CANINE CONGESTIVE HEART FAILURE

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Simon Swift explains how quick, decisive action against the underlying causes of this disease can prove to be effective, despite the risks of renal failure

TO be able to treat congestive heart failure, it must first be recognised.

Typically, it occurs when cardiac output is not sufficient to meet metabolic demands, or when it can only do so with elevated filling pressures (see Table 1). Many causes have a long asymptomatic period before failure develops. However, it may only be when the dog more obviously develops failure that it is presented in the clinic. For example, the murmur of mitral regurgitation in a cavalier King Charles spaniel may be detected at routine vaccinations years before heart failure is seen.

Heart failure signs include indicators of backward failure due to elevated filling pressures. Examples include ascites from right-sided failure or pulmonary oedema from left-sided failure. Forward failure manifests as low cardiac output, which can result in syncope, exercise intolerance and pre-renal renal failure. Weight loss and breathlessness are also common signs. Severe failure can cause cardiogenic shock with weak pulses, pallor and tachycardia.

Coughing is not a common sign of heart failure, as the cough receptors are not in the alveoli and terminal bronchioles, and, therefore, are not stimulated by early cardiogenic pulmonary oedema. However, left atrial enlargement causing main stem bronchial compression is a common coughing cause in dogs with heart disease (not failure). This is important, as there is no evidence – despite the SVEP and VETPROOF studies – that early treatment with an angiotensinconverting enzyme (ACE) inhibitor is beneficial in dogs with degenerative mitral valve disease before failure develops.

In dogs with preclinical DCM, a small study suggested that an ACE inhibitor might be beneficial. The role of pimobendan in such cases is being evaluated in the PROTECT study.

There have been various attempts to classify the degree of heart failure. Some have been based on human schemes, such as the modified New York Heart Association and International Small Animal Cardiac Health Council Classification schemes. The modified American Heart Association and American College of Cardiology (AHA/ACC) scheme is perhaps more appropriate to veterinary medicine (see Table 2), and may help stratify treatment options.

Acute heart failure

Severe cardiogenic pulmonary oedema (Figure 1) demands urgent treatment if death is to be averted. These patients have no reserve capacity, so stress should be avoided.

Some diagnostic tests may need to be delayed until the patient is stable. In addition, cage rest should be provided to reduce movement. The mnemonic FONZ, also a character from *Happy Days*, can be useful as a way of remembering the management of these cases:

• Furosemide can be used at 2mg/kg to 5mg/kg IV, repeated every one to four hours until the respiratory rate reduces. A constant-rate infusion of furosemide can be used instead, and may provide more effective diuresis. However, it needs careful management, or the patient can become severely volume depleted, with life-threatening electrolyte imbalances.

• Oxygen supplementation will improve tissue oxygenation, initially at 50 to 100 per cent, but reduced to 40 per cent or lower in a few hours to reduce lung damage. It should be delivered via a route that is the least stressful to the patient. This may involve nasal prongs, a mask or an oxygen tent.

• Nitroglycerine ointment can be applied to the skin in a hairless area, such as the skin or groin, at a half to one inch every six hours. It increases venous capacitance and so reduces cardiac filling pressures. However, its effectiveness in congestive heart failure in dogs remains unclear. Wear gloves when applying it.

• Z (ZZZ) stands for sedation, which is used to reduce anxiety. Butorphanol (0.2mg/ kg to 0.3mg/kg IM, morphine (0.025mg/kg to 0.1mg/kg) and diazepam (5mg to 10mg IV) may be used.

Sodium nitroprusside, a potent arteriolar and venodilator, can be used as a constant-rate infusion instead of nitroglycerine. Blood pressure must be closely monitored to maintain a systolic pressure of 90mmHg to 110mmHg. It must be protected from the light, and should not be infused with other drugs. It is usually continued for 12 to 24 hours, and prolonged treatment over 48 hours is associated with cyanide toxicity.

