Chapter 36

Cardiovascular Disease

Ralph C. Harvey and Stephen J. Ettinger

Introduction

The anesthetic management of a patient with cardiovascular dysfunction can be very challenging, because most preanesthetic and anesthetic agents capable of depressing the central nervous system can also produce cardiovascular depression. Patients with cardiovascular dysfunction may be more prone to fluid overload and dysrhythmias. Extremes in heart rate may cause severe problems, including heart failure. Patients with cardiovascular dysfunction may lack sufficient cardiac reserve to compensate for anesthetic-induced depression. Because of the diversity of pathophysiological conditions, no single anesthetic management technique or protocol can be recommended for all animals with cardiovascular dysfunction.[1] Appropriate patient monitoring with arterial blood pressure, central venous pressure, electrocardiogram, end-tidal carbon dioxide, pulse oximetry, and other parameters is essential to reduce anesthetic and surgical risk.

Cardiovascular Physiology

The function of the myocardial cell is to contract and relax rhythmically with other myofibers so that the heart will act as a pump. The basic contractile unit of heart muscle is the sarcomere, which is composed of interdigitating protein filaments of actin and myosin. Muscle shortening begins in the myocardial muscle when the actin and myosin filaments are activated. This activation is regulated by tropomyosin and troponin. Tropomyosin prevents the interaction of actin and myosin during diastole. When tropomyosin is no longer at its blocking position, systole is initiated. The availability of ionized calcium in the area of the troponin-tropomyosin protein unit acts as an immediate catalyst for the contraction-relaxation cycle. The contraction of the heart muscle depends on the amount of free calcium ions available around the myofibril. Part of the contractile-dependent calcium originates from superficial sites on cell membranes that are in equilibrium with extracellular calcium and therefore can be affected by drugs that do not penetrate the myocardial cell.

Few clinically used drugs affect the actin and myosin proteins, but many drugs can alter the availability of calcium for activation of the contractile process.[1] Digitalis increases calcium movement to the troponin-tropomyosin protein unit and thus increases contractile strength. Barbiturates and inhalant anesthetic agents seem to disrupt calcium movements and thus cause reduced contractile strength. Myocardial intracellular acidosis also inhibits the binding of calcium to the troponin-tropomyosin unit, causing decreased myocardial contractile strength. Disease conditions or drugs that produce metabolic or respiratory acidosis may decrease contractility. Most anesthetics depress respiration and predispose patients to respiratory acidosis.

Blood pressure is the product of peripheral vascular resistance and cardiac output. Cardiac output is the product of heart rate and stroke volume. Drugs that alter any or all of these parameters may greatly affect blood pressure and tissue blood flow. Preanesthetic and anesthetic agents can alter vascular resistance (e.g., phenothiazine tranquilizers, α_2-adrenergic agonists, barbiturates, and inhalant agents), heart rate (e.g., opioids, α_2-adrenergic agonists, dissociative agents, and inhalants), and stroke volume (e.g., inhalant anesthetics). Patients that suffer from diseases causing impaired cardiac output, patients with congenital heart disease, and those suffering from hypotension, hypovolemia, anemia, and/or heartworms are at a higher anesthetic risk.

Impaired Cardiac Output

Anesthetic management of patients with impaired cardiac output depends greatly on the underlying pathology, which may require echocardiography for accurate diagnosis. General guidelines are to avoid bradycardia and tachycardia, decreased preload, and hypovolemia, as well as unnecessarily high left-atrial pressures and volume overload. The decreased tolerance of these patients for improper fluid therapy and anesthetic management emphasizes the need for adequate perioperative monitoring and support. These patients should be preoxygenated for 5 to 7 min prior to anesthetic induction. If cardiovascular function is adequate, the choice of anesthetic drugs may not be specific for these patients; however, drugs that may produce tachycardia (anticholinergics

and dissociative agents) or large changes in vascular resistance should be used judiciously.

Narcotics are often used as preanesthetic medication because of their minimal effects on the myocardium, and they can be readily antagonized. Indeed, opioids are the mainstay of cardiac anesthesia. Opioids tend to maintain, and may even indirectly improve, myocardial function. They can be used in combination with acepromazine or a benzodiazepine tranquilizer for additional sedation. Use of acepromazine may be contraindicated in some forms of cardiovascular diseases (e.g., hypertrophic cardiomyopathy) but may be beneficial in others (e.g., mitral valve insufficiency) because of the potential for decreased afterload. Opioids can increase vagal afferent activity, which may decrease heart rate. If significant bradycardia occurs, atropine or glycopyrrolate should be given as needed.

