Monitoring Congestive Heart Failure

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Congestive heart failure (CHF) is a clinical syndrome, which is commonly encountered in small animal practice. It is characterised by the accumulation of fluid as a result of severe heart disease. This fluid accumulation manifests as pulmonary oedema in left sided CHF and in cavitary effusions (pleural, pericardial and ascites) in right sided CHF. The clinical consequences of both CHF itself and its medical management can have a significant impact on the patient's welfare; appropriate monitoring is therefore crucial to maintaining a good quality of life. This article reviews the goals of monitoring in CHF and discusses the methods available in veterinary practice.

Introduction

Congestive heart failure (CHF) results from the increased left and/or right atrial pressures that develop in advanced heart disease in dogs and cats. The underlying causes of these increased

atrial pressures differ according to the underlying heart disease; in myxomatous mitral valve disease (MMVD) the cause is mitral regurgitation, in dilated cardiomyopathy (DCM) it is systolic dysfunction and in hypertrophic and restrictive cardiomyopathies (HCM and RCM, respectively) diastolic dysfunction results in impaired atrial emptying. Regardless of the underlying cause, however, increased left and/or right atrial pressures result in increased pulmonary and/or systemic venous pressures, respectively. The resultant increase in venous hydrostatic pressure favours accumulation of fluid in the extravascular space (Figure 1); this is initially compensated for by increased lymphatic clearance, but once this is overwhelmed pulmonary oedema and/or cavitary effusions develop.

Regardless of the underlying heart disease, the main goal of treatment for CHF is therefore to decrease atrial pressures and thereby decrease the venous hydrostatic pressures driving fluid accumulation. This is achieved primarily by treatment with diuretics, which decreases circulating volume by increasing urinary salt and water loss, thus decreasing cardiac preload. Treatment with ACE inhibitors may further decrease preload, by blunting the renin-angiotensin-aldosterone system, while also decreasing afterload, through their vasodilatory effects. Although some heart diseases are associated with specific treatment considerations (e.g. positive inotropes such as pimobendan should be avoided in cases with outflow tract obstructions such as subaortic stenosis), the cornerstone of treatment remains diuresis. Similarly, the goals of monitoring are similar regardless of the underlying heart disease.

General Monitoring Considerations

Reported survival times for veterinary patients with CHF vary markedly between studies, with much of this variation attributable to differences in treatments (e.g. in one study, median survival in dogs with CHF secondary to MMVD treated with pimobendan was 127 days

longer than in those treated with benazepril (Haggstrom, Boswood et al. 2008)). Nevertheless, it seems reasonable to assume that optimal patient monitoring will also improve both quality of life and survival times, although there is no published data to support this. The overall goals of monitoring in CHF are as follows:

i) To ensure that congestion is well controlled and the clinical consequences of pulmonary oedema/ cavitary effusions are minimised

ii) To ensure that any negative consequences of medical therapy are minimised

iii) To ensure that other consequences of severe cardiac disease (e.g. cardiac cachexia, arrhythmias, decreased cardiac output, etc.) are identified and addressed

Clearly, the first two of these goals require a delicate balance to be struck. The best way to minimise the negative consequences of medical therapy is to minimise the doses of the drugs used; however, the doses chosen need to be adequate to control congestion. Like Goldilocks, we therefore need to find the dose that is "just right" for our patient. However, this decision is complicated by the fact that the optimal dose will vary according to individual patient responses to drugs (especially diuretics) and with disease severity. It is important to remember that dose escalations are typically required as the underlying disease progresses and becomes increasingly difficult to control over time.

a) Response to Diuretics

A range of factors influence the response of an individual patient to diuretics. Firstly, loop diuretics, such as furosemide, need to be delivered to the renal tubule in order to have their action and the oral bioavailability of furosemide is low. It is reported to be 77% in healthy dogs (El-Sayed, Atef et al. 1981), but in dogs with CHF this will be further decreased by factors such as compromised gastrointestinal absorption and impaired renal

perfusion. As a result, the clinician cannot be certain what proportion of the dose administered is actually reaching the renal tubule. Additionally, a phenomenon known as diuretic resistance develops in response to diuretic therapy. Briefly, the mechanisms of diuretic resistance include:

i) Activation of the renin-angiotensin-aldosterone system (RAAS), resulting in increased salt and water retention

ii) Increased proximal tubular reabsorption of salt and water due to the diuretic-induced decrease in extracellular volume

iii) Increased solute delivery to the distal nephrons results in their hypertrophy, allowing compensatory increases in reabsorption

iv) The kidneys become less responsive to the natriuretic peptides, which are released by the heart in response to increased filling pressures and normally result in increased natriuresis and diuresis.

