Left-Sided Congestive Heart Failure in Dogs: Treatment and Monitoring of Emergency Patients*

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ABSTRACT: As clinicians' knowledge about congestive heart failure (CHF) improves, so does the ability to treat and monitor patients with this condition. Initiating effective treatment at an early stage of cardiac disease may prevent or delay progression of CHF, thereby enabling the most favorable long-term outcome. This article focuses on the treatment and monitoring of animals that present with clinical signs related to decompensation despite therapy or to previously undiagnosed CHF.

Early diagnosis of congestive heart failure (CHF) and rapid initiation of basic therapies to improve hemodynamics are vitally important in the emergency setting, although the patient's condition can dictate how much diagnostic testing is possible. Once a diagnosis has been made, aggressive treatment is often imperative to be able to hope for a favorable outcome. Of paramount importance in aggressively treating pulmonary edema are the administration of parenteral diuretics and oxygen supplementation to help improve hemoglobin oxygen saturation. However, placement of a central venous catheter or a nasal oxygen tube can be fatal in a patient with extreme cardiopulmonary instability. In these cases, the use of intramuscular injections and flow-by or hood oxygen is the best therapeutic choice. Most canine patients tolerate placement of a peripheral catheter without experiencing undue stress. If vascular access is achieved, the available medical arsenal includes constant-rate infusion (CRI). Supplemental oxygen and loop diuretics are still considered the mainstays of primary treatment.

INITIAL ACUTE TREATMENT

Diuretic Therapy

Diuretics are considered to be the most critical therapy in acute and chronic CHF.[1] The goals of using a loop diuretic are to reduce intravascular fluid volume and to rapidly decrease preload venous capillary pressures, thereby reducing cardiogenic pulmonary edema.[2] Loop diuretics act on the thick ascend-

*A companion article on pathophysiology and diagnosis begins on page 79.
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ing loop of Henle and inhibit sodium, potassium, and chloride cotransport.[3,4] The end result is loss of urinary water, hydrogen ions, potassium, sodium, chloride, calcium, and magnesium.[3–5] The goal of diuretic therapy in the emergent patient is to decrease the patient's body weight by 5% to 7%.[6]

Furosemide is one of the most commonly used loop diuretics in veterinary and human medicine.[3–6] The dose, route, and frequency of administration should depend on disease severity and patient stability[5]; the initial dose should be 2.2 to 4.4 mg/kg IV or IM (Table 1). This dose can be repeated every 30 to 60 minutes until the patient's body weight has decreased by 5% to 7%.[6] The dosing interval can then be changed to every 4 to 6 hours until the patient can tolerate switching to oral therapy. Another option is to administer 2.2- to 4.4-mg/kg boluses every 60 to 120 minutes until the patient's respiratory rate and distress start to decrease.[7] This approach can be useful for patients in which the stress of repeated weighing should be avoided. The diuretic effect of IV furosemide lasts only 1 to 2 hours after each bolus.[3,4,7,8] Neurohormonal activation may cause rebound sodium and water retention between doses.[3,4,7]

Recent literature has demonstrated that using a CRI of furosemide leads to greater diuresis, natriuresis, and calciuresis with less kaliuresis in healthy dogs than does using intermittent boluses.[9] A loading dose of 2.2 to 4.4 mg/kg (maximum: 8 mg/kg) should be given before starting the CRI at a dose of 0.5 to 1 mg/kg/hr. When an animal is placed on a CRI, a syringe pump can be used to deliver the concentrated form of the drug. The CRI should be set up with the highest concentration possible and a flow rate sufficient to keep the IV catheter patent without significantly increasing the patient's intravascular fluid volume. Unless other drugs need to be administered by CRI, the administration of supplemental fluids is contraindicated in patients with fulminant CHF.[6] If IV fluids are considered necessary, a fluid that is low in sodium content, such as lactated Ringer's solution (130 mEq sodium/L), 0.45% saline with 2.5% dextrose, or 5% dextrose in water, should be administered at the lowest rate possible.

The use of diuretics in an azotemic patient may present clinicians with a dilemma. The azotemia may be prerenal due to severe failure of forward flow (i.e., poor renal perfusion due to decreased cardiac output), or it may be secondary to chronic diuretic treatment for CHF with subsequent renal insufficiency. Regardless of the cause, patients with CHF require a diuretic to decrease pulmonary vascular fluid overload. The diuretic dose should be selected based on the severity of CHF rather than on the presence or degree of azotemia. The best option is to use the smallest dose of drug required to achieve diuresis. If the clinical response to an initial dose is poor, the dose should be increased. Concurrent renal and heart failure is a therapeutic reality in which the treatment for one condition can adversely affect the other until organ failure occurs.

