to high FiO_2 by further decreasing alveolar ventilation; severe respiratory depression may result from restoration of SaO_2 to normal or near-normal levels. Frequent ABG sampling is essential to titrate the FiO_2 against arterial tension of both oxygen and carbon dioxide.

ANALGESIA

Systemic opioids should be used with extreme caution as they can result in frequent obstructive apnoeas with pronounced oxygen desaturations.[80] Epidural morphine has been successfully used in one patient with OSAS,[127] but delayed depression of central respiratory drive is serious and may contraindicate this technique in patients with CSAS.[102] Patient-controlled analgesia has also been successfully used in morbidly obese SAS patients after gastric bypass procedures.[128] However, careful attention to details is necessary if avoidance of serious complications is sought.[122] Clearly, further studies are required to define the role of newer approaches of opioid administration in SAS patients. Regional analgesia is associated with a low incidence of apnoea and is completely free of hypoxic episodes,[80] thus may be the most ideal form of analgesia in SAS patients. Non-steroidal antiinflammatory drugs (NSAIDs) have considerable analgesic effects[129] and they are increasingly used in postoperative SAS patients.

CPAP THERAPY

Patients who are already being treated with nCPAP should resume that treatment as early as possible postoperatively.[57] Nasal CPAP may also be used to maintain pharyngeal patency in patients demonstrating recurrent apnoeas after extubation, and has been shown to prevent apnoea-related blood pressure fluctuations.[119]

VENTILATORY THERAPY

Because of their pathophysiology, several SAS patients may need a variable period of postoperative mechanical ventilation in the ICU. Examples of such patients are those with morbid obesity,[99] awake hypercapnia,[116] chronic lung disease,[130] myotonic dystrophy,[131] post-obstructive pulmonary oedema,[91] or prolonged central apnoea after tracheostomy.[24] Prudence also dictates, when relatively large doses of opioids are used intraoperatively[91] or are needed postoperatively, maintenance of the tracheal tube with or without mechanical ventilation.

Anaesthetic implications of specialized surgical treatment of OSAS

Site-specific UAW procedures are often prolonged, particularly when multiple procedures are concomitantly made to address multiple levels of obstruction. A relaxant-based anaesthetic is, therefore, most appropriate. As access by the anaesthetist to the patient's head will be restricted during surgery, the tracheal tube and all relevant connections must be firmly taped and a disconnection alarm used. To allow the anaesthetic machine to be further from the operative field, the Bain system can be used with advantage. A simple arrangement consists of connecting a suitable ventilator to the reservoir bag mount of the Bain system with the valve closed. An additional advantage of this arrangement is that different combinations of fresh gas flow and minute volume can be selected to produced a desired $PaCO_2$.

Uvulopalatopharyngoplasty

The UPPP is performed with the patient supine and the head slightly elevated to enhance venous drainage. In

addition, localized ischaemia may be produced by local infiltration of epinephrine. The patient should be carefully observed for dysrhythmias, particularly if halothane is used; isoflurane is a better choice of volatile agent. The addition of a local anaesthetic to the infiltrate may be helpful to reduce postoperative pain and analgesic requirements.[132] A smooth recovery is important, as coughing or straining may result in disruption of palatal suture line and healing with stenosis, a serious complication which often necessitates tracheostomy.[57,98] Considerable worsening of OSA during the early postoperative period may result from residual anaesthetic effects or UAW compromise from oedema. Acute UAO has been reported immediately after extubation[91,100,120] and has resulted in several deaths.[100] A nasopharyngeal tube may be left in place to maintain airway patency after extubation.[98] This may be supplemented with CPAP and/or oxygen therapies.[133] For postoperative analgesia NSAIDs have been generally advocated, including acetaminophen,[134] diclofenac[135] and oxicam derivatives.[129] Respiratory monitoring for one or two nights postoperatively is often pursued.[136]

Lingual surgery

A tracheostomy is mandatory before surgery involving the base of the tongue. Microlaryngoscopy and lingualplasty are performed using the CO_2 laser[63,64] and a laser-safe tube should be used. Appropriate safety precautions must also be taken to prevent inadvertent tissue injury of both the patient and operating room personnel. Odynophagia is a significant problem postoperatively.

Mandibular and maxillomandibular surgery

A cuffed armoured tracheal tube should be passed nasally to permit occlusion of the teeth (intermaxillary fixation). In MMA, an extra long latex reinforced tube may be used so that all connections may be away from the immediate facial region. The tip of the tracheal tube should be sited just above the carina to prevent accidental extubation during surgical advancement of the maxilla.[137] Controlled hypotension may be used to reduce blood loss in patients who appear to bleed excessively.[138] Postoperatively, all patients are managed in the ICU with oxygen supplementation, analgesics, antiemetic/H_2 blocker therapy, and monitored with oximetry. CPAP is frequently used and should be applied through a nasopharyngeal airway to avert aggravation of any subcutaneous emphysema produced by maxillary incisions (Dr. R.W. Riley, personal communication).

Tracheostomy

This procedure can safely be performed under local infiltration anaesthesia in well-selected OSAS patients. However, because tracheostomy may be exceedingly difficult in the markedly obese patients with short necks, preoperative control of the airway by elective awake intubation has been recommended.[24] Careful monitoring after surgery is advisable since some patients may be at risk for continued oxygen desaturation resulting from exacerbation of a previously subclinical central apnoea,[24] associated COPD[139] or rebound excessive REM sleep.[57]

Conclusion

Sleep apnoea syndrome presents the anaesthetist with many challenges. In addition to the difficulties of tracheal intubation imposed by the anatomical features, these patients are sensitive to central depressant drugs, particularly opioids. Anaesthetic care, postoperative analgesia and cardiorespiratory management need to be handled with care.

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