Chapter 53

Anesthetic Emergencies and Procedures

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Introduction

In many veterinary practices, after the induction of anesthesia, no one is assigned the task of anesthetist to monitor anesthesia and be vigilant for untoward events that might result in accidental morbidity and mortality. As with most unwanted events, the anticipation of possible complications and having a plan of action already prepared will facilitate successful resolution of the problem. Since the onset of general anesthesia upsets the physiological equilibrium of patients and can bring them closer to harmful outcomes, preparation to manage these problems is even more critical. Monitors that display vital parameters such as oxygen saturation of hemoglobin, end-tidal carbon dioxide, blood pressure, and heart rhythm are available to facilitate early detection of critical events such as bradycardia, changes in oxygen availability, and hypoventilation. Veterinarians who vigilantly monitor have a better opportunity to respond quickly to a harmful trend before a disaster occurs.

Anesthetic Risk

The risk of death from disease or related surgery is usually greater than the risk of death from anesthesia. However, anesthesia involves the controlled administration of potentially toxic drugs and thus carries a risk of organ dysfunction and damage, delayed recovery, and death. Mistakes are not necessarily reversible, and death can occur suddenly and often without warning when patients are not appropriately monitored. The goal of anesthetists should be to manage the risks associated with anesthesia and the perioperative period, affording patients the best chance of a successful outcome. *Risk management* is a term developed by the insurance industry and adopted by the health care industry to describe processes used to prevent injury, litigation, and financial loss.[1,2] The real aim of this process is to use analysis of adverse events to prevent similar injuries to subsequent patients. Risk management starts with an unbiased and nonjudgmental review and analysis of all "critical events" causing real or potential patient harm. The next step is formation or modification of standard operating procedures. For example, in aviation, accident investigation begins with discovery of the facts by an independent board (National Transportation Safety Board) and then analysis and publication of the findings. There is also an anonymous reporting system (Aviation Safety Reporting System) that involves the documentation and analysis of events that were considered hazardous by the participants but did not lead to an accident. These aviation review procedures provide a model for the improvement of anesthesia safety in both human and veterinary medicine. A commitment to the highest-quality patient care will ultimately lead to the routine performance of such analyses by medical providers.

Species-Related Risk

Advances in medical technology and pharmacology, as well as the increase in training of anesthesiologists, veterinarians, and licensed technicians, have done much to decrease the inherent risks associated with anesthesia. The risk of anesthetic-related death in people is estimated at between 1:10,000 and 1:200,000.[3–5] The rate of anesthetic-related death among dogs and cats anesthetized in private practice has been assessed at 0.1%.[6] Horses present an inherently greater challenge during anesthesia because of unique anatomical, physiological, and behavioral factors. Several studies suggest an anesthetic-related mortality rate of between 0.08% and 0.9% in healthy horses.[7,8] Postanesthetic lameness caused by myopathies and neuropathies reportedly occurs in 6.4% of anesthetized horses.[9] Emergency cases, including colics, are associated with an apparent mortality rate of 31.4%.[10] Another retrospective analysis of cases from a single facility using a fairly standardized anesthetic protocol found an incidence of anesthetic-related mortality of 0.12% (L.

Bidwell, personal communication, 2004). In this study of 21 deaths from among 17,961 equine anesthetics, the incidence of cardiac arrest was 0.06% (10 deaths), that of long-bone fractures was 0.04% (8 deaths), and that of myoneuropathies 0.02% (3 deaths). When interpreting studies of comparative anesthetic-related morbidity and mortality, it should be remembered the definitions of the anesthetic period may vary and often include additional surgical and disease risk factors.

High-Risk Patients

Based on clinical experience, the small animal patients that are associated with a high risk of adverse outcome from anesthesia and surgery include geriatric (especially hyperthyroid) cats; post-trauma cases with pulmonary pathology, hemothorax, or pneumothorax or pulmonary hemorrhage; and cases of acute head trauma and severe intra-abdominal hemorrhage. Patients requiring a high level of care and commitment to achieve a good outcome include neonates; those with low body weight or morbid obesity; and patients undergoing portosystemic shunt occlusion or cardiac, intracranial, or intraocular surgery.

The procedures or conditions associated with high risk of adverse outcome in equine patients include advanced age, heavily muscled young horses, extreme emaciation, ethmoidal hematoma, or guttural pouch mycosis with severe hemorrhage, septic shock, and intra-abdominal hemorrhage. Periparturient mares also have a greater risk of adverse outcome.[11,12]

Cardiovascular Emergencies

Hemorrhage and Fluid Loss

Blood loss during surgery may be insidious or obvious. Body fluids may also be lost during surgery to transudation, sequestration, or evaporation. Extravasation of fluid to a nonfunctioning or sequestered edema space is commonly referred to as *loss to the third space*, the first and second spaces being the intracellular and extracellular spaces. These losses may reduce circulating blood volume significantly. Regardless of cause or route of loss, a decrease in circulating blood volume is not well tolerated by anesthetized patients.

Quantifying blood loss is important but can be difficult, so the severity of hemorrhage is often assessed by its impact on the patient. Severe blood loss causes tachycardia, reduced arterial pressure, pale mucous membranes, decreased pulse pressure, and decreased area under the arterial pulse wave.[13,14] Packed cell volume decreases only during resuscitation or fluid shift into the vascular space, but base deficit increases as changes in bicarbonate, and venous pH correlate with blood volume lost.[15] All of the aforementioned changes have been reported in anesthetized horses, with one difference: Tachycardia in response to blood loss is not usually observed in horses that are under anesthesia.[16] Physiological responses to blood loss may be blunted or masked by anesthetic and anesthetic adjunctive drugs (e.g., α_2-agonists), further emphasizing the need for appropriate monitoring for early detection and correction of hypovolemia.

Shed blood can be replaced with crystalloids, colloids such as plasma, hemoglobin-based oxygen-carrying solutions, dextrans, whole blood, or a combination of these solutions (Table 53.1). In most situations, hypertonic solutions do not seem to have a distinct advantage over isotonic crystalloid solutions.[17] Crystalloid solutions such as lactated Ringer's or Plasmalyte are usually administered at threefold the volume of shed blood, as a rough guideline for resuscitation. The main advantage of crystalloid solutions is their low cost. Colloid solutions such as whole blood, plasma, hydroxyethyl starch, and hemoglobin-based oxygen carriers can be used as a substitute for crystalloids.[18] Hemoglobin-based oxygen-carrying solutions (e.g., Oxyglobin) are relatively

Table 53.1. Management of complications associated with anesthesia.

Complication	Treatment	Trade Name	Dosage	Side Effects
Excitement, delirium	Acepromazine	PromAce	0.05–0.2 mg/kg IV, IM	Prolonged recovery
	Medetomidine	Domitor	1–2 µg/kg IV	Bradycardia
	Diazepam	Valium	0.25–0.5 mg/kg IV	Hypothermia
	Midazolam	Versed	0.05–0.2 mg/kg IV, IM	
Hypoventilation	Oxygen	—	—	Respiratory depression
	Ventilation	—	—	Additional hypoventilation if too aggressive ventilation
				Resisting mask
Laryngospasm	Topical lidocaine	Xylocaine 2%	—	
	Lidocaine jelly	—		
	Lidocaine IV		1–2 mg/kg IV	
Hypoxemia	Oxygen		—	
	Tracheostomy	Portex[a]		Subcutaneous emphysema
	Ventilation			Hyperventilation
Pneumothorax	Oxygen			
	Chest tubes	Sherwood[b]	—	Infection
	Thora-Seal III			
	Ventilation			Hyperventilation

(*continued*)

Table 53.1. Management of complications associated with anesthesia (*continued*).

