European Society of Veterinary Cardiology

ESVC Precongress - September 17th 2025 – MECCC Maastricht

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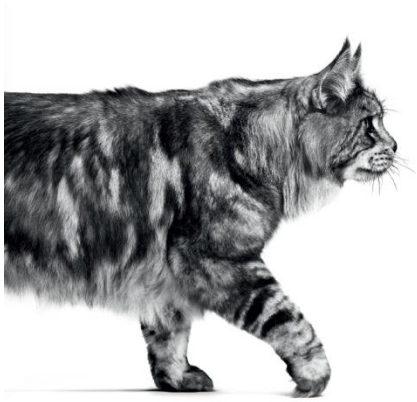

European Society of Veterinary Cardiology (ESVC) thanks Speakers and Sponsors for their support to the ESVC pre-congress day 2025

PRECONGRESS PROGRAM

Time	Title	Speaker
09.00-09.30	Registration	
09.30-10.10	Ventriculo-arterial coupling and hemodynamic evaluation in the intensive-care setting	*F. Guarracino*
10.15-10.55	Echocardiographic assessment of hemodynamically unstable patients during the perioperative period	*F. Guarracino*
11:00-11.30	Coffee break	
11.30-12.10	Diagnostics in acute heart failure in dogs and cats	*T. DeFrancesco*
12.15-12:55	Treatment of acute heart failure in dogs and cats	*T. DeFrancesco*
13:00-14:00	Lunch	
14:00-14:40	Collapsing episodes – head or heart?	*J. Loureiro*
14:45-15:25	Code red: Tackling critical canine arrhythmias	*R. Willis*
15.30-16:00	Coffee break	
16:00-16.40	Mechanical ventilation in cardiac patients	*C. R. Kennedy*
16.45 18.00	Panel discussion	*All Speakers*
18.00	End of the meeting	

Ventriculo-arterial coupling and hemodynamic evaluation in the intensive-care setting

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Head of the Department of Cardiothoracic and Vascular Anesthesia and Intensive Care at Azienda Ospedaliero Universitaria Pisana in Pisa, Italy

Ventriculo-arterial coupling (VAC) describes the dynamic interaction between the left ventricle (LV) and the arterial system, reflecting how effectively the heart pumps blood into the circulation. It is a fundamental concept in cardiovascular physiology that links myocardial performance to arterial load, ultimately determining cardiovascular efficiency and energetic cost.

The core of VAC lies in the relationship between two parameters: end-systolic ventricular elastance (Ees) and effective arterial elastance (Ea).

- End-systolic elastance (Ees) represents the contractile strength of the left ventricle at the end of systole. It is a load-independent measure of myocardial contractility, defining the stiffness of the ventricle when it has completed contraction.
- Effective arterial elastance (Ea) is an integrated measure of arterial load, encompassing arterial resistance, compliance, characteristic impedance, and heart rate. Ea represents the net arterial "stiffness" or afterload against which the ventricle must eject blood.

The ratio Ea/Ees provides a quantitative index of VAC. An optimal ratio (typically around 1.0) reflects a state where the heart and arterial system are well-matched, maximizing mechanical efficiency and stroke work while minimizing myocardial oxygen consumption.

When ventriculo-arterial coupling is optimal, the LV ejects blood efficiently into the arterial system, preserving stroke volume and maintaining adequate perfusion pressure with

minimal energy expenditure. Conversely, a mismatch in coupling — such as increased arterial elastance (higher Ea) or reduced ventricular contractility (lower Ees)
— leads to impaired cardiovascular performance (uncoupling), increased myocardial workload, and can contribute to heart failure and other cardiovascular diseases.

Understanding VAC is clinically important for assessing cardiovascular health and guiding therapy. Interventions such as vasodilators, inotropes, or device-based therapies can modulate Ees and Ea to restore optimal coupling, improve cardiac efficiency, and provide adequate stroke volume and organs perfusion.

Echocardiographic assessment of hemodynamically unstable patients during the perioperative period

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Echocardiography plays a pivotal role in the evaluation and management of perioperative hemodynamic instability. By providing direct, non-invasive visualization of cardiac structures and real-time assessment of function, it enables targeted evaluation of key determinants of circulatory failure, including ventricular systolic and diastolic performance, right and left ventricular loading conditions, intravascular volume status, and pericardial pathology.

Integrated with functional hemodynamic monitoring, echocardiography allows dynamic assessment of fluid responsiveness through parameters such as velocity-time integral changes and inferior vena cava variation, complementing conventional indices like pulse pressure and stroke volume variation. It facilitates differentiation between primary cardiac dysfunction, inadequate preload, excessive afterload, and extrinsic constraints, thus guiding precise therapeutic interventions.

In the perioperative setting, where rapid decision-making is essential, echocardiography adds significant value by enabling individualized diagnosis and optimization of hemodynamic status, ultimately supporting timely and effective management of unstable patients.

Diagnostics in acute heart failure in dogs and cats

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Heart failure (HF) is one of the most common causes of acute dyspnea in dogs and cats presenting in an emergency setting. Unfortunately, respiratory distress can be due to many different etiologies. Regardless of the etiology, managing a dog or cat in respiratory distress is challenging. Excessive handling, restraint and diagnostics can be counterproductive, especially in cats. Prompt recognition of heart failure and any comorbidities will aid in providing appropriate therapeutics to stabilize the patient in the short term and long term. The **initial diagnostic evaluation needs to be efficient, high-yield and minimally stressful** for the patient. It is important to remember that there is **NO single diagnostic test for congestive heart failure**. The diagnosis is based on a combination of findings including signalment, historical interrogation of clinical signs and results of diagnostic tests, primarily including physical examination, thoracic and cardiac imaging.

History and physical examination

History and physical examination remain the cornerstone in the assessment of patients with HF. A thorough history by a team member (while other team members assess the patient) helps to identify the potential etiology of the respiratory distress, precipitating factors or co-morbidities. While a good proportion of patients with acute HF are due to worsening of chronic HF, others present with HF for the first time. Acute decompensated HF can occur without known precipitant factors but often occurs with one or more precipitating factors such as the development of a tachyarrhythmia, systemic infection or anemia or administration of IV fluids or sodium retaining medications. Good questions to ask include:

- Is this a recurrence or worsening of previously diagnosed HF? If so, current treatments?
- Previous history heart disease, or murmur? If so, current treatments?
- Events leading up to the signs? (i.e., stressful event, steroid administration)
- When did these signs start and progression of signs? Any previous treatments?

- Any other previously identified conditions or current therapies?
- Any other abnormal clinical signs? (anorexia, lethargy, vomiting…)
- Heartworm prevention? Retrovirus status? Indoors or outdoors? Supervised or unsupervised?
- Diet? Does the pet eat a non-traditional grain-free diet?

The physical examination is targeted to the cardiovascular and pulmonary systems. Increased respiratory rate and effort of varying degrees should be assessed and evaluated for signs of impending respiratory failure. Typical lung auscultation findings for cardiogenic pulmonary edema are increased breath sounds and pulmonary crackles. Dogs with HF secondary to myxomatous mitral valve disease typically have loud systolic murmurs and tachycardia. Dogs with dilated cardiomyopathy on the other hand typically have soft or barely audible systolic murmurs, gallops sounds and tachyarrhythmias, such as atrial fibrillation or ventricular arrhythmias. The physical examination of a cat with HF is variable with respect to heart rate, rhythm and presence of murmur and oftentimes is not helpful to corroborate or refute a CHF diagnosis. **Blood pressure** (BP) in the ER world is considered the 4th vital sign. In a patient with signs of low cardiac output (cold extremities, tachycardic, weak), a corroborating low BP (< 90 mmHg systolic or 60 mmHg mean) is worrisome for low cardiac output and need for inotropic support. Alternatively, a persistently high BP (if deemed real vs situational) in a HF patient could potentially exacerbate LV afterload and hinder management of HF.

