TREATMENT OF ACUTE CONGESTIVE HEART FAILURE IN SMALL ANIMALS

UK VET - Volume 7 No 2 March 2002 Peer Reviewed Article CPD

SMALL ANIMAL Cardiology

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Introduction

A good understanding of the underlying pathophysiology is required to enable a sensible approach to the treatment and management of heart failure. Systolic dysfunction predominates in most heart failure patients, but others suffer from diseases that primarily impair diastolic performance. The general approach to treatment is greatly influenced by these distinctions, which must be clinically determined.

In patients with acute, decompensated congestive heart failure, immediate priorities include:

1. Relief of the life-threatening pulmonary oedema and/or pleural effusion
2. Maintenance of adequate systemic arterial blood pressure
3. Adequate delivery of blood to the vital tissues

It should be emphasised that the efficacy of drugs that are used to treat heart failure depends very much on drug type, underlying disease and individual patient response. There are no set rules and it is important to closely monitor a patient with acute heart failure. The treatment should be adapted depending on repetitive clinical evaluation of the patient.

Congestive heart failure

Several classifications have been used to characterise heart failure. From a practical and therapeutic point of view it is preferable to subdivide congestive heart failure into its acute, life-threatening form and more chronic, congestive heart failure. This paper will concentrate on the treatment of acute congestive heart failure.

Acute congestive heart failure

Acute congestive heart failure can develop suddenly with the onset of a new and rapidly progressing disease or from exacerbation of a pre-existing chronic disorder. Acute congestive heart failure being life threatening should be treated aggressively. One should select drugs with a rapid onset of action and proven haemodynamic benefits.

Pulmonary oedema associated with mitral regurgitation or dilated cardiomyopathy

Cage rest

Strict **cage rest** is an essential part of the treatment in acute heart failure. Stress must be avoided. Ancillary investigations to confirm the cause of the acute congestive heart failure may need to be postponed until the patient is stable.

Oxygen supplementation

Arterial hypoxaemia can best be remedied by providing an oxygen rich environment and by rapidly decreasing left atrial and pulmonary venous pressure. An **oxygen** rich environment can be obtained by the means of an oxygen cage. Alternatively and ideally, oxygen

Fig. 1
Intranasal oxygen delivery in a Great Dane with dilated cardiomyopathy.

Fig. 2
Oxygen should preferably be humidified before administration.

(humidified) can be administered via an intranasal catheter providing that it does not cause distress to the animal (Figs. 1 and 2).

Intravenous diuretics and/or thoracocentesis

A potent diuretic will reduce total blood volume and therefore pulmonary oedema. In the presence of life-threatening pulmonary oedema **frusemide** (Lasix® 50 mg/ml) should preferably be administered intravenously (initially 2-4 mg/kg IV q 1-4 hrs in dogs, 1-2 mg/kg IV in cats) as it has an additional and very beneficial venodilator effect when administered by this route (Fig. 3). Dosage should then be reduced (dogs: 2-4 mg/kg q 8-12 hrs; cats: 1-2 mg/kg q 8-12 hrs). Excessive dosing can lead to dehydration, electrolyte depletion, renal failure, low cardiac output and circulatory collapse. Monitoring of renal function including serum electrolytes and urine production is essential.

Fig. 3
Acute heart failure treatment: intravenous frusemide and percutaneous nitroglycerine

Thoracocentesis is required to alleviate respiratory distress due to severe pleural effusion. It is not advisable to drain ascites unless the effusion is compromising respiration.

Vasodilators

Acute reduction of pulmonary venous pressure is achieved by the use of a potent vasodilator to redistribute intravascular fluid volume. Because of the general lack of haemodynamic monitoring in veterinary medicine, topical administration of **nitroglycerine** (Percutol® 2%; Dominion 2.5 cm/20 kg BW q 8 hrs) is the most practical and safe vasodilator. It is a venodilator. It is applied to a hairless, well perfused area of skin (medial pinna or groin) (Figs. 4a and 4b). Gloves should be worn to apply ointment. In addition, a warning sign with information about the site of the nitroglycerine administration should be left on the animal's kennel to protect other staff from inadvertent skin contact, which can cause headaches. It should be avoided in patients with cardiogenic shock. Patients become refractory to nitrates with long-term administration, therefore this product should only be used for three to five days for the initial control of pulmonary oedema.

