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REVIEW

Management of acute heart failure in cats☆

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Abstract Acute heart failure in cats represents a complex clinical situation in feline practice and this review has been designed to focus on the description of acute heart failure in cats, the diagnostic approach and clinical management of acutely decompensated feline cardiac patients. The authors acknowledge the lack of scientific evidence regarding many treatments used for heart disease in cats, and hence their approach may differ from recommendations given by other cardiologists. Every individual cardiac cat is also different, and it is important that all treatments are carefully tailored to the individual. Therefore this review provides generic advice based on the authors' personal experience but should not provide prescriptive guidelines on when to use particular drugs and doses and readers are encouraged to seek the latest information when managing these challenging cases.

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Abbreviations

AHF	acute heart failure
AO	aorta
ATE	arterial thromboembolism
CHF	congestive heart failure
HF	heart failure
LA	left atrium
LV	left ventricle
RA	right atrium
VHS	vertebral heart score

Introduction

Heart failure (HF) is defined as a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.[1] The clinical manifestations of HF may result from a variety of cardiac disorders, which may include diseases affecting the myocardium (i.e., cardiomyopathies), endocardium (i.e., valvular diseases), or great vessels (i.e., systemic and pulmonary hypertension, embolisms). The majority of cats with HF present clinical signs relating to an impairment of left ventricular (LV) myocardial function. LV functional abnormalities in cats are often observed in patients with hypertrophic ventricular wall and reduced ventricular lumen; however, significant myocardial dysfunction can also be observed in cats with normal myocardial thickness and normal LV lumen and in cats with severe LV dilatation and markedly reduced ejection fraction.[2] Although diastolic dysfunction is the predominant pathophysiological mechanism in cats with cardiomyopathy, systolic and diastolic dysfunction can coexist, so a stringent differentiation between systolic and diastolic failure may be less relevant for the clinical management of these patients.[3]

As observed in humans and other animal species, the cardinal manifestations of HF in cats are dyspnoea and fatigue, which often limit exercise tolerance and interaction. However, exercise intolerance is frequently unnoticed in domestic cats because of their common sedentary life style. Consequently, signs of fluid retention (congestive failure) are often the only abnormalities observed by the owners, such as tachypnoea/dyspnoea secondary to pulmonary oedema and/or pleural effusion and abdominal enlargement caused by ascites. Therefore, the terms HF and congestive heart failure (CHF) are almost interchangeable in feline cardiac patients.[3]

Indeed, acute heart failure (AHF) is most commonly observed in cats with cardiomyopathy (CM), but it has also been reported in a variety of other pathologies, such as degenerative valve disease,[4] endocarditis,[5,6] congenital abnormalities,[7–12] tachycardiomyopathy,[13] myocardial infarction,[2,14] hyperthyroidism[15] and even iatrogenic volume overload following aggressive fluid replacement, steroid therapy[16] and hyperviscosity syndrome.[17] Overall, it would be more appropriate to consider CHF as a clinical syndrome characterised by specific signs (i.e., tachypnoea/dyspnoea) in the medical history and signs on physical examination, thoracic radiography and ultrasonography (pulmonary oedema, pleural effusion and ascites). Cardiac biomarkers are very useful to detect the presence of myocardial damage (i.e., high-sensitivity cardiac troponin-I) or myocardial stress (NT-proBNP) although they cannot provide definitive confirmation of CHF. Therefore, a single diagnostic test for acute CHF does not exist and its diagnosis remains largely a clinical judgement based on a careful history collection and thorough clinical examination.[1]

Acute heart failure vs. chronic heart failure

According to recent European Society of Cardiology guidelines for the diagnosis and treatment of HF in people, AHF can be defined as a rapid onset of signs of HF, and this often represents a life-threatening condition, which requires immediate hospitalisation and medical attention.[18] Such description can comfortably be imported as a definition of acute CHF in feline cardiology. Unlike dogs, in which the vast majority of presenting patients progressively develop CHF following deterioration of a previously recognised cardiac condition (e.g. diagnosis of chronic degenerative mitral valve disease following detection of an audible heart murmur), cats with CHF may have never been previously diagnosed with a cardiac disease or even been suspected to have a heart problem, and they can present more unexpectedly in acute or hyper-acute failure.

The inception of clinical signs is often triggered by a stressful event (e.g. car journey, hospitalisation) or by a simple clinical procedure (e.g. restraint, forced recumbency for radiographic examination or echocardiography, blood sample, intravenous catheter placement). The sudden onset of CHF in these cases could be attributable to a rapid release of catecholamines, which induces generalised vasoconstriction and increased cardiac output (increased

stroke volume and heart rate). The result of these combined effects is a ventricular pressure overload, increased atrial pressure and, eventually, pulmonary capillary hypertension, pulmonary oedema and/or pleural effusion. Hence, patients suspected of having, or known to have, an underlying cardiac disease should always be examined gently and cautiously and considerations of the risks of any procedure borne in mind.[2,19] Conversely, a cat that has been previously diagnosed with CHF and has been clinically managed for at least a few weeks is commonly said to have chronic CHF. Cardiologists often refer to 'stable' chronic CHF if the clinical signs of a cat previously diagnosed and managed with CHF have remained well-controlled and generally unchanged for several weeks or months. On the other hand, when a stable CHF cat deteriorates, the patient may be described as 'decompensated,' and this may happen again 'acutely,' because of the patient becoming refractory to therapy or because of the onset of a clinical complication. The correct use of the above terminology may provide consistency for medical standard of care.

Clinical presentation

According to a recent retrospective study, the median age of cats presented with an episode of acute heart CHF is 10.7 years (range 2.0—22.5 years).[8] A similar age at presentation was reported in a prospective study by Smith and Dukes-McEwan,[11] who indicated a median age of 9.0 years (range 0·75—18 years). In both the above studies, dyspnoea and/or tachypnoea were cardinal signs of acute CHF. Tachypnoea and dyspnoea in CHF may have different causes, the most obvious being the presence of pulmonary oedema or pleural effusion. Some cats with pleural effusions may display paradoxical breathing, which is observed as a discordant movement of chest and abdominal wall during respiration. Ascites may also affect the respiratory rate and pattern. However, tachypnoea may also result from metabolic acidosis secondary to hypo-perfusion of peripheral tissue[20] or from ascites, since the pressure on the diaphragm induced by the fluid accumulated in the peritoneal space may interfere with the respiratory function and cause substantial discomfort.[19] Finally pain can contribute to tachypnoea in cats presented with acute onset of CHF complicated by arterial thromboembolism (ATE).[21]

Approximately half of the cats presenting in acute CHF do not have auscultatory signs relatable to an underlying cardiac disease, such as an audible heart murmur, gallop sounds, or arrhythmias and this explains the challenging diagnosis of CHF in cats on physical examination.[8,11]

