THE UNIVERSITY OF MELBOURNE

Melbourne Veterinary School

Case Study 3

Jack’s pumping, but he’s not jumping

10th August 2023

VETS30014 / VETS90124

Learning Objectives

- Explain the physiological basis of the ventricular function curve and relate the ventricular function curve to Jack's cardiac function.
- Explain the pressure changes that occur in the heart and major vessels when the force of ventricular contraction is reduced
- Identify the cardiac shadow, major vessels and lung fields in normal lateral and ventrodorsal radiographs. Understand the parameters that are used to assess cardiac size and shape, and apply these to examination of radiographs supplied
- Describe the modes of echocardiography that are employed to assess cardiac function
- Predict the consequence of ventricular tachycardia (with VPC's) for cardiac output
- Postulate some mechanisms by which Jack's cardiac output might be improved with drugs.

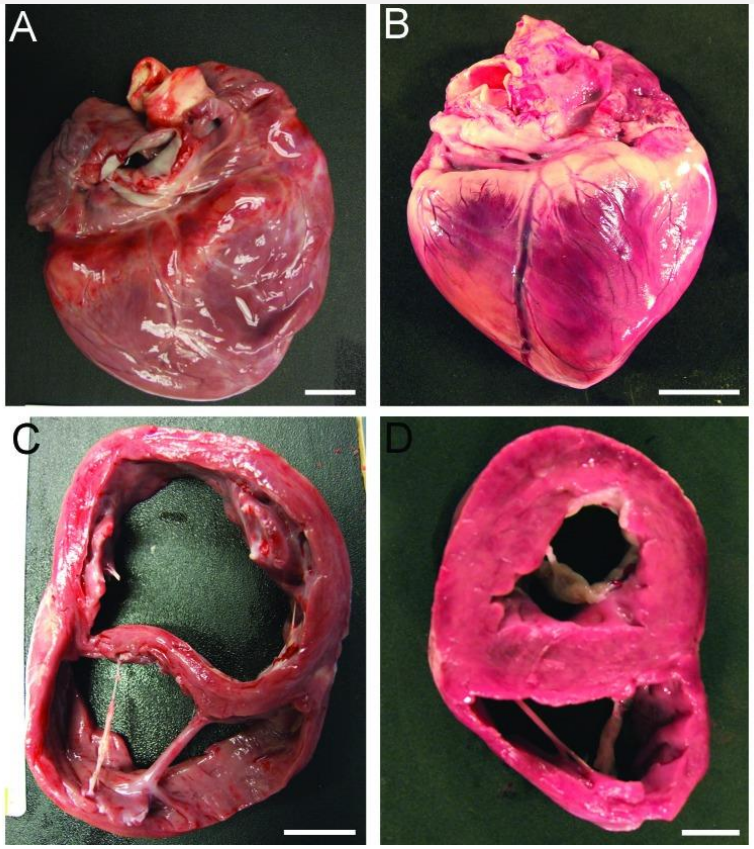
In physiological terms, what is Jack's primary problem?

Decreased Contractility

- Jack is suffering from Dilated CardioMyopathy (DCM)
- The heart is dilated, and the heart muscle is weak

  (myo= muscle; pathy= disease) systolic failure and ventricular dilation

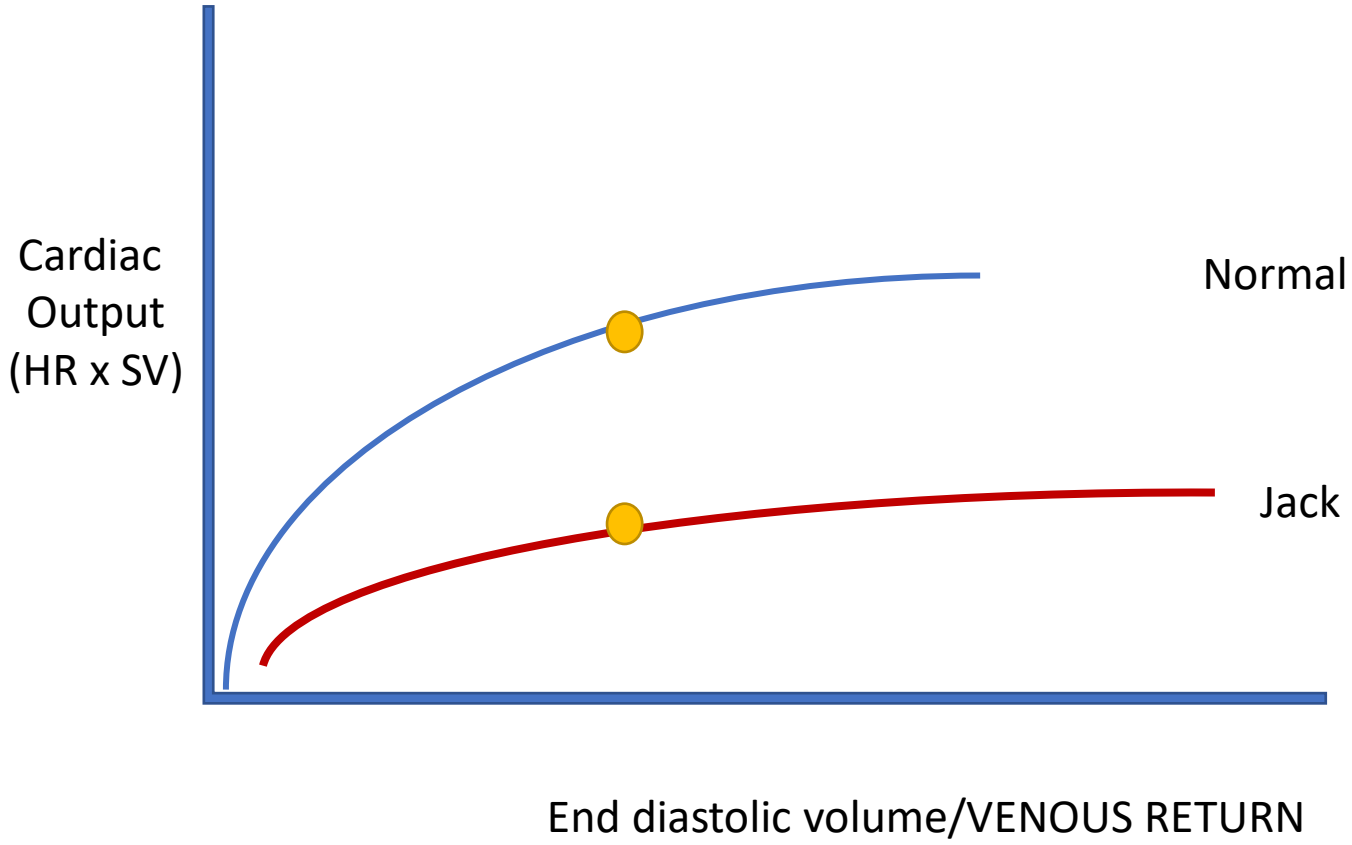
- ‘DCM Phenotype’



https://europepmc.org/article/pmc/4549678

Contractility… the amount of tension that can be developed at any given length of cardiac myocyte

How does Jack's condition change the ventricular function curve?



