

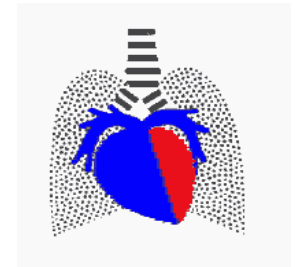
The Heart II.

(CO, preload, afterload, contractility...)

Milan Chovanec

Department of Physiology

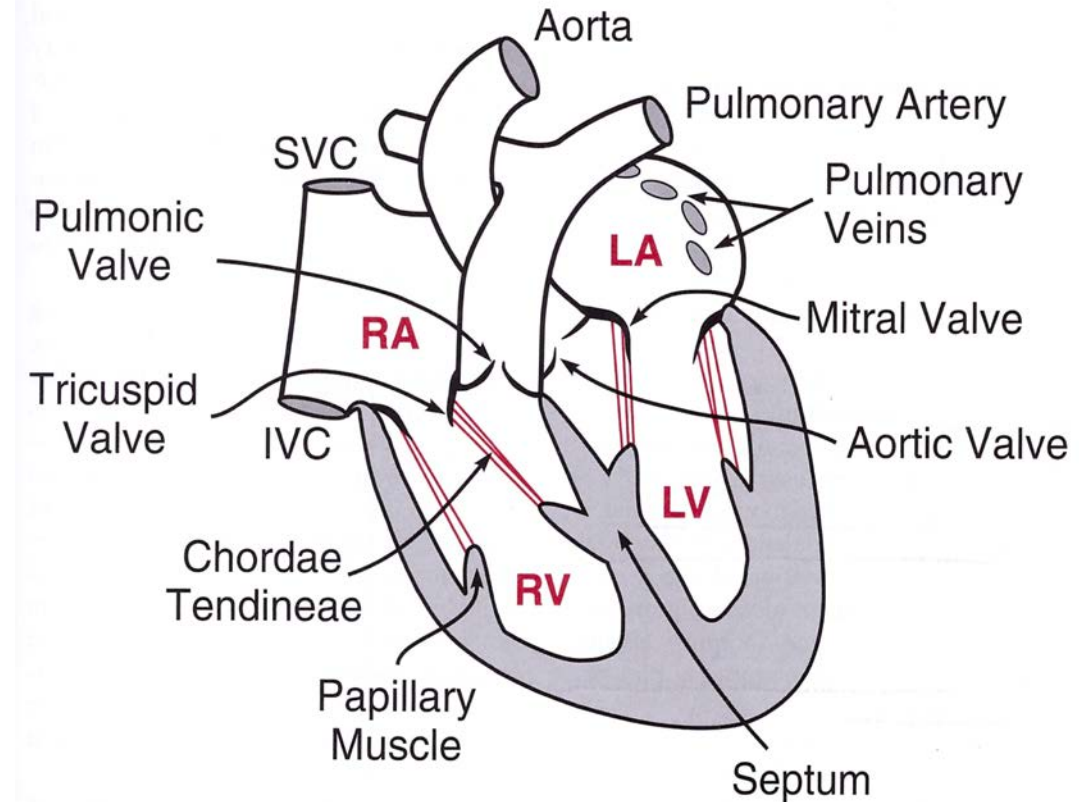
2nd Medical School, Charles University, Prague



- Cardiac cycle, cardiac output
- Preload
- Afterload
- Contractility – inotropy
- Relationship between CO, preload, afterload, contractility, venous return...

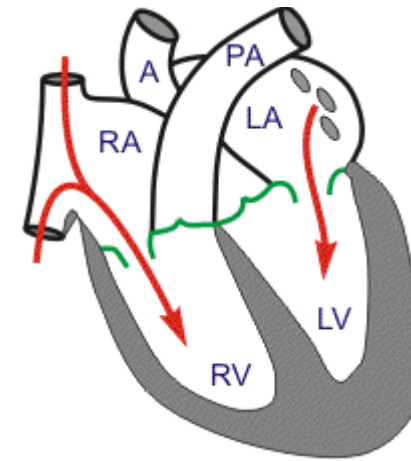
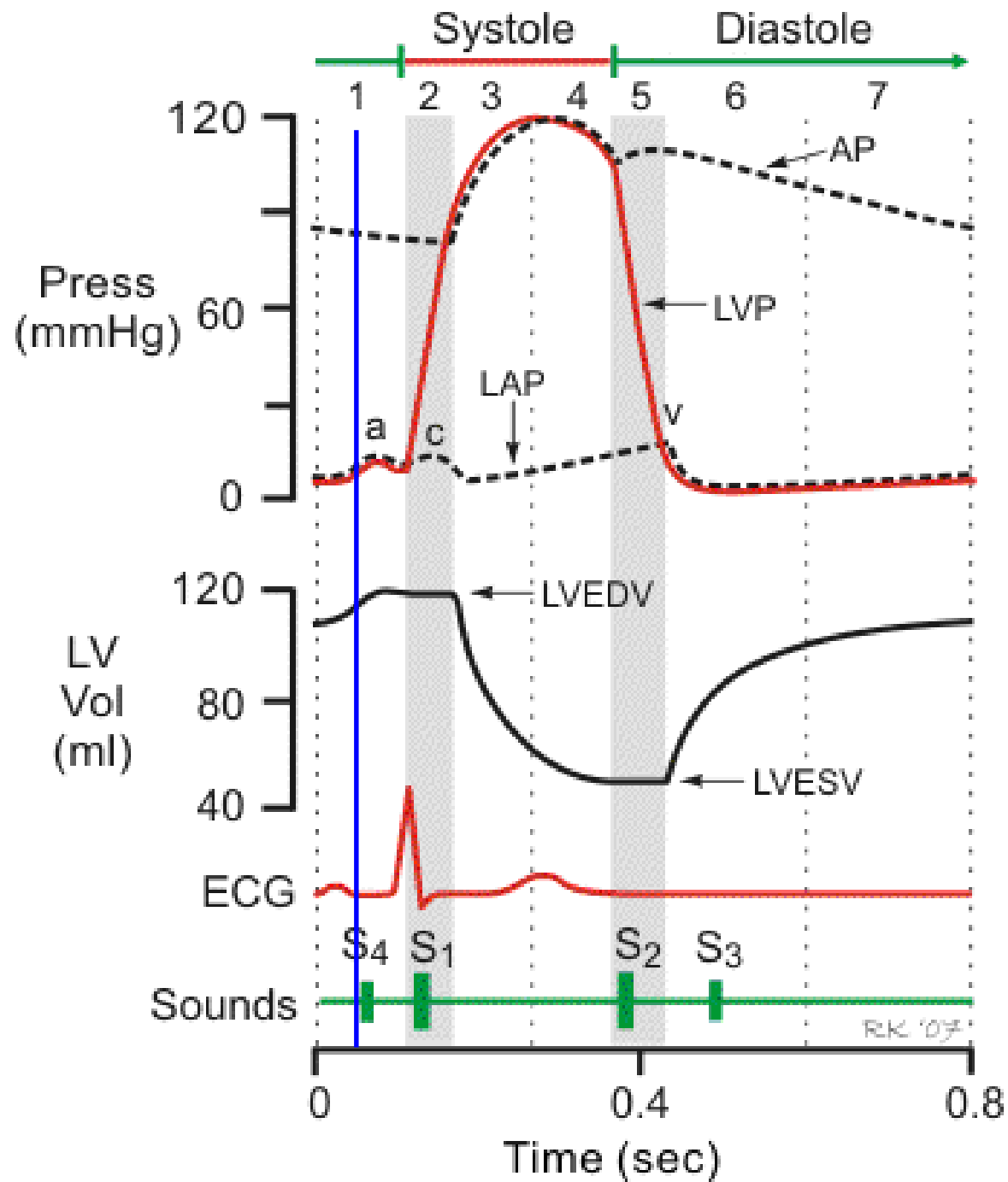
The Heart = pump

- Device pumping blood in cycles
- Filling (diastole) / ejecting (systole)
- pressure / volume work
- isotonic } contraction
- isometric } relaxation

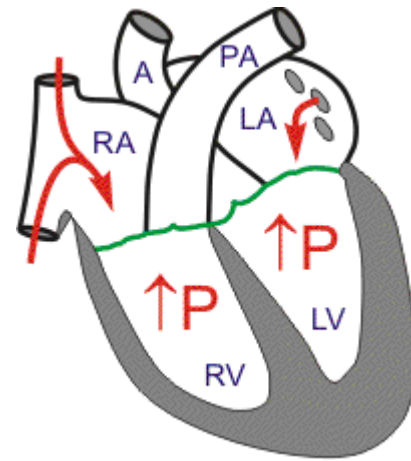
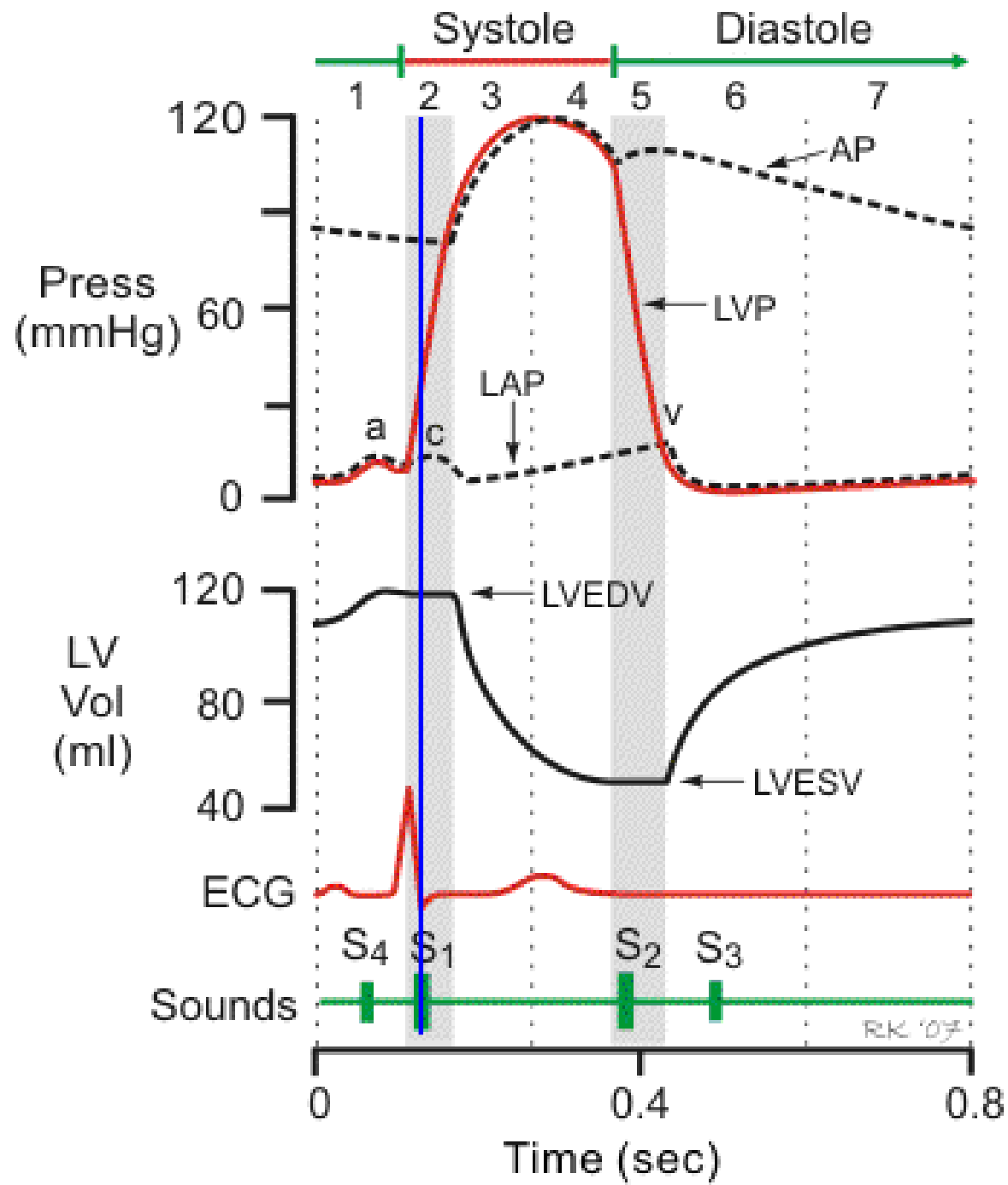


The Heart Cycle

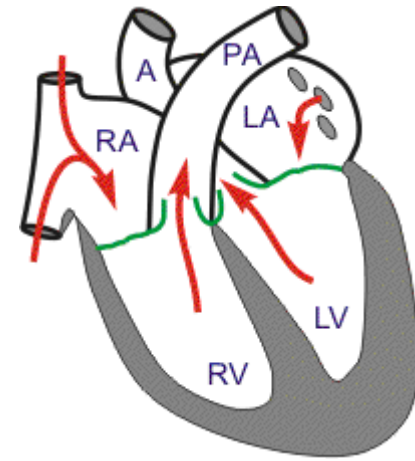
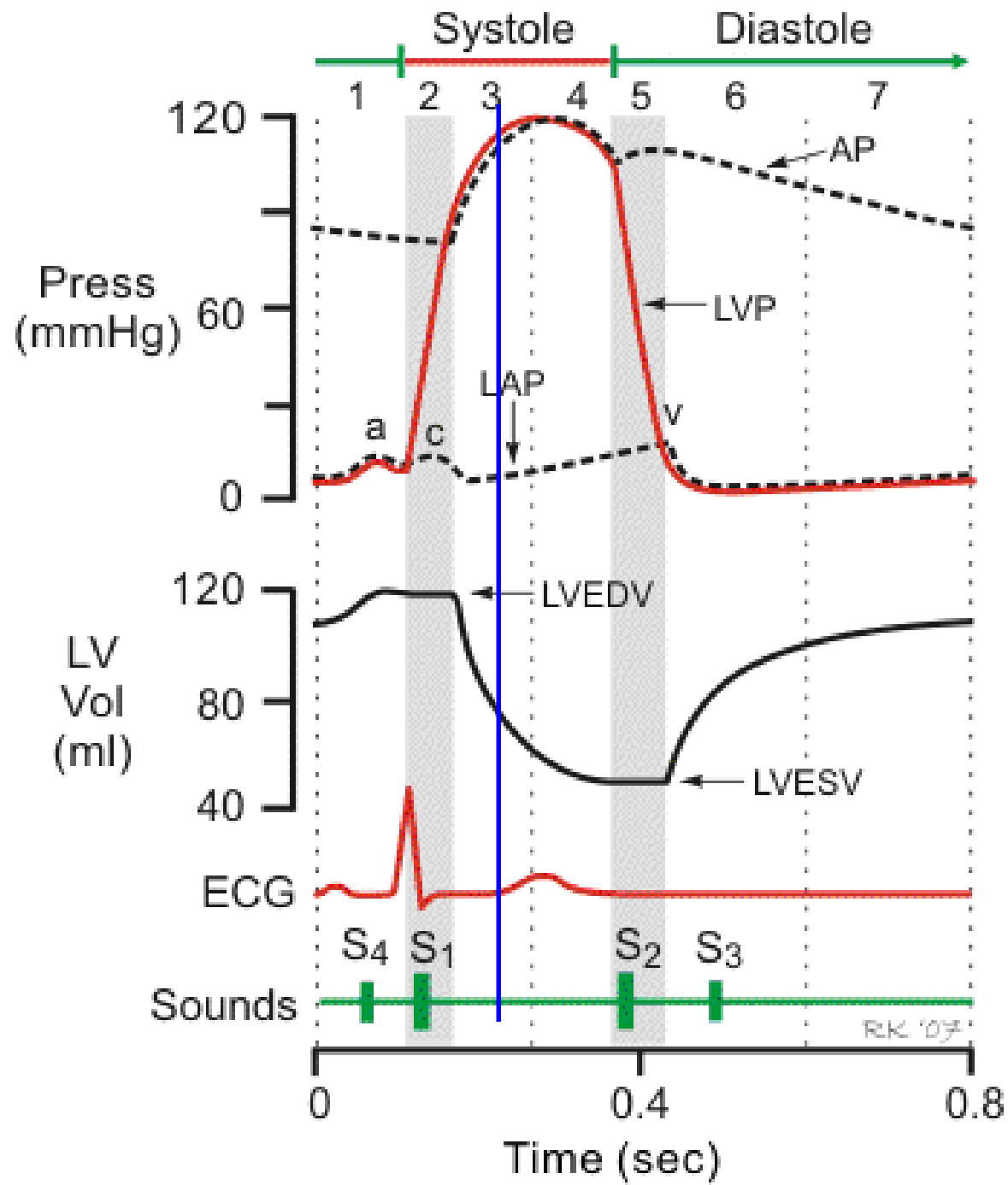
- Atrial contraction
- isovolumetric ventricular
- Ejection
- Isovolumetric ventricular relaxation
- Passive ventricular filling