Chest percussion can identify a typical horizontal line of dullness consistent with pleural effusion, if present (Video 1).[19]

Rectal temperature has been reported to be lower than normal in cats presenting in acute CHF and their hypothermia appears to be inversely associated with survival, in analogy to what has been reported in cats with clinical presentation of ATE.[8,21]

Unlike dogs, significant tachycardia is rarely observed in cats presenting in acute CHF, even when they are affected by concomitant atrial fibrillation.[11,22] With some exceptions offered by sustained supraventricular or ventricular tachycardia, where resting heart rate can often be above 250 bpm, heart rate of cats in CHF tends to be normal or lower than normal.[11,23,24] Bradycardia in cats in CHF can be secondary to bradyarrhythmias or conduction abnormalities, such as atrial standstill and atrioventricular blocks. However, sinus bradycardia may also be present and its mechanism could potentially be attributed to concomitant hypothermia, current or pre-existing pharmacological treatment with beta-receptor antagonists (i.e., atenolol or propranolol), down-regulation of myocardial beta-receptors, altered arterial baroreflex sensitivity, or any other dysfunction affecting the cardiac autonomic nervous system.[25,26] A more profound bradycardia (average 145 bpm) has been reported in a published case series of 12 cats affected by acute onset of CHF following a relatively short course of systemic corticosteroid therapy. Although hypothermia may have had a contributing effect in reducing resting heart rate in those cats, it has been proposed that this is a feature of corticosteroid associated-CHF.[16] Based on these observations, lower than normal heart rate and mild hypothermia might represent useful physical findings in support of a diagnosis of feline CHF.

Pale mucous membranes, weak femoral pulses, weakness, jugular venous distension or pulsation and abdominal distension are other common physical findings in cats with acute CHF, although none of these signs can be considered pathognomonic.[4]

Diagnostic testing

Cats presented with acute signs of CHF are extremely vulnerable and every attempt should be made to reduce their stress and anxiety before attempting any diagnostic test or therapeutic

procedure. Effective sedation protocols are explained below. The patient should be maintained in a comfortable sternal recumbency, which facilitates respiratory movements. Dullness on chest percussion can raise suspicion of pleural effusion and the presence of fluid in the pleural space can be easily confirmed by trans-thoracic ultrasonography, performed with the patient in a comfortable sternal position (Video 1). If ultrasound facilities and expertise are available, a rapid bedside ultrasound examination can be performed before thoracic radiography (Video 2). This emergency technique is commonly addressed as 'point-of-care thoracic ultrasound examination' and it allows rapid identification of signs of relevant underlying cardiac disease, such as cardiac chamber enlargement, ascites, pleural and pericardial effusion.[27] This ultrasonographic exam is normally performed with the cat in sternal recumbency, while receiving oxygen supplementation typically after a low dose of sedation. Learning how to perform a point-of-care thoracic ultrasound examination is relatively intuitive and simple and, with minimal supervised training, general practitioners can achieve proficiency in identifying left atrial enlargement, pleural and pericardial effusions.[28,29] Increased left or right atrial size associated with respiratory signs is highly suggestive of CHF, especially if accompanied by signs of pleural effusion. Maximal left atrial size measured in B-mode from the right parasternal four-chamber long axis view is readily achievable by untrained first-opinion practitioners and a cut-off value of 16·5 mm has been reported to have a sensitivity and specificity of 87% for a diagnosis of HF.[11] If the left atrial (LA) size is indexed to the aortic diameter (Ao) in a right parasternal short-axis view at the level of the heart base (Fig. 1), a left atrium/aorta ratio greater than 1.5 suggests LA enlargement and values between 1.51 and 1.79, 1.79 and 1.99 and $\geq$2.0 are defined as mild, moderate and severe LA dilation respectively.[4,30] LA enlargement in cats with CHF is usually moderate to severe.[4] Conversely, if LA size is normal, noncardiac causes of dyspnoea should be sought in that patient. The emergent focused ultrasound examination has become an extension of the physical examination and has been termed by some as the 'visual stethoscope'.[27] Nevertheless, it is mandatory to consider some important exceptions, which can be related to the intrinsic capacity of the feline heart chambers to rapidly adapt to variations in

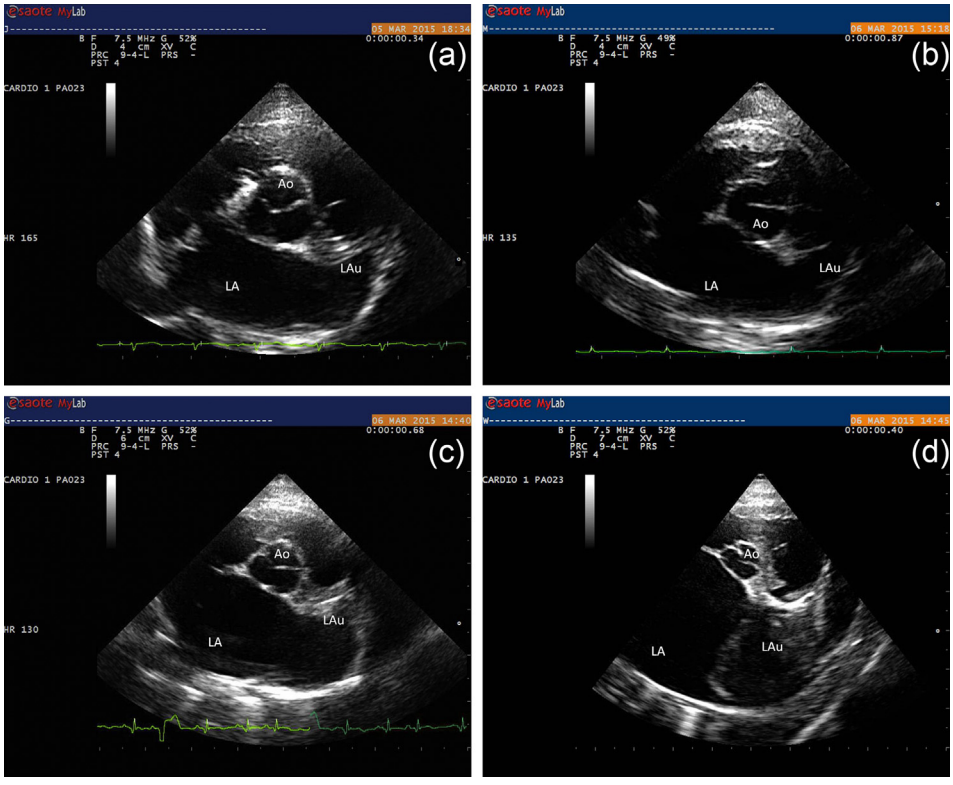

Fig. 1 Right parasternal short-axis view of the heart base of cats showing different left atrial (LA) dimensions. (a) Normal LA size (La/Ao ratio = 1.3); (b) mild LA enlargement (LA/Ao = 1.7); (c) moderate LA enlargement (LA/Ao = 2.0); (d) severe LA enlargement (La/Ao = 4.4). An echo-dense structure is also visible in the left auricular (LAu) cavity, suggesting an intracavitary thrombus.