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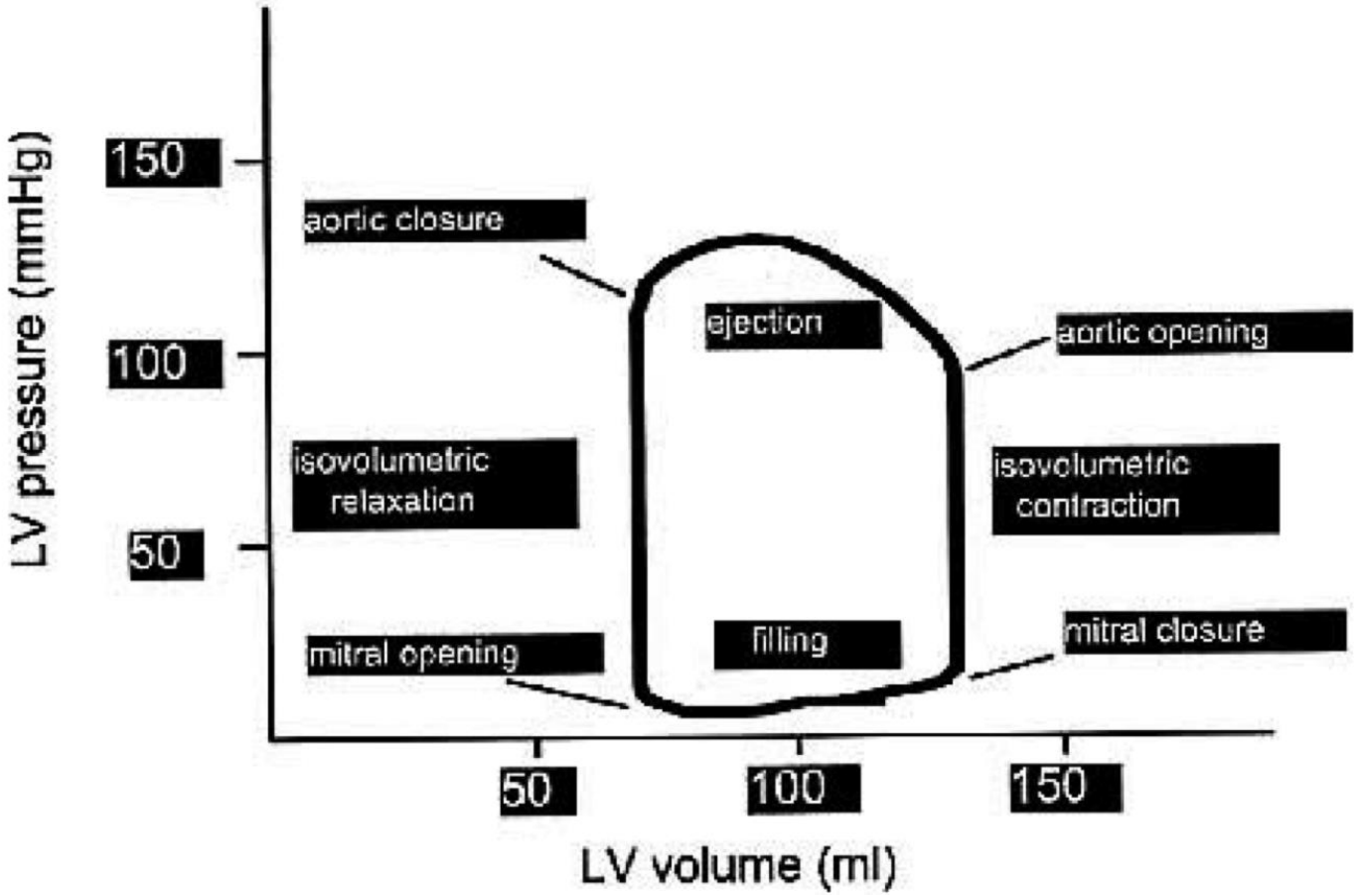

