Heart Fauiler

Dr Nooshin Derakhshandeh

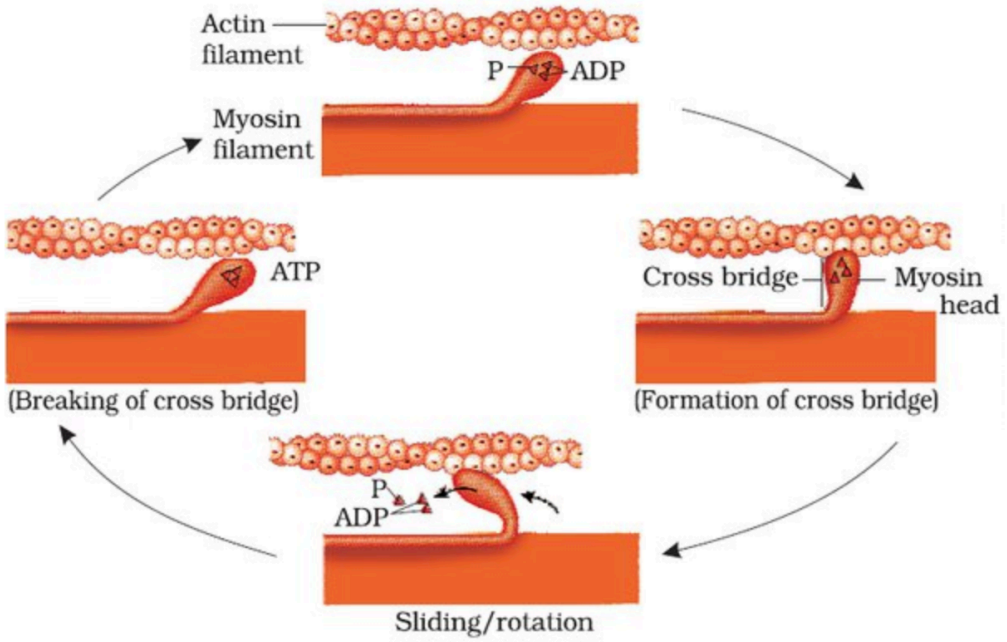
- Heart failure occurs when the heart is unable to deliver sufficient oxygen to the body's tissues due to impaired cardiac function. Despite adequate venous return to the heart, there is a maldistribution of blood.

- Congestive Heart Failure (CHF) involves increased venous and capillary pressures, causing fluid accumulation in tissues and organs.

- The heart undergoes biochemical, mechanical, and functional changes during failure.
- **Biochemical Change:**

Decreased ATP hydrolysis affects cardiac contraction:

decreased myocardial fiber shortening. Decreased contractility or pump function



- A decrease in contractility or pump function that is observed with heart failure.

- In a healthy heart, increased ventricular filling (or preload) results in a more forceful contraction.

- The failing heart, however, generates a lower force of contraction (systolic function) than expected despite an increased preload present prior to contraction. for any given preload, the systolic function is less than anticipated when compared with normal.