Thoracic point-of-care ultrasound

In the author's practice, every dog or cat with respiratory distress will have a thoracic point-of-care ultrasound (POCUS) exam performed shortly after intake functioning as an extension of the physical exam. The POCUS exam is performed with the patient in sternal recumbency while receiving flow-by oxygen supplementation. The POCUS exam is a comprehensive exam of the heart, lung and pleural space. Thoracic (POCUS) can provide immediate, low-stress and high-yield diagnostic information to guide therapy, especially in the cat as physical exam and radiographic findings are often non-specific. The POCUS findings suggestive of left-sided HF are an **enlarged left atrium** (usually with LA:Ao ratio > 2:1) **and numerous, bilateral strongly positive B-lines sites**. Although B-lines are not specific for

pulmonary edema, when combined with an enlarged left atrium and clinically integrated, these POCUS findings increase one's confidence for the diagnosis of HF. B-lines can also be monitored during therapy but resolution of b-lines will lack behind clinical response and respiratory rate. The POCUS exam can also rapidly identify findings suggestive of a left atrial rupture (pericardial effusion), a devastating complication of myxomatous mitral valve disease. Cats with congestive heart failure can manifest with any combination of pulmonary edema (B-lines), pleural effusion and pericardial effusion. The finding of pericardial effusion is highly specific for a HF diagnosis. In a severely decompensated patient, one would postpone further diagnostics and start treatment based on these initial findings. In a dog or cat that presents with abdominal distention and ascites, the finding of distended hepatic veins and an enlarged caudal vena cava with minimal respiratory fluctuations is suggestive of increased right heart filling pressures and right HF.

Thoracic radiographs

Thoracic radiographs also play an important role for the diagnosis of HF. In our hospital, thoracic radiographs are postponed until after initial treatment and stabilization. In two recent meta-analysis in humans, the diagnostic accuracy of lung ultrasound for congestive heart failure was better than thoracic radiographs. The radiographic diagnosis of HF is typically based on the finding of pulmonary venous distention, presence of left-sided cardiomegaly and a consistent pulmonary pattern. In the dog, cardiogenic pulmonary edema appears as increased interstitial to alveolar pulmonary opacities with typically a caudal-dorsal distribution. A recent study of **dogs presenting with respiratory signs, vertebral left atrial size (VLAS) was a significantly more accurate predictor of congestive HF** as compared to vertebral heart size (VHS), (AUC, 0.92; 95% CI, 0.85-0.96). Radiographic findings in cats in HF can be variable and nonspecific. One study showed that **a VHS of > 9.3 vertebrae in the cat was very specific for the presence of heart failure**. Normal cats' VHS are typically less than 8 vertebrae. Cats with HF often have some degree of pleural effusion, that can obscure will the cardiac silhouette, pulmonary veins and pulmonary parenchyma. In cats with HF, pulmonary arteries can be more prominent than pulmonary veins. However, a recent study showed that right-sided caudal pulmonary venous distension at the level of the 9^{th} rib is 75% sensitive and 100% specific for the diagnosis of HF in cats.

Cardiac troponin and NTproBNP

Used in conjunction with other diagnostic tests, cardiac biomarkers can be helpful in the diagnosis of HF. In the acute setting, the point-of-care **proBNP** test in cats (of both plasma and pleural effusion) has been shown to be helpful to differentiate cardiac from non-cardiac causes of respiratory distress. The point-of-care BNP test in the cat is better to rule out HF than ruling it in as some cats without HF may have elevated SNAP proBNPs. A point-of-care quantitative proBNP test is now available in dogs (and cats), however clinical trials assessing its accuracy in diagnosis heart failure are not published to the authors knowledge. Send out tests for proBNP, may still be useful but the results may take several days. In the author's experience, most sendout proBNPs when in active HF are > 3000 pg/ml in the dog and > 1000 pg/ml in the cat.

The usefulness of **cardiac troponin** in the acute HF setting is to potentially identify cats with possible Transient Myocardial Thickening (TMT) or possible myocarditis. The diagnosis of TMT is made when reverse remodeling is identified echocardiographically, several weeks to months after an initial diagnosis of HF, often severe HF. Many cats with TMT are young and an antecedent or predisposing event is often identified.

Salient laboratory analysis

In the ER setting, point-of-care lab tests using a small volume of blood are usually performed when placing an IV catheter. In our hospital, these quick assessment tests would include a packed cell volume (PCV), total protein, blood glucose, lactate and a venous blood gas with electrolytes. **Lactate** functions as a marker of hypoperfusion and anaerobic metabolism. In acute HF, an elevated lactate level often signals circulatory insufficiency and predicts adverse outcomes. **Elevated blood glucose, hyponatremia and hypochloremia** have also been associated with worse outcomes. Once stabilized, all patients should ideally have complete blood count, and chemistry profile evaluating serum electrolytes, renal parameters (BUN, creatinine, SDMA) and T4 in cats performed to optimize management of diuretics and RAAS inhibitors. In some dogs (Golden Retriever, English Cocker Spaniels, eating nontraditional diets) or cats with dilated cardiomyopathy and HF, testing for possible taurine deficiency is advised.

Electrocardiography

ECG is the best test to evaluate heart rate and rhythm abnormalities. In the setting of acute HF, arrhythmias are not uncommon, especially in large breed dogs with dilated cardiomyopathy. The presence of atrial fibrillation in a dog or cat with suspect HF is very suggestive of heart failure. Conduction disturbances such a left bundle branch block is also worrisome for severe structural heart disease.

Echocardiography

Once the patient is stabilized and can lay comfortably on its side, quantitative echocardiographic parameters can suggest increased left heart filling pressure. In humans, an increased mitral inflow E/A ratio > 2:1, E/e' > 14, tricuspid regurgitation velocity > 2.8 m/s, and left atrial volume index of 34 ml/m^2 are suggestive of increased LV filling pressure. In the dog, in addition to LA size, increased TR velocity suggesting post capillary pulmonary venous hypertension, and high MV inflow peak E velocity, the Doppler echo ratio of peak E to LV isovolumic relaxation time (IVRT) may be one of the better predictors of increased LV filling pressures. In an experimental model of left-sided heart failure, E:IVRT ratio outperformed various other commonly used doppler echo variable including the E:E'. Normal peak E:IVRT is generally < 2. As LA pressure increases > 15-20 mmHg, E:IVRT also increases (2.5 – 4).

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Treatment of acute heart failure in dogs and cats

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The immediate goals of HF therapy are to reduce abnormal fluid accumulations and restore comfort in breathing while providing an adequate or improved cardiac output. While treating pathologic arrhythmias are paramount to the management of acute HF, it will not be discussed in this lecture.

Oxygen therapy and/or ventilatory support

Oxygen therapy may be given based on clinical judgment. In humans HF patients with respiratory distress, respiratory rates >25 breaths/min, and oxygen saturation <90%, oxygen therapy and non-invasive ventilation (ex. CPAP) are recommended. In veterinary medicine, **high flow nasal oxygen** is a safe and feasible way to support oxygenation in dogs and possibly cats beyond conventional oxygen supplementation. High flow nasal oxygen provides continuous positive airway pressure at flow rates of 1-2 L/kg/min (compared to 50-100ml/kg/min for conventional oxygen therapy). The benefits of continuous positive airway pressure allow recruitment of collapse alveoli and is associated with an increase in function reserve capacity. The oxygen supplied by the high flow is heated, humidified and can be blended with medical air. While routine use of anxiolysis is not routinely recommended in humans with acute HF, in the author's experience, **anxiolysis** with a low dose butorphanol is helpful in our veterinary patients especially when placing nasal prongs, an IV catheter and performing ER diagnostics.

