Cardiac Emergencies in Dogs

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ABSTRACT
Cardiovascular emergencies are life-threatening. The clinical presentation is often spectacular and therapy demands some snap decisions based on a solid theoretical knowledge. Some of the common emergencies are pericardial effusion, bradyarrhythmias, supraventricular tachycardia and acute congestive failure. Diagnosis aids such as pulse oximetry, non-invasive blood pressure monitoring, CVP measurement, electrocardiography, radiography and echocardiography would help in evaluating the critically ill patient. Procedures such as thoracocentesis and pericardicentesis could help the patient tide over the critical condition.

KEYWORDS: Arrhythmias; cardiac tamponade; cardiogenic shock; mitral regurgitation; ventricular tachycardia.

Introduction
Life threatening cardiac disorders include severe congestive heart failure, severe tachyarrhythmias, severe bradyarrhythmias and cardiac tamponade. It is important to recognize these conditions and stabilize the animal as quickly as possible before further investigations are carried out.

History and Physical Examination
Assess quickly but efficiently. Always handle gently and quietly-any stress in handling can kill the patient, but you must be able to assess the patient effectively.

Sedation
If the animal is distressed and becoming frantic, sedation may help in decreasing oxygen consumption thereby improving the patient’s condition, as well as allowing you to conduct your assessment. However, on the other hand, if the animal is using all its ventilatory reserve at rest, sedation may depress respiration enough to cause further decompensation. For dogs, combinations of acepromazine (0.03 mg/kg) and an opiate (buprenorphine 0.015 mg/kg or butorphanol 0.2-0.4 mg/kg) can be given intramuscularly.

Non-invasive Monitoring of Hypoxemia
(Pulse Oximetry)
The saturation of haemoglobin with oxygen in arterial blood (SaO₂) is a useful indicator of hypoxemia. Pulse oximetry is a non-invasive technique to allow continuous monitoring of arterial oxyhemoglobin saturation. Blood contains 4 species of hemoglobin (Hb): 1) oxyhemoglobin (HbO₂), 2) reduced Hb, 3) methemoglobin (MetHb), and 4) carboxyhemoglobin (COHb). In healthy individuals, the latter two are in small concentration. Pulse oximetry measures functional hemoglobin saturation \[\text{SaO}_2 = \frac{\text{HbO}_2}{\text{HbO}_2 + \text{Hb} \times 100}\] and thereby assesses arterial oxygenation. It does not assess ventilation (CO₂ elimination). Hypoxemia may be a late onset sign of deterioration in some cases of respiratory failure, especially when compensatory tachypnea has maintained normal oxygen levels. Accurate pulse oximeter readings are not always possible in every animal owing to probe placement issues, thick or pigmented skin, movement artifact and other factors. Thus, hemoglobin saturation determined by pulse oximetry should always be evaluated in light of the patients clinical condition. Arterial blood gas analysis should be considered whenever pulse oximetry estimation is in question.

Non-invasive Blood Pressure Monitoring
Hypertension may predispose certain ‘target’ organs to injury, particularly the eyes, kidneys and cardiovascular and neurovascular systems. Hypotension is a common consequence of shock, dehydration and certain drug toxicities. Systolic
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Blood pressure >160 suggests hypertension; SBP>200 mmHg recorded on two occasions at least 24 hours apart indicate hypertension, unless the animal was excited. End-organ injury provides supportive evidence of hypertension. SBP<90 indicates hypotension.

Central Venous Pressure (CVP)
CVP directly measures pressure in the great thoracic veins as blood returns to the right heart. Serial or continuous CVP measurement helps assess right heart filling and status of intravascular volume. Evaluation of the direction of change in CVP measurements over time is more relevant than basing diagnostic/therapeutic changes on isolated measurements. CVP generally decreases as venous return decreases. Low CVP suggests hypovolemia. Elevated CVP measurements suggest either right ventricular failure or intravascular volume overload.

Electrocardiography
Assessment of heart rate and rhythm provides information about cardiac chamber enlargement, implies the presence of severe pericardial or pleural effusion and can help assess certain suspected systemic and metabolic disorders (e.g. marked disturbances of potassium or calcium, ischemia, infarction). Continuous ECG monitoring, event recorders or Holter recordings are useful to detect transient arrhythmias.