Fig. 4a

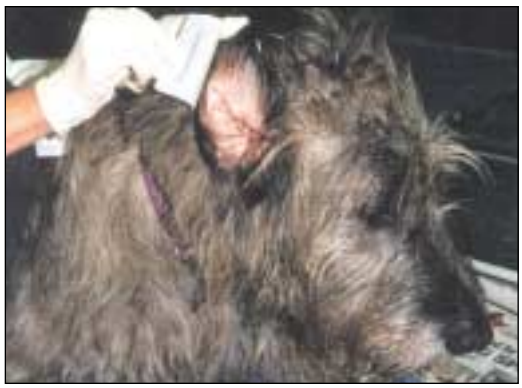


Fig. 4b
Fig. 4b and 4b. Application of nitroglycerine ointment (WEAR GLOVES !!!) on the medial side of the pinna and warning sign.

Intravenous nitroprusside and oral hydralazine should only be used when haemodynamic monitoring is available. Sodium **nitroprusside** (Nipride® 10 mg/ml) is an extremely potent, direct acting mixed vasodilator. Because of its short half-life a continuous rate infusion is necessary to achieve effect. The dose (1-10 µg/kg/min diluted in 5% dextrose) should carefully be titrated towards effect but avoiding profound hypotension. Adverse effects include hypotension, tachycardia, nausea, vomiting and with chronic administration cyanide poisoning.

Alternatively, **hydralazine** tablets (0.5-3.0 mg/kg PO q 8-12 hrs; start low, titrate) can be administered. Hydralazine (Apresoline® 25 and 50 mg tablets) is a very potent arteriodilator. It is more efficient in treating pulmonary oedema secondary to mitral regurgitation than to dilated cardiomyopathy. Hydralazine should be administered with extreme care in dogs already receiving ACE-inhibitors. Common side effects include first dose hypotension and anorexia, vomiting and diarrhoea.

Positive inotropes

Inotropic support is very important with acute heart failure due to dilated cardiomyopathy, and it is also used to treat severely decompensated mitral regurgitation.

Dobutamine is a synthetic catecolamine (β_1-sympathicomimetic drug) that is more efficacious than digoxin for acute management of profound myocardial failure. It can only be used in an intensive care setting. Dobutamine increases contractility with little change in heart rate or afterload. It is very short lived and therefore best suited as a continuous rate infusion (Dobutrex®, Lilly; dogs: 2.5-15 µg/kg/min dobutamine hydrochloride, titrate up to effect). Efficacy is limited following chronic administration because of down-regulation of β-adrenergic receptors, but administration over 3 days may prolong the effect for up to two to three weeks. Serious side effects include ventricular arrhythmias and therefore constant ECG monitoring during dobutamine administration is strongly recommended.

Digoxin is a comparatively weak positive inotrope with a narrow margin of safety. In most cases digitalis glycosides can be administered orally, starting at maintenance dosage (Lanoxin, tablets, GlaxoWellcome; dogs 0.22 mg/m^2). Rapid intravenous digitalisation is rarely necessary in dogs with acute heart failure except to control certain supraventricular arrhythmias.

Treatment of serious arrhythmias

The most commonly encountered arrhythmias in acute heart failure are atrial fibrillation, supraventricular tachycardia and ventricular tachycardia. Substantial haemodynamic and clinical improvement can often be realised by successful treatment. Further details are discussed in a separate article.

Low-output heart failure

Low-output heart failure is often associated with acute cardiac tamponade (pericardial effusion), massive pulmonary thromboembolism, or sudden myocardial depression due to an anaesthetic or due to circulating toxins. Low-output heart failure is treated by resolving the precipitating event (**pericardiocentesis** in case of pericardial tamponade), by optimising preload (intravenous fluid therapy), and by providing inotropic support (see above).