Diuretic resistance develops within 14 days of treatment with furosemide in healthy dogs (Hori, Ohshima et al. 2010); its impact on the dose of furosemide required to control congestion in clinical patients is difficult to assess, but it is likely to contribute to the need to escalate therapy over time.

Torasemide is a loop diuretic with a potency approximately 10 times that of furosemide. It also has a longer half-life and duration of action and higher bioavailability (80-100%) compared with furosemide (Hori, Takusagawa et al. 2007). These characteristics mean that torasemide produces more reliable diuresis and is better able to counteract diuretic resistance, although its potency means that the risk of adverse effects is potentially higher.

b) <u>Negative Consequences of Medical Therapy</u>

The majority of the adverse drug effects seen in animals being treated for CHF relate to diuretic therapy. These include:

i) Dehydration - which, if excessive, may result in decreased cardiac output and hypotension due to the resultant decrease in preload

ii) Hypokalaemia and hypomagnesaemia – which may result in muscle weakness and arrhythmias. Spironolactone, an aldosterone receptor antagonist, is a potassium-sparing diuretic which can help mitigate hypokalaemia.

iii) Azotaemia – a mild pre-renal azotaemia secondary to mild dehydration is common and should not be a cause for concern. As a rough guide, this would include increases in BUN of up to 50% above the upper reference interval and increases in creatinine of up to 25% above the upper reference interval. However, more aggressive diuresis can result in acute kidney injury and/or exacerbation of existing chronic kidney disease

iv) RAAS activation – this can result in increased salt and water retention, sympathetic activation and vasoconstriction (which increases afterload and therefore cardiac work). In experimental models, RAAS activation is also associated with adverse cardiac remodelling and the development of fibrosis.

v) Ototoxicity – although this side-effect is listed in many formularies, it is rarely seen in dogs and cats.

Adverse effects associated with angiotensin converting enzyme inhibitors (ACEi) include hypotension, azotaemia and hyperkalaemia, although the latter is rarely encountered in animals receiving loop diuretics. Pimobendan is generally well-tolerated, although gastrointestinal upsets are occasionally encountered, and some dogs may become

tachycardiac or experience a decrease in systemic blood pressure (BP) (although this is most likely at doses significantly above the recommended range).

From the above it is clear that monitoring of hydration status, BP, renal parameters and electrolytes is crucial in patients being treated for CHF.

c) Cardiac Cachexia

Cardiac cachexia is characterised by loss of muscle mass, with or without overall weight loss, as a result of decreased energy intake, increased protein catabolism, decreased muscle protein synthesis and increased energy requirements. Cardiac cachexia is associated with decreased survival times in both dogs and cats with CHF (Ineson, Freeman et al. 2019, Santiago, Freeman et al. 2020). Monitoring of appetite, body and muscle condition scores and body weight is therefore recommended. Interventions to improve appetite (e.g. to address drug-induced anorexia) and to maintain adequate caloric and protein intake (e.g. avoiding low-protein diets, where possible) should be made proactively.

d) Specific Considerations for Monitoring of Cats

HCM is by far the most common heart disease in cats (Payne, Brodbelt et al. 2015). Both HCM and RCM are characterised by diastolic dysfunction, which results in impaired cardiac filling. The time spent in diastole (and hence the time available for ventricular filling) shortens disproportionately to the time spent in systole as heart rates increase. Diastolic function will therefore be further impaired in cats with HCM or RCM when sympathetic tone increases, leading to further increases in left atrial pressures and a significant risk of decompensation of CHF. This means that a cat that has been well controlled in their home environment may decompensate when exposed to the stress of being brought to the clinic for a check-up. A pragmatic decision to monitor these patients

remotely and only recommend blood tests, etc. if there has been a change in medication doses and/or clinical status (rather than at pre-specified intervals) may be in their best interests.