Radiography
Positioning animals for radiography often causes distress, which may prove fatal in dyspneic animals. It is often better to make an initial assessment based on physical exam and delay radiography until the animal is more stable. If radiography is essential, avoid placing the animal on its back. Pleural Effusion (Fig.2): Perform thoracocentesis if necessary. Rather than obtaining radiographs, it is often safer to attempt thoracocentesis if you suspect there is a large pleural effusion. This can be a life-saving measure if an effusion is present, and generally does little harm if there is no effusion (but use a small butterfly cannula).

<table>
<thead>
<tr>
<th>Cardiogenic Pulmonary Edema</th>
<th>Pleural Effusion</th>
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<tbody>
<tr>
<td>Respiratory distress</td>
<td>Increased Respiratory effort</td>
</tr>
<tr>
<td>No stridor/ stertor</td>
<td>Distended chest</td>
</tr>
<tr>
<td>Pale or cyanotic mucous membranes</td>
<td>Pale or cyanotic mucous membranes</td>
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<tr>
<td>Weak pulses</td>
<td>Quiet lung sounds ventrally</td>
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<tr>
<td>± Murmurs / gallops / arrhythmias</td>
<td>Loud breath sounds dorsally</td>
</tr>
<tr>
<td>± Inspiratory crackles</td>
<td>Ventral dullness on percussion</td>
</tr>
<tr>
<td>(alveolar edema)</td>
<td></td>
</tr>
<tr>
<td>Nasal frothing / coughing</td>
<td>'Scallop' or 'leafing' of lung lobes on radiographs</td>
</tr>
<tr>
<td>pink frothy fluid</td>
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Echocardiography
Diagnostic ultrasound assists cardiac examination when the heart is obscured by pleural effusion; diagnoses pericardial effusion; provides quantitative assessment of cardiac structure (valves; chamber dimensions, wall thickness); assesses systolic (contractile) and diastolic function; quantifies gradients via Doppler echocardiography; detects disturbances of blood flow; detects intracavitary masses (clots, tumors) and helps characterize congenital and acquired heart diseases.
Congestive Heart Failure

In dogs, CHF results most commonly from volume overload caused by chronic degenerative valvular disease (severe mitral regurgitation) or dilated cardiomyopathy. Treatment requires aggressive measures to resolve the congestive state and improve cardiopulmonary function. Furosemide is given as IV boluses (2-4mg/kg q 30-60min) or by constant rate infusion. Vasoactive drugs are added to promote venodilation and/or arterial dilation. Typically, this may include nitroglycerin ointment for mild to moderate edema. In states of life threatening edema in the dog, the potent vasodilator sodium nitroprusside is administered by CRI (2-20µg/kg/min with constant arterial blood pressure monitoring). Alternatively, hydralazine, a potent arteriolar dilator, can be given (2mg/kg PO bid) when pulmonary edema results from mitral regurgitation. Inotropic support using dobutamine (5-15µg/kg/min constant rate infusion) is indicated when severe myocardial failure or cardiogenic shock is present (e.g. dilated cardiomyopathy). The potential role of pimobendan in this circumstance has not been clarified but may provide benefit as well. Digoxin is often considered (dog - 0.005-0.01 mg/kg lean body weight q 12 hrs; cat - 1/4 of 0.125 mg tablet q 24-48 hrs), especially when right-sided heart failure or atrial fibrillation is present. Antiarrhythmic therapy is administered when needed to suppress or abolish ventricular tachyarrhythmias, or to control ventricular rates with supraventricular tachyarrhythmias such as atrial fibrillation. Supplemental oxygen administration is provided. Mechanical removal of effusion is performed if necessary. Volume overload secondary to patent ductus arteriosus is correctable by surgical or occlusion techniques. Other systemic and metabolic disorders may cause or contribute to heart failure including endocarditis, myocarditis, pheochromocytoma, diabetes and hyperthyroidism. Heartworm disease is a treatable cause of right-sided CHF.