If systolic pressures are already low, systolic support may be required. Intravenous sympathomimetic agents, such as dobutamine or dopamine, can be used at 1mcg/kg/minute to 10mcg/kg/minute. Start with a low dose and titrate upwards with blood pressure. Dobutamine, a synthetic analogue of dopamine, is more commonly used, as it has a lesser effect on heart rate and afterload.

Dobutamine stimulates beta-1 receptors, with only weak effects on beta-2 and alpha receptors. Both are potentially arrhythmogenic, and can produce increased pulmonary and systemic vascular resistance.

Pimobendan is a calcium inodilator with both positive inotropic and vasodilatory properties. Given orally, it is active after one hour and can be used with sympathomimetic agents. Digoxin is not usually used in the acute setting, due to its slow onset of action.

If a large effusion is present, the dog will benefit from drainage of a pleural effusion. Ascites can be partially drained if the volume is causing pressure on the diaphragm, restricting respiratory function. Complete drainage is not recommended, as it is a protein-rich fluid and protein loss is to be avoided. A pericardial effusion should also be drained if cardiac tamponade and rightsided heart failure is present (Figure 2). Cardiac arrhythmias may need treating if the heart rate is affecting output.

Lidocaine would be the drug of choice for ventricular tachycardia, and a third-degree atrioventricular (AV) block may require pacemaker implantation.

Ongoing treatment

Close monitoring of the patient is essential. Respiratory and heart rates, blood pressure and urine output should be recorded initially every 15 minutes, and then less frequently as the heart failure becomes controlled.

Serum biochemistry and blood gas analysis are useful, if available. The frequency and doses of further medication depend on the response to therapy, and the trends are as important as the actual values.

Chronic heart failure

Once acute failure is controlled, diuretics are usually continued to keep the signs of congestive failure under control.

However, treatment goals have changed to try to negate the adverse effects of the neurohormonal activation in response to heart failure and, as a result, the myocardial remodelling and progression of disease that otherwise would occur.

• **Diuretics**

The dose of the loop diuretic, furosemide, can be reduced once the heart failure is controlled. Owners can often become adept at adjusting the dose, based on breathing rate. Although hypokalaemia is a concern, if the dog is eating, it is unlikely. More potent loop diuretics, such as torasemide, have been used.

Spironolactone has become a popular adjunct to furosemide for its anti-aldosterone action, following the RALES trial in humans. It promotes sodium loss and potassium retention, and is called potassium sparing. Spironolactone may decrease cardiac remodelling and fibrosis. Clinical trials suggest a significant improvement in survival in dogs with mitral valve disease. Studies are investigating its role in DCM. The dose is 2mg/kg once daily with food.

Thiazide diuretics reduce sodium and chloride resorption, and increase calcium absorption. Hypokalaemia and dehydration are common adverse effects, and thiazides should be used with caution, introduced at a low dose and titrated upwards to effect. Again, they are added to furosemide as part of the sequential nephron blockade.

• **ACE inhibitors**

ACE converts angiotensin-1 to the more potent angiotensin-2 (Figure 3). Angiotensin-2 is a potent vasoconstrictor that also causes sodium and water retention and aldosterone secretion. In addition, ACE cleaves bradykinin, a vasodilatory peptide.

Studies such as LIVE, IMPROVE, BENCH and COVE have proved the efficacy of ACE inhibitors in canine congestive heart failure with lower mortality and sustained clinical improvement. As a result, they are now standard therapy once dogs develop congestive heart failure. ACE inhibitors result in vasodilation, decreasing the workload of the heart. There may also be beneficial effects in decreasing myocardial remodelling. Although some are more lipophilic, and so should have better tissue penetration, evidence of superiority of one ACE inhibitor over any other is lacking.

• **Calcium inodilators**

Pimobendan increases contractility by increasing the sensitivity of troponin C for calcium.

