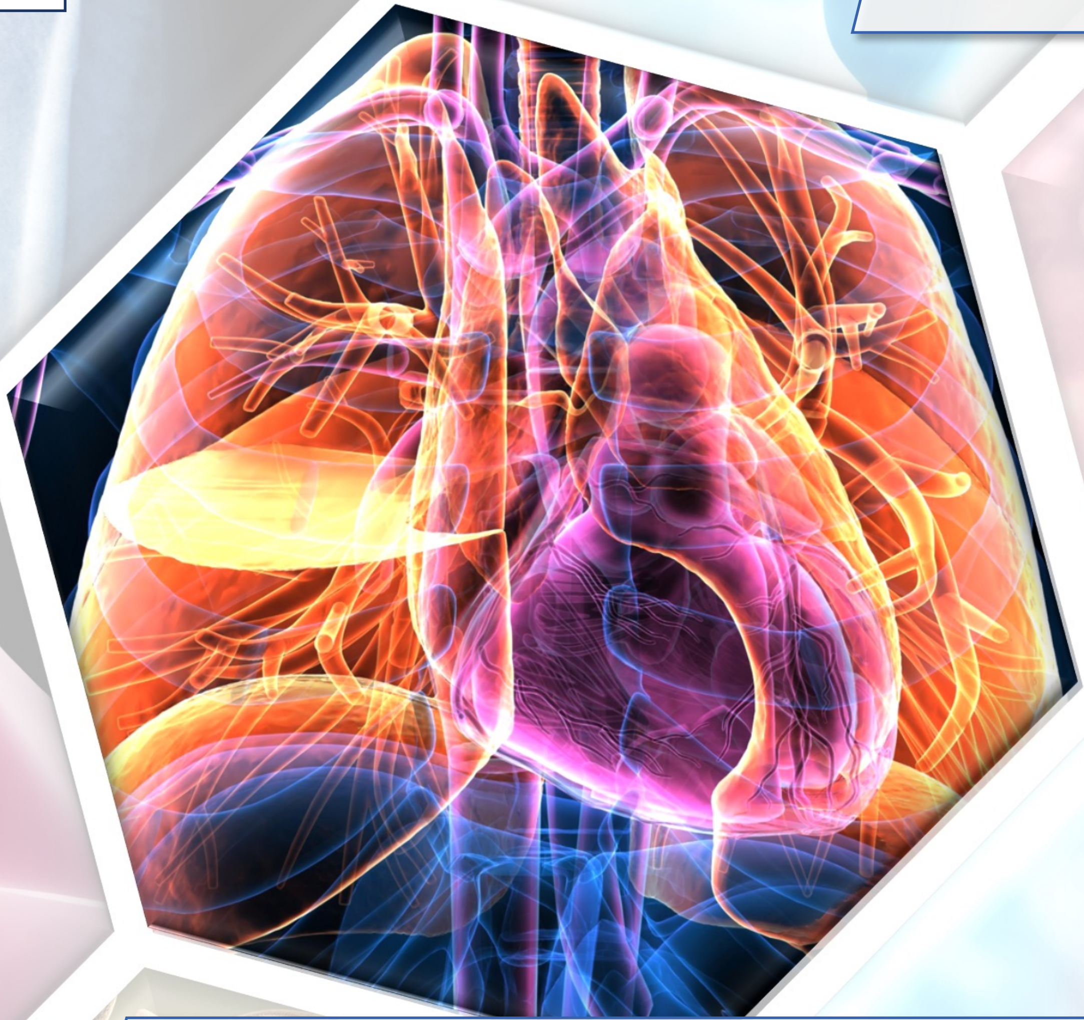
IMPERIAL

The Cardiac Cycle

20th January 2025



Dr. Alicia D’Souza a.dsouza@imperial.ac.uk

Session Plan

The cardiac cycle

- **Systole** (contraction) and **diastole** (relaxation)
- **Seven distinct phases** can be identified
- **Pressure changes** in the atria, ventricles and outflow arteries govern valve movement
- Valve closure and rebound pressure produces healthy heart sounds **S1** and **S2**

Preload & afterload

- Preload (stretch) determined **by volume of blood returning to the heart**
- Afterload (pressure the heart has to work against) determined **by diastolic blood pressure**
- Changes in preload and afterload affect the shape of the PV loop

Pressure volume loops

- **Graphical representations** of ventricular pressures and volumes as they change during the cardiac cycle

Extrinsic stimulation

- Increased **sympathetic stimulation increases cardiac myocyte [cAMP]** and allows the **delivery of more Ca^{2+} to myofilaments**. Activation of symp. beta receptors by:
  - **Circulating catecholamines** from adrenal gland
  - **Noradrenaline released** from nerves

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The cardiac cycle

Each heart beat is split into **two main phases**

Diastole

Lasts approximately 2/3 of each beat

Ventricular relaxation
The ventricles fill with blood

Split into **4 distinct phases**

Systole

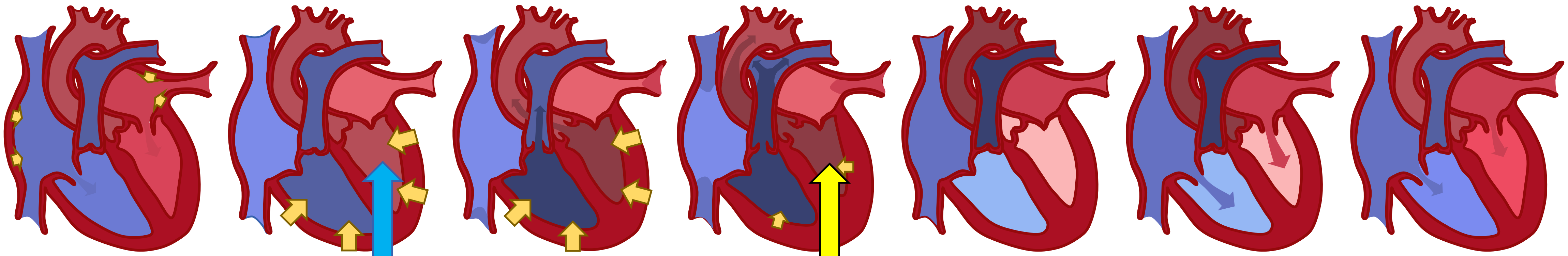
Lasts approximately 1/3 of each beat

Ventricular contraction
Ventricles generate pressure then eject blood into the arteries

Split into **3 distinct phases**

The cardiac cycle

	SYSTOLE			DIASTOLE		



Atrial systole	Isovolumetric contraction	Rapid ejection	Slow ejection	Isovolumetric relaxation	Rapid passive filling	Slow passive filling

End-diastolic volume − **End-systolic volume** = **Stroke volume (mL)**

120 mL − **50 mL** = **70 mL**

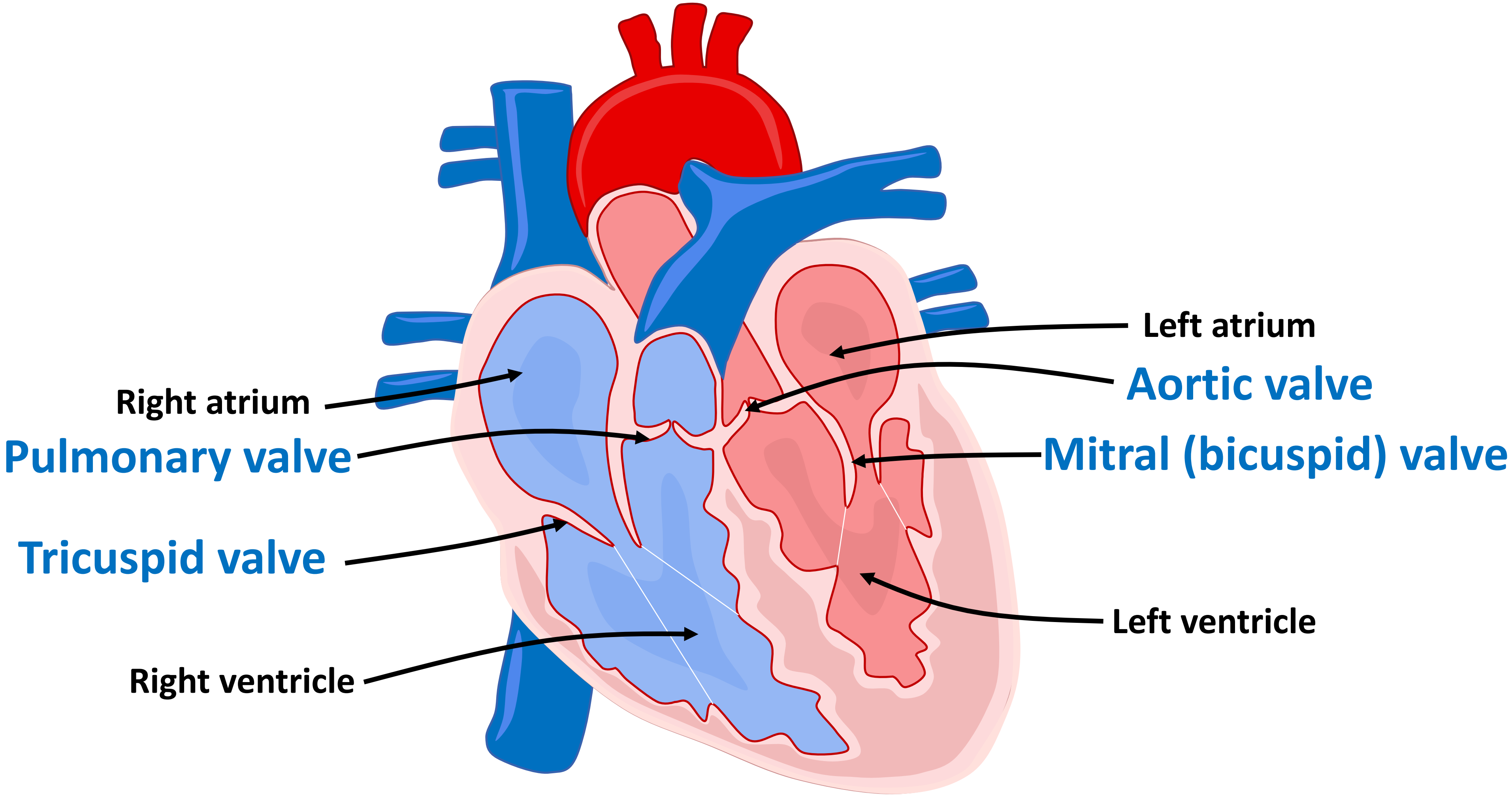
100 x **Stroke volume** ÷ **End-diastolic volume** = **Ejection fraction (%)**

70 mL ÷ **120 mL** = **58%**

Normal range 52-72%

Heart and lung mechanics: Explain the mechanical factors associated with heart and lung function.

Cardiac valve



Heart and lung mechanics: Explain the mechanical factors associated with heart and lung function.