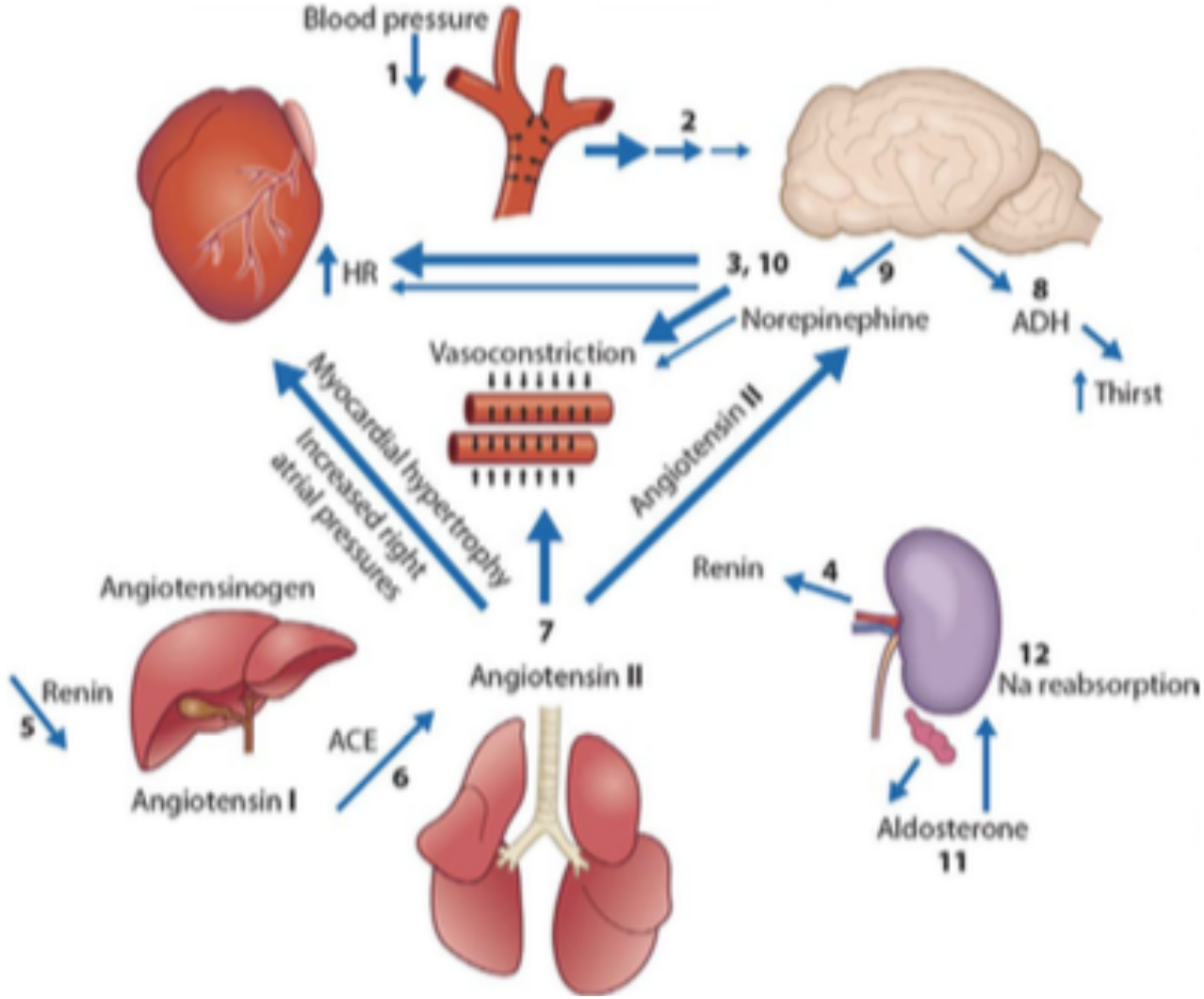
- **Hormonal Responses:**

Baroreceptors: Small decreases in cardiac output trigger adrenergic system activation: Increases heart rate, contractility, and causes peripheral vasoconstriction.

Renin-Angiotensin System:

Decreased renal blood flow releases renin, leading to angiotensin II production.

Angiotensin II Effects: Causes vasoconstriction, ADH release (reduces urine production), and aldosterone release (increases sodium and water retention).


Blood pressure
1
2
HR
3, 10
9
8
Norepinephine
ADH
Thirst
Vasoconstriction
Myocardial hypertrophy
Increased right atrial pressures
Angiotensin II
Renin
4
12
Na reabsorption
Aldosterone
11
Angiotensinogen
Renin
5
Angiotensin I
ACE
6
7
Angiotensin II

Definitions

- End-diastolic volume (EDV):
  Volume of blood in ventricles at the end of diastole = 110-130 mL.

- Stroke volume (SV):
  Amount of blood ejected from ventricles during systole (per each beat) = EDV-ESV = around 70 ml in an average adult at rest = 70-80 mL/beat

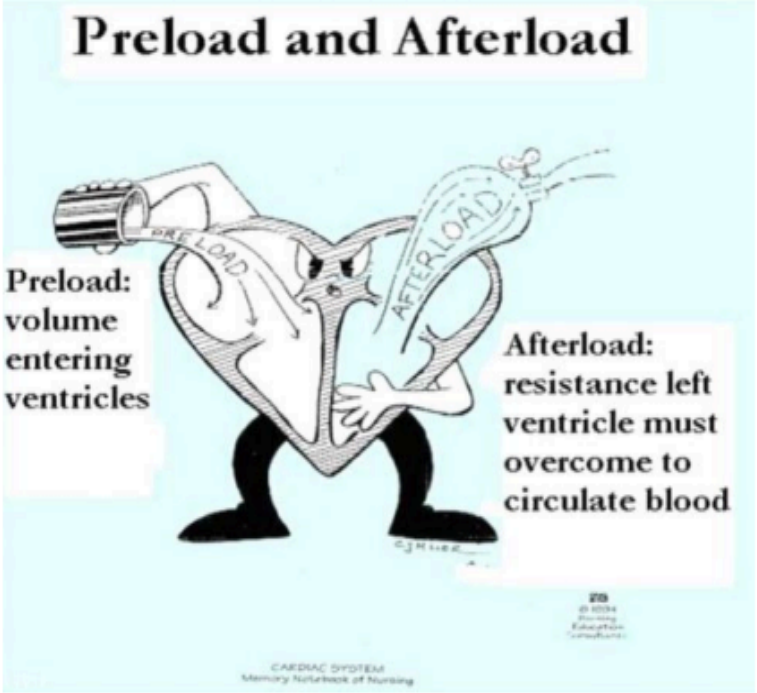
  - End Diastolic Volume and End Systolic Volume determine the Stroke Volume.

- End-systolic volume (ESV):
  Amount of blood left in ventricles at the end of systole = 40-60 mL.

- Ejection fraction (EF):
  Fraction of end-diastolic volume ejected during a heart beat =stroke volume/end diastolic volume = 60-65 %.

