Chapter 43

Airway Disease

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Introduction

Airway obstruction may be associated with trauma, congenital anatomic abnormalities, aspiration of foreign material, or laryngospasm. Specific management of airway obstruction in the perianesthetic period is determined by the severity of the obstruction and the underlying factors associated with its cause.

Respiratory Depression in the Perianesthetic Period

When possible, the use of potent respiratory-depressant medications should be avoided in patients with respiratory disease. When sedative/analgesics with respiratory-depressant effects (e.g., opioids) are used for premedication, the patient's respiration should be closely monitored (e.g., pulse oximetry, end-tidal CO_2, and arterial blood gas analysis). Mixed agonist-antagonist opioids such as butorphanol minimize respiratory depression. Butorphanol is described as having a "ceiling effect" on respiratory depression and analgesia.[1] This terminology implies that the dose-response curve for butorphanol will plateau at some point, thus imparting no further respiratory depression/analgesia as the dose is increased. Naloxone can be used to antagonize respiratory depression associated with opioid analgesics but will also antagonize analgesia.[2] Analgesia may be preserved to some extent by titrating naloxone "to effect" when reversing opioid-induced respiratory depression. However, *rapid* intravenous administration of naloxone has been associated with development of cardiac dysrhythmias and even sudden death.[3,4] Administration of a mixed–agonist-antagonist opioid such as butorphanol may effectively antagonize severe respiratory depression while maintaining some analgesic action.[5] Incremental dosing of butorphanol (0.05 mg/kg at a time) for antagonism of full-agonist opioids is advised to prevent sudden arousal and dysphoria.

Regional analgesia is gaining in popularity for improving postoperative analgesia. This technique causes less respiratory depression than do parenteral opioids. Following thoracotomy, analgesia may be enhanced by intrapleural injection of bupivacaine (1.5 mg/kg).[6] For control of pain associated with procedures involving the hind limbs (and possibly procedures more rostral), epidural analgesia is a useful adjunct to general anesthesia. Epidural analgesia prior to surgery will decrease general anesthetic requirement.[7] Inhalation anesthetics are associated with dose-dependent respiratory depression. Therefore, use of epidural analgesia as an adjunct to inhalation anesthesia may decrease respiratory and cardiovascular depression. Epidural analgesia in dogs is often accomplished using morphine (0.1 mg/kg) in sterile water or isotonic saline solution in sufficient quantity to yield 1 mL per 5 kg of body weight.[8]

Airway Trauma

Trauma to the head and neck can cause progressive respiratory distress. Airway occlusion may result from collapse or obstruction of the nasal or oral passages accompanied by tissue swelling, hemorrhage, and aspiration of tissues, blood, or foreign materials. Head trauma and secondary cerebral edema can decrease ventilation via neurological mechanisms independent of physical obstructions. Additional respiratory depression must be avoided to prevent associated increases in intracranial pressure and neurological morbidity. In a retrospective study of 85 dogs undergoing cervical spinal decompressive surgery, respiratory arrest was a significant factor in three (42%) of seven that died of complications arising during surgery.[9]

The airway and laryngeal function should be examined during a light plane of general anesthesia or while the animal is sedated. Care must be taken to avoid excessive depression from anesthetics with resultant inhibition of respiration or laryngeal activity. A clinical investigation found that observation of laryngeal function during light anesthesia with propofol or thiopental was superior to anesthesia induced with the combination of diazepam and ketamine.[10]

Surgical procedures on the nasal airway, pharynx, larynx, or trachea can be followed by obstructive postoperative swelling. Delicate surgical technique combined with perioperative anti-inflammatory doses of corticosteroids minimize this potential. Administration of pediatric strength phenylephrine nose drops in each nostril will counteract nasal passage hyperemia, improving ventilation and often stimulating increased swallowing.

Animals with thick or copious secretions in the respiratory tract have increased risk for airway obstruction. Endotracheal intubation restricts the diameter of the trachea and increases the likelihood of airway obstruction from viscid secretions. Parti-

cular attention should be given to patients with small endotracheal tubes (e.g., internal diameter < 5 mm). Decreased compliance detected by difficult positive-pressure ventilation (by squeezing the rebreathing bag) in a patient with abnormal respiratory tract secretions should prompt the anesthetist to inspect the airway. Suction of secretions from the endotracheal tube may be required periodically during anesthesia. In some cases, the best solution may be to reintubate the trachea with a different endotracheal tube. Pharyngeal suction prior to anesthetic recovery from nasal, pharyngeal, or oral surgery decreases risk of aspiration of blood and debris. When regurgitated material or blood has accumulated in the pharynx, the risk of aspiration can be reduced further by gently withdrawing the endotracheal tube with the cuff partially inflated. After delivery to the pharynx, matter can be removed by suction.