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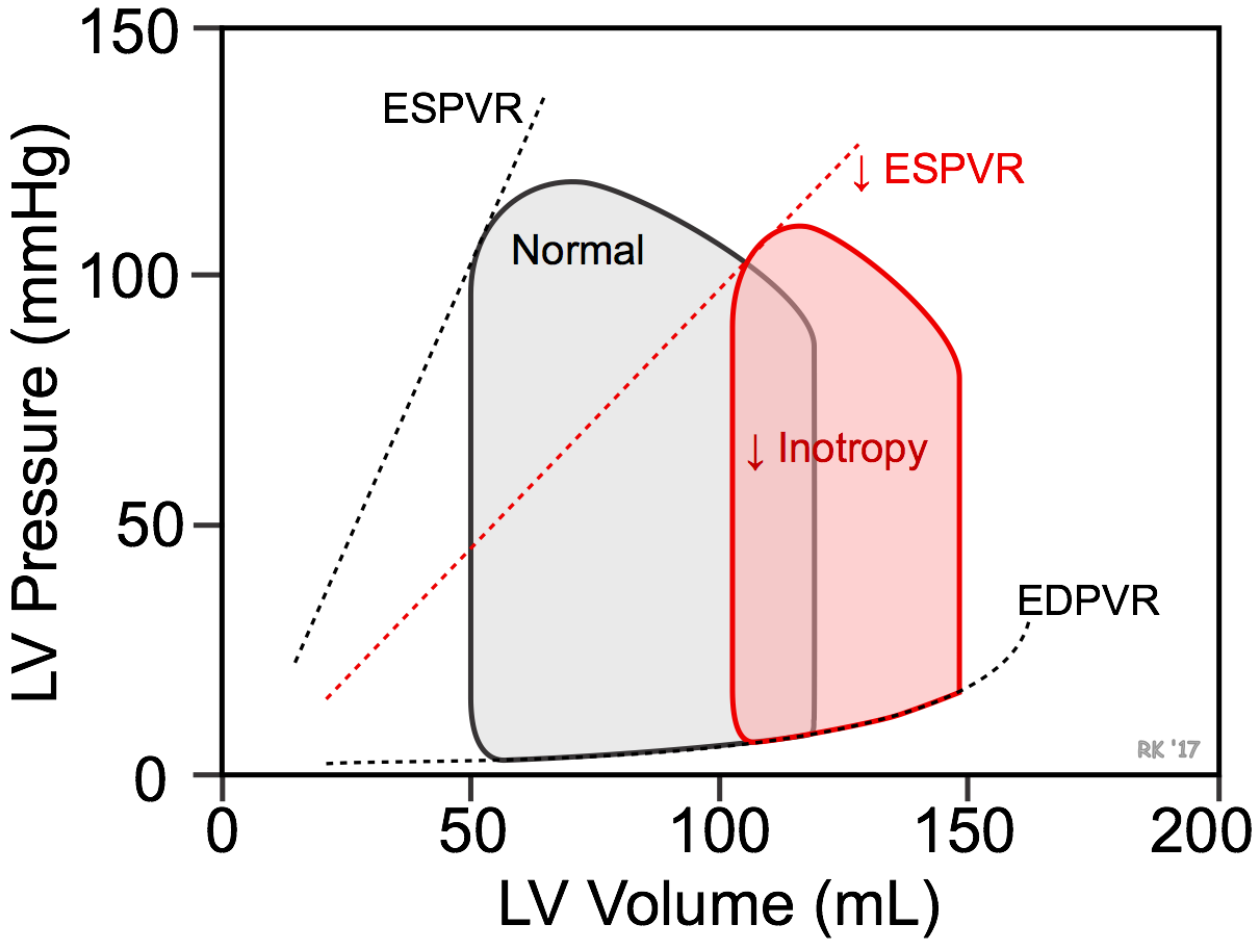
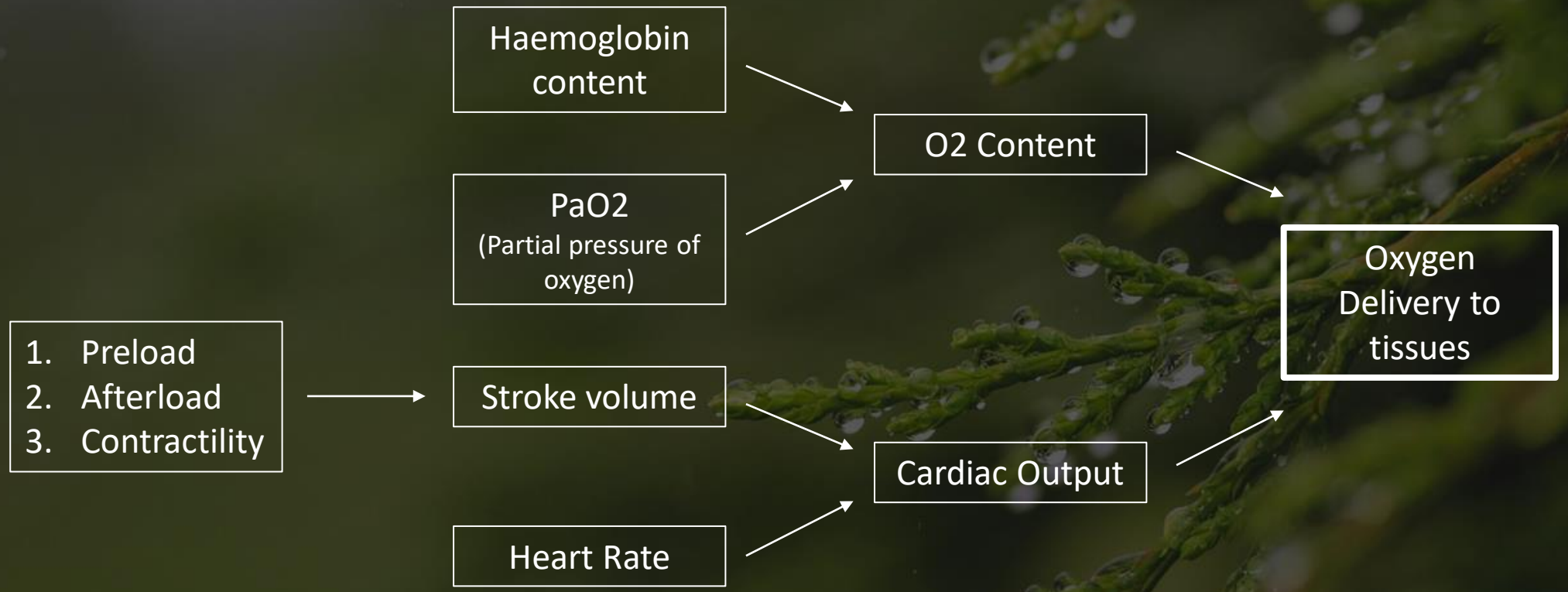


https://www.cvphysiology.com/Heart%20Failure/HF005

“lethargic and breathing heavily”..

Can we now explain these signs?

Tree of life



DCM → decreased contractility (systolic function) → decreased stroke volume→ decreased cardiac output

→ insufficient CO to match tissue oxygen requirements→ **lethargy and poor exercise tolerance**

“lethargic and breathing heavily”..

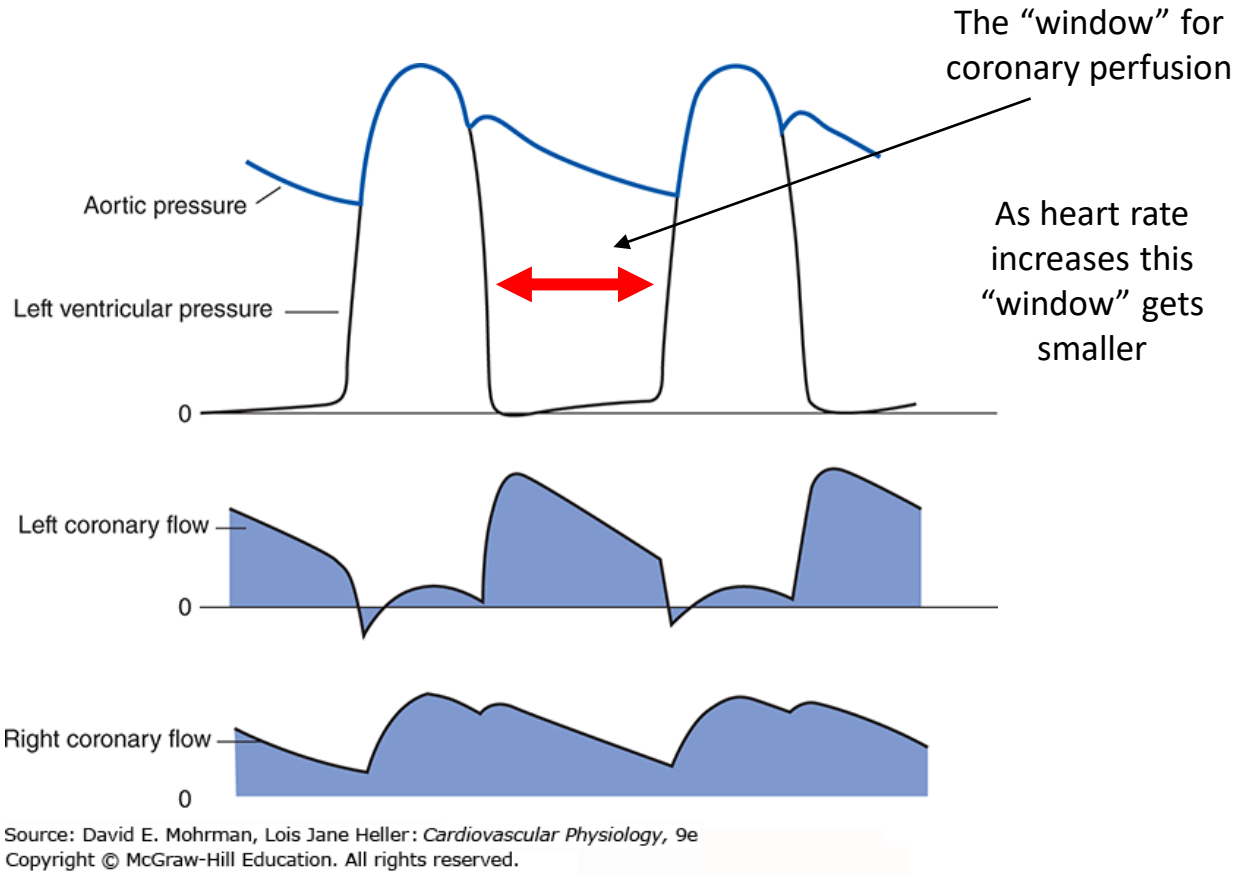
Can we now explain these signs?