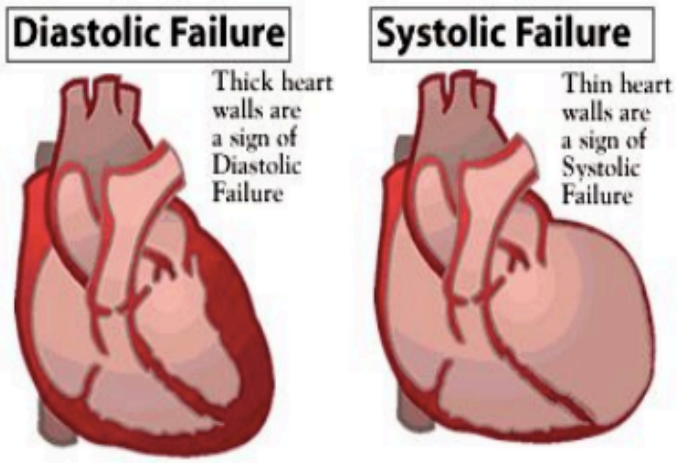
Preload and Afterload

- **Preload:** it is the amount of blood that returns to the heart from veins. (end diastolic pressure)
- Preload is the load on the muscle in the relaxed state.
- Is end-diastolic volume, which is related to right atrial pressure. When venous return increases, end-diastolic volume increases and stretches or lengthens the ventricular muscle fibers.
- **Afterload:** it is the resistance against which the ventricles contract.
- Increased afterload= increased cardiac workload.
- For the left ventricle is aortic pressure. Increases in aortic pressure cause an increase in afterload on the left ventricle and for the right ventricle is pulmonary artery pressure. Increases in pulmonary artery pressure cause an increase in afterload on the right ventricle.



Female slides only	Preload	Afterload
Increased in :	Hypervolemia Heart failure	Hypertension Vasocon-striction

Heart Failure



Systolic failure

This is the most common cause of HF

The heart loses its ability to contract or pump blood into the Circulation

In turn, it may not have the muscle power to pump the amount of oxygenated and nutrient-filled blood the body needs into the circulation

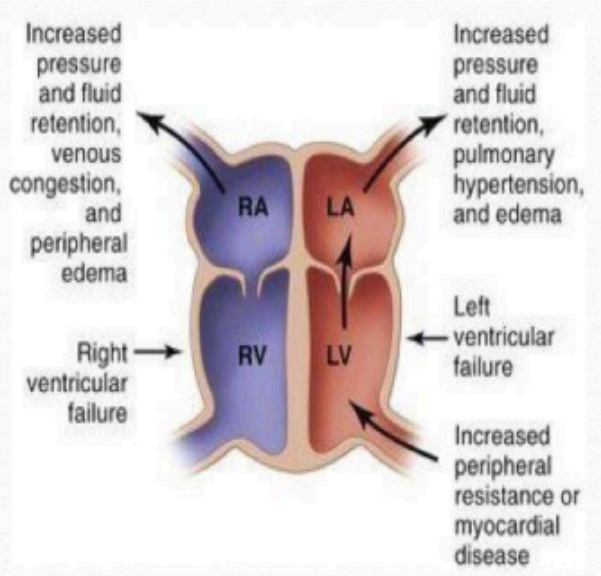
Diastolic failure

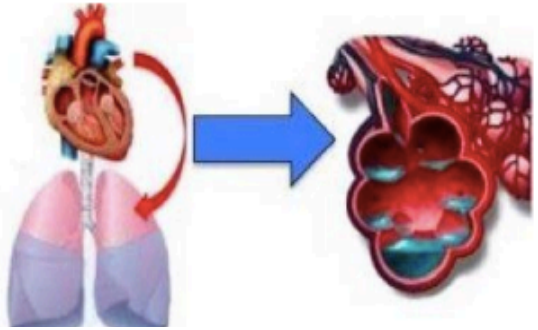
The heart loses its ability to relax because it becomes stiff
Heart cannot fill properly between each beat.

Often the walls of the heart thicken, and the size of the chamber may be normal or reduced.

Types of heart failure

- Left sided heart failure
  - Inadequate output of LV causing decreased CO to body and back pressure to the lungs. The left side of the heart is usually where heart failure begins.
- Right sided heart failure
  - Inadequate output of RV causing decreased CO to lungs and back pressure to venous system. It may occur alone but is usually a result of left- sided failure.
- Congestive heart failure
  - Chronically, left HF results in secondary pulmonary hypertension and right HF.





Heart failure Causes

Intrinsic myocardial causes (These result in reduction in ventricular contractility):

- myocardial infarction (death of cardiac myocytes due to blockage of the coronary arteries)
- Cardiomyopathy
- Myocarditis

Cardiac arrhythmias: e.g., complete heart block

Extrinsic causes (These make it more difficult to eject blood into aorta)

- systemic hypertension
- aortic stenosis

1- Impaired cardiac function

- Coronary heart disease
- Cardiomyopathies (muscle disease)
- Rheumatic fever
- Endocarditis

2- Increased cardiac workload

- Hypertension
- Valvular disorders
- Anemias
- Congenital heart defects

3- Acute non-cardiac conditions

- Volume overload
- Hyperthyroidism, Fever, Infection

Causes of left Sided HF

1. Systolic Dysfunction

Impaired Contractility

- Myocardial infarction
- Transient ischemia
- Chronic volume overload
- Mitral/Aortic Regurgitation