If only tranquilization is needed, a low dose of acepromazine (e.g., 0.02 mg/kg) may be administered intramuscularly. Acepromazine decreases peripheral vascular resistance and very often leads to arterial hypotension and reduced preload. Acepromazine can have significant negative inotropic effects. Because of direct myocardial depression, and prominent vasodilatation and hypotension, the value of inducing sedation/tranquilization must be weighed against the potential adverse effects. Phenothiazines must be used cautiously in most cardiac patients.[2] If acepromazine is administered, patients must be monitored closely and appropriate supportive care used should adverse effects become significant.

The use of α_2-adrenergic agonists should be avoided in patients with impaired cardiac output. These drugs can produce significant dysrhythmias, including severe sinus bradycardia and sinoatrial and atrioventricular nodal blocks. Bradycardia, reduced contractility, and increased afterload are particularly disadvantageous effects of α_2-adrenergic agonists in many cardiac patients.[2,3]

Cardiomyopathy

Cardiomyopathy can be classified as hypertrophic or congestive. Hypertrophic cardiomyopathy is characterized by ventricular hypertrophy, decreased ventricular compliance, and impaired ventricular filling, which result in reduced cardiac output. Ventricular contractility (pump function) is usually not impaired.[4] Congestive (dilated) cardiomyopathy is characterized by marked ventricular dilation, increased ventricular end-diastolic and systolic volumes, and poor myocardial contractility. Often, congestive heart failure is present.[4] Since most anesthetic drugs worsen existing myocardial performance, to reduce the risk during the perianesthetic period, treatment of cardiomyopathy is warranted prior to anesthesia. Commonly employed cardiovascular medications used for treatment of congestive heart failure are listed in Table 36.1. In dogs with dilated cardiomyopathy, anesthesia is best induced with agents that have minimal direct myocardial depressant effects. Etomidate or alphaxalone would be preferable induction agents over either thiopental or propofol. The direct depressant effects of ketamine on the myocardium may be clinically significant if sympathetic nervous system efferent activity is already maximal or exhausted. The lowest possible concentration of an inhalant should be used for maintenance of anesthesia. Anesthesia is less depressing to myocardial performance when an opioid is coadministered, resulting in lower inhalant anesthetic requirements.

Pericardial Tamponade and Constrictive Pericarditis

These are associated with impaired cardiac output caused by reduced stroke volume secondary to reduced end-diastolic ventricular volume. There is limited expansion of the cardiac chambers, resulting in decreased ventricular filling such that heart rate must increase to maintain cardiac output. Pulse pressure is usually decreased, and peripheral pulses may feel abnormal. Myocardial contractility might not be impaired.[5]

Valvular Heart Disease

The heart contains four valves: mitral, tricuspid, aortic, and pulmonary. Valve changes usually can be classified as insufficiency or stenosis. Valvular heart disease is associated with impaired cardiac output and, when severe, can cause congestive heart failure. When a murmur is asculted or valvular heart disease is suspected, the preanesthetic evaluation should include thoracic radiographs and possibly an echocardiogram in addition to the routine preanesthetic screening. The value of these diagnostic tests is to facilitate anesthetic planning and intraoperative responses to abnormal monitored parameters since the management of valvular disease can vary significantly depending on which valve (valves) is (are) affected.

Although both mitral and tricuspid insufficiencies are often clinically insignificant, ventricular ejection fraction is reduced. It is useful to maintain heart rate, maintain contractility, and avoid arteriolar constriction.[6] Antimuscarinics should be used conservatively, but lower doses of atropine or glycopyrrolate are often used to inhibit anesthetic-associated bradycardia. α_2-Adrenergic agonists are contraindicated in patients with valvular insufficiency, because they can affect heart rate and peripheral vascular resistance. Opioids are a principle component of balanced anesthesia for these patients and are generally chosen based on analgesic and sedative requirements. If these patients are stable, anesthesia may be induced with ketamine and diazepam or propofol. Less stable patients may be anesthetized by induction with etomidate or high doses of opioids in combination with a benzodiazepine. To avoid pronounced vasodilatation and hypotension, lower doses of inhalants are used. Use of the inhalants helps to prevent arteriolar vasoconstriction and increased afterload. Fluid therapy should be conservative and based on continual monitoring of central venous pressure and arterial blood pressure. As with other aspects of anesthetic care, postoperative monitoring and support are individualized.

