• Inotropy / contractility = change in SV at a given EDV. (gives shift in Starlings curve)
  o Happen as result of Ca2+ 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
  o 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:
  o 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
  o 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
  o No change in ESV by increasing EDV just increase in SV
• Also; can change SV by changing contractility so we fill heart to same resting but eject more blood (EDV doesn’t change but ESV goes down)
• In reality; neither of these happen independently
• Regulation of CO
  o $CO = HR \times (EDV - ESV)$
    • EDV intrinsic factors:
      o Pre-load
      o VR/CVP
    o Blood volume
    o Skeletal muscle pump
    o Respiratory pump
    o Venous tone
    o Gravity
    o Atrial contraction
    o HR (>180bpm)
  • ESV extrinsic factors:
    o Contractility / inotropy
      o Sympathetic nerve activity
      o Circulating adrenaline / noradrenaline.