Diagnosis and Treatment of Respiratory Problems in Sedation and Anesthesia for Dentistry

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This paper will define those respiratory problems that are relevant to the practice of outpatient general anesthesia and sedation for dentistry. The respiratory problems of local anesthesia are confined to the hazards of aspiration of foreign bodies and to the central effects of the local anesthetic, the latter particularly in overdose. Even in acceptable dosage, the local anesthetic will potentiate the effects of the sedative or general anesthetic, and thus in discussing sedation, the local anesthetic effects will be included.

PREDISPOSING FACTORS

The increased airway reactivity seen with either acute infections or with allergic reactions poses problems for the administration of sedation and anesthesia. Thus, the avoidance of all irritants to the airway before beginning a procedure is relevant, and problems have been noted to occur in patients exposed to such irritants.[1] Increased airway reactivity in the presence of the common cold is well established, with the additional concern of possible postoperative pulmonary problems. The avoidance of sedation and anesthesia in such cases in the acute phase is well recognized. Not only does proceding cause a hazard for the patient, but also for the operator and assistants, and raises additional problems in adequately sterilizing any inhalation equipment. It, therefore, would be sensible to delay the administration of anesthesia in such patients until the airway is no longer reactive, although some studies seem to suggest that in the adult such concern is unwarranted.[2] Any evidence of secondary infection other than viral would definitely require that the procedure be postponed. Treatment of bacterial infection with regular antibiotics is then indicated.

Increased airway reactivity may follow if the airway is instrumented, as can occur during oral surgery. Increased secretions can also be noted, and bronchospasm is more likely to develop. Bronchodilators will help prevent bronchospasm and aid in the removal of secretions. Anesthesia time should be minimized and postoperative opioids avoided.

Chronic obstructive pulmonary disease (COPD) is a well recognized hazard. Not only is the airway probably more reactive, but viscid secretions can cause further respiratory obstruction. Inhalation anesthesia becomes difficult, and the problems of controlling spasmodic attacks during the procedure should be noted. In addition, there are problems postoperatively in obstruction of the airway by plugs of mucous and subsequent development of atelectasis. Care in administering oxygen (O_2) is recommended in order not to depress the hypoxic respiratory drive. Problems with mechanical ventilation and COPD occur, and this is an inappropriate anesthetic technique for outpatient dental anesthesia.

Obesity causes problems, as decreased lung volume is evident together with a diminished airway. In the obese patient, the semisupine position is the position of choice, because the obese patient, when supine, has a considerable decrease in lung capacity. In addition, the potential for hypertension and myocardial disease in such patients is an ever-present threat.

Pregnancy causes unique problems for respiratory patients. As with the obese patient, the large uterine mass can compromise lung capacity.[3] In addition, dilution of the blood, and thus relative anemia, requires increased oxygenation during the procedure, in particular to avoid fetal damage.

INTRAOPERATIVE FACTORS

A number of variables can induce respiratory problems in dental anesthesia and sedation. In reviewing the data presented on drug and other factors, it is important to evaluate respiratory effects by noting the ventilatory re-

sponse to changes in the arterial carbon dioxide tension ($PaCO_2$). Static measurements of blood gases, tidal volume, or respiratory frequency do not present the total picture.

Sleep and Posture

Sleep and posture initiate major changes in lung physiology. Significantly, sedation parallels the effects of sleep, and atelectasis occurs when sleeping or lying, which has been demonstrated by computer-assisted tomography. The dependent lung is always the most affected.[4] Nitrous oxide (N_2O) compounds the problem, as the changes occur more rapidly than when breathing room air or oxygen. Sleep decreases the tone of the upper airway, and the musculature of the tongue and pharyngeal tube may collapse inward. Central and obstructive apnea is common when opioids are used in conjunction with regional anesthesia. There is impaired function of the intercostal muscles during sleep, and impaired ventilatory responses to carbon dioxide (CO_2) during sleep. Thus, the problems of sedation can be compared with those noted with sleep. Even without general anesthesia, respiratory problems can still occur.