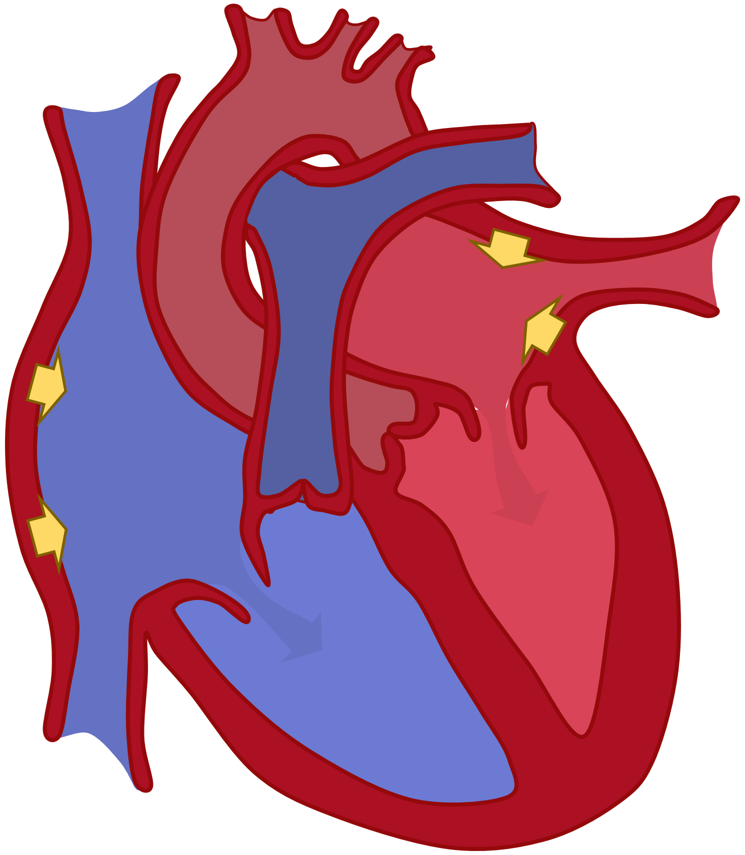
Cardiac cycle: **Atrial systole**

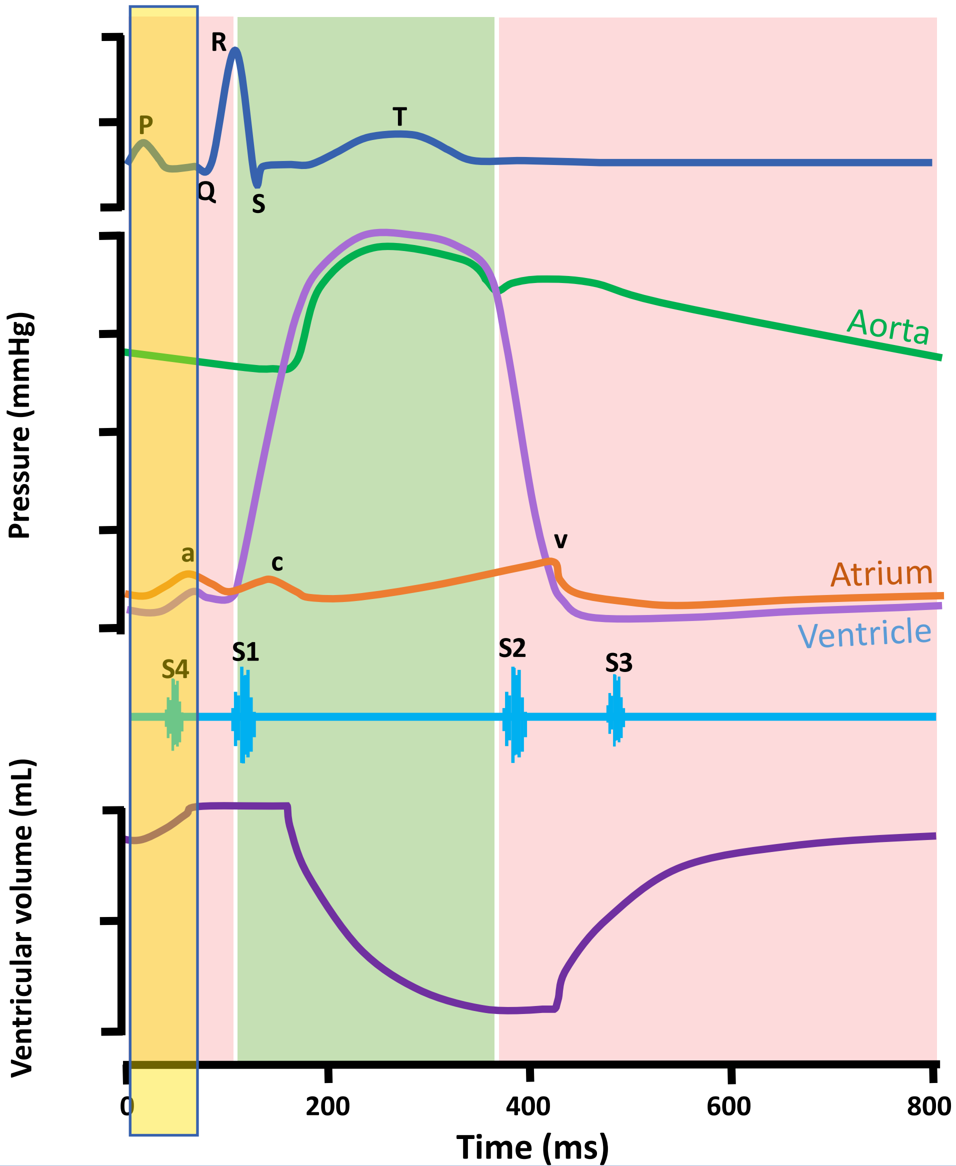
Atrial systole

P-wave on ECG signifies start of atrial systole

Atria **already almost full** from passive filling driven by pressure gradient. Atria contract to **'top-up' the volume of blood in ventricle**

4th heart sound – **abnormal**, occurs with congestive heart failure, pulmonary embolism or tricuspid incompetence





Heart and lung mechanics: Explain the mechanical factors associated with heart and lung function.

Cardiac cycle: **Isovolumetric contraction**

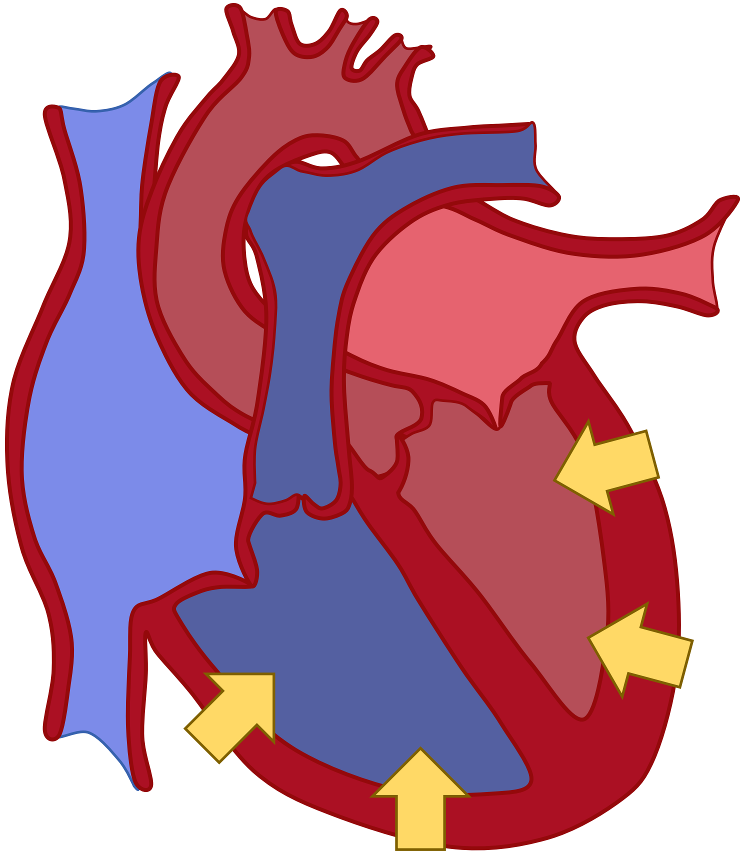
Isovolumetric contraction

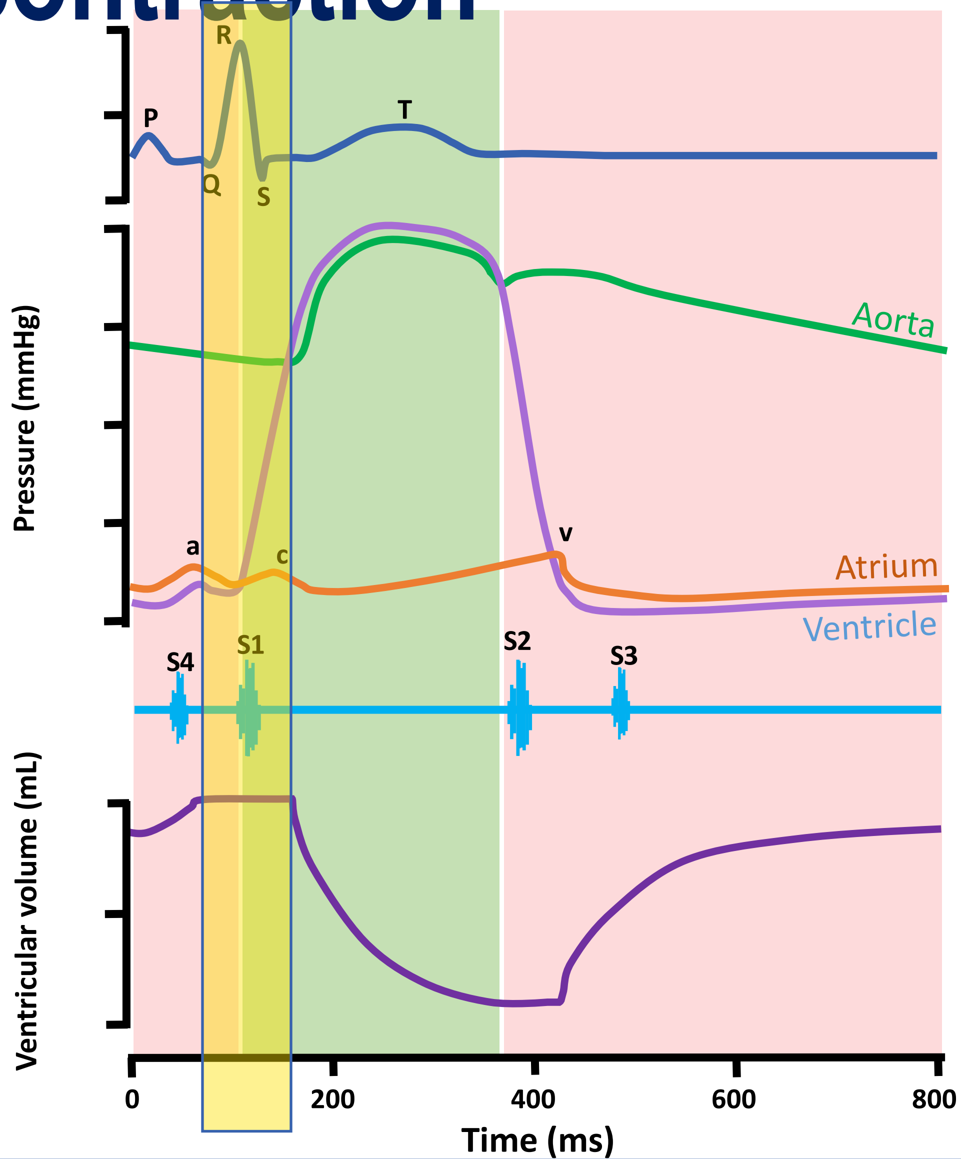
QRS complex marks the **start of ventricular depolarisation**

This is the interval between AV valves (tricuspid & mitral) closing and semi-lunar valves (pulmonary & aortic) opening

Contraction of ventricles with **no change in volume (isometric)**

1st heart sound (‘**lub**’) due to **closure of AV valves** and associated vibrations





Heart and lung mechanics: Explain the mechanical factors associated with heart and lung function.

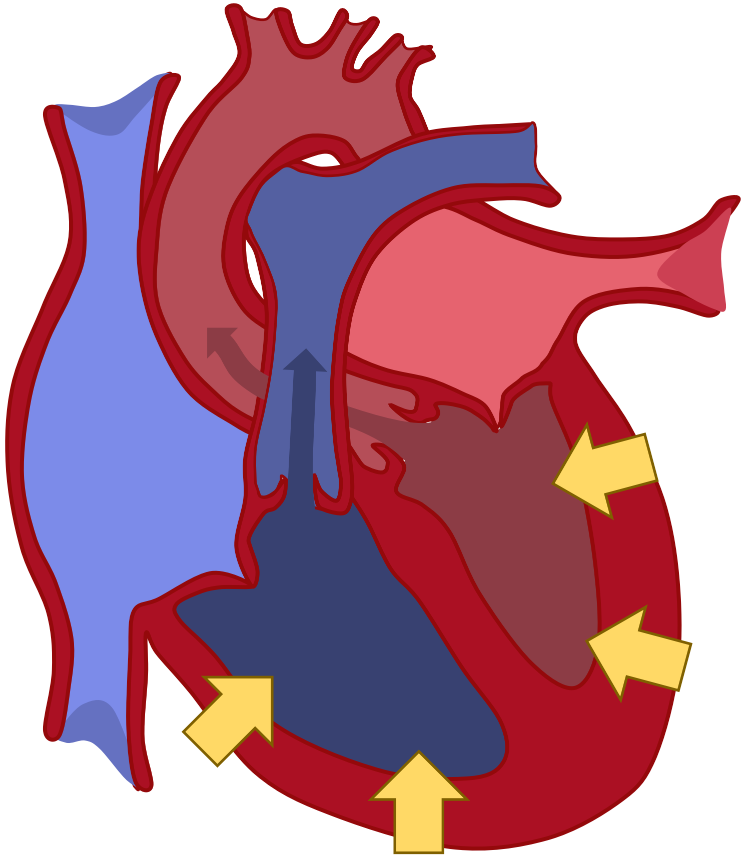
Cardiac cycle: **Rapid ejection**

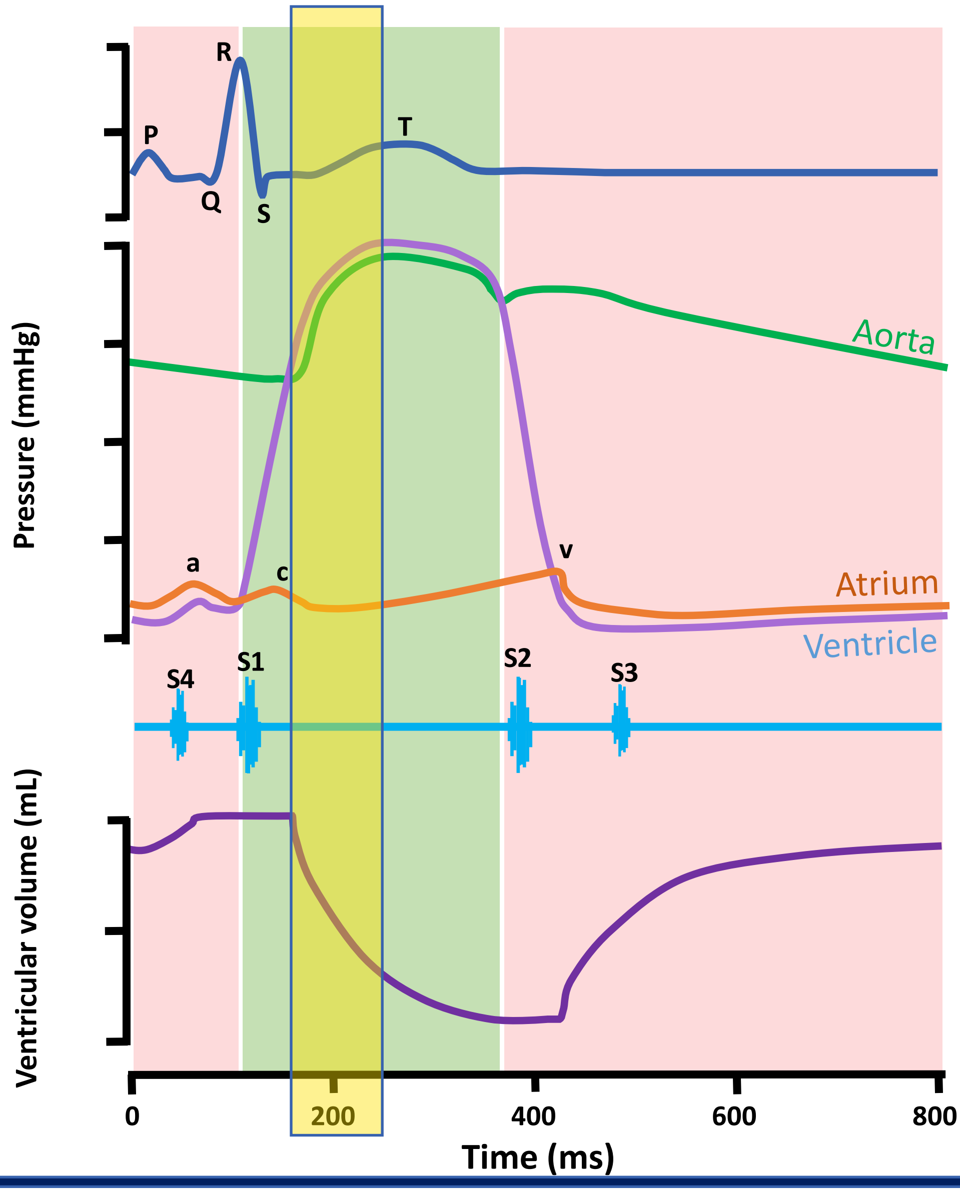
Rapid ejection

Opening of the aortic & pulmonary valves mark the start of this phase

As ventricles contract pressure within them exceeds pressure in aorta and pulmonary arteries. Semilunar valves open, blood pumped out and the volumes of ventricles decrease (isotonic contraction).