Increased Afterload

- Atrial Stenosis
- Uncontrolled HTN

2. Diastolic Dysfunction

Obstruction of LV filling

- Mitral Stenosis
- Pericardial constriction or tamponade

Impaired ventricular relaxation

- Hypertrophic or restrictive cardiomyopathy
- Transient ischemia

- In both types, blood may "back up" in the lungs causing fluid to leak into the lungs (pulmonary edema)
- Fluid may also build up in tissues throughout the body (edema)

Causes of Right Sided HF

Cardiac Causes

- Usually occurs as a result of left HF
- Pulmonary stenosis
- Right ventricular infarction

Pulmonary Vascular Disease

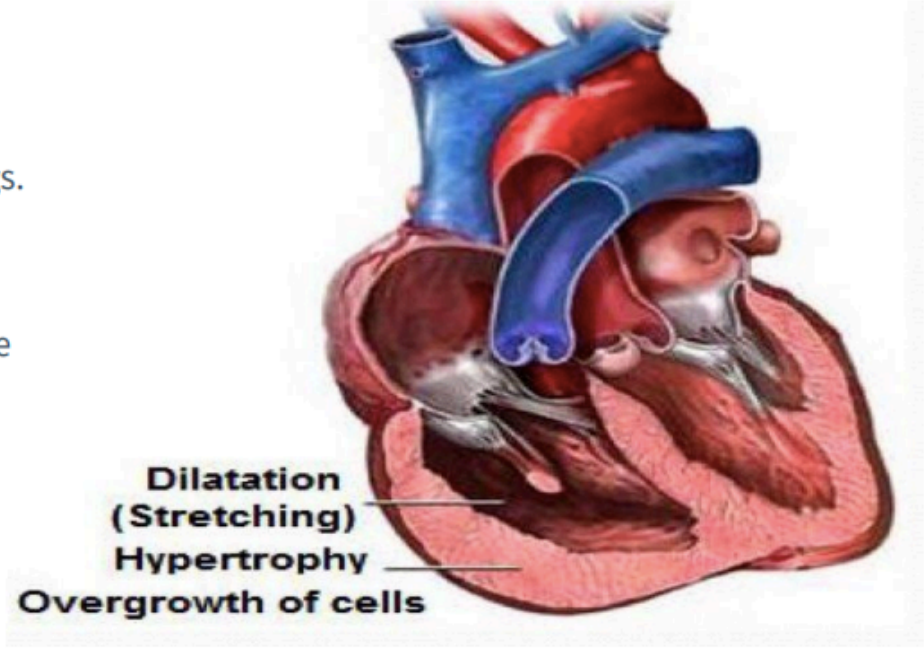
- Pulmonary embolism
- Pulmonary HTN Right ventricular infarction

Pulmonary Parenchymal disease

- COPD
- Interstitial lung disease
- Chronic infections
- Adult respiratory distress syndrome

Can a person have RVF without LVF? (COR PULMONALE)

- Cor pulmonale, or right-sided HF, is an enlargement of the right ventricle.
- It is due to high blood pressure in the lungs.
- It is usually caused by chronic lung disease



Left-Sided CHF (Dogs)

- Tachypnea and dyspnea
- Tachycardia (occasionally bradycardia in some cats and rarely dogs)
- Heart murmurs, gallop sounds, arrhythmias
- Increased respiratory sounds, possibly crackles
- Cough (more reliable in large breed dogs)
- Syncope
- Severe signs: Coughing up pink-tinged sputum

Right-Sided CHF (Dogs)

- Ascites
- Abdominal distension
- Jugular pulsation and distension
- Positive hepatojugular reflex
- Swollen subcutaneous tissue, limb edema in advanced disease
- Focal facial and neck swelling
- Lung and heart sounds may be decreased with pleural effusion

CHF (Cats)