In a patient with severe dyspnea that is not responsive to therapy and on the brink of respiratory failure, one should be prepared to take over the airway and ventilate if owners are so inclined emotionally and financially and if the pet could have a meaningful recovery (ex. First time HF). **Ventilation** supports their respiratory function while allowing their left atrium time to stretch (if ruptured chordae tendineae) and time for the medications to improve their cardiovascular function. Ventilation also allows one to perform further diagnostics in a safe environment if the cause of the dyspnea is uncertain. Most heart failure

patients (~> 60%) respond well to medical management and are able to be extubated within 12 - 36 hours.

Diuretics

Loop diuretics are the cornerstone of acute HF treatment because of their rapid onset of action and efficacy for the treatment of fluid overload and congestion. The challenges of diuretic use are optimal dosing, timing and method of administration for the individual patient and manifestation of HF. Ideally furosemide should be started with an initial IV bolus, however if IV administration is too stressful, an IM/SQ injection is a reasonable alternative. If the patient is already on oral diuretics prior to the acute HF episode, the dose should be 1.5 or 2 times the oral dose taken by the patient before presentation. If the patient was not previously on oral diuretics, then the initial **furosemide** dose is typically in the range of 1-3 mg/kg. In severe cases, furosemide dose can be repeated hourly for 3-4 hours to effect. Continued furosemide treatment can be given as either scheduled boluses every 6-12 hours or as a continuous infusion. Daily single bolus administrations are discouraged because of the possibility of post-dosing rebound sodium retention. In both human and veterinary clinical studies, no clear advantage has been identified between furosemide bolus and continuous infusion administration. There are similar decreases in respiratory rates and no significant short term mortality differences between furosemide administration given as **intermittent boluses or continuous infusion**. However, in a small experimental study of normal dog, the continuous infusion resulted in significantly higher urine output in 8 hours as compared to a single bolus dose. The author typically uses a continuous infusion of 4-8 hours after an initial bolus dose, followed by scheduled IV bolus dosing prior to switching to oral administration.

Diuretic response should be evaluated shortly after the start of diuretic therapy by assessing respiratory rate, respiratory effort and urine output. In the 2021 European Society of Cardiology guidelines, assessing a spot urine sodium content measurement after 2 or 6 h is also recommended. In humans, a satisfactory diuretic response can be defined as **a urine sodium content >50-70 mEq/L at 2 h and/or by a urine output >100-150mL/h during the first 6 h**. Urinary Na excretion and urine volume are complementary but distinct aspects of diuretic responsiveness. In a retrospective study of 51 dogs with acute HF, dogs with low urinary Na excretion had significantly longer mean time in supplemental oxygen oxygen than dogs with high urinary Na (uNa <87 mmol/L, 24.2 ± 2.6 hours vs uNa ≥87 mmol/L, 16.6 ± 1.7

hours; P = .02). In this study, urine Na concentration outperformed other metrics of diuretic responsiveness including weight loss.

A concern that we all have in the management of acute HF is worsening renal function. Traditionally, worsening renal function has been associated with worse outcome and a poor prognostic indicator. However, several recent studies in both humans and veterinary medicine have challenged this assertion. Worsening renal function may simply be reflective of a normal renal response to effective decongestion. In fact, worsening renal function and hemoconcentration have been associated with better outcomes, such as greater improvements in decongestion, decreased mortality and HF re-hospitalization. This is corroborated by two retrospective veterinary studies showing how worsening renal failure did not affect the long-term outcome of dogs and cats treated with diuretics for hospitalized heart failure.

In situations in which loop diuretics have minimal response, concern for an incorrect diagnosis or diuretic resistance should be considered. Despite repeated diuretic dosing, the kidneys become less responsive due to adaptive mechanisms, and the drug's ability to promote sodium and water excretion diminishes. Factors contributing to diuretic resistance and diuretic braking include poor delivery of drug to the renal tubules; activation of the RAAS system; increased sodium reabsorption in the distal nephron; sympathetic nervous system activation; and renal structural changes, such as hypertrophy of the distal tubules. Pharmacologic strategies to improve diuretic response include increasing the diuretic dose, changing or combining diuretic agents; administering hypertonic saline with high dose furosemide to transiently increase intravascular volume and improve sodium delivery to the tubules of the nephron and lastly IV vasodilators and inotropes to improve cardiac output and renal perfusion. A recent meta-analysis of **hypertonic saline** in HF showed promising results including shorter mean hospital stays, lower mortality rates, fewer readmissions, and significant improvements in serum creatinine levels, 24-h urine output, and weight loss compared with intravenous furosemide therapy alone. Switching to torsemide or the addition of a **thiazide** diuretic should be considered in patients with resistant edema in patients that do not respond to an increase in diuretic dose. **Torsemide** has increased bioavailability and a longer half-life than furosemide. Despite these theoretical advantages, meta-analyses and a large clinical trial (TRANSFORM) of hospitalized HF human patients treated with torsemide vs furosemide did not result in a significant difference in all-cause mortality. Another diuretic

that may be helpful in the acute management is **acetazolamide**. Acetazolamide, a carbonic anhydrase inhibitor, that reduces proximal tubular sodium reabsorption, can improve the efficiency of loop diuretics, potentially leading to more and faster decongestion in patients with acute decompensated heart failure with volume overload. Initial studies have shown faster decongestion, as well significant reduction in serum pH, increase in natriuresis with acetazolamide among HF patients. Acetazolamide is considered a "chloride-sparing" diuretic that may help to reduce the development of hypochloremia, that been proposed as an indicator and mediator of diuretic resistance.

Another consideration for diuretic resistance and treatment failure is that **high capillary pressure for an extended period can also cause increased capillary permeability** and fluid transfer into the alveoli, leading to edema and atelectasis. Although cardiogenic pulmonary edema usually occurs in the absence of change in the permeability of the alveolar-capillary barrier, the capillary wall's permeability may also be affected during CPE, especially in cases of a sudden increase in pulmonary capillary hydrostatic pressure (i.e., flash pulmonary edema) or in chronic markedly increased hydrostatic pressure. This increased in hydrostatic pressure can cause mechanical damage on the alveoli–capillary barrier through a process known as "stress failure". The breakdown of the alveolar-epithelial barrier is a consequence of multiple factors that include dysregulated inflammation, intense leukocyte infiltration, activation of procoagulant processes, cell death, and mechanical stretch. Reactive oxygen and nitrogen species can damage ion channels, such epithelial sodium channels that are responsible for keeping the alveoli dry.

As veterinary medicine expands its use of extracorporeal therapies, **ultrafiltation** may be an alternative treatment strategy, especially in the setting of acute HF with poor diuretic response and renal dysfunction. Studies of human patients with decompensated HF and cardiorenal syndrome with inadequate diuretic response, ultrafiltration vs. diuretic intensification can be an effective and safe option. It reduces readmissions, creatinine levels and increases volume depletion without affecting mortality. Ultrafiltration may be useful in a decompensated HF patient with a potentially reversible cause of kidney injury as it is an invasive technique that carries a high economic cost.

Vasodilators

Intravenous vasodilators induce potent vasodilation within minutes of administration, leading to potentially rapid relief of dyspnea and reduced congestion. Intravenous

vasodilators dilate veins, arteries or both. In the author's practice, **nitroprusside** is the commonly used IV vasodilator because of its balanced vasodilation to dilate veins leading to a reduction in venous return to the heart, and arteries, leading to lowered afterload, increased stroke volume and consequent relief of symptoms. Because of their mechanisms of action, IV vasodilators may be very effective in those patients whose acute pulmonary edema is caused by increased afterload, or in flash pulmonary edema and minimal hypervolemia, such an in a dog with an acute chordae tendineae rupture. The 2021 European Society of Cardiology human guidelines state to consider using IV vasodilators as an adjunct to diuretics in acute HF patients with SBP >110 mmHg. The guidelines do not make a specific recommendation for specific IV vasodilator. Other vasodilators options include IV nitroglycerin, IV isosorbide dinitrate, nesiritide and clevidipine. Interestingly, while IV vasodilators are commonly associated with improved short-term outcomes, such as reduced intubation rates, no studies have shown an effect on mortality.