No heart sounds for this phase

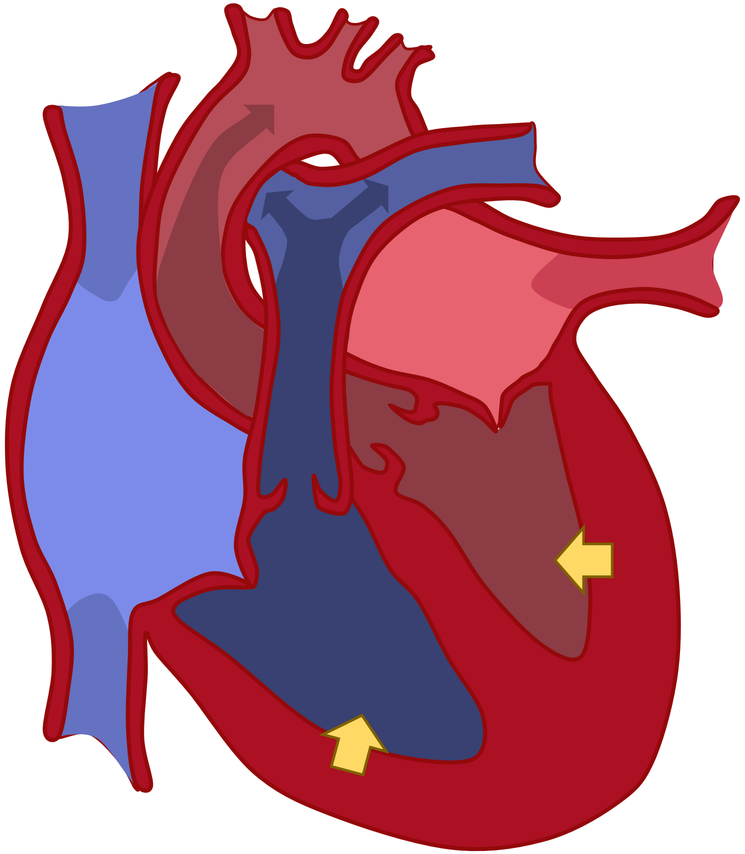


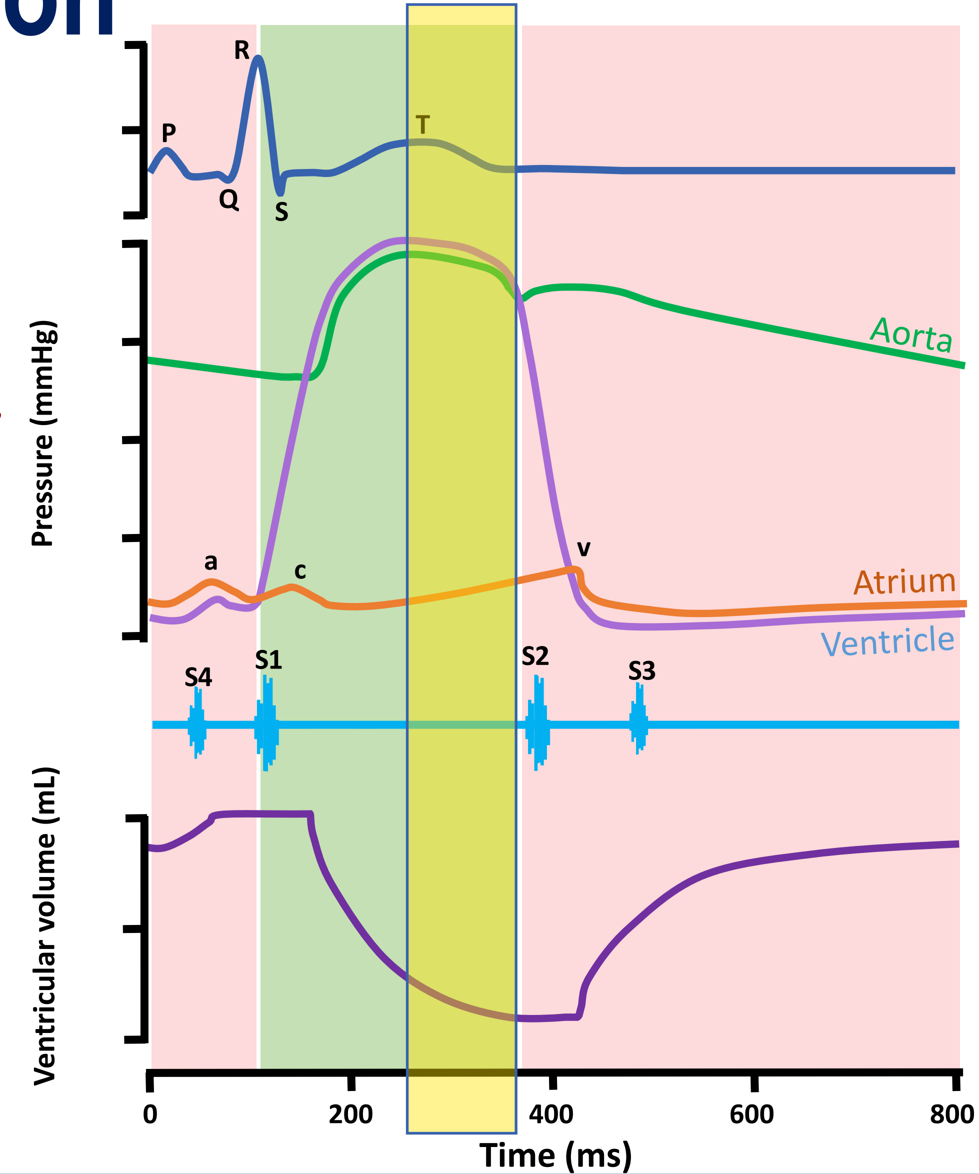


Heart and lung mechanics: Explain the mechanical factors associated with heart and lung function.

Cardiac cycle: **Reduced ejection**

Reduced ejection
This phase marks the **end of systole**
Reduced pressure gradient means **aortic & pulmonary valves begin to close**
Blood flow from ventricles decreases and **ventricular volume decreases more slowly**
As pressures in ventricles fall below that in arteries, blood begins to flow back **causing semilunar valves to close**
Ventricular muscle cells repolarize producing T wave





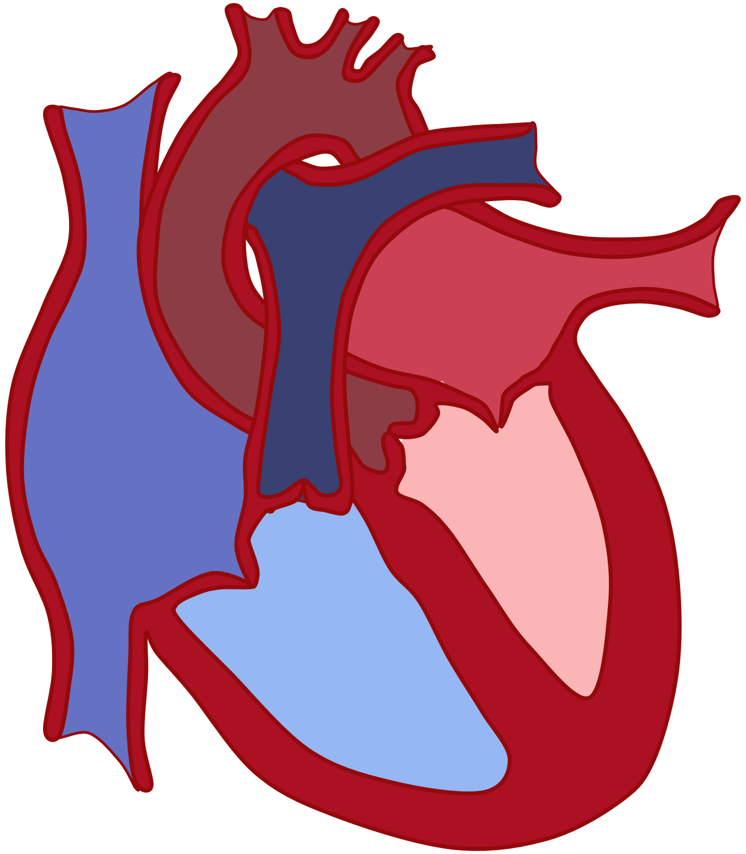
Cardiac cycle: **Isovolumetric relaxation**

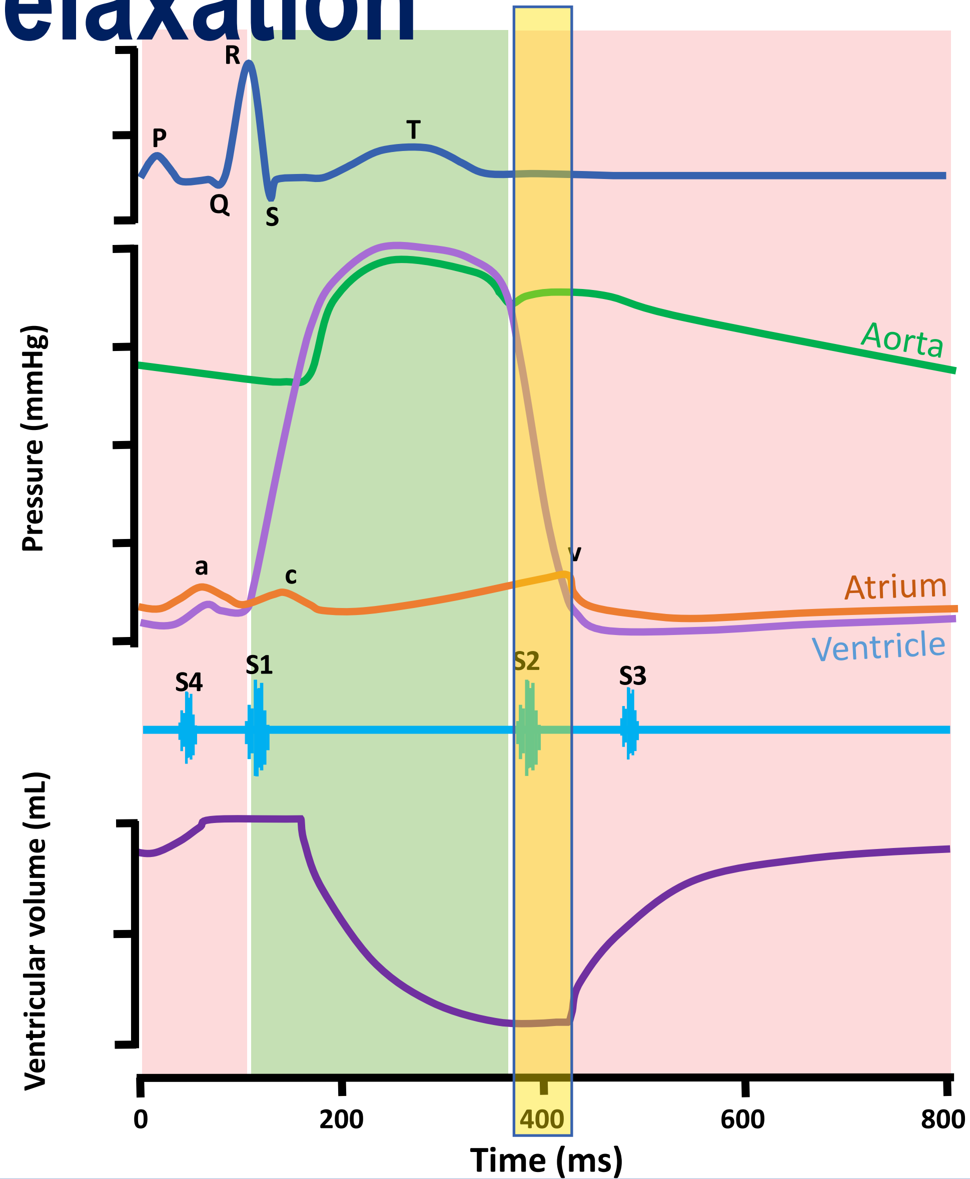
Isovolumetric relaxation

The aortic & pulmonary valves shut, but the **AV valves remain closed** until **ventricular pressure drops below atrial pressure**.

Atrial pressure continues to rise. **Dichrotic notch** (green line) caused by rebound pressure as **distended aortic wall relaxes**.