The phosphodiesterase-3 activity results in vasodilation, reducing cardiac workload and increasing output. Many dogs show marked clinical improvement once pimobendan is started. Some initial studies, such as PITCH and VetSCOPE, suggest an increase in quality of life and longevity. It has been used as an adjunct to ACE inhibitors, but the question as to whether pimobendan is superior to an ACE inhibitor has been answered by the QUEST trial.

This study compared pimobendan with benazepril in dogs on standard therapy for heart failure

caused by degenerative mitral valve disease. The dogs on pimobendan clearly lived longer than those on benazepril (median survival times of 267 days versus 140 days). Pimobendan should not be given with food as this decreases absorption significantly.

Digoxin is a mild positive inotrope acting by inhibiting the Na+/K+/ATPase membrane enzyme. The increased sodium is exchanged for calcium, thereby increasing calcium available for contraction. Perhaps more importantly, digoxin increases vagal tone, and hence it is most useful in treating supraventricular tachycardias, such as atrial fibrillation. It takes five to seven days to reach steady-state levels when given orally, and serum concentrations eight to 10 hours after pilling should be assayed at seven to 10 days. Toxicity results in gastrointestinal signs, depression and arrhythmias.

Amlodipine is a calcium channel blocker that acts peripherally to cause peripheral vasodilation. It has been used to lower blood pressure in dogs with severe mitral regurgitation, in an attempt to reduce the regurgitant fraction and left atrial size. It should be used with caution in dogs already receiving pimobendan, as the interaction between the two has not been evaluated and the increased contractility produced by pimobendan may be adversely affected.

Dietary advice

High-salt diets should be avoided. Several cardiac diets that have moderately restricted sodium levels are available, which may be beneficial once congestive heart failure develops.

Severe sodium restriction is not recommended, as this can stimulate the renin-angiotensin-aldosterone system. Poor appetite is common in dogs with congestive heart failure, and is one component of cardiac cachexia. However, pro-inflammatory cytokines, such as TNF and interleukin-1, also play a role and, as a result, omega-3 fatty acid supplementation may be beneficial. Taurine and L-carnitine may be beneficial in breeds with DCM suspected to be caused by deficiencies in one of these amino acids.

Conclusion

Acute congestive heart failure treatment is an emergency situation requiring urgent action to address the oedema or effusions. Frequent reassessment will help assess response and guide further treatments.

Chronic heart failure is a battle against the harmful neurohormonal stimulation that perpetuates and exacerbates the disease. If the underlying causes can be addressed, the outlook can be favourable (Figure 4). As the disease progresses, doses of diuretics may need to be increased and more diuretics introduced. Eventually, there may be a balance between controlling the heart failure or causing renal failure.

Ultimately, if the underlying cause cannot be addressed, the battle will be lost, resulting in the

death of the patient.

