Anesthesia, Sleep, and Upper Airway Collapsibility

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- Anesthesia • Sleep • Upper airway collapsibility
- Obstructive sleep apnea

Smooth anesthetic induction and emergence is held as an anesthesia management ideal. The term implies a seamless change from the awake to the anesthetized state and back again, based on skillful management of the rate and magnitude of drug administration and attendant issues, including airway management. It conjures up concepts of careful titration to varying concentrations at the site of drug action to produce dose-dependent effects and of the orderly pharmacokinetic processes of absorption, distribution, tissue and receptor binding, and elimination that modulate these changes.

The term does, however, mask the fact that the transition from consciousness to unconsciousness during anesthetic induction is, in neurophysiologic terms at least, quite abrupt,[1] as it is during sleep onset,[2] with a relatively stable state of consciousness or unconsciousness on either side of the transition. Indeed, the thalamocortical pathways involved have been thought to have the characteristics of a bistable flip-flop switch.[2] The transition to unconsciousness is accompanied by an abrupt decrease in upper airway muscle activity and increase in upper airway collapsibility.[3] Hence, on one side of the divide (moderate [conscious] sedation) there is some protection against obstruction afforded by muscle activation and rousability, whereas on the other side (deep [unconscious] sedation) there are vulnerabilities associated with muscle relaxation and, when anesthetized, lack of rousability. Understanding this dichotomy is important in helping plan safe perioperative airway management,

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especially where there is particular vulnerability to obstruction, as with obstructive sleep apnea (OSA).

REGULATION OF CONSCIOUSNESS

An ascending arousal system originating in the brainstem is crucial in maintaining wakefulness with well-defined cell groups involved in 2 major pathways.[2] The major inputs into one of these are the upper pontine pedunculopontine and laterodorsal tegmental nuclei. These activate thalamic relay neurons and the thalamic reticular nucleus that are crucial for transmission of information to the cerebral cortex. The other pathway originates in centers in the upper brainstem and caudal hypothalamus, including the locus coeruleus, dorsal and median raphe nuclei, ventral periaqueductal gray matter, and the tuberomammillary nucleus. This ascending arousal pathway bypasses the thalamus, activating pathways in the lateral hypothalamus and basal forebrain and then the cerebral cortex.

The activity of these wakefulness-promoting pathways is inhibited by a system of gamma-aminobutyric acid (GABA)-containing neurons, in which the lateral hypothalamic ventrolateral preoptic nucleus (VLPO) appears to play a key role. It both receives afferents from and has outputs to the major cell groups in the brainstem and hypothalamus that participate in arousal. These pathways have mutually inhibitory influences on each other (**Fig. 1**).[4] During wakefulness the activity of the VLPO is strongly inhibited by the locus coeruleus and other wakefulness-promoting centers. As the activity of these decreases at sleep onset, the VLPO becomes active, in turn reciprocally inhibiting their activity. These mutually inhibitory elements set up a self-reinforcing loop whereby activity on one side inhibits activity on the other, acting to disinhibit its own activity. This activity has the characteristic of a bistable flip-flop switch that acts to produce stable states of wakefulness or sleep with sharp transitions between them.[2] Relevant to anesthesia, the VLPO is heavily populated by GABA type A-ergic neurons and anesthetic agents (eg, propofol) potentiate its GABAergic inhibition of