2nd heart sound ('**dub**') due to **closure of semilunar valves** and associated vibrations





Cardiac cycle: **Rapid passive filling**

Rapid passive filling

Occurs during isoelectric (flat) ECG **between cardiac cycles**

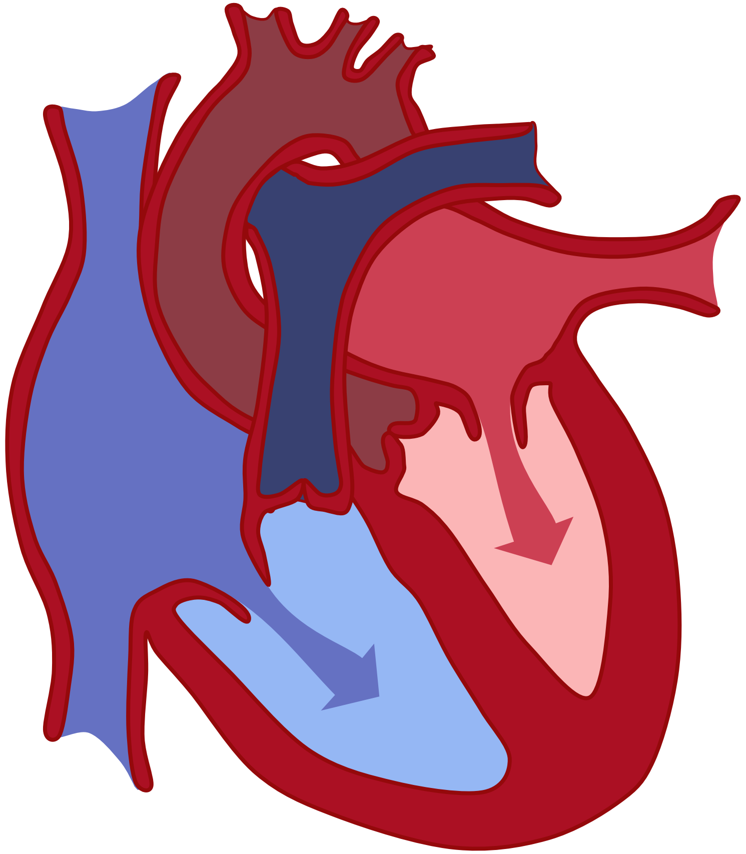
Once AV valves open blood in the atria flows rapidly into the ventricles.

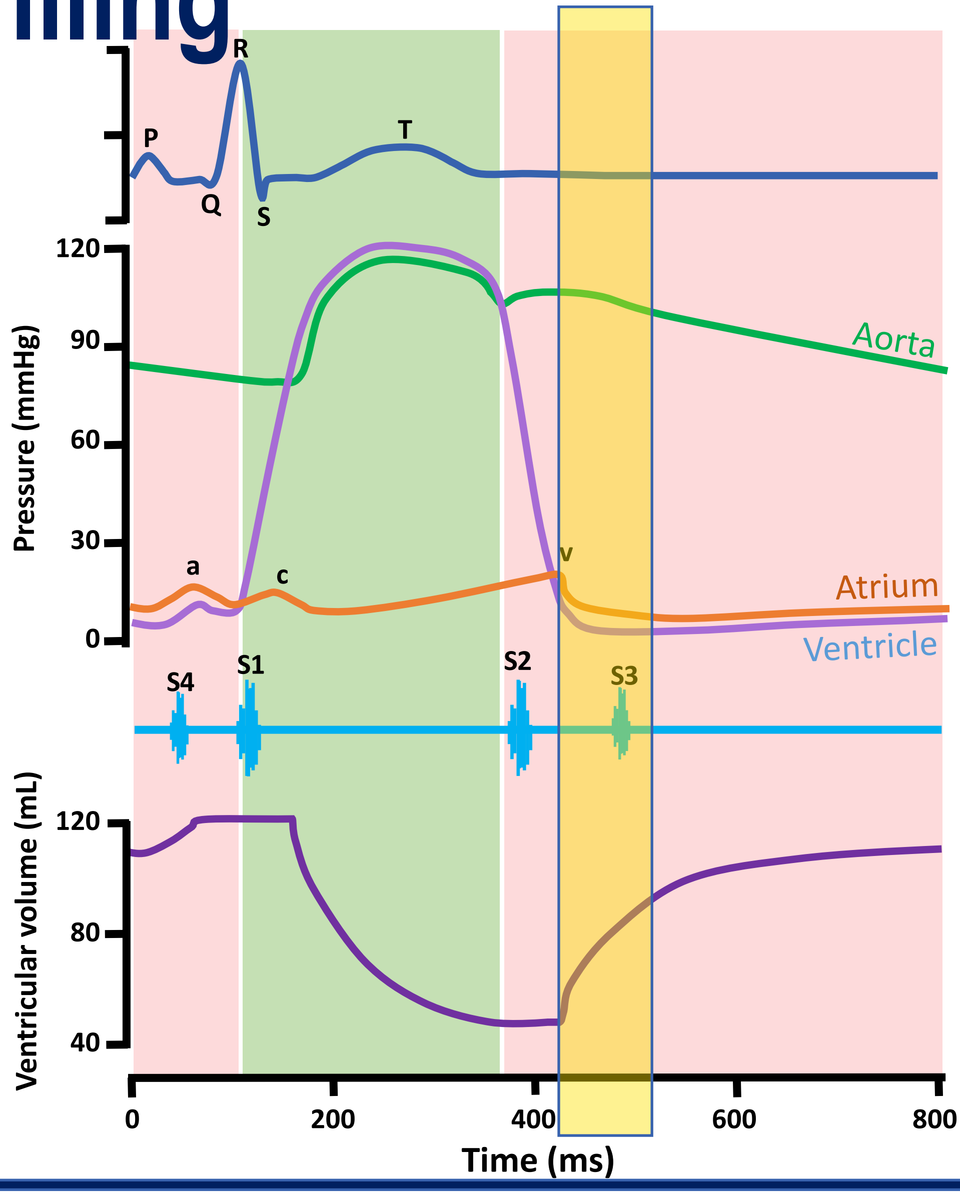
3rd heart sound – **usually abnormal** and may signify turbulent ventricular filling

Can be due to severe hypertension or mitral incompetence

Normal

S3





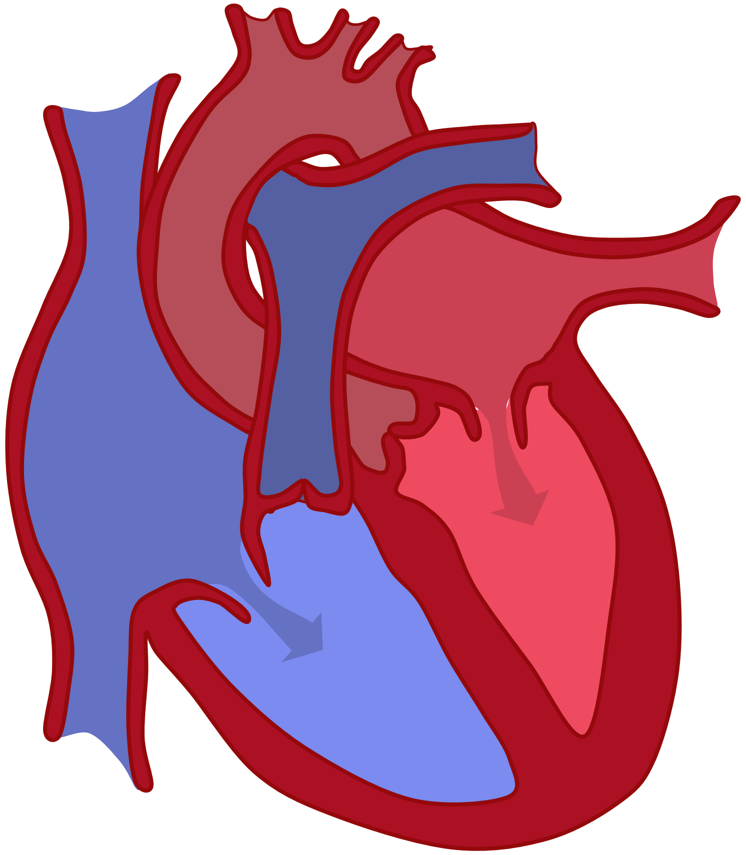
Cardiac cycle: **Reduced passive filling**

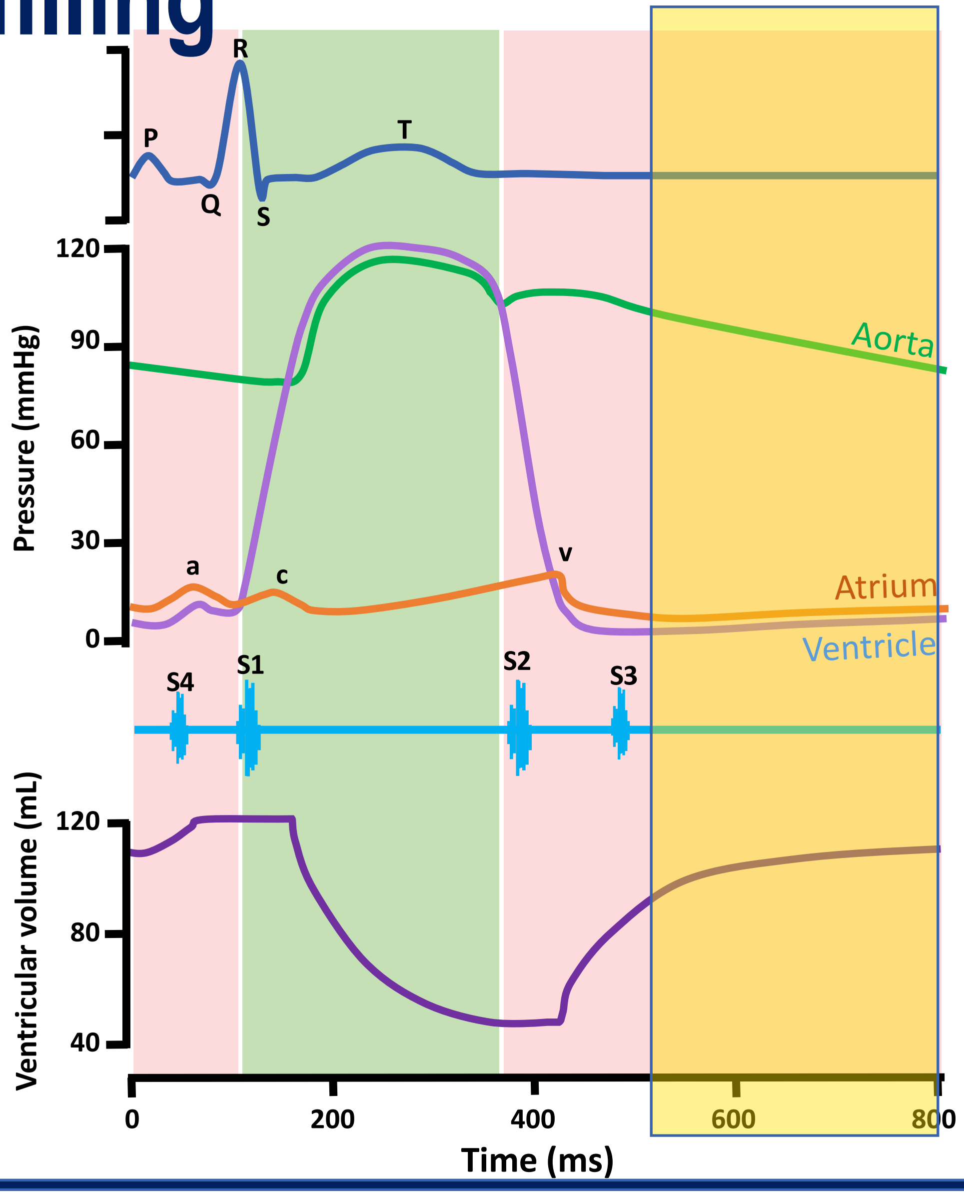
Reduced passive filling

This phase can be called **diastasis**

Ventricular volume fills **more slowly**

The ventricles are able to **fill considerably** without the contraction of the atria.



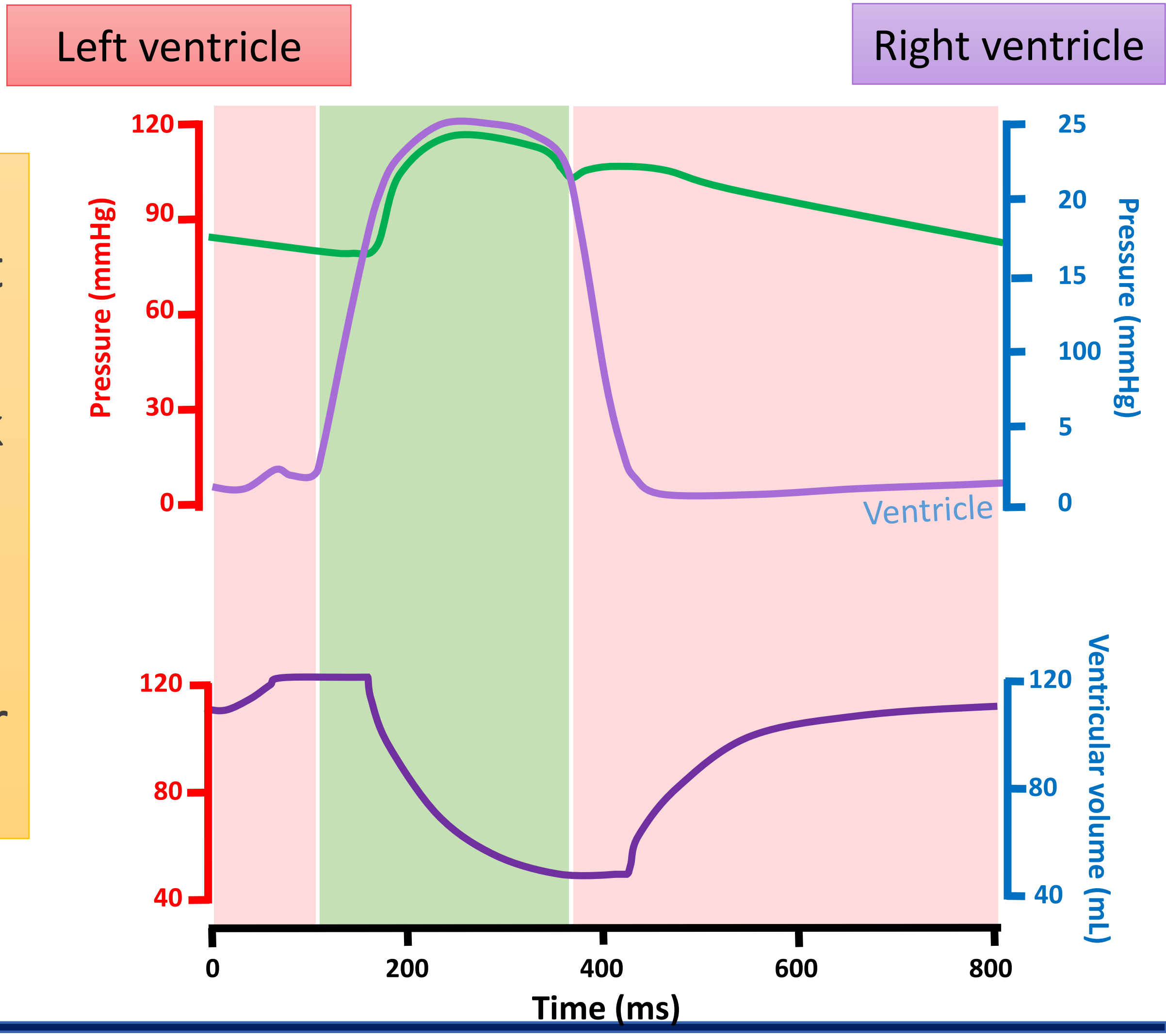


Pulmonary circuit pressures

The **patterns of pressure changes** in the right heart are essentially **identical** to those of the left

