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Cardiovascular Therapeutics for Advanced Practitioners Mini Series

Session Three: Management of acute congestive heart failure in dogs and cats

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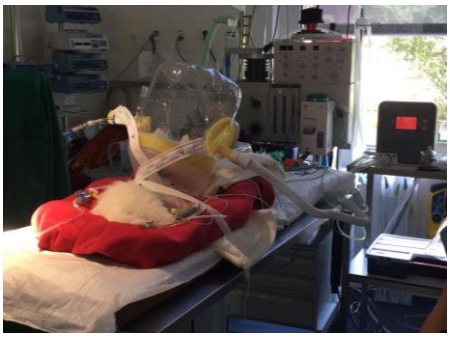
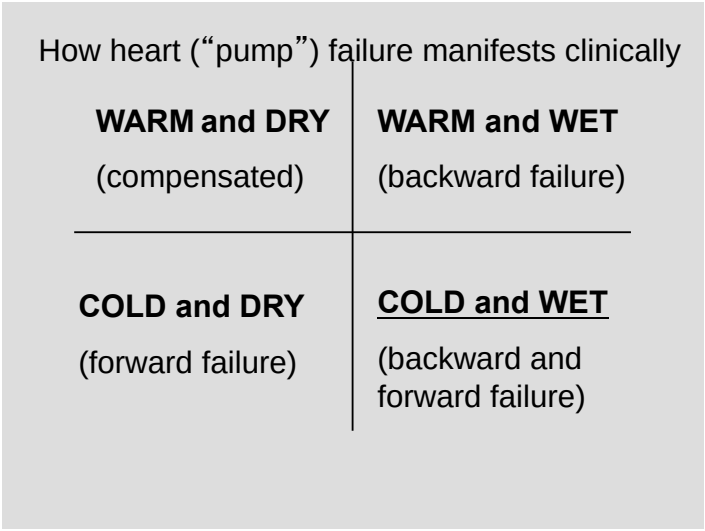


Acute Heart Failure Therapy

Clinical presentation

Clinical presentation of heart failure (HF) will depend on whether the patient is in backward heart failure (congestive), forward heart failure (low cardiac output), or a combination of the two.



Goals of heart failure therapy

Regardless of underlying cardiac pathology (e.g. myxomatous mitral valve disease, dilated cardiomyopathy, hypertrophic cardiomyopathy) the principles for treating HF are similar. The priorities are clearing fluid accumulations, supporting myocardial function (i.e. attempting to improve systolic or diastolic function (or both), decreasing myocardial work load, and controlling heart rate and rhythm to optimise cardiac output).

Severe pulmonary oedema (acute left sided CHF):

- Pulmonary oedema (dogs) and pulmonary oedema and/or pleural effusion (cats) are the most common cardiac emergencies; these patients are typically WARM AND WET.
- Respiratory rate (RR) is very useful indicator of left sided CHF. Resting RR should be monitored in the hospital, and sleeping RR at home should be monitored serially by the owner.

Summary of acute stabilisation (ACVIM Consensus Statement 2009):

- Hospitalisation for cage rest and close monitoring (in a quiet, minimum stress environment if possible)
- Oxygen therapy
- Opioid sedation if patient very anxious
- Furosemide intravenously (or intramuscularly)
- Consideration of whether to use vasodilators +/- positive inotropes
- Thoracocentesis and abdominocentesis as required

Methods of administering oxygen therapy

The choice will depend on practicality and the method that is tolerated best by patient. Arterial blood gas analysis, if available, can be used to evaluate hypoxia. Any level of hypoxia warrants at least temporary oxygen supplementation. A PaO_2 of < 60 mmHg or oxygen saturation of < 90% is an indication for aggressive oxygen therapy. Mechanical ventilation is a consideration, if practical, for any patients with progressive respiratory fatigue, lack of PaO_2 response to oxygen supplementation, or progressive hypercapnia.

Oxygen cages provide 40-90% inspired oxygen in a humidified and temperature controlled environment. Care must be taken to avoid overheating, especially in large or panting patients.