Methods for Monitoring Congestive Heart Failure

a) Sleeping Respiratory Rate

An ideal monitoring method would be accurate, cost-effective and readily available; criteria that are fulfilled by monitoring of sleeping respiratory rate (SRR) in patients with CHF. SRR is more accurate than resting respiratory rate, as respiratory rate is more likely to be influenced by the environment when animals are awake. Most dogs and cats with medically well-controlled CHF have SRR < 30 breaths /minute, when counted by their owners in their home environment (Porciello, Rishniw et al. 2016). Anecdotally, it appears that involving owners in this way gives them a valuable sense of ownership and partnership in optimising their pet's care. Use of smartphone apps, such as the Cardalis App, makes the process of counting breaths per minute more straightforward (a breath is logged each time the user touches the smartphone screen) and also makes the following of trends over time easier. However, some owners prefer a paper-based log system, or to create their own spreadsheets.

Given the potential adverse effects of the drugs used to manage CHF outlined above, ideally the lowest effective doses (i.e. the minimum doses that control congestion) should be chosen. This is most important in relation to the loop diuretics; these drugs contribute significantly to the potential adverse effects seen and they have wide dose ranges, allowing the dose to be tailored to the needs of the patient. In contrast, the doses of most other drugs used to manage CHF (e.g. ACEi, pimobendan and spironolactone) are

relatively standardised and less likely to be adjusted than those of the loop diuretics. SRR can be used to help the clinician and the owner determine the lowest effective dose of the loop diuretic. This is achieved by asking the owners to sequentially reduce the loop diuretic dose every 3-5 days while vigilantly monitoring SRR; if the SRR starts to increase, the dose should revert to that prior to the most recent dose reduction.

The author recommends that owners start to monitor SRR in dogs and cats with known heart disease once they develop evidence of cardiomegaly (e.g. left or right atrial enlargement). This is a valuable way to identify the development of CHF at the earliest possible opportunity. Early identification allows early intervention, so that medical control of CHF may be achieved before animals become dangerously dyspnoeic and unstable. As well as improving patient quality of life, this early intervention can result in significant financial savings for the owners by minimising the length of hospitalisation. However, in some cases (especially in cats) there is no clinical evidence of underlying heart disease prior to the onset of CHF, and so pre-emptive monitoring of SRR is not always possible. Once an animal is stabilised on medical therapy for CHF, the author recommends daily monitoring of SRR. If the SRR is suddenly and persistently >40 breaths/ minute the author recommends that the owner give an additional dose of diuretics and contact a veterinary surgeon. If the SRR is trending consistently upwards over several days, the author recommends that the client contacts a veterinary surgeon before making any dose adjustments.

b) Blood Tests

As mentioned above, the monitoring of hydration status, renal parameters and electrolytes is crucial in patients being treated for CHF. Additionally, circulating N-terminal pro-B-type natriuretic peptide (NT-proBNP) and cardiac troponin I (cTnI) can be measured as markers of myocyte stretch (i.e. increased filling pressures) and injury, respectively.

i) Packed cell volume and total solids (PCV/TS) – measurement allows assessment of the degree of dehydration. Modest increases in PCV and TS are expected in response to diuretic therapy, as the goal is to decrease circulating volume in order to improve congestion. However, excessive dehydration is counterproductive, potentially leading to greater RAAS activation, azotaemia, etc.