circulating plasma volume.[31,32] For example, intravenous fluid therapy in the preceding 48 h can potentially cause significant LA enlargement even in the absence of an underlying cardiac condition. Similarly, LA size may appear normal or near-normal despite the presence of CHF following aggressive diuresis, hypovolaemia/dehydration, long-acting glucocorticoid injection in the preceding week or any acute exacerbating event such as a rapid formation of an intracavitary thrombus.[4,32]

The right atrial (RA) size is assessed subjectively on focused assessment with sonography for trauma echocardiographic examination, using the right parasternal long axis four-chamber view. In normal cats, the RA appears smaller than the LA and therefore RA enlargement simply refers to RA which subjectively becomes larger than the LA (Fig. 2).

Another promising user-friendly ultrasound technique that can potentially be applied to cats with suspected acute CHF is lung ultrasonography, in which identification of a linear ultrasound artefact named 'lung comets or rockets' seems reliably associated with pulmonary oedema.[33,34] However, to the best of the authors' knowledge, this technique has not yet been validated in cats.[c]

More advanced echocardiographic techniques are also available to determine LA function, which is significantly reduced in cats with CHF.[35,36] However, despite their foreseeable attractiveness, such techniques are less relevant to practical emergency considerations.

In life-threatening cases of dyspnoea and where ultrasound facility is not readily available, parenteral administration of furosemide could be justified before radiographic confirmation of pulmonary oedema, should the full clinical presentation suggest acute onset of CHF.

Thoracic radiography is still considered the 'gold standard' test for confirming the presence of cardiogenic pulmonary oedema in cats, and this test can also show signs compatible with right-sided HF. Using a simplistic approach, the feline heart size is comparable to a small egg with the apex on the sternum and the long axis leaning forward with an angle of about 45 degrees, although there is a high variability between subjects. The vertebral heart score (VHS) of normal cats has values below 8 (7.5 ± 0.3) while, with the exception of pericardial effusion, a VHS between 8.0 and 9.3 is more consistent with cardiomegaly. Moreover, a VHS greater than 9.3 in dyspnoeic cats is highly suggestive of HF.[37,38] Clinicians should also be aware of pseudocardiomegaly that can be caused by pericardial fat accumulation in the pericardium, although a good radiographic exposure can often distinguish the different densities of the two structures (heart and fat). Pericardial-peritoneal diaphragmatic hernia is another cause of pseudocardiomegaly to consider in the differential diagnosis. Examination of the falciform ligament and subcutaneous fat can help the clinician to differentiate real cardiomegaly from pseudocardiomegaly caused by accumulation of fat.[4]

The radiographic diagnosis of CHF in cats can be challenging, but is mainly based on the presence of clinical signs supported by a triad of radiographic changes: cardiomegaly (interpreted as increased VHS or LA enlargement or both), pulmonary venous engorgement and any of the following signs of congestion, such as pulmonary oedema, pleural effusion and ascites. Pulmonary venous congestion is usually seen in the early stages of CHF and it is characterised by dilated veins that appear more pronounced and prominent than normal. With the progression of CHF this finding is not always easily appreciated, especially when the cat has already received treatment with diuretics or when signs are masked by concomitant presence of pleural effusion. The presence of LA enlargement can be appreciated as a rounded opacity caudal to the tracheal bifurcation in lateral view or, even better, as a 'valentine-shaped' heart on the dorso-ventral or ventro-dorsal projections. However, LA enlargement in cats is sometimes difficult to appreciate on thoracic radiography even in patients with CHF and therefore a normal LA size on thoracic radiographs does not rule out CHF in dyspnoeic cats.[39]

Radiographically, cardiogenic pulmonary oedema appears initially as an interstitial pattern and due to the peri-vacular nature of the initial effusion, blood vessels appear with less distinct borders. With the progression of congestion, the fluid tends to invade the alveolar lumen, creating an image of alveolar pattern with poorly defined borders ('cotton fluff-like') and this is usually the pattern observed with an acute clinical presentation. The anatomical distribution of alveolar pulmonary oedema in cats is rather peculiar and it may reflect different stages in the development of the lesion at the time of the radiographic study. According to one study, cardiogenic pulmonary oedema in cats can present as non-uniformly diffuse (61% of cases), uniformly diffuse (17%), multifocal (17%) and focal (4%)

[c] Evaluation of Point-of-care Lung Ultrasound (VetBLUE Protocol) for the Diagnosis of Cardiogenic Pulmonary Oedema in Dogs and Cats with Acute Dyspnoea. Late Breaking Research Abstract. ACVIM Forum 2015.

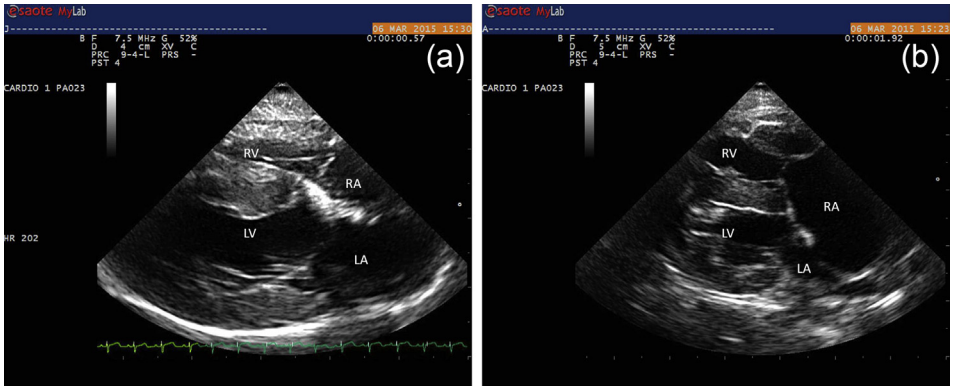

Fig. 2 Right parasternal four-chamber long axis view of a normal cat (a) and a cat affected by severe right sided heart enlargement (b). In cat b, the right ventricle (RV) and right atrium (RA) appear severely dilated when compared to the corresponding left ventricle (LV) and left atrium (LA).