In addition, the position of the patient is significant in the development of respiratory problems. Forsyth's definitive study demonstrated clearly the best match of ventilation-perfusion occurs in the semisupine position, which makes it the best position for treatment under sedation as well as general anesthesia in regard to cardiorespiratory changes.[5]

Drugs

A side effect of all anesthetics is to decrease ventilation in a dose-related manner. Indeed, respiratory depression is used as a measure of anesthetic depth. All anesthetics decrease metabolism and hence CO_2 production. Therefore, the $PaCO_2$ is important in determining the effects of drugs, and particularly the ventilatory changes noted in the $PaCO_2$ response curve. Elevated $PaCO_2$ is important, as arrhythmias are common and intercranial pressure is increased. The CO_2 response with 1.1 MAC halothane is 40% of control.[6] Surgical stimulation, however, will maintain the $PaCO_2$ near normal levels. Inhalation agents tend to depress the depth of respiration, while sedatives and opioids tend to decrease the frequency.

Secretions

Secretions are increased by the use of many drugs, such as pungent general anesthetics, succinylcholine, and the anticholinesterases. The increased secretions impair mu-

cociliary transport; the cough reflex is impaired with anesthesia and sedation. Thus the ability of the patient to get rid of secretions is depressed. The use of the anticholinergic drugs, such as atropine and scopolamine, might be considered useful to decrease airway secretions. However, there is controversy whether these agents do more harm by drying secretions and causing mucous plugs in the airway. The routine use of the anticholinergic drugs is no longer recommended. If indicated, the drug of choice is glycopyrrolate. Few studies have been done to demonstrate that it is more effective in decreasing secretions, but the cardiovascular effects are less than the major changes effected by atropine.

Instrumentation

The problem of instrumentation in dentistry is compounded by the shared operating site and airway. Many consider that endotracheal intubation is a necessity for even outpatient general anesthesia for dentistry. The hazards of nasal endotracheal intubation are also well recognized.

The sites of obstruction for passage of the nasal endotracheal tube to the larynx are the inferior turbinate, the adenoidal tissue, the posterior pharyngeal recess, Passavant's ridge, and finally the false cords. Mortality and morbidity due to endotracheal intubation account for 30% of the anesthesia problems due to faulty technique. In a review published in the *Canadian Anaesthetists' Society Journal*, it was noted that of 50 patients who suffered problems due to faulty endotracheal technique, death occurred in 37, and cerebral damage in 13.[7] In addition to the recognized fatalities with endotracheal intubation, problems of morbidity have been noted. Aspiration occurs more frequently (although the cuffed endotracheal tube prevents aspiration during the treatment, the occurrence of postoperative aspiration is much greater). The electrocardiographic abnormalities that occur with endotracheal intubation have promoted the practice of deep anesthesia, a hazard in itself. Trauma is frequent with endotracheal intubation, to the mouth, pharynx, and dentition. The endotracheal tube does not guarantee an airway, as mechanical problems, such as kinking, overinflation and herniation of the cuff, and blockage of the cuff with secretions, occur that require constant vigilance.

The avoidance of endotracheal intubation can often be satisfactorily overcome by the use of the laryngeal mask. This mask is frequently utilized to provide satisfactory airway conditions during oral surgery or restorative dentistry, allowing the airway to be shared. The mask will not prevent aspiration and does not guarantee an airtight airway, however, and thus cannot be used in the presence of a full stomach. It has been shown to be a satisfactory

alternative to endotracheal intubation, though not to be confused as a complete substitute for endotracheal intubation.

PREVENTIVE THERAPY

Cessation of cigarette smoking, even on the day of administration, helps reduce the incidence of airway problems. Bronchodilators should be used preoperatively if the patient exhibits any degree of bronchospasm; decongestant nasal spray may be useful for uncomplicated nasal stuffiness.

MONITORING

The most common clinical signs of respiratory problems in dental anesthesia and sedation can be detected with a stethoscope. Using the pretracheal stethoscope is simple and satisfactory. Respiratory obstruction and secretions interfering with the airway can readily be detected, while the rate and depth of respiration can be routinely monitored.