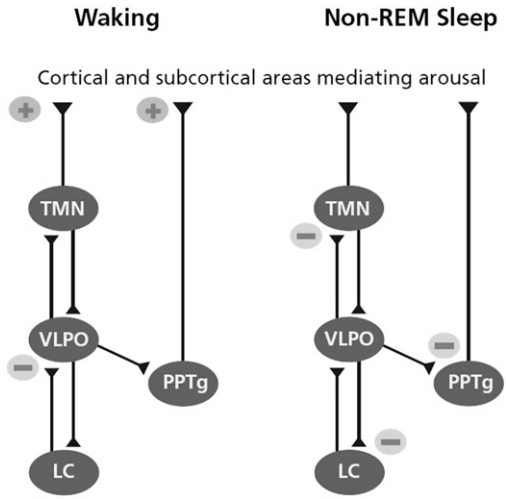

Fig. 1. Some connections of the ventrolateral preoptic nucleus involved in the regulation of sleep and wakefulness. TMN, tuberomammillary nucleus; LC, locus coeruleus; PPTg, pedunculopontine tegmental nuclei. (*From* Harrison NL. General anesthesia research: aroused from a deep sleep? Nature Neuroscience 2002;5:928; with permission.)

arousal pathways through stimulatory effects on $GABA_A$ receptors, and perhaps by facilitating excitatory inputs into the VLPO.[5] Hence, the sleep switch is also activated by anesthetic agents and may constitute a narcotic mechanism that is common to sleep and anesthesia.[1,4]

Consistent with this threshold-related switching behavior wake-sleep and sleep-wake transitions are abrupt as are transitions from consciousness to unconsciousness during induction of anesthesia.[1,3] An essential difference between the sleep and anesthetized states is, of course, that with sleep following the flip to unconsciousness, moderate stimulation is then sufficient to disturb sleep causing a flop to consciousness; whereas once anesthetic drug-induced unconsciousness is induced, the capacity to arouse requires at least some drug elimination to occur before it is restored.

Consciousness provides protection for upper airway patency and respiration. The wakeful state provides a nonspecific behavioral drive to respiration and to the upper airway musculature, enhancing tonic and phasic neuronal activation. It is the loss of this activation with sleep or anesthesia-induced unconsciousness that is responsible for increased collapsibility of the upper airway[3] and the obstruction that ensues in predisposed, unprotected individuals. In sleep obstruction of the upper airway is terminated by arousal, accompanied by muscle activation and restoration of patency with repetitive events forming the basis of OSA. In anesthesia, such obstructive events require active intervention by attending staff at least until the ability to arouse spontaneously is restored.

DETERMINANTS OF UPPER AIRWAY PATENCY

Upper airway obstruction during sleep or anesthesia results from a combination of an anatomically predisposed airway and the permissive effect of the muscle relaxation that is an inevitable consequence of these states. Anatomic predisposition can be thought of in terms of caliber and shape of the airway, extraluminal tissue pressure, and airway wall compliance. Obstruction is most likely to occur where substantial anatomic predisposition is present, posture is unfavorable (supine, mouth open, neck flexed), and muscle relaxation is profound (as in anesthesia or rapid eye movement [REM] sleep). The tendency to collapse is countered by upper airway muscle activation.

Caliber of the Airway

There are several reasons why a narrow airway is more vulnerable to collapse. First, and most obviously, the smaller the airway the less is the absolute change in luminal cross section required for airway closure. Second, Laplace's law dictates that at equilibrium the transmural pressure across a concave surface is directly proportional to wall tension and inversely proportional to its radius of curvature. It follows that the transmural pressure gradient required to prevent collapse of the airway varies inversely with its radius of curvature. Third, increased resistance of the narrowed airway necessitates generation of more negative intraluminal pressures. Fourth, airway wall compliance is increased at lower calibers (**Fig. 2**).[6]

Transluminal Pressure Gradient

The pressure gradient across the pharyngeal wall is determined by the difference between the pressure in the tissues surrounding the airway (the extraluminal tissue pressure) and the pressure within the airway lumen (intraluminal pressure). The extraluminal tissue pressure increases with obesity and other increases in tissue volume, such as edema, or with narrowing of skeletal confines, as with neck flexion or micrognathia or retrognathia.[7] Intraluminal pressure decreases during inspiration and