(Fig. 3).[40] The radiographic heterogeneity of cardiogenic pulmonary oedema in cats contributes to make a diagnosis of CHF more challenging than in dogs. Although not specific to CHF, pleural effusion is often radiographically observed in cats with CHF and it appears as focal areas of radiopacity in the chest cavity, which tend to separate the lung lobes creating distinctive grooves (fissures lines) and outlining the lung lobes (scalloping). Ideally, significant pleural effusions should be diagnosed and managed via thoracocentesis before a radiographic examination. Finally, hepatomegaly and ascites can also be detected in cats with CHF, and they may represent a sign of right-sided CHF, especially when jugular vein distension or pulsation is observed on physical examination. Hepatomegaly appears as a liver shadow protruding beyond the

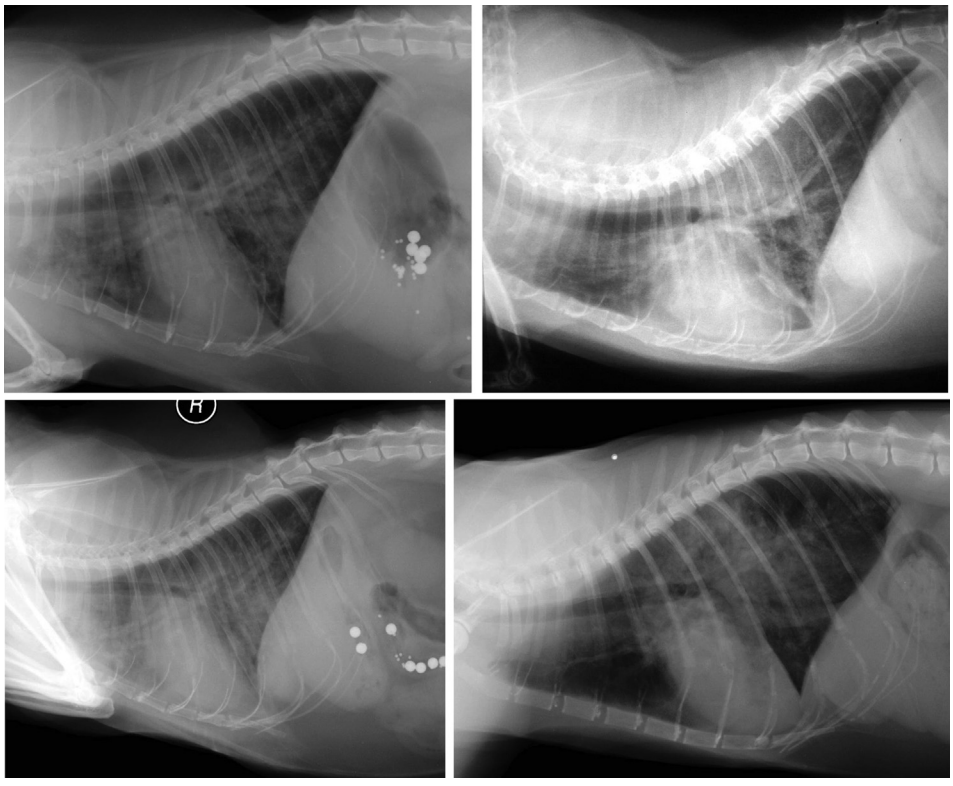

Fig. 3 Examples of highly variable radiographic appearance of cardiogenic pulmonary oedema in four cats presenting with clinical signs of acute congestive heart failure. These variations may reflect different stages in the development of the lesion at the time of the radiographic study.

last costal arch, while ascites is detected as diffuse radio-density with loss of radiographic details of the cranial viscera on abdominal radiographs, and a pendulous abdomen on physical examination.

Determination of plasma concentration of the amine terminal pro B-type natriuretic peptide (NTproBNP) is another valuable tool to assist the clinician in the diagnostic stratification of cats presented with acute dyspnoea.[41,42] Measurement of NT-proBNP in pleural fluid may also be useful to distinguish cardiac from noncardiac causes of pleural effusion in cats.[43] For many years, this test has been neglected in veterinary emergency and critical care because of the unavailability of point-of-care testing. However, the recent worldwide release of the SNAP® Feline proBNP Test (IDEXX Laboratories, Inc, Westbrook, Maine, United States) has provided a very rapid (10 minutes) screening test. According to this test, a dyspnoeic cat with a normal result has an NTproBNP concentration less than 100 pmol/L, and therefore the cause of dyspnoea is most likely noncardiac in origin. Conversely, a positive result ($\geq$270 pmol/L) could be compatible with HF but it could be elevated in noncardiac causes of dyspnoea.

Finally, plasma cardiac troponin-I (cTnI) level, a sensitive and specific marker of myocardial injury, can also be considered in the emergency setting for differentiation of cardiac from noncardiac causes of dyspnoea in cats.[44] In an ideal situation, plasma troponin should be measured via a rapid validated point-of-care assay, such as the Biosite Triage Meter [45] or the i-Stat 1 analyser,[46] and cTnI concentrations less than 0.24 ng/mL can be used to rule out CHF as the cause of dyspnoea, whereas concentrations above 0.66 ng/mL are more suggestive of a cardiac cause.[45,46] Plasma cTnI and troponin T (cTnT) have been recently indicated as predictors of cardiac death in cats with hypertrophic cardiomyopathy when measured by high-sensitivity (HS) assays and this can add useful information in clinical decision making, although HS-cTnI and HS-cTnT tests need to be performed via external laboratories.[47,48]

Clinical management

While the therapeutic recommendations outlined likely reflect current state-of-the-art clinical medicine, it is important to emphasize that no prospective clinical trials have been published to date in cats with HF. As such, recommendations made herein are based on the authors' anecdotal experiences, retrospective or experiment studies in cats and borrowed evidence from canine and human clinical trials, guidelines and consensus statements.[1,18,49]

Minimizing stress is of the utmost importance in a cat in AHF. Because cats with respiratory distress can decompensate quickly with excessive handling, often times empiric therapy may be necessary prior to making a definitive diagnosis of HF. It is prudent to allow cats in respiratory distress to have sufficient time to rest in between diagnostic tests or treatments, and this approach can often be lifesaving. The immediate goals of AHF therapy are to alleviate dyspnoea and reduce abnormal fluid accumulations while supporting or improving cardiac output. Generally, for a presumptive diagnosis of severely and acutely decompensated HF, initial empiric therapy includes cautious sedation, IV or IM furosemide, oxygen therapy and thoracocentesis (if needed).

Sedation

Despite the minimal risk of depressing the respiratory drive, sedation is generally associated with reduced metabolic demand, reduced anxiety and neuro-humoural response to stress, which in turn results in improvement of the respiratory muscle work, heart rate and blood pressure.[50,51] In the authors' opinion, butorphanol is an effective sedative for patients in respiratory distress, at dose ranges from 0.05 to 0.30 mg/kg IV, IM or SQ but is generally dosed at 0.1 mg/kg. Buprenorphine (0.005—0.02 mg/kg IV, IM or SQ) is another safe and effective sedative that has a longer duration of action and better analgesic effects than butorphanol.[52—54] For these reasons, buprenorphine represents a better option in cats presenting with concurrent signs of ATE.