DCM→ increasing end diastolic length→ stroke volume ≠ venous return→ increased pulmonary venous pressure→ pulmonary venous congestion → pulmonary oedema→ increased respiratory effort

What other factors could be contributing to poor cardiac output in Jack?

What other factors could be contributing to poor cardiac output in Jack?

- Poor contractility
  - Intrinsic due to DCM
  - low coronary perfusion and low arterial oxygen saturation
- Incompetent mitral valve, so lower ejection fraction
- Irregular filling time and decreased filling time



Source: David E. Mohrman, Lois Jane Heller: *Cardiovascular Physiology*, 9e
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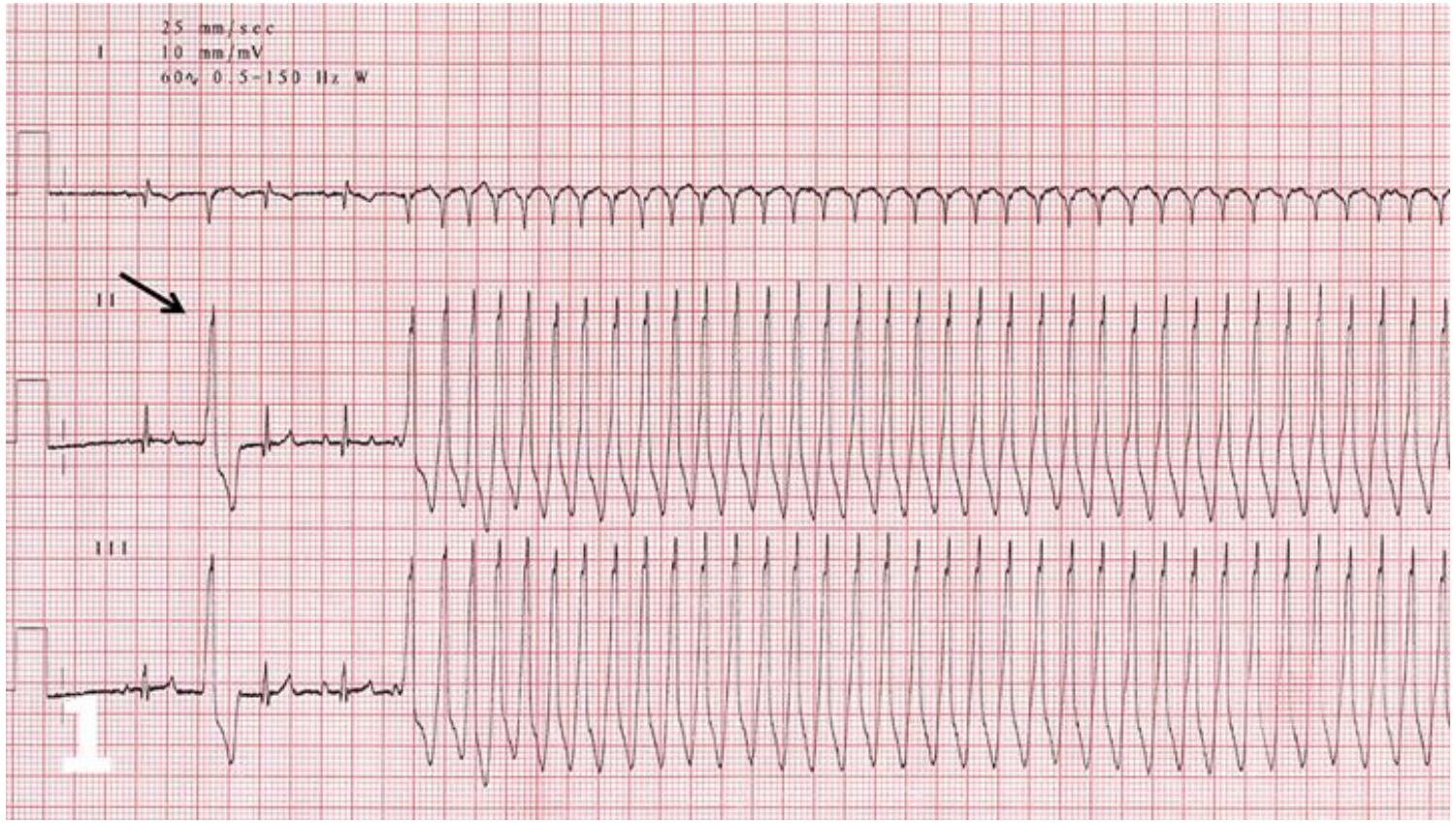
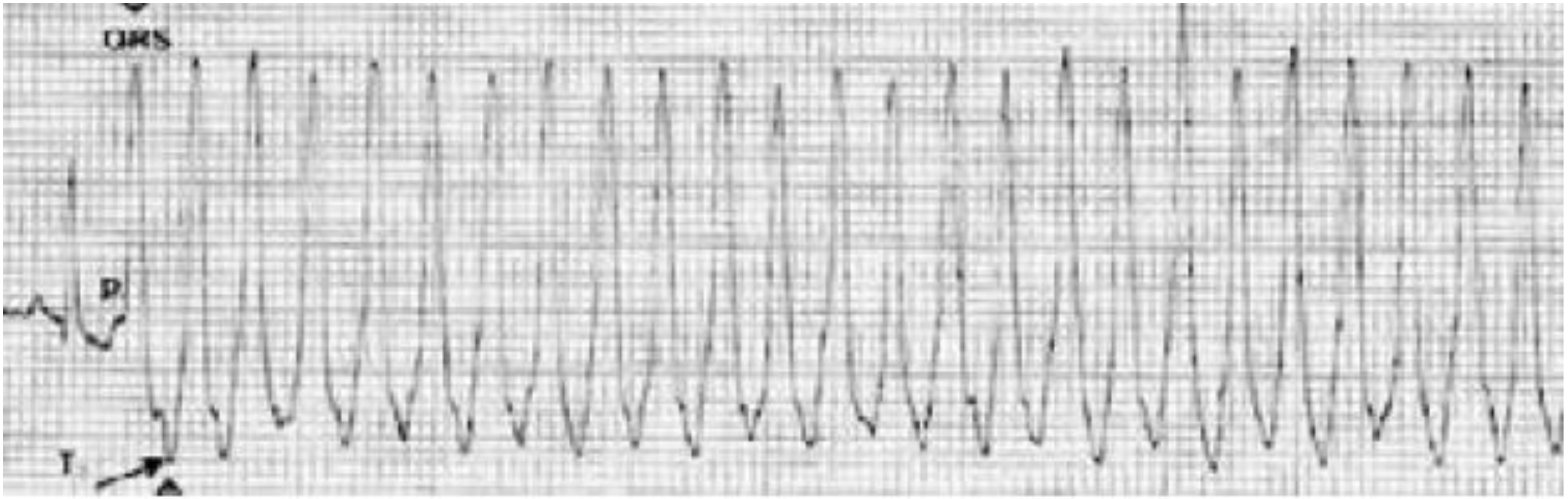
What about the arrythmia?
ECG Interpretation