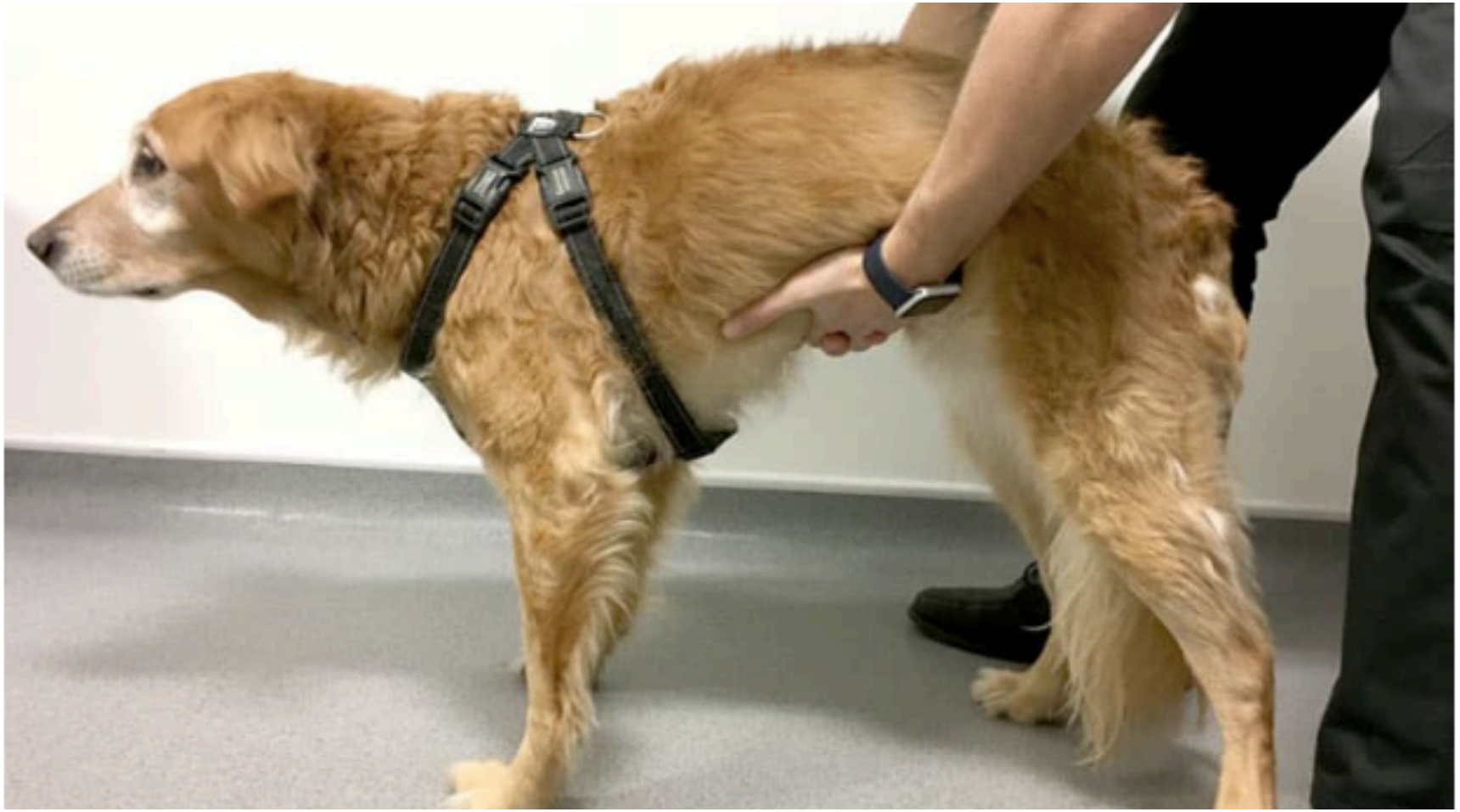
- Bradycardia, hypotension, hypothermia
- Pleural effusion, pericardial effusion
- Cardiac auscultation abnormalities (murmurs, gallop sounds)
- Tachycardia

In cats, right-sided CHF is rare, and ascites alone in a cat is usually not cardiac related. Pleural effusion is the most common presentation for right-sided CHF in cats.

How to interpret the hepatojugular reflux test?

In a normal patient (normal right atrial pressure and no restriction dilation) the pressure created to the caudal vena cava will be accommodated by the right atrium and right atrial appendage with no pressure transmitted to the jugular vein (the jugular vein will not be raised). This means that the patient's ascites is not caused by increased hydrostatic pressures resulting from heart disease/cardiac tamponade and further non-cardiac investigation should therefore be prioritised.

In a patient with markedly increased right atrial pressures, cardiac tamponade or any process compromising right atrial or right auricular appendage dilation (i.e. space occupying mass) the pressure applied to the caudal vena cava will be directly transmitted to the jugular veins causing it to dilate/raise. This means that the patient's ascites is caused by heart disease (increased hydrostatic pressures) and cardiac investigation (echocardiography) should be prioritised.