Local anaesthesia (e.g. lidocaine 2%) or additional sedation may be needed to safely perform thoracocentesis. This can be achieved using an additional small dose of butorphanol combined with a low-dose acepromazine or midazolam. If buprenorphine was initially administered, simply adding in a low-dose acepromazine or midazolam might be sufficient. Fractious cats may require additional sedation with low-dose ketamine (3—5 mg/cat IV or IM), which might be sufficient when combined with an opioid and either acepromazine or midazolam to safely perform the clinical procedure. Low-dose fentanyl or remifentanil would also be reasonable options for sedation in a cat with HF.

Oxygen supplementation

Supplemental oxygen therapy is recommended to reduce the breathing effort. Interestingly though, in human medicine, oxygen supplementation

alone, without end expiratory pressure with tight-fitted face mask, does not seem associated with significantly improved outcomes.[55–57] That being said, oxygen may transiently help the cat during times of handling or could provide a quiet environment while waiting for the medications to work. Supplemental oxygen would be of most benefit for a cat that becomes hypoventilatory because of respiratory muscle fatigue.[58] With noninvasive modalities, the aim is to achieve an increase in inspired oxygen of 40–50%. Oxygen can be delivered by 'flow-by', face mask, nasal prongs or oxygen cage. Although 'flow-by' and face mask methods are reasonable options, these methods are not always accepted by cats in distress and they are not very efficient and high flow rates (2–5 L/min) are recommended to effectively increase inspired oxygen concentration. Nasal prongs may also be tolerated by some cooperative cats. Based on studies in dogs with nasal cannulas placed for inspired oxygen, flow rates of 50–100 mL/kg/min are recommended with this technique.[59] Placing the cat in a quiet and oxygen rich environment, such as an industry manufactured oxygen cage, is ultimately the most efficient solution. While there are other makeshift methods to deliver oxygen (e-collar, 'baggie' method, cage front, or infant incubator), these should be used with caution because of issues with unpredictable oxygen enrichment, the potential of overheating and inadequate CO_2 removal with subsequent accumulation which could all be dangerous to the cat. Finally, any long term oxygen supplementation requires adequate humidification and warming.

Diuresis

Regardless of the underlying aetiology, furosemide plays a pivotal role in the pharmacologic treatment for AHF, although it does not directly improve cardiac output and can potentially lead to diminished renal perfusion, electrolyte abnormalities and further activation of the RAAS system.[60] Furosemide decreases preload by blocking sodium, potassium, chloride and secondarily water reabsorption in the ascending limb of the loop of Henle in the nephron. The increased urine output leads to a decreased circulating plasma volume that causes a decrease in hydrostatic pressure at the level of pulmonary capillaries. The net filtration of oedema decreases allowing the lymphatics to remove the pulmonary oedema from the interstitial and alveolar spaces in the lungs into the intravascular space resulting in improved breathing effort and patient's comfort. Furosemide is most helpful in cats with pulmonary oedema, while in cats with large volume pleural effusion and respiratory difficulties, furosemide alone will not result in a significant clinical benefit without concomitant therapeutic thoracocentesis.

The dose of furosemide needs to be tailored to the individual patient, because excessive doses can lead to deleterious effects on renal perfusion (azotaemia) and electrolytes depletion (hypokalaemia, hyponatraemia, hypochloraemia, hypomagnesaemia, and hypocalcaemia) especially in an older cat.[60,61] Conversely, an insufficient dose of diuretic can lead to therapeutic failure, prolonged hospitalisation, and potential euthanasia because of refractory or recurrent HF. In a severely decompensated HF cat, furosemide should be administered intravenously to provide the most rapid onset of action and more predictable bioavailability.[62,63] However, because IV administration may often be challenging and stressful in critical patients, intramascular administration represents a valid alternative. The initial recommended bolus dose is approximately 2–4 mg/kg for a severely decompensated cat and 1–2 mg/kg for mildly decompensated patients. If given IV, a clinical improvement should be expected peak effect in approximately 30 minutes after administration, while the result might be expected in 1–2 hours after administration if given IM. An empirical approach is to consider a cumulative maximal dose of 12 mg/kg/day. Cats with preexisting HF that decompensate with recurrent pulmonary oedema should receive higher doses of furosemide than their chronic oral dose.[1] Depending on the severity of the underlying pulmonary oedema, repeated boluses or initiation of continuous infusion of furosemide may be indicated. Monitoring the respiratory rate and effort on an half hour to hourly basis will help guide frequency and dose of additional furosemide acutely. If the furosemide is effective, one should notice a gradual decrease in the respiratory rate and effort over the next few hours. Based on the response to the first or second dose of furosemide, one may estimate the subsequent doses and frequency of furosemide administration.

In human medicine, there continues to be some debate regarding repeated bolus vs. continuous rate infusions (CRIs) of furosemide.[64–67] Some studies have demonstrated that, at similar doses, continuous infusion of furosemide improves diuresis while diminishing fluctuations in intravascular volume accompanied by a constant urine production over time. Constant infusions may also limit the risk of renal ischaemic injury caused by abrupt changes in vascular volume. Additionally a study in dogs showed diminished kaliuresis and improved diuresis with furosemide administered

via CRI compared to bolus dosing.[63] In the best of the authors' knowledge, results of similar studies in cats are not available. However, it should also be noted that while CRI dosing may have some benefits over standard administration of boluses, a continuous infusion requires the use of syringe pumps which are expensive and not always available. Finally, despite improved diuresis with CRI in smaller studies, large clinical trials in humans have failed to show significant clinical benefit over repeated bolus dosing.[64] The authors' preference is a combination of initial bolus dosing and short term CRI of 2—8 hrs tailored to the individual cat and clinical scenario.

Centesis

Pleural effusion, pericardial effusion and ascites are common presentations in cats with HF.[19,29] The decision to perform a centesis should take into consideration careful calculation of the risk/benefit ratio of the procedure. For example, mild ascites and small volume of pleural or pericardial effusion, often associated with concurrent pulmonary oedema, may successfully respond to diuresis alone, without the need of any mechanical drainage.