References and further reading

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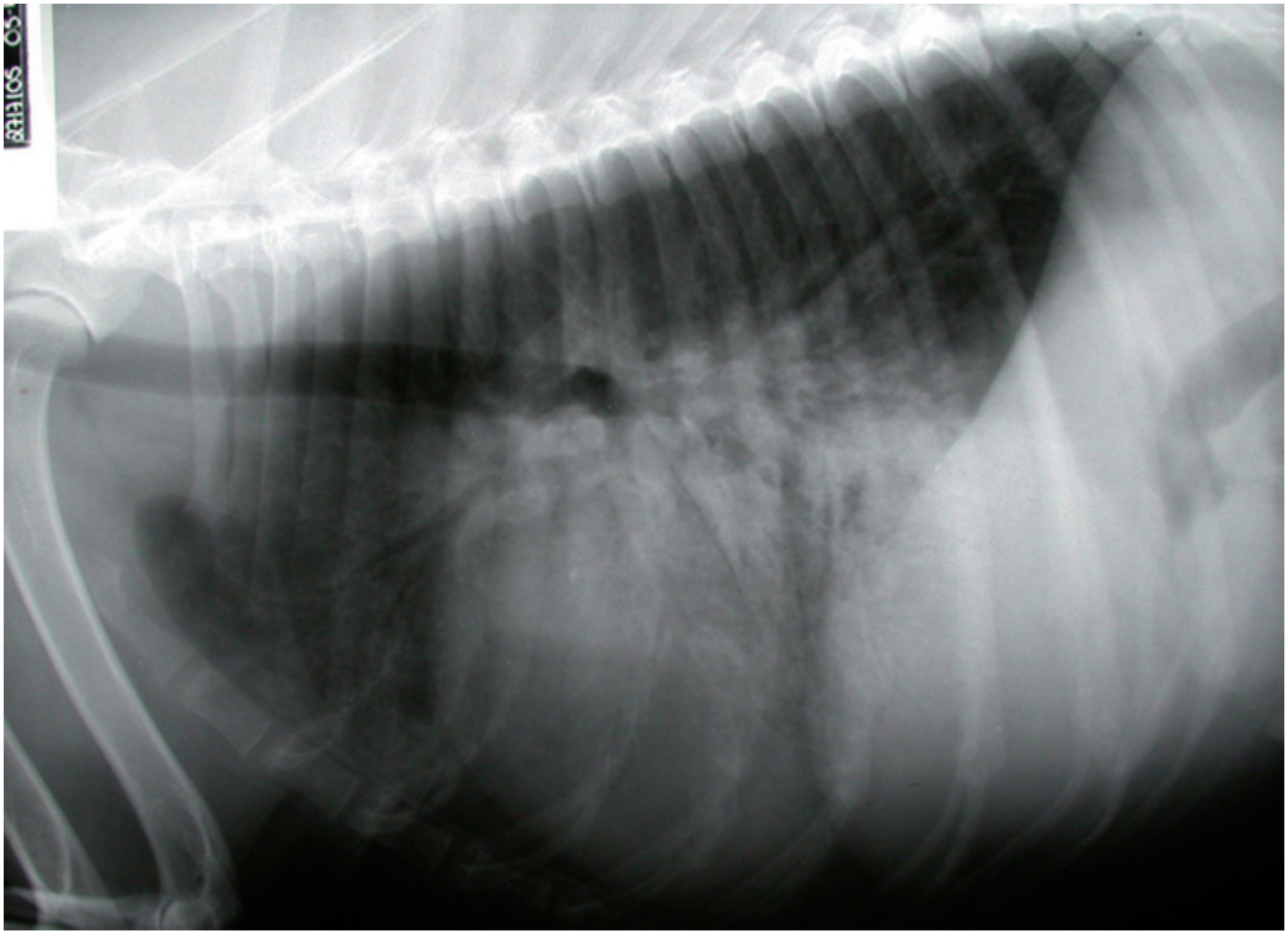

Figure 1 (left). Right lateral thoracic radiograph of a Dobermann with DCM in acute congestive failure showing perihilar and cranioventral pulmonary oedema, with air bronchograms.