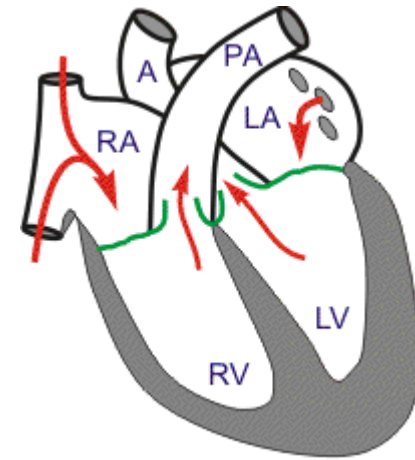
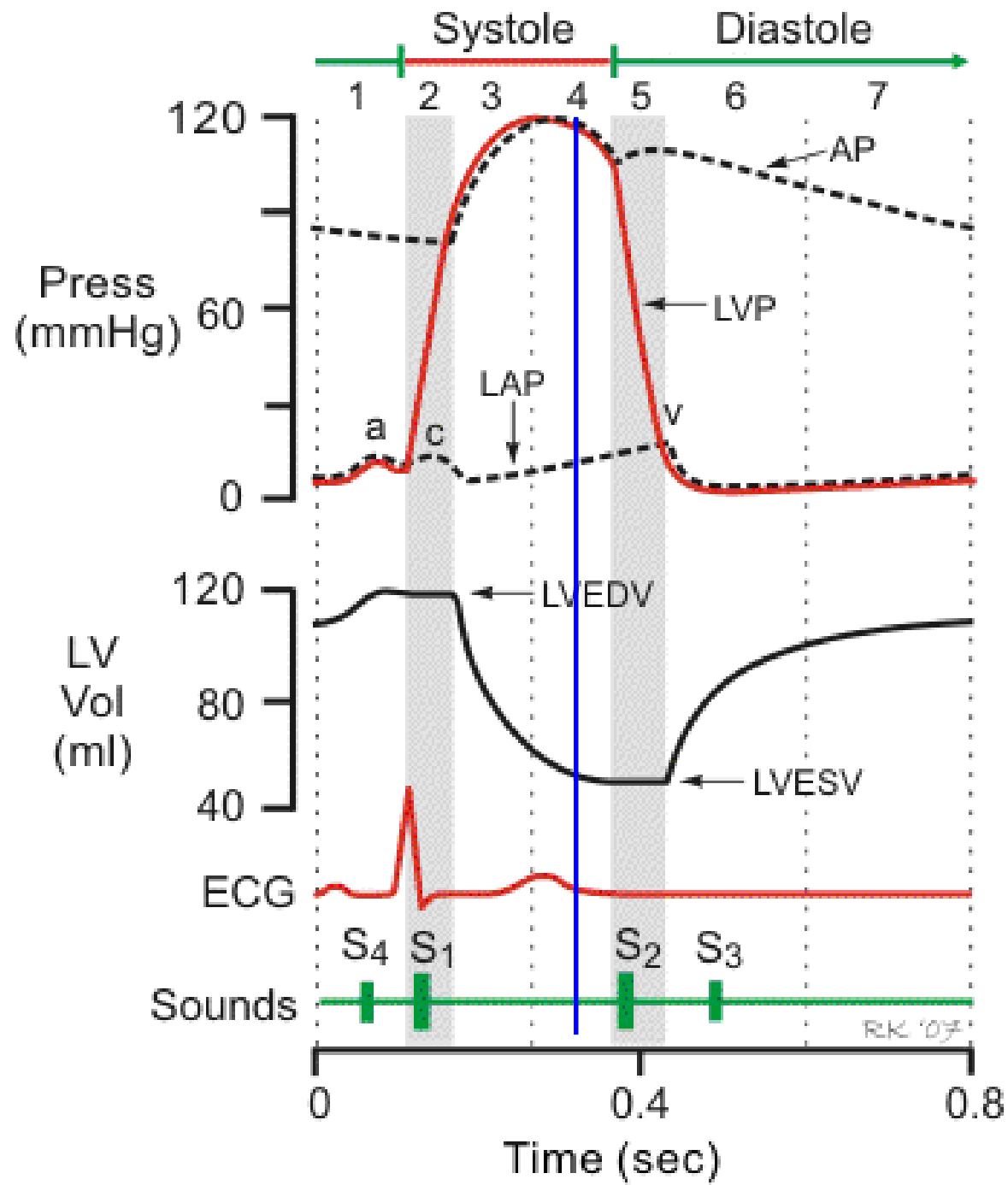
1. Atrial contraction



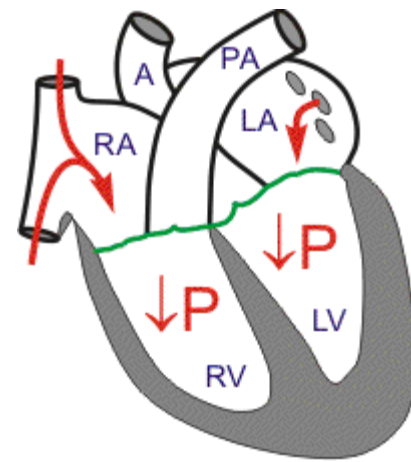
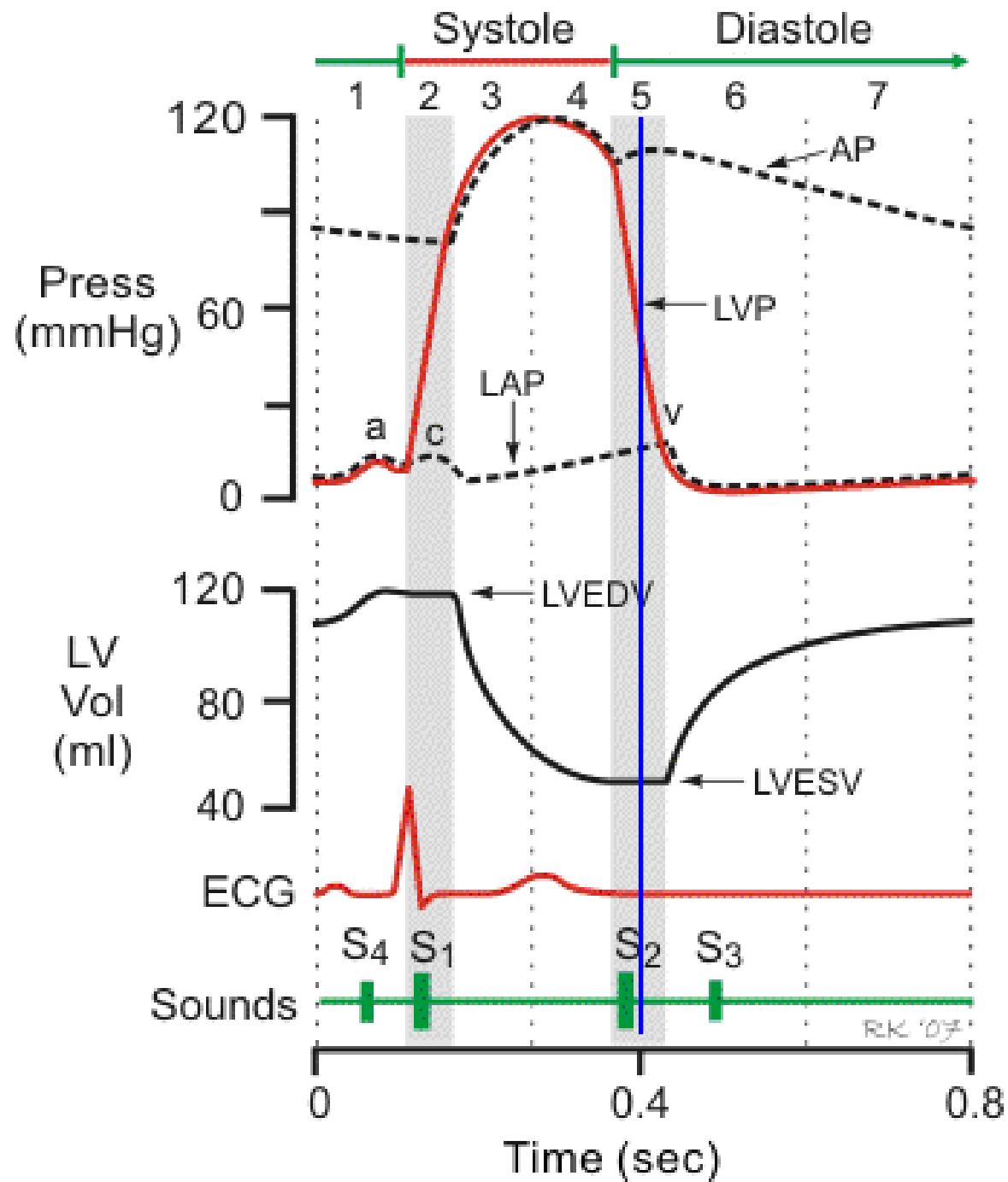
2. Isovolumetric contraction



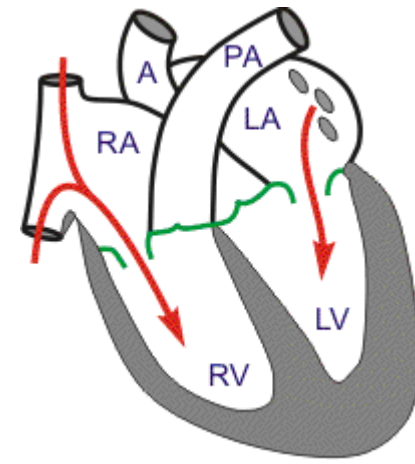
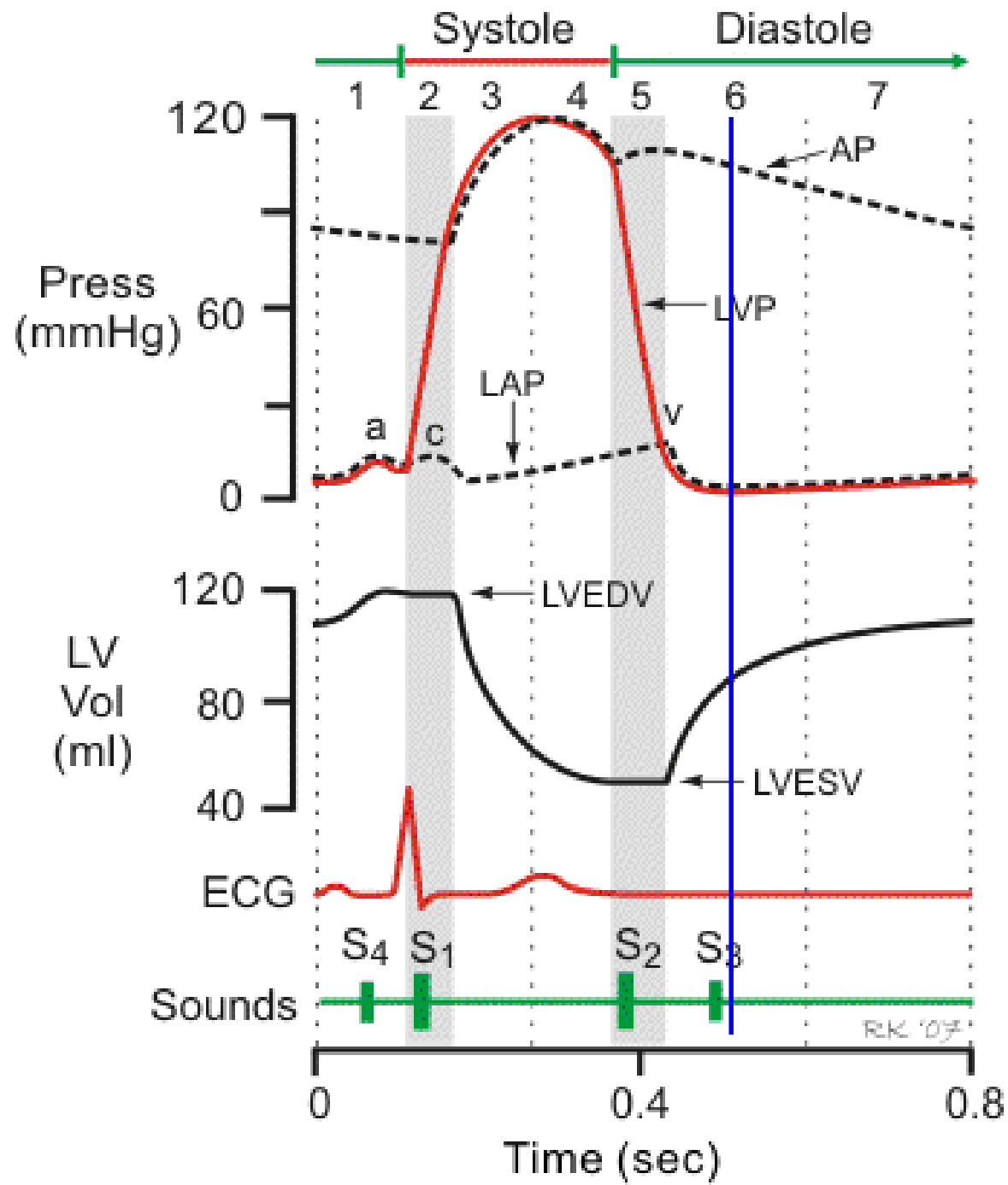
3. Fast ejection