Thoracocentesis

It is the most effective therapeutic manoeuvre to relieve respiratory distress caused by a large volume of pleural effusion. The procedure is relatively safe and iatrogenic complications, such as pneumothorax, are rarely observed. Needle puncture of a large vessel or even the heart is also possible but much less likely with careful technique. In a cat with chronic chylous effusion, pneumothorax could also be caused by a tear in the fibrotic visceral pleura with rapid re-expansion of the lungs.[68] The needle insertion can be guided by physical exam, thoracic radiographs or optimally point-of-care thoracic ultrasound. Most cats can be tapped just on one side because of the presence of sufficient mediastinal fenestration and bilateral procedure is rarely necessary. In general, the needle is inserted into the chest at the level of the 7th—8th intercostal space, lower and middle third, entering the pleural space in front of the rib to avoid vessels and nerves running along the caudal aspect of the rib. Once the site of the centesis is identified, the fur is clipped and the skin is prepared aseptically. The procedure is typically performed with the cat in sternal recumbency with adequate manual restraint while providing supplemental oxygen if needed.

The procedure can be performed by using a butterfly-needle catheter or an over-the-needle IV catheter attached to extension tubing, a three-way stopcock or one-way drainage valve and a syringe. With either technique, at least 1 mL of constant negative pressure with the syringe should be applied in order to visualize the pleural fluid in the catheter and tubing system once the pleural space is entered. Each method has its advantages and disadvantages. The butterfly needle catheter is quick and efficient and works well for most cats because of their relatively thin chest walls. Most 23—21 G butterfly catheters are only ½—3/4 inch in length which may not be long enough to safely enter and position the needle in an obese cat. The main disadvantage is the persistence of a sharp needle in the chest during a therapeutic thoracocentesis which may increase the risk of pneumothorax if proper technique is not used. Some important finesse points with the butterfly catheter are to advance the needle with the bevel facing cranially (mouth of bevel facing mouth of cat). Then once the pleural space is penetrated, the needle should be wrapped caudally around the rib as advancing it deeper so that the bevel is facing the pleural space. Once inside the chest cavity with the needle, one should hold the needle as flat as possible against the pleural space until all fluid is drained (Fig. 4). When removing the needle, this should be retracted as flat as possible. The alternative technique with the over-the-needle catheter offers the advantage of potentially more complete and safer removal of pleural fluid once the catheter is within the chest because the sharp needle stylet is removed after penetration. Typical over-the-needle catheter sizes and lengths used in the cat range from 18 G 1.75 in to 16 G 3.25 in. A local anaesthetic block with a small dose of

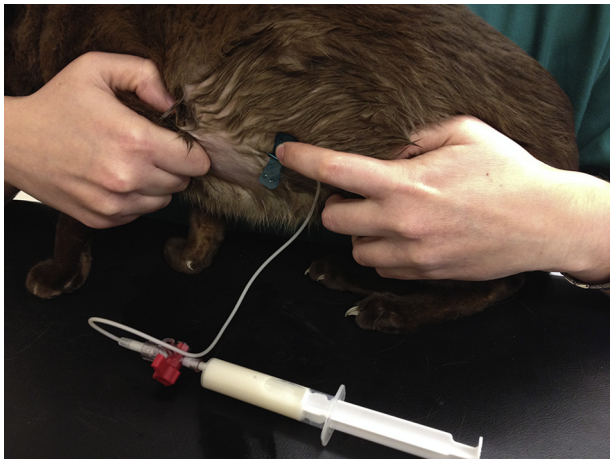

Fig. 4 Thoracocentesis in a cat using the butterfly needle technique. The image shows the needle position once inserted inside the chest cavity, holding on to the wings of the butterfly to keep the needle as flat as possible against the pleural space, until all fluid is drained.

lidocaine or lidocaine topical jelly will help to decrease the patient discomfort associated with the needle insertion. The disadvantages of the over-the-needle catheter system relate to the longer preparation time especially if fenestrations are made, the need for a releasing incision and local block in the skin and difficulties in threading the catheter into the chest. If suboptimal technique is used for making the fenestrations, for advancing or removing the catheter, there is also a risk for fragmentation of the catheter resulting in a small piece of the catheter remaining in the chest cavity. The main finesse point with the over-the-needle catheter system is that once the needle stylet penetrates the pleural space, one should keep the needle absolutely still as one advances the soft catheter over the needle into the pleural space. The most common mistake is pulling the needle stylet back as one advances the catheter resulting in kinking or loss of positioning in the chest cavity. Both techniques for thoracocentesis work well in experienced hands and the decision is ultimately a pure clinician's preference. After thoracocentesis, respiratory rate and effort should be monitored carefully and a significant improvement should be expected within a few minutes in case of successful procedure without complications (Video 3). Fluid analysis is recommended to ensure that the nature of the fluid is compatible with HF. Radiographs after therapeutic thoracocentesis are prudent to better evaluate the heart size, lungs and pulmonary vasculature.

Pericardiocentesis

It is rarely performed in the cat with HF and typically only at a speciality referral centre after an echocardiogram has confirmed the presence of significant pericardial effusion. Usually the pericardial effusion present in HF is small volume and will resolve with medical therapy alone. Rarely though, the risk/benefit ratio may be in favour of pericardiocentesis if the pericardial effusion is disproportionately large volume and echocardiographic criteria for cardiac tamponade, such as diastolic collapse of the right atrium, are present.

When performed properly, the complication rate of pericardiocentesis is low. However, the risk of myocardial damage and death should be discussed as a rare but potential complication of the procedure. Death may result from a lethal arrhythmia from cardiac contact from the catheter or needle or coronary artery laceration. Human studies show that ultrasound guided pericardiocentesis is associated with a reduced complication rate.[69] Other complications include less serious arrhythmias, pneumothorax or intracardiac puncture. Because cats need to be absolutely still during the procedure, heavy sedation or even light anaesthesia is recommended for pericardiocentesis. The optimal site for pericardiocentesis is identified by echocardiography and is typically on the right side at the 4th—5th intercostal spaces just at or above the costochondral junction. A continuous electrocardiogram (ECG) is used to detect catheter induced arrhythmias. Typically, an over-the-needle catheter system (with no fenestrations), similar to thoracocentesis, is used. The patient is placed in sternal or lateral recumbency depending on the patient's demeanour and clinician's preference.

Abdominocentesis

This technique should be considered when an excessive fluid accumulation in the peritoneal cavity interferes with the mechanics of respiration and causes excessive patient's discomfort. In analogy with the previously described thoracocentesis techniques, a successful abdominocentesis can be performed by using a butterfly needle or an over-the-needle catheter. The point of needle insertion depends on clinician's preference, although ultrasound-guided procedure will allow the identification of the largest pocket of fluid in order to maximise the drainage and avoid puncture of the spleen or other abdominal organs and major vessels.