Observation of the reservoir bag is also of great value and is one of the additional advantages of the administration of N_2O/O_2 sedation. Cyanosis, which is a late sign of respiratory insufficiency, is most readily detected in the oral mucosa. However, action should have been taken before this occurs. The pulse oximeter is an extremely useful device that should be available to all who administer sedation and anesthesia for dentistry. If the O_2 saturation value falls below 90%, then the arterial oxygen tension (PaO_2) can be readily calculated by subtracting 30 from the O_2 saturation value. The shape of the O_2 dissociation curve indicates the hazard of waiting for prolonged falls in O_2 saturation below 90%, as by that time the PaO_2 has fallen precipitously. Pulse oximetry is theoretically not required for conscious sedation, as patient status can be adequately evaluated by talking to the patient together with the use of a pretracheal stethoscope. However, legislation dictating its use is becoming commonplace.

The third technique of evaluating ventilation during sedation and anesthesia is by measuring the end-tidal CO_2 tension. Unless there is an airtight fitting, as for an endotracheal tube, changes only can be noted, and absolute values are not available due to leakage. However, a valuable guide can be obtained by attaching a catheter adjacent to the airway to assess end-expiratory CO_2, noting changes in the waveform or deviations in the levels. An absent or low CO_2 does not solely reflect changes in ventilation; low CO_2 may be due to a low cardiac output.

SEQUELAE

The general sequelae that develop following respiratory complications during dental anesthesia are hyperpnea, hypercarbia, and hypoxia.

Hyperpnea

Two factors frequently lead to hyperventilation in the clinical setting: fear and pain. A sequence of events then may ensue, including hypocarbia and respiratory alkalosis; cerebral vasoconstriction, decreased release of oxygen from hemoglobin, and altered cerebral function; and decreased plasmic ionic calcium and tetany. If intravenous sedation is used to overcome the hyperventilation, apnea may ensue because of the hypocapnia and lack of respiratory drive. In the case of fear-induced hyperventilation, the buildup of CO_2 will restore ventilation before hypoxia develops. However, if the hyperventilation was in response to noxious stimulation, such as scraping of the periosteum, the apnea that develops upon cessation of the stimulus may persist until hypoxia develops. Thus the traditional technique of allowing excess buildup of expired CO_2 in the ventilatory system should not be attempted until the initiating factor has been determined. Apneic oxygenation will prevent hypoxia in these instances.

Hypercarbia

Hypercarbia, in the main, is due to decreased elimination of CO_2. Rarely, except in malignant hyperthermia, is it due to increased production. This depression of respiration is usually related to depression of the respiratory center with anesthetic, sedative, and opioid agents. Obstruction of the airway in such patients compounds the problem, resulting in further increases in $PaCO_2$. Hypercarbia stimulates respiration and will increase blood pressure and pulse rate together with peripheral vasodilation. The response to increased catecholamines under anesthesia may include arrhythmias. However, elevation of CO_2 will also produce anesthesia, and consequently further increases in $PaCO_2$ will result in further deepening of the anesthetic with progressive increases in CO_2 tension. Treatment should be directed to reversal of the hypercarbia by artificial ventilation until respiratory acidosis is corrected. Respiratory acidosis responds to adequate ventilation; sodium bicarbonate is not indicated. It must be remembered that hypercarbia is rarely a fatal event, except indirectly by the myocardial effects of tachycardia, hypertension, and arrhythmias.