Other vasodilators, such as **hydralazine or amlodipine**, may also be helpful in dogs with refractory or recurrent congestive heart failure especially in dogs with myxomatous mitral valve disease and maintained blood pressures. These drugs may be good options (instead of sodium nitroprusside) depending on the severity of the patient's edema and clinical status. As with nitroprusside, one should start at low doses and titrate upwards based on clinical response and blood pressure. Hydralazine has a quicker onset of action than amlodipine and can be given IV. Amlodipine is only available as an oral medication and is the author's preferred additional long-term vasodilator because of its efficacy and it's once to twice a day dosing schedule.

ACE-inhibitors such as enalapril and benazepril are typically withheld in the per acute management of HF due to possible lowering of intrarenal perfusion and the glomerular filtration rate but are important in the chronic management to counteract the RAAS that can lead to diuretic resistance.

Inotropes

Inotropic agents are currently recommended in human patients in the inpatient setting presenting with acute HF with low cardiac output and hypotension. **In dogs, pimobendan, a positive inotrope and vasodilator, is initiated is virtually in all dogs with**

acute heart failure regardless of etiology and blood pressure. In a dog receiving pimobendan prior to onset of HF, the dose and/or frequency are escalated.

The clinical evidence for the usefulness of pimobendan in cats with acute HF is not robust. In a prospective randomized placebo-controlled double-blind multicenter nonpivotal field study of 83 cats with HCM and recently controlled HF, the addition of pimobendan (to furosemide) did not result in improved outcome. However, a subgroup analysis of this study did show a benefit for cats without left ventricular outflow tract obstruction and worse outcome for cats with left ventricular outflow tract obstruction. These results contrast with a previously published retrospective case-controlled study of pimobendan that showed an improved survival in cats with and without left ventricular outflow tract obstruction.

Other IV inotropic agents such as **dobutamine and milrinone** may be used in a refractory hypotensive patient, especially if IV pimobendan is not available. Milrinone is a phosphodiesterase 3 inhibitor that increases cardiac inotropy, lusitropy, and peripheral vasodilatation. In contrast, dobutamine is a synthetic catecholamine that acts as a β_1- and β_2-receptor agonist and improves blood pressure by increasing cardiac output. Both agents are considered inodilators, although milrinone may have more potent vasodilator effects. In human medicine, current clinical practice is to use milrinone in patients with severe pulmonary hypertension because of a suggested mechanism of reducing pulmonary-artery pressures and improving right ventricular function. Additionally, concerns regarding the effects of dobutamine on heart rate and myocardial oxygen consumption have mitigated its use in patients who are at risk for tachyarrhythmias or myocardial ischemia. However, a recent prospective randomized comparator study (the DOREME trial) showed no significant difference in outcomes between milrinone and dobutamine in patients with cardiogenic shock. Both are given as a continuous infusion, starting at low doses and titrate upward based on arterial blood pressure and heart rate.

A new positive inotrope, **omecamtiv mecarbil,** a direct cardiac myosin activator, improves cardiac function through an increase in actin–myosin interaction without affecting calcium transients. The drug increases stroke volume and ejection time in healthy volunteers and HF patients dependent on plasma concentration. The drug can be administered orally. In the GALACTIC-HF trial of 8256 human patients with HF with reduced ejection fractions, omecamtiv slightly reduced the primary endpoint of cardiovascular death (8% relative risk reduction) and HF hospitalizations. The effects were larger in the patients

with lower LVEF as well as in those with criteria for severe HF. In addition, the drug was well tolerated, with no significant changes in blood pressure or kidney function. However, the US Food and Drug Administration (FDA) has declined to approve omecamtiv mecarbil for treatment of adults with chronic heart failure with reduced ejection fraction (HFrEF), citing a lack of evidence on efficacy.

Vasopressors

Norepinephrine, a peripheral arterial vasoconstrictor, may be used in patients with severe refractory hypotension. The aim is to increase perfusion to the vital organs. However, this is at the expense of an increase in LV afterload. Therefore, low doses such as 0.2-1.0 µg/kg/min and, a combination of norepinephrine and inotropic agents are used in patients with advanced HF and cardiogenic shock. In human patients with cardiogenic shock, epinephrine, compared with norepinephrine, was associated with a three-fold increase in the risk of death, higher incidence of refractory shock, higher heart rates and lactic acidosis. There is a lack of information about the use of vasopressor in dogs and cats with cardiogenic shock.

Centesis

If the pleural or abdominal effusion is of significant quantity contributing to respiratory compromise, the most effective, immediate therapeutic maneuver is the removal of the fluid, in addition to diuretic therapy. This is usually performed under sedation, local block and ultrasound assistance.

Thromboembolism prophylaxis

Thromboembolism prophylaxis with **clopidogrel** or another antithrombotic agent is recommended for all cats in congestive heart failure due to the increased risk for arterial embolic events. A recent pharmacogenomic study of cats with HCM had a genetic variant of the ADP receptor resulting in a reduced response to clopidogrel. These cats may benefit from dual antithrombotic therapy or another anticoagulant. Genetic testing of this genetic variant is advised for cats at high risk for arterial thromboembolism.

Pre-discharge assessment and post discharge management planning

Once comfort in breathing and hemodynamic stabilization is achieved with IV and in-hospital therapy, treatment should be optimized before discharge. Initiating or restarting oral is ideal as doses can be optimized before discharge. However, due to stress and cost of hospitalization, the time spent on oral medications while in hospital is often short. Therefore, client education and early post discharge monitoring in the home environment are key to optimizing medical treatment. Educating the owner to monitor clinical signs of recurrent heart failure and establishing scheduled rechecks will be important. Spend some time with the owner to educate them as to how to monitor home resting (ideally sleeping) respiratory rate. Work with the client to optimize medication compliance and economic impact on the family. An outpatient recheck within 1-2 weeks after hospitalization is advised to assess efficacy and optimize therapy.

Recent human studies have shown that early (prior to or within 1-2 weeks of hospital discharge) introduction of sodium–glucose cotransporter 2 inhibitors (SGLT2is) and/or angiotensin receptor-neprilysin Inhibitor in patients hospitalized for acute HF improves all-cause death and rehospitalization rates. The utility of these drugs in veterinary heart failure management is unknown currently.

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Collapsing episodes – head or heart?

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Collapsing episodes are defined as acute losses of postural tone followed by spontaneous recovery representing a complex and often diagnostically challenging clinical presentation. The complexity of these cases arises from the wide range of potential underlying causes, transient nature of these events and frequent lack of physical examination abnormalities between episodes contrasting with the life-threatening potential of some aetiologies. A thorough and systematic approach is essential to tackle this challenging clinical presentation.

The diagnostic process must start with obtaining an impeccable history, focusing on when the episodes occur, the relation to a possible trigger such as exercise, excitement, cough, rest or feeding and whether the episodes can be induced and/or pre-empted. Following this, a detailed description of the event and recovery period should be pursued. Questioning should be directed to the details of how the episode starts, (i.e. tonic-clonic from the start, or flaccid initially progressing to stiffness), how it develops, how long it lasts and the details of the recovery period and duration. These steps are crucial, as further investigations will be guided by these details and may include a vast array of tests such as blood analysis, electrocardiography (ECG), echocardiography, Holter monitoring, advanced imaging (MRI), and, in some cases, electromyography (EMG) or muscle/nerve biopsies. Despite this relevance, this process is not without intrinsic challenges.