Complication	Treatment	Trade Name	Dosage	Side Effects
Cardiac dysrhythmias				
Tachycardia	LRS	—	10–20 mL/kg per hour	Bradycardia
	Esmolol	Brevibloc	0.01–0.1 mg/kg IV	Bradycardia
	Propranolol	Inderal	0.05–0.1 mg/kg IV	Hypotension
	Increase anesthesia	—	—	Bradycardia
Bradycardia	Atropine	—	0.02 mg/kg IV	Tachycardia
	Glycopyrrolate	Robinul V	0.005 mg/kg IV	
Ventricular dysrhythmias	Lidocaine	Xylocaine	Dogs: 0.5 mg/kg IV	Bradycardia
			Cats: 0.2 mg/kg IV	Convulsions
	Procainamide	Pronestyl	10–20 mg/kg IM	Hypotension
			10–20 mg/kg IV per hour	
	Amiodarone	Cordarone	5 mg/kg IV	Liver toxicity
				Hypothyroidism
Hypotension	Fluids (LRS)	—	10–20 mL/kg IV	
	Dopamine	Intropin	3–5 µg/kg per minute	Dysrhythmias
	Dobutamine	Dobutrex	3–5 µg/kg per minute	Tachycardia
				Hypertension
Blood or fluid loss	Fluids (LRS)		40–90 mL/kg IV per hour	Pulmonary edema
	Blood		20–40 mL/kg IV	Allergic reaction
	Hydroxyethyl starch 6%	Hetastarch	10–20 mL/kg IV per day	Circulatory overload
	Hemoglobin glutamer 200	Oxyglobin	10–30 mL/kg IV	Circulatory overload
Hypothermia	Warmed fluids		5–10 mL/kg IV per hour	Overhydration
	Water-heating pad	Gaymar[d]		
	Forced air warming	Bair Hugger[c]		Hyperthermia
Hypoglycemia	Dextrose 5%	—	1–2 mL/kg IV	Hyperosmolality
Metabolic acidosis	Sodium bicarbonate	—	1–2 mEq/kg IV every 10 min	Metabolic alkalosis
				Hypokalemia
				Hyperosmolality
Hyperkalemia	Sodium bicarbonate	—	0.5–1.0 mEq/kg IV	As above
	Sodium chloride 0.9%	—	10–40 mL/kg per hour	
	Calcium chloride	—	10 mg/kg IV	Tachycardia
Hyperpyrexia	Oxygen			
	Fluids (LRS)		5–10 mL/kg IV	
	Tranquilizers	PromAce	0.05–0.1 mg/kg IM	
	Dantrolene sodium		2–4 mg/kg IV	
Prolonged recovery	Doxapram	Dopram V	1–2 mg/kg IV	Excitement
	Yohimbine	Yobine	0.5 mg/kg IV	
Postoperative pain	Morphine sulfate		0.1–1.0 mg/kg IM	Respiratory depression
	Buprenorphine	Buprenex	0.01 mg/kg IV, IM	Slow recovery
	Butorphanol	Torbugesic	0.2–0.4 mg/kg IM	Slow recovery
	Meloxicam	Metacam	0.2 mg/kg SC	Vomiting

IM, intramuscularly; IV, intravenously; LRS, lactated Ringer's solution; SC, subcutaneously.
[a]Shiley, Irvine, CA.
[b]Sherwood Medical, St. Louis, MO.
[c]Arizant Healthcare, Eden Prairie, MN.
[d]Gaymar, Orchard Park, NY.

expensive but have a long shelf life, and do not require cross-matching.[19] The use of colloids has the advantage of sustaining colloid osmotic pressure while preserving plasma volume but has the disadvantage of being more expensive than crystalloid solutions.

Acute hemorrhage of greater than 20% of the blood volume or a decline in pack cell volume to less than 20% because of the combined effects of blood loss and crystalloid fluid administration can be treated with an appropriate mass of red blood cells by either transfusion of whole blood, packed red cells, or 10 to 30 mL/kg of a hemoglobin-based oxygen carrier.[20,21] Hemoglobin-based oxygen-carrying solutions or red blood cells are preferred because of the need for restoring adequate hemoglobin concentrations to carry oxygen to the tissues. Smaller amounts of surgi-

cal hemorrhage, not associated with severe decreases in the hemoglobin concentration, can be replaced with crystalloids (e.g., lactated Ringer's) or colloids rather than blood.

Cardiac Dysrhythmia

Most dysrhythmias are caused by preexisting medical conditions, administration of premedications, anesthesia induction and maintenance agents, and surgical stimulation. Dysrhythmias require treatment if they reduce cardiac output, cause sustained tachycardia, or are likely to initiate dangerous ventricular dysrhythmias.

Canine gastric dilation/volvulus or multiple trauma often precipitates dysrhythmias that may require treatment prior to induction of anesthesia.[22,23] Dysrhythmias following gastric dilation/volvulus presumably have their origin in acid-base imbalance, electrolyte disturbance, myocardial ischemia, circulating cardiac stimulatory substances, and/or autonomic nervous system imbalance. Treatment involves correcting physiological abnormalities and administering lidocaine or procainamide. It is absolutely imperative that ventricular premature contractions (VPCs) be differentiated from ventricular escape beats before administration of antiarrhythmic drugs, because suppression of an escape rhythm can cause immediate asystole and death. If the sinus rate is low, an intravenous atropine injection of 0.02 mg/kg may increase the sinus rate and invoke overdrive suppression, which may inhibit the dysrhythmia. VPCs and ventricular tachycardia resulting from a traumatized myocardium are commonly treated with lidocaine or procainamide. If possible, surgery should be delayed 2 to 4 days or until the dysrhythmias have subsided.

Several of the popular drugs used as preanesthetic medication can predispose patients to conduction abnormalities. Atropine or glycopyrrolate can cause sinus tachycardia and increase myocardial work and oxygen consumption. Phenothiazine tranquilizers reportedly predispose the heart to sinus bradycardia, sinus arrest, and, occasionally, first-degree and second-degree heart block, although it has also been shown to protect against VPCs. Xylazine causes bradycardia and second-degree atrioventricular blockade and decreases the epinephrine threshold for VPCs. The μ-receptor agonist opioids morphine, hydromorphone, fentanyl, and oxymorphone will also precipitate a slowing of heart rate via increased vagal efferent activity. The anesthesia induction agents thiopental and ketamine have been reported to increase the likelihood of dysrhythmia formation after epinephrine administration during halothane anesthesia.[24,25] This multidrug interaction has also been described for thiopental and isoflurane.[24]

Other factors responsible for the development of the dysrhythmias during the surgical period include altered arterial carbon dioxide partial pressure ($PaCO_2$), altered PaO_2, altered pH, and autonomic reflexes from surgical manipulation, as well as central nervous system disturbances and cardiac disease. Because most perioperative dysrhythmias do not seriously affect cardiac output, treatment can be discrete. Changing to a different inhalation anesthetic, using intermittent positive-pressure ventilation, or increasing the depth of anesthesia may eliminate the dysrhythmia.[26,27] Other treatments for controlling ventricular dysrhythmias include correcting blood-gas abnormalities or administering a small quantity of intravenous lidocaine (0.5 mg/kg) or procainamide (1.0 mg/kg).

Allergic Reactions

Allergic reactions involving anesthetics are uncommon but could occur after sensitization to a drug. Allergic or anaphylactic reactions are mediated by the immune system. They are more commonly associated with repeated exposure to an allergen, but cross-reactivity may be seen with some preexisting allergies (e.g., allergies to eggs and to egg proteins in propofol). Anaphylactic reactions following thiopental administration have been reported.[28,29] Intravenous injection of the intravenous contrast agent diatrizoic acid (Hypaque; Amersham Health, Princeton, NJ) has caused tachypnea, bronchoconstriction, and mucoid diarrhea in dogs. Allergic reactions are treated with intravenous fluids, antihistamines, and corticosteroids. Epinephrine should be administered in severe reactions accompanied by severe bronchoconstriction or cardiovascular collapse. Many unexpected responses to anesthetic and anesthetic adjunctive drugs have been labeled as "allergies" by veterinarians; however, proper diagnosis is crucial because it may have serious ramifications for future anesthetic delivery.