With recurrent heart failure, upward drug titration may be necessary. Serum digoxin concentrations should be monitored. Diuretic resistance may occur as heart failure progresses. Some animals are likely to benefit from intravenous furosemide therapy which has higher bioavailability, or a second and third diuretic (e.g. thiazide, 5 to 20 mg daily or spironolactone - 12.5 to 25 mg once to twice daily). It is prudent to assess BUN, creatinine, electrolytes and blood pressure during chronic therapy.

Cardiogenic shock (Myocardial failure) is most commonly associated with dilated cardiomyopathy. Less frequent etiologies include chronic volume overload (e.g. mitral regurgitation, left-to-right shunts) or sepsis. The principal hemodynamic feature of cardiogenic shock is systemic hypotension associated with reduced ventricular pumping (i.e. myocardial failure/ systolic dysfunction). Pulmonary edema, systemic congestion, hypotension and tissue hypoxia result. Acute management may require inotropes (dobutamine CRI), diuretics to reduce congestion, vasodilators such as sodium nitroprusside. ACEI, digoxin, pimobendan and control of sepsis and arrhythmias.

Hospital treatment of CHF

Initial management of cardiogenic pulmonary edema is the same, regardless of cause.

- Furosemide 2-6 mg/kg IV, repeat initial dose hourly until response
- Oxygen by bag, cage or intranasal
- Nitroglycerin patch 1/2-4 cm percutaneously q 8 hours
- Carry out further work-up once stable; may need to continue for 24-48 hours while starting oral therapy. After initial therapy, further investigations are indicated to determine the cause of CHF.

Mitrail regurgitation

In dogs with mitral regurgitation, the mitral regurgitant volume can be significantly reduced (by 50% in some cases) by using an arterial dilator such as hydralazine. This cannot be used if the arterial pressure is already low. Start therapy with furosemide, oxygen and nitroglycerin. If no response within an hour, repeat furosemide and hydralazine at 0.5-3.0 mg/kg PO q 12 hours.

Dilated cardiomyopathy / cardiogenic shock

Start therapy with furosemide, oxygen and nitroglycerin. Allow the patient 15-30 minutes at rest to stabilize and prepare dobutamine.
solution. Start dobutamine at 2.5 µg/kg/min and increase up to 10 µg/kg/min. Watch for adverse effects (tachycardia, arrhythmias, seizures). After 48 hours of therapy, reduce the dobutamine rate by 50% each two hours then stop.

**Congestive heart failure with pleural effusion**
Furosemide, oxygen and nitroglycerin should be administered. Thoracocentesis should be performed using a butterfly cannula or angiocath. One side of the chest is often sufficient.

**Arrhythmias**
Tachyarrhythmias may depress cardiac output, cause hemodynamic impairment or hypotension and result in organ ischemia. Shortened diastolic filling decreases coronary blood flow, reduces myocardial oxygen supply, causes ischemia and results in more serious arrhythmias. Certain tachyarrhythmias may deteriorate by becoming electrically unstable. Hemodynamic impact of tachyarrhythmias are influenced by factors related to underlying cardiac disease and the particular type of arrhythmia (i.e. (a) loss of synchronized atrial systole, (b) altered ventricular activation sequence, (c) rapidity of ventricular rate, (d) timing of ectopic beats relative to preceding P-QRS-T complexes, (e) background vasomotor tone, (f) cardiac effects of antiarrhythmic drugs and (g) underlying cardiac dysfunction or health). Because cardiac output = heart rate x stroke volume, sustained tachycardia may reduce cardiac output and arterial blood pressure.

In atrial fibrillation with rapid ventricular response, ventricular filling shortens due to loss of atrial contraction, variation in cycle length and high ventricular rate. This is worsened by concurrent myocardial dysfunction (e.g. dilated cardiomyopathy) or exercise. Impulses originating in the ventricle (e.g. ventricular tachycardia) alter patterns of electrical activation and reduce stroke volume. Rapid sustained ventricular tachycardia decreases cardiac output, results in hypotension and organ ischemia. Ventricular flutter causes precipitous deterioration and all circulation ceases with ventricular fibrillation. Paroxysms of atrial tachycardia with normal ventricular activation may not cause clinical consequences; multifocal atrial or ventricular tachycardia are more likely to compromise hemodynamics, especially if ventricular function is abnormal.