Questioning about collapsing episodes is difficult. First, people often struggle to provide precise descriptions of events due to the sudden, alarming nature of these episodes. Many report only the dramatic "end result" (e.g., "he just fell over") while missing critical details about pre-collapse behaviour or subtle prodromal signs and often overestimate the duration of the episode. Second, the terminology used can be misleading - terms like "seizure," "fainting," or "spacing out" may be applied inconsistently or inaccurately to describe fundamentally different phenomena. Third, recall bias frequently distorts histories, with people understandably focusing on the most dramatic aspects while overlooking potentially diagnostic details like exact body position, eye movements, or duration of recovery. The emotional distress surrounding these events further complicates history-taking, as people

may unintentionally emphasize certain aspects while minimizing others. Effective questioning requires patience, use of non-leading questions, and sometimes multiple interviews to reconstruct events accurately. These challenges underscore why video documentation has become increasingly vital in these cases and their role cannot be over-emphasised.

Once as much information as possible is gathered, the planning of further investigations and prioritisation of diagnostic tests is often assisted by considering the main causes of collapse as potentially metabolic, cardiovascular and neurological/neuromuscular.

Metabolic disorders typically cause persistent clinical signs until addressed clinically but may present as episodic collapse in early stages. Key metabolic aetiologies of collapse include anaemia, hypoglycaemia and electrolyte disturbances such as hypocalcaemia and hyperkalaemia. These conditions are often diagnosed with initial minimal database, but intermittent hypoglycaemia can be missed in non-fasted blood samples.

Cardiovascular aetiologies represent the most critical category due to their life-threatening potential and are often investigated after initial minimal database, unless the features of the episode are strongly indicative of a non-cardiac aetiology. Cardiovascular collapsing episodes are typically acute, brief, flaccid, involving the whole body and with rapid recovery to normal but in more severe or prolonged episodes, abnormal rigid postures such as opisthotonus and tonic-clonic features may develop due to brain hypoxia. Cardiovascular collapses can occur at any time but are usually more severe during periods of higher cardiac demand (exercise/excitement). While any significant structural heart disease (e.g., AS, PS, MMVD, DCM) may predispose patients to collapse due to reduction in cardiac output, three primary mechanisms typically occur to trigger the episodes:

1. Arrhythmias:
    - Bradyarrhythmias: High-grade 2nd degree AV block, 3rd degree AV block, sinus arrest
    - Tachyarrhythmias: Ventricular tachycardia, supraventricular tachycardia

Arrhythmias usually result in classic cardiac syncope features (acute, brief, and fast recovery to normal) and can occur at any time even at rest.

2. Pericardial Effusion/Cardiac Tamponade:
    - Acute: Hemangiosarcoma, left atrial rupture

- Chronic: Idiopathic pericardial effusion, infiltrative neoplasia

Cardiac tamponade usually results in a more prolonged collapse and recovery time.

3. Hypoxia:
    - Pulmonary hypertension
    - Right to left shunting

Collapses due to hypoxia can usually be elicited with excitement or exercise and may lead to opisthotonus with tonic-clonic activity often confused for seizures.

Neurological and neuromuscular causes of collapse typically involve either significant alteration in mentation/consciousness (seizures), abnormal involuntary movements (paroxysmal dyskinesias), or progressive weakness with preserved mentation (neuromuscular disease). The episodes are often prolonged with deficits persisting between episodes or in the peri-episode period. These features are in contrast with cardiovascular collapse and careful observation of episode characteristics assist this distinction:

1. Seizures:
    - Classic phases: Prodrome → Ictus → Post-ictus
    - Tonic-clonic movements
    - Prolonged post-ictal period (minutes to hours)
2. Paroxysmal Dyskinesias:
    - Breed-specific (CKCS, Scottish Terriers, etc)
    - Dystonic movements with or without subtle mentation alteration
    - Normal interictal periods
3. Neuromuscular Disorders:
    - Myasthenia gravis (exercise-induced weakness)
    - Polymyositis (progressive muscle pain/weakness)
    - Typically show persistent clinical signs between episodes
    - No change in mentation

In addition to the classic metabolic, cardiac, and neurologic causes of collapse, Exercise-induced collapse (EIC), Border Collie collapse (BCC), and vasovagal syncope are unusual yet important causes of collapsing episodes in dogs, each presenting distinct characteristics.

EIC is a genetic disorder primarily affecting working breeds like Labrador Retrievers, triggered by intense exercise and linked to a DNM1 gene mutation that disrupts neural transmission during high activity.

Border Collie collapse shares similarities with EIC as another exertion-related condition, but it occurs specifically in Border Collies without the DNM1 mutation and often involves more pronounced neurological signs like disorientation during episodes.

Vasovagal syncope differs fundamentally from both, being a reflex-mediated response to a specific stress/excitement trigger rather than physical exertion, though like EIC and BCC, it results in transient collapse without progressive inter-episode deterioration.

While EIC and BCC are exercise-dependent syndromes with breed-specific predispositions, vasovagal syncope is trigger-dependent and not limited to any specific breeds, but brachycephalic breeds may be predisposed. All three conditions share the hallmark of being paroxysmal events in otherwise normal dogs, but they require different diagnostic approaches - genetic testing for EIC, exclusionary diagnosis for BCC, and trigger identification for vasovagal syncope. Their management likewise diverges, with EIC and BCC requiring exercise modification, while vasovagal syncope focuses on trigger reduction and avoiding condition that may decrease blood pressure such as dehydration.

In summary, the diagnostic approach to collapsing episodes in dogs requires a systematic and thorough evaluation due to the diverse potential causes spanning metabolic, cardiovascular, and neurological/neuromuscular systems. A detailed history focusing on episode triggers, characteristics, and recovery patterns forms the cornerstone of the initial approach, though challenges in information recall and terminology often necessitate video documentation for accurate assessment. The key to successful management lies in careful episode characterisation, leveraging video evidence when available, recognizing signalment predispositions, and employing targeted diagnostics based on clinical features. Ultimately, a methodical approach combining thorough history-taking with appropriate diagnostic testing and collaboration of all involved is the best strategy for optimal outcomes for these complex cases.

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Code red - Tackling critical arrhythmias

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Introduction / General approach

Coumel's concept of a triangle of arrhythmogenesis states that three components are required to generate arrhythmias – a substrate, trigger and modulating factors. (FARRE, 2004) The substrate is often a damaged myocardium which may be remodelled anatomically (eg fibrosis, adipose tissue), electrically (eg action potential shortening or heterogenicity) or inflamed. Triggers include early or late afterdepolarisations, re-entrant activity and premature beats. Modulator factors principally involve the autonomic nervous system but can include drugs and genetic pre-dispositions. (Cheniti et al., 2018)

To assess these parameters in a clinical setting we would consider the following:

- Substrate: echocardiography, troponin
- Modulating factors: signalment / genetic predisposition, electrolytes
- Triggers: drugs, toxins, premature beats / pauses, R-on-T

Naturally there is interplay between these factors – for example a damaged substrate resulting in congestive heart failure tends to increase sympathetic nervous system tone. (Coumel, 1993) When attempting to predict success of treating an arrhythmia the substrate is of key importance – for example treating a ventricular arrhythmia post-GDV surgery with a structurally normal myocardium is much more likely to be successful than treating a similar arrhythmia in a Doberman with underlying cardiomyopathy. (Muir & Bonagura, 1984)

The effect of an arrhythmia on the patient can be very variable – some dogs with extreme tachycardia or bradycardia can appear normal whereas others may present collapsed.

To evaluate the effect of an arrhythmia on the cardiovascular system we will often require a "minimum database" including:

- Subjective assessment of responsiveness and demeanour
- Heart rate
- Respiratory rate

- Blood pressure
- ECG – 6 or 12 lead
- Echocardiography – often a brief echo
- Bloods – electrolytes and troponin

If we decide to treat then the next stages are to:

- Administer drug(s) – intravenous or oral
- Establish monitoring – clinical v ECG
- Establish success criteria

Tachycardias

Tachycardias can be sub-divided into categories:

1. Wide QRS complex tachycardia
    a. usually ventricular tachycardia (VT)
    b. less commonly supraventricular tachycardia (SVT) with aberrancy
2. Narrow QRS tachycardia usually supraventricular tachycardia (SVT)

Determining an ECG diagnosis can help to select an appropriate treatment and influence risk stratification.