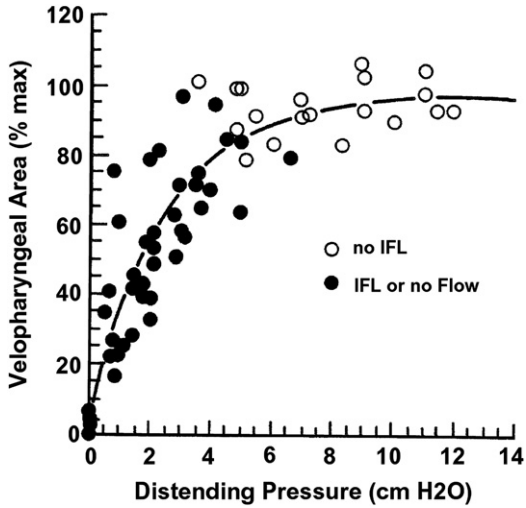

Fig. 2. Relationship between velopharyngeal area and distending pressure in humans. Compliance (slope of the relationship) increases at low calibers. IFL, inspiratory flow limitation. (*Adapted from* Isono S, Morrison DL, Launois SH, et al. Static mechanics of the velopharynx of patients with obstructive sleep apnea. J Appl Physiol 1993;75:148–54; with permission.)

increases during expiration. The magnitude of the inspiratory decrease is a function of inspiratory flow rate and of resistance upstream of the site of interest. It becomes greater, for example, with increased nasal resistance.

Compliance of the Airway Wall

Pharyngeal wall compliance is a measure of its degree of flaccidity and, in the absence of muscle activity, reflects its intrinsic elastic properties and the degree of radial (transverse) and axial (longitudinal) tension to which it is subjected. A more compliant (flaccid) airway is more likely to collapse and, indeed, compliance is increased in OSA.[6] The properties of connective tissue, bone, and fat all influence intrinsic compliance, as does surface tension.[8] Compliance tends to increase at low calibers, increasing collapsibility (see **Fig. 2**).[6] Longitudinal tension on the airway increases with increasing lung volume (tracheal tug), acting to decrease compliance.[9] This activity, along with reflex muscle activation (see later discussion), stabilizes the airway during inspiration.

Upper Airway Muscle Activation

Inspiratory activation of pharyngeal dilator muscles, of which the genioglossus is the most important and best studied, counteracts the narrowing effect of the inspiration-associated decreases in intraluminal pressure. In addition to this phasic inspiratory activity, tonic activity is present during wakefulness to help stiffen and stabilize the airway wall. The activity of the genioglossus (and other extrinsic tongue muscles, apart from palatoglossus) is mediated via the hypoglossal nerve. Its nucleus is situated in the medulla and receives a variety of inputs that together determine its output.[10] These inputs include negative pressure reflexes initiated by mechanoreceptors principally situated in the larynx, phasic inspiratory input from respiratory neurons arising from the pontomedullary central pattern generator, and a tonic excitatory stimulus related to the wakeful state. Each of these inputs is depressed by sleep or anesthesia to

a varying degree through central effects, with muscle relaxants providing an additional peripheral component in cases where they are used. Hence, accounting for all of the inputs is important when considering the effects of sleep and anesthesia on upper airway behavior.

THE NATURE OF OBSTRUCTIVE SLEEP APNEA

With sleep, upper airway collapsibility increases as a result of a reduction in pharyngeal dilator muscle activation and loss of lung volume, which decreases longitudinal traction on it. The reduction in muscle activation appears to be the combined result of loss of the stimulatory effect of wakefulness, reduction in respiratory drive, and depression of negative pressure reflexes. These changes are evident at sleep onset but vary in intensity with sleep state, being more profound in REM than non-REM sleep. Although the accompanying increase in upper airway collapsibility with sleep is present in everyone, in normal individuals without anatomic compromise, the upper airway is robust and patency is not significantly compromised. However, in individuals with anatomically vulnerable airways, these changes can precipitate partial or complete upper airway obstruction. Indeed patients with OSA appear to have a relative increase in wakeful upper airway muscle activation to compensate for their anatomic predisposition to obstruction.[11]