Spironolactone is an aldosterone antagonist that acts on the distal nephron and collecting duct by blocking the exchange of potassium and sodium, thereby sparing potassium and promoting the excretion of sodium.[10] Aldosterone is released in the final step of the renin-angiotensin-aldosterone system,[c] the prolonged activation of which ultimately leads to the detrimental effects of heart failure, such as the promotion of sodium retention, myocardial fibrosis, and increased sympathetic and decreased parasympathetic activity.[11] Spironolactone has been shown to decrease heart rate and mortality in humans with CHF and plays a significant role in the treatment of human heart failure.[1,10]

Spironolactone is not often used in the acute management of acute left-sided CHF in dogs because its diuretic efficacy in dogs is unknown. Therefore, it should not be considered as an alternative to a loop diuretic. It may be better used as an adjunct to furosemide on a long-term basis, not for its diuretic effects, but for its antialdosterone properties.

Oxygen Supplementation

Oxygen supplementation is integral to correcting arterial hypoxemia. The goals of oxygen therapy are to increase arterial oxygenation (PaO_2), prevent anaerobic metabolism, and decrease myocardial workload during hypoxemic states.[12,13] Ventilation/perfusion mismatch is the leading cause of arterial hypoxemia in animals with pulmonary edema secondary to CHF.[14] Numerous oxygen delivery systems can be employed to enrich the patient's fractional inspired oxygen, including flow-by tubing, a face mask or hood, nasal or nasopharyngeal intubation, an oxygen cage, tracheal insufflation, and mechanical ventilation. The oxygen supplementation technique employed should be based on the patient's clinical stability. When choosing the method of oxygen supplementation, the following must be considered:

- What will the animal tolerate in terms of insufflation?
- How long is oxygen supplementation likely to be necessary?

[c]See Figure 1 in the article beginning on page 79.

Table 1. Drugs Used in Emergency Treatment of Dogs with CHF

Drug/Therapy	Dose	Route	Mechanism of Action	Benefits	Side Effects
Carvedilol	1.5 mg/kg q12h. Titrate up as needed	PO	Nonselective α- and β-blocker; antioxidant	β-Blockade, vasodilation	Bradycardia, hypotension, gastrointestinal signs
Digoxin	0.005–0.01 mg/kg q12h. Do not exceed 0.375 mg/day. Therapeutic levels 1–2 ng/ml	IV or PO	Sodium pump inhibition, resulting in increased cytosolic calcium	Weak positive inotropy, decreased ventricular rate	Gastrointestinal signs, digoxin toxicity, drug interactions
Dobutamine	2–20 µg/kg/min	CRI	β_1- and β_2-agonist; mild, dose-dependent α-agonist effects	Positive inotropy, indirect increase in blood pressure	Tachycardia, facial twitching, seizures, tachyphylaxis
Furosemide	2–8 mg/kg; 0.5–1 mg/kg/hr CRI	IV, IM, CRI	Loop diuretic	Diuresis, natriuresis, calciuresis	Hypokalemia, volume depletion
Morphine	0.025–0.2 mg/kg	IV, IM	Pure µ agonist	Sedation, dilation of splanchnic vasculature, respiratory	Depression, nausea, excessive sedation, bradycardia
Nitroglycerin paste	¼–1 inch q8h	Cutaneous	Mixed venous and arteriolar vasodilator	Predominant venous dilation and preload reduction	Hypotension, local dermal reaction
Nitroprusside	1 µg/kg/min; titrate up to effect	CRI	Mixed venous and arteriolar vasodilator	Preload and afterload reduction	Profound hypotension, cyanide toxicity
Oxygen	50–100 ml/kg/min	Inhaled	—	Corrects arterial hypoxemia	At a high setting, lung injury is possible
Pimobendan	0.1–0.3 mg/kg q12h	PO	Arteriodilator and venodilator, positive inotrope, phosphodiesterase-III inhibitor, calcium sensitizer	Efficient positive inotropy, low myocardial oxygen consumption, can be used in acute and chronic failure	Arrhythmias, gastrointestinal signs

- What is the hospital's capacity to administer and monitor oxygen therapy?
- What level of fractional inspired oxygen should be administered?