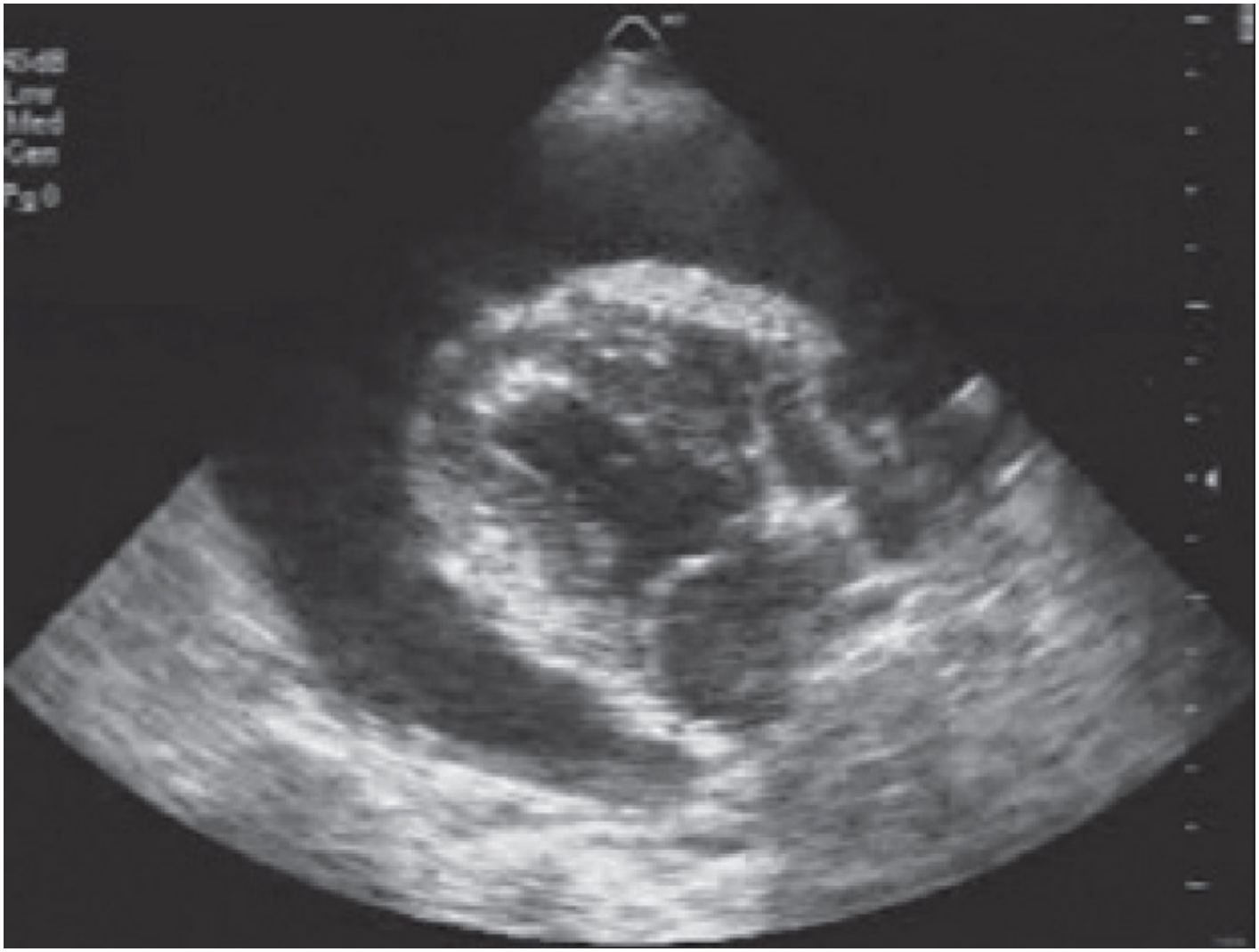

Figure 2. Right parasternal lng axis echocardiogram of a bull mastiff with a pericardial effusion showing cardiac tamponade, with collapse of the right atrium.