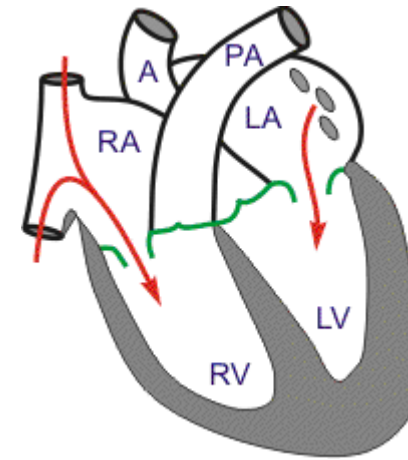
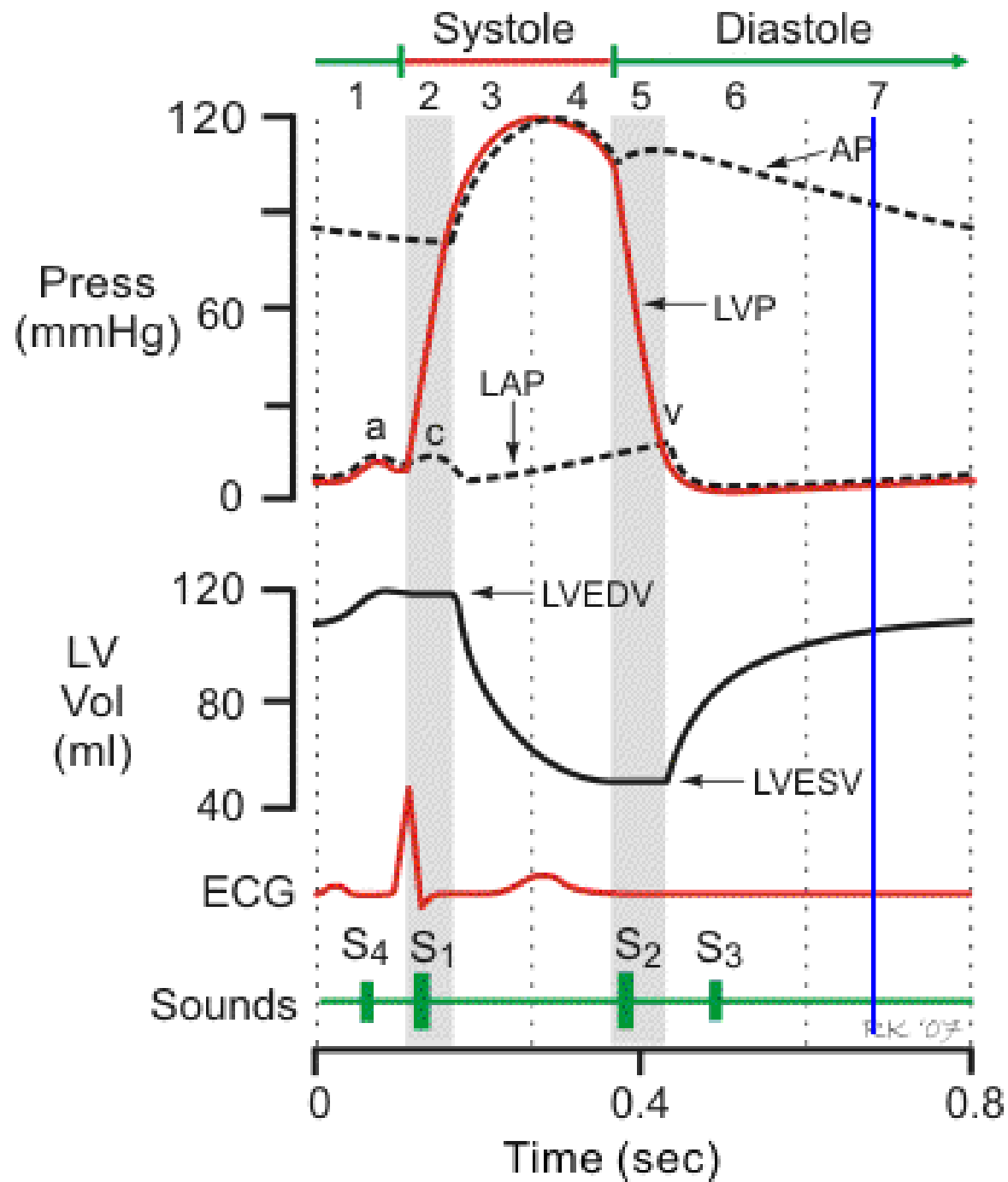
4. Slow ejection



5. Isovolumetric relaxation

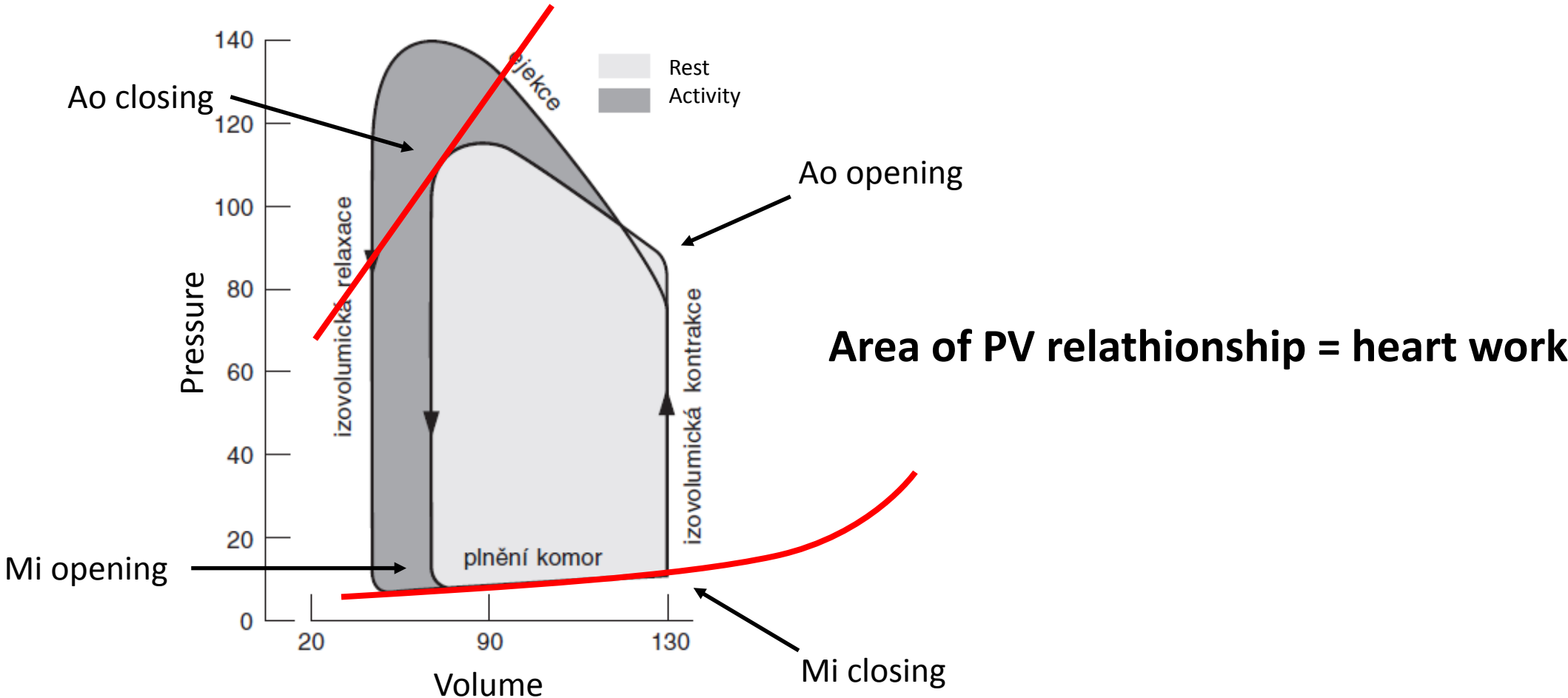


6. Fast filling



7. Slow filling

Pressure / Volume Relationship



Cardiac Output

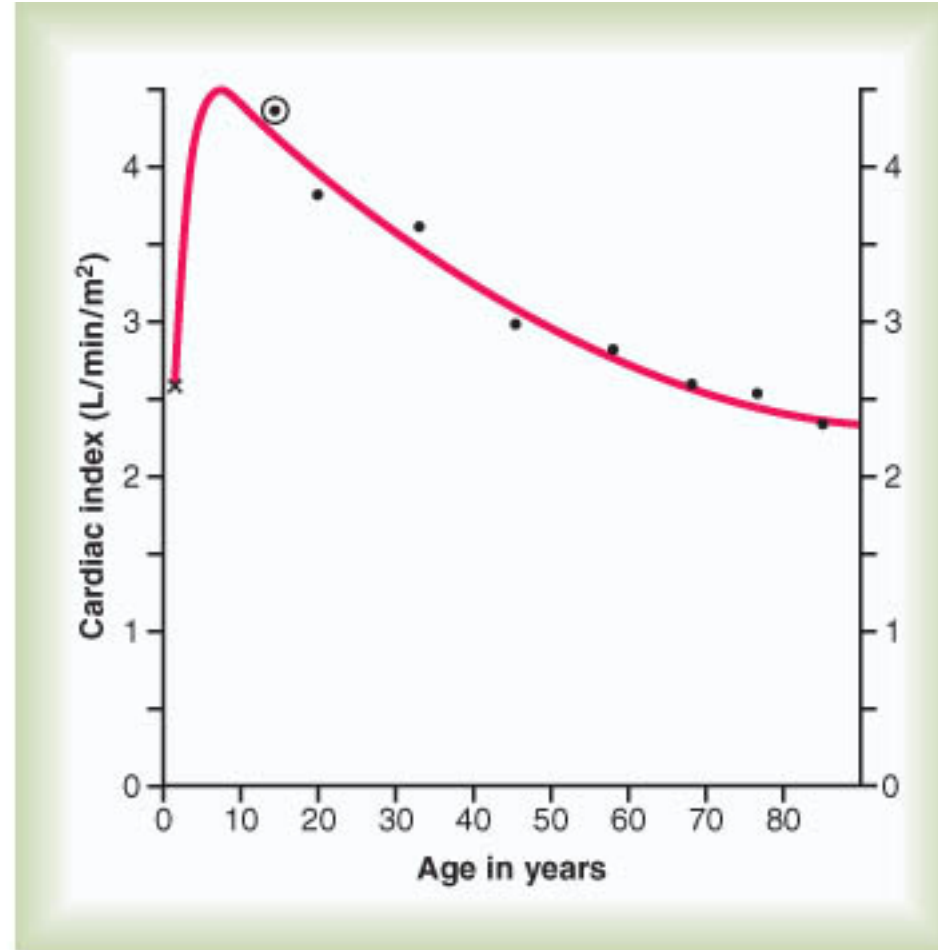
$$CO = SV \cdot HR$$

$$\text{Cardiac Index (CI)} = CO / \text{BSA}$$

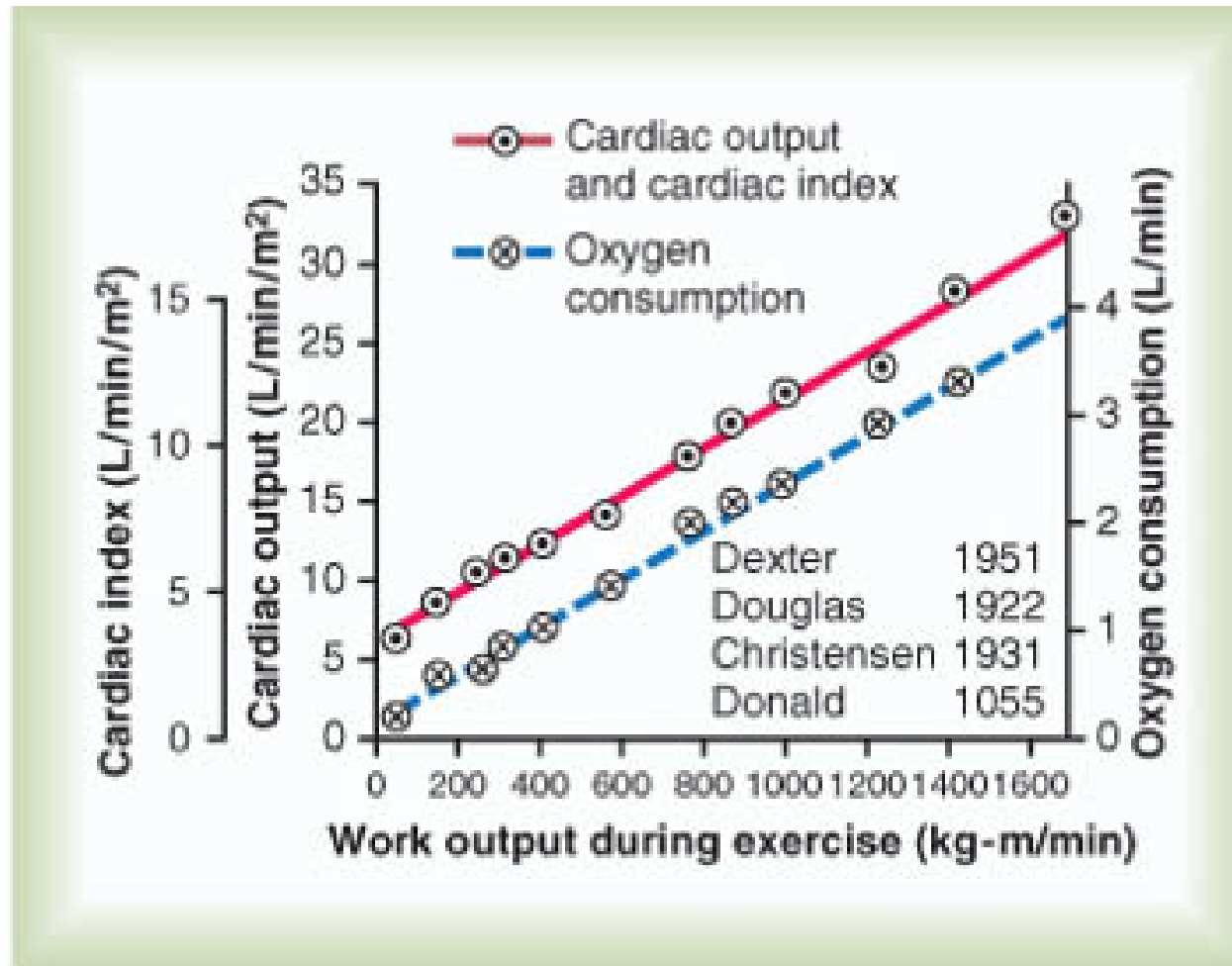
When the heart is unable to pump sufficiently to maintain blood flow to meet the body's needs = the heart failure

Increasing of the heart rate leads to shortening of diastole (filling). Since some the heart rate more increase of HR leads to decreasing of CO!!!