A complete discussion of oxygen supplementation is beyond the scope of this article, but the topic has been discussed in depth elsewhere.[12,14] In general, regardless of the means of supplying oxygen, patient struggling should be avoided. When an oxygen cage is used, a setting of 65°F with a flow rate of 6 to 10 L/min is often adequate in normothermic animals.[15,16] Although the oxygen setting may initially need to be higher, it should be reduced to 40% or less within a few hours to avoid lung injury.[16] When nasal oxygen is used, delivery of humidified oxygen at a rate of 50 to 100 ml/kg/min is reasonable.

Venodilators

Initial management of cardiogenic pulmonary edema should include the use of an organic nitrate. Topical nitroglycerin is the initial drug of choice. Topical nitroglycerin acts primarily as a coronary artery and venous

dilator, with less of an effect on systemic arterioles.[8,17] By causing dilation, venodilators increase the volume of fluid that the capacitance vessels can accommodate before pulmonary capillary pressure and edema increase. Nitroglycerin comes in a paste formulation that can be spread on the skin, most commonly inside the pinnae. With very poor cardiac output, peripheral circulation is poor and absorption may be diminished. Alternative sites include the axilla, inguinal region, and lateral thoracic wall. The drug should be administered every 8 hours for the first 24 hours, rotating the placement site at each treatment to improve absorption. Human oral formulations and transdermal patch delivery systems are also available; however, the safety and efficacy of transdermal delivery of nitroglycerin in dogs have not been studied.[8,17,18]

Oxygen supplementation plays an integral role in correction of arterial hypoxemia.

ADJUNCT THERAPY

Morphine

Parenteral morphine is used as an adjunct therapy for CHF and cardiogenic pulmonary edema. Although morphine has no direct cardiac effects, it may have beneficial secondary effects. Reported benefits of morphine use in patients with cardiogenic pulmonary edema include the drug's action as a sedative and respiratory center depressant that results in slower and potentially deeper breaths. Additionally, the drug dilates the splanchnic vasculature in dogs and therefore redistributes blood away from the lungs.[19–21] Some cardiologists elect not to use morphine due to its potential emetic side effects. The use of other opiates may be beneficial for their sedative effects; however, these drugs have no documented effect of splanchnic vasculature dilation.

Hydralazine

Hydralazine is a potent pure arteriolar vasodilator that has been used successfully for short-term management of CHF due to valvular disease. Dilation of arterioles results in a decrease in vascular resistance and thus an increase in forward flow, markedly improving cardiac output. Unfortunately, hydralazine has not been shown to be as useful if myocardial disease is present. The major side effect of hydralazine is hypotension. Therapy should be directed to achieve a mean arterial pressure (MAP) of between 60 and 80 mm Hg. Clinical effects should be noted within 1 hour of dosing, with peak levels at 3 hours after administration. We recommend an initial dose of 1 mg/kg PO. The patient's blood pressure should be monitored; if the desired goal is not achieved with the first 1 mg/kg dose, an additional 0.5 to 1 mg/kg can be administered, to a maximum cumulative dose of 3 mg/kg. Once the desired blood pressure has been achieved, the dose can be administered orally every 12 hours.

Additional Therapy for Refractory Cases

Extreme cases of pulmonary edema and respiratory distress may be refractory to standard oxygen, diuretic, and venodilator therapy. In patients with refractory pulmonary edema, CRI of other drugs, such as dobutamine and nitroprusside, should be considered. Therapy should be directed to maintain the MAP above 60 mm Hg, with a target MAP of 80 mm Hg.[6]

Dobutamine

The use of positively inotropic agents in patients with severe, decompensated CHF can be very helpful. The premise of therapy with these agents, based on the Frank-Starling law of the heart, is to reduce ventricular filling pressures and increase cardiac output.[22] The Frank-Starling law of the heart states that the greater the stretch of the myocardium before contraction, the greater the force of contraction will be. Dobutamine is a synthetic catecholamine that acts mainly as a β-adrenergic agonist.[22] Because dobutamine primarily acts on β_1 receptors, its selective inotropic effect appears to be greater than its chronotropic effect.[22] The result is improved myocardial contractility without an increase in heart rate and myocardial oxygen demand. Dobutamine is therefore preferred to dopamine for the treatment of CHF.[22]