Cardiac Arrest

Successful treatment of cardiac arrest requires early diagnosis. The brain is the organ most susceptible to hypoxia or ischemia, because serious brain injury develops after only 4 or 5 min of cardiac arrest. The brain injury can be multifactorial, including the rapid loss of high-energy phosphate compounds during ischemia, cell structural damage during reperfusion, progressive brain hypoperfusion especially in certain areas, and suppression of protein synthesis in selectively vulnerable neurons.[30] Once the diagnosis of cardiac arrest has been confirmed, all efforts must be toward developing effective blood flow and reestablishing a heartbeat. Cardiopulmonary resuscitation (CPR) with external cardiac massage appears to be ineffective in protecting the brain from injury and should be only part of the initial resuscitation protocol. If unsuccessful, time should not be wasted with external CPR in lieu of more effective internal techniques.[31]

Cardiac arrest is diagnosed when some or all the signs listed in Table 53.2 are present. When the heartbeat or peripheral pulse cannot be palpated, the systolic blood pressure is generally less than 50 mm Hg. In this circumstance, the heart may actually have a weak beat, but cardiac output is probably very low and true cardiac arrest imminent. A nonpalpable weak heartbeat along with a regular rhythm has been termed *pulseless electrical activity* (PEA), formerly known as electrical mechanical dissociation. This type of functional cardiac arrest occurs with anesthesia overdose and from many other causes, such as hypovolemia, acute cardiogenic decompensation, severe acidosis, or hypoxemia. It is important to look for correctable causes of PEA during the first moments of resuscitation to improve the odds of success. Other forms of cardiac arrest include asystole and ventricular fibrillation. The three types of cardiac arrest can be

Table 53.2. Signs of cardiac arrest.

1. No palpable heart beat
2. No palpable pulse
3. Apnea
4. Lack of surgical hemorrhage
5. Cyanosis
6. No muscle tone
7. Dilated pupils (later)

differentiated with an electrocardiogram (ECG) or by direct observation of the heart during thoracic surgery or internal CPR.

Cardiopulmonary Resuscitation

When any or all of the signs listed in Table 53.2 are present, the traditional *ABCD* protocol for treatment of cardiac arrest must be started immediately. *A* refers to *airway* and reminds the resuscitator that a patent airway is a necessity. Endotracheal intubation is the best method of insuring a patent airway. The goal of *B*, *breathing*, is to supply high concentrations of oxygen to the alveoli and to eliminate carbon dioxide. Intermittent positive-pressure ventilation is usually instituted in intubated patients, although, when breathing room air and using chest compressions only (no artificial ventilation), dogs have maintained adequate gas exchange and oxygen saturation greater than 90% for longer than 4 min.[32] The real value of artificial breathing has been questioned for routine resuscitation in people.[33] The current recommendations for a breathing rate of 10 to 24 breaths/min may be too high.[34] Assuming there is enough blood flow to provide a reading, the pulse oximeter can be useful as a guide to determine respiratory rate. Simply ventilate at a rate that maintains hemoglobin saturation at 90% or higher.

C refers to *cardiac massage*, which can be either external (thoracic) or internal. External thoracic massage is thought to produce cardiac output by one or a combination of two methods. The *thoracic pump theory* holds that blood moves out of the thoracic cavity during the compression half of the CPR cycle because of a buildup of internal thoracic pressure (Fig. 53.1). This mechanism is thought to occur primarily in animals with a body weight greater than 15 to 20 kg. Evidence for the thoracic pump theory includes the phenomenon of cough CPR in humans and artificial cough CPR in dogs.[35,36] The *cardiac pump theory* explains blood flow in smaller animals or animals with a narrow side-to-side thoracic width and refers to actual mechanical compression of the myocardium by the thoracic wall during CPR systole (Fig. 53.2). Blood flow in some patients may be produced by a combination of the cardiac and thoracic pump mechanisms. Whatever the reason for forward blood flow, it appears that external thoracic massage is not very protective of the brain, because CPR performed for more than 3 or 4 min is often associated with significant neurological injury.[37] Because traditional external thoracic massage is apparently ineffective in many patients, various maneuvers have been proposed to improve blood flow during CPR. For example, *interposed abdominal compression* (IAC)[38] involves manually compressing the abdomen in counterpoint to the rhythm of the chest compression. The physiological reason for improvement of blood flow is that compression of the abdominal aorta responds like an intra-aortic balloon pump and that pressure on the abdominal veins primes the right heart and pulmonary vasculature in preparation for the next thoracic compression.[39] This method of augmenting external CPR has been asso-

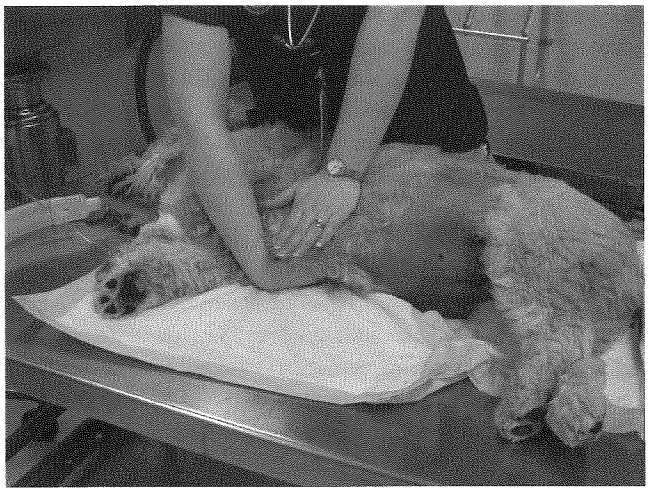

Fig. 53.1. External thoracic massage administered to a larger dog that probably derives blood flow primarily from the thoracic pump mechanism. The resuscitator, standing at the dog's back, is applying thoracic compressions over interspace 4 or 5 at the level of the costochondral junction. In larger dogs, the thoracic compressions may not mechanically contact the heart, so all blood flow is derived from increased intrathoracic pressure. The right hand is supplying a counterforce for thoracic compressions with the palm of the left hand. The compression rate for this dog should be from 80 to 100 beats/min.

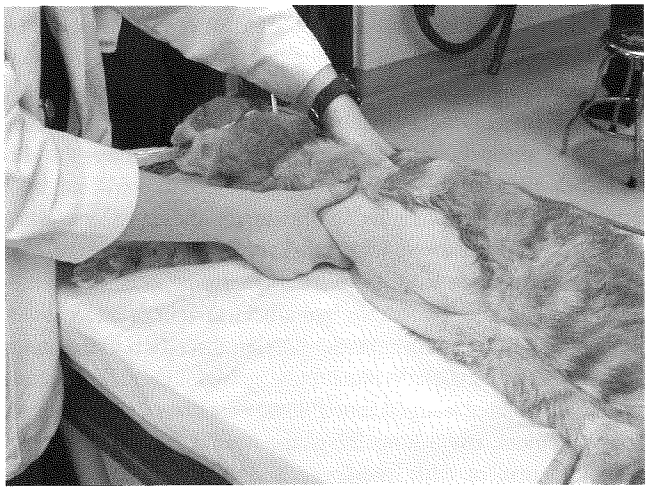

Fig. 53.2. External thoracic massage administered to a cat with blood flow derived from the cardiac pump. The thoracic walls contact the heart with each compression. Note that only the thumb and fingers of the right hand compress the thorax, while the left hand stabilizes the cat. The compression rate should be from 100 to 120 beats/min.

ciated with improved survival in people and vital organ perfusion in dogs.[40,41] Utilization of IAC-CPR in over 100 dog CPR labs as part of a clinical anesthesia rotation demonstrated that venous return and arterial blood pressure improved for about 1 min, after which hemodynamics began to fail again (A. T. Evans, personal observation). Another way of improving blood flow during CPR is to simultaneously ventilate at the time of thoracic compression. Simultaneous ventilation-compression (SVC) CPR has improved carotid blood flow during resuscitation of animals.[42] Opposing evidence has also been presented that shows that the mitral valve of dogs may actually close in response to rhythmic increases in intrathoracic pressure.[43] Despite this evidence to the contrary, SVC-CPR probably improves blood flow during CPR of large dogs when the thoracic pump is the primary mechanism in generating blood flow.