Administration of oxygen should continue throughout recovery when there is risk of postoperative airway obstruction or significant respiratory depression. If obstruction develops, supplementary oxygenation increases the time available for institution of airway control. Rapid-sequence induction of anesthesia with an injectable anesthetic may be necessary to regain control of the upper airway via an endotracheal tube. After reestablishment of a secure airway, it may be possible to resolve the underlying problems and then successfully recover the patient from anesthesia. In brachycephalic animals, resection of an elongated soft palate may be necessary to prevent airway obstruction associated with tissue swelling that has developed during the procedure. In severe cases of obstruction, an emergency tracheostomy may be necessary.

Brachycephalic Airway Syndrome

Anatomical abnormalities of the upper airway in brachycephalic dogs can severely compromise their ability to ventilate adequately. The primary defects of stenotic nares, elongated soft palate, and hypoplastic trachea are exacerbated in extreme cases with eversion of laryngeal saccules and redundant pharyngeal tissues. Dogs with arytenoid cartilage, laryngeal, or tracheal collapse present similar anesthetic challenges. Reduction of the cross-sectional area of the trachea greatly increases resistance to airflow and the work of breathing. In addition, vagal tone is frequently high in brachycephalic dogs. Vagal stimulation associated with pharyngeal manipulation (difficult intubation) or vagotonic drugs can contribute to significant bradycardia and further airway narrowing. Preanesthetic administration of anticholinergic agents is indicated in these cases. The anesthetist's goals are to avoid deep sedation, especially in animals that are not continuously monitored; to intubate the trachea by using a rapid intravenous induction technique when practical; and to maintain tracheal intubation until the dog demonstrates adequate recovery from anesthesia. Anesthesia induction should be preceded by having the dog breathe oxygen through a face mask. Respiratory support during anesthesia may be required, especially in overweight animals. Obesity further impairs ventilatory function by decreasing tidal volume and functional residual capacity of the lung.

Recovery from anesthesia is judged to be adequate when a dog strongly objects to the presence of the endotracheal tube. Reversal of the effect of opioids by naloxone administration may aid in rapid return of a dog's ability to maintain its airway. However, painful or distressed animals may experience more respiratory difficulty following extubation, so the benefits of opioid reversal should be carefully weighed against the potential complications of the increased pain and stress that may accompany reversal. Because of the presence of redundant tissue in the pharynx, most brachycephalic breeds (e.g., bulldog and shar-pei) benefit from having the anesthetist hold the dog's tongue and/or extend its neck immediately after extubation of the trachea. A source of 100% oxygen, an endotracheal tube, a laryngoscope, and an intravenous anesthetic-induction drug such as propofol should be immediately available. Ventilatory function of brachycephalic dogs should be closely monitored for at least 1 h following recovery from anesthesia.

Laryngeal Abnormalities

Abnormal laryngeal anatomy due to congenital malformation or acquired disease may affect respiration, especially during sedation. Tracheal intubation may be hindered by masses protruding into the glottis or by strands of tissue that cross the glottis. Smaller-diameter endotracheal tubes may be placed to solve this problem in some instances. However, an excessively narrow endotracheal lumen may severely affect ventilation, leading to hypercapnia or hypoxia.

Laryngospasm during the perianesthetic period occurs most frequently in cats, swine, rabbits, and primates, but has also been observed in dogs and horses. Laryngospasm may occur after irritation of laryngeal tissues by secretions or blood. Spasm of the larynx may be caused by touching the larynx during a light plane of anesthesia. Topical application of a local anesthetic such as lidocaine is recommended prior to tracheal intubation in these species to minimize tactile stimulation–induced laryngospasm. Neuromuscular blocking agents such as atracurium may be useful for preventing laryngospasm during tracheal intubation, although their use in domestic veterinary species is seldom needed. Extubation of the trachea can also trigger laryngospasm in susceptible animals. In one case, a Vietnamese potbellied pig apparently developed laryngospasm 4 h after tracheal extubation in spite of an uneventful recovery from inhalation anesthesia. Treatment of laryngospasm that occurs during tracheal extubation (or later) includes reintubation using anesthetics and neuromuscular blocking agents, if necessary. Nasotracheal intubation is an effective means of reestablishing a patent airway in some species, such as horses. Oxygen should be administered and the patient should be evaluated for subsequent development of pulmonary edema over the next 1 to 4 h. Pulmonary edema may accompany laryngospasm with clinical signs of dyspnea and tachypnea and production of pink frothy material in the airway becoming apparent during recovery from anesthesia.[11] Dogs with upper-airway obstruction can develop life-threatening pulmonary edema.[12] Factors associated with generation of pulmonary edema following airway obstruction are listed in Table 43.1. The extreme negative pressure produced by an animal with airway

Table 43.1. Factors associated with development of pulmonary edema following acute airway obstruction