OSA is characterized by recurrent episodes of partial or complete upper airway obstruction during sleep. These obstructive hypopneas and apneas are associated with hypoxemia and sympathetic activation and are terminated by arousals, which disrupt sleep and are responsible for the excessive daytime sleepiness that usually accompanies it. Loud habitual snoring is a usual, but not invariable, associated feature that signifies the presence of a narrow, floppy airway. Hypoxia may also accompany the respiratory disturbances; its magnitude varies with the length of the disturbance, lung volume (and therefore size of oxygen stores), and degree of intrapulmonary shunt. Although magnitude of hypoxemia and extent of symptoms are important, severity of OSA is usually expressed in terms of the number of apneas and hypopneas per hour of sleep, the apnea-hypopnea index. In adults, less than 5 respiratory events per hour of sleep is considered normal, 5 to 15 mild, 15 to 30 moderate, and greater than 30 severe OSA.[12] Sleep is not a homogenous state and severity of OSA varies with sleep stage, body posture, and neck position. It is worse in REM than non-REM sleep and is aggravated in the supine posture or when the neck is flexed. In milder cases, the problem may only be apparent when the individual is in supine REM sleep.

A total of 4% of adults have OSA to a clinically significant degree.[13] Everyday consequences of the problem include the social costs of the loud snoring that often accompanies it; the excessive daytime sleepiness that results from recurrent arousals with its negative implications for safety, productivity and social interactions; and various comorbidities, including hypertension and vascular disease, diabetes and metabolic syndrome, and mood disorders. Addressing predisposing lifestyle issues, such as obesity and sedative or alcohol abuse, are important. Beyond addressing these issues, as long as no medically or surgically correctible predisposing abnormality exists, the treatment of choice is continuous positive airway pressure therapy delivered by a nasal or face mask.[14] Dental splints designed to hold the mandible forward during sleep are a useful but less predictably effective alternative.[15]

The primary site of collapse within the upper airway during sleep is the velopharynx in the majority of patients (approximately 80%), as it is during anesthesia.[16,17] The other common site of collapse is retrolingual. These vulnerable segments correspond to the narrowest levels within the upper airway.[18]

Factors that act to narrow the airway predispose to OSA. These factors include increasing age,[19] male gender[20] (with male [central] distribution of body fat important), menopause,[21] obesity,[22] increased neck circumference,[23] macroglossia,[24] retrognathia,[25] and maxillary constriction.[26] These factors may be present to varying degrees in otherwise normal individuals or they may be part of a disease syndrome, such as acromegaly, Down's syndrome, Pierre-Robin syndrome, or other syndromes associated with craniofacial abnormality. There is an association with difficult intubation and indeed many of the features that suggest a difficult airway from the anesthesia point of view, either difficult intubation or difficult mask ventilation, also suggest OSA.[27,28]

Obesity exerts its effects through increasing local compressive forces on the upper airway and by decreasing functional residual capacity, and therefore longitudinal traction on the upper airway, particularly when recumbent.[22] There are familial predispositions to OSA.[29] Neuromuscular conditions affecting the upper airway muscles also predispose to OSA[30] as do endocrine (hypothyroidism, acromegaly), connective tissue, and storage diseases that decrease upper airway caliber. Specific pathologies in the upper airway also predispose to obstruction at discrete sites. These pathologies include nasal obstruction,[31] tonsillar and adenoidal hypertrophy,[32] pharyngeal tumors, foreign bodies, hematomas, and edema. Stroke and head injury can increase vulnerability to OSA by depressing muscle tone and arousal responses, as can alcohol[33] and sedative consumption.[34] Various sleep postures increase vulnerability to obstruction, including supine recumbency,[35] neck flexion,[36] and mouth opening.

OSA AND PERIOPERATIVE RISK

The factors that predispose to OSA also predispose to obstruction under anesthesia and those with OSA are prone to difficulties with tracheal intubation and with airway maintenance under anesthesia.[27] There is a relative paucity of studies of perioperative risk relating to OSA and so published guidelines, such as the American Society of Anesthesiologists guidelines,[37] remain heavily dependent on expert opinion rather than high-level evidence. Nevertheless, the available literature does suggest some trends that appear consistent with pathophysiological considerations.