Open thoracic or internal CPR is more effective at perfusing the heart and brain during the critical beginning minutes of CPR.[44–46] Higher blood pressure and cardiac output can be achieved with internal CPR. Most veterinary practices are well equipped to perform internal CPR because controlled ventilation and thoracotomy can be performed. The limiting factor in its employment is often the surgical inexperience of the attending veterinarian or the "do not resuscitate" wishes of the animal owner. Although it can be a difficult subject to broach, it is desirable to ascertain prior to the procedure the owner's wishes concerning CPR, in writing, in the event that cardiac arrest should occur during anesthesia and surgery. Valuable time may be lost trying to contact owners. Whichever method of CPR is chosen, there are some guidelines for CPR technique that, when followed, can improve success. The animal should be in right lateral recumbency with the resuscitator standing at its back (Fig. 53.1). The thoracic or cardiac compression rate should be 80 (large dogs) to 120 (cats) per minute.[47,48] A longer compression time will augment forward blood flow when using the thoracic pump mechanism.

The recommendations for *D, definitive* or *drug therapy*, start with the immediate use of epinephrine. Epinephrine should be administered early, preferably into a central vein or alternatively into a peripheral vein, intrabronchially, or directly into the chamber of the left ventricle.[49] For intrabronchial administration, use a flexible catheter wedged into a distal bronchus.[50] For intracardiac placement, use a long, 22-gauge needle inserted at the left thoracic fourth or fifth interspace and costochondral junction. For intravenous administration, a dose of 0.05 to 0.1 mg/kg is used, whereas bronchial administration requires 0.05 to 0.1 mg/kg diluted to a 2- to 3-mL volume with saline. The dose for intracardiac epinephrine is 0.025 to 0.05 mg/kg. Even though intracardiac epinephrine seems appealing as a way of efficiently delivering the drug to the heart, the technical difficulty of positioning the needle in the chamber of the left ventricle when the heart cannot be palpated, along with the potential for myocardial or coronary vascular injury, makes this technique the least advantageous. Since the goal of CPR is to revive the heart as soon as possible, early administration of epinephrine is crucial, and it should be given immediately after diagnosis of cardiac arrest.

The use of vasopressin in asystolic cardiac arrest has been recommended as a new standard of care in people.[51] The interest in vasopressin as treatment for cardiac arrest was due to an observation in the early 1990s that endogenous vasopressin levels were greater in survivors of cardiac arrest than in patients that died.[52] The resuscitation success from the injection of vasopressin compared with epinephrine may be because the heart continues to consume oxygen after epinephrine injection (especially with tachycardia that often follows successful epinephrine-assisted resuscitation), whereas vasopressin augments coronary blood flow through an increase in systemic vascular resistance and increased diastolic perfusion pressure without an accompanying tachycardia.[53,54] In people, epinephrine may be potentially detrimental in early asystolic cardiac arrest because exogenous epinephrine could be expected to potentiate hypoxemia and advancing acidosis, which could further impair the pressor effects of epinephrine.[51] Tracheal administration of vasopressin (1.2 units/kg) in anesthetized dogs has resulted in systolic, diastolic, and mean blood pressure increases that last longer than 1 h.[55] Although research into the effects of vasopressin in treating cardiac arrest in dogs is scarce, an intravenous dose of 0.8 units/kg has been suggested for treatment of shock-refractory ventricular fibrillation, pulseless ventricular tachycardia, asystole, and PEA.[56]

Lidocaine is used after resuscitation if ventricular dysrhythmias are compromising cardiac output. The use of lidocaine during ventricular fibrillation to improve the results of electrical defibrillation is being reevaluated.[57] Lidocaine is usually given as an intravenous bolus at a dose of 0.5 mg/kg. Amiodarone has also been recommended for shock-refractory ventricular tachycardia or fibrillation in people.[58] Because of its vasodilatory effects on the coronary circulation, amiodarone (5 mg/kg intravenously) is best administered in combination with epinephrine.[59] Metabolic acidosis from hypoxia and ischemia, and respiratory alkalosis caused by iatrogenic hyperventilation during treatment of cardiac arrest, commonly occur during resuscitation.[60] The immediate use of bicarbonate is controversial, because metabolic acidosis is slow to develop during CPR and is somewhat neutralized by an ensuing respiratory alkalosis. Respiratory alkalosis is caused by external thoracic compression and controlled ventilation during CPR. Sodium bicarbonate (1 mEq/kg) administered after 10 min of resuscitation will improve the chance of return of spontaneous circulation and may play a role in mitigating postresuscitation cerebral acidosis.[61,62] However, bicarbonate administration can result in production of carbon dioxide as metabolic acid is neutralized. Careful monitoring of $PaCO_2$ can be a guide to adequate ventilation postresuscitation to avoid paradoxical cerebral acidosis.

Atropine or glycopyrrolate are important drugs to administer during CPR because reflex bradycardia may have contributed to the initial cardiac arrest. In addition, bradycardia often occurs after a heartbeat has been established. Atropine at 0.02 to 0.04 mg/kg or glycopyrrolate at a dose of 0.01 mg/kg intravenously will enhance the automaticity and conduction of both sinoatrial and atrioventricular nodes.[63]

In dogs and cats, pulseless electrical activity is apparently more common than ventricular fibrillation.[64] Asystole, observed as a flatline ECG, is the next most common form of cardiac arrest, with ventricular fibrillation the least common. It is fortu-

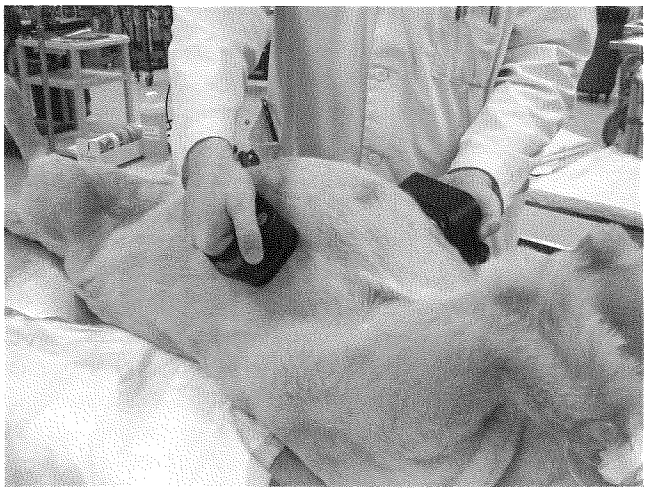

Fig. 53.3. Placement of direct-current paddles for defibrillation of the heart during cardiac arrest. An area under the paddles has been shaved and electrode jelly applied to the paddles. Administer a shock of approximately 3 to 5 joules (watts/second) per kilogram of body weight. Sequential discharges of increasing energy of 50% at each shock may be more effective at converting fibrillation.

itous that ventricular fibrillation is the least common expression of cardiac arrest, because most veterinary practices do not have access to a direct-current defibrillator. If a direct-current defibrillator is available, clip the hair from a small area from each side of the thorax. After applying electrode gel to each paddle, firmly apply the paddles to the thorax (Fig. 53.3) and administer a shock of approximately 3 to 5 joules (watts/seconds) per kilogram of body weight. Sequential discharges of increasing energy may be more effective at converting fibrillation.[56] Internal defibrillation requires a smaller electrical discharge: a total of 10 to 50 joules. Alcohol should not be used for ECG lead placement during CPR because alcohol is highly flammable and may be ignited by a defibrillator.