Relationship between CO and age



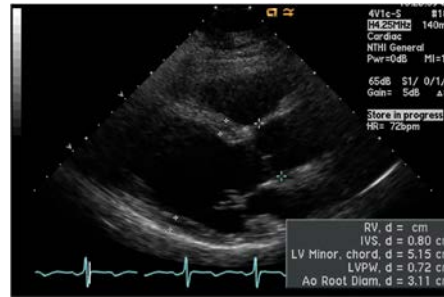
Relationship between CO and exercise



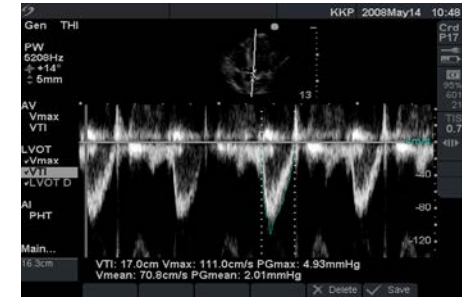
Measurement of CO

- Echo:

Diameter of LVOT= calc.of area of LVOT

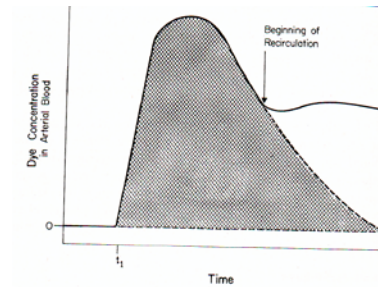


Velocity of flow in LVOT = VTI in LVOT

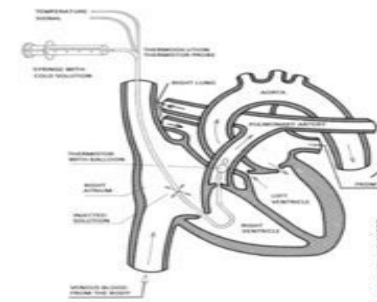


- Diluting methods:

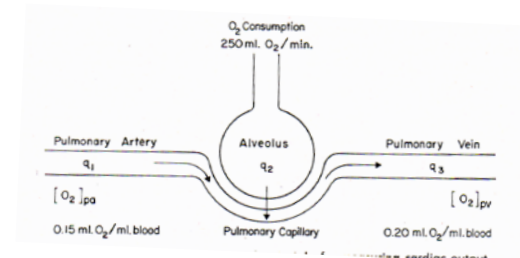
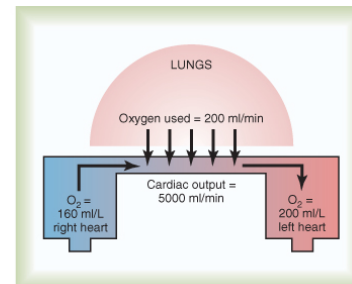
Dye dilution – experiments



Thermodilution – common in critical ill patients



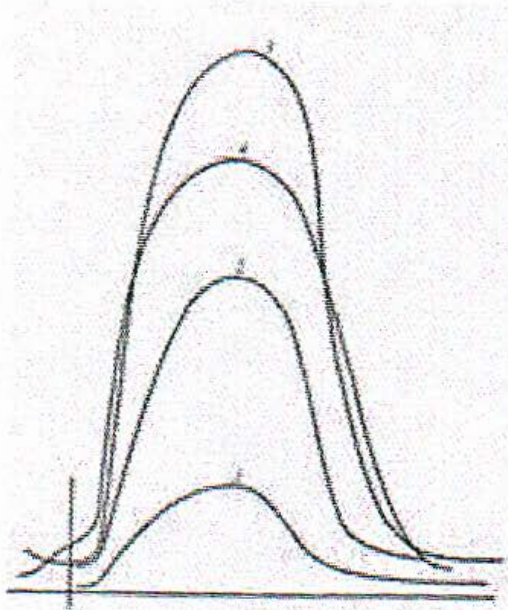
- Fick principle:



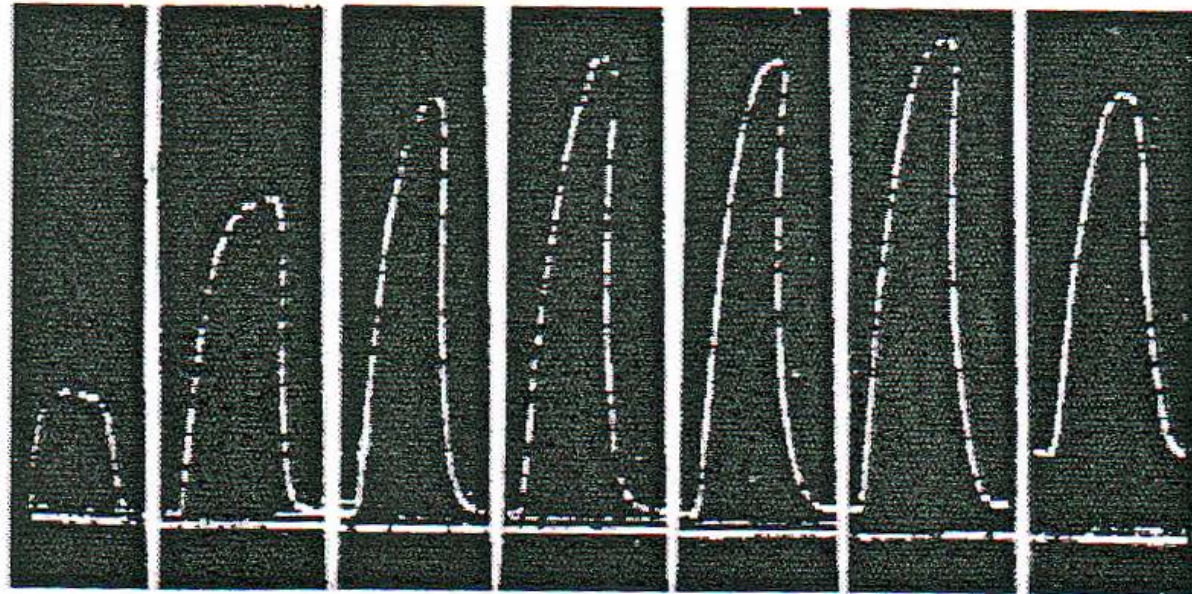
Preload

- Tension of contraction increases on the length of sarcomere at end of diastole
- Increased filling leads to faster and stronger contraction
- Venous return, CVP
- HETEROMETRIC REGULATION OF CONTRACTION
- Frank-Starling relationship

Frank-Starling



Frank (frog, 1895)



0.5

1.0

1.5

2.0

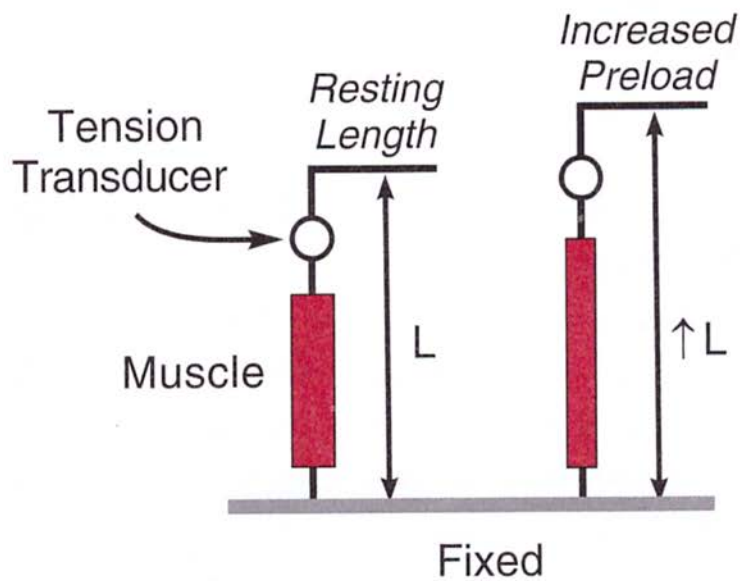
2.5

3.0

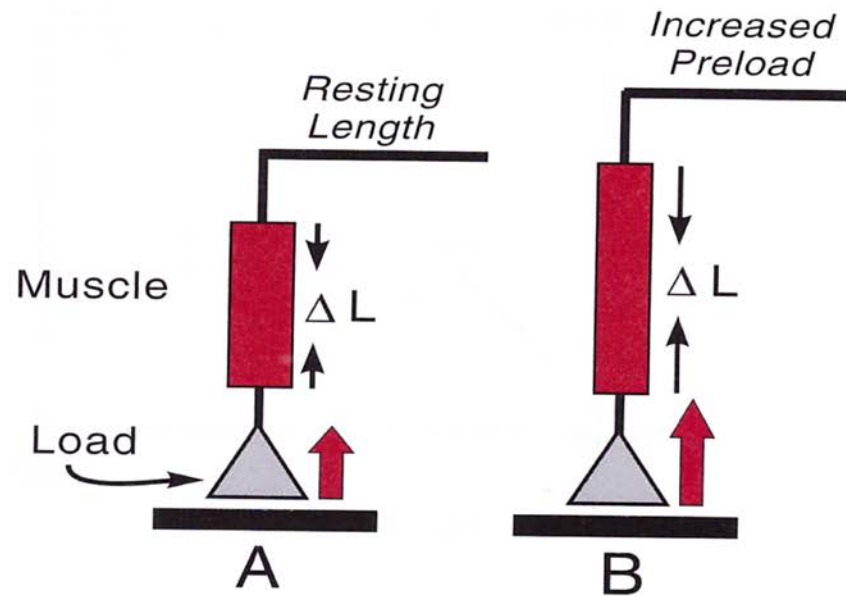
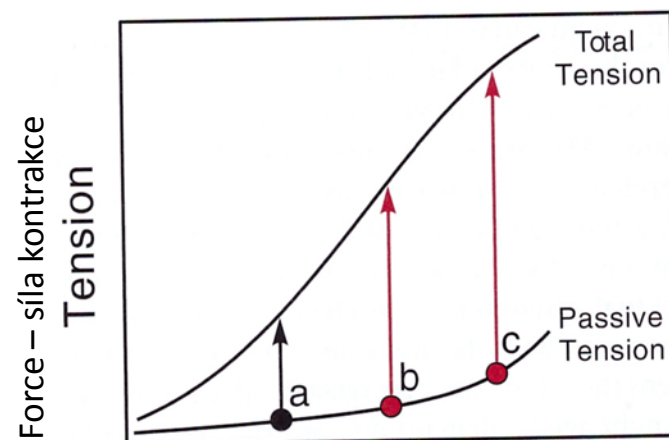
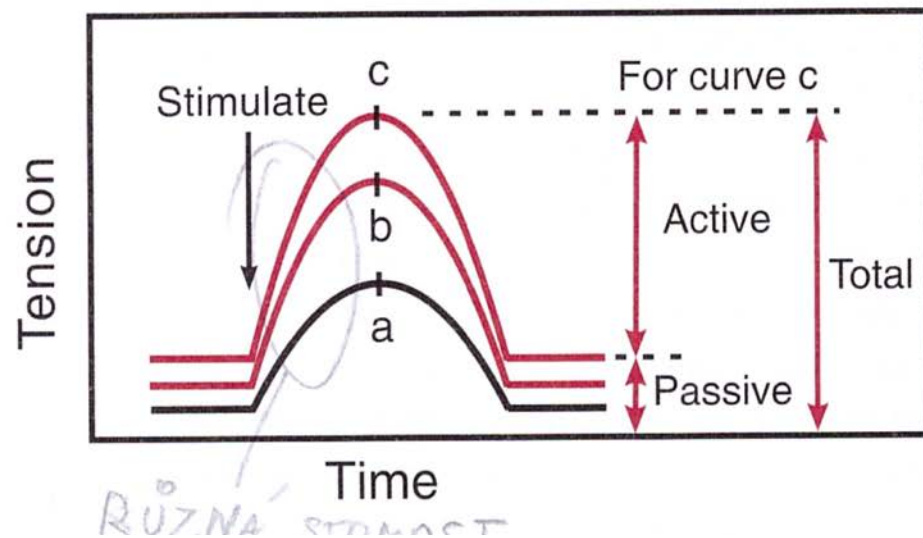
4.0 cc

Starling (dog, 1914)

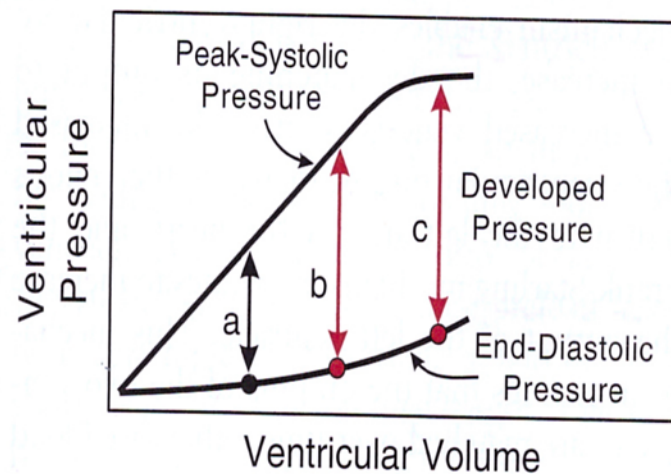
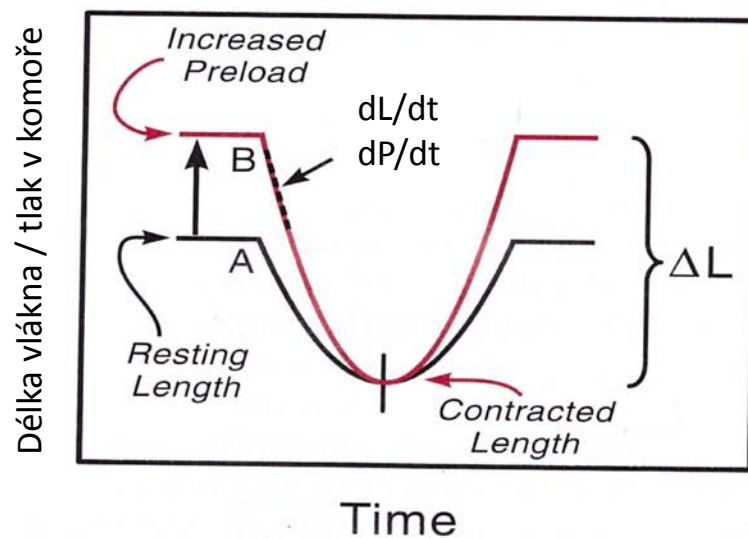
“Experiments carried out in this laboratory have shown that in an isolated heart [...] (within physiological limits) the larger the diastolic volume [...] the greater is the energy of its contraction.”



