

- Inotropy / contractility = change in SV at a given EDV. (gives shift in Starlings curve)
 - Happen as result of Ca^{2+} handling in cell; things that change in heart failure.

After-load affecting Stroke Volume:

- After-load = the force/load against which the heart must contract to eject the stroke volume
 - E.g. arterial blood pressure
 - High ABP gives high afterload; heart having to produce even greater pressure to project blood into aorta
 - High afterload/ aortic pressure makes it more difficult to eject SV e.g. in systemic or pulmonary arterial hypertension.
- Affected by:
 - Peripheral resistance:
 - High PR = higher BP makes it more difficult for blood to be ejected into aorta.
- Pulmonary artery pressure also affects afterload on right side of heart
 - Patient with heart failure & pulmonary oedema; their pulmonary pressure will be higher so pressure for RV (right ventricle) higher.

Summary:

- Increase SV by Starlings law of heart, by changing EDV
 - No change in ESV by increasing EDV just increase in SV
- Also; can change SV by changing contractility so we fill heart to same as resting but eject more blood (EDV doesn't change but ESV goes down)
- In reality; neither of these happen independently
- Regulation of CO
 - $\text{CO} = \text{HR} \times (\text{EDV} - \text{ESV})$

EDV intrinsic factors:

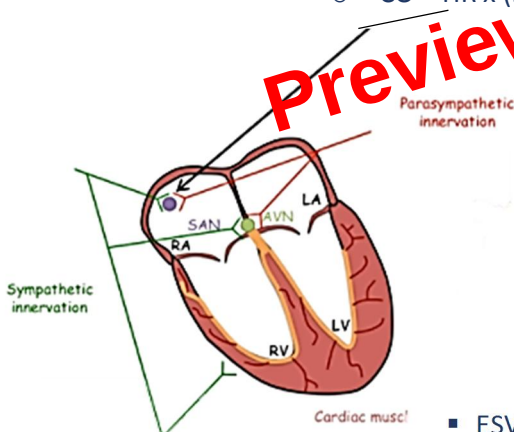
- Pre-load
- HR/CFR

- Blood volume
- Skeletal muscle pump
- Respiratory pump
- Venous tone
- Gravity

- Atrial contraction
- HR (>180bpm)

■ ESV extrinsic factors:

- Contractility/ inotropy
 - Sympathetic nerve activity
 - Circulating adrenaline / noradrenaline.



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