Intranasal oxygen is provided at a flow rate of 0.5 - 1 litres per minute. If administered for more than a few hours, oxygen should be humidified, and an E-collar used to prevent the dog from removing the nasal catheter.

Diuretics

Diuretics are used to control oedema formation. Oedema in CHF is due to increased circulating blood volume. Total circulating blood volume can be increased by as much as 30 % in severe CHF, due to chronic RAAS activation.

Important considerations when using diuretics include the possibility of development of azotaemia or electrolyte imbalances. Renal function should be monitored closely (24 - 48 hours after initiation or a dose increase). Some degree of azotaemia may be unavoidable in patients with advanced heart disease and compromised cardiac output. Consider increasing the frequency of diuretic dosing i.e. from q 12 hr to q 6 - 8 hr, to spread the diuretic effect, rather than increasing the dose. Particularly in inappetant, dehydrated patients on high diuretic doses, it is important to monitor serum potassium levels at least once daily.

Furosemide acts in the thick portion of ascending Loop of Henle. It is the most important diuretic for treatment of acute CHF, with a rapid onset of action. The dosage range is very wide, and initial IV boluses can be followed up with CRI if necessary. The severity of dyspnoea and improvement in RR should be monitored as a guide to treatment success. It is also important to monitor renal parameters and electrolytes, especially potassium. Always aim to maintain the patient on the lowest dose of furosemide necessary to control CHF signs, and avoid severe dehydration and pre-renal azotemia. Furosemide also has some bronchodilating properties when given IV and mild antitussive action by affecting laryngeal nerve sensitivity. This explains why some dogs wrongly treated with furosemide stop coughing, even though they are not in CHF.

Furosemide CRI can be effective in dogs (and cats) with severe left-sided CHF that have a poor response to furosemide boluses. Studies in humans (healthy and CHF) and healthy dogs and horses, suggest that compared to IV boluses, CRI furosemide results in more diuresis, less volatility in plasma volume, more natriuresis and calciuresis, less kaliuresis and possibly less activation of the RAAS.

Positive inotropes

These drugs have beneficial haemodynamic effects in patients with systolic heart failure (HF) due primarily to a direct increase in myocardial contractility and therefore, cardiac output. By increasing the force of myocardial contraction, it is easier for the failing left ventricle to eject blood into the circulation. They are not indicated in cases where augmentation of cardiac output via increased contractility is not possible (e.g. fixed aortic stenosis). There is recent data suggesting that pimobendan has a beneficial effect on survival in cats with HCM and

CHF (Reina-Doreste *et al* 2014), but this is an off-label use. Although this study supports the use of pimobendan in cases with HCM, further prospective studies will be needed before pimobendan is used as a first-line therapy in this scenarium. Pimobendan benefit in cases of obstructive HCM is unknown at the moment, and till further studies are done HOCM should be considered a counterindication for the use of a positive inotrope.

Pimobendan is a calcium-sensitizing drug, acting as a positive inotrope to improve contractility. It is a phosphodiesterase III inhibitor that also decreases cardiac afterload by causing vasodilation, therefore making it easier for the heart to pump blood into the circulation this is why pimobendan is referred to as an inodilator. Pimobendan is available in oral and intravenous form.

Dobutamine is a selective beta-1 adrenergic agonist. It increases contractility with a minimal increase in heart rate and blood pressure. It must be administered via CRI using a syringe pump, and titrated to effect. It has a short half-life, thus rapid onset and offset of action. There is a risk of supraventricular or ventricular tachyarrhythmias with higher doses. Dogs under dobutamine should be under continuous ECG monitoring and blood pressure should be regularly measured. Tolerance to its inotropic effects occurs with prolonged infusion.

Vasodilators

Vasodilators, in particular arterial dilators, allow rapid 'unloading' of the ventricle. They are indicated in patients with pulmonary oedema due to impaired left ventricular function or mitral insufficiency. By decreasing the systemic vascular resistance, it is easier for the failing left ventricle to eject blood into the circulation (lower afterload). In patients with significant mitral regurgitation, the regurgitant fraction is decreased, thus lowering left atrial pressure.