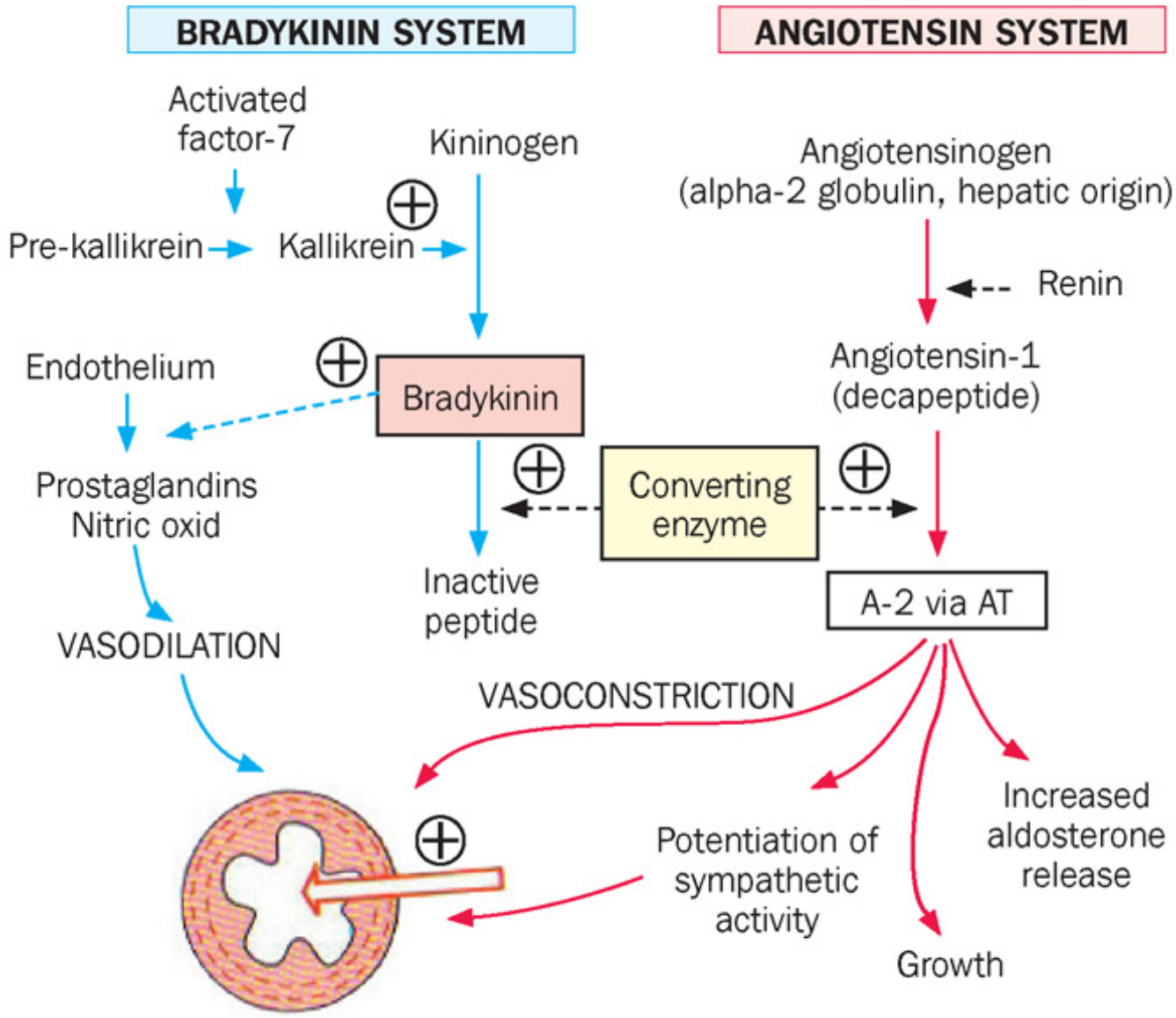

Figure 3. The role of ACE inhibitors in treating chronic congestive heart failure.

Image: OPIE (2004).