Acute heart failure in feline cardiomyopathy

In cats, the most common reason for acute congestive heart failure is diastolic dysfunction (secondary to hypertrophic or restrictive cardiomyopathy). They do not benefit from positive inotropes or arteriodilators and therapy should be designed to relieve pulmonary congestion and improve ventricular filling. Intravenous **frusemide** (Lasix® 1 mg/kg IV q 1-2 hrs until improvement) combined with topical **nitroglycerine** (Percutol® 2 %; 0.5-0.75 cm q 8 hrs) are advocated in case of pulmonary oedema (Figs. 5a and 5b). Renal function should be closely monitored because cats are very sensitive to diuretics. Thoracocentesis should be performed if the respiration is compromised by pleural effusion. An oxygen cage is preferred over an intranasal catheter because excessive handling and stress can tilt the balance in the already severely compromised feline. Additional drugs can be administered to prevent thromboembolism, reduce heart rate and improve diastolic filling. β-blocking agents (**propanolol** Inderal® 2.5-5.0 mg/cat PO q 8 hrs; **atenolol** Tenormin® 6.25-12.5 mg/cat q 12-24 hrs) and calcium channel blockers (**diltiazem**, Hypercard® 10 mg PO q 8hrs) have been advocated to improve left ventricular filling and cardiac performance in cats with hypertrophic cardiomoypathy. β-blockers are generally more effective than calcium-channel blockers in reducing heart rate, but excessive heart rate reduction can be detrimental in acute congestive heart failure. β-blockers are contra-indicated if concurrent asthma is present because of their bronchoconstrictive effect.

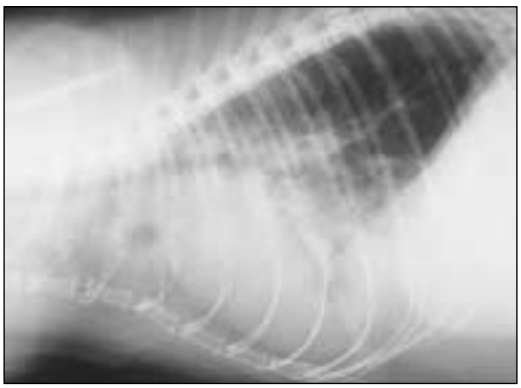
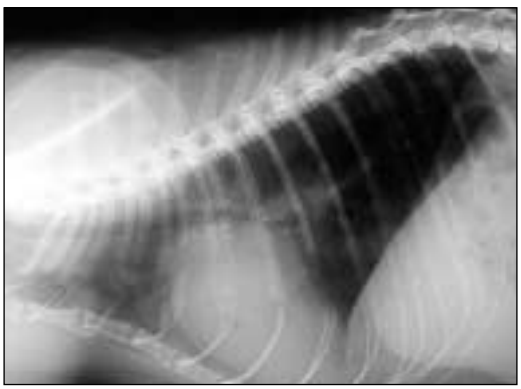


Fig. 5a



Figs. 5b

Figs. 5a and 5b. Lateral thoracic radiographs from a domestic short-haired cat pre- and .post frusemide and nitroglycerine administration for the treatment of severe pulmonary oedema secondary to acutely decompensated hypertrophic cardiomyopathy.

Further reading

Kittleson, M. D. and Kienle, R.D. (1998) **Small Animal Cardiovascular Medicine**. Mosby.

Fox, P. R., Sisson, D. and Moise, N. S. (1999) **Textbook of Canine and Feline Cardiology**. Second Edition. W.B. Saunders company.

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These multiple choice questions are based on the above text. Readers are invited to answer the questions as part of the RCVS CPD remote learning program. ***Answers appear on the inside back cover****. In the editorial panel's view, the percentage scored, should reflect the appropriate proportion of the total time spent reading the article, which can then be recorded on the RCVS CPD recording form.*

1. What is the primary goal in the treatment of acute heart failure?

a. Controlling the arrhythmia.
b. Relief of the life-threatening pulmonary oedema and/or pleural effusion.
c. Avoiding concurrent renal failure.
d. Intranasal oxygen delivery

2. What is the major benefit from intravenous frusemide administration?

a. Rapid onset of action.
b. Additional beneficial venodilator effect.
c. Decreased risk of renal impairment.
d). Anti-arrhythmic properties.

3. Why should nitroprusside and hydralazine only be used in the intensive care setting?

a. Haemodynamic monitoring is essential because they can cause profound hypotension.
b. They cause vomiting and therefore the animal is at risk from aspiration pneumonia.
c. They induce renal failure.
d. They have arrhythmogenic properties.

4. What is the most practical and safe vasodilator in veterinary medicine for the treatment of acute congestive heart failure?

a. Enalapril tablets
b. Nitroprusside continuous rate infusion
c. Hydralazine tablets
d. Nitroglycerine ointment

5. What is the most common reason for acute congestive heart failure in cats?

a. Diastolic dysfunction
b. Systolic dysfunction
c. Arrhythmias
d. Pericardial effusion