In order to obtain this response, the left ventricle must be able to sustain an adequate stroke volume. Therefore, potent arterial vasodilation is contraindicated in animals with fixed left ventricular stroke volume (e.g. hypertrophic cardiomyopathy, mitral stenosis, aortic stenosis).

Severe pulmonary oedema in animals with fixed left ventricular stroke volume must be managed primarily by preload reduction (e.g. furosemide, dietary salt restriction, venodilators).

Clinical signs of effective arterial vasodilation include bright pink mucous membranes with rapid capillary refill time, warm extremities and gradual decrease in RR and respiratory effort (RE). Clinical signs of hypotension include weakness, collapse, tachycardia and worsening renal biochemistry values.

Balanced vasodilators

Sodium nitroprusside is a rapid-acting and potent 'mixed' vasodilator, causing both preload and afterload reduction. It is mandatory to administer nitroprusside via CRI, using a syringe pump as this drug is extremely potent and the dose must be accurately controlled. It has rapid onset and offset of action and with a half-life of minutes, can be titrated to effect. Use of nitroprusside requires close blood pressure monitoring. Direct arterial blood pressure monitoring is preferable, although Doppler or oscillometric methods can be used if there is no alternative, BUT be aware that these indirect methods tend to be inaccurate at low arterial blood pressures. Target mean arterial blood pressure should be around 70-80 mmHg. Nitroprusside can be used to effectively reduce arterial pressures and provide rapid relief of acute pulmonary oedema. It is especially indicated for treatment of animals with severe and refractory CHF that do not respond to initially therapy.

Known hypotension is a contraindication for administration of nitroprusside, but normal to mildly elevated arterial blood pressure accompanying low cardiac output and CHF signs is an indication for its use. After 48–72 hours of CRI, the risk of toxicity (cyanide) increases. Rapid improvement in clinical signs is usually noted in the first 3–6 hours when nitroprusside is administered concurrently with furosemide and other adjunctive therapies.

Pimobendan see above.

Angiotensin- converting enzyme inhibitors are important in the therapy of chronic CHF but are less helpful in the therapy of acute pulmonary oedema, due to their slower onset of action and relatively mild arterial dilation compared with other, more potent vasodilators.

Arterial vasodilators
Hydralazine is a potent arterial dilator that can be used to treat both acute and chronic heart failure. Indications for use in acute pulmonary oedema are the same as those for sodium nitroprusside. Hydralazine may be chosen for acute therapy if sodium nitroprusside is not readily available, invasive arterial blood pressure monitoring or use of an infusion pump is not possible, or financial constraints preclude the use of a CRI. Contraindications for use of hydralazine are similar to those for sodium nitroprusside. Arterial blood pressure should be obtained prior to therapy to exclude hypotension in an individual patient. Animals with fulminant pulmonary oedema are usually given a higher initial dose of hydralazine than patients with more chronic signs or animals already receiving chronic vasodilator therapy.

Blood pressure is assessed approximately 1 hour after dosing. The target response is a mean (if measured invasively) or systolic (if measured by Doppler sphygmomanometry or oscillometry) arterial blood pressure decrease of up to 20 mmHg, as long as the SAP remains higher than 90 mmHg and MAP remains higher than 60 mmHg. When an effective dose has been documented, that dose is repeated at 12-hour intervals.

Amlodipine is a 2nd generation calcium channel blocker that acts as an arteriodilator by inhibiting calcium influx into the arterial vascular cells. Indications and contraindications for its use are similar to those of hydralazine.