A large retrospective case control study found that OSA did not appear to be a risk factor for unanticipated admissions in outpatient surgery; there was no difference in perioperative adverse events or unplanned admissions between subjects with OSA and matched controls.[38] When the reasons for such unplanned admissions were analyzed there was no clear difference in profiles of causes between the groups. However, when major surgery is considered a different picture emerges; a retrospective case control study of postoperative morbidity after hip or knee arthroplasty demonstrated a substantial increase in complications, particularly serious complications, including those requiring unplanned intensive care unit admission, in subjects with OSA.[39] It showed an increased length of hospital stay for these subjects. These findings are supported by another retrospective matched cohort study that suggests that the risk of postoperative complications for patients with diagnosed OSA is double that of patients without this diagnosis.[40] Risks associated with upper airway surgery appear to increase in the presence of OSA; in adults post-uvulopalatopharyngoplasty respiratory complications appear to be associated with more severe OSA.[41] A study of children undergoing adenotonsillectomy for OSA versus those undergoing this procedure for other reasons demonstrated an increase in complications in the immediate perioperative period for the OSA subgroup, including problems during induction of anesthesia, emergence from it, and in early recovery.[42] Although a far greater body of literature is required, these findings suggest that, at least for minor procedures,

the major risks relating to OSA status are likely to be found in the immediate perioperative period, with patients undergoing upper airway surgery at particular risk. This finding may particularly be the case with children given their narrow upper airways and low functional residual capacities, which act to limit oxygen stores relative to metabolic requirement when in the obstructed state. It may be that the risks relating to major surgery relate to the requirement for postoperative sedative and narcotic analgesics in many of these patients, with an attendant risk of impaired consciousness (either through direct drug effect or through respiratory depression and hypercapnia) and depressed arousal responses. These observations are consistent with the notion that the perioperative and postoperative problems for patients with OSA particularly relate to unconsciousness and suppression of arousal responses and that their risk profile diminishes substantially with return of consciousness and rousability following anesthesia, providing they are not further compromised by subsequent drug administration.

The return of ready rousability is an important criterion to be met before discharge from the recovery room following anesthesia for patients without other cause for prolonged unconsciousness. Although emergence from anesthesia can be accompanied by abrupt return of consciousness[43] (again suggesting the threshold switchlike behavior at the interface between consciousness and unconsciousness), often this is not the case, which reflects persistent sedative drug activity in some cases. However, it is also probable that anesthesia frequently transposes into natural sleep when postoperative pain in not an issue, particularly late at night when propensity to natural sleep is the greatest. This factor may not be readily recognized given the similarities in behavioral appearances between sleep and anesthesia.

In outpatient surgery, discharge from the recovery room is followed by discharge home, within a few hours, once the effects of the anesthetic drugs have dissipated. However, with more invasive surgery, the substantial postoperative analgesia/sedation that is often required means that vulnerable patients remains at risk of upper airway obstruction through its depressant effects on rousability. Regional anesthetic techniques allow some of this potential difficulty to be circumvented. Although anesthesia itself appears to have some restorative properties when unmodified by the effects of surgery,[44] sleep is usually disturbed in the postoperative period. The degree of this disturbance varies with type and extent of surgery, pain and its treatment, personality, and the nursing environment. It is characterized by sleep restriction or deprivation, sleep fragmentation, and distorted sleep architecture with loss of REM sleep and may persist for many days postoperatively.[45] These disturbances can result in cognitive and psychomotor dysfunction.[46] These disturbances cause 2 problems for patients with OSA: (1) the effects of the postoperative sleep fragmentation compound those of fragmentation caused by OSA itself, and (2) re-establishment of normal sleep after a substantial period of sleep deprivation is characterized by a state of "REM-sleep rebound" during which the proportion of REM sleep increases above the usual adult proportion of 20% to 25% of total sleep time.[45] This state may last 1 or more nights. Given that REM sleep is the most vulnerable stage of sleep for depression of neural drive to the upper airway, the overall severity of OSA can be aggravated by it.