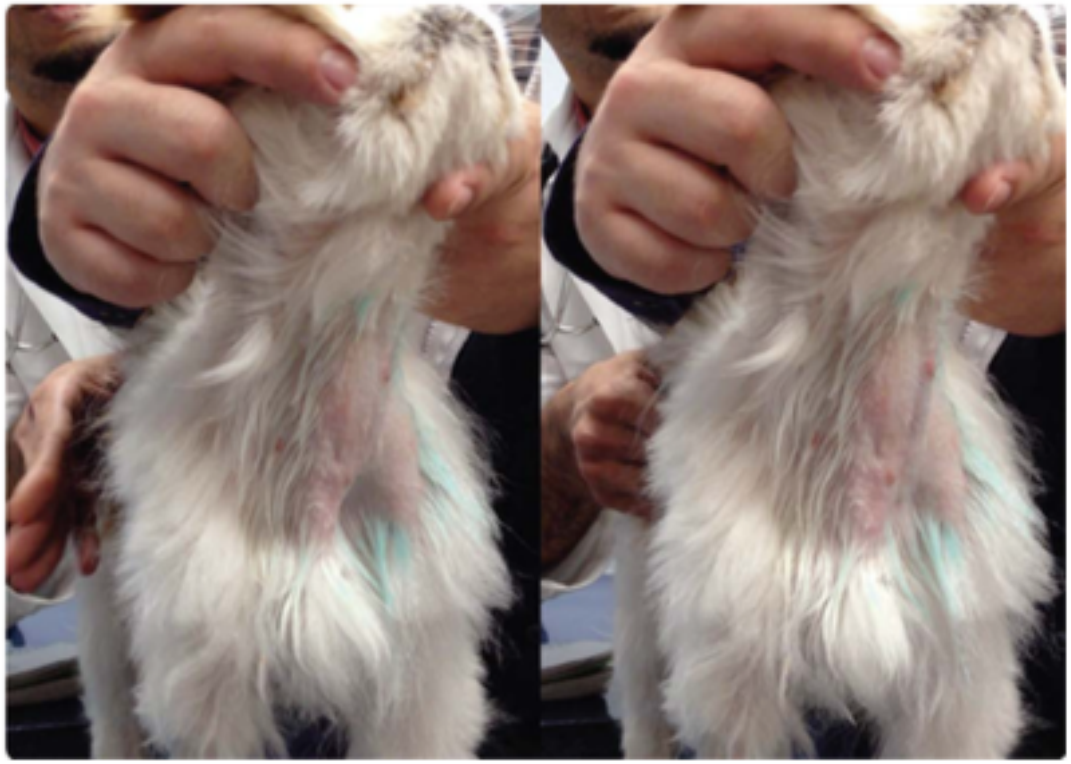


FIGURE 8.2 Hepatojugular reflux sign. There is marked distension of the jugular veins (right) when pressure is applied to the cranial abdomen, compared with baseline (left).

TABLE 8.9 Classification of heart disease stages (based on American College of Veterinary Internal Medicine [ACVIM] consensus statement)

Stage of heart disease	Description
A	Animals at high risk for developing heart disease but currently have no identifiable structural disorder of the heart
B	Animals with structural heart disease (e.g., the typical murmur of mitral valve regurgitation is present) but have never developed clinical signs caused by heart failure – *Stage B1:* Asymptomatic animals that have no radiographic or echocardiographic evidence of cardiac remodeling in response to CVHD MMVD as well as those in which remodeling changes are present but not severe enough to initiate medical therapy (pimobendan) – *Stage B2:* Asymptomatic animals that have hemodynamically significant valve regurgitation, as evidenced by radiographic or echocardiographic findings of left-sided heart enlargement and for whom medical therapy is warranted (pimobendan)

C	Animals with past or current clinical signs of heart failure associated with structural heart disease May or may not require hospitalization and aggressive therapy
D	Animals with end-stage disease with clinical signs of heart failure caused by MMVD that are refractory to standard therapy Such patients require advanced or specialized treatment strategies in order to remain clinically comfortable with their disease As with stage C, some animals in stage D will require acute, hospital-based therapy, and others can be managed as outpatients

CVHD, chronic valvular heart disease; MMVD, myxomatous mitral valve disease.

Classification Systems for Heart Failure Severity

CLASSIFICATION	DEGREE OF SEVERITY
Modified AHA/ACC Heart Failure Staging System	
A	No apparent structural disease, yet considered "at risk" for developing heart disease (for example, breed-associated risk for DCM in Doberman Pinschers, and CMVD in Cavalier King Charles Spaniels)
B	Structural cardiac abnormality is evident (such as a murmur), but no clinical signs of heart failure have occurred
B1	Asymptomatic disease, with no/minimal radiographic or echo evidence of cardiac chamber enlargement/remodeling
B2	Asymptomatic disease, but cardiac chamber enlargement is evident
C	Structural cardiac abnormality evident, with clinical signs of heart failure either in the past (resolved with therapy) or currently present *Note:* Some clinicians subdivide stage C based on current signs of CHF into C1 – No current signs; C2 – mild congestive signs (low/medium grade); C3 – overt/severe CHF (high grade)
D	Persistent or end-stage heart failure signs, refractory to standard therapy (e.g., require ≥ 8-12 mg/kg/day of furosemide)

Modified NYHA Functional Classification	
I	Heart disease is present but no evidence of heart failure or exercise intolerance; cardiomegaly is minimal to absent
II	Heart disease present but clinical signs of failure only with strenuous exercise; radiographic cardiomegaly is usually present
III	Signs of heart failure with normal activity or mild exercise (e.g., cough, orthopnea); radiographic signs of cardiomegaly and pulmonary edema or pleural/abdominal effusion
IV	Severe clinical signs of heart failure at rest or with minimal activity; marked radiographic signs of CHF and cardiomegaly
International Small Animal Cardiac Health Council Functional Classification	
I	Asymptomatic patient
Ia	Signs of heart disease without cardiomegaly
Ib	Signs of heart disease and evidence of compensation (cardiomegaly)
II	Mild to moderate heart failure; clinical signs of failure evident at rest or with mild exercise and adversely affect quality of life
III	Advanced heart failure; clinical signs of CHF are immediately obvious
IIIa	Home care is possible
IIIb	Hospitalization recommended (cardiogenic shock, life-threatening edema, large pleural effusion, refractory ascites)

AHA/ACC, American Heart Association and American College of Cardiology; *CHF*, congestive heart failure.

authors are solely responsible for the content of the statements.