Additional information to distinguish ventricular tachycardia from supraventricular tachycardia with aberrancy can be obtained using 12 lead ECG criteria as described in humans (Dresen & Ferguson, 2018) and, more recently, in dogs. (Perego et al., 2025)

Table 1: Six lead ECG features of wide and narrow QRS complex tachycardias:

	VT	SVT with aberrancy	SVT
Heart rate	>180bpm	>180bpm	>180bpm
QRS duration	Usually >80ms	>80ms	<70ms
QRS appearance	Wide / bizarre	Wide / bizarre	Similar to sinus beats
Duration	>4 consecutive beats	>4 consecutive beats	>4 consecutive beats
P:QRS	Atrioventricular dissociation	Consistent coupling of P:QRS	Consistent coupling of P:QRS
QRS morphology and rhythm	Monomorphic (single QRS morphology and single cycle length) or polymorphic (variable QRS morphologies and cycle lengths)	Generally monomorphic, regular or irregular rhythm	Generally monomorphic, regular or irregular rhythm
Capture / fusion beats	May be present	Unlikely to be present	Unlikely to be present

(Tilley, 1992) (Fox et al., 1999) (Willis et al., 2018) (Romito et al., 2024) (Santilli et al., 2018)

Risk stratification in cases with tachycardia

The risks associated with sustained tachycardia include sudden death and arrhythmia induced cardiomyopathy. However, it may be pertinent to consider whether our therapeutic target (eg reducing the risk of sudden death) is the same as the owners who may be more concerned about episodic collapse and also keen to avoid the side effects associated with some anti- arrhythmic drugs.

Factors to consider when assessing the risk of sudden death:

- Current signs compatible with haemodynamic compromise eg hypotension / collapse
- Breed
- Age (channelopathies tend to affect young dogs)
- Underlying disease
- History of collapse
- Presence of congestive heart failure
- Presence of R-on-T
- Frequent ventricular ectopy
- Presence of ventricular tachycardia

Treatment of tachyarrhythmias

The adage recommending treating the dog, not the ECG bears repeating! If the dog is stable despite the tachycardia, then oral treatment may be a reasonable option especially if using drugs that are rapidly absorbed such as sotalol however, in unstable cases, intravenous therapy may be indicated. There is geographical variation in the intravenous drugs available and those currently available in the UK include lidocaine, amiodarone, magnesium, sotalol, diltiazem and verapamil. This presentation will focus on emergency intravenous therapy rather than chronic oral therapy.

Lidocaine

i) Ventricular tachycardia

Lidocaine is classed as a Ib drug in the Vaughan-Williams classification system. (Williams, 1984) to systemic illness (GDV) receiving lidocaine showed that successful cardioversion occurred in 33 of them.(Muir & Bonagura, 1984). Additionally, even if lidocaine does not convert ventricular tachycardia to sinus rhythm, it does raise the threshold for ventricular fibrillation. (Gerstenblith et al., 1972) (Echt et al., 1989)(Allen et al., 1971)

However, lidocaine is not effective in all cases (Lynch et al., 1990) and toxicity can be seen especially after repeated doses – around 10mg/kg is likely to result in toxicity in dogs but, in some cases, it may be seen at lower doses such as 5mg/kg.(Lemo et al., 2007) (Wilcke et al., 1983). Reported adverse effects include

gastrointestinal signs (eg, hypersalivation, lip smacking, and vomiting) and neurologic signs (eg, sedation, head tremors, opisthotonus, body tremors, and seizures) however these adverse effects tend to be brief and self- limiting.(Wright et al., 2019) Typically 2mg/kg is given as repeated intravenous boluses 5-10 minutes apart to a cumulative dose of 8mg/kg and, if effective, followed by a continuous rate infusion. Lidocaine is less effective in cases with hypokalaemia and therefore if there is reduced response to lidocaine then checking serum potassium is advisable.(Singh & Williams, 1971) In some experimental models, lidocaine has a hypotensive effect which could be detrimental in dogs with tachycardia and low cardiac output.(Edouard et al., 1986)(Fazekas et al., 1994)

ii) Supraventricular tachycardia

Lidocaine is considered to be less effective for treatment of atrial tachyarrhythmias despite a study in isolated atrial and ventricular myocytes showing that lidocaine's sodium current inhibition was essentially identical in these 2 cell types.(Furukawa, 1995) This has been attributed to atrial myocytes having a lower (more depolarised) resting membrane potential resulting in less available inward sodium channels compared with ventricular myocytes. The combination of a smaller inward sodium current combined with the lack of a plateau phase in atrial myocytes also results in fewer inactivated sodium channels available to be blocked by lidocaine.(Furukawa, 1995)

However, lidocaine has been shown to be effective at converting orthodromic atrioventricular reciprocating tachycardia (OAVRT) to sinus rhythm (Wright et al., 2019) as the accessory pathway may exhibit similar properties to ventricular cardiomyocytes. Analysis of the ultrastructural morphology of cardiomyocytes within three surgically excised APs identified connexin 43 distribution matching that in ventricular cardiomyocytes and dissimilar to atrial or AV nodal cells.(Peters et al., 1994). The majority of the dogs in the study described by Wright et al responded to lidocaine and the non-responders were more likely to have antegrade conduction through the accessory pathway and / or postero-septal accessory pathways.(Wright et al., 2019)

Lidocaine has also been used for the conversion of vagally mediated atrial fibrillation to sinus rhythm.(Moïse et al., 2005)(Pariaut et al., 2008) Proposed mechanisms for this effect include prolonging the atrial refractory period, increasing the excitable gap in the re-entrant wave, or directly blocking the cardiac muscarinic receptors.(Nattel, 2003) However in the case series reported by Seo et al whilst two of the four cases converted to sinus rhythm, a Dobermann with dilated cardiomyopathy phenotype developed ventricular tachycardia then ventricular

fibrillation which was fatal; in two dogs, atrial fibrillation converted to atrial flutter prior to sinus rhythm and, in the fourth dog there was significant bradyarrhythmia prior to conversion to sinus rhythm so case selection and knowledge of prior medication may merit consideration prior to treatment.(Seo et al., 2022)

Magnesium sulphate

Intracellular and extracellular concentrations of magnesium have important effects on the electrophysiology of cardiomyocytes.(Perry & Illsley, 1986)(DiNicolantonio et al., 2018) (Humphrey et al., 2015)(Margn et al., 1993) Diuretic therapy during heart failure could also deplete body magnesium. The use of magnesium infusions in dogs with ventricular tachycardia has been reported and, although it was not shown to be effective as a sole agent, in some cases it may enhance the efficacy of lidocaine so merits consideration especially in refractory cases.(Ranninger et al., 2019)(Schoeller et al., 2020)(Gatson et al., 2022)

Amiodarone

Amiodarone is a class III antiarrhythmic although it possesses characteristics of all four classes so also blocks sodium, potassium and calcium channels resulting in slowed conduction and prolongation of the refractory period.(Desai et al., 1997) In healthy anaesthetised dogs, intravenous amiodarone had a mild depressant effect on myocardial contractility however this effect was much more marked after experimentally induced myocardial infarction.(Ware et al., 1991) Since 2008, Nexterone has been available and has been shown to be effective in dogs for the treatment of tachyarrhythmias without the negative side effects seen with earlier preparations that contained polysorbate or benzyl alcohol carriers.(Cober et al., 2009)(Somberg et al., 2002) Reversible hepatotoxicity was reported in 10/22 Dobermanns on oral therapy (Kraus et al., 2009) however in another study adverse effects were less frequent.(Pedro et al., 2012)

i) Ventricular tachycardia

A case series reporting the use of intravenous amiodarone in dogs with tachyarrhythmias showed that the drug terminated ventricular tachycardia in 7 out of 9 cases and was well tolerated with no significant change in blood pressure or acute adverse effects in all cases however a wide range of doses was used in these patients. (Levy et al., 2016)

ii) Supraventricular tachycardia

Intravenous amiodarone has been used in the conversion of acute atrial fibrillation in two dogs. (Oyama &