Hypertrophic Cardiomyopathy

This is the most commonly diagnosed cardiac disease of cats.[7] An inherited form of the disease has been identified in humans and in Maine coon cats. Hypertrophic cardiomyopathy (HCM) is characterized by a stiff left ventricle with poor diastolic function. As

Table 36.1. Chronic therapy for congestive heart failure

Classes	Trade Name	Mechanism of Action	Effects on Contractility	Effects on Afterload	Maintenance Dose
Nitrovasodilators					
Na nitroprusside	Nitropress	Activation of EDRF or NO. These drugs act as substrates for the formation of NO. Nitroprusside is primarily an arterial dilator, whereas nitroglycerin is a venodilator.	—	↓	2–5 µg/kg/min IV (monitor pressure) CRI suggested Protect drug from light
Nitroglycerin	Nitrostat; Nitropaste	Preload-reducing agent.	—	↓	1–5 µg/kg/min IV recommended only for acute-care situations with continuous blood pressure monitoring. Available in oral and transdermal paste formulations, but oral is rarely if ever used in animals. Pastes are considered third-line therapy. More likely to use oral hydralazine acutely. *Dog*: ¼–½ inch/kg QOD *Cat*: ⅛ inch/kg QOD
Isosorbide dinitrate	Many formulations available	Formation of NO.	—	↓	0.5–2.0 mg/kg PO, BID; also available as an ointment; uncommonly used in animals
Hydralazine	Apresoline	Hydralazine interferes with Ca^{+2} transport in smooth muscle; acts as an afterload-reducing agent.	—	↓	*Dog*: 0.5–2.0 mg/kg PO, BID *Cat*: not commonly recommended
ACE inhibitors					
Captopril	Capoten	Prevents conversion of angiotensin I to angiotensin II, which decreases blood pressure and produces some venodilation. Produces balanced vasodilation, prevents renal fluid retention, reverses cardiac fibrosis, and slows heart rate through decreased ß-adrenergic stimulation.	—	↓	This drug is rarely used in VM and is less reliable than are the other ACE I agents below. *Dog*: 0.5–2.0 PO, TID *Cat*: the same
Enalpril	Enacard		—	↓	*Dog*: 0.5 mg/kg PO, SID-BID *Cat*: 0.25–0.5 mg/kg PO, SID-QOD
Benazepril	Lotensin Foretaker		—	↓	*Dog*: 0.25–0.5 mg/kg PO, SID-BID *Cat*: 0.25–0.5 mg/kg PO, SID
Lisinopril	Zestril Prinivil	ACE I, as above for captopril		↓	*Dog:* 0.5 mg/kg PO, SID *Cat:* 0.25 mg/kg PO, SID
Diuretics					
Acetazolamide		Inhibits Na^+ from passing into the proximal tubule. The osmotic effect is at the glomerulus.	—	↓	*Dog*: 10 mg/kg q 6 h Rarely used in cardiovascular medicine
Aminophylline		Increases the vascular perfusion of the glomerulus.	↑	↓	*Dog*: 11 mg/kg PO, BID-TID *Cat*: 5 mg/kg PO, BID-TID
Spironolactone	Aldactone	Inhibits the aldosterone receptor in collecting tubule (CT). Prevention of ACE escape is the principal use of this agent now.	—	↓	*Dog*: 0.25 mg/kg SID for preventing ACE escape. Higher dosages may be considered as a diuretic. *Cat*: the same
Furosemide	Lasix	Inhibits the Na^+, K^+, and Cl^{-2} cotransporter in the thick ascending loop of Henle.	—	↓	*Dog*: 1–4 mg/kg PO, SC, IM, IV, SID-QID *Cat*:1–2 mg/kg PO, SC, IM, IV, SID-QID

(*continued*)