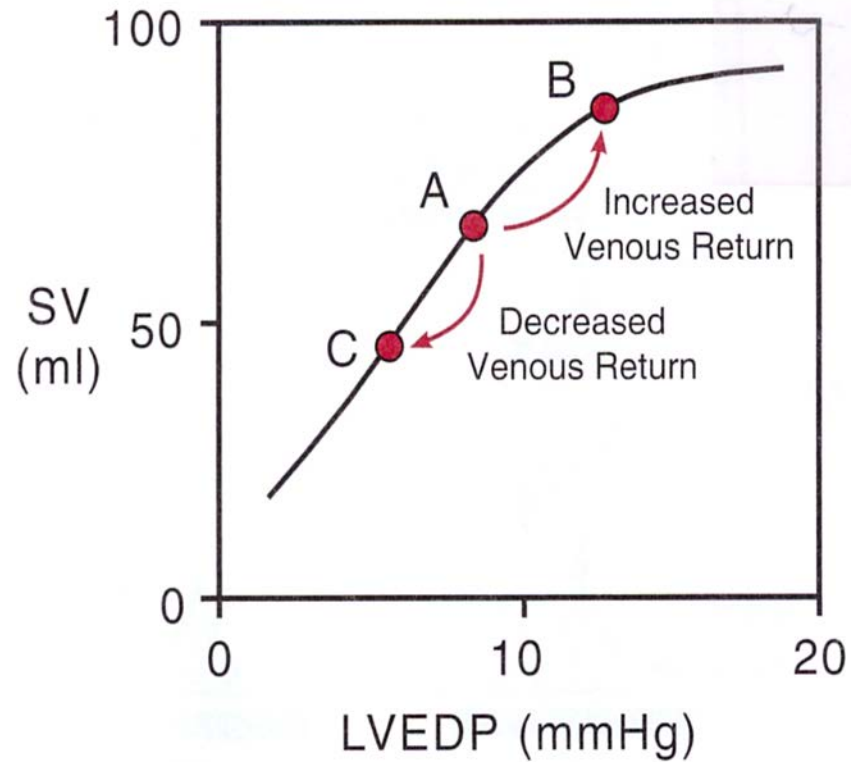
Isometric contraction



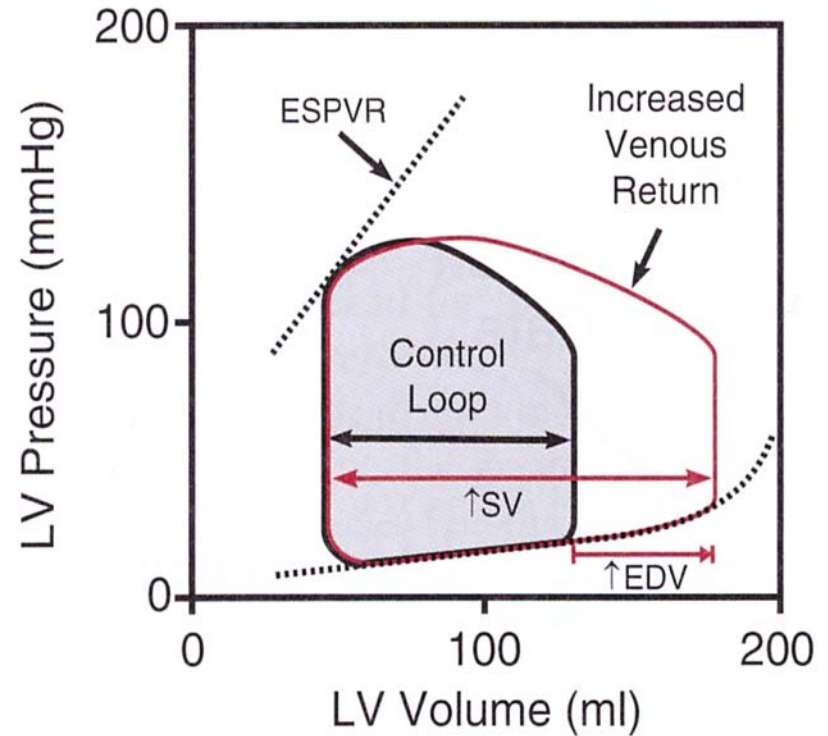
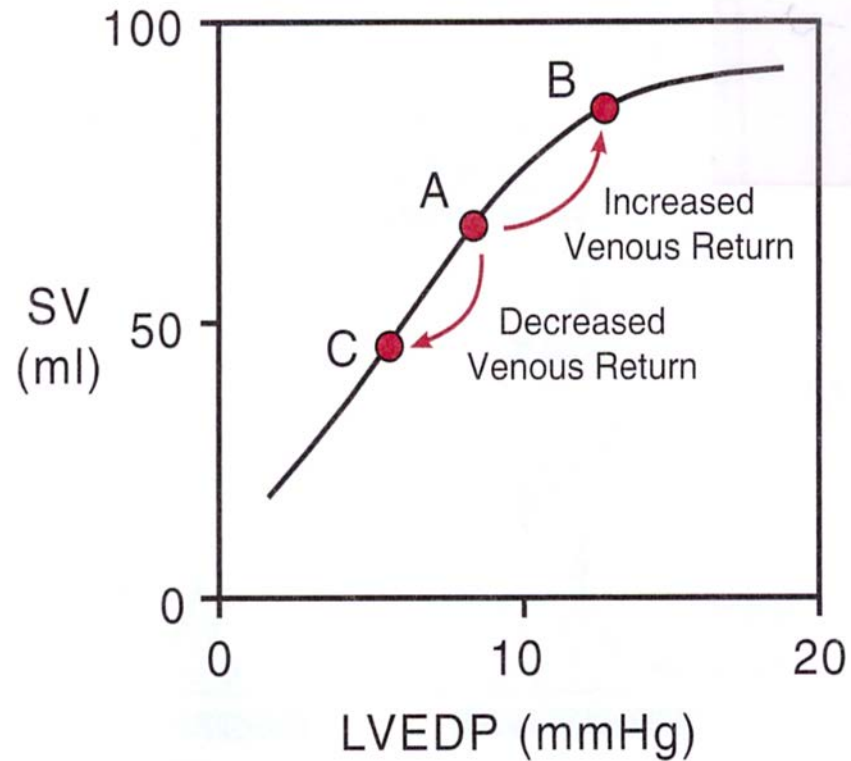
Isotonic contraction



Frank-Starling mechanism (Heterometric regulation of contraction)



Frank-Starling mechanism (Heterometric regulation of contraction)



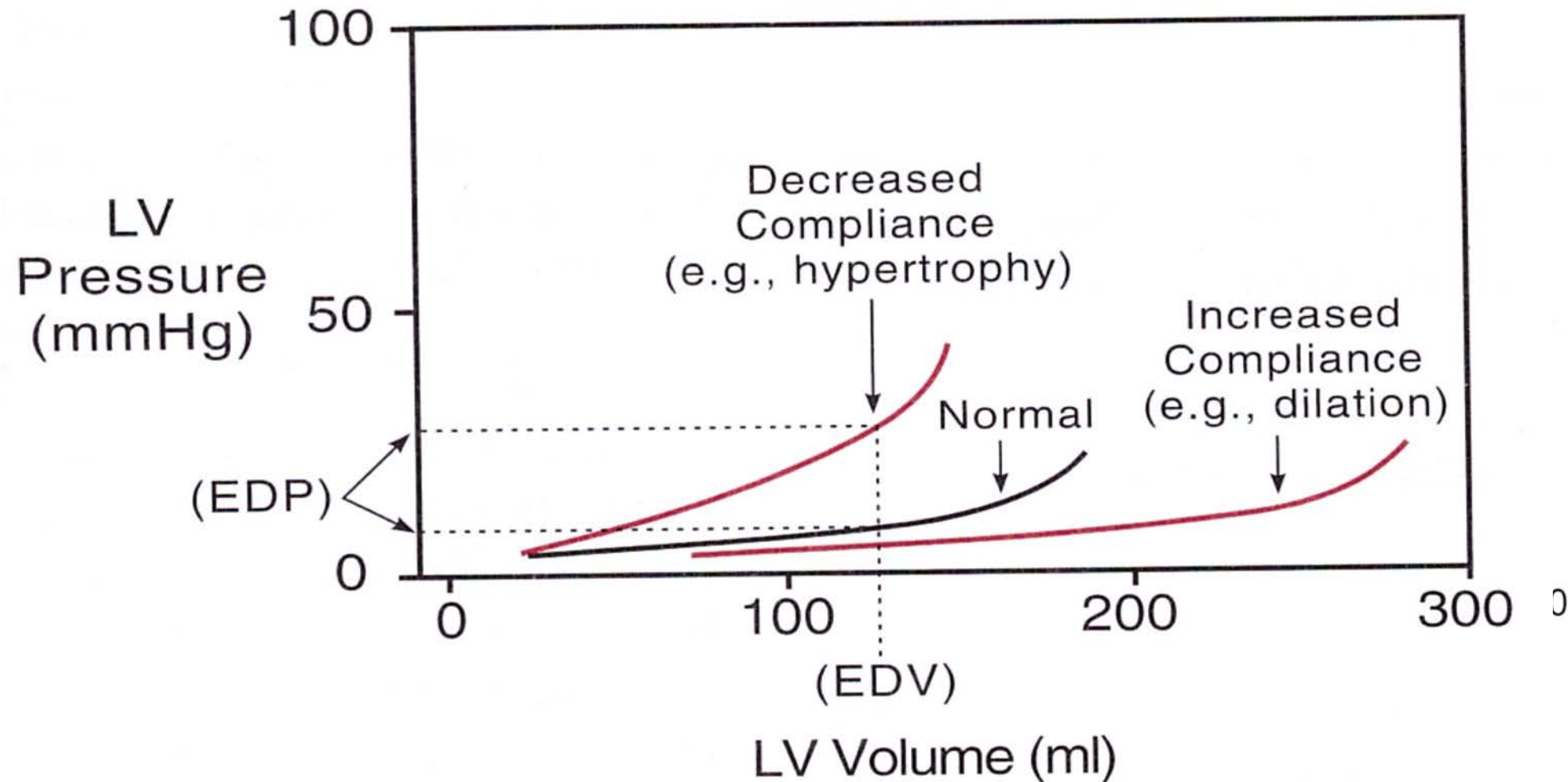
Frank-Starling mechanism

Increased filling – faster and stronger contraction, why?

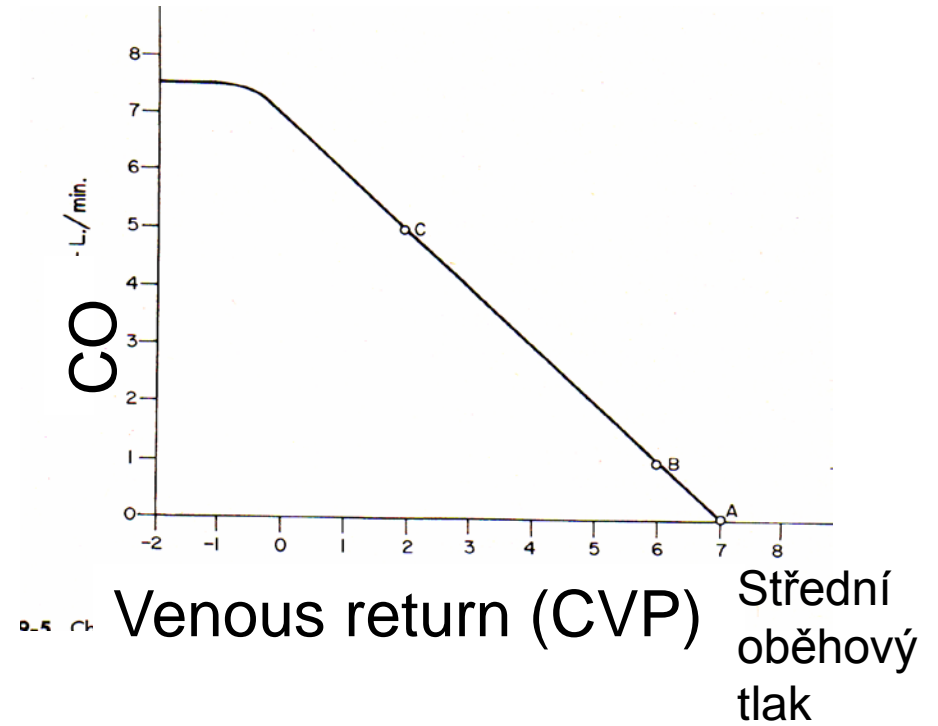
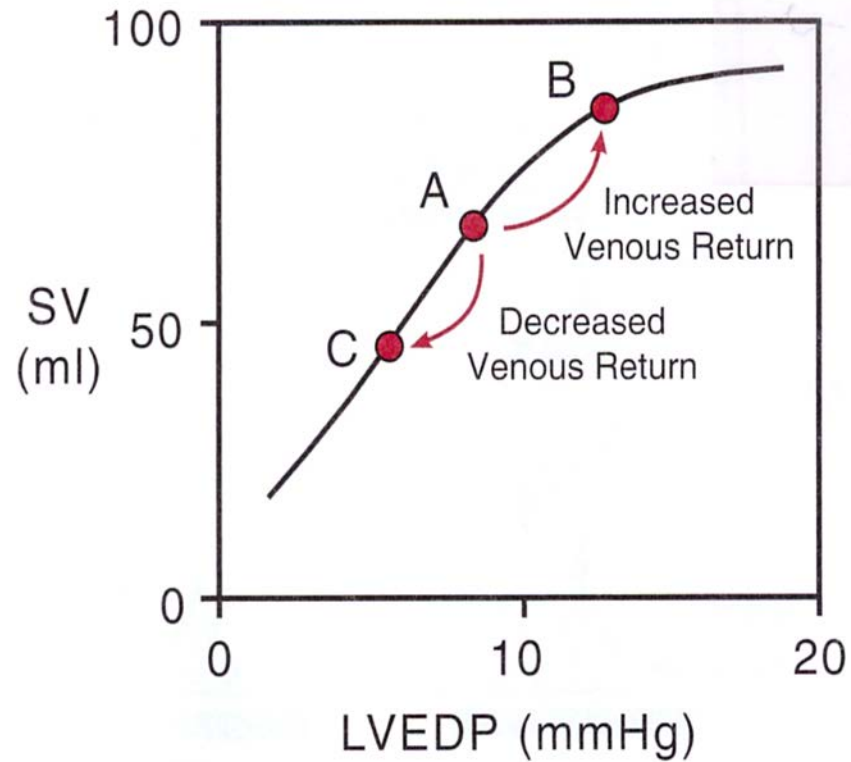
- Prolonged sarcomere:
 - More actin-myosin interactions – more ATP, more energy
 - Increased sensitivity of troponin C to Ca^{2+}
 - Increased intracellular $[\text{Ca}^{2+}]$
 - Decreased diameter of muscle fiber – actin+myozin closer together