https://www.cliniciansbrief.com/article/ventricular-tachycardia

- Can you identify the R wave?
- Is the R-R interval constant?
- Can you identify a P wave?
- Is every P wave related to a QRS complex?
- Does the form of the QRS complex appear normal?



“rapid heart rate with highly variable rhythm”

What is ventricular tachycardia?

What is a tachycardia?
"tachy"= rapid

What is a ventricular tachycardia?

→ a succession of ventricular premature complexes (VPC's)

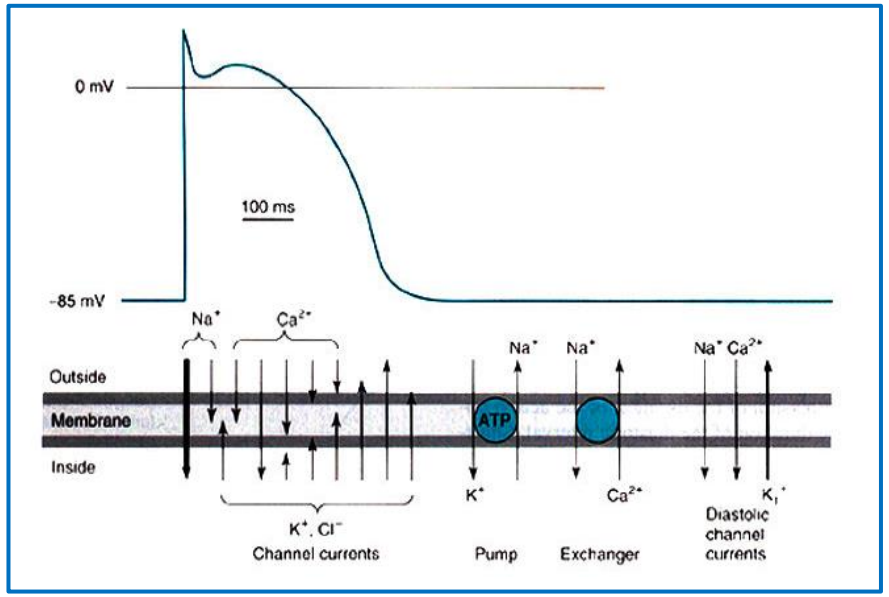
Why do these occur?

Because of the activity of a ventricular ectopic pacemaker

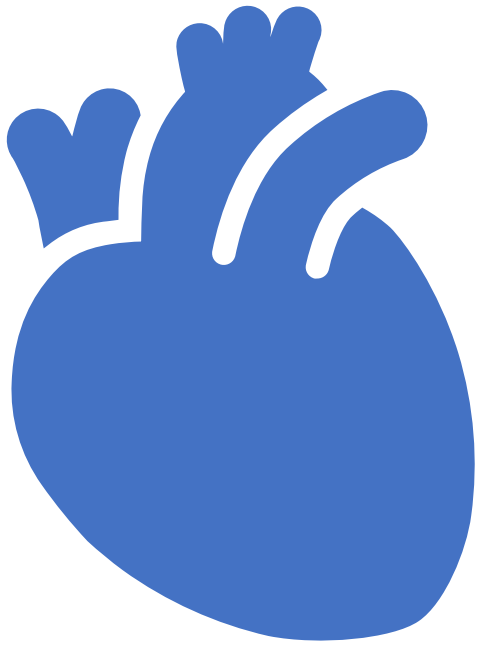
Why do ectopic pacemakers appear?

What does it take to maintain a stable resting membrane potential?

- Adequate perfusion - how is this impacted in DCM?
- Adequate oxygenation and nutrient supply
- Appropriate generation of ATP
- Where is the ATP used?
- Active transport of ions:
  - Na/K ATPase
  - Ca uptake into sarcoplasmic reticulum
  - Ca ATPase



To restore and maintain
Stable resting membrane potential

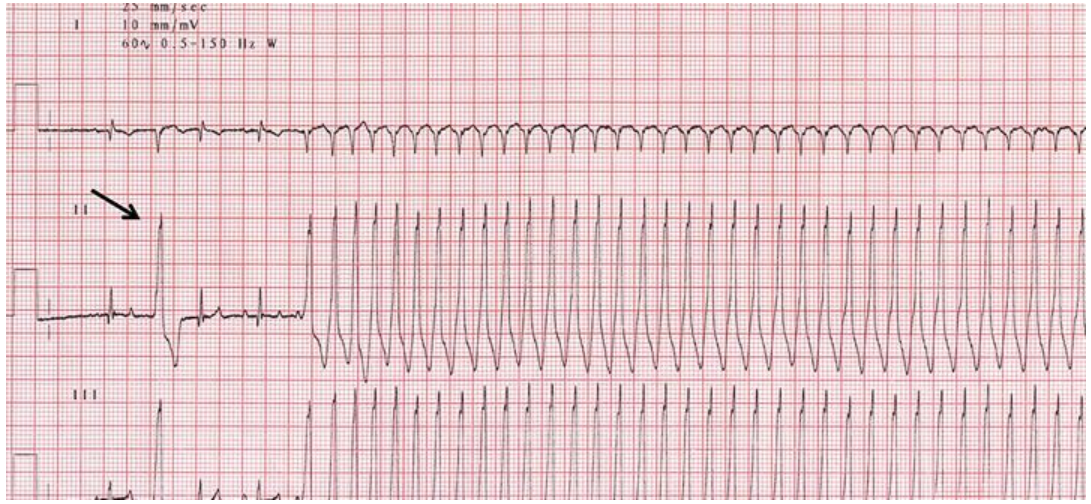


What is the aetiology of dilated cardiomyopathy phenotype?