Table 36.1. Chronic therapy for congestive heart failure (*continued*)

Classes	Trade Name	Mechanism of Action	Effects on Contractility	Effects on Afterload	Maintenance Dose
Hydrochlorothiazide	Hydrodiuril	Blocks resorption in the distal convoluted tubule by inhibiting the Na^+ and CT cotransporter.	—	↓	*Dog*: 2–4 mg/kg PO, SID-BID *Cat*: 1–2 mg/kg PO, SID-BID
Inotropes					
Digitalis	Cardoxin; Lanoxin	Inactivates the Na^+/K^+ ATPase pump, increasing Ca^{+2} intracellularly.	↑	↑	*Dog*: 0.005–0.008 mg/kg PO, BID in smaller dogs and 0.22 mg/m^2; also twice daily in larger dogs *Cat*: ¼ to ½ 0.125-mg tablet PO q 48–72 h, based on size and clinical response
T3	Tristat	Increases Ca^{+2} adenosine ATPase activity; upregulates beta-receptor activity.	↑	—	Not likely to be used and not a primary inotrope but may be part of the protocol along with traditional agents in the event of a low thyroid value
"Inodilators"					
Amrinone	Inocor	Class III phosphodiesterase enzyme inhibitors (PDEIs) prevent breakdown of cAMP, which results from the stimulation of ß-adrenergic receptors.	↑	↓	1–3 mg/kg IV slow to effect, then CRI at 10–100 µg/kg/min
Milrinone	Primacor		↑	↓	1–3 mg/kg/min IV slow, then CRI at 10–100 µg/kg/min to effect
Pimobendan	Vetmedin	Ca^{+2} sensitization and PDE inhibition.	↑	↓	*Dogs*: 0.3–0.6 mg/kg PO, SID *Cats*: not known if applicable
Beta blockers					
Metoprolol	Lopressor; Toprol XL	Upregulates damaged beta sites; reduces the adrenergic barrage; slows the cardiac rate.		↓	*Dogs*: 0.25–1.0 mg/kg PO BID-TID; begin low and titrate upwards slowly. *Cats*: very low dosages used usually for heart rate control and not CHF
Carvedilol	Coreg	Beta-blocking agent and peripheral vasodilator (blocks $ß_1$, $ß_2$, and α_1).	↑	↓	*Dogs*: 0.05 mg/kg PO initially once daily: slowly titrate weekly to maximum 0.3 mg/kg

ACE, angiotensin-converting enzyme; CRI, constant-rate infusion; EDRF, endothelium-derived relaxing factor; IV, intravenous; NO, nitric oxide; PO, per os (orally); and SC, subcutaneously.
This table enlists a variety of classes of medications including digitalis, "inodilators," vasodilators, ACE inhibitors, and diuretics. Dosages taken from *Textbook of Veterinary Internal Medicine*, 6th ed.[16]

ventricular wall thickness increases, end-diastolic volume and ventricular function are decreased. Congestive heart failure develops as ventricular stiffness increases, end-diastolic ventricular volume decreases, and mitral regurgitation and hypertension develop. Early HCM is often asymptomatic. Signs of progressive HCM (murmurs, arrhythmias, dyspnea, and thromboembolic disease) are consistent with the development of heart failure. Sudden death during the stresses of hospitalization, anesthesia, and medical or surgical procedures can occur with more advanced HCM.

For patients with early signs of cardiomyopathy, anesthesia can be induced by using propofol, etomidate, or a neuroleptanalgesic combination. Mask induction using one of the potent volatile inhalant anesthetics is also an acceptable technique, although the stress of induction may be detrimental to patients with impaired cardiac function. Inhalant anesthetics are most often the maintenance agents for these patients. Isoflurane is one of the preferred inhalants because of preservation of a near-normal cardiac index and minimal dysrhythmic effects in healthy animals, when compared with the effects of halothane. However, in animals with diastolic dysfunction (e.g., HCM) isoflurane may be associated with reduced preload and afterload leading to reduced end-diastolic ventricular volume and increased end-systolic ventricular to aortic pressure gradients when dynamic outflow-tract obstruction is present. Sevoflurane minimally reduces cardiac output and is associated with less vasodilation than is isoflurane at typical anesthetic doses in healthy animals. Animals may maintain a lower heart rate when anesthetized with sevoflurane than with isoflurane, although preanesthetic drugs may alter the

cardiovascular responses to inhalant anesthetics. Based on the lower blood solubility and reduced pungency, sevoflurane may provide for a less stressful inhalant induction compared with either halothane or isoflurane. When a lower heart rate is desired (e.g., for cats with HCM), sevoflurane may be preferred over isoflurane.[8] Among the injectable general anesthetics, etomidate is unique in maintaining cardiac output without increasing myocardial oxygen consumption.