Internal Cardiopulmonary Resuscitation

After administration of epinephrine, and after attention to airway (A) and breathing (B) of the CPR protocol, begin external thoracic massage. It seems reasonable to start external CPR even though success rates are low with this method. Some animals respond positively to one or two doses of epinephrine and 1 or 2 min of external CPR. These appear to be primarily animals in PEA or asystole. If there is no response after 2 min, one should quickly begin the more productive internal CPR technique.[65] Unfortunately, many practitioners may not feel confident about performing a thoracotomy when they have little or no previous experience with this procedure. There is little to lose, however, when a patient is in cardiac arrest and has not responded to initial resuscitation attempts. Emergency thoracotomy can be accomplished quickly in an arrested animal. Clip the hair from the left thorax at the fifth interspace. Spray or wipe the area with an antiseptic solution and incise the skin starting 1 inch from the spine to within 1 inch of the sternum. With surgical scissors, continue incision through the various tissue layers, avoiding the internal thoracic artery near the sternum. Bluntly penetrate the pleura, extend the incision, and spread the ribs. If the abdomen is open during surgery, a transdiaphragmatic approach has been used (especially during diaphragmatic hernia surgery) to reach the heart in a timely manner. Reach into the thorax and begin cardiac massage at a rate of 80 to 120 compressions per minute. Depending on its size, the heart can be massaged with fingers, one hand, or two hands.[66] Epinephrine can now be easily administered into the left ventricle as required. If the resuscitation is successful and mental alertness improves, the patient can be anesthetized to complete closure of the thoracic incision. The thorax should be flushed with warm, sterile physiological saline and closed in a routine manner. Infection is rare after emergency thoracotomy in people and, from clinical experience, uncommon in dogs.[67,68] An algorithm for patients with confirmed cardiac arrest is presented in Fig. 53.4.

Postresuscitation Care

Once there is a return of spontaneous circulation, attention must be directed toward limiting the neurological injury and other sequelae produced by the cardiac arrest. Intensive care must be provided to address blood-gas abnormalities, respiratory insufficiency, hypotension, cardiac dysrhythmias, and temperature. Clinical trials in people have demonstrated neurological benefit of mild therapeutic hypothermia (32°C to 34°C) in survivors of out-of-hospital cardiac arrest.[69] Mild hypothermia should be instituted as soon as possible after resuscitation and maintained for at least 12 h. Cooling methods that may work in smaller animals involve surface cooling of the head and neck, as well as circulation of cool air over the patient's body. Tympanic membrane temperature can be used as a proxy for brain temperature. Application of mild hypothermia may improve the rather dismal success rate of cardiac and brain resuscitation in animals.

After successful CPR, the use of intravenous antibiotics has been recommended to counter the potential septicemia that can follow ischemic insult of the integrity of the lining of the gastrointestinal tract. Administration of osmotic diuretics such as mannitol (0.5 to 1.0 g/kg intravenously) after resuscitation has also been recommended to counter cerebral edema secondary to ischemia.

Perivascular Injection

Among all of the injectable anesthetics in use today, the perivascular injection of thiopental has likely caused more local tissue damage than all other anesthetics put together, primarily because of its very alkaline pH. It is, however, unusual for a perivascular slough to occur if the concentration of thiopental is 2.5% or less. If thiopental is inadvertently injected, perivascular treatment should consist of infiltration of the area with saline to dilute the thiopental, lidocaine to vasodilate capillaries and increase absorption, and corticosteroids to decrease the inflammatory response. Propofol, ketamine, and etomidate normally do not cause tissue sloughing if accidentally injected perivascularly. In horses or cattle, glycerol guiacolate is irritating and will likely cause a tissue

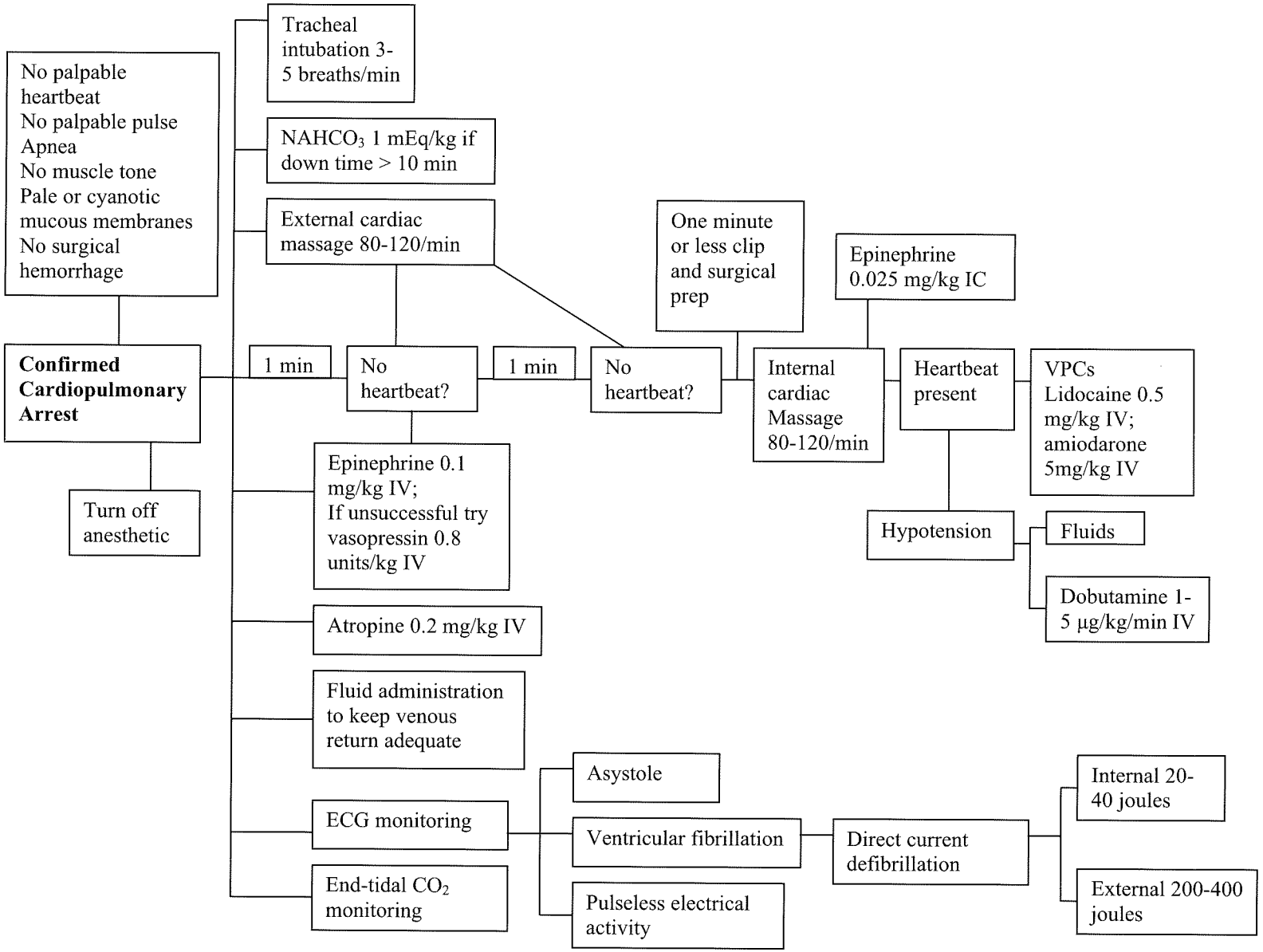

Fig. 53.4. An algorithm for cardiopulmonary resuscitation (CPR). This simplified protocol for CPR is used to resuscitate animals that might be able to survive cardiac arrest. Because early restoration of brain perfusion is the most important goal, a quick decision for internal cardiac massage is required. ECG, electrocardiogram; IC, intracardiac; IV, intravenously; $NaHCO_3$, sodium bicarbonate; and VPCs, ventricular premature contractions.

slough if a large volume is injected or infused outside the intended vessel. Many catecholamine solutions including dopamine can lead to tissue necrosis after perivascular injection. Intense α_1-receptor–mediated vasoconstriction is likely the cause.