ii) Renal parameters – measurement of BUN and creatinine is most commonly used to monitor the renal effects of treatment for CHF. It is important to remember that diuretics have their effects by compromising the ability of the renal tubules to concentrate urine; urinary specific gravity (USG) will therefore be decreased in animals receiving treatment for CHF and USG cannot be used to assess underlying renal function in these patients. A mild prerenal azotaemia is a commonly anticipated change, secondary to dehydration, as described above. This often manifests as a mild increase in serum BUN, often with little change in serum creatinine. However, the development of a more marked azotaemia is a cause for concern (especially if the increases in BUN and creatinine are rapid, if the patient has known underlying chronic kidney disease (CKD), or if the patient is showing clinical signs referable to uraemia (nausea, inappetence, vomiting, etc.). Management in such cases often involves a careful balance between achieving sufficient diuresis to control congestion while minimising the impact of therapy on renal perfusion. It is often possible to address this by withdrawing ACEi treatment and reducing diuretics to the lowest effective dose (see SRR section above). In cases with sudden onset uraemic signs and marked, rapid increases in BUN and creatinine, consistent with an acute kidney injury (AKI), diuretic and ACEi therapy may need to be withdrawn completely, at least temporarily; in some cases, fluid administration may be required (careful enteral administration, e.g. via a naso-oesophageal tube, is generally safer than intravenous

fluid administration). In some cases, especially those that have sustained an AKI or with pre-existing CKD, it may not be possible to find a balance between congestion and uraemia that allows an acceptable quality of life, and so euthanasia may be the most appropriate course of action.

iii) Electrolytes – generally, sodium, potassium and chloride are routinely monitored in dogs and cats being treated for CHF. Although hypomagnesaemia may result from diuretic therapy and may increase the risk of arrhythmias, its measurement is typically only considered if severe arrhythmias develop, as this test is less widely available. As mentioned previously, the most common electrolyte abnormality associated with loop diuretic and ACEi treatment is hypokalaemia, which may result in muscle weakness and arrhythmias (severe hypokalaemia is associated with ventricular arrhythmias, including ventricular tachycardia). If hypokalaemia develops, management options include decreasing loop diuretics to the lowest effective dose (see SRR section above), addition of spironolactone to the treatment regimen (if the patient is not already receiving this drug), withdrawal of ACEi, oral supplementation of potassium, or a combination of these strategies. Dilutional hyponatraemia may develop in patients with severe CHF, due to increased antidiuretic hormone release and insufficient tubular flow through the distal nephron. Although it is generally recommended that care is taken with salt intake in patients with heart disease (e.g. feeding a diet with a healthy salt content and avoiding salty treats), it may be necessary to supplement the diet with salt if dilutional hyponatraemia develops. Additionally, it should be remembered that markedly salt restricted diets tend to be unappetising, which can exacerbate cardiac cachexia.

iv) Cardiac biomarkers – both NT-proBNP and cTnI have been shown to be useful in the differentiation of cardiac from non-cardiac respiratory distress and for prognosis in

CHF in both dogs and cats (Hezzell, Boswood et al. 2012, Borgeat, Payne et al. 2014, Fox, Oyama et al. 2015, Hezzell, Rush et al. 2016). However, their usefulness for monitoring of CHF is less well characterised. It appears that SRR is a more useful method of monitoring for worsening CHF than circulating NT-proBNP measurements (Schober, Hart et al. 2011); SRR has the added advantage of being achievable by the owner in their home environment, as frequently as they wish, without incurring any financial cost. NT-proBNP measurements have also been used to guide therapy in human CHF patients, with the goal of improving outcome. An observational study of dogs with CHF showed that those with lower plasma NT-proBNP measurements following initial treatment for CHF had longer survival times (Wolf, Gerlach et al. 2012), and a second study showed that the use of a prespecified treatment algorithm results in decreases in plasma NT-proBNP (Hezzell, Block et al. 2018). However, whether the use of this algorithm to guide therapy results in improved outcomes in dogs remains unknown. It is also important to remember that there is significant day-to-day and week-to-week variability in plasma NT-proBNP measurements in an individual patient, such that values must change by at least 60% before a true change can be considered to have occurred (Winter, Saunders et al. 2017).