Congenital Heart Disease

When considering the anesthetic management of patients with congenital heart disease, the problems encountered are often similar to those in patients with congestive heart failure. The most common surgically correctable problems are patent ductus arteriosus (PDA) and persistent right aortic arch (PRAA).

PDA is usually recognized early in life before patients develop signs of heart failure. Typically, if diagnosed early, shunt blood flow is from the systemic circulation to the pulmonary artery (left-to-right shunt). However, when systemic vascular resistance decreases after induction of anesthesia, shunt flow may reverse, especially when significant pulmonary hypertension is present. Pulse oximetry is useful for rapid detection (rapid reduction in arterial hemoglobin saturation) of this reversion to a right-to-left shunt. Phenylephrine can be useful in this situation to increase systemic pressure and reestablish left-to-right flow. If the patient is normal in other respects, the anesthetic protocol is designed for the pediatric patient undergoing a thoracotomy with attention toward maintaining heart rate and cardiac output. Surgical manipulation around the heart may cause ventricular ectopic beats. These are usually transitory and do not require treatment. When the PDA is ligated, increased blood pressure may cause a reflex slowing of the heart rate. This is a normal physiological response.[8] In some instances, antimuscarinic drugs (e.g., atropine or glycopyrrolate) may be needed to counteract the sinus bradycardia. To minimize the potential for bradycardia, an anticholinergic may be administered as a preanesthetic medication. Because of the size of some patients, intraoperative hypothermia is often a problem when they undergo PDA surgery. Every effort should be made to minimize the loss of body heat.

PRAA is also usually recognized and corrected early in life. If a patient is normal in other respects, the anesthetic protocol is designed for the pediatric patient undergoing a thoracotomy. It is important to remember that a patient with PRAA may be suffering from aspiration pneumonia. As with a PDA, surgical manipulation around the heart may cause ventricular ectopic beats, and intraoperative hypothermia is of concern.

Hypotension, Hypovolemia, or Shock

Patients with hypotension and hypovolemia should be stabilized with intravenous fluids and/or whole blood prior to anesthesia. Many preanesthetic and anesthetic drugs are potentially hypotensive; therefore, these drugs can exacerbate preexisting hypotension.

Shock can be defined as an acute clinical syndrome characterized by progressive circulatory failure that leads to inadequate capillary perfusion and cellular hypoxia.[9] Shock is a complex, multisystem disorder that may be caused by a variety of insults. Shock may be classified as hypovolemic, cardiogenic, or vasculogenic. If one thinks of the cardiovascular system as a pump, fluid, and pipes to carry the fluid, then the three classifications reflect which component of the cardiovascular system is affected.

Hypovolemic shock occurs when there is an inadequate volume of fluid (blood) being pumped through the cardiovascular system. Hemorrhage, fluid loss, and trauma can all cause hypovolemic shock.

Cardiogenic shock, which occurs when the heart is no longer an effective pump, can be caused by a failure in ventricular filling (cardiac tamponade, tension pneumothorax, or collapse of the vena cava caused by inadvertent closure of the pop-off valve, resulting in airway pressure buildup) or by a failure of ventricular ejection (ruptured chordae tendineae, cardiac dysrhythmias, severe myocardial depression, or severe and prolonged increase in systemic vascular resistance).

Vasculogenic shock occurs when there are changes in venous capacitance or peripheral resistance. Numerous causes can lead to vasculogenic shock, including sepsis (vasodilation is caused by release of vasoactive substances such as histamine, prostaglandins, and bradykinin), anaphylaxis (vasodilation occurs because of histamine release), neurogenic factors (loss of vasomotor tone caused by excessive general anesthesia, trauma of the central nervous system, and spinal anesthesia), and a severe and prolonged increase in peripheral resistance.