Electrical instability is increased by rapid ventricular rates and multifocal impulse origination. Depolariza-tions occurring within the preceding T wave are extremely dangerous. The underlying state of ventricular function, systemic and metabolic alterations and concurrent drug or anesthetic agents influence electrical stability. Additional factors include timing of the ectopic impulse (i.e. the earlier the premature complex relative to the preceding T wave, the greater the electrical liability). Tachycardia = >180 bpm in small breed dogs; >160 bpm in large breeds and >220 bpm in puppies. With supraventricular tachycardias, vagal maneuvers may occasionally convert the arrhythmia. Supraventricular arrhythmias may be treated with digitalis glycosides, calcium channel blockers, beta blockers and other agents. Acute management of ventricular tachycardia includes treatment of the underlying cause and lidocaine. Pacemaker implantation may be required to treat high grade AV block (Fig. 3).

**Fig. 3: Third degree Atro-Ventricular block**

**Ventricular Tachycardia**
Rapid, repetitive ventricular extrasystoles can decrease arterial blood pressure and lead to signs of hypotension; in addition, some ventricular tachycardias are electrically unstable and may deteriorate to ventricular fibrillation (Fig. 4).

**Fig. 4: ECG showing Ventricular tachycardia**

In the absence of CHF, treat sustained ventricular tachycardia if:

- Very rapid (>200/min)
- Causing hypotension (<90 mmHg systolic)
- Animal is symptomatic
- Suspect risk of ventricular fibrillation
- Confirm the rhythm diagnosis of VT with an ECG
- Check serum potassium levels
- For sustained VT, administer lidocaine (2 mg/kg/minute bolus; repeat up to 8 mg/kg total dose over 10 minutes)
- If successful, start constant rate infusion at 50 to 70 µg/kg/min
- Avoid propranolol and cimetidine
- Alternative to lidocaine in dogs - procainamide 2 mg/kg/minute

**Bradyarrhythmias**
Sinus bradycardia with ST segment changes. May be associated with hypoxia, may be warning sign of impending cardiopulmonary arrest. Check airway/ventilation, anesthesia/sedation, body temperature, electrolytes. Consider atropine/epinephrine.

**Atrial standstill (hyperkalemia)**
Counteract adverse effects of hyperkalemia. Intravenous fluids (0.9% NaCl) and Calcium gluconate (0.5ml/kg of 10% solution slowly over 5-10 mins) (Fig. 5).

![Fig. 5: ECG showing Atrial stand still](image]

**Cardiac tamponade**
Tamponade occurs when sufficient pericardial fluid accumulates within the pericardial space to increase the intrapericardial pressure above right atrial pressure, causing compression of the right heart. Affected animals may present with weakness (acute pericardial effusions-low output or right-sided heart failure (chronic accumulation of pericardial effusion). Physical findings include muffled heart sounds, distended jugular veins, arterial hypotension and ± pulses paradoxicus.

![Fig. 6: Radiograph showing pericardial effusion](image]

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**Pericardiocentesis**
A catheter or trocar system is used via a right-sided approach (ideally guided by echocardiography initially) or by the palpable cardiac impulse. The ECG is monitored for arrhythmias. Lidocaine is infiltrated locally around the entry site. The patient is placed in a slightly oblique lateral position. The needle/catheter is advanced through the skin and deliberately into the pleural and pericardial space using on hand as a “stop” to prevent sudden penetration. The pericardium can often be detected as it is punctured. If the heart is struck, the needle will “grate” and premature ventricular beats will occur. Once fluid is moving into the catheter hub, the needle is advanced 1-3 mm further and then held stable while the catheter is advanced into the space and manually secured. Owing to the relatively inelastic properties of the pericardium, the removal of even small amounts of effusion may be very beneficial and cause a rapid fall in the intrapericardial pressure.

**References**