Hypoxia

Hypoxia is a frequent occurrence in anesthesia and sedation that must be avoided. The use of the pulse oximeter gives valuable information in this regard. Hypoxia may

develop due to an inadequate inspired O_2 tension, as could occur in high altitudes, or with failure of the delivery system when the patient is attached to an anesthetic circuit. It occurs during general anesthesia unless 30% O_2 is administered. During sedative techniques, depression of respiration is such that the inspired O_2 falls due to inadequate ventilation, and thus compensation should be made with the administration of O_2. During early induction with 80% N_2O and 20% O_2, a condition of hyperoxia exists due to the second gas effect. However, 30% O_2 for prolonged administration should be considered the norm. Any problems of airway obstruction will result in a low inspired O_2 and the consequent development of hypoxia. It is, however, with the development of shunting during anesthesia and sedation that hypoxia unassociated with ventilatory problems can develop. The ideal posture is semisupine, as ventilation is improved in this situation and dependent atelectasis is less likely to occur. An interesting feature was noted by Wise in the administration of anesthesia with methohexital when the cardiac output failed to rise in the presence of hypoxia, as normally occurs.[8]

While the ventilatory response to a PaO_2 of 40 is eliminated by 1.1 MAC halothane, 0.1 MAC halothane for analgesia and sedation depresses the hypoxic response to 25% of control.[9] Other inhalation agents similarly depress the ventilatory response to hypoxia, as do opioids and some sedatives. A similar ventilatory response has been noted under halothane anesthesia to metabolic acidosis. Droperidol has been shown to increase the hypoxic sensitivity of the respiratory center, and can be considered a useful sedative for dentistry when it is desirable to avoid respiratory depression.[10]

The significance of hypoxic respiratory depression is principally noted in the postoperative period, when the O_2 tension is lowered, and then further depressed by the inability of the respiratory center to respond with increased ventilation. A recent study by Dahan and Ward[11] has shown that diazepam and midazolam also decrease the ventilatory response to hypoxia, which could account for the fatalities that have occurred in the elderly with these drugs.

SPECIFIC PROBLEMS

Apnea

The major hazard of general anesthesia or deep sedation is apnea. It can be readily treated by ventilation with oxygen or even room air. During the period of ventilation, attention should be paid to the diagnosis of the problem. If it is excess inhalation agent, then artificial ventilation will eliminate the high concentrations administered. If it is an intravenous opioid, then consideration should be given to administering either naloxone or nalbuphine.

These only work in patients who have been narcotized. Naloxone (Narcan) works on the mu receptors; naloxone drip, which was frequently recommended for treatment of nonopioid-induced apnea, has proved to be ineffective. Drugs such as diazepam and midazolam can be reversed with flumazenil. A nonspecific reversal of apnea occurring with general anesthesia can sometimes be influenced by analeptics. The reversal agents are not a primary response; rather ventilation with O_2 is the cornerstone of treatment. It is not enough to suggest that narcotic apnea is no problem, as an emergency room physician misstated, "Anybody could hold their breath long enough until the Narcan had been given."

Upper Airway Obstruction

Probably the most common cause of respiratory problems in all anesthesia and sedation is upper airway obstruction. Soft tissue obstruction of the airway can be corrected in the majority of instances by lifting behind the angle of the jaw and thrusting the jaw forward, pushing the soft tissues anteriorly. This increases the anterior-posterior dimension of the airway and pushes the tongue out of the way. Placement of an airway, either nasopharyngeal or oral, is also useful if the patient is sufficiently obtunded. The nasopharyngeal airway is more readily tolerated than the oral airway. A significant study was done by Moore et al, who demonstrated that the addition of N_2O to chloral hydrate sedation initiated airway problems in 25% of pediatric patients.[12] In the United Kingdom, regulations have been promulgated that only a single drug administration can be considered conscious sedation.

Aspiration

Aspiration of stomach contents or foreign bodies is not confined to sedation and anesthesia. Early reports of aspiration of tooth fragments in the 1950s described calculus and tooth fragments that entered the right mainstem bronchus even though the patient received only local anesthesia. Aspiration has been suggested to be a common problem at the present time. It is suggested that 40% of the population suffer with hiatal hernia, which can result in regurgitation and aspiration of stomach contents; hence, the vogue for the administration of preoperative H_2 blockers and antiemetic agents. It has not, however, been demonstrated to be a clinical problem.