Respiratory Insufficiency

Respiratory depression is defined by an increase in $PaCO_2$, and not by a decrease in respiratory rate alone. It is common for respiratory rate to decrease with a decrease in the level of activity and awareness (e.g., sleep), but tidal volume typically increases to compensate, resulting in no net change in $PaCO_2$. During anesthesia, respiratory insufficiency is common because many factors alter the chemoreceptor responsiveness to carbon dioxide, leading to elevated $PaCO_2$. Causes include the administration of opioids and other sedatives prior to anesthesia, the relative overdose of induction agents, positioning for surgery, respiratory depressant effects of inhalants, surgical trauma, recovery from bronchial alveolar lavage, and excessive use of opioids during recovery. The use of opioids with or without tranquilizers prior to induction of anesthesia to provide sedation and analgesia often results in a patient being well sedated but with depressed ventilatory drive. High doses of μ-receptor agonists such as oxymorphone, hydromorphone, and morphine are more likely to produce respiratory depression than is the κ-receptor agonist butorphanol. In addition, the decreased responsiveness to increased carbon dioxide tensions during halothane, isoflurane, or sevoflurane anesthesia tends to cause hypoventilation, although surgical stimulation often overrides some anesthetic-induced loss in respiratory drive.[70,71] In addition to the depressant effects of inhalants on responsiveness to increased $PaCO_2$, subanesthetic doses depress the peripheral chemoreceptors such that hypoxia does not stimulate a ventilation response.[72,73] Hypoxia often occurs during recovery from anesthesia after diagnostic bronchial alveolar lavage (BAL). Although BAL with volumes of up to 4 L have only transient effects in healthy dogs, BAL can lead to increased morbidity and mortality in dogs and cats with severe respiratory disease.[74–76] During BAL, supplemental oxygen can be administered by insufflation with a small rubber tube placed in the trachea alongside the bronchoscope (Fig. 53.5). After the procedure, oxygen should be administered by endotracheal tube, mask, or chamber until the pulse-oximeter readings remain at 90% saturation or higher while room air is breathed. Airway obstruction may also occur after ear-ablation surgery. Soft tissue swelling in the posterior pharynx may be severe enough to require a tracheostomy for relief.

Apnea is common during routine anesthesia. It occurs during induction after the administration of thiobarbiturates or propofol, during maintenance of anesthesia with ketamine, when controlled ventilation is discontinued, and as a consequence of deeper-inhalation anesthesia. Apnea occurring at induction is generally transient and is treated by low-frequency intermittent ventilation that is adequate to maintain hemoglobin-oxygen saturation at greater than 90%. Apnea occurring later during anesthesia, especially in spontaneously breathing animals, must be quickly recognized and treated with decreasing anesthetic concentrations and/or high-frequency positive-pressure ventilation. Apnea late in anesthesia is usually caused by excessive depression of the respiratory centers of the brain secondary to high anesthetic concentrations, or because of decompensation associated with severe neurological disease such as hydrocephalus or intracranial neoplasia.

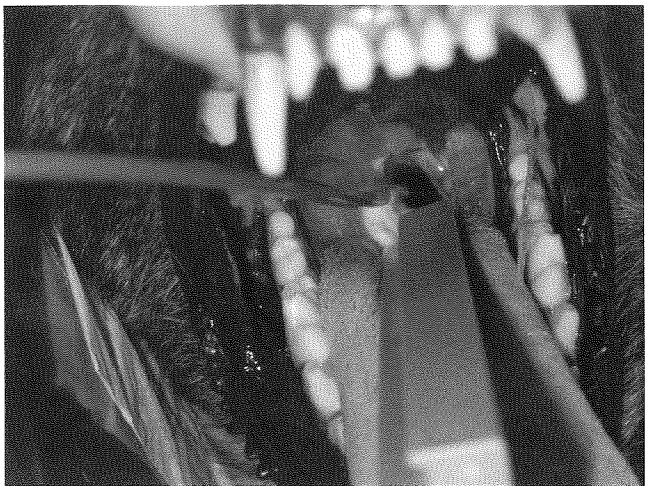

Fig. 53.5. Placement of a red rubber tube into the trachea for insufflation of oxygen during bronchial alveolar lavage. The red rubber tube via an extension is connected to the oxygen line that normally connects the anesthesia machine with the circle.

Generally, apnea during induction of anesthesia with thiobarbiturates or propofol is caused by a relative overdose or a fast bolus injection.[77] Ketamine and diazepam in a 1:1 mixture by volume is commonly used for anesthesia in cats. Apnea often occurs, especially when anesthesia is maintained with supplemental isoflurane. This combination of drugs, each of which is a respiratory depressant alone, can induce a persistent respiratory depression. If, in response to respiratory depression, assisted or controlled ventilation is employed, $PaCO_2$ will often be reduced below the arterial or alveolar PCO_2 level at which cats will remain apneic (apneic threshold). Decreased functional residual capacity (FRC) during anesthesia can increase hypoxia by lowering alveolar ventilation-perfusion ratios (V_A/Q) and expanding atelectatic areas. This occurs because the FRC is close to or less than the closing volume (CV) of the lung. The CV is the volume of the lung at which small airways begin to close. When the tidal volume is less than the CV, small airways remain closed throughout the breathing cycle, and atelectasis increases. If the CV of some airways remains within the tidal volume range, then there is some air exchange during inspiration and expiration, though not the normal amount. This partial ventilation decreases the V_A/Q. These lung changes are prevalent in older animals and during anesthesia. Intermittent positive-pressure breathing and positive end-expiratory pressure (PEEP) can be used to diminish the hypoxia that occurs from changes in FRC.

Equipment Malfunction

Routine equipment maintenance and leak tests should be used to reduce the chances of anesthesia-machine malfunctions. Com-

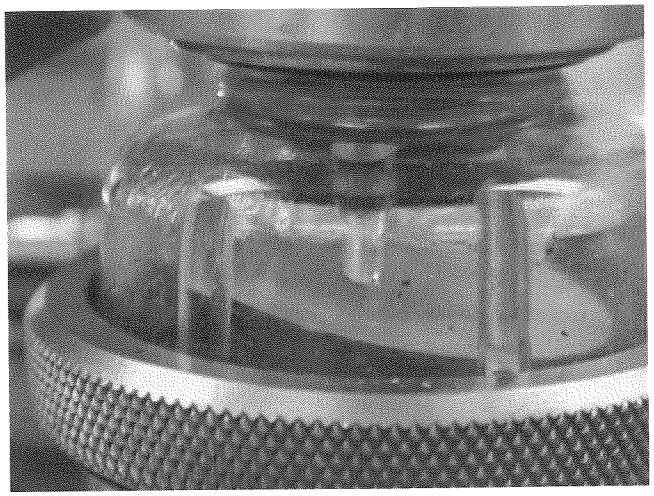

Fig. 53.6. Because of condensation of humidified exhaled gases on the dome and valve, this exhalation check valve has become lodged in the open position, enabling rebreathing of expired gases. An end-tidal carbon dioxide monitor would detect this equipment problem.