Prosek, 2006) Amiodarone has also been used to successfully convert supraventricular tachycardia and atrial flutter.(Levy et al., 2016)

Sotalol

Like amiodarone, sotalol is a class II ang-arrhythmic but also has I, II and IV properties.(Williams, 1984) This wide range of actions suggests that sotalol may be effective for multiple mechanisms of arrhythmia resulting in VT or SVT including OAVRT.(Romito et al., 2024)(Wright et al., 2018) Sotalol shows reverse use dependence and therefore may be less effective at higher heart rates.(Peralta et al., 2000) Similar to other drugs with beta adrenergic antagonistic activity, sotalol has a negative inotropic effect which in experimental models has been shown to be around 1/5 that of propranolol.(Hoffmann & Grupp, 1969)(Treseder et al., 2019) However a more recent study showed that oral sotalol was well tolerated in clinical cases even in the setting of systolic dysfunction.(Visser et al., 2018)

Diltiazem and verapamil

Calcium channel antagonists prolong atrioventricular conduction velocity and therefore can be effective in supraventricular arrhythmias (eg atrial fibrillation, atrial flutter and OAVRT), especially at high heart rates, but are unlikely to be effective for ventricular arrhythmias. (Atkins et al., 1995)(Miyamoto et al., 2001) (Melis et al., 2024)(Wright et al., 2018) Although diltiazem did not significantly lower systolic function in healthy anaesthetised dogs and has less negative inotropic effect than verapamil, caution is advised in cases with systolic dysfunction.(Kapur et al., 1988)(Whitehouse et al., 2023)

Esmolol

Esmolol is a 1 receptor antagonist with rapid and short duration of action.(Wiest & Haney, 2012) As well as the favourable negative chronotropic properties, esmolol also has negative inotropic properties and therefore extreme caution is advised in hypotensive patients with systolic dysfunction. In a case series, the majority of patients treated successfully had non-cardiac disease (eg toxin ingestion) resulting in supraventricular tachycardia.(Verschoor-Kirss et al., 2022). Esmolol has also been used for the conversion of atrial fibrillation to sinus rhythm in dogs.(Hassan et al., 2007)

Cardioversion

In patients with supraventricular tachycardia showing severe haemodynamic compromise, a manual cardioversion (also known as a precordial thump) can be performed. This manoeuvre involves exerting a sharp, controlled blow to the chest wall at the level of the heart in the hope that the energy delivered provokes a premature beat to interrupt a re-entrant circuit and restore sinus rhythm.(Atkins et al., 1995) (Melis et al., 2024)

Transthoracic electrical cardioversion involves delivering an electric shock synchronized with the intrinsic activity of the heart. As delivery of the shock is painful, electrical cardioversion should always be performed in anaesthetised patients. Synchronisation ensures that the electrical shock is not delivered during the vulnerable period of the cardiac cycle, i.e. from 60- 80ms before and from 20-30ms after the apex of the T wave. Electrical cardioversion can be used to convert all abnormal heart rhythms except ventricular fibrillation to sinus rhythm and is more likely to be successful if the substrate is normal (eg acute onset atrial fibrillation under GA).(Bright et al., 2005) (Bright & ZumBrunnen, 2008) Other factors that determine success include the positioning of the transthoracic pads to ensure as much of the myocardium as possible is depolarised simultaneously, the voltage delivered and also thoracic impedance.

Cats

Whilst arrhythmias are commonly seen in cats with heart disease, we often assume they are well tolerated which could in part be due to cats having a more sedentary, independent lifestyle that conceals overt clinical signs. Cats are often less compliant with medications than dogs and this could also contribute to our reluctance to administer anti-arrhythmic therapy. In cats with haemodynamically significant ventricular tachycardia the use of esmolol and amiodarone have been described. (Verschoor-Kirss et al., 2022) (Berlin et al., 2020)(Burkitt- Creedon et al., 2024) In cats receiving chronic oral amiodarone therapy the medication appeared to be well tolerated – transient gastrointestinal signs were noted in approximately 1/3 of the cats studied but no clinically significant laboratory abnormalities were documented.(Rossi et al., 2025) As cats are more susceptible to lidocaine toxicity, if this drug is used then lower doses than dogs and careful monitoring is required.

How do we evaluate success of anti-arrhythmic therapy?

An important area for future research is how we evaluate the success (or otherwise) of our ang arrhythmic therapy – for example are we aiming for resolution of clinical signs or a reduced risk of sudden death; or a significant change in ECG parameters such as complete suppression of the arrhythmia or a reduction in the Lown-Wolf grade? (Romito et al., 2024) Future therapies may seek cure of the arrhythmia via increased availability of EP studies and ablation for both supraventricular and ventricular arrhythmias; revisit the feasibility of implantable cardiac defibrillators and also increased use of stereotactic radiation therapy. (Sanglli et al., 2011) (Santilli & Perego, 2015)(Sanglli et al., 2018) (Hsue & Gagnon, 2023)

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Mechanical Ventilation for Cardiac Patients

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Mechanical ventilation (MV) uses a machine to generate positive pressure, forcing air into the respiratory system, filling the lungs, and generating a *tidal volume* (the volume of air that moves in or out of the lungs in a normal breath).[1,2] As the polar opposite to natural, spontaneous breathing that utilizes negative pressures to generate tidal volumes, mechanical ventilators use positive pressure and the term *positive pressure ventilation (PPV)* is used. In the healthy lung, convention dictates that the normal tidal volume for a dog is approximately 10 – 15 ml/Kg.[3] With diseased pulmonary parenchyma, and following trends in human critical care, tidal volumes have been trending towards lower volumes, i.e. < 10 ml/Kg.[1,2,3,4,5,6] Patients with pulmonary edema are considered to have diseased pulmonary parenchyma.

Mechanical ventilation can be highly effective. When indicated, it is a life-saving intervention that can provide possibilities for treatment and patient management that otherwise would not exist. However, it must be remembered that MV alone is not a therapy for the underlying disease, rather it supports the patient whilst the underlying disease is addressed or runs its time course.

Indications for mechanical ventilation include[1,2]:

- Hypoxic respiratory failure despite supplemental oxygen (partial pressure of arterial oxygen, PaO_2 < 60 – 80 mmHg; peripheral oxygen saturation of hemoglobin, SpO_2 < 90 – 93%).
- Hypercapnic respiratory failure ($PaCO_2$ > 60 mmHg).
    - Key point: patients that have increased reason to breathe, such as those experiencing cardiac failure, should *never* have an elevated or even normal $PaCO_2$. Hypercapnia is a sign of *extreme* crisis in cardiac failure patients.
- ***Impending respiratory fatigue***
    - Key point: waiting for a patient to fatigue prior to initiating MV is too late; it permits patient suffering, risks their life and reduces the successfulness of MV.

- Cardiovascular collapse, i.e., shock states.
    - In shock, breathing can account for > 20% of the oxygen utilization, which is unhelpful, as shocked patients breathe to obtain oxygen for other systems.

How Does Mechanical Ventilation Benefit the Heart Failure Patient – Respiratory Focus

PPV can be very effective when managing patients with cardiogenic pulmonary edema (CPE). In CPE, lung *compliance* – how easy the lung is to inflate – is decreased due to interstitial and alveolar edema, and the volume of usable lung for gas exchange is reduced.[4,5] Both of these increase the *work of breathing* – how much energy is required to breathe – thus the oxygen required to maintain breathing; they also lead to hypoxemia, progressive respiratory fatigue and hypercapnia. CPE patients often meet the clinical criteria to initiate PPV.