At higher doses, dobutamine can have positive chronotropic effects that result in tachyarrhythmia. Therefore, high-dose dobutamine should be used with caution in tachycardic animals. ECG monitoring during dobutamine infusion is very important in critical patients. If dysrhythmias occur as a result of dobutamine infusion, the selected dose should be reduced as long as the systemic blood pressure remains stable.[23] If additional antiarrhythmic therapy is necessary, β-blockers should be avoided because they can be negatively inotropic and antagonize

the effects of the dobutamine. In some patients, tachyphylaxis may develop after 24 to 72 hours of dobutamine therapy due to down-regulation of β receptors.[22,23] This is an unpredictable phenomenon that results in a rapidly decreasing response to a drug after administration for an extended period of time or multiple doses. Another detrimental side effect that has been reported in dogs is facial twitching that may progress to seizures.[24] Dobutamine must be administered as a CRI at a dose of 2 to 20 µg/kg/min. Clinicians should start therapy at the low end of the dose range and titrate to the desired effect.

Sodium Nitroprusside

Sodium nitroprusside is a balanced venous and arteriolar vasodilator that results in decreased systemic and pulmonary vascular resistance.[23,25] The drug is metabolized to nitric oxide inside smooth muscle cells, which activates cyclic guanosine monophosphate and results in vasodilation.[22,24] Nitroprusside causes an immediate decrease in preload, afterload, and left ventricular end-diastolic pressure.[23,25] Additionally, myocardial oxygen delivery is increased relative to demand.[23] In cases of acute, catastrophic CHF that is refractory to standard medical management, nitroprusside should be administered as long as the patient is not hypotensive. Because the drug has a very short half-life, it must be administered by CRI. The CRI should be titrated to effect, starting at the lowest dose possible (1 to 10 µg/kg/min IV). Blood pressure should be monitored at all times because sodium nitroprusside can cause rapid changes in blood pressure and result in profound hypotension. If hypotension develops, the CRI should be slowed or stopped until blood pressure returns to normal. Caution must be exercised when continuing nitroprusside therapy because of potential cyanide toxicosis after approximately 72 hours.[23] Clinical signs of cyanide toxicity include seizures, depression, vomiting, metabolic acidosis, and resistance to the drug. Deleterious side effects unassociated with drug toxicity include hypotension, tachycardia, and vomiting.[22]

It has been proposed that dobutamine and sodium nitroprusside work synergistically in patients with severe, decompensated CHF.[23] Dobutamine increases contractility, while sodium nitroprusside decreases afterload. Because sodium nitroprusside decreases arterial blood pressure, the dose rate may be limited. However, the dobutamine may provide enough forward flow to allow increases in the sodium nitroprusside dose, resulting in better tissue perfusion than that achieved with either drug used alone.[23] In patients with dilated cardiomyopathy, administering dobutamine first to improve contractility and increase cardiac output can offset the hypotensive effects of sodium nitroprusside.

Pimobendan

If the case is not overtly critical and a strong positive oral inotrope is needed, the use of pimobendan (Vetmedin, Boehringer Ingelheim) at 0.1 to 0.3 mg/kg q12h may be helpful in dogs. Pimobendan is a benzimidazole–pyridazinone derivative that is approved for the treatment of CHF due to dilated cardiomyopathy and mitral valve insufficiency in dogs.[26] It has four distinct pharmacotherapeutic properties that make it useful for the management of cardiovascular disease: more positive inotropic activity than digoxin, inhibition of phosphodiesterase-III, sensitization of the myocardium to calcium, and arteriolar and venous dilation.

Because pimobendan has both dilatory and inotropic properties, it has been called an *inodilator*. The positive inotropic activity of pimobendan is unique. Most other positive inotropes increase the amount of calcium in the cytosol, thus increasing calcium availability at the expense of myocardial oxygen consumption. Pimobendan increases the cardiac myocyte's sensitivity to calcium by altering the binding of calcium to the troponin C complex.[26–28] This effect increases sarcomere shortening for a given concentration of cytosolic calcium, thereby increasing contractility.[26–28] This is done with little to no increase in myocardial oxygen consumption. In other words, the calcium-sensitizing property improves efficiency without the deleterious effects seen with other positive inotropes.[26–28]

Initial management of cardiogenic pulmonary edema should include the use of an organic nitrate/venodilator.

Digoxin

Although it is a very weak positive inotrope, digoxin has long been a popular drug for both acute and chronic heart failure therapy. Unfortunately, as with most other cardiac drugs, little meaningful research has demonstrated

a clinical benefit of digoxin in veterinary CHF patients. Digoxin does have some potentially detrimental side effects to consider before use in animals with CHF, including numerous drug interactions, increased myocardial oxygen consumption, cardiac dysrhythmias, vomiting and diarrhea, and worsening of renal function. If used, it should be administered at a dosage of 0.005 to 0.01 mg/kg q12h.