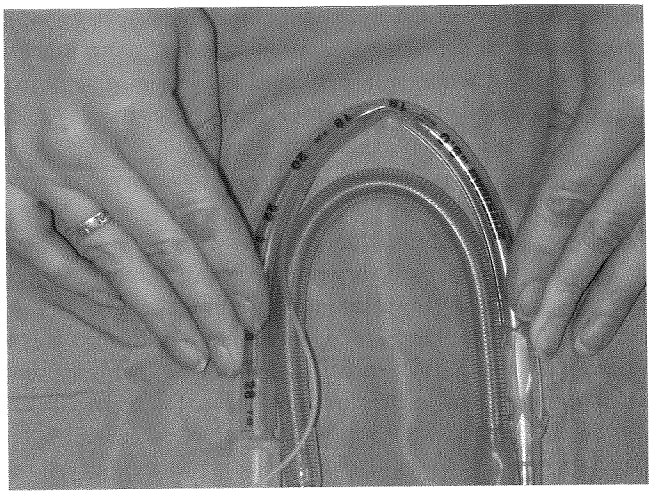

Fig. 53.7. The use of a wire-reinforced endotracheal tube will prevent kinking of the tube and obstruction of oxygen flow to patients. The protected endotracheal tube should be used when extreme flexion of the animal's head and neck or of the endotracheal tube is anticipated.

mon equipment malfunctions include "channeling" of gas flow through the carbon dioxide–absorbent canister, "sticking" of the exhalation check valve in the open position, interruption of oxygen supply, and kinking of the endotracheal tube. All of these equipment malfunctions can be rapidly detected with the routine use of a capnograph and pulse oximeter during inhalant or injectable anesthesia. *Channeling* occurs when gas flow through the carbon dioxide–absorbent canister is uneven, resulting in early termination of carbon dioxide absorption. If the pathway is through the center of the canister, there is not likely to be any observable change in color of the carbon dioxide absorbent, although the end-tidal carbon dioxide monitor should indicate increased rebreathing of exhaled gases (i.e., elevation of inspired carbon dioxide). Another cause of increased rebreathing of exhaled gases involves a malfunction of the exhalation check valve. The accumulation of moisture from humidified exhaled gases condensing on the cooler anesthesia machine parts can cause the check valve to remain in the open position (Fig. 53.6). In this situation, patients will rebreathe more exhaled gas and higher levels of carbon dioxide. The oxygen supply to a patient can be mistakenly interrupted when the oxygen lines have been pressurized before the oxygen cylinder has been turned off. When the oxygen flowmeter is turned on again, oxygen flow is initially present, giving the impression the oxygen cylinder is open, but will cease when the oxygen in the lines is exhausted. This is more likely to occur when switching from a central supply of oxygen to smaller oxygen cylinders mounted onto the anesthetic machine. The endotracheal tube can kink during extreme flexion of the animal's neck during positioning for cerebral spinal fluid tap, cervical spine radiographs, or ophthalmologic procedures. One should always determine patency of the endotracheal tube after extreme flexion of the head and neck. Use of a wire-reinforced endotracheal tube can reduce the incidence of obstruction (Fig. 53.7). If any problem is suspected with the anesthesia machine, the patient should be disconnected from the machine and temporarily ventilated with room air until the problem is solved.

Delayed Recovery

Occasionally, an animal will fail to recover normally from anesthesia. Common causes of this problem include hypothermia, hypoglycemia, and heavy narcotization. Hypoglycemia has been shown to decrease the minimum alveolar concentration of halothane in rats.[78] Hypoglycemia can be clinically silent in anesthetized patients, which emphasizes the importance of glucose monitoring in susceptible patients. During anesthesia, signs of sympathetic overactivity or ventricular arrhythmias may be the only detectable evidence of life-threatening hypoglycemia.[79] Patients at high risk of developing hypoglycemia in the perianesthetic period include neonates, very small patients, fasting diabetics treated with their usual insulin dose, and dogs with glucocorticoid deficiency.[80]

Occasionally, coma or blindness can follow anesthetic-related insult to the central nervous system. If persistent neurological deficit follows an apparently uneventful anesthetic procedure, likely causes include hypoxia, severe hypotension, undiagnosed hydrocephalus, other preexisting neurological dysfunction, or an idiosyncratic drug-related response. In these cases, the exact etiology may be harder to determine. Treatment of these patients is primarily supportive, and the prognosis has to be guarded. In cases of anesthesia-related cortical blindness, vision may return as long as 2 weeks later, so cautious optimism is appropriate.

Reports of poor or delayed recoveries from anesthesia abound in popular canine and feline breed journals, and many breed societies relay stories of anesthetic-related problems. There are indeed situations where breed-specific anatomical or physiological peculiarities may complicate anesthetic management. Also, ge-

netic differences in specific populations of a species or breed can perhaps increase the risk of performing anesthesia in some individual animals. Nevertheless, most animals presented with nonspecific warnings about delayed recovery from anesthesia respond normally to the careful dosing of commonly used anesthetic agents. Inappropriate dosage of anesthetic or inadequate patient monitoring is more likely the common culprit in many cases of reported "anesthetic sensitivity."

Gastroesophageal Reflux and Regurgitation

Reflux of gastric contents into the esophagus has occurred if esophageal pH decreases below 4 (reflux of gastric acid) or above 7.5 (reflux of bile). This reflux is clinically silent and usually acidic.[81,82] The lower esophageal sphincter is considered to be the primary barrier to the development of this reflux.[83] Lower esophageal sphincter pressure in dogs is decreased with the use of isoflurane, atropine, acepromazine, and xylazine.[84,85] The effects of many other anesthetic agents on sphincter function have not been determined. Gastroesophageal reflux (GER) reportedly occurs during anesthesia in approximately 17% of dogs receiving thiopental, halothane, and other agents; in 50% of dogs receiving propofol; and in up to 60% of dogs receiving preanesthetic morphine.[81,82,86,87] A 5% incidence of GER has been reported in a population of anesthetized people, which suggests that anesthetic-induced GER occurs less frequently in people than in dogs, even though opioids are commonly used in both species.[88] In some cases, the refluxate is of sufficient volume to reach the pharynx and even drain from the mouth (regurgitation). The incidence of regurgitation is currently estimated at around 0.1% in animals anesthetized at the Michigan State University Veterinary Teaching Hospital.[89] This material may be aspirated into the lungs, leading to pneumonitis, or may cause local irritation of the esophagus as a prelude to development of ulcerative esophagitis and stricture formation.[83,88,90,91]

Hypothermia

This occurs commonly in anesthetized patients because of depressed thermoregulation, excessive heat loss relative to metabolic production, and mixing of core and peripheral blood by indiscriminant vasodilation. Heat is lost to the environment through convection, conduction, radiation, and evaporation, and occurs more rapidly when body surface is larger relative to body mass. Many anesthetic drugs, including opioids, the inhalant anesthetics, and α_2-agonists, interfere with thermoregulation and contribute to prolonged postoperative hypothermia.[92–94] Inhalation anesthetics lower the threshold for response to hypothermia in people to about 34.5°C, and presumably this occurs in animals, as well.[92] The rate of core temperature decrease in horses under halothane anesthesia has been shown to be 0.37°C/h, which is reduced to 0.19°C/h with the application of a forced-air warmer to help maintain patient body temperature.[95] Anesthetized dogs have been shown to have a decrease in rectal temperature of 1.9°C/h in the first hour of anesthesia.[96] Hypothermia has been associated with pain, suppressed phagocytic activity including decreased migration of polymorphonuclear cells, reduced superoxide anion production, and reduced bacterial killing, and thus may contribute to systemic suppression of immune reactivity in the perioperative period.[97] In a retrospective study of dogs, mild decreases (1°C) in body temperature during surgery were not related to increased risk of incisional infections.[98] Accidental surgical hypothermia can be limited by increasing the ambient temperature, but this is seldom feasible. Circulating warm-water pads, especially applied to the legs, have been shown to help preserve body heat in dogs.[99] Forced-air warming systems currently are the most efficient and effective means of preserving or increasing body heat in anesthetized patients. This warming technique has been shown to delay or reduce the rate of heat loss in horses, dogs, and parrots.[95,100] Humidification and warming of inhaled gas has been shown to be ineffective as a sole means of maintaining core temperature in dogs or cats.[96,101,102] The use of uncovered electrical heating pads or "hot-water gloves" is discouraged because of potential for thermal injury.[103,104]

Hyperthermia

Drug-induced hyperthermia is rare in the practice of veterinary anesthesia. However, μ-receptor opioid agonists such as hydromorphone and fentanyl have been associated with moderate hyperthermia in some cats. The most commonly used drugs in human practice that can cause hyperthermia include antipsychotic agents, serotonin antagonists, sympathomimetic agents, inhalation anesthetics, and agents with anticholinergic properties.[105] The resultant hyperthermia is frequently accompanied by intense skeletal muscle rigidity (contracture), rhabdomyolysis, and hyperkalemia. Neuroleptic malignant syndrome is a rare but potentially lethal reaction to antipsychotic drugs, including phenothiazines and lithium.[96] Dopaminergic antagonism, a direct myotoxicity, altered thermoregulation, or extrapyramidal hyperactivity are postulated to contribute to the development of this syndrome. It is very possible that this syndrome could even occur in phenothiazine-treated animals placed in a very warm environment.