Bronchodilation

In addition to furosemide, some cats with refractory pulmonary oedema and respiratory distress may benefit from inhaled salbutamol (INN)/albuterol (USAN). Indeed, some cats with HF may develop peribronchiolar pulmonary oedema potentially associated with bronchoconstriction, although this sequel has not been clearly demonstrated. Generally, one or two 'puffs' (100 micorgrams per puff) are administered with a dedicated mask and spacer chamber (i.e. Aerokat[d]). Additional administration could be repeated after 15—30 minutes to ensure delivery of the drug to the smaller airways. Inhaled salbutamol/albuterol may also represent a low risk empiric treatment option in cats in which a confirmative diagnosis of HF or feline asthma has not been achieved. It is important to emphasise that bronchodilators should be used cautiously, as they may promote tachyarrhythmias, especially if administered at high doses.

Positive inotropes

In humans with acute pulmonary oedema, the short-term use of positive inotropic agents may aid in the resolution of HF regardless of the aetiology

[d] Aerokat©, Trudell Medical International, Ontario, Canada.

because of the ability of these agents to improve myocardial function.[70]

Pimobendan
Although pimobendan is not currently licenced for use in cats, it has been used with increased frequency in the management of feline HF. Pimobendan has a dual mechanism of action and is often termed an 'inodilator.' Specifically, the drug has calcium-sensitising property that improves contractility (positive inotrope) with minimal effects on myocardial oxygen consumption. The other mechanism of action is phosphodiesterase inhibition, primarily leading to a balanced vasodilation (arterial and venous) and possibly improved relaxation.[71,72] Pimobendan is primarily available for dogs as oral formulation. Most recently injectable pimobendan (Vetmedin 0.75 mg/mL solution) has become available in the United Kingdom. Compared to dogs, pimobendan in cats has a substantially longer elimination half-life and maximal drug plasma concentration and a recommended therapeutic dose is not available.[77] Nevertheless, many clinicians decide to administer pimobendan in cats with AHF, especially in the presence of LV systolic dysfunction, significant pleural effusion, renal insufficiency, or severe refractory pulmonary oedema. While there are not published prospective randomised clinical trials evaluating pimobendan in cats with HF, there is a growing body of retrospective case series suggesting its safety and potential clinical benefit as compared to conventional therapy.[73–76] Dose and frequency escalation are not uncommon in dogs with recurrent or refractory HF with good clinical response and a similar phenomenon might be expected in cats.[78]

Dobutamine
If a cat with severely decompensated HF is unable to take oral medications and has signs of low cardiac output, intravenous administration of dobutamine should be considered, especially for the first-time HF patient. Dobutamine is an adrenergic positive inotrope with primarily beta-1 effects. The dose ranges from 1 to 10 mcg/kg/min CRI, starting at a lower dose and titrating upward based on blood pressure and ECG monitoring, because hypertension and tachyarrhythmias represent the main adverse effects.

Vasodilators
Deceasing afterload with vasodilatory drugs enhances forward stroke volume and cardiac output in failing hearts because the afterload is often elevated in HF. Vasodilators have been found to be very effective in the treatment of systolic dysfunction in dogs and humans.[1,18,79] However, there is no clear consensus on vasodilatory therapy in cats with acute and chronic congestive HF.

Nitroglycerine
It is available in different formulations but the 2% ointment is the most commonly used in small animals, generally applied topically (1/4–1/2 in) in conjunction with diuretics in the acute management of severe HF to further reduce preload. Reapplication can be used in as frequently as q 8 hours for up to 24–48 h. Nitroglycerin dilates the splanchnic vasculature and redistributes the blood into the abdomen away from the heart and lungs.[80,81] Although there is little risk for harmful effects of nitroglycerin, its therapeutic benefit in cats has never been established.

Sodium nitroprusside
It is a potent venous and arterial vasodilator, which dilates both the systemic and pulmonary vasculature. Nitroprusside has the potential to cause marked hypotension, thus starting at a low dose (0.5–2 mcg/kg/min), and titrating upward based on blood pressure, targeting a mean blood pressure of 70 mmHg or systolic blood pressure 90–100 mmHg.[1,18,49] Owing to its potent vasodilatory effects, continuous arterial blood pressure monitoring is recommended. Generally infusions are given for <24 hrs. Longer infusions of the drug have been rarely associated with thiocyanate toxicity, particularly in the setting of renal insufficiency. Nitroprusside must be protected from light during infusion and further diluted in 5% dextrose in water to a concentration that is suitable for the dosage required and size of the patient (typically ~100–300 mcg/mL for a cat).

ACE inhibitors

ACE inhibitors (enalapril, benazepril, ramipril, imidapril) are currently licenced for the management of HF in dogs but not in cats. Furthermore, in AHF, they could potentially reduce intrarenal perfusion and glomerular filtration rate in and therefore they are rarely indicated in this phase.[49,82] Although there is lacking scientific evidence of beneficial effects of ACE-I in cats with HF, many clinicians would still advocate the use of ACE inhibition in the chronic management of all HF patients once the patient is stable and eating. [1,18,49]

Antiarrhythmic drugs
With atrial and ventricular arrhythmias contributing to the morbidity and mortality of HF, various classes

of antiarrhythmic agents have been repeatedly studied in large randomized clinical trials in humans. Instead of conferring survival benefit, however, nearly all antiarrhythmic agents increase mortality in the HF population.[1,18,83] Often times, control of the pulmonary oedema and improving cardiac output will improve the arrhythmia indirectly. Low grade arrhythmias are generally not treated. Nevertheless, if haemodynamically significant supraventricular or ventricular tachyarrhythmias are present, then specific antiarrhythmic treatment is recommended. For rapid atrial fibrillation (e.g. ventricular response rate higher than 250 bpm), diltiazem might be considered for rate control. Oral diltiazem is given either as a nonsustained-release formulation (10 mg/cat PO q8h), or as a sustained-release oral formulation (e.g. Dilacor® 30 mg/cat/day). Diltiazem is also available in some countries as an injectable formulation for urgent control of a supraventricular arrhythmia in a cat that cannot take oral medications (0.05—0.1 mg/kg slow IV, repeated up to 0.25 mg/kg). If rapid and sustained ventricular tachycardia, lidocaine slow IV 0.2—0.5 mg/kg (repeat once or twice) or sotalol PO 2 mg/kg q12h is recommended. Risks of diltiazem and sotalol are related to their potential to decrease cardiac output because of their negative inotropic effects or potentially their effects to lower heart rate. Serious side effects of lidocaine in the cat result from effects on the central nervous system and the cardiovascular system. Clinically, these can be evident as lethargy, unconsciousness, coma, convulsions, seizures, hypotension, bradycardia, and cardiovascular collapse and can result in death.[84]

Cats with pre-existing subclinical hypertrophic cardiomyopathy and already on beta blocker therapy are often presented with decompensated HF and clinicians should evaluate very carefully the risk/benefit ratio of continuing the beta blocker therapy. A slow progressive discontinuation of atenolol over a period of several weeks, rather than a sudden withdrawal, is generally preferred. Similarly, starting beta-blockade in a cat with acute CHF should be discouraged.