Endotracheal intubation does not prevent aspiration except during the procedure. Endotracheal intubation in head and neck cases is the most common cause of postoperative aspiration. The adoption of the semisupine position is a useful technique to prevent aspiration. Regurgitation is a passive process, and gravity plays a role in its development. It does not require total suppression of the

cough reflex for aspiration to occur. Simple sedation with excessive alcohol is a frequent cause of aspiration. Depression of the airway reflex with an opioid may be considered an additional hazard in sedative techniques with the supine position, particularly when the administrator has not been trained in airway management and treatment of regurgitation.

Laryngeal Stridor and Spasm

A distinction must be made between laryngeal stridor and spasm. Laryngeal stridor is common; laryngeal spasm very rarely occurs and rarely requires treatment. It is important to appreciate that in laryngeal stridor excessive positive pressure within the ventilatory system is not the ideal method of treatment. Fink[13] noted in 1956 that further closure of the airway results as the soft tissues are pressed into the airway. Rather, the administration of 100% O_2 will help relieve the problem. Hypoxia tends to potentiate the stridor and, thus, any gas within the airway should be 100% O_2. Stridor is frequently made worse by excessive positive pressure within the upper airway. Significantly, only the posterior cricoarytenoid muscle opens the airway; teleologically humans and animals have developed laryngeal musculature to prevent aspiration. If, however, laryngeal spasm does occur, then one treatment is to attempt to open the airway with positive pressure, and allow some ingress of O_2. A useful measure for those who are skilled in ventilation and can assuredly manage endotracheal intubation under these circumstances is to administer a small dose, perhaps 10 mg, of succinylcholine. This will open the larynx in spasm and allow ventilation with 100% O_2. Forward traction on the tongue as a treatment for laryngospasm should never be done. Pleasants[14] suggested that all who administer N_2O/O_2 sedation should be required to perform laryngeal intubation, not having seen the results of the damaged airways and obstruction of the airways that occur when those who do not perform this task every day attempt laryngeal intubation. Nitrous oxide sedation does not require this form of treatment. Finally, in this regard, it is worth recalling Sir Robert Macintosh's dictum that the patient will always take the penultimate breath before expiring, and if this is 100% O_2, then the day will have been saved.

Bronchial Asthma and Bronchospasm

If the patient is intubated, it is important to distinguish the reactive airway from the tight chest of light anesthesia. Succinylcholine will control the tight chest, whereas intravenous lidocaine will treat the reflex-induced bronchospasm.

The development of an asthmatic attack under sedation or anesthesia requires instant active therapy. The diagno-

sis is made by the typical retraction of the ribs and the wheezing heard by stethoscope. If the patient is a known asthmatic and uses an inhaler, then a therapeutic advantage is for them to bring their inhaler with them. If the administrator has an anesthetic or analgesia circuit, then the inhaler can be fitted into the circuit and the drugs administered via a T tube. Of the many drugs available, albuterol and terbutaline are selective β_2 agonists. The choice of drug really depends on what the patient has previously found most suitable. If the condition persists, hand ventilation should be given and the anesthetic deepened while β_2 agonists are given. Modern adrenergic bronchodilators are quite safe, but the use of isoproterenol is no longer warranted. Theophylline is not indicated in an acute attack, though it may be used prophylactically, as may cromolyn.

If the patient has a known history of bronchial asthma, a premedication with a H_1-histamine blocker, such as diphenhydramine, is an extremely useful technique, providing sedation as a side effect. Possible consideration should be given to premedication with glycopyrollate, which will decrease secretions that may occur, particularly if the airway is instrumented. An attempt to decrease the amount of saliva is significant under such circumstances. Corticosteroid may also be given a few hours before in a preparation regime. Inhalation of metered doses of ipratropium, an anticholinergic bronchodilator, have no significant effects except in COPD.