OSA AND ANESTHESIA MANAGEMENT

Anesthesia management of patients with OSA must ensure that their particular vulnerability when unconscious or under the influence of sedative drugs is adequately addressed (**Box 1**). The potential vulnerability of patients with OSA is a matter of concern to anesthesiologists and has led to the development of guidelines for the

Box 1
OSA and anesthesia management

- Try to identify OSA preoperatively.
- When not previously diagnosed, refer patients for preoperative evaluation of sleep where the probability of OSA is high, surgery is elective, and there is a likely need for postoperative narcotic analgesia or sedation.
- When previously diagnosed and compliant with CPAP, ensure it is available for perioperative use.
- Where previously diagnosed but not compliant with CPAP, reinstruct in its use.
- Avoid sedative premedication.
- Use regional anesthesia and analgesia where feasible.
- When general anesthesia is used, be prepared for difficult intubation and other difficulties in airway maintenance. Use techniques that allow early return of consciousness.
- Try to minimize postoperative sedation.
- Have CPAP available for early postoperative use.
- Nurse in a high-dependency area with continuous monitoring until patients are sentient and able to self-administer CPAP. Patients requiring ongoing narcotic analgesia or sedation should remain in a high-dependency area until this need abates.
- Use lateral positioning, a nasopharyngeal airway, and oxygen therapy where CPAP is refused and upper airway obstruction is problematic.
- Consider OSA in patients with difficult airways perioperatively. Inform patients and refer for investigation for the possibility where clinically indicated.

perioperative management of OSA by organizations, such as the American Society of Anesthesiologists.[37] Principles addressed in such guidelines include systematic identification of patients at risk; avoidance of sedation in unsupervised surroundings; minimization of use of sedative and narcotic drugs; preparation for difficulties in intubation and airway management intraoperatively and immediately postoperatively; careful postoperative supervision until sentient; use of postoperative aids, such artificial airways or continuous positive airway pressure (CPAP) therapy where airway compromise exists; and particular care following upper airway surgery.

Identifying OSA Preoperatively

Patients with OSA may present with the diagnosis made and, better still, treatment instituted. However, at least as commonly given the notorious underdiagnosis of the problem, individuals with OSA present undiagnosed. A fairly simple collection of symptoms and signs will alert the vigilant anesthesiologist to the possibility of the disorder during preoperative evaluation. The cardinal symptoms of OSA are habitual snoring, witnessed apnea, disrupted and unrefreshing sleep, and excessive daytime sleepiness. Not all of these features are present in all patients, but any combination of them in an individual is highly suggestive of the presence of this common and potentially disabling disorder. Signs include obesity, increased neck circumference, mandibular or maxillary hypoplasia, oropharyngeal crowding (high Mallampati scores, decreased pharyngeal width), and hypertension. Any signs that suggest to an anesthesiologist that there may be difficulties with tracheal intubation or with airway maintenance perioperatively should also suggest OSA. The converse also holds; symptoms and signs of OSA should alert the anesthesiologist to the possibility of

difficulties with airway management. Clearly these symptoms and signs should be sought at preoperative evaluation, as should the history of prior diagnosis of the problem. Indeed, apart from the importance of observations made during preoperative evaluation and intraoperatively for perioperative management, they can also help identify previously undiagnosed OSA, with long-term benefits for patients. The relationship between OSA and airway collapsibility under deep sedation is utilized in sleep nasendoscopy. This procedure is performed under drug-induced sedation to assess upper airway collapsibility and identify the likely primary site of obstruction for subsequent surgical attention.[47]

When Possible OSA has not been Previously Diagnosed

When the possibility of previously undiagnosed OSA has been raised by preoperative evaluation, the probability of its presence can be assessed using a prediction rule approach, such as that of Flemons.[48] Surgery should be postponed when this assessment indicates an intermediate or high likelihood of OSA or the anesthesiologist is otherwise concerned and surgery is elective and likely to require narcotic analgesia or sedation postoperatively. When postponed, patients should be referred to a sleep physician for evaluation and, where indicated, initiation of appropriate treatment.