Regardless of the underlying cause, a common pathway of circulatory failure is present in shock. Reflex mechanisms may compensate for early circulatory failure and result in recovery of a patient with mild or moderate shock. However, reflexive compensatory mechanisms may become deleterious to a patient if they are prolonged and may result in microcirculatory changes and further cellular hypoxia.

All forms of shock eventually result in decreased blood flow and hypoperfusion of the body tissues. Baroreceptors in the aorta and carotid artery and low-pressure receptors in the atria respond to the decreased cardiac output and blood pressure. This results in activation of the sympathoadrenal system. Hypothalamic sympathetic nerve centers increase release of norepinephrine from postganglionic sympathetic nerve endings and increase liberation of epinephrine and norepinephrine from the adrenal medulla into the blood. This results in splenic contraction and a release of blood into the circulation. Epinephrine and norepinephrine stimulate alpha and beta receptors. Alpha-receptor stimulation results in vasoconstriction of both arteries and veins. Beta-receptor stimulation causes vasodilation in skeletal muscle and increased force and rate of cardiac contraction. This results in a redistribution of blood flow to the heart and brain. Blood flow to the splanchnic, renal, and cutaneous vessels is markedly decreased. The catecholamines produce tachycardia, increase myocardial contractility, and stimulate hepatic glycogenolysis. Venous constriction causes decreased vascular capacity. The decreased vascular capacity improves venous return and thus cardiac output.

An important compensatory mechanism in shock is the ex-

travascular fluid shift. Owing to vasoconstriction, there is decreased capillary blood flow and thus capillary pressure. The decreased capillary pressure allows extravascular fluid to enter the blood vessels. This is very important in expanding circulating fluid volume. Endocrine factors are also important in the compensatory mechanism of shock. Renin is released from the ischemic kidney to activate the renin-angiotensin-aldosterone system. This results in vascular constriction; renal absorption of sodium, chloride, and water; and renal excretion of potassium. Antidiuretic hormone is released because of hypovolemia, and this also promotes water retention. The overall effect is to increase extracellular fluid volume.

These compensatory mechanisms cause a significant redistribution of blood flow to the heart, brain, and adrenal glands, and may aid recovery of patients in mild to moderate shock. However, they may not be adequate in severe shock, and it may become irreversible. Irreversibility is characterized by inadequate tissue perfusion to vital organs that results in cardiac failure, disseminated intravascular coagulation, depression of the reticuloendothelial system, and peripheral vascular failure. Hypotension and decreased capillary perfusion lead to cellular hypoxia, decreased delivery of energy substrates to the cell, and increased concentration of cellular metabolites.

Glucose is first used anaerobically by the cells as an energy source with production of pyruvate and limited amounts of adenosine triphosphate (ATP). Pyruvate is then aerobically utilized to produce large amounts of ATP, or it may be released into the circulation after being reduced to lactic acid. Large amounts of oxygen are needed by cells to produce the ATP. In shock, cellular hypoxia occurs, and although ATP can be produced anaerobically, it may not be produced in adequate amounts.[9] The establishment of membrane ionic gradients depends on adequate ATP generation. Cellular edema may result if ionic gradients are not maintained. The lactic acidemia that occurs in shock results from the release of anaerobic energy in tissues unable to support adequate oxidative processes. Individual cells and then organs begin to die.

Increased cellular metabolites (lactic acid) in the capillary bed cause precapillary sphincters to relax, but postcapillary sphincters remain constricted. Blood flows into the capillary bed but is slow to leave, resulting in an increased hydrostatic pressure with net flow into the tissues and further volume deficits. Decreased perfusion of the splanchnic vasculature results in pancreatic ischemia and the release of myocardial depressant factor. Myocardial depressant factor decreases myocardial contractility.[10] Splanchnic vasoconstriction and decreased capillary perfusion depress the reticuloendothelial system in the spleen and liver. With impaired function of the reticuloendothelial system, endotoxins, bacteria, and microemboli accumulate and produce further circulatory failure. Slow-moving (stagnant) acidic blood is hypercoagulable. Clot-initiating factors are common in shock and include bacterial toxins and thromboplastin of red cells (released by hemolysis).[11] These factors result in disseminated intravascular coagulation, which results in a consumption of clotting factors, bleeding, and focal tissue infarcts due to microthrombi. Multiorgan failure (multiorgan dysfunction syndrome) occurs, and the patient dies. Induction of anesthesia in patients with any level of shock is ill-advised.