Digoxin slows ventricular rate, especially during atrial fibrillation, to allow better ventricular filling. For this reason, its use is considered by some to be standard practice in the treatment of CHF with concurrent atrial fibrillation.[29]

MONITORING

It is important to monitor all patients with CHF for inadequate perfusion and poor forward flow. Clinical signs of inadequate tissue perfusion include cool extremities, hypothermia, poor pulse quality, cyanosis, prolonged capillary refill time, generalized weakness, mental dullness, and decreased urine output. Parameters that should be monitored include urine output, arterial blood pressure, and oxygen saturation (using pulse oximetry or arterial blood gas measurement). Before any monitoring technique is performed, the benefits of the information to be gained should be balanced against the risks of stressing the critical patient.

Urine Output

Urine output can be measured either by placing an indwelling urinary catheter or by weighing bedding before placing it into the cage and subtracting this amount from the weight of the bedding after the patient has voided. One milliliter of water (urine) weighs approximately 1 g. Normal urine output in a euvolemic patient should be no less than 1 ml/kg/hr. Weight gain may indicate that the patient is continuing to retain fluid and has not had adequate diuresis. As mentioned above, weight gain or loss can be used to direct diuretic therapy.

Blood Pressure

Blood pressure monitoring gives the veterinarian important information that can be used to tailor therapy to improve preload, decrease afterload, and improve contractility. To avoid exacerbation of hypotension, baseline blood pressures should be obtained in all animals with CHF before the initiation of parenteral venodilator therapy.

In veterinary medicine, the most commonly used indirect means of measuring arterial blood pressure are the Doppler and oscillometric methods. Direct blood pressure measurement is not as frequently used because it entails cannulation of an artery and the patient may be too stressed to withstand the physical restraint required for catheter placement. Local anesthetic nerve blocks can be used to reduce patient stress if arterial catheterization is absolutely necessary. The oscillometric technique has the advantage of providing heart rate, systolic and diastolic pressures, and MAP. The Doppler method provides reliable and repeatable measurements of systolic pressure and heart rate. Although techniques that allow measurement of diastolic pressure using Doppler have been described, they are very user dependent and subjective and are, in our opinion, invalid. The target MAP should be 80 mm Hg, with a minimum MAP of no less than 60 mm Hg.

Oxygen Saturation

A pulse oximeter is a noninvasive tool that measures arterial hemoglobin saturation (SaO_2) at the level of the capillary. All patients that may have circulatory compromise should be monitored with a pulse oximeter.[30] These devices provide clinicians with the patient's SaO_2 without serial arterial blood sampling. An SaO_2 of 95% is equivalent to a PaO_2 of 80 mm Hg, as seen on the oxyhemoglobin dissociation curve. An SaO_2 of 90% is equivalent to a PaO_2 of 60 mm Hg.

Like any other piece of equipment, pulse oximeters are not fail-safe. It may be impossible to obtain an accurate reading in patients that are in severe circulatory compromise and therefore peripherally vasoconstricted. One means of double-checking pulse oximeter readings is to evaluate the patient's heart rate using a second monitoring method—if the heart rate displayed on the oximeter does not match that of the patient, the SaO_2 likely is not accurate either. Pulse oximeter readings of less than 90% require intervention with supplemental oxygen. If the oximeter reading for a patient on oxygen supplementation is less than 80%, aggressive intervention such as mechanical ventilation and positive end-expiratory pressure should be considered.

Heart Rate and Rhythm

Electrocardiography is useful in monitoring patients' responses to therapy. It also allows identification of dysrhythmias, which can develop as a result of any condition that involves poor tissue perfusion, hypoxemia, or ischemia.[2] Electrocardiographic monitoring should be

considered for all patients with CHF, particularly those that have demonstrated evidence of a dysrhythmia on thoracic auscultation or pulse palpation and those that are being treated with a potentially proarrhythmogenic drug, such as dobutamine. Electrocardiography has been used as a prognostic indicator in humans with dilated cardiomyopathy; it has also been used to identify predictors for mortality.[31] The use of standard bipolar leads I, II, and III allows monitoring of rhythm and rate. If the unipolar limb leads aVR, aVL, and aVF are added, the mean electrical axis can be evaluated, thus assisting in diagnosing chamber enlargement and intraventricular conduction defects.

