- Inotropy / contractility= change in SV at a given EDV. (gives shift in Starlings curve)
    - Happen as result of Ca2+ handling in cell; things that change in heart failure.

<u>After-load affecting Stroke Volume:</u>

- 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
    - CO = HR x (EDV – ESV)
        - EDV intrinsic factors:
            - Pre-load
            - TR/CVP
                - 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.