The preanesthetic treatment should be aimed at minimizing any signs of COPD. This would allow the use of breathing exercises preoperatively and the use of antihistaminic drugs. Sedation and regional anesthesia would be the anesthetics of choice. General anesthetic induction should be smooth and given with a minimal amount of barbiturate, which would sensitize the airway. Ketamine is a useful drug for induction, and 1.5 mg/kg intravenous lidocaine should be given prior to intubation. Maintenance with volatile anesthetic agents would probably be the technique of choice, as this tends to produce bronchodilatation. On emergence, the patient should be carefully monitored to make sure that bronchospasm does not occur. The availability of an endotracheal tube for such patients is necessary.[15]

PEDIATRIC CONSIDERATIONS

Low oxygen reserve, because of the small size of pediatric patients, makes respiratory problems more hazardous in this age group than in adults.[16] In addition, the sensitivity of the pediatric patient to opioids is probably one of the reasons that there is a high incidence of cardiac arrest reported in pediatric sedation. Review of many of the pediatric cases indicates that the airway became lost and

respiratory depression occurred without the operator noticing. Development of cyanosis is a late sign and cannot be relied on to safeguard the pediatric patient, due to the low oxygen reserve. Attention to apnea that occurs in pediatric sedative practice suggests that opioids should be avoided in all pediatric sedation cases. The practice of injecting the opioids in such cases into the buccal mucosa can only be condemned. Research performed with alphaprodine (no longer available) utilizing this technique indicated that blood concentrations approximate to those with intravenous administration are achieved.

Anatomic Considerations

The infant has a relatively large head. The small mandible complicates lifting the jaw, and the small nares makes difficult nasal intubation. There is frequently a high arched palate, which accounts for the difficulty in achieving intubation, as does the large, floppy epiglottis. Macroglossia will often result in soft tissue upper airway obstruction, and consequently attention must be paid to the pediatric airway at all times. If laryngeal intubation is considered, then it is important to understand that the larynx is not the smallest part of the airway, that there is subglottic narrowing. Forcing an endotracheal tube through the small subglottic area is hazardous. The larynx is placed anteriorly and cephalad, and consequently visualization of the airway is frequently difficult.

Respiratory Infections

The frequency of respiratory infections in children is significant, as is difficulty in distinguishing the pediatric runny nose from either respiratory allergy or acute viral infections. By definition, upper airway infections primarily affect structures above the larynx, and lower respiratory tract infections below the larynx. Clinically however, the upper and lower portions are usually involved simultaneously. The diagnosis of a respiratory infection is consistent with the following: mild sore throat, sneezing, rhinorrhea, congestion, mild malaise, productive cough, a fever of 37.5° C to 38° C, laryngitis, and a white blood count over 10,000. The presence of three or more indicators is sufficient to define an upper respiratory infection. The important feature, before deciding to postpone or cancel a case, is to have a set of guidelines, as cancellation often poses major problems for the family. The noninfectious nose includes allergic rhinitis, either seasonal or perennial, and vasomotor rhinitis, seen in crying patients. The temperature is normal. Infectious runny noses are viral infections, such as the common cold, flulike syndromes, or laryngotracheitis. Contagious diseases, such as chicken pox, measles, epiglottitis, meningitis, and tonsillitis, are fre-

quent causes. Acute bacterial infections would preclude any form of dental care. How then is the infectious disease to be distinguished? History and examination are significant. If there are any signs or symptoms, then laboratory data should be considered. Primarily, it is important to involve the parent in the decision. If the patient is considered to have a cold, the parent will know. If it is allergic rhinitis, then it is appropriate to proceed with the procedure. Rescheduling is suggested for upper respiratory infections after 2 wk, and for lower respiratory tract infections after 6 wk. These recommendations are somewhat stringent; however, one of the common causes of undiagnosed and unsuspected cardiac arrest is attributable to viral myocarditis. As noted by Tomlin[17] viral myocarditis is a common cause of the rare fatalities which are seen with dental anesthesia. In addition, my own series, reported in *Complications of Dental Sedation and Anesthesia*, noted this potential disease as being significant.[18]

CONCLUSION

It can be stated that respiratory problems are not only the most common complication of dental sedation and anesthesia, but the most easily prevented. Careful evaluation excludes patients who are unsuitable for anesthesia; preparation prevents the occurrence of complications; and the trained and studied operator does not allow complications to become dangerous. No medication, no technique, no equipment can replace the knowledge and skill of the doctor.

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