Electrolyte Levels

Monitoring of serum electrolytes should play an important part in the acute management of heart failure patients. It also plays an important part in chronic medical management. Renal disease, acid–base disturbances, increased catecholamines, and the use of diuretics such as furosemide must all be considered as potential causes of electrolyte disturbances, which can further exacerbate underlying clinical disease. Electrolyte abnormalities should be identified and treated before further morbidity or mortality results.

Potassium

Hypokalemia has primary arrhythmogenic effects, decreasing the refractory period and coupling interval for ventricular extrasystolic beats.[32] Conduction through the AV node can be depressed due to hypokalemia.[32] Caution should be exercised when using digoxin in patients with abnormal potassium levels. When serum potassium is decreased, the heart can become sensitized to digoxin-induced dysrhythmias. On the other hand, hyperkalemia can result in tolerance of higher serum digoxin levels. Electrocardiographic changes associated with hyperkalemia tend to be a concentration-dependent continuum. The first changes may be larger, peaked T waves. Then decreased R-wave amplitude, followed by prolonged QRS and PR intervals and possible ST-segment depression, may be noted. Finally, P-wave amplitude may decrease, eventually resulting in atrial standstill (absent P waves).[33]

Magnesium

Magnesium depletion appears to play a clinical role in CHF patients because it may exacerbate clinical signs, especially when diuretic therapy is used.[32] The documented incidence of hypomagnesemia in dogs with CHF that have been administered diuretics is low. In one study,[34] there was no significant difference in serum magnesium concentration between control dogs and dogs with heart failure treated with diuretics with or without digoxin. Reported clinical signs of hypomagnesemia and depletion of intracellular stores in humans include cardiac dysrhythmias, poor contractility, and vasoconstriction.[33] Electrocardiographic changes associated with hypomagnesemia may include a widened QRS complex and peaked T waves followed by a prolonged PR interval.[33] The role of magnesium is established in human CHF and likely will become more of an issue in veterinary medicine when additional animal studies are done.

CONCLUSION

Regardless of the underlying cause of CHF, the initial catastrophic events that result in presentation of the emergency patient should be handled similarly. Stabilizing the patient, correctly diagnosing the problem, and initiating aggressive treatment and management are important. Standard treatment has changed little over the years. The most critical therapy includes loop diuretics and oxygen. More recent additions to the therapeutic arsenal include morphine, nitrates, sedatives, and vasodilators. Refractory cases require more intense and aggressive therapy, possibly including intravenous positive inotropic agents and intravenous vasodilators, and should be monitored accordingly.

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ARTICLE #2 CE TEST

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1. Which combination of drugs constitutes the most critical therapy in treating CHF?
a. diuretics and oxygen
b. diuretics and sedatives
c. diuretics and positive inotropes
d. oxygen and sedatives

2. _____ are considered to be the most critical therapeutic drug class in acute and chronic CHF.
a. Diuretics
b. Sedatives
c. Positive inotropes
d. Vasodilators

3. Respiratory depression, sedation, and splanchnic vessel dilation are all properties of
a. acepromazine.
b. morphine.
c. nitroglycerin.
d. dobutamine.

4. Dobutamine is a synthetic catecholamine that mainly acts as a
a. diuretic.
b. vasodilator.
c. β-adrenergic agonist.
d. none of the above

5. Doppler blood pressure monitoring provides reliable and repeatable measurements of
a. systolic pressure and heart rate.
b. diastolic pressure and heart rate.
c. systolic and diastolic pressures and heart rate.
d. none of the above

6. An Sao_2 of 95% as measured by pulse oximetry is equivalent to a Pao_2 of
a. 60 mm Hg.
b. 70 mm Hg.
c. 80 mm Hg.
d. 90 mm Hg.

7. Side effects of sodium nitroprusside therapy may include
a. vomiting.
b. hypotension.
c. cyanide toxicity.
d. all of the above

8. _____ may indicate that the patient is continuing to retain fluid and has not had adequate diuresis.
a. Development of dysrhythmia
b. Weight gain
c. Cool extremities
d. Vomiting

9. _____ should not be considered as a potential cause of an electrolyte disturbance.
a. Diuretics (e.g., furosemide)
b. Hypertension
c. Renal disease
d. Acid–base disturbances

10. _____ is a potassium-sparing diuretic that may have a role as adjunct therapy in dogs with CHF.
a. Digoxin
b. Furosemide
c. Pimobendan
d. Spironolactone