- Cardiomyopathy can be the result of several different factors
  - Eg viral infection, antineoplastic drug therapy, taurine deficiency
- Naturally occurring DCM in both humans and dogs shows strong familial tendencies
- Underlying genetic and intracellular mechanisms are still poorly defined, but include:
  - Abnormal mitochondrial activity → impaired energy generation
  - Altered intracellular calcium handling → increased risk of arrhythmias

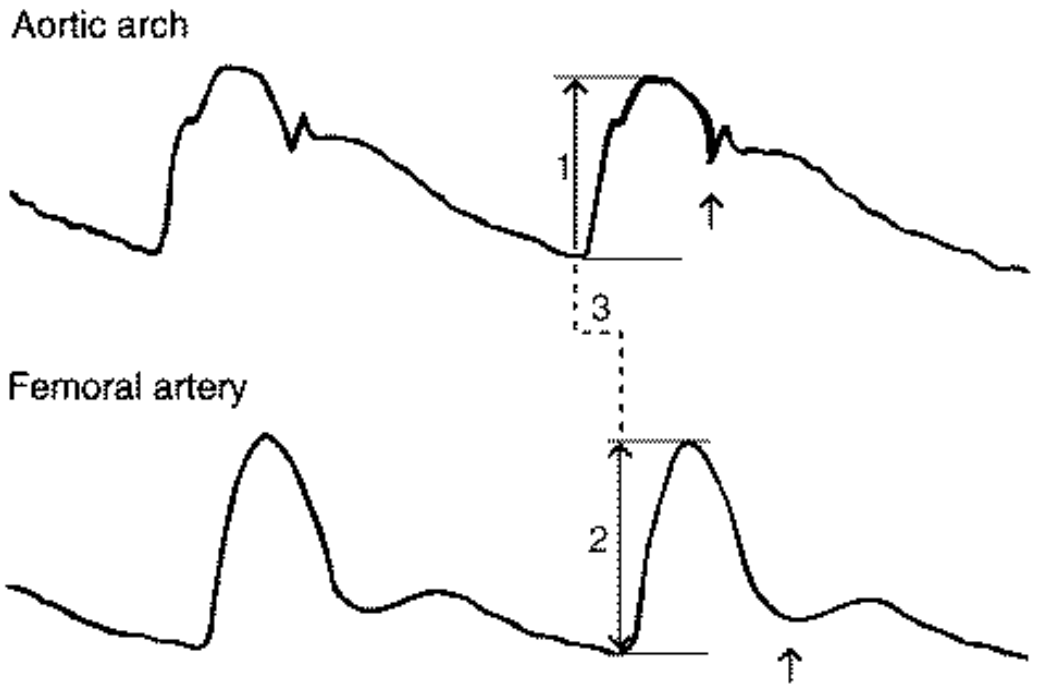
Why is the heart rhythm irregular?

- “Random” activity of ectopic pacemaker →
- Irregular conduction pattern across ventricles that is dependent on refractory/ resting state of cardiac myocytes
- Conduction/depolarization of ventricles not following normal path →
- Abnormal wave forms on ECG (VPC’s)



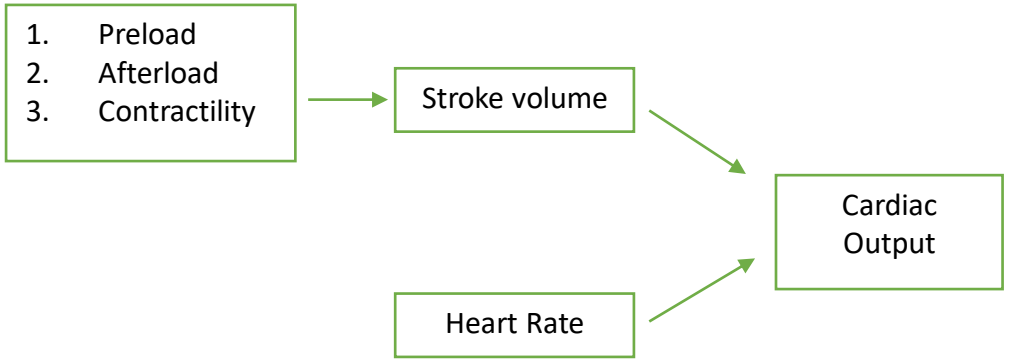
“weak pulse and pulse deficits”

What determines pulse strength?



- The strength of the pulse we feel depends on the AMPLITUDE of the pulse
- Pulse amplitude depends on STROKE VOLUME
- So, what has happened to Jack's stroke volume?

The determinants of stroke volume...