Frank-Starling mechanism – ventricular compliance

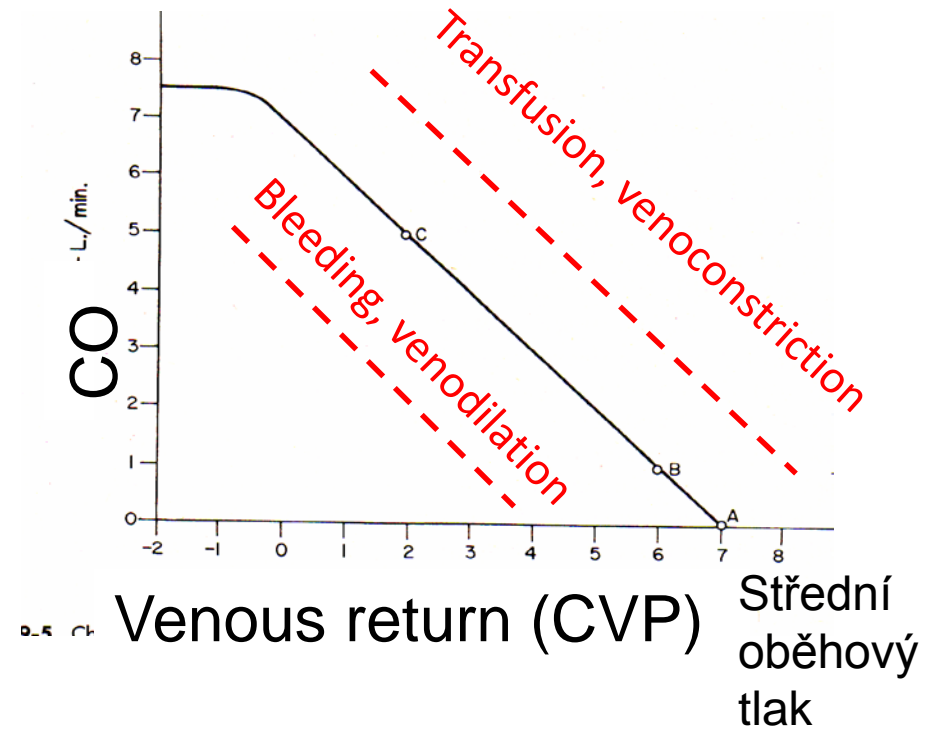
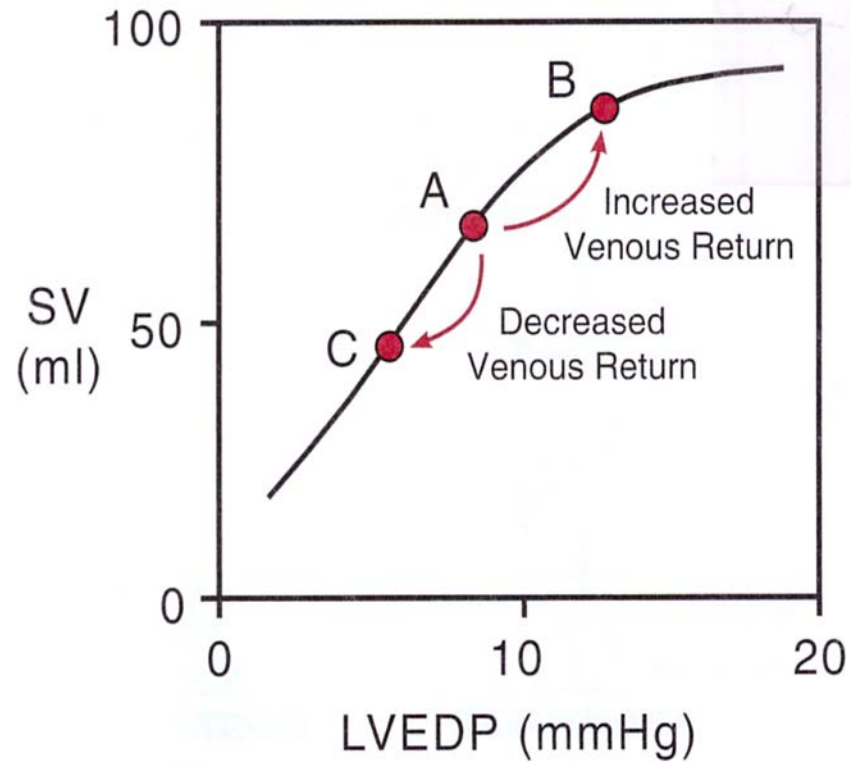
(Heterometric regulation of contraction)



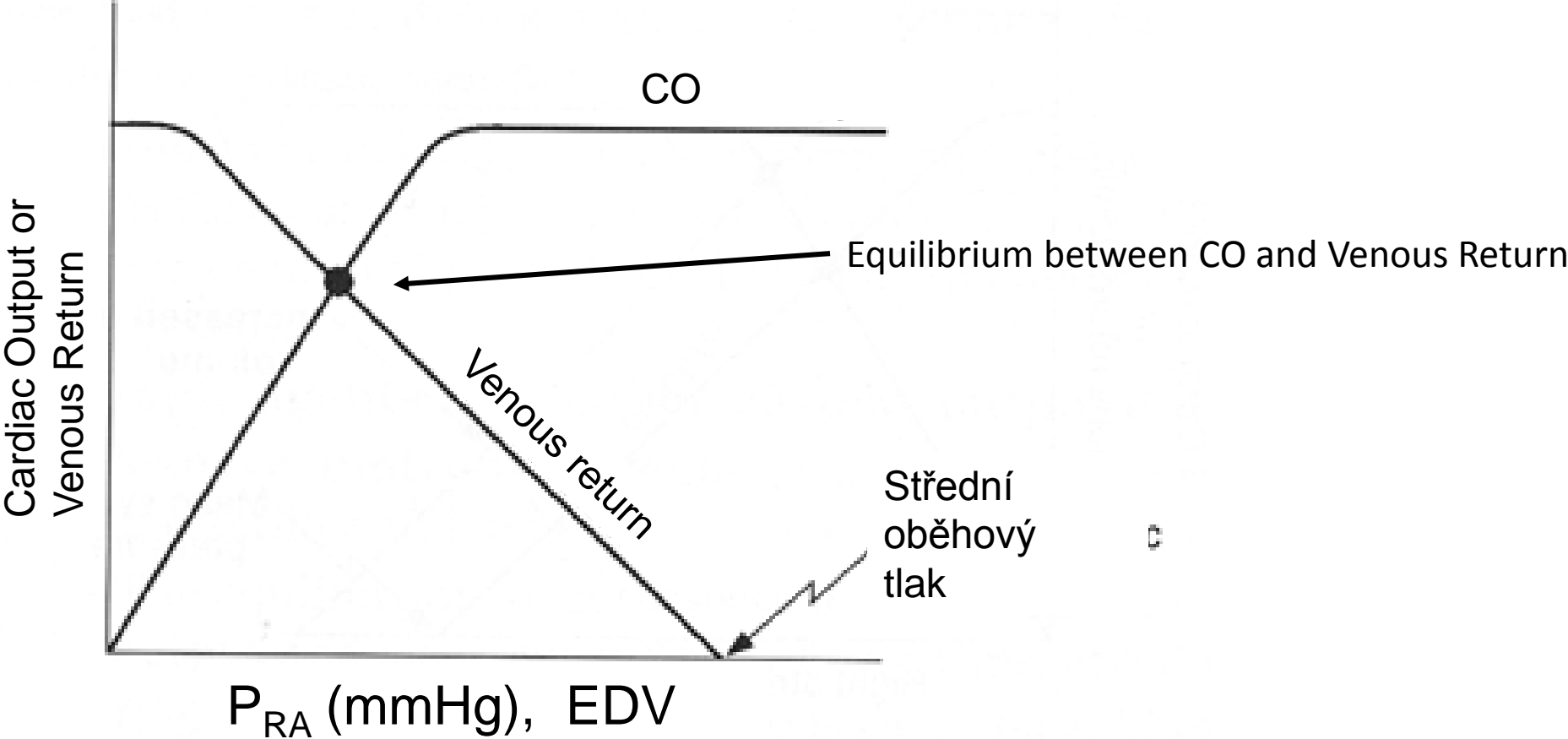
CO and Venous Return



CO and Venous Return



Relationship between CO and Venous Return



Faktors Determining Ventricular Preload

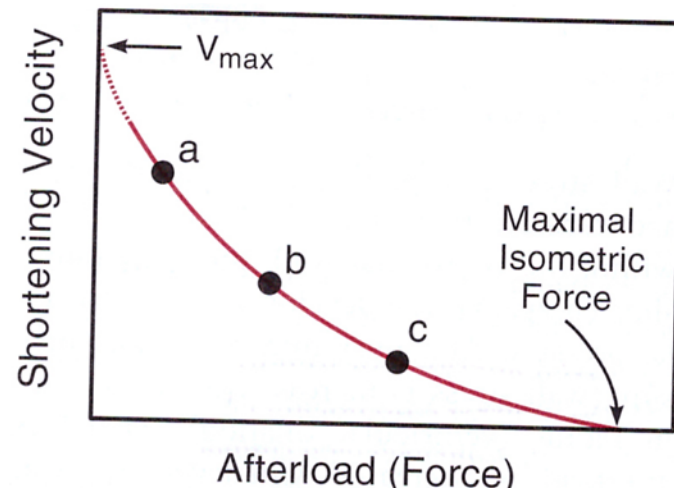
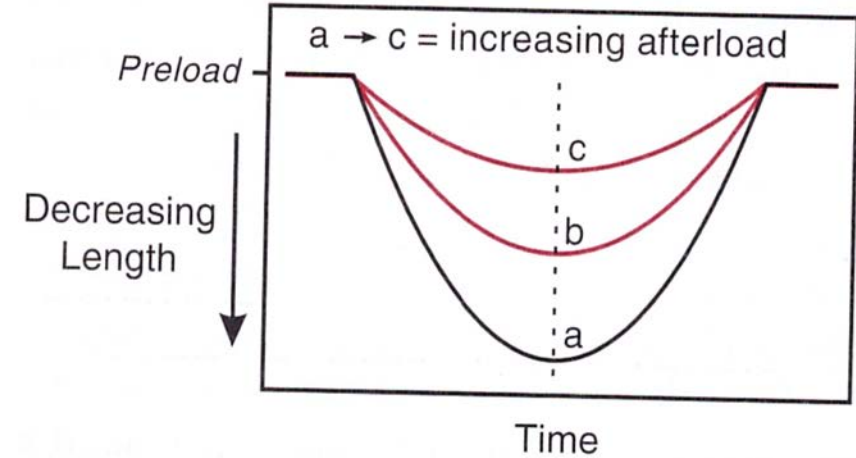
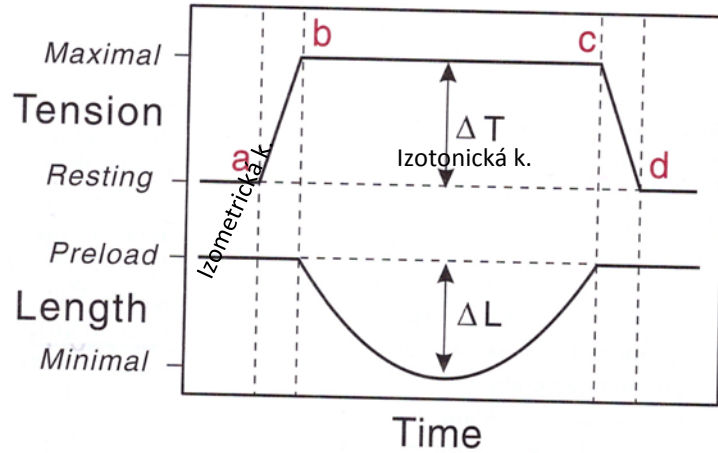
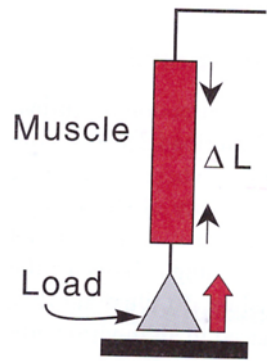
- Venous Pressure, venous return, CVP
- Ventricular compliance
- Heart Rate – filling time, time of diastole
- Atrial contraction – more important in tachycardia, atrial fibrillation
- Inflow resistance_ – Tri stenosis, Mi stenosis
- Outflow resistance_ – PAP, Pu stenosis, hypertension, Ao stenosis
- Ventricular contractility – decreased contractility leads to increased preload

Afterload

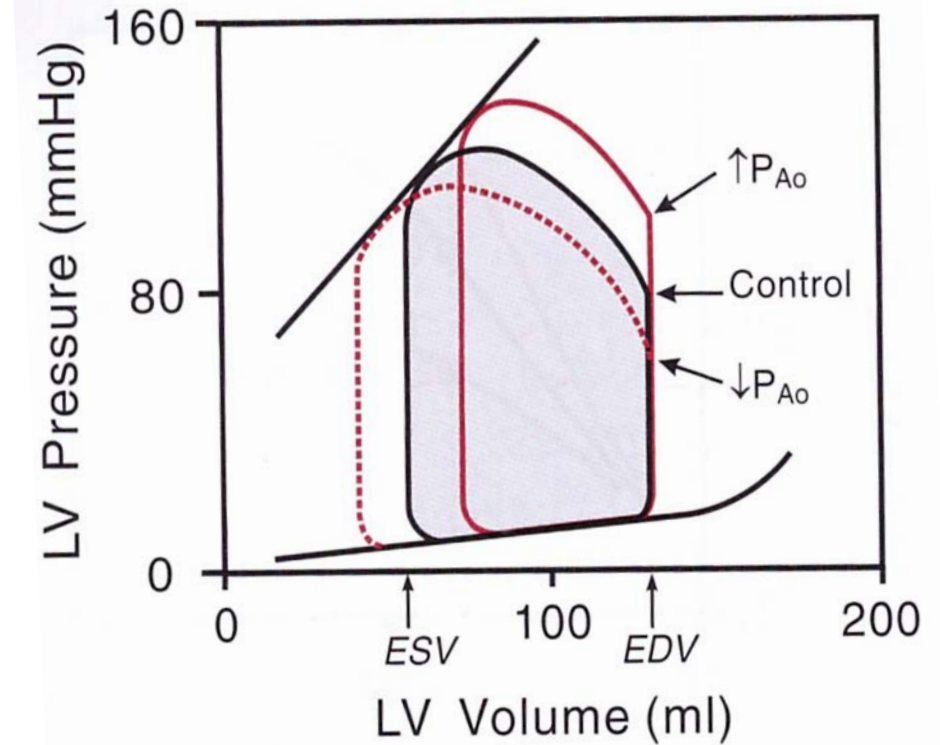
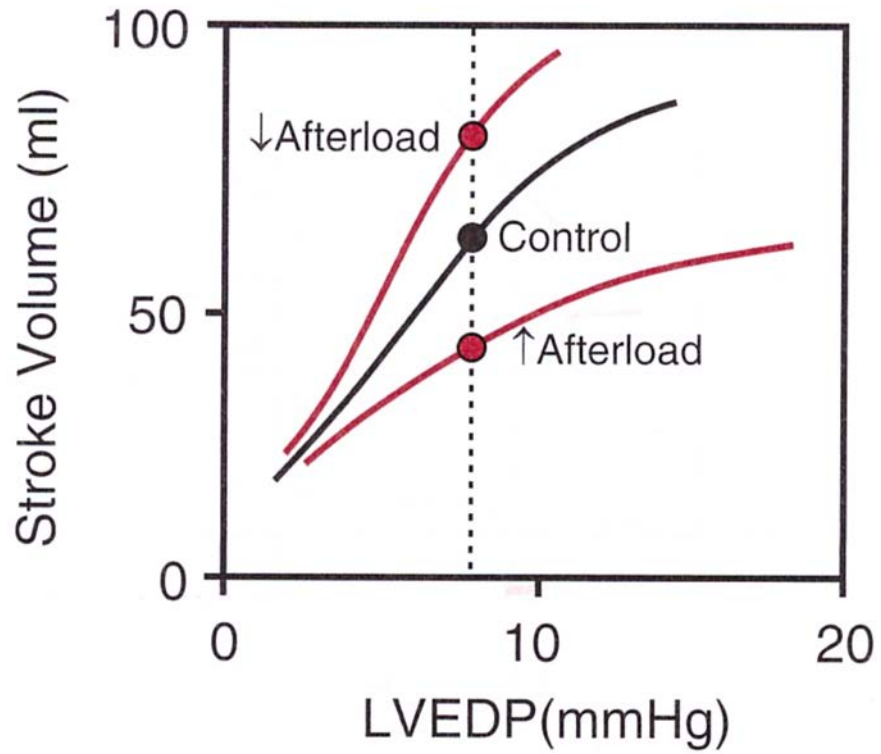
- Load against which the heart must contract to eject blood
- Characterized by SVR (small arteries, arterioles)
- Can be measured like wall stress:

$$\sigma \propto \frac{P \cdot r}{h}$$

Afterload: force / velocity relationship



Afterload

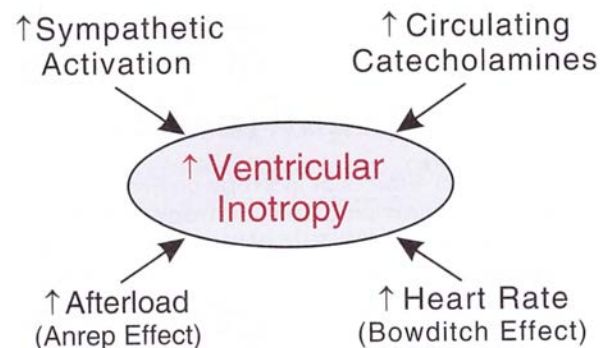


↓ CO
↑ ESV

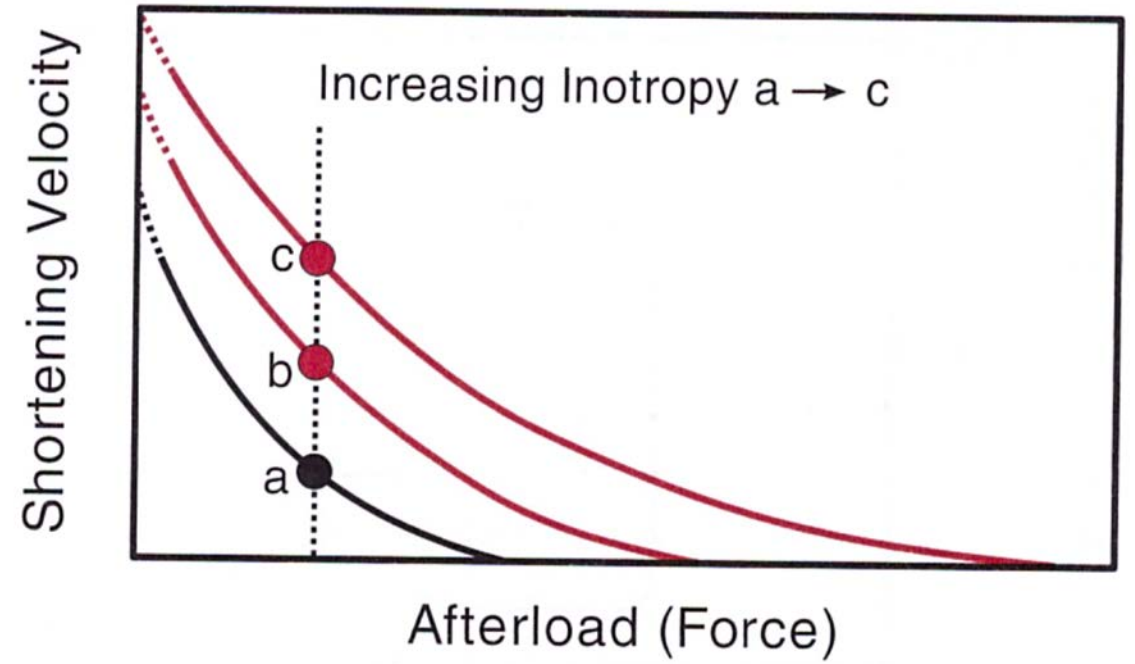
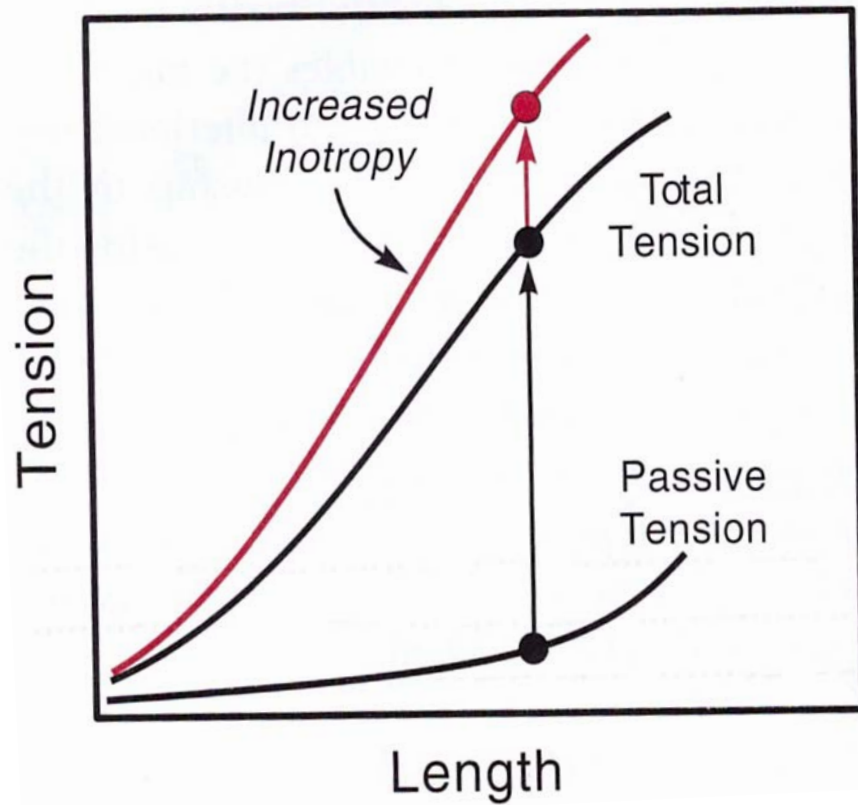
Contractility - inotropy

- Contractility (inotropy) = the force of contraction
- Sarcomere length is changing by different mechanism then changing of interaction actin-myozin:
 - Increased influx Ca^{2+}
 - Increased release of Ca^{2+} from SR
 - Increased sensitivity of troponin C to Ca^{2+}
- Independent on the length of sarcomere = HOMEOMETRIC REGULATION OF CONTRACTION

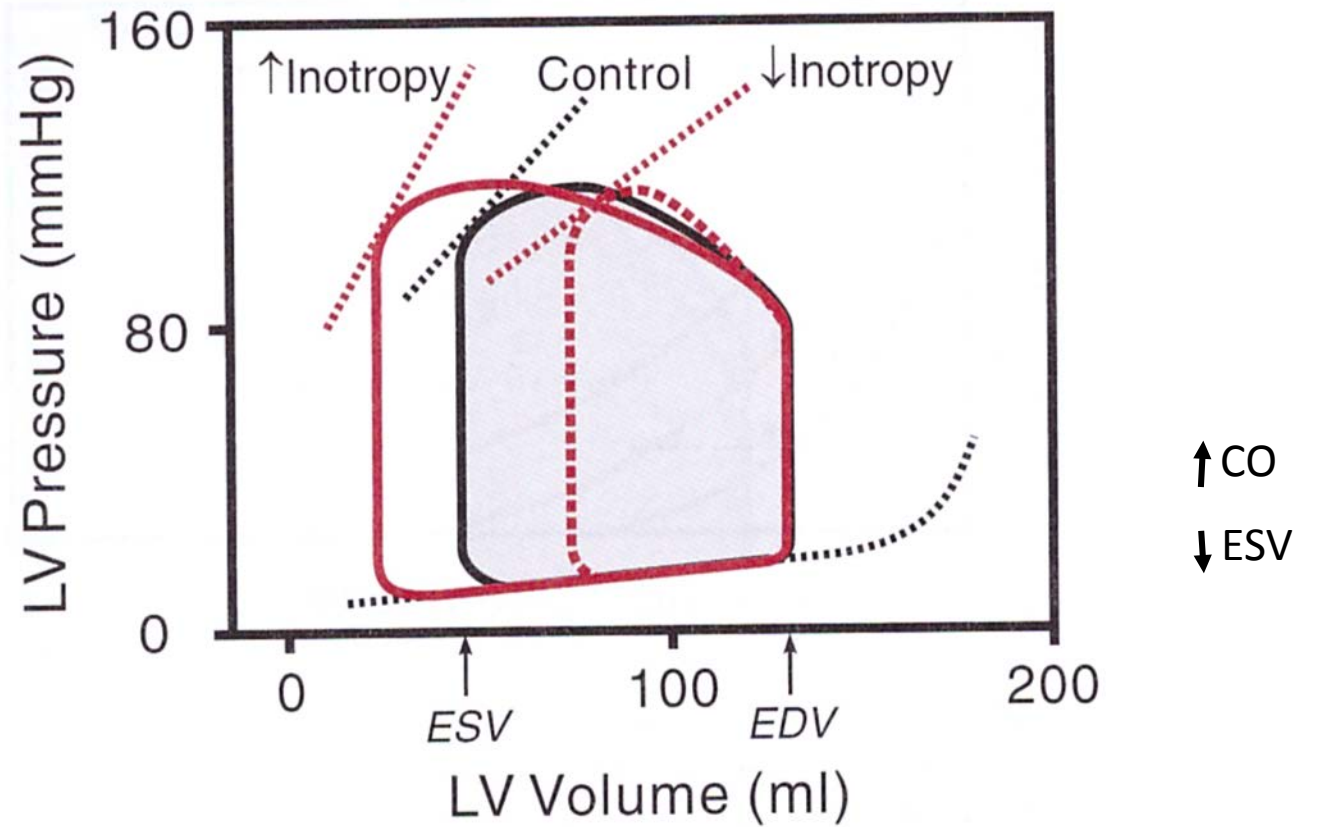
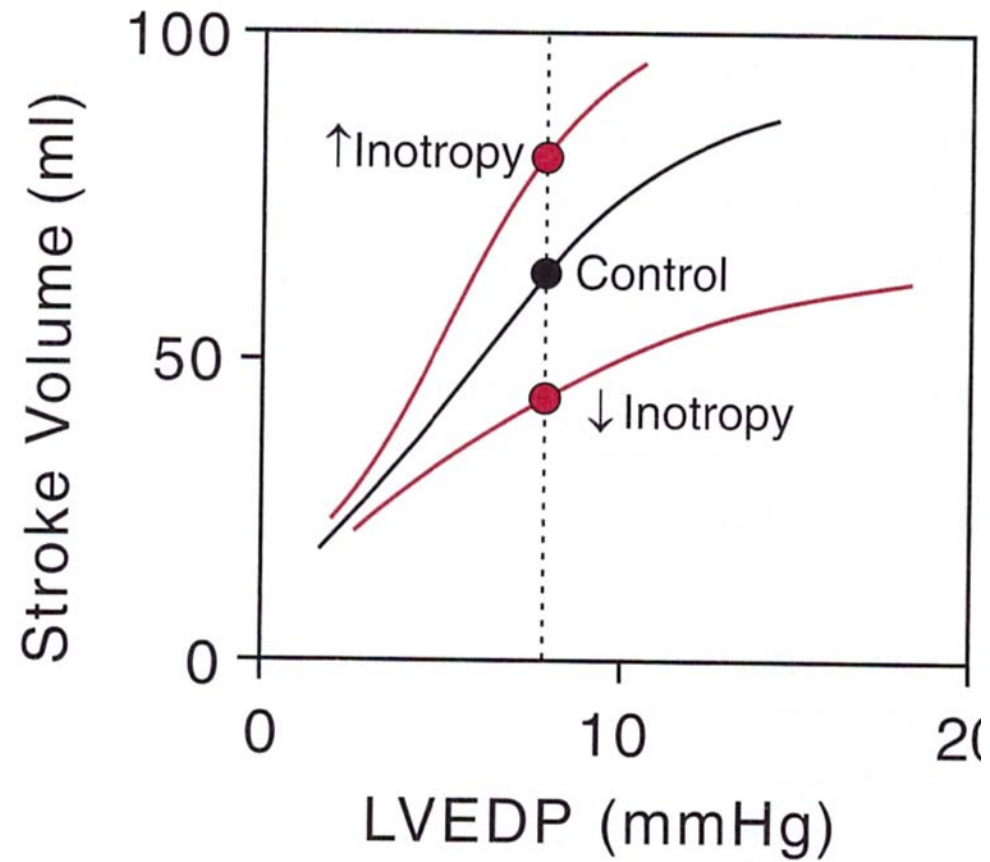
- Factors influencing inotropy:



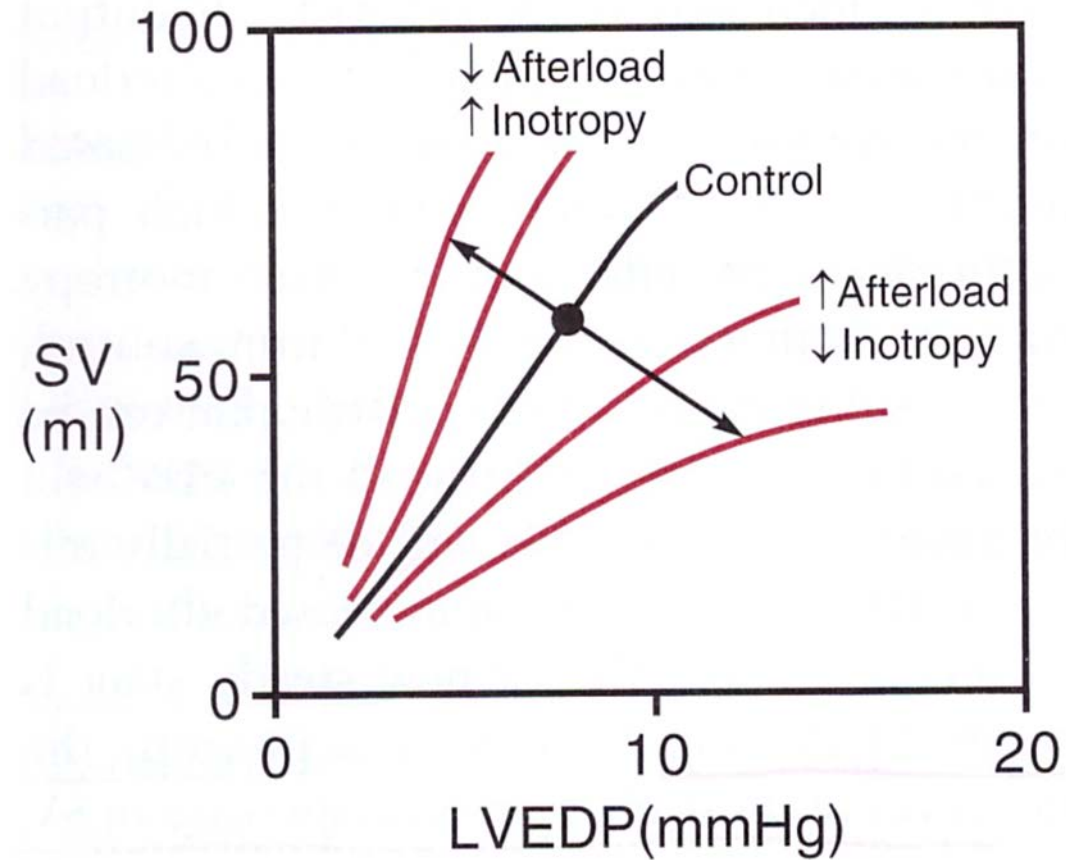
Inotropy



Inotropy



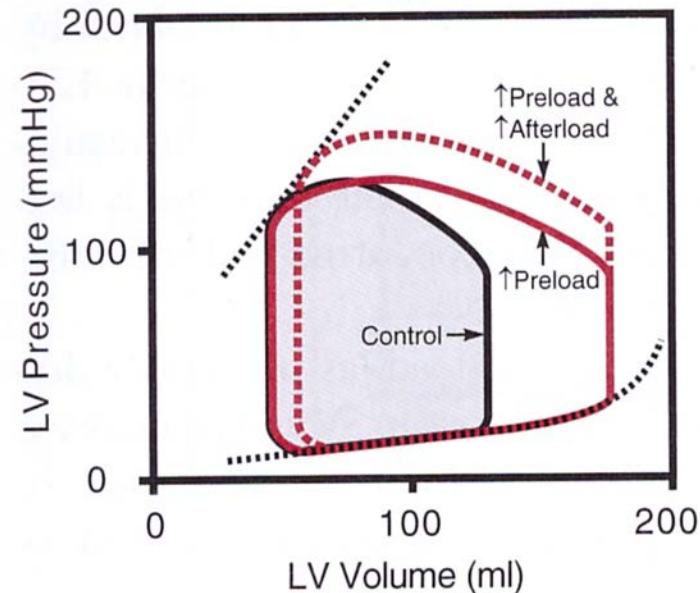
Inotropy vs. afterload



Interaction: preload, afterload, inotropy

Increased preload:

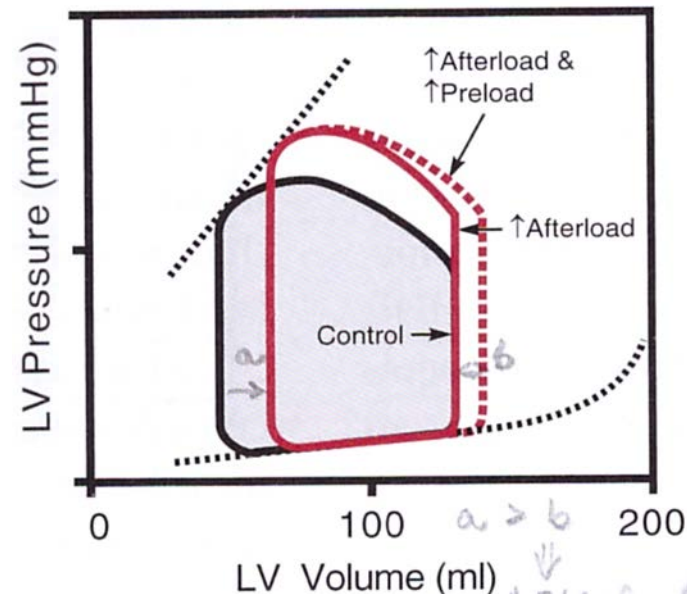
- increased SV and CO
- SVR not changed
- Primal vascular tone caused to relative increase of afterload:



Interaction: preload, afterload, inotropy

Increased afterload:

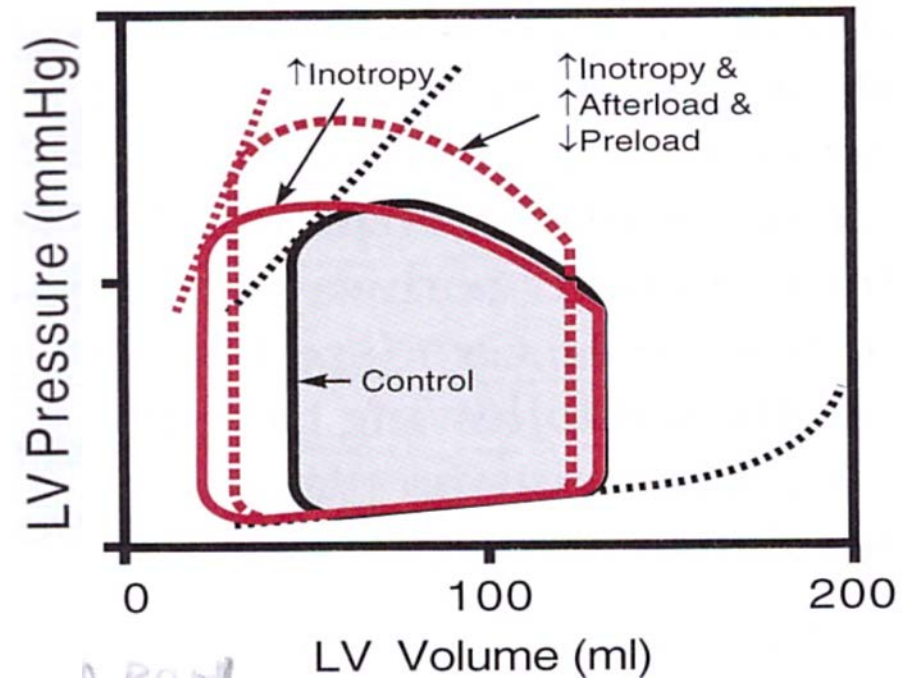
- decreased SV and CO
- decreased CO is not able pumps whole Venous Return
- Increased Venous Return leads to increase preload



Interaction: preload, afterload, inotropy

Increased inotropy:

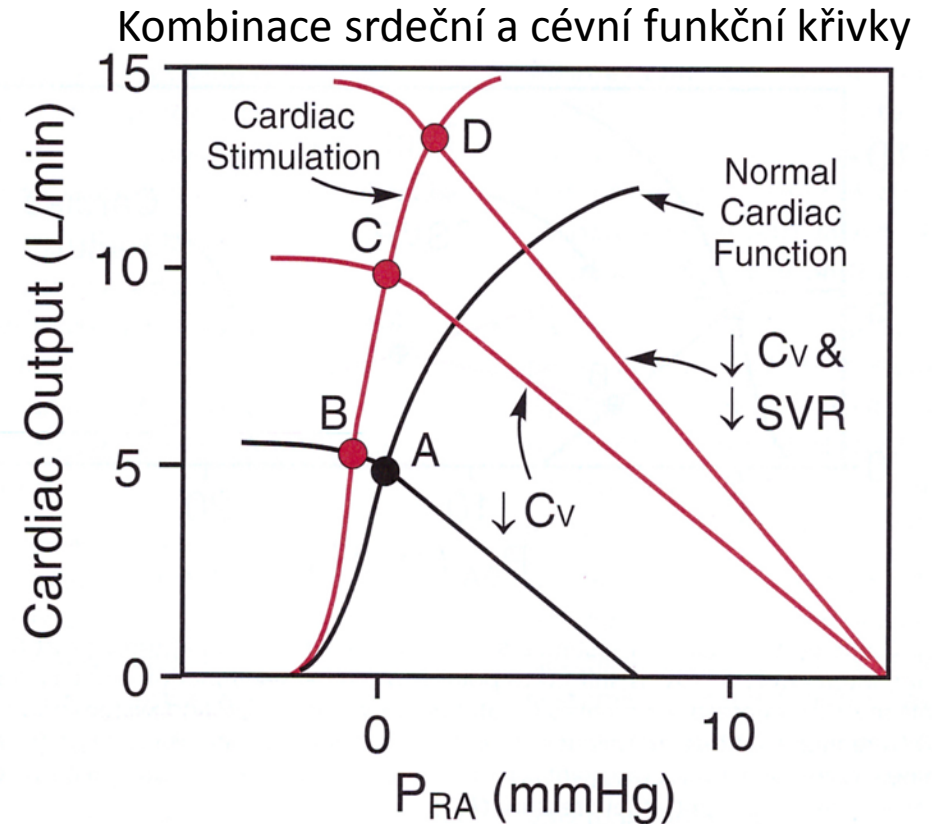
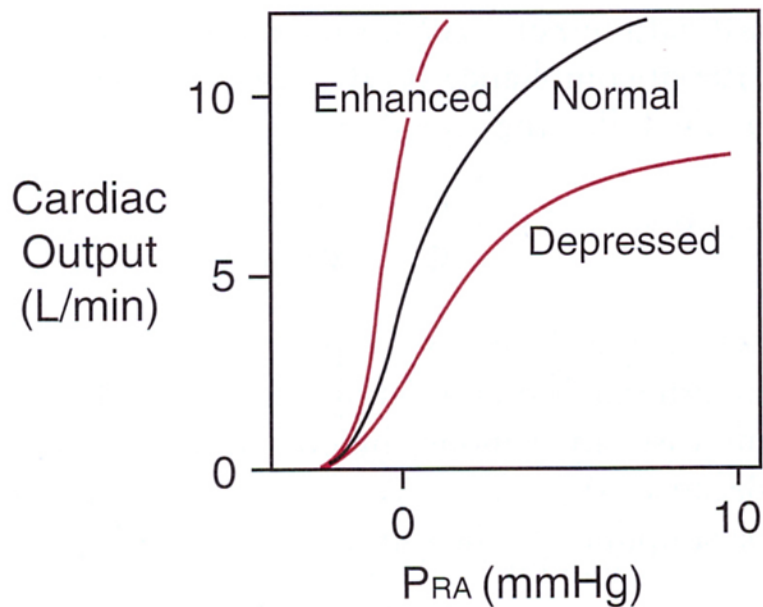
- Increased CO and decreased ESV
- SVR not changed
- Relative increased afterload
- Increased afterload caused decreased contractility
- Decreased contractility caused decreased preload



Increased sympathetic activation

(increased heart stimulation + venous splanchnic vasoconstriction + arteriolar vasodilation)

Srdeční funkční křivka

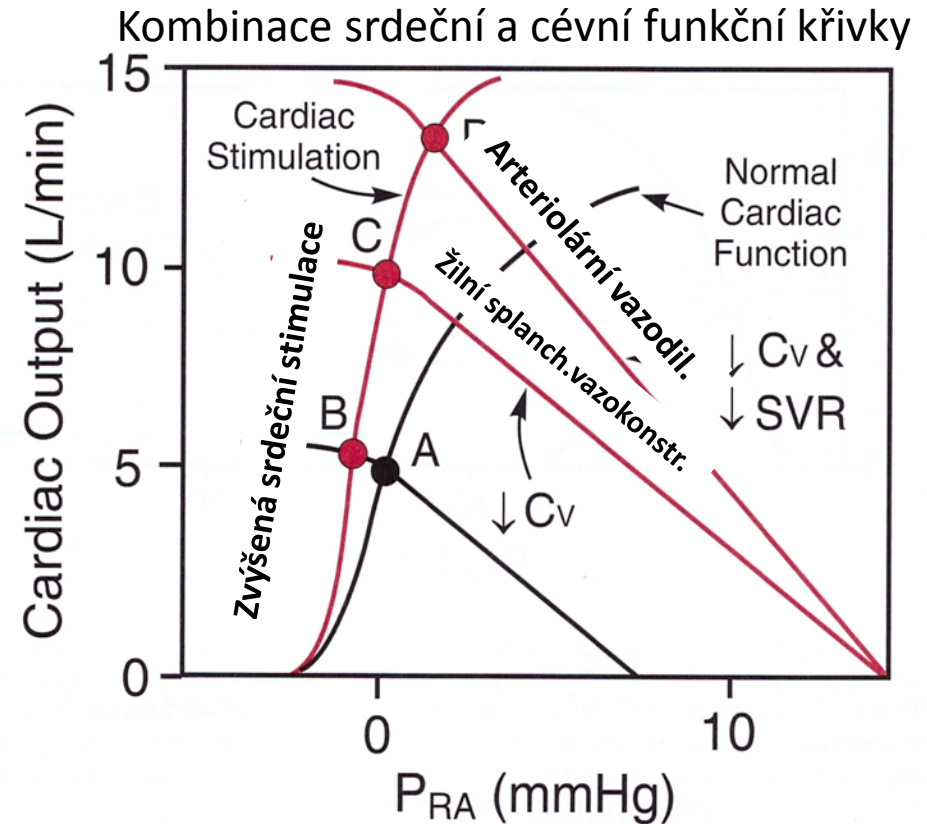
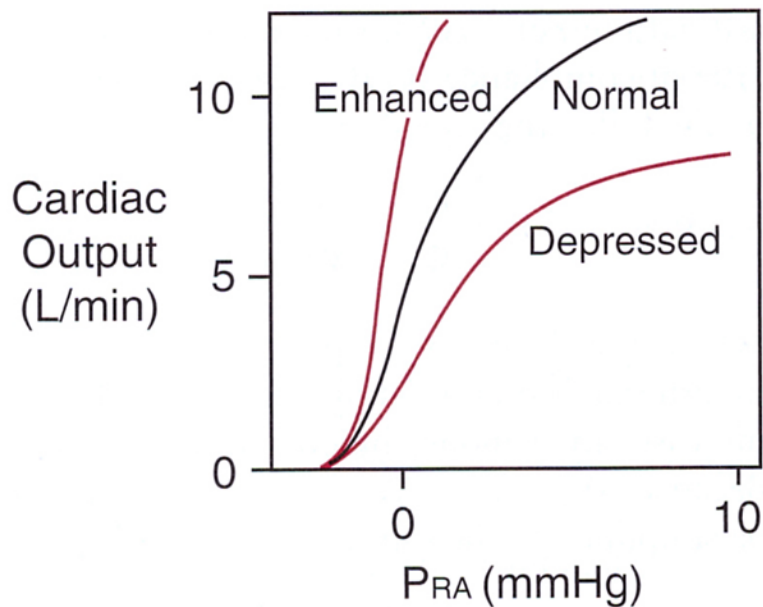


A – equilibrium between CO and Venous Return

Increased sympathetic activation

(increased heart stimulation + venous splanchnic vasoconstriction + arteriolar vasodilation)

Srdeční funkční křivka



A – equilibrium between CO and Venous Return

Heart failure