Anti-thrombotic drugs

Cats with HF often have enlarged left atria placing them at an increased risk for ATE, which represents the most devastating complication of feline heart disease. Cats with concurrent ATE and HF have a lower survival rate (~77 days) than cats without HF (~233 days).[21,85] Despite the lack of scientific evidence, many cats with HF often receive aspirin or clopidogrel as a prophylactic anti-platelet aggregation therapy even before the onset of ATE and such intervention is often prompted by the detection of intracavitary spontaneous echocardiographic contrast ('smoke') during echocardiographic evaluation of the cat in AHF.

Mechanical ventilation

In selected severe cases of AHF, intubation and mechanical ventilation may be a lifesaving intervention, especially in those cases with respiratory muscle fatigue refractory to medical therapy. Mechanical ventilation supports the pulmonary system to maintain an adequate level of alveolar ventilation, restore normal acid-base balance and oxygenation to the organs and tissues while giving the patient time to respond to medical therapy. Respiratory muscle fatigue may be diagnosed by a decrease in respiratory rate, associated with hypercapnea and declining level of consciousness. Another consideration for ventilation is to allow further diagnostic workup in a declining patient with suspect HF and lack of improvement with conventional HF medication. Mechanical ventilation in these patients will support of the respiratory system and increase the safety of further diagnostic testing such as an endotracheal wash with cytology and culture and thoracic imaging.

The decision to offer mechanical ventilation needs to consider both the prognosis for a meaningful recovery of the patient, hospital's equipment and expertise, as well as client's finances and expectations. In a recent small case series of small animals undergoing positive pressure ventilation for HF, the overall survival-to-discharge rate in cats was 66%, which is considerably higher than previously reported.[86] According to this report, all four ventilated cats survived to hospital discharge. However, ventilator therapy is expensive as it requires extensive nursing care, ventilator equipment and expertise. Ventilator therapy has complications and even a few hours of mechanical ventilation can put a significant strain on available personnel.

Monitoring and discharge

The most helpful monitoring parameters in the acute management of HF in cats are respiratory rate, respiratory effort and level of patient's consciousness. Generally a rapid improvement is expected after thoracocentesis but continued monitoring is important for the following few hours to ensure that iatrogenic pneumothorax has not developed. A cat with severe cardiogenic pulmonary oedema would be expected to improve over the course of a few hours to a day following diuretic therapy. If no improvement is noted in respiratory rate and effort after the initial dose of

injectable furosemide within a couple of hours, then a repeated dose of diuretic is recommended. Adjunctive therapies such as pimobendan, nitroglycerin or salbutamol/albuterol inhalation may also be considered. Intensification of adjunctive therapies would be recommended if respiratory distress persisted or worsened.

For the in-hospital patient, in addition to respiratory rate monitoring, hourly monitoring of heart rate and rhythm is also recommended via continuous ECG recording, if feasible. Blood pressure monitoring every 4—6 h is also recommended, especially when considering adjunctive vasodilator therapy. Measurement of renal and electrolyte parameters should ideally be performed before starting treatment and repeated a few days later. For the cat being managed as an in-patient, repeat imaging in 12—36 h is also recommended as it can be a helpful to finely titrate the dose of diuretics. For a cat that primarily manifested with pleural effusion, a brief fluid check with a point-of-care ultrasound exam may be helpful to reassess recurrence of effusions after a thoracocentesis. For a cat with pulmonary oedema, a repeat thoracic radiograph typically just prior to discharge will be helpful to assess the dose of furosemide at discharge. Chronically, the best furosemide dose to administer is the lowest effective dose.

Treating the first episode of acutely decompensated HF is usually successful. A recent study showed an estimated 80% survival rate to discharge for dogs and cats with AHF that were admitted to a university emergency department in an urban setting.[8] Once a cat is breathing normally and has the desire to drink water, this is generally a good indication that it may be ready for hospital discharge. Because cats are often unsettled in veterinary hospitals, waiting to discharge a cat until it eats on its own may be a mistake as most cats will eat better in their home environment. While many cats with HF are typically not drinking or eating at presentation, one should resist the temptation to use IV or SQ fluids (e.g. LRS, 0.9% NaCl) in the initial treatment as it may be harmful and worsen cardiogenic pulmonary oedema. Most cats will start eating and drinking after successful management of HF, although tempting a patient to eat its favourite food is always sensible. One should postpone any implementation of a sodium restricted diet until the cat is stable and eating well. If the cat is not eating at the time of discharge, it may also be prudent to withhold ACE inhibitors transiently until it is eating. Serum potassium level should also be measured before discharge, especially in cats displaying inappetance. If hypokalaemia is detected, this can be treated effectively by concomitant administration of potassium supplementation and/or potassium-sparing agents such as spironolactone (2 mg/kg orally q24h; this takes a few days to have maximal effect), although sufficient data of clinical efficacy of spironolactone in symptomatic cats are currently unavailable.[19]

An important and often overlooked part of the successful emergency management of HF is open communication with the owner regarding the emotional, practical, and financial ability to deal with the long term management of the animal's heart disease. Survival times for most cats in HF with treatment vary from 6 months to 1 year depending on the underlying aetiology of the heart disease and comorbidities.[14,76,87—89] Finally, it should be noted that administration of any forms of medication may be an insurmountable obstacle to treatment in many cats and a pragmatic approach is often needed in some cases. It is a good practice to contact the owners for follow-up a few days after discharge and arrange a clinical recheck after approximately 1—2 weeks, depending on individual circumstances.

Conflict of Interest

The authors do not have any conflict of interest to disclose.

Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jvc.2015.09.007.

Video	
Video 1	Chest percussion performed on a 14-year-old female neutered domestic shorthaired cat presented for acute onset of tachypnoea and dyspnoea. The sound becomes duller when percussion is performed on the ventral part of the chest, on both sides. A point-of-care thoracic ultrasound examination reveals severe right ventricular and right atrial enlargement and an anechoeic area in the pleural space consistent with pleural effusion.

Video 2	Point-of-care thoracic ultrasound exam in a cat presented with acute onset of tachypnoea/dyspnoea. Ultrasound images show moderate amount pleural effusion, scant pericardial effusion and severely diminished systolic myocardial function with an irregular endocardial surface with slight left ventricular hypertrophy, most likely representing an end-stage form of cardiomyopathy. A more confident diagnosis of congestive heart failure was achieved after the point-of-care ultrasound exam.
Video 3	Clinical presentation of a cat with acute onset of tachypnoea/dyspnoea before and after successful thoracocentesis, which yielded approximately 220 mL of fluid. A marked improvement of respiratory rate and effort is observed just a few minutes after the procedure.

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