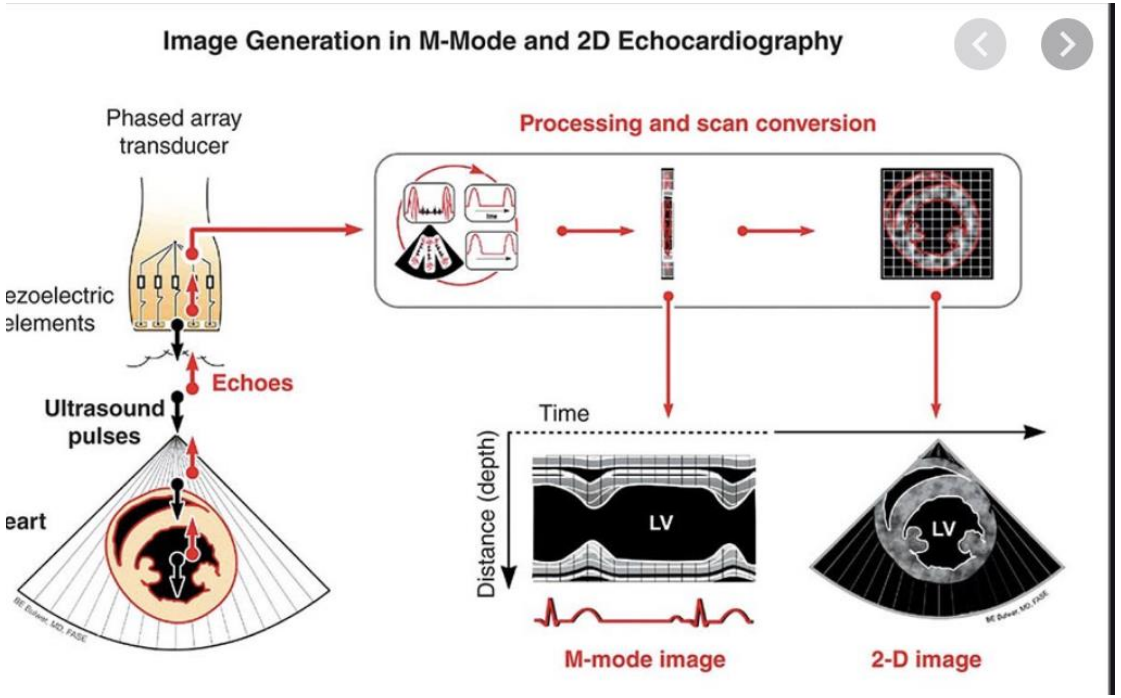


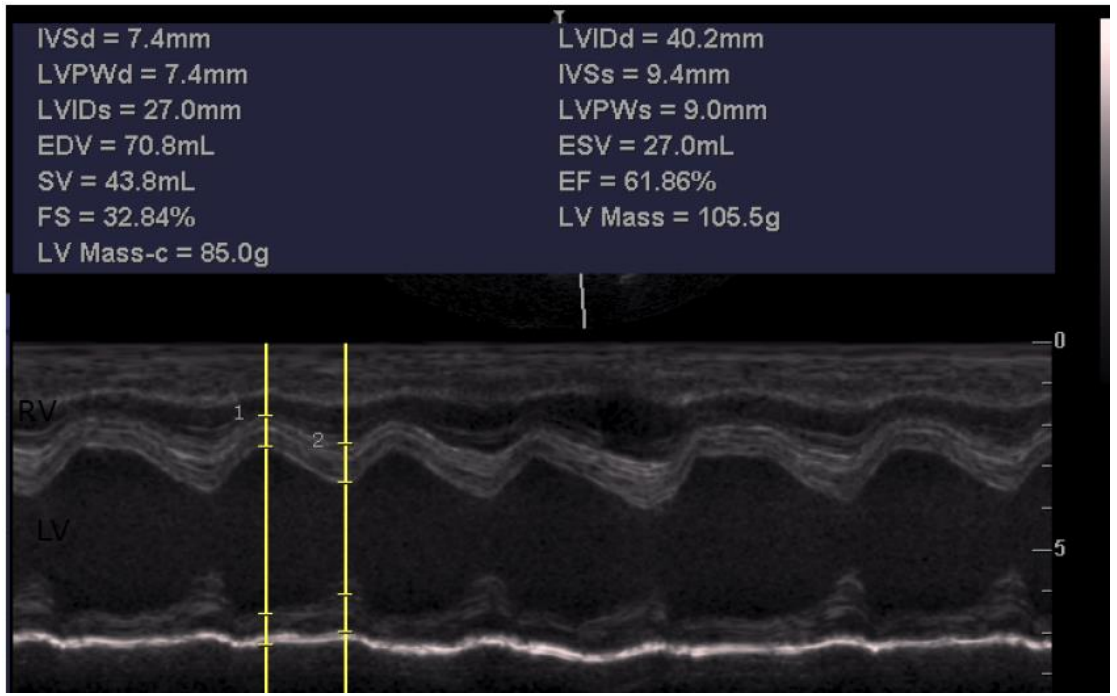
In order to have appropriate cardiac output we must have

- Adequate filling time (Jack has a very rapid heart rate so reduced filling time)
- Adequate venous return (not a problem for Jack)
- Adequate contractility (Jack's DCM results in him having reduced contractility)

What is a pulse deficit?

- A heartbeat occurs but is not accompanied by a palpable arterial pulse
- This is different from saying ”absence of a pulse”… (which would be inconsistent with life…)
- Pulse deficit occurs when ventricular filling time is shortened such that stroke volume is insufficient to generate a palpable pulse





How do we assess contractility?

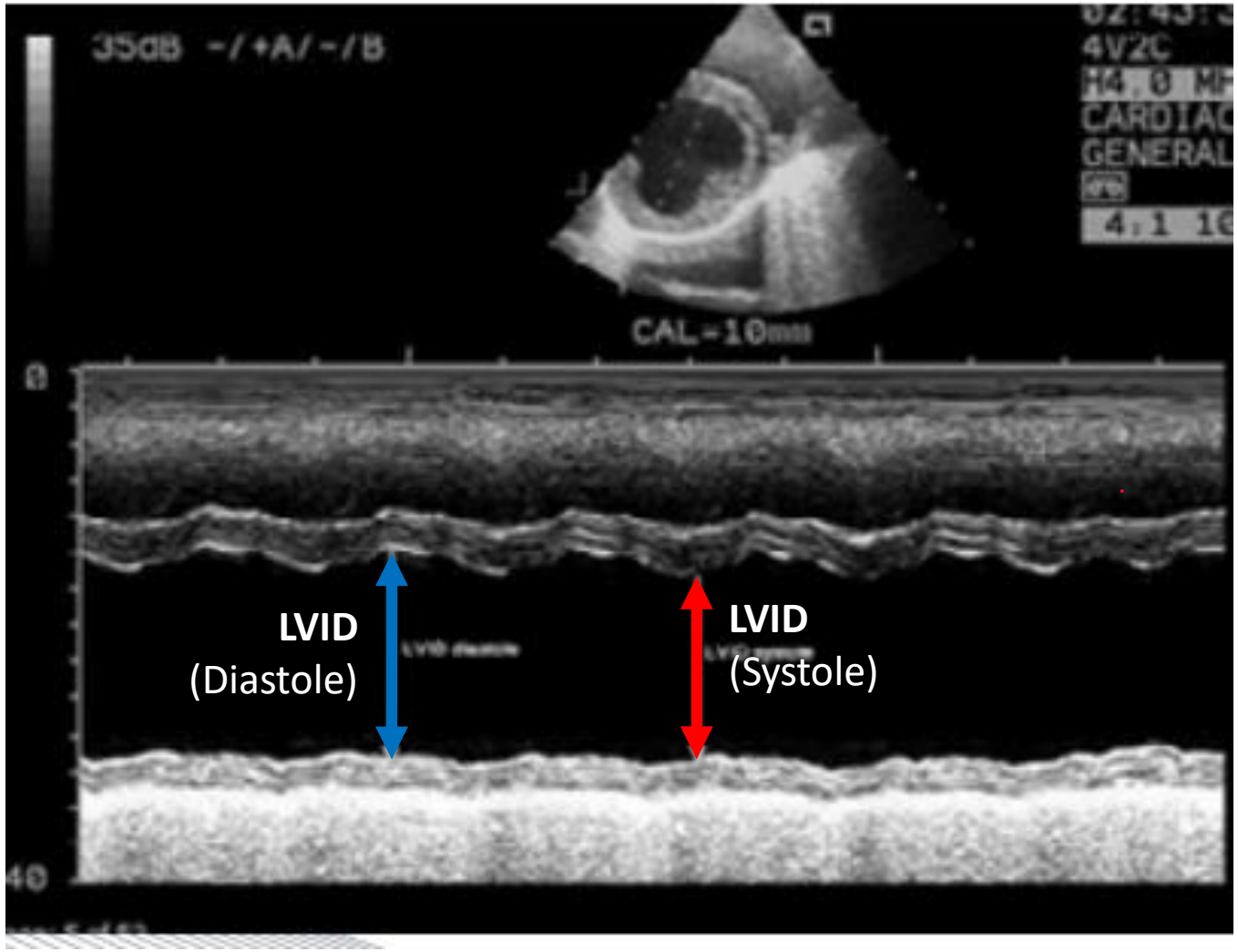
M mode echocardiography and fractional shortening

Measuring Fractional shortening

Fractional Shortening=

$$\frac{\text{Left ventricular internal diameter in diastole- Left ventricular internal diameter in Systole}}{\text{Left Ventricular internal diameter in diastole}}$$

Jack's Fractional Shortening



$$\mathbf{FS} = \frac{\mathbf{LVID}\ (\text{Diastole}) - \mathbf{LVID}\ (\text{Systole})}{\mathbf{LVID}\ (\text{Diastole})}$$

$$FS = \frac{4.6 - 3.9}{4.6} = \mathbf{15\%}$$

What is normal FS?