Malignant hyperthermia is an inherited membrane-linked abnormality (ryanodine receptor mutation) that has been documented in several species, including pigs, dogs, cats, and horses.[106–109] Susceptible patients should be anesthetized with barbiturates, propofol, opiates, and tranquilizers, and may be pretreated with dantrolene. Avoidance of known triggering agents—such as potent inhalation anesthetics, depolarizing muscle relaxants and stress—is advised.

Accidental iatrogenic hyperthermia can develop during warm ambient temperatures, in animals with thick hair coats, and with the use of forced-air warming systems. It is important to monitor body temperature in patients where active heating strategies are being used. Some smaller patients when treated with forced-air warming on the highest setting (43°C) heat up rapidly. In most situations, iatrogenic hyperthermia subsides rapidly after the heat source is removed.

Injuries

A number of other conditions can lead to injury during anesthesia:

Swollen feet: Limbs can be secured by ties placed so tight that they reduce venous drainage.

Corneal ulcers: Anesthetics reduce or eliminate the palpebral and corneal reflex and reduce tear formation. Chemical irritants, physical trauma, or drying can lead to ulceration. Artificial tears are important in preventing these problems.

Tracheal mucosal injury: Overinflation of the cuff or moving the cuff while it is inflated can cause mucosal injury, tracheal rupture, or tracheal chondromalacia. Tracheal rupture is an uncommon sequela to intubation in cats and can usually be treated medically.[110] When changing patient positions, the endotracheal tube should be disconnected from the Y adapter, and the patient's head and neck supported to prevent sliding or movement of the endotracheal tube cuff. To prevent pressure-induced mucosal necrosis, it is wise to inflate the cuff of the endotracheal tube only sufficiently to seal a leak at 10 to 20 cm H_2O. It is not recommended to simply put "some air" in the cuff without checking its pressure.

Joint pain: Older animals with arthritic joints that are placed on their backs for surgery may have joint pain for days following anesthesia.

Pulmonary barotrauma: Overinflation of the lungs will damage the pulmonary structures significantly if pressures exceed 30 cm H_20.[111–113] Inadvertently leaving the adjustable pressure-limiting valve (pop-off valve) closed or using the oxygen flush when the patient is on a Bain's system can create pulmonary overpressurization. One simple way to provide protection for the patient if the pop-off valve inadvertently remains in the closed position is to place a commercially available PEEP valve in the breathing circuit (Fig. 53.8).

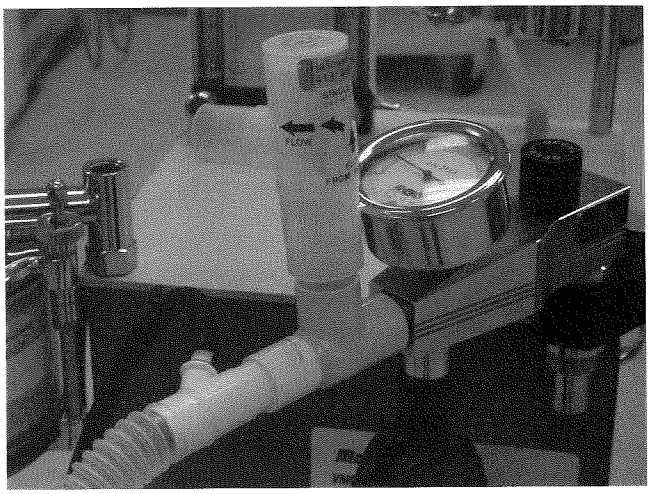

Fig. 53.8. A 20-cm water positive end-expiratory pressure (PEEP) valve can serve as an emergency release valve in case the adjustable pressure-limiting valve (pop-off valve) is accidentally closed and pressure begins to build in the anesthesia system. The PEEP valve can be used with circle and Bain's anesthesia delivery systems.

Epidural Analgesia and Regional Nerve Block

The use of the epidural route for delivery of opioids and local anesthetics is becoming increasingly popular, especially with prolonged drug delivery by epidural catheter.[114–118] There are reports of epidural catheters having been placed and left in dogs for 7 days and in horses for up to 20 days, with the main complication being catheter dislodgement and local tissue response.[116–118] Meticulous attention to aseptic technique is essential when drugs or catheters are placed in the epidural space. Epidural abscessation and discospondylitis have been reported following epidural injection.[119] Other complications reported following epidural injection in dogs include urinary retention, prolonged cerebrospinal fluid levels of morphine, myotonus, and pruritus.[114,120–122] Subarachnoid injection of preservative-free morphine in a dog caused such severe pruritus and myoclonus that the dog had to be anesthetized for several hours until the reaction resolved.[121] The authors have observed one horse with severe pruritus of the hind feet after epidural injection of xylazine and local anesthetic. Sedation with detomidine was sufficient to calm this horse. It appears from anecdotal reports that when a local anesthetic is combined with xylazine for epidural injection in horses, hind-limb ataxia and weakness are more likely to occur.

Regional nerve block has some risk of causing local anesthetic toxicity, although combining local anesthetics with epinephrine (5 μg/mL) will dramatically reduce this risk. Direct needle trauma to the nerve being blocked can cause a prolonged or permanent neural deficit. Local hemorrhage may result in hematoma formation, but this is generally self-limiting.

Electrolyte Abnormalities

Hyperkalemia is one electrolyte abnormality that can be associated with acute death.[123,124] The causes of rapid-onset hyperkalemia during anesthesia and surgery include transfusion of old stored blood, chronic heparin therapy (dogs), uroperitoneum (especially foals and cats), iatrogenic administration (potassium penicillin or potassium chloride), and hyperkalemic periodic paralysis (HPP). Horses that are homozygous or heterozygous for HPP should be closely monitored. Diet change or the stress of fasting, anesthesia, and pain may lead to an attack in the perianesthetic period. Signs of an HPP attack are obvious even in anesthetized horses as hyperkalemia produces very characteristic ECG changes.[125] Arrhythmias caused by hyperkalemia can lead to cardiac arrest and may not respond to conventional antiarrhythmic therapies, but do respond to the rapid treatment of hyperkalemia.[125,126] Aggressive lowering of serum potassium by using acetazolamide, furosemide, dextrose, and sodium bicarbonate, and the reversal of hyperkalemic effects on cell membrane potential by calcium administration can help resolve an HPP crisis if it occurs during anesthesia.[125,126]

Hypokalemia caused by hemodilution and decreased intake is the most common cause of postoperative arrhythmias in people.

Arrhythmias (ventricular ectopy) associated with this electrolyte disturbance are not commonly observed in other animals. Hypocalcemia is an insidious electrolyte disturbance that can also be a problem for anesthetized patients. Hypocalcemia can lead to muscle weakness, which can be particularly problematic for horses trying to stand while recovering from anesthesia.

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