c) Blood pressure monitoring

As mentioned above, hypotension is a potential consequence of treatment with diuretics, ACEi and, rarely, pimobendan. Additionally, low output failure (i.e. compromised cardiac output) may develop as a result of diseases associated with systolic dysfunction (e.g. DCM, arrhythmogenic cardiomyopathy (ARVC) and end-stage disease of any aetiology), severe outflow tract obstruction (e.g. subaortic stenosis) or severe diastolic dysfunction leading to sufficient compromise of preload. Monitoring of non-invasive BP measurements and intervention to ensure adequate perfusion is maintained is therefore

important in patients receiving treatment for CHF, with a goal of maintaining systolic BP >100 mmHg. Non-invasive measurement using either Doppler sphygmomanometry (systolic BP) or the oscillometric method (systolic, diastolic and mean BP) is available (Figure 2). Options to address hypotension include decreasing the doses of loop diuretics to the lowest effective dose (see SRR section above), withdrawal of ACEi therapy, increasing the dose of pimobendan (if the cause is believed to be compromised systolic function some veterinary cardiologists will increase the dose of pimobendan by 50%, although it is important to note that this recommendation is off license). If in hospital treatment is required, pressors (e.g. dobutamine) can be used to maintain perfusion; however, if systolic BP >100 mmHg cannot be maintained outside the hospital environment and the patient's quality of life is compromised, euthanasia should be considered for the welfare of the patient.

d) Radiography

The author does not recommend routine thoracic radiography at pre-specified intervals, although the initial diagnosis of CHF should be radiographically confirmed in all patients, unless it is not safe to do so (e.g. it is often unsafe to restrain severely dyspnoeic cats for radiographs) (Figure 3). Instead, radiographs are taken if the patient's clinical status has changed and the underlying cause is uncertain (e.g. an increase in SRR does not respond to an increase in diuretic dose as expected, or an animal starts to cough without an increase in SRR). It is important to keep an open mind and not assume that changes in respiratory signs are necessarily attributable to worsening CHF; many dogs and cats with heart disease have concurrent airway disease, and other differentials (e.g. neoplasia, pneumonia, etc.) may also be investigated radiographically.

e) Electrocardiography

The prevalence of arrhythmias in dogs and cats varies widely according to the underlying heart disease. For example, ventricular arrhythmias are a cardinal feature of ARVC, but are documented far less frequently in dogs with MMVD. The author recommends that an electrocardiogram (ECG) is recorded in any patient in which an abnormal rhythm and/ or pulse deficits is appreciated on physical examination. Additionally, an ECG should be recorded if clinical signs potentially explained by a persistent or intermittent arrhythmia are reported (e.g. weakness, syncope, etc.). In the case of an intermittent arrhythmia a Holter monitor (allowing recording of an ECG in the patient's normal environment for 24 hours or more) may be required to document the rhythm at the time of an episode (e.g. of syncope) (Figure 4). A Holter monitor is also useful to assess and monitor the effectiveness of antiarrhythmic medication (e.g. to assess the adequacy of rate control in patients with atrial fibrillation, or the frequency of ventricular arrhythmias in patients with ARVC).

f) Echocardiography

In current veterinary practice, echocardiography is the central modality used for the diagnosis and staging of heart disease in dogs and cats. It allows cardiac structure and function to be assessed in real time and the identification of risk factors for future adverse events, such as the onset of CHF (e.g. left atrial enlargement and increased filling pressures) or arterial thromboembolism (e.g. left atrial enlargement, decreased left atrial function and the presence of spontaneous echocontrast +/- intracardiac thrombus). However, once the underlying heart disease has been characterised and the presence of CHF confirmed (ideally radiographically), echocardiography is less important for routine monitoring than the methods described above.

SRR is a more useful method of identifying worsening CHF than echocardiographic changes (or plasma NT-proBNP measurements, as mentioned previously) (Schober, Hart et al. 2011).

In general, the author recommends repeating echocardiography if the patient's clinical status has changed, rather than at routine, pre-specified intervals, similar to the recommendations for repeating radiography outlined above. Examples of how echocardiography can contribute valuable information in this setting include the identification of a ruptured chorda tendineae causing acute decompensation of CHF or the development of pulmonary hypertension leading to the development of right-sided congestive heart failure.