Low cardiac output with pulmonary oedema (COLD AND WET)

This clinical presentation may occur in the setting of severe systolic or severe diastolic dysfunction:

Severe systolic dysfunction is typical of dogs with dilated cardiomyopathy. Decreased systolic function is accompanied by increased filling pressures, but cardiac output is still inadequate. These patients present with clinical weakness, collapse or signs of shock accompanied by pulmonary oedema, with or without arrhythmias. They are most often normotensive or mildly hypotensive, with systolic arterial blood pressures in the range of 80–90 mmHg, but are poorly perfused with cold extremities due to peripheral vasoconstriction. The combination of arterial dilators and positive inotropes often increases cardiac output enough to decrease left atrial pressures and relieve pulmonary oedema, making high doses of furosemide unnecessary.

If oedema does not resolve rapidly enough, low to moderate doses of furosemide may be used to decrease preload. Rapid, aggressive diuresis should be avoided unless oedema is life threatening, because it is important to avoid hypovolaemia/dehydration which might result in worsening of low output signs.

Severe diastolic dysfunction is most commonly seen in the setting of hypertrophic or restrictive myocardial diseases in cats. Acute low cardiac output due to diastolic dysfunction (e.g. HCM or RCM) is usually accompanied by pulmonary oedema. In diseases typified by diastolic dysfunction, systolic function is normal or increased. Relief of pulmonary oedema is achieved by administration of diuretics and venodilators as outlined above, but care must be taken to avoid over-diuresis and arterial hypotension. Arterial vasodilation is contraindicated in acute heart failure caused by diastolic dysfunction.

In severe cases, dobutamine infusions may be indicated to increase cardiac output and relieve oedema. Although dobutamine is usually referred to as a positive inotrope, it also has powerful lusitropic (promoting ventricular relaxation) effects. Administration of a dobutamine CRI to cats with severe pulmonary oedema, together with cautious diuretic therapy, can lead to rapid resolution of pulmonary oedema without induction of severe dehydration. Pimobendan may be useful in such cases as well, although this is currently an off-label use in cats (see above).

In cats that have sinus or other supraventricular tachycardias, administration of a calcium channel blocking agent, such as diltiazem, may improve diastolic function by slowing heart rate. Once pulmonary oedema has resolved, beta-blocking agents can be used instead of calcium channel blockers to slow the heart rate if preferred, but use of beta-blocking agents is contraindicated if CHF is present, or if calcium channel blockers or dobutamine are already being used.

Severe low output failure / cardiogenic shock (COLD AND DRY):

Low output cardiac failure is a challenging emergency presentation. Certain serious cardiac arrhythmias (fast tachyarrhythmias or slow bradyarrhythmias) can lead to clinical signs of low-output failure, so an ECG is mandatory in these patients. Other important differential diagnose is acute pericardial effusion with cardiac tamponade. Low cardiac output can be exacerbated by hypovolaemia in patients with cardiac disease, and is usually the result of complications of CHF therapy (e.g. over-diuresis leading to dehydration).

In an arrhythmic patient a prompt ECG diagnosis and immediate treatment is necessary, e.g. ventricular tachyarrhythmia give lidocaine bolus +/- CRI or in a 3rd AV block with a slow and unstable escape rhythm refer for emergency pacemaker implantation.

Initial assessment of the hypovolaemic patient with low cardiac output failure should include serum biochemical analysis to document evidence of dehydration, organ dysfunction and any electrolyte imbalances. Serum digoxin concentration should be measured if the patient has been receiving digoxin.

The therapeutic plan includes cautious intravenous rehydration. Any electrolyte disturbances or drug toxicities must be addressed, while encouraging cardiac output with positive inotropic medications. Fluids are supplemented with potassium based on measured blood concentrations of this electrolyte. In most cases, ‘maintenance’ rate fluid administration rates (2 ml/kg/h) are sufficient for initial supplementation, and the use of fluid pumps for accurate control of fluid volume administered is highly recommended.

Most patients with clinical signs of hypovolaemia and no current evidence of CHF will benefit from reduction or temporary discontinuation of diuretic therapy.

RR and RE must be monitored very closely as the patient with cardiac disease is rehydrated, as CHF secondary to fluid overload may develop suddenly. Success of fluid therapy is

monitored through physical examination, weight, arterial blood pressure, urine output and blood tests.