Anemia

Anemic patients are at higher risk from an anesthetic standpoint because the oxygen-carrying capacity of the cardiovascular system is diminished. Either packed red blood cells, whole blood, or hemoglobin-based oxygen-carrying solutions should be considered if the dog or cat has a packed cell volume (PCV) of less than 25% to 30% before surgery or less than 20% after surgery. Patients with chronic anemia seem to be able to cope better with the problem than those with acute anemia. Whole blood, packed red blood cells, or a hemoglobin-based oxygen-carrying solution (e.g., Oxyglobin) should be readily available. The rate and total amount of blood administered should be tailored to the requirements of the patient. Patients with acute blood loss and hypovolemia can usually tolerate faster rates of colloid administration than can normovolemic patients that are anemic.

Anemic and/or hypoproteinemic patients should have serial PCV and total plasma protein concentration measurements during and after surgery. If an animal is anemic, supplemental oxygen may be beneficial in the preanesthetic period as well as the postoperative period to maintain maximal hemoglobin saturation. Although dissolved oxygen content is very minor compared with hemoglobin-bound oxygen content, a high inspired oxygen tension will enable more oxygen to be dissolved into the plasma and thus help counteract the decreased oxygen-carrying capacity due to low red blood cell numbers. A mask, nasal catheter, or oxygen cage can be used to deliver 40% to 100% oxygen to patients.

A pulse oximeter should be used during anesthesia and during recovery when patients are anemic. One of the periods of greatest risk for anemic patients is when anesthesia is discontinued and the fraction of inspired oxygen suddenly decreases from near 1.0 to 0.21 (room air) in the presence of anesthetic drug-related respiratory depression. In addition, as a hypothermic patient recovers, shivering will occur, dramatically increasing oxygen demands. Since approximately 5 g/dL of desaturated hemoglobin is needed for visible cyanosis to develop, anemic patients (PCV less than 15%) would not be expected to appear cyanotic even though hemoglobin oxygenation is dangerously low. Pulse oximeters are more sensitive at detecting hemoglobin desaturation in anemic patients, although a particular model's accuracy under these conditions may vary.

Hypoproteinemia

Many preanesthetic and anesthetic drugs are reversibly bound to plasma proteins, especially albumin. If plasma protein concentration is decreased, a greater fraction of highly protein-bound drug (i.e., protein binding in excess of 80%) is pharmacologically active and therefore will have an increased effect. Plasma protein, primarily albumin, is also required to maintain plasma oncotic pressure. Hypoalbuminemic patients are less tolerant of crystalloid fluid administration and more prone to volume overload and

pulmonary edema. Total plasma protein concentration should be maintained above 3.5 to 4.0 g/dL. If the plasma protein concentration falls below this number, the administration of plasma or colloidal fluid substitutes should be considered.

Heartworm Disease

A positive heartworm test in itself does not contraindicate any particular anesthetic regimen or protocol. If the patient is not exhibiting clinical signs, any standard anesthetic protocol is probably satisfactory, provided patient monitoring is appropriate. One should be aware that patients with heartworms may be more prone to spontaneous and catecholamine-induced cardiac dysrhythmias while under anesthesia.[12] Additionally, pulmonary hypertension and/or pulmonary embolic disease may be present, affecting pulmonary and cardiovascular function. If a significant number of heartworms are present, cardiac output may also be decreased.

Cardiac Dysrhythmias

Ventricular dysrhythmia is relatively common in dogs and cats during anesthesia, and its incidence is increased with certain anesthetics (e.g., halothane) and diseases (e.g., splenic neoplasia). Arrhythmia is more common with inappropriate levels of inhalant anesthetics.[13–15] Ventricular tachyarrhythmia can be resolved through use of antiarrhythmics (typically lidocaine or sotalol), by providing deeper anesthesia if the patient is inadequately anesthetized, or by changing to a less arrhythmogenic anesthetic (e.g., changing from halothane to isoflurane). Catecholamine-induced arrhythmia may be more common during illness, after injury, and with other stressors.

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