ACVIM consensus statement guidelines for the classification, diagnosis, and management of cardiomyopathies in cats

Virginia Luis Fuentes[1] | Jonathan Abbott[2] | Valérie Chetboul[3] | Etienne Côté[4] | Philip R. Fox[5] | Jens Häggström[6] | Mark D. Kittleson[7] | Karsten Schober[8] | Joshua A. Stern[7]

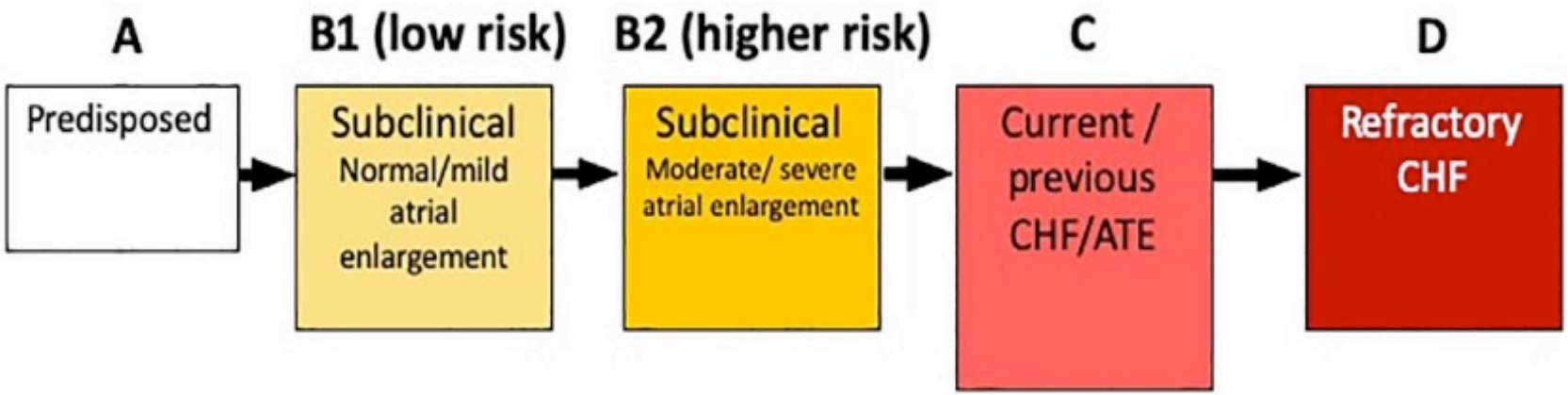


FIGURE 2 Stages of feline cardiomyopathy. Within stage B2, additional risk factors include a gallop sound, arrhythmia, decreased left atrial function, extreme left ventricular hypertrophy, left ventricular systolic dysfunction, spontaneous echo-contrast/thrombus, regional wall motion abnormalities. ATE, arterial thromboembolism; CHF, congestive heart failure

Heart management

TABLE 8.10 Drugs commonly used in treatment of congestive heart failure*

Diuretics	– Furosemide 0.5–4 mg/kg p.o., IM or IV q.8–12h.; usually 0.3–0.5 mg/kg/h CRI (up to 1 mg/kg/h in refractory cases) – Spironolactone 0.5–2 mg/kg p.o. q.12–24h. – Hydrochlorothiazide 0.5–1 mg/kg p.o. q.12–48h. – Torsemide—total daily furosemide dose divided by 10–20 and split into p.o. q.12–24h. dosing (author currently uses total daily furosemide dose divided by 10 and split into q.12h. dosing)
ACE inhibitors	– Benazepril 0.25–0.5 mg/kg p.o. q.24h. – Enalapril 0.25–0.5 mg/kg p.o. q.12–24h.
Positive inotropes	– Pimobendan 0.2–0.4 mg/kg p.o. q.12h. – Digoxin 0.005 mg/kg p.o. q.12h. – Dobutamine 3–10 µg/kg/min IV CRI
Calcium channel blockers	– Amlodipine 0.1–0.5 mg/kg p.o. q.24h. – Diltiazem 0.5–2 mg/kg p.o. q.8–12h. – Diltiazem extended release 2–5 mg/kg p.o. q.12h.
Beta-adrenergic receptor blockers	– Atenolol 0.5–2 mg/kg p.o. q.12–24h. – Propranolol (beta 1 & 2) 0.25–2 mg/kg p.o. q.8h.
Vasodilators	– Nitroprusside 2–5 µg/kg/min CRI – Hydralazine 0.5–3 mg/kg p.o. q.12h.
Nitrates	– Nitroglycerin small dogs: 1/4–1/2 inch topically; medium dogs: 1/2–1 inch; large dogs 1–2 inches q.6–12h. for 24–48 hours (wear gloves) – Isosorbide dinitrate 0.2–2 mg/kg p.o. q.12h.
PDE-5 inhibitor	– Sildenafil 1–3 mg/kg p.o. q.8–12h.

* All doses are for canines unless otherwise indicated. CRI, continuous rate infusion; PDE-5, phosphodiesterase-5.

- **Monitoring and Early Intervention**

Dogs with Stage B1 and B2 heart disease should be monitored every 6-12 months.

Owner Education: Teach recognition of CHF signs.

Resting Respiratory Rate: Daily monitoring advised; a rate above 40 bpm indicates the need for veterinary evaluation.