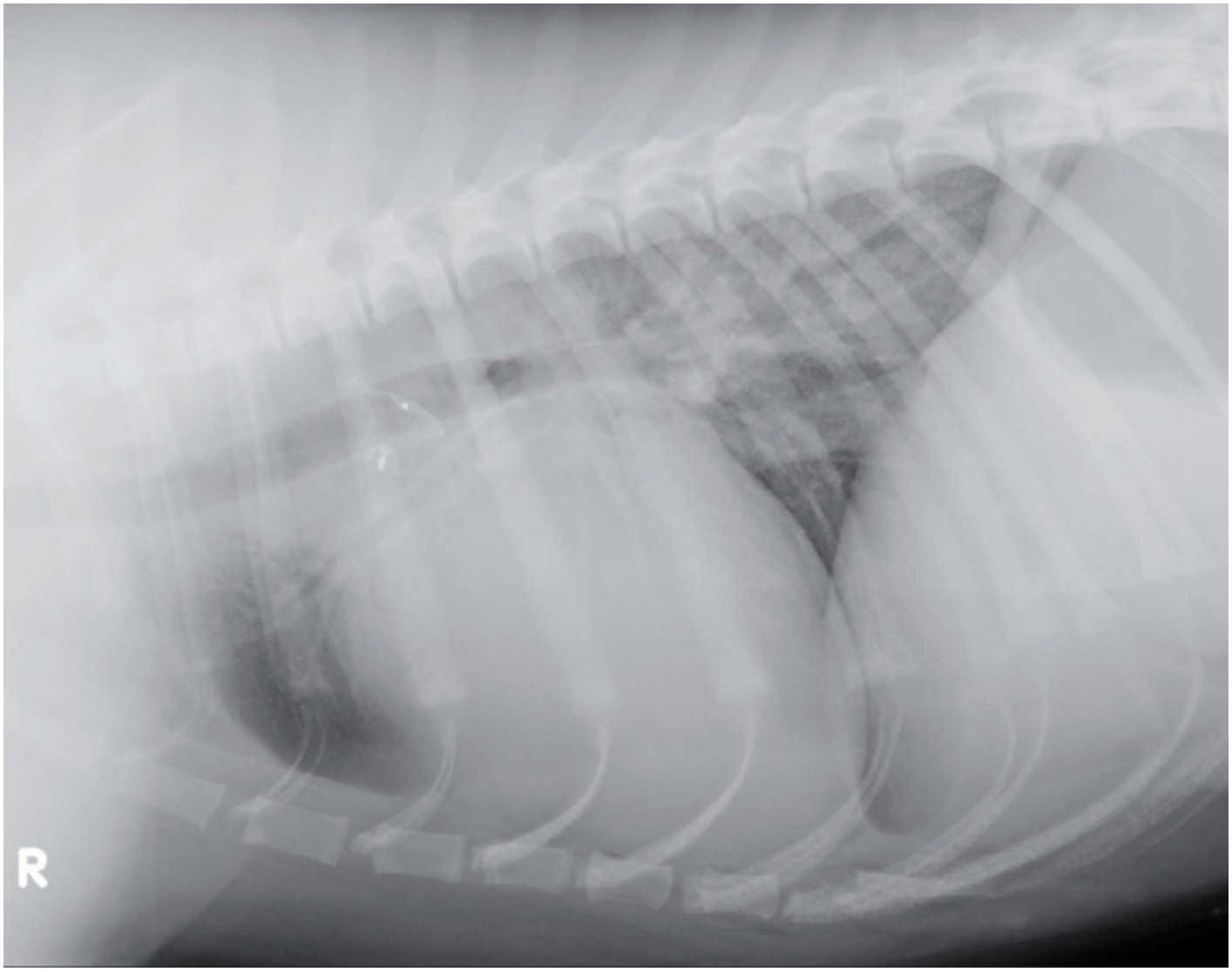

Figure 4. Right lateral thoracic radiograph from a Rottweiller with a patent ductus arteriosus that has been occluded with an Amplatzer device.

Cause	Examples
Primary myocardial failure – systolic	Dilated cardiomyopathy
Volume overload	Degenerative mitral valve disease, patent ductus arteriosus
Pressure overload	Aortic or pulmonic stenosis
Reduced ventricular compliance	Hypertrophic cardiomyopathy Restrictive cardiomyopathy

Table 1. Causes of congestive heart failure

Class	Definition
A	Patient "at risk" of developing heart failure, but with a structurally normal heart
B	Structural cardiac abnormality but no clinical signs of heart failure
C	Structural cardiac abnormality with past or present clinical signs of heart failure
D	Persistent or end-stage heart failure signs, refractory to standard therapy

Table 2. AHA/ACC classification scheme