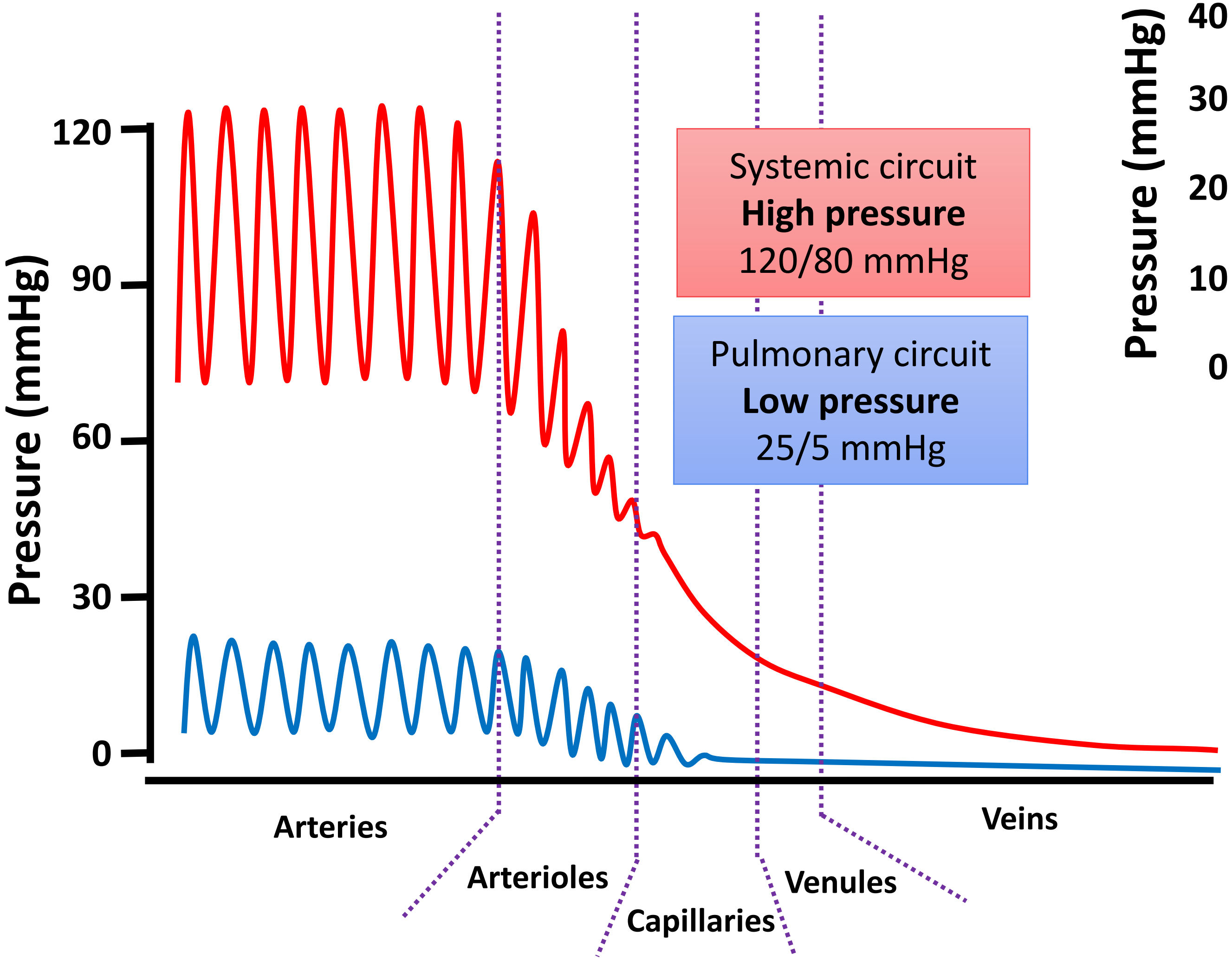
Quantitatively, the pressures in the right heart and pulmonary circulation are **much lower** (peak of systole – 25mmHg in pulmonary artery)

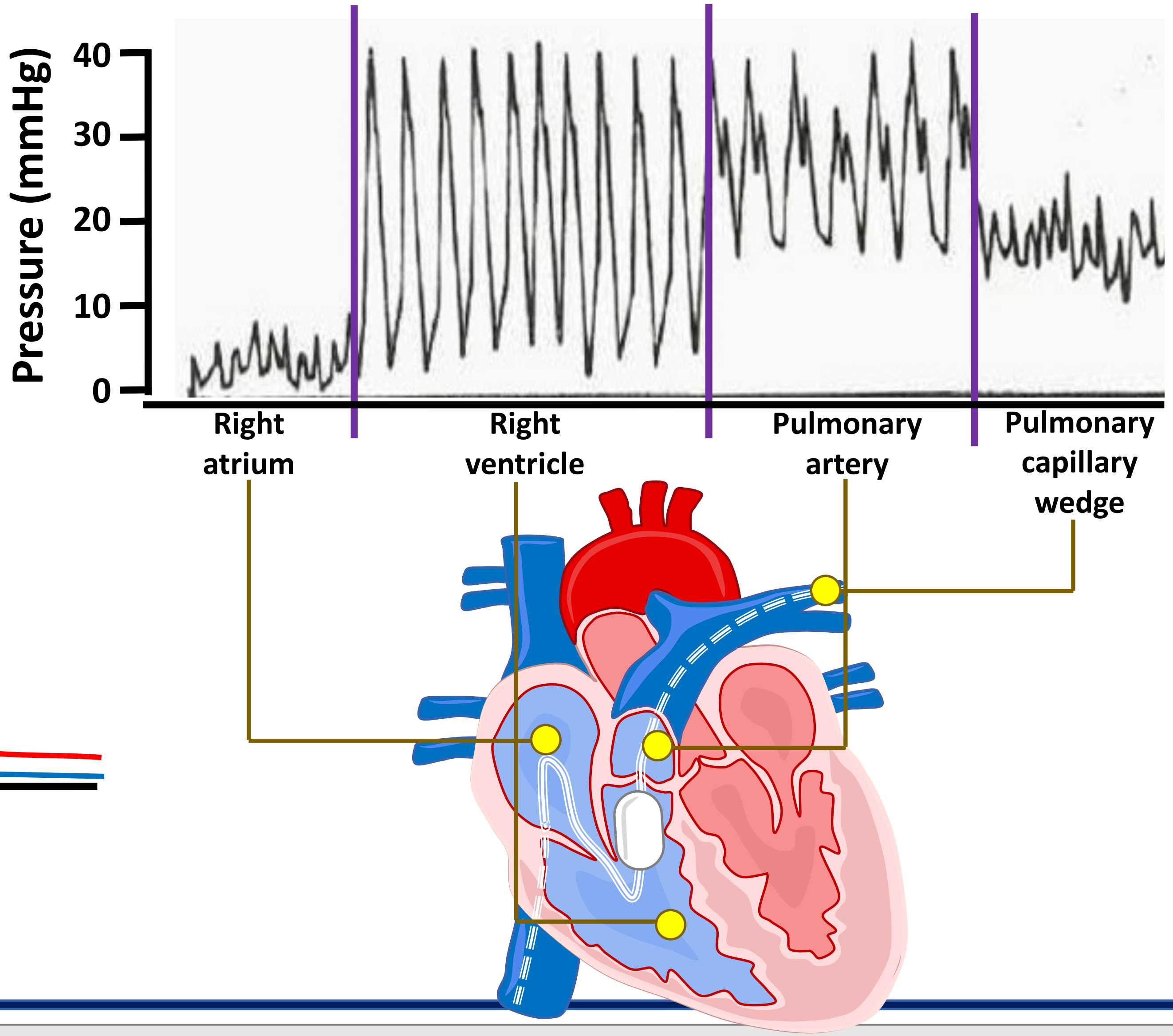
Despite lower pressures right ventricle ejects **same volume of blood** as left (it is simply pumping the same quantity of blood into a lower pressure circuit)!



Cardiovascular and respiratory systems regulation: Outline the regulation and of the cardiovascular and respiratory systems.

Pulmonary circuit pressures





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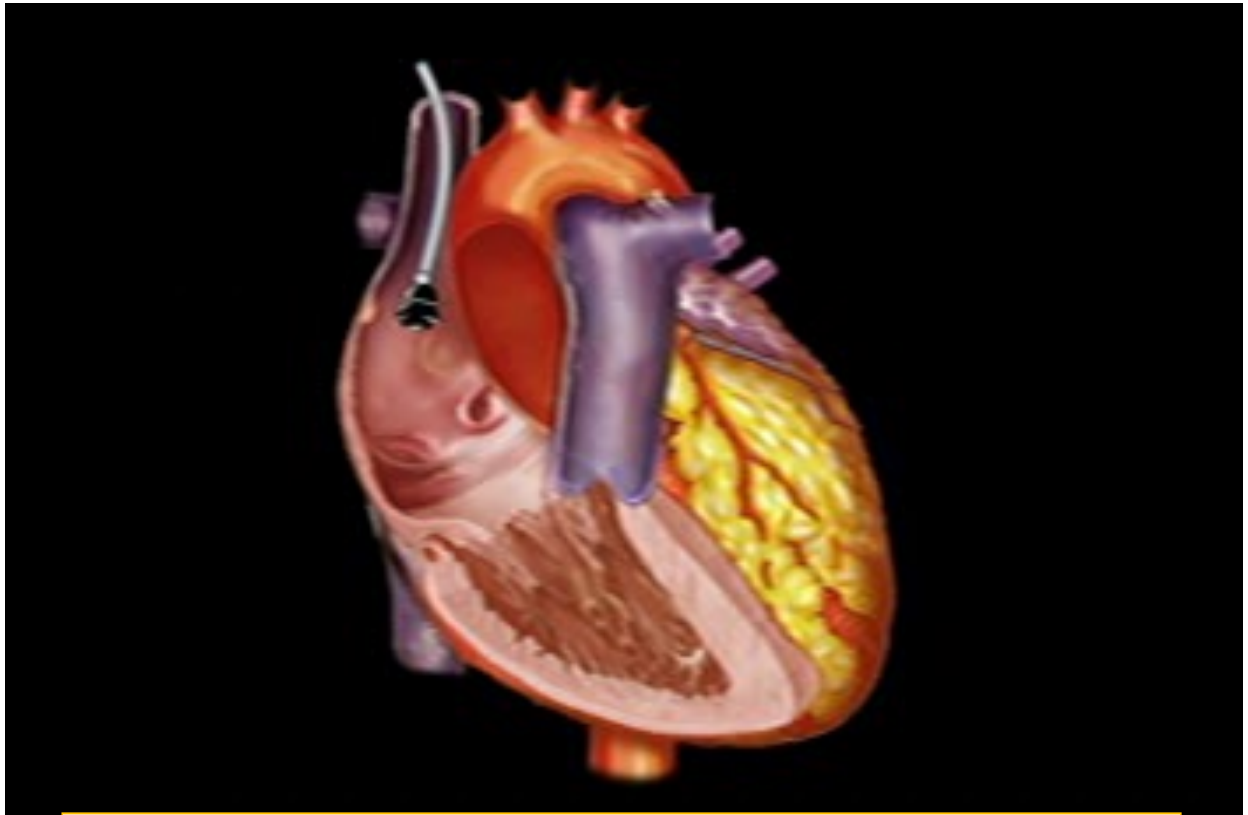
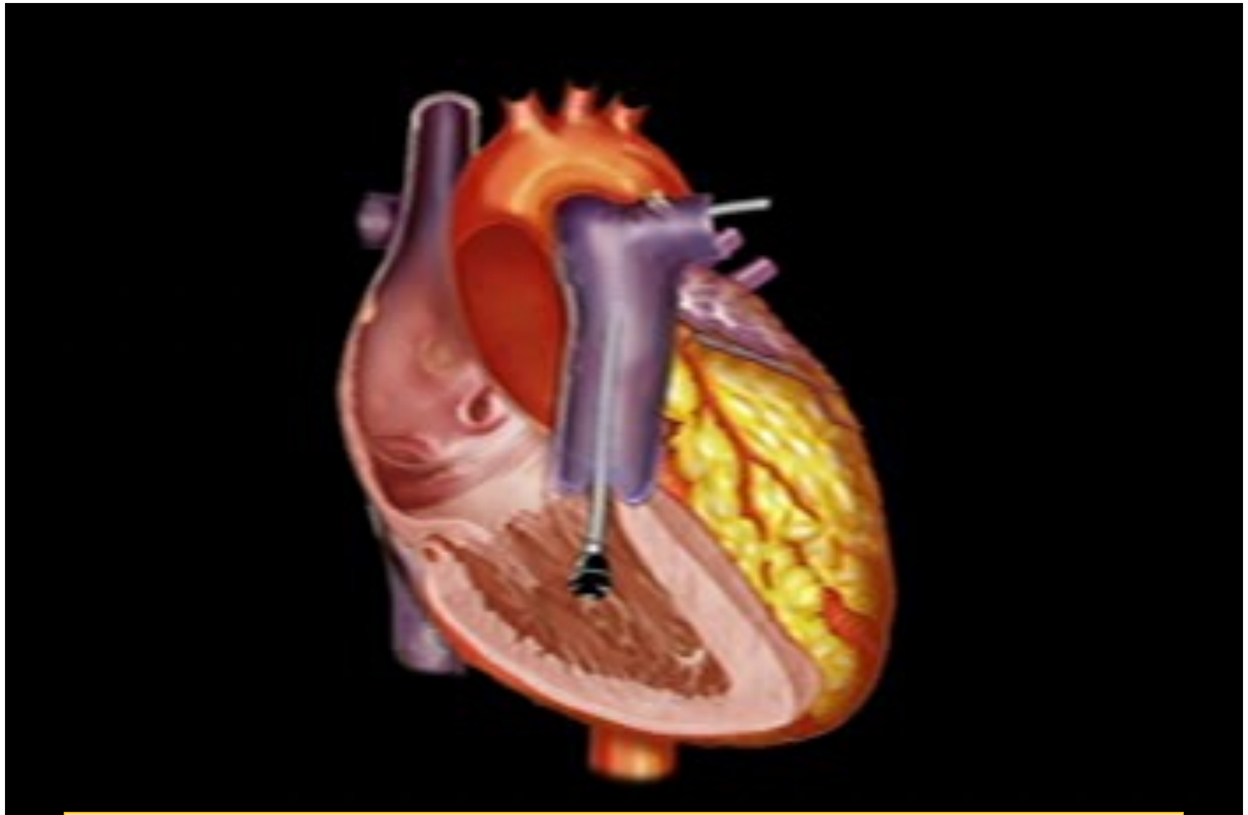
Pressure changes