28%- 42%

Jack’s murmur

What has caused the grade 2/6 systolic murmur?

- Annular dilation (secondary/functional regurgitation)

What could we do to improve Jack's cardiac output?

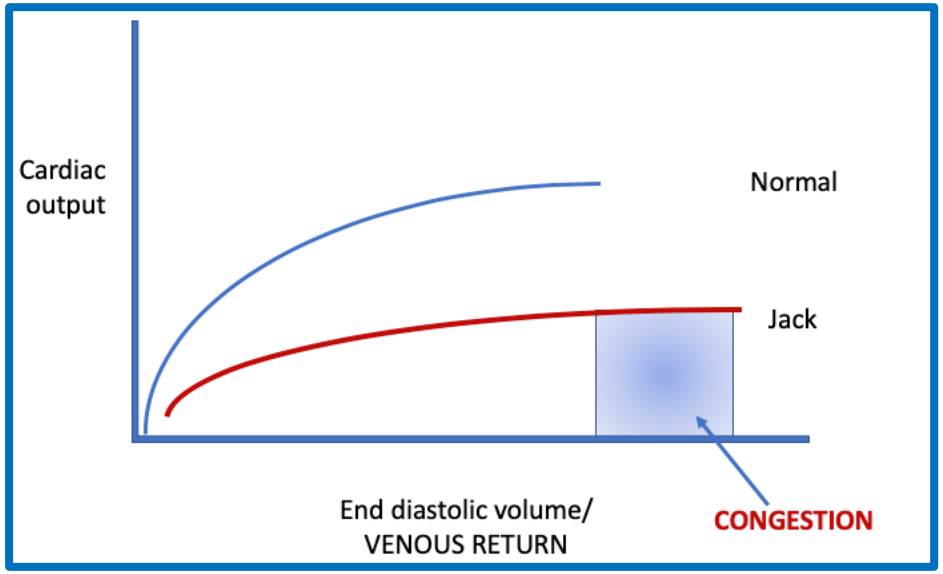
ie how can we support a failing heart?

What could we do to improve Jack's cardiac output?

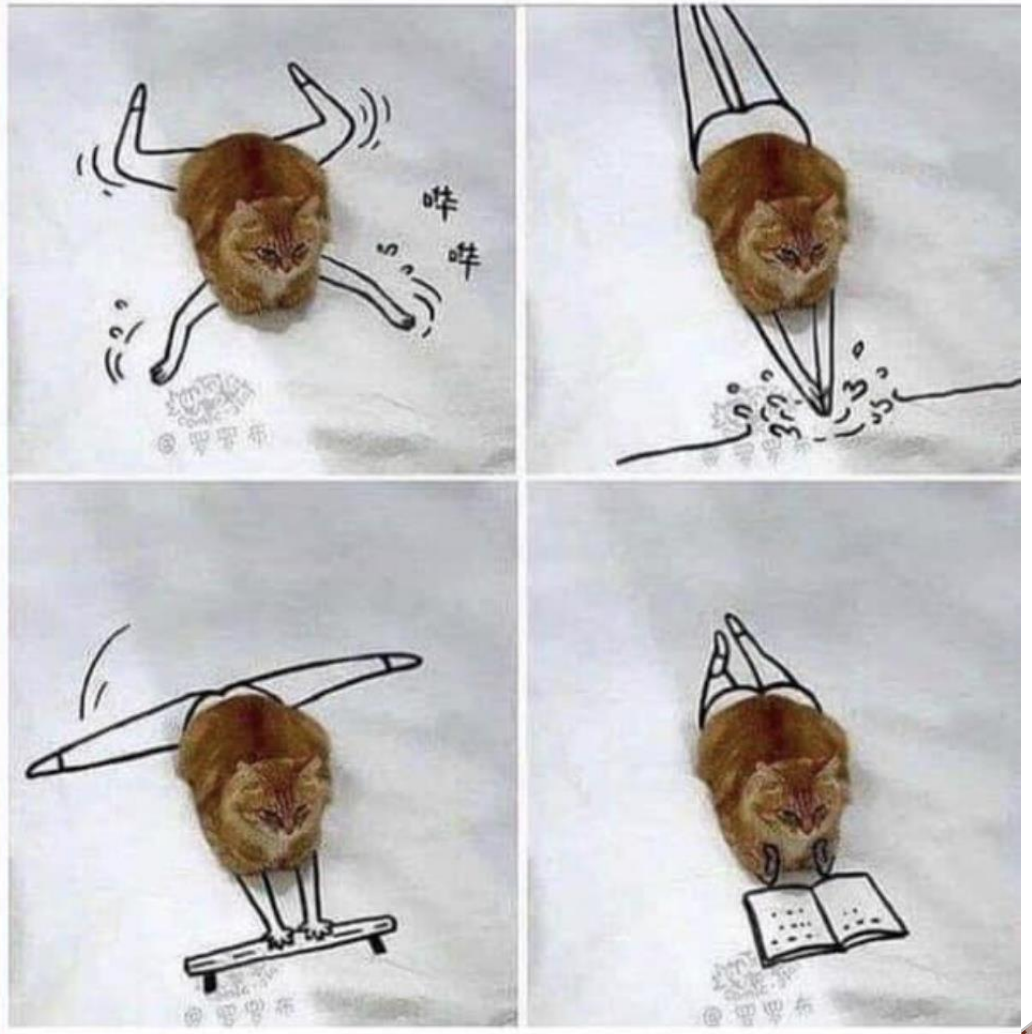
- Contractility
- Length tension relationship
- Ejection fraction
- Increase filling time

What could we do to improve Jack's cardiac output?

- Contractility. Positive INOTROPE
- Length tension relationship. Reduce PRELOAD
- Ejection fraction. Reduce AFTERLOAD
- Increase filling time. Antiarrythmics



Trying to make your cat look active



Case Study Quiz this week

- Available on canvas now
- Due 11.59pm next Tuesday – don’t forget!

Cardiovascular Clinical Exam Prac: tomorrow

- Group allocations available on canvas
- If you need to change groups; find someone to swap with
- Do the preparatory activities for the Stations you will be completing tomorrow
- Bring a stethoscope and a watch (with a second hand!) if you have one
- Sign-in will be available at equine and in the LTB

Cardiovascular: Intra-semester test

- Monday 21st August 9am: 60 minutes, online, open book
- Content from Weeks 1-4: lectures, case studies, prac classes
- Section A: MCQ questions (30)
- Section B: Short answer questions (20 marks)