g) Lung Ultrasound

Lung ultrasound is a useful method to identify extravascular fluid in the lungs and is commonly used as an adjunct to echocardiography in patients with CHF and a standardised examination protocol, known as Vet BLUE, has been described (Lisciandro, Fosgate et al. 2014, Vezzosi, Mannucci et al. 2017). Normal air-filled lung creates reverberation artefacts on ultrasound, known as A lines, which appear as horizontal lines across the image. In contrast, the presence of fluid in the lungs creates B lines on ultrasound, also known as comet tail artefacts or lung rockets, which are orientated parallel to the ultrasound beam. Dogs and cats with healthy lungs have very few or no B lines on lung ultrasound; in contrast, patients with CHF have large numbers of B lines (Lisciandro, Fosgate et al. 2014, Lisciandro, Fulton et al. 2017). Lung ultrasound can be performed quickly, with the patient standing or in sternal recumbency and with minimal restraint; this technique is therefore particularly suitable for dyspnoeic, unstable patients. Lung ultrasound can be combined with a brief, focussed cardiac ultrasound examination,

to allow assessment for changes consistent with significant cardiac disease and right-sided CHF (e.g. atrial enlargement, decreased ventricular systolic function and the presence of pericardial and/ or pleural effusions). Additionally, a brief abdominal ultrasound examination will allow detection of ascites.

It is important to remember that B lines are also seen in other causes of increased extravascular fluid in the lungs, including pneumonia and haemorrhage; thoracic radiography therefore remains the preferred modality for definitive differentiation of cardiac from non-cardiac causes of respiratory distress. Nevertheless, if significant numbers of B lines are seen in combination with left atrial enlargement in a patient presenting with respiratory distress, it is reasonable to instigate treatment for CHF. This is especially true in patients that cannot tolerate lateral recumbency due to dyspnoea and are too unstable to be restrained for radiography; many cats will fall into this category. A positive response to diuretic therapy usually confirms the presence of CHF and further investigations, such as more extensive echocardiography and thoracic radiography, can be performed once the patient is more stable.

h) Specific Monitoring Considerations for Right-Sided Congestive Heart Failure

Much of the advice given above applies equally to the monitoring of left- and right-sided CHF, particularly the recommendations for monitoring of BP, hydration status, renal parameters and electrolytes. It is also important to remember that cats with left-sided heart disease often present with pleural effusions. However, there are some differences, which are outlined below.

i) Thoracic and abdominal ultrasound are excellent modalities for the identification and monitoring of pleural and pericardial effusions and ascites, with the advantage of being safer to perform in dyspnoeic patients.

ii) Ascites is typically better tolerated than pulmonary oedema or significant pleural effusion and will consequently have less impact on SRR until a large enough volume has accumulated to significantly compromise breathing.

iii) Measurement of the abdominal circumference can be a useful method for at home monitoring of ascites.

iv) The presence of significant arrhythmias (e.g. rapid atrial fibrillation) or pericardial effusion typically results in right-sided CHF.

Frequency of Monitoring

As with any clinical test, the clinician should always consider how the results will guide their therapeutic plan. If the plan will remain unchanged regardless of the results, then the clinician should question their motivation for performing the test. This is particularly important in the case of cats, as the benefit of any monitoring test must outweigh the potential risk of decompensation inherent in exposing the patient to the stress of a visit to the clinic. With these principles in mind, it is clear that any monitoring plan should ideally be tailored to the clinical needs of the patient; a cat that has a stable SRR, good appetite and good energy levels but becomes very stressed when presented to the clinic would be monitored less frequently than a dog with uncontrolled CHF and rapid atrial fibrillation that tolerates clinic visits well. Nevertheless, some general principles are outlined in Table 1.

Summary

Dogs and cats can continue to have a good quality of life for months or even years after the onset of congestive heart failure. However, judicious monitoring is important to optimise their wellbeing, by ensuring that CHF remains adequately controlled while minimising the

potential adverse effects of treatment, including excessive dehydration, hypotension, azotaemia and electrolyte abnormalities, and the negative effects of the underlying cardiac disease, such as cachexia. Ideally the monitoring regime should be tailored to the needs of the individual patient, with tests being performed in order to answer specific clinical questions, rather than according to a set routine.

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Table 1: Recommendations for monitoring of congestive heart failure in dogs and cats.