Right atrium to right ventricle via tricuspid valve



Right ventricle to pulmonary artery via pulmonary valve

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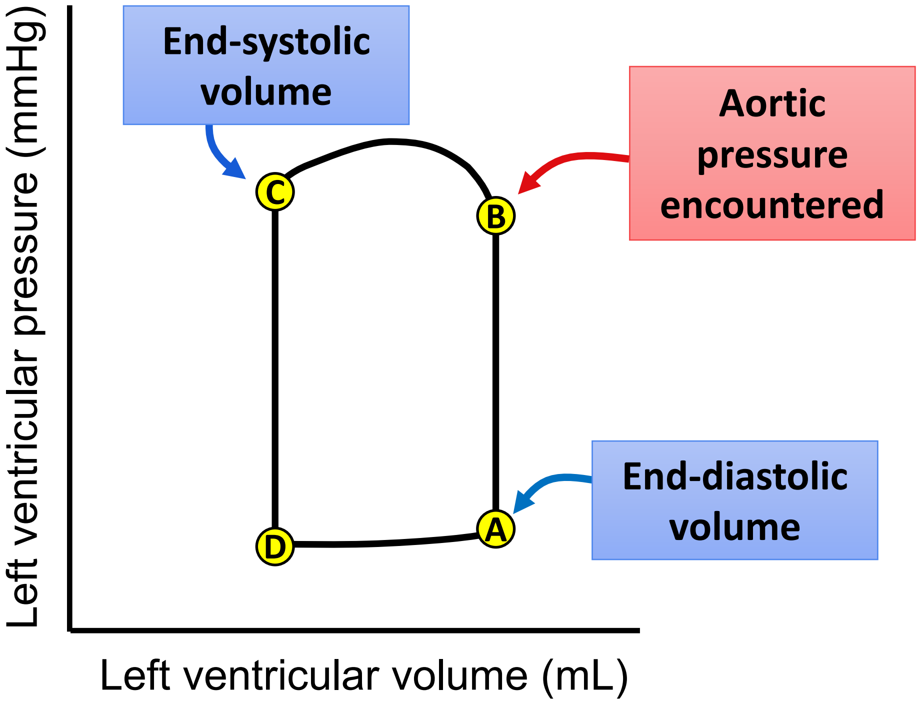

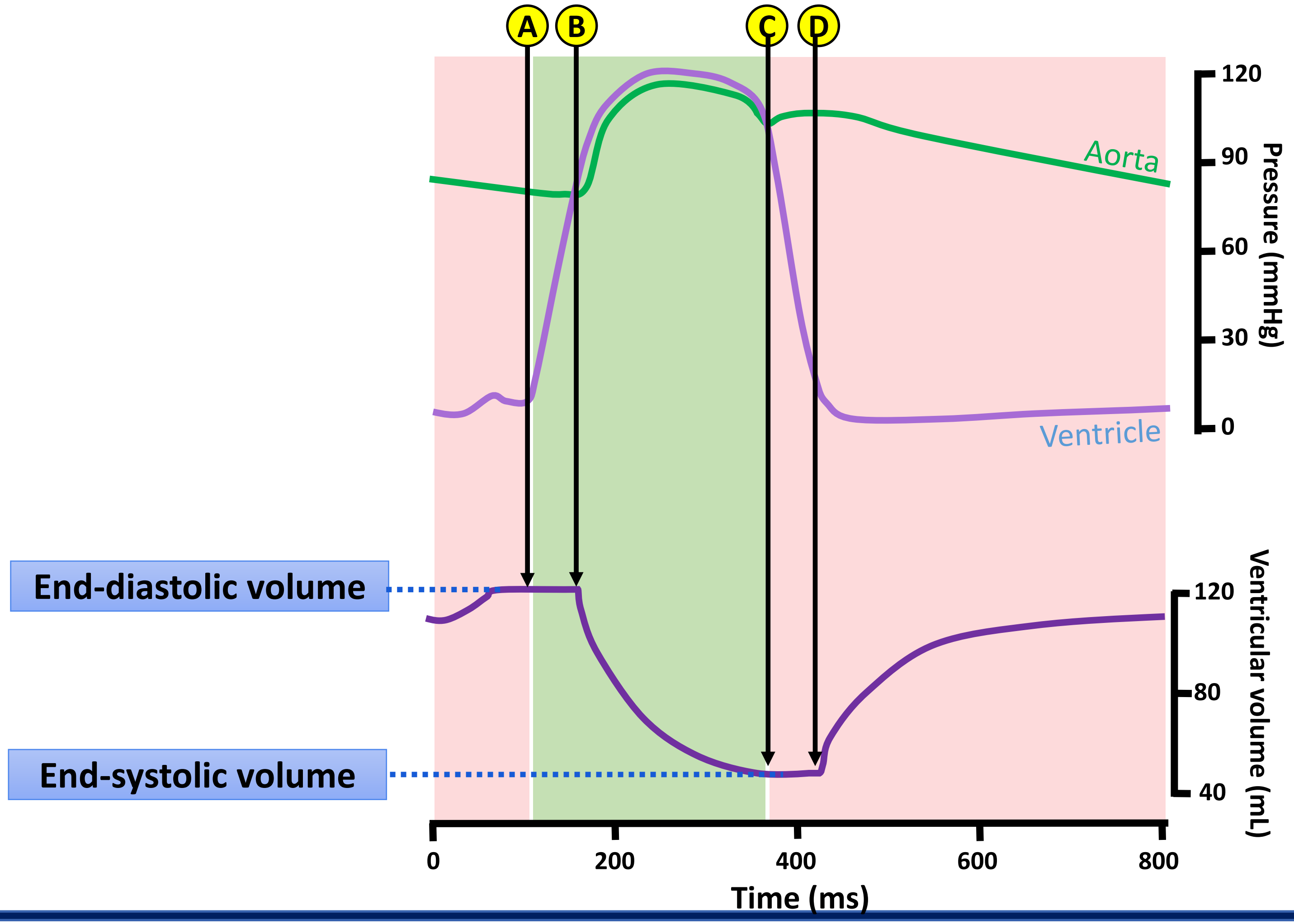

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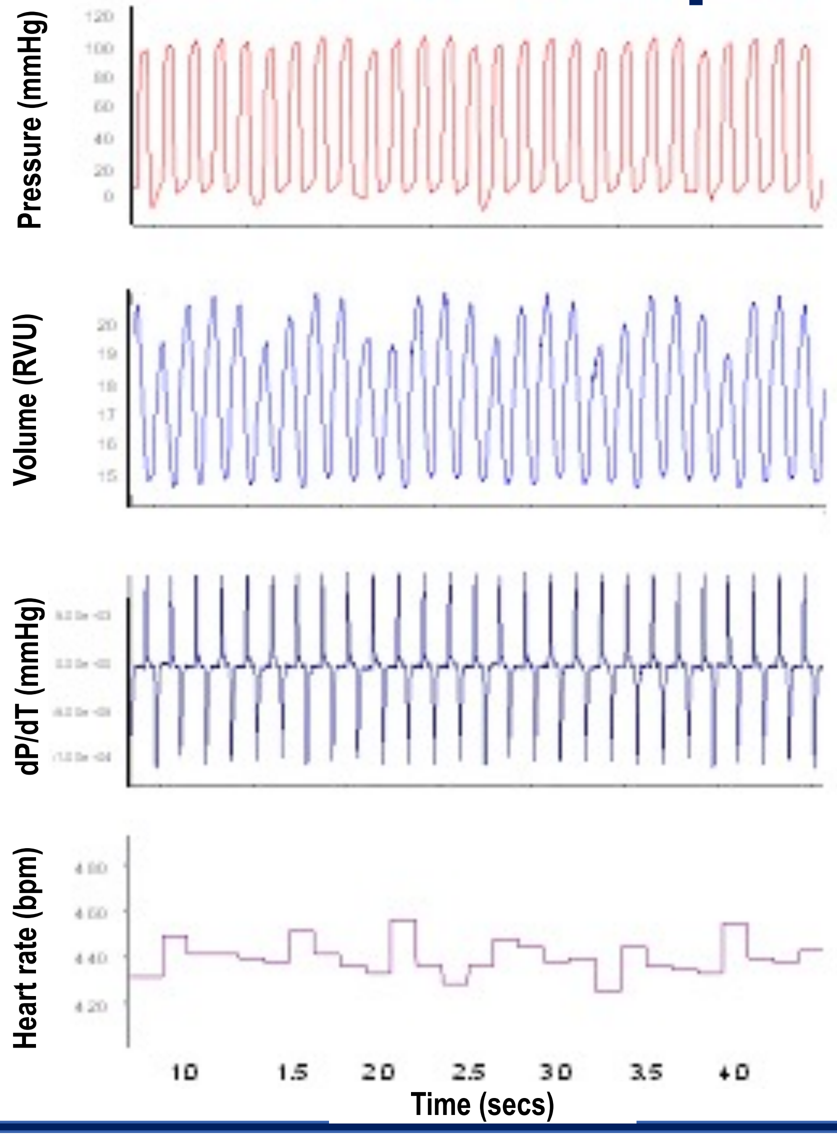