Pimobendan: Initiated in dogs with valvular disease or dilated cardiomyopathy upon evidence of heart enlargement (Stage B2).

ACE Inhibitors: Use in severe cardiac disease (Stage B2) is controversial.

- # Treatment of Left-Sided CHF in Dogs (stage C)

Furosemide Administration: Always part of treatment; consider IV, SC, or IM routes for moderate-to-severe pulmonary edema.

IV Furosemide: Rapid edema clearance and increased renal blood flow through prostaglandin release.
CRI of Furosemide: **An effective method. (4–8 mg/kg IV) or (0.66–1 mg/kg/hour IV)**

Oxygen Therapy: Recommended alongside furosemide for clinical signs of pulmonary edema.

IV Dobutamine: Considered for positive inotropic support in patients with DCM or advanced mitral valve disease.

Pimobendan: Administered during hospitalization for acute heart failure management.

Afterload Reduction: Consider IV **nitroprusside** for direct vasodilation in refractory cases.

Spironolactone: Considered for further RAAS blockade; **acts as an aldosterone blocker and weak potassium-sparing diuretic.**

Arrhythmia Management: Necessary if present.

- **Managing Chronic, Refractory Left-Sided CHF**

Multinephron Blockade: Use additional diuretics like **spironolactone and hydrochlorothiazide to target different kidney levels.**

Spironolactone: Acts as an aldosterone blocker and potassium-sparing diuretic.

Hydrochlorothiazide: Use with caution in patients with kidney disease or low sodium, as it affects urine dilution and solute-free water excretion.

Torsemide: Considered a potent alternative to furosemide in chronic cases, being 10 to potentially 20 times more potent.

Special Considerations for Severe Pulmonary Hypertension

Sildenafil Administration

- **Treatment of Right-Sided CHF in Dogs:**

Similar to left-sided CHF, though typically less urgent.

Furosemide: Injectable form may be beneficial in cases with ascites for better absorption compared to oral administration.

Fluid Removal: Essential to remove as much fluid as possible from abdominal and/or pleural spaces.

Use of Spironolactone in Right-Sided CHF

Spironolactone: Recommended by some due to potential inhibition of normal aldosterone breakdown by a congested liver in right-sided CHF.

- Atrial Tachyarrhythmias in Right-Sided CHF

- Investigation and Treatment: Essential to address atrial tachyarrhythmias, as loss of atrial contraction can worsen right-sided CHF more than left-sided CHF.

- **Key Differences in Treating CHF in Cats**

Furosemide Dosage: Lower and more conservative compared to dogs.

Hypothermia and Hypotension: Efforts should be made to correct these conditions.

Pimobendan

Hypokalemia: Monitor potassium levels frequently; initiate oral supplementation before signs appear.

Caution with Other Diuretics: Use diuretics other than furosemide and spironolactone with extreme caution.

TABLE 8.11 Treatment of clinical signs of heart failure in the absence of congestive heart failure

Clinical sign	Treatment
Syncope	Rule out CHF with radiographs and consider ruling out an arrhythmia via Holter monitor Start or adjust dosage of ACE inhibitor, spironolactone, or pimobendan or increase current dose of furosemide to alter loading of the heart
Pulmonary hypertension	Sildenafil 1–3 mg/kg p.o. q.8–12h.
Cough	Hydrocodone 0.25 mg/kg p.o. q.6–12h. or butorphanol 0.2–0.4 mg/kg p.o. q.6–12h.

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STANDARD ARTICLE

Journal of Veterinary Internal Medicine
Open Access
ACVIM
American College of Veterinary Internal Medicine

Efficacy of oral torasemide in dogs with degenerative mitral valve disease and new onset congestive heart failure: The CARPODIEM study

Beatrice Besche[1] | Thomas Blondel[1] | Emilie Guillot[1] | Catherine Garelli-Paar[1] | Mark A. Oyama[2]

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Journal of Veterinary Cardiology
Volume 37, October 2021, Pages 52-61

Effect of a single dose of pimobendan on right ventricular and right atrial function in 11 healthy cats ☆

L.V. Kost DVM [a], T.M. Glaus DVM, PhD [a], A. Diana DVM, PhD [b], M. Baron Toaldo DVM, PhD [a b]

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DOI: 10.1111/jvim.16187

STANDARD ARTICLE

Journal of Veterinary Internal Medicine Open Access

ACVIM American College of Veterinary Internal Medicine

Clinical relevance of serum electrolytes in dogs and cats with acute heart failure: A retrospective study

Marine Roche-Catholy[1] | Iris Van Cappellen[1] | Laurent Locquet[1] | Bart J. G. Broeckx[2] | Dominique Paepe[1] | Pascale Smets[1]

- A significant and moderate negative correlation was identified between serum Cl concentrations and furosemide doses both at discharge and at end-stage disease in dogs.

- This finding advocates for assessment of serum Cl concentration in dogs with acuteCHF, and suggests that, even during a first episode of CHF, this variable might help identify dogs who could require larger doses of diuretics, illustrating the potential of Cl as a marker for disease severity and response to diuretic therapy. Several large clinical studies have established this relationship in people, and have advanced that **hypochloremia could serve as a marker of impaired diuretic responsein HF patients**