Monitoring test	**Frequency**	
Sleeping respiratory rate	Daily (or more frequently if concerned about increases or upward trends)	
Blood tests	PCV/TS, renal parameters and electrolytes:	• Ideally at the onset of CHF • 5-7 days after initial stabilisation of CHF • 5-7 days after adjusting medications (especially diuretics and ACEi) • Every 3-6 months thereafter
	Cardiac biomarkers	• Ideally at the onset of CHF • 5-7 days after initial stabilisation of CHF • Consider measurement every 6 months (some evidence of prognostic value)
Blood pressure measurement	• At each clinic visit • If clinical signs develop that are potentially referable to hypotension (e.g. weakness or syncope)	
Electrocardiography	Standard ECG	• If an arrhythmia is detected on physical examination • If clinical signs potentially referable to a persistent or intermittent arrhythmia are reported

	Holter ECG monitor	• If clinical signs potentially referable to an arrhythmia are reported • 7-14 days after initiation of antiarrhythmic therapy • 7-14 days after any dose adjustment of antiarrhythmic drugs
Radiography	• If new or worsening clinical signs develop, especially those potentially referable to the respiratory system	
Echocardiography	• If new or worsening clinical signs develop, especially those potentially referable to deterioration in cardiac function (e.g. ruptured chorda tendineae, development of pulmonary hypertension, etc.)	

Figure 1: An illustration of Starling's forces under normal conditions. At the proximal end of the capillary (on the left of the diagram), the lower oncotic pressure in the interstitium created by the lower protein concentration favours extravasation of fluid. At the distal end of the capillary (on the right of the diagram), the lower intravascular hydrostatic pressure favours movement of fluid back into the capillary. However, if pulmonary venous pressure increase, the distal capillary hydrostatic pressures also increase, preventing this inward movement of fluid and leading to the development of interstitial pulmonary oedema.

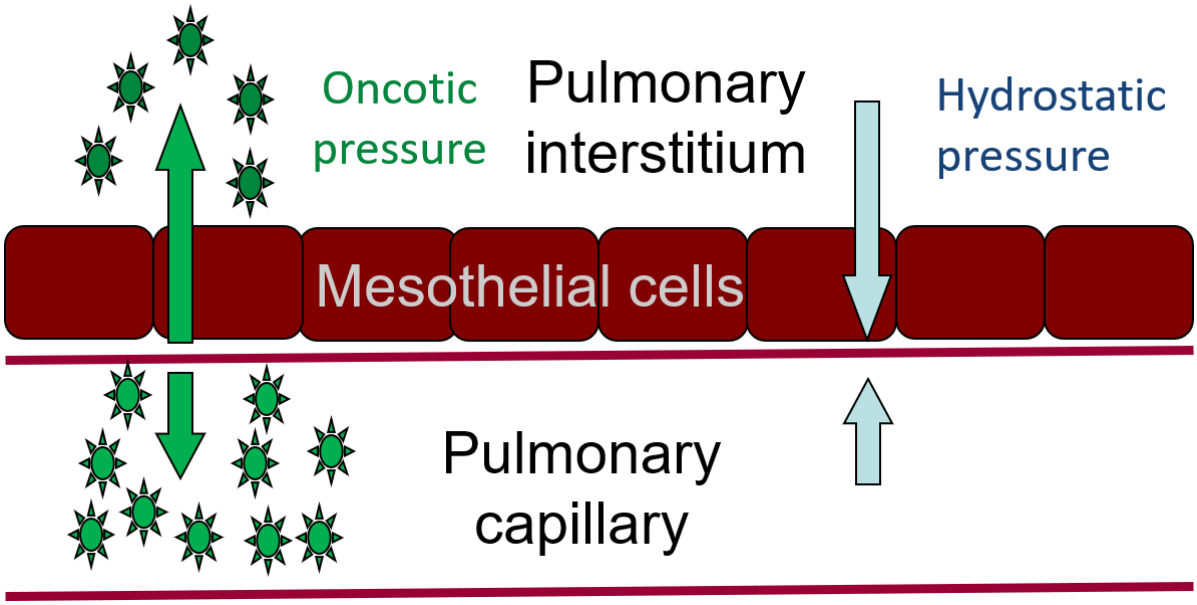
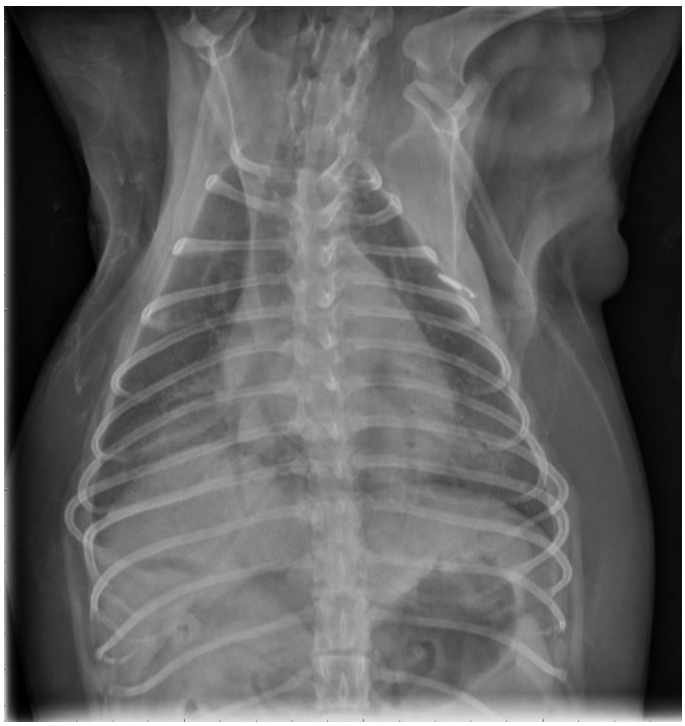