Severe negative intrathoracic pressure
Decreased interstitial hydrostatic pressure
Catecholamine release
Vasoconstriction
Increased vascular hydrostatic pressure
Hypoxia
Increased permeability of pulmonary vasculature
Net accumulation of interstitial fluid
Inadequate lymphatic removal of interstitial fluid

obstruction when attempting to inspire causes decreased interstitial hydrostatic pressure in the lung. Simultaneous release of catecholamines causing increased vascular hydrostatic pressure may cause a net accumulation of interstitial fluid. Hypoxia associated with an acutely obstructed airway may promote fluid movement into the interstitial spaces by increasing permeability of pulmonary vasculature. Pulmonary edema formation after airway obstruction is probably multifactorial, so treatment is symptomatic. Oxygen supplementation to maintain an SpO_2 greater than 90%, diuretics, and corticosteroids are used to treat pulmonary edema. Emergency tracheostomy may be required to establish a patent airway rapidly. Tracheal intubation and positive-pressure ventilation are routinely used in human patients with pulmonary edema, for whom the syndrome is rarely fatal.[13,14]

Horses may develop airway obstruction during tracheal extubation after anesthesia. Potential causes include laryngospasm, paralysis of the arytenoid cartilages, and mechanical obstruction. Epiglottic retroversion has been described as a cause of upper-airway obstruction in horses.[15] Syncope as a consequence of complete upper-airway obstruction caused by a subepiglottic cyst has also been reported.[16] Laryngospasm is associated with cessation of airflow, whereas paralysis of the arytenoid cartilages is characterized by stridor and decreased airflow. Laryngospasm may result from laryngeal irritation during anesthesia and/or extubation. Paralysis of the arytenoid cartilages has been associated with poor function of the recurrent laryngeal nerve. In horses, the recurrent laryngeal nerve may be susceptible to damage by hyperextension of the neck for a prolonged period during anesthesia.[17] Postanesthetic paralysis of the arytenoid cartilages has also been observed following 2- to 4-h procedures in which a horse's trachea was intubated with an excessively large (in retrospect) endotracheal tube. Mechanical obstruction of the airway may occur during recovery from anesthesia if a horse becomes cast or positioned such that the neck is improperly flexed. Pulmonary edema following transient mechanical airway obstruction in a horse has been reported.[18] Horses anesthetized with the head lower than the body may be at risk for accumulation of fluid in laryngeal tissues. These horses may develop airway obstruction by an edematous glottis after tracheal extubation. Many anesthetists prefer to recover horses at risk for upper-airway obstruction with a nasotracheal tube in place until the horse is standing. Treatment of a horse with postanesthetic airway obstruction is initiated by placement of a nasal or oral endotracheal tube. If one of these techniques is not successful, an emergency tracheostomy is indicated. Supportive treatment includes administration of oxygen, diuretics, corticosteroids, antibiotics, and analgesics.

Mishaps Involving the Airway

Unfortunately, mishaps or accidents involving management of the airway during anesthesia increase morbidity and mortality among animal patients. It is therefore imperative that anesthetists become familiar with the anesthetic machine, ventilator, and intubating equipment available to prevent such mishaps. Vigilance in preventing excessive pressure buildup within an anesthetic circuit is required. The function of the "pop-off" valve should be continuously monitored. Sudden or unexplained increases in circuit or airway pressure should be immediately investigated. Equipment failure should be ruled out while anesthesia is maintained using a different delivery system. An unusual cause of increased inspiratory plateau pressure has been attributed to a leak in the bellows of a mechanical ventilator that allowed the driving gas of the bellows to enter the breathing circuit.[19] Increased airway pressure may also occur from development of tension pneumothorax.

Venipuncture has also been associated with airway obstruction caused by iatrogenic trauma to vital structures in the neck. Injury of the recurrent laryngeal nerve may cause temporary paralysis of the arytenoid cartilage in horses. Aspiration pneumonia secondary to choke caused by a periesophageal hematoma following jugular venipuncture has been reported in a llama.[20]

Airway obstruction has occurred in intubated patients following administration of nitrous oxide, which may diffuse into the air-filled endotracheal tube cuff, causing increased cuff pressure and endotracheal tube collapse.[21] Overexpansion of the endotracheal tube cuff when administering nitrous oxide may be prevented by filling the cuff with an appropriate nitrous oxide–oxygen mixture (i.e., gas aspirated from the breathing circuit) rather than with air. Nitrous oxide may also diffuse into small bubbles of air found in some endotracheal tubes as a manufacturing defect. Expansion of these air bubbles with nitrous oxide has caused airway obstruction in guarded endotracheal tubes (those with a spiral wire used to prevent kinking of the tube).[22] Prior to use, endotracheal tubes should be examined for material defects such as presence of air bubbles that contraindicate administration of nitrous oxide.

Inadvertent displacement of the endotracheal tube may cause airway obstruction. Subtle changes in position of the endotracheal tube may occur during radiography or surgical positioning. Flexion of the neck of anesthetized dogs has resulted in caudal displacement, endobronchial placement, or total occlusion of the endotracheal tube.[23]

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