PPV can provide 100% oxygen, take over the work of breathing, and control patient ventilation such that CO_2 is effectively eliminated.[1,2,3] Simply, it can do what the patient is trying – and failing – to do. With the addition of Positive End-Expiratory Pressure (PEEP), the collapsed alveolar units can be recruited and alveolar edema even cleared, providing greater lung volume available for gas exchange.[1,2,4,5] PEEP can be thought of as CPAP (continuous positive airway pressure) for the intubated patient: in exactly the same way as a CPAP mask works, PEEP provides a continuous pressure such that the pressure within the respiratory system never drops below a set level, maintaining open alveolar units. By opening fluid-filled and collapsed alveolar units, PPV also improves lung compliance.

Importantly, PPV results in positive intrathoracic pressure, which reduces right heart preload. The reduction in preload decreases pulmonary capillary hydrostatic pressure, reducing the gradient for fluid efflux out of the capillaries into the interstitium and ultimately the alveolar space.[4,5,6] PPV also increases intra-alveolar pressure, providing a resistive force to fluid efflux from the pulmonary capillaries.[5] PPV can assist in clearing CPE.

How Does Mechanical Ventilation Benefit the Heart Failure Patient – Cardiac Focus

PPV, particularly with PEEP, has favorable effects on hemodynamics in patients with cardiac failure. The hemodynamic effects of PPV are related to increased intrathoracic pressure, which is a necessary consequence of using positive pressures. When intrathoracic pressure is

increased, right atrial pressure is also increased, which is the downstream pressure for venous return. As such, the gradient for venous return is decreased and preload is reduced.[4,5,6] This helps to unload the heart and reduce the formation of CPE.

PPV decreases left ventricular stroke work. The increased intrathoracic pressure pressurizes the left ventricle and intrathoracic aorta, such that left ventricular afterload is decreased.[4,5,6] This can improve left ventricular performance and is particularly helpful in patients with reduced systolic function. By reducing afterload, left ventricular myocardial oxygen demand is decreased. Positive pressures also reduce functional mitral regurgitation.[7]

PPV has been shown to improve myocardial perfusion and relieve ischemia, partly by providing increased oxygen and by relieving pulmonary hypoxic vasoconstriction, and partly by reducing sympathetic nervous activity.[5]

What Are the Risks or Negative Effects of Mechanical Ventilation?

Despite its benefits, PPV has the potential to negatively affect cardiac patients. Most of these negative effects are the consequence of increased intrathoracic pressures.

PPV and PEEP decrease venous return by increasing right atrial pressure: this decreases preload. While this is considered beneficial, particularly in the earlier stages of patient management, there is the potential for excessive preload reduction when combined with aggressive diuretic therapy. For example, a previously Stage B1 chronic valve disease patient that experiences an acute chordal rupture experiences severe, acute CPE; however, this patient is not systemically volume overloaded. While diuresis may be necessary, excessive diuresis can underload this patient: initiating MV can further reduce preload and consequently cardiac output and organ perfusion. However, while PPV can potentially reduce cardiac output, in patients with elevated filling pressures, PEEP *increases* cardiac output.[5] This is particularly relevant to the patients we see in small animal medicine, as most of our cardiac failure patients are volume overloaded. It also highlights the need for careful and frequent patient and pharmacological assessments when managing critical heart failure patients.

PPV increases right ventricular afterload. The lungs normally rest at an equilibrium point called *functional residual volume (FRC),* which is the volume of air remaining in the lungs and the end of normal, passive expiration. At FRC, the pulmonary vascular resistance (PVR), as

determined by the intrathoracic pressure and thoracic structure recoil pressures, is at its lowest.[4] With PPV, increasing intrathoracic pressure progressively collapses the alveolar pulmonary vasculature, leading to decreased usable vascular volume and increased PVR.[4,5,6] As sicker patients generally require higher pressures, this detrimental effect on right ventricular afterload is expected to be worse in sicker patients. Additionally, due to interventricular dependence, increases in right ventricular volumes and pressures can negatively affect left ventricular performance.[4,5] However, PVR, from a respiratory viewpoint, exists as a U-shaped curve (figure 1). When the alveolar units are collapsed, PVR is increased: the application of PPV and PEEP can open alveolar units, opening collapsed vessels that are tethered to these units and *decreasing* PVR.[4,5,6] Some of the increase in right ventricular afterload can also be offset by the reduction in hypoxic pulmonary vasoconstriction with the improved oxygenation achieved by PPV.

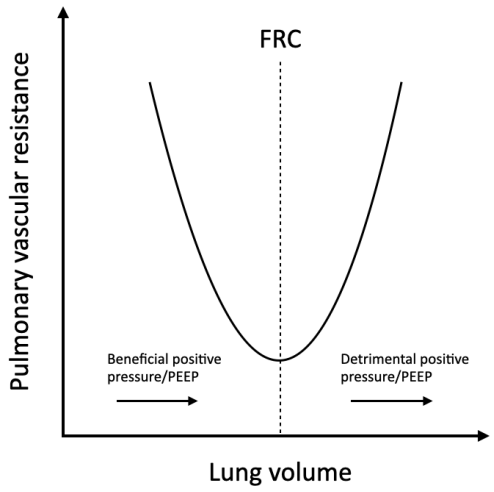

Figure 1: The U-shaped curve of pulmonary vascular resistance. At both low and high lung volumes, pulmonary vascular resistance is increased. If positive pressure/PEEP move the lungs from an excessively low volume toward FRC, pulmonary vascular resistance decreases; if positive pressure/PEEP move the lungs to higher volumes, pulmonary vascular resistance increases.

Dynamic hyperinflation leads to the phenomenon of auto-PEEP. In patients with expiratory airflow obstruction or those that require a longer time to fully breathe out the previous tidal volume, inspiration initiated before complete exhalation can lead dynamic hyperinflation (the

progressive accumulation of air in the lungs).[1,2,3,4,5,6] Also termed breath-stacking, this trapped and accumulating air increases the end-expiratory volume and thus pressure in the respiratory system above the set PEEP level, termed auto-PEEP. Progressive auto-PEEP worsens venous return, cardiac output and systolic blood pressure, and increases PVR.[4,5,6] Ultimately, death can occur if this problem progresses unnoticed. It is also one explanation for the medically mysterious Lazarus effect whereby a patient declared dead can auto-resuscitate once cardiopulmonary resuscitation has been stopped.

PPV reduces renal blood flow and glomerular filtration, leading to renal sodium and water retention.[4,6] Additionally, PEEP increases antidiuretic hormone secretion.[4] Both of these are unhelpful in the congested cardiac failure patient.

What Alternatives Are There to Mechanical Ventilation?

MV can be effective and lifesaving in cardiac failure patients. Perhaps most importantly, it can relieve dyspnea and the associated suffering, and can prevent fatigue. Given the expense, possible complications and psychological block many clinicians and owners experience to the idea of intubation and ventilation, success rates are artificially decreased and outcomes are pre-ordained to be poor. In many cases, MV is initiated too late – a patient should never be allowed to fatigue before MV is initiated.

Considering the reluctance to intubate, what are the options available for the struggling patient? While non-invasive ventilation is in its infancy in veterinary medicine, there are some options available. High-flow nasal cannulae can deliver humified, heated, pure oxygen at high rates and can also generate a small amount of CPAP. Oxygen helmets can also provide CPAP and can, in theory, but set-up to provide bi-level non-invasive ventilation by connecting these helmets to a mechanical ventilator. Limitations of these modalities include a lack of experience, poor patient selection, ineffectiveness for a given patient and, most importantly, they can delay necessary intubation, promoting patient suffering and risking life. In some cases, however, non-invasive augmented oxygen modalities can be highly effective: a positive response, if there will be one, is typically seen within 1 – 2 hours.

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