When OSA has been Previously Diagnosed and Patients are Compliant with CPAP Therapy

Patients with diagnosed OSA on CPAP therapy should be instructed to bring their equipment to the hospital for use whenever asleep or sedated.

When OSA has been Previously Diagnosed and Patients are not Compliant with CPAP Therapy

Patients who have been diagnosed with OSA, either independently or as part of preoperative workup, but who do not use CPAP regularly, should be reinstructed in its use preoperatively, so that it is readily applicable whenever under the influence of narcotics or sedatives.

Avoidance of Sedative Premedication

Premedication with sedatives or opioids should be avoided wherever possible when OSA is known or suspected. When these substances are required because of anxiety or pain, patients should be observed in a high-dependency area.

Anesthetic Technique

Regional anesthetic and analgesic techniques should be used where feasible. When general anesthesia is needed, the possibility of difficult intubation and difficulties with airway management must be considered. Technique and drugs used should be selected to allow early return of consciousness and minimal postanesthetic sedation whenever possible. CPAP must be available for immediate use postoperatively in all patients with known or suspected OSA. Indeed, CPAP or noninvasive bilevel ventilation should be considered for use during procedures involving moderate/deep sedation to maintain airway patency, particularly when patients are familiar with these therapies.

Postoperative Nursing Environment

Patients diagnosed with OSA, or when the suspicion of it has arisen preoperatively (but not been investigated because of emergency), intraoperatively (because of difficulty with tracheal intubation or maintenance of airway patency), or in the recovery

room, must be nursed in a high-dependency area postoperatively with appropriate monitoring, including continuous oximetry. This monitoring should continue until patients are sentient and able to reliably administer CPAP unassisted or, in cases where OSA has only been suspected, airway stability during sleep or sedation has been confirmed. Patients with ongoing requirement for postoperative narcotic analgesia or sedation should remain in a high-dependency nursing environment until this need abates. Particular care is required after upper airway surgery because postoperative edema may temporarily worsen upper airway patency, even after operations aimed at increasing pharyngeal caliber, such as laser-assisted uvulopalatoplasty.[49]

When CPAP therapy is refused and upper airway obstruction continues to be problematic, use of lateral positioning, a nasopharyngeal airway, and oxygen therapy are alternate but less satisfactory strategies. In some settings, such as when the nose is packed postoperatively, a face mask rather than a nasal mask will need to be used to deliver CPAP. In other settings, such as surgery involving the sino-nasal tract and skull base (eg, pituitary surgery), early use of CPAP postoperatively may be undesirable because of concerns regarding a potential for pneumocephalus.

Postdischarge Management

When suspicion of previously undiagnosed OSA has arisen as a result of preoperative, intraoperative, or postoperative events, patients should be informed and referred to a sleep physician for further investigation.

SUMMARY

Patients with OSA have airways that are difficult when unconscious, whether the unconsciousness is a result of sleep or anesthesia. Anesthesia presents particular problems for such patients because, unlike during sleep, protection afforded by the ability to arouse is suppressed. Furthermore, anesthesia is associated with profound muscle relaxation, whereas during sleep some muscle activation is retained during non-REM sleep. These particular vulnerabilities are present until consciousness returns and must be accounted for in perioperative anesthesia management. OSA is underdiagnosed and its prevalence continues to increase in advanced economies as obesity and age increases. Careful preoperative evaluation and insightful perioperative observation is likely to identify patients at risk, highlighting the need for both careful postoperative management and specific follow-up to ensure that the sleep-related component of the difficult airway receives appropriate care.

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