Figure 2: Non-invasive measurement of systolic blood pressure by Doppler sphygmomanometry.

Figure 3: Lateral and ventrodorsal radiographs of the thorax of a dog with cardiomegaly, pulmonary venous congestion and a caudodorsally distributed heavy interstitial to alveolar pulmonary pattern, consistent with cardiogenic pulmonary oedema. These are the three changes typically seen in dogs with CHF; it should be remembered that the distribution of cardiogenic oedema is more variable in cats.



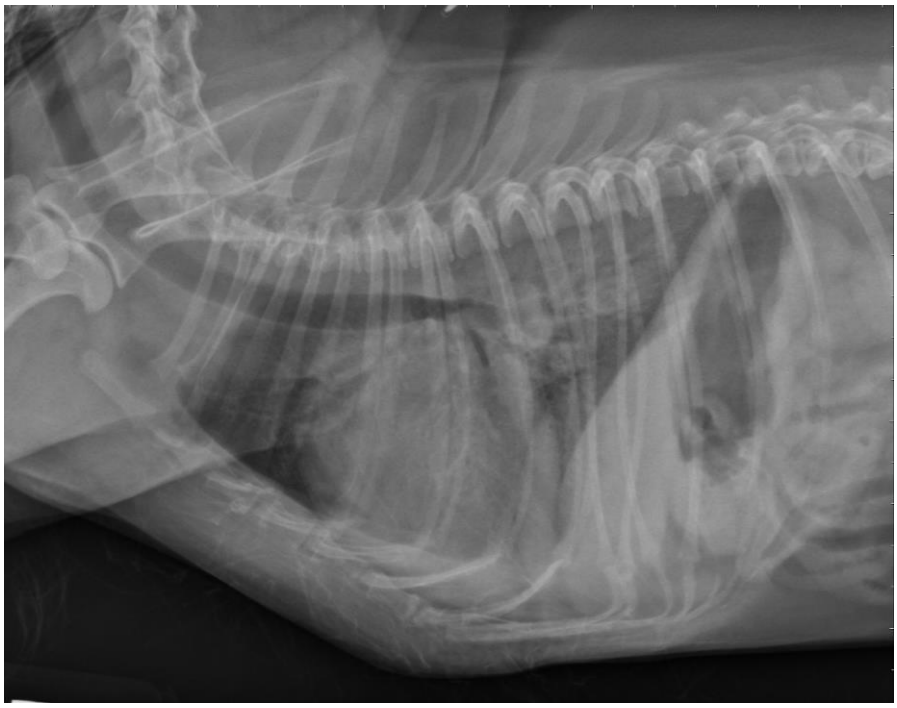


Figure 4: A Holter ECG monitor.