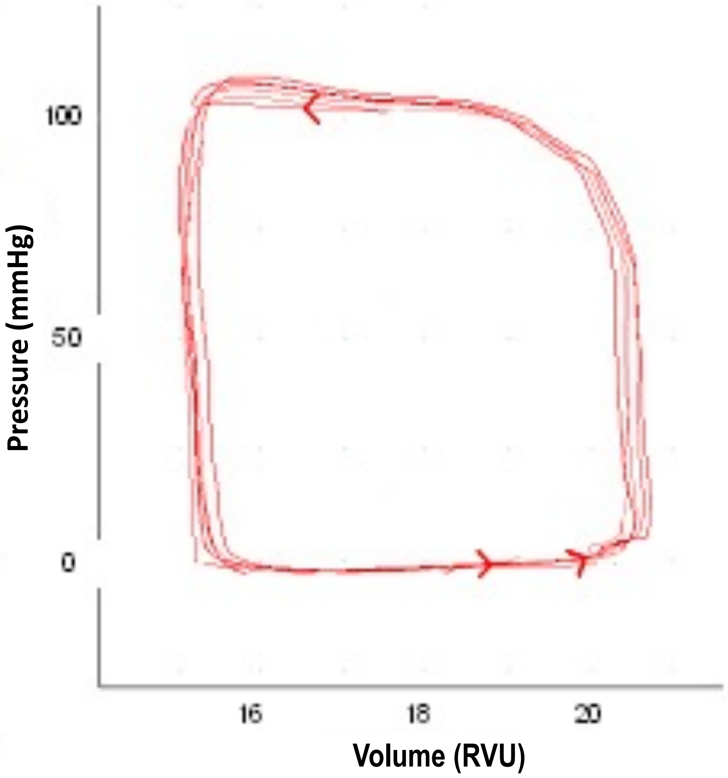

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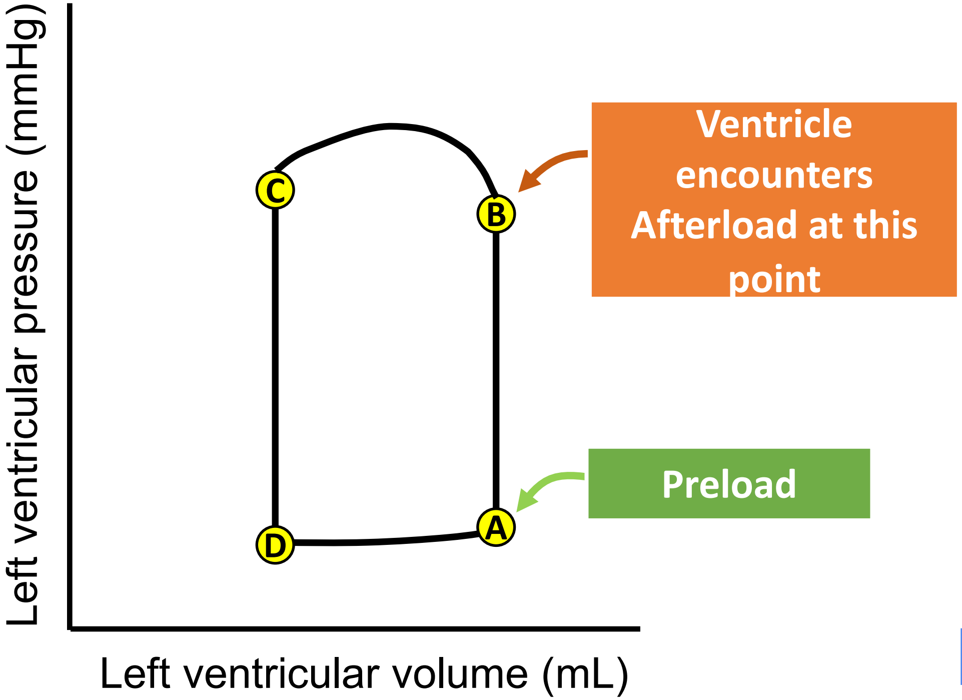
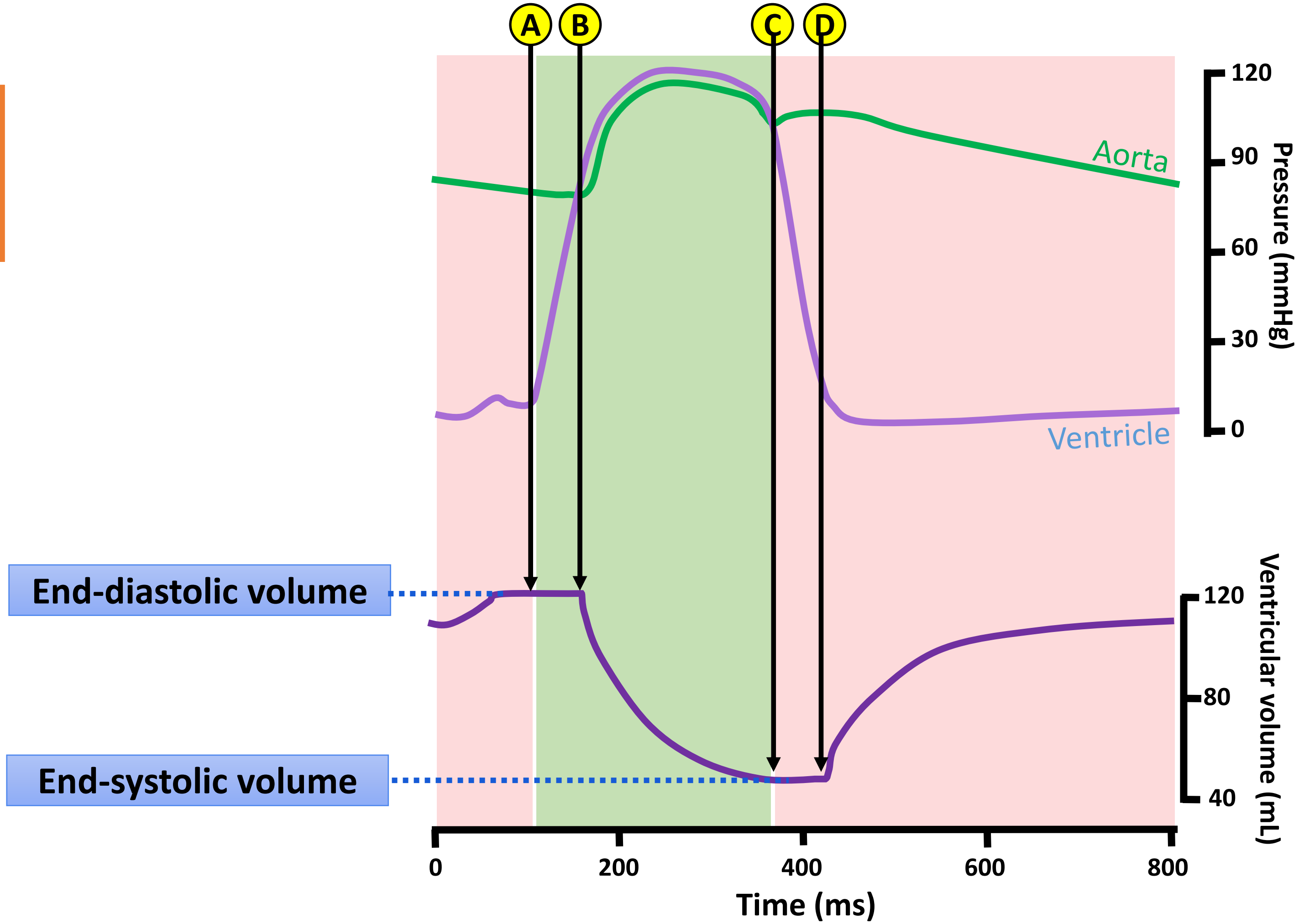
Preload and afterload on the pressure volume loop



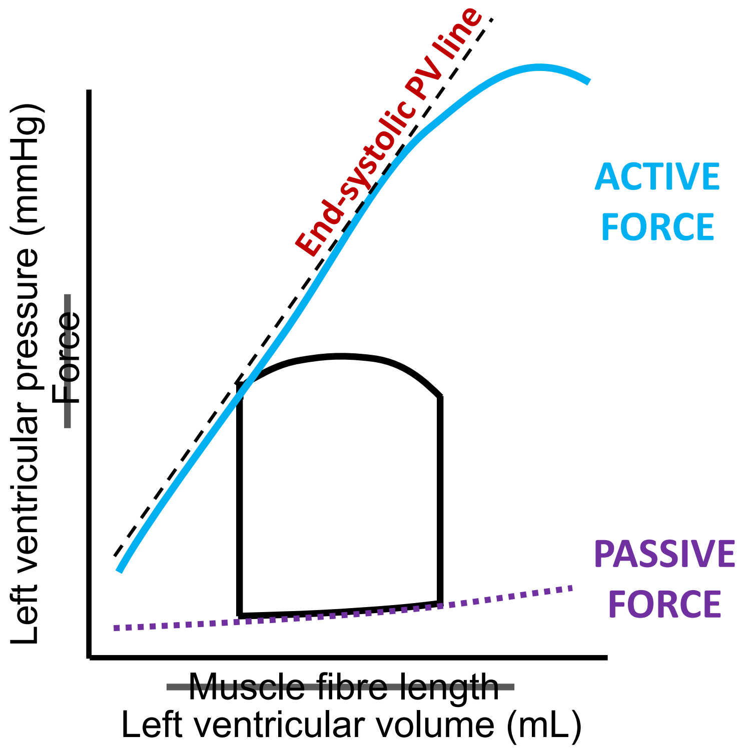


Blood filling the ventricles during diastole determines the **PRELOAD** that stretches the resting ventricular muscle

The blood pressures in great vessels (aorta and pulmonary artery) represent the **AFTERLOAD**

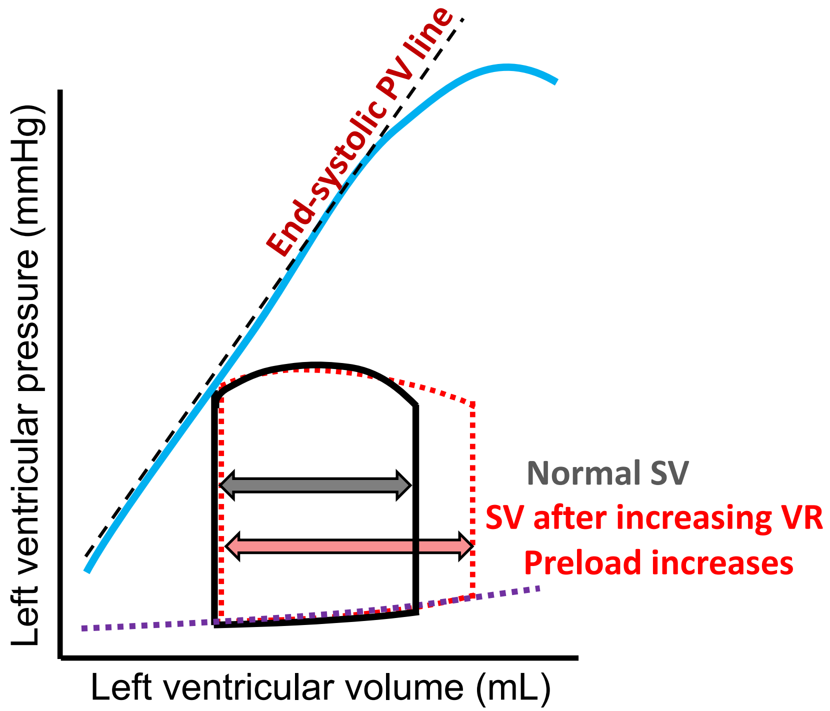
Preload and afterload on the pressure volume loop

ESPVR is the **maximal pressure that can be developed by the ventricle at any given volume**



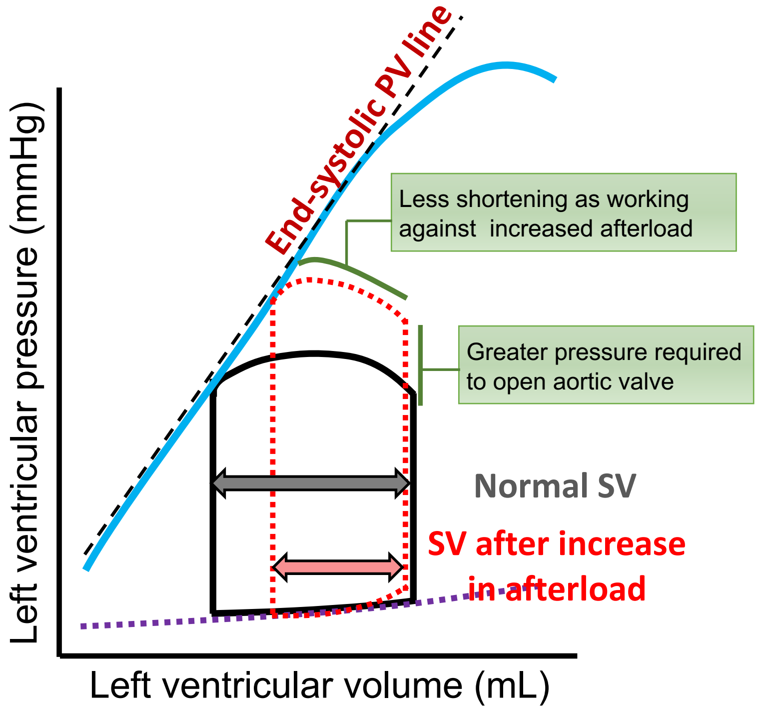
Increases in preload result in **increased stroke volume**

This is the **Frank-Starling** relationship



Increases in afterload result in **decreased stroke volume**

Remember from previous lecture that as **afterload increases**, the **amount of shortening that occurs decreases**



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Changing cardiac output

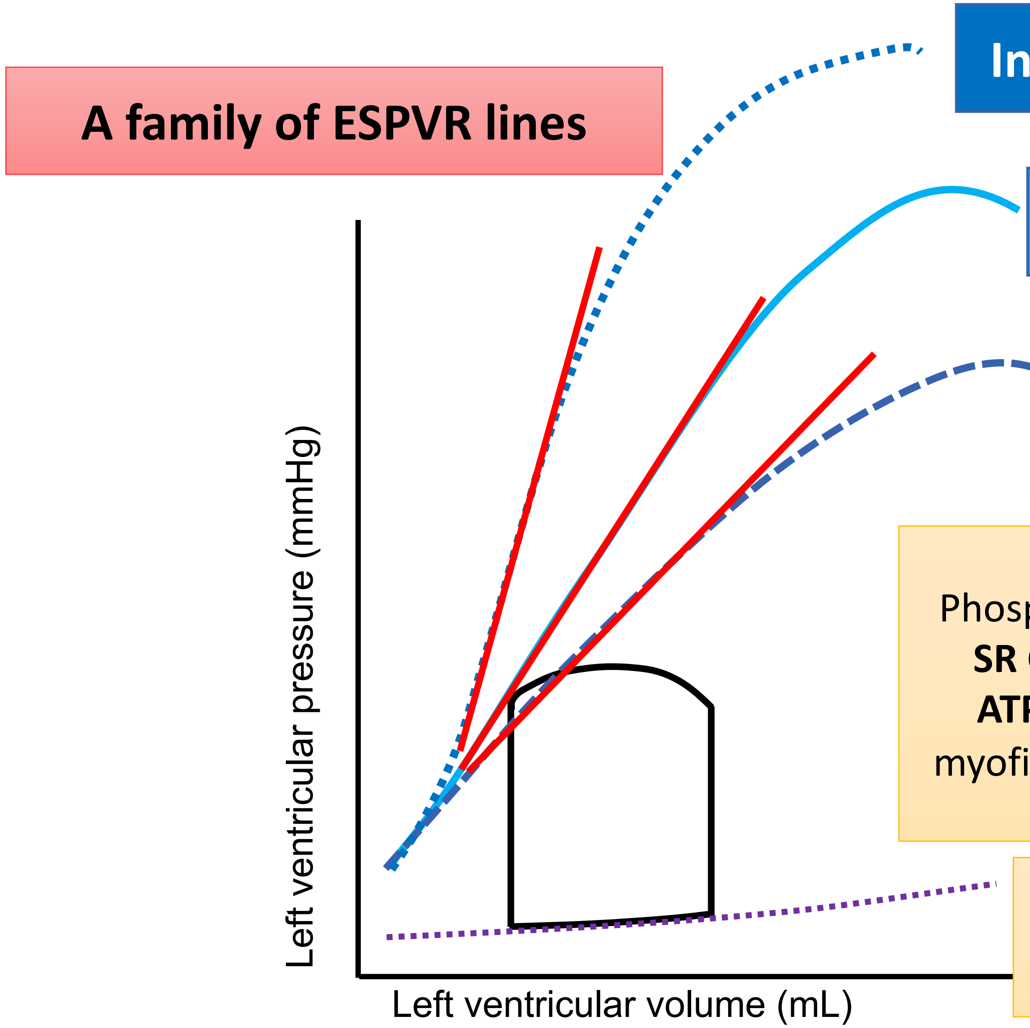
Cardiac output = Heart rate x Stroke volume

Stroke volume → Preload | Afterload | **Contractility**

Definition: Contractile capability (or strength of contraction) of the heart
Increased by: Sympathetic stimulation
Extrinsic mechanism: Changes Ca^{2+} delivery to myofilaments

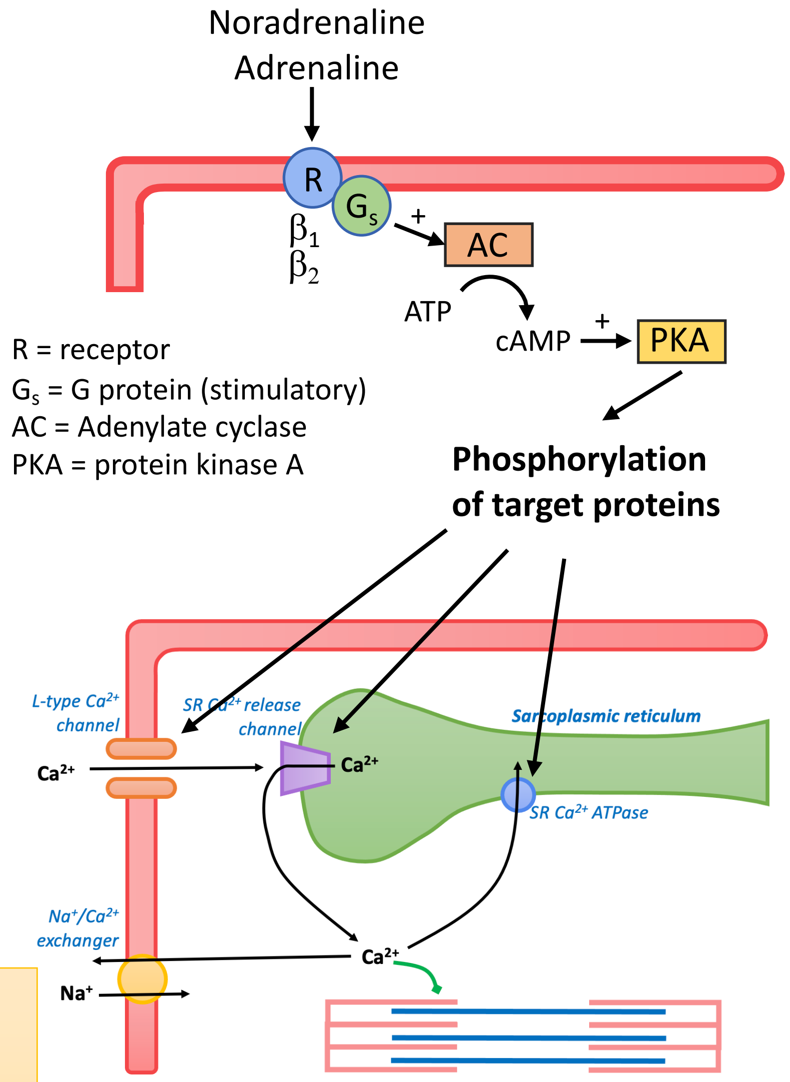
As contractility changes, a 'family' of **Frank-Starling relations** become apparent

Cardiac contractility affects steepness of the Frank-Starling relationship



Phosphorylation of **L-type Ca^{2+} channel**, **SR Ca^{2+} release channel** and **SR Ca^{2+} ATPase** increases delivery of Ca^{2+} to myofilaments and increases **contractility**

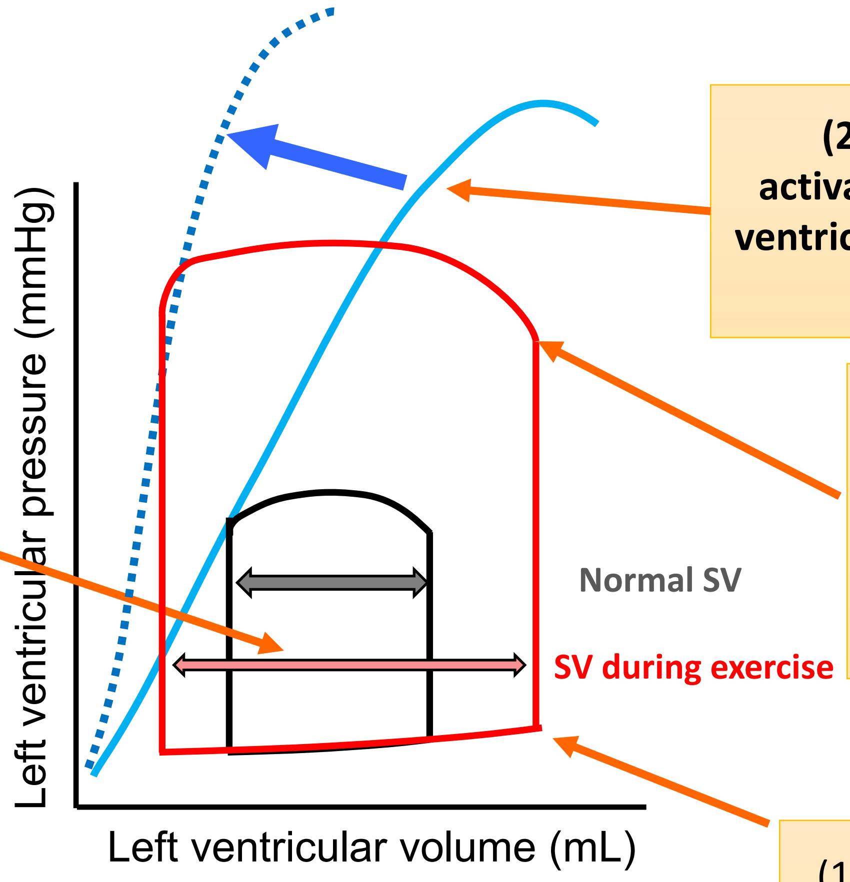
Increased contractility = **increased gradient of ESPVR lines**



What happens to PV loops during exercise?

(4) **Combination** of **increased cardiac contractility and increased VR generate increased SV** (and EF)

FINAL NOTE: If HR increases to very high rates, diastolic filling time can be reduced and this decreases EDV



(2) Main factor: Sympathetic activation of the myocytes increases ventricular contractility, that decreases end-systolic volume.

(3) The **increase in arterial pressure that occurs during exercise increases afterload** (and can lessen the reduction in end-systolic volume but offset by large increase in contractility)

(1) Increased VR aided by muscle and respiratory pump **increases EDV**

Session review

The cardiac cycle

- Can be broadly split into **systole** (contraction) and **diastole** (relaxation) – most often in reference to the ventricles
- **Seven distinct phases** can be identified
- **Pressure changes** in the atria, ventricles and outflow arteries govern valve movement
- Valve closure and rebound pressure produces healthy heart sounds **S1** and **S2**

Pressure volume loops

- A **graphical representation** of how ventricular pressures and volumes change during the cardiac cycle
- Has a typically ‘box-like’ profile bordered at the top left by **end-systolic pressure volume relation (ESPVR)**

Preload & afterload

- Changes in preload affect the **width** of the PV loop
  - **Preload affected by volume of blood returning to the heart**
- Changes in afterload affect the **height** and **left border** of the PV loop
  - The upper left point follows the **ESPVR**
  - **Afterload affected by volume of blood capable of being ejected**, due to obstruction of pressure gradient

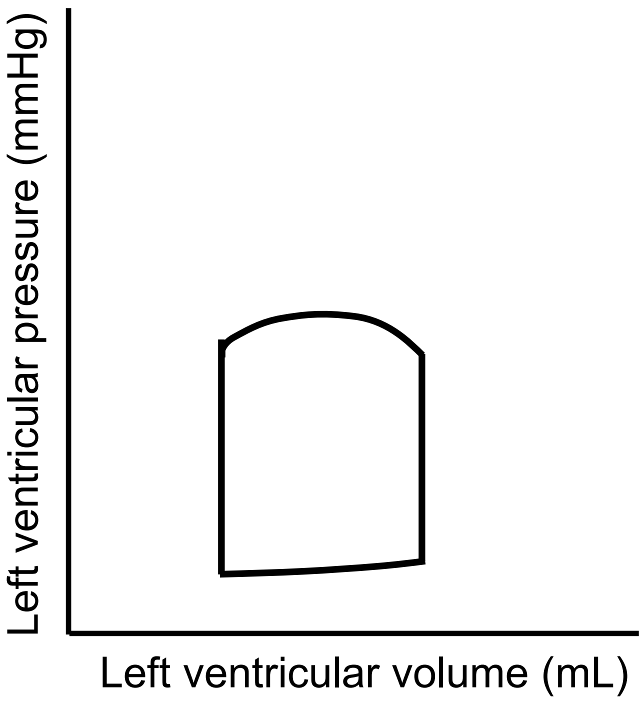
Extrinsic stimulation

- Parasympathetic stimulation is present at rest, which **slows the SA node rate** from 110 bpm to 70 bpm
- Sympathetic stimulation increases SA node rate via:
  - **Hormonal**: Circulating adrenaline from adrenal gland
  - **Neural**: Noradrenaline released from nerves

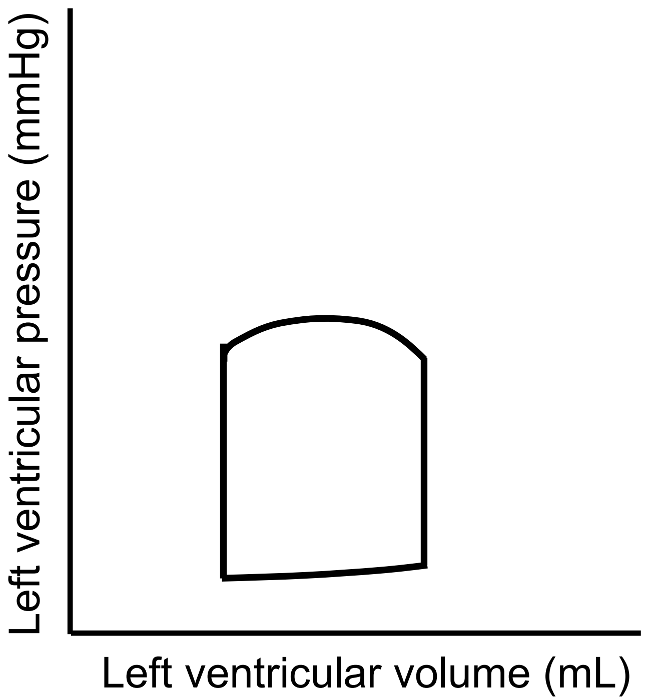
Review questions

Q1: Draw the normal PV loop below. Add to it what you think the PV loops for the following may look like

a) Hardening of the aortic valve



b) Exercise



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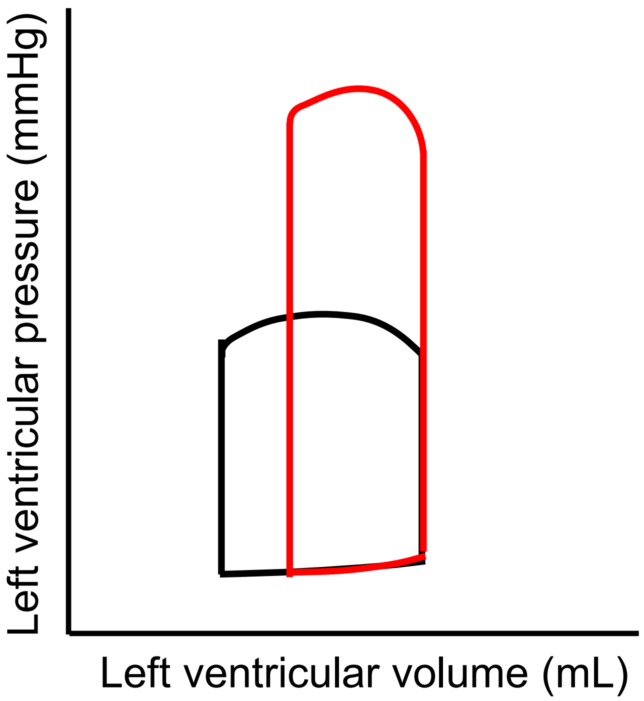
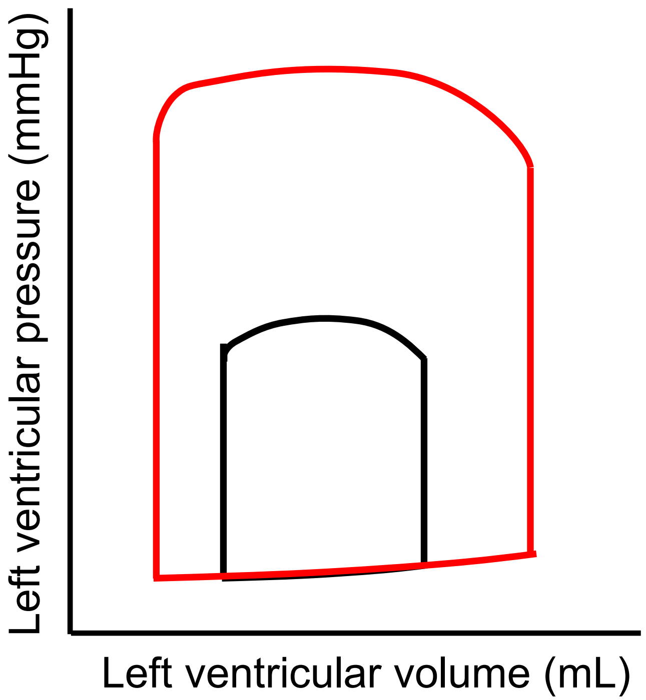


Hardening and narrowing of the aortic valve reduces flow and **increases afterload**



Venous return increases due to venoconstriction and skeletal muscle pump, and **contractility is increased** via sympathetic nervous system

Review questions

Q2: Which of the following will not affect preload?

A: Increased adrenaline secretion during fight or flight response

B: Decreased ventricular compliance

C: Hardening and narrowing (stenosis) of the pulmonary valve

D: Right atrial pressure

E: Decreased central venous pressure

Review questions

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C: Hardening and narrowing (stenosis) of the pulmonary valve

D: Right atrial pressure

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Review questions

Q3: Which is the longest phase of the cardiac cycle?

A: Reduced passive filling

B: Isovolumetric contraction

C: Reduced ejection

D: Atrial systole

E: Isovolumetric relaxation

Review questions

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