The Heart II.

(CO, preload, afterload, contractility…)

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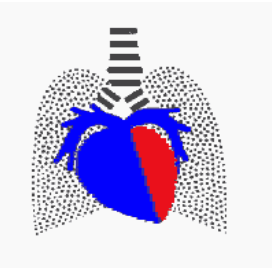
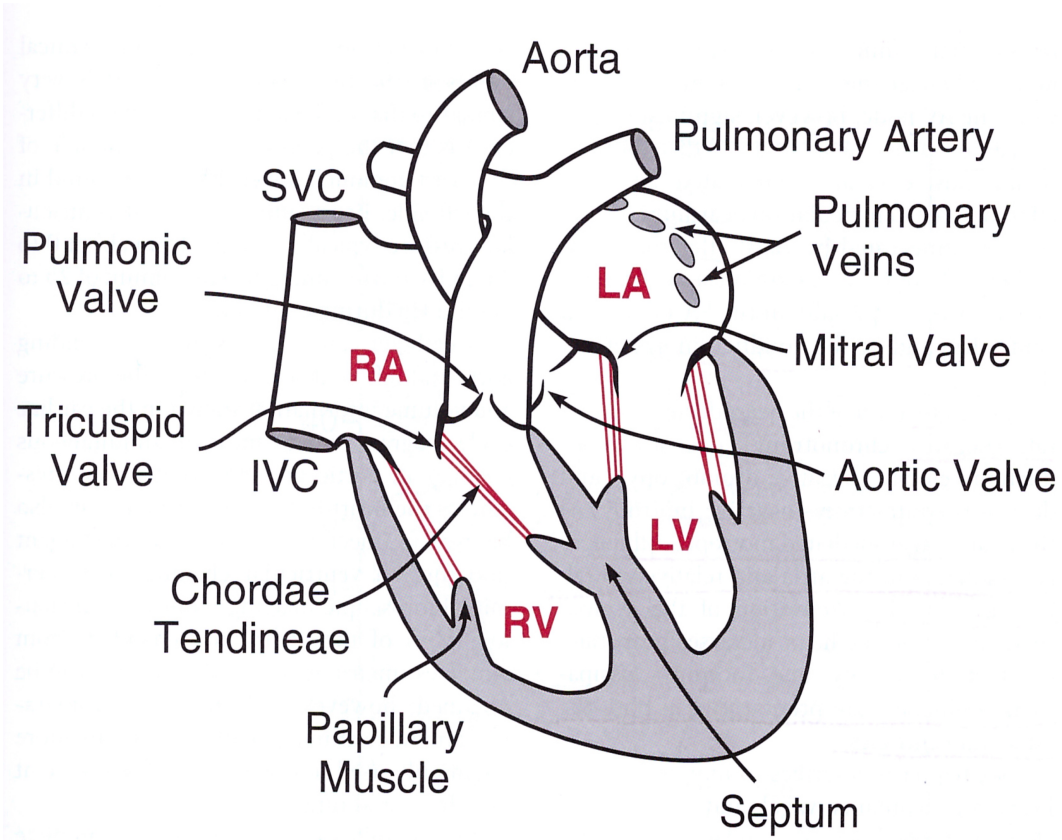


- Cardiac cycle, cardiac output
- Preload
- Afterload
- Contractility – inotropy
- Relationship between CO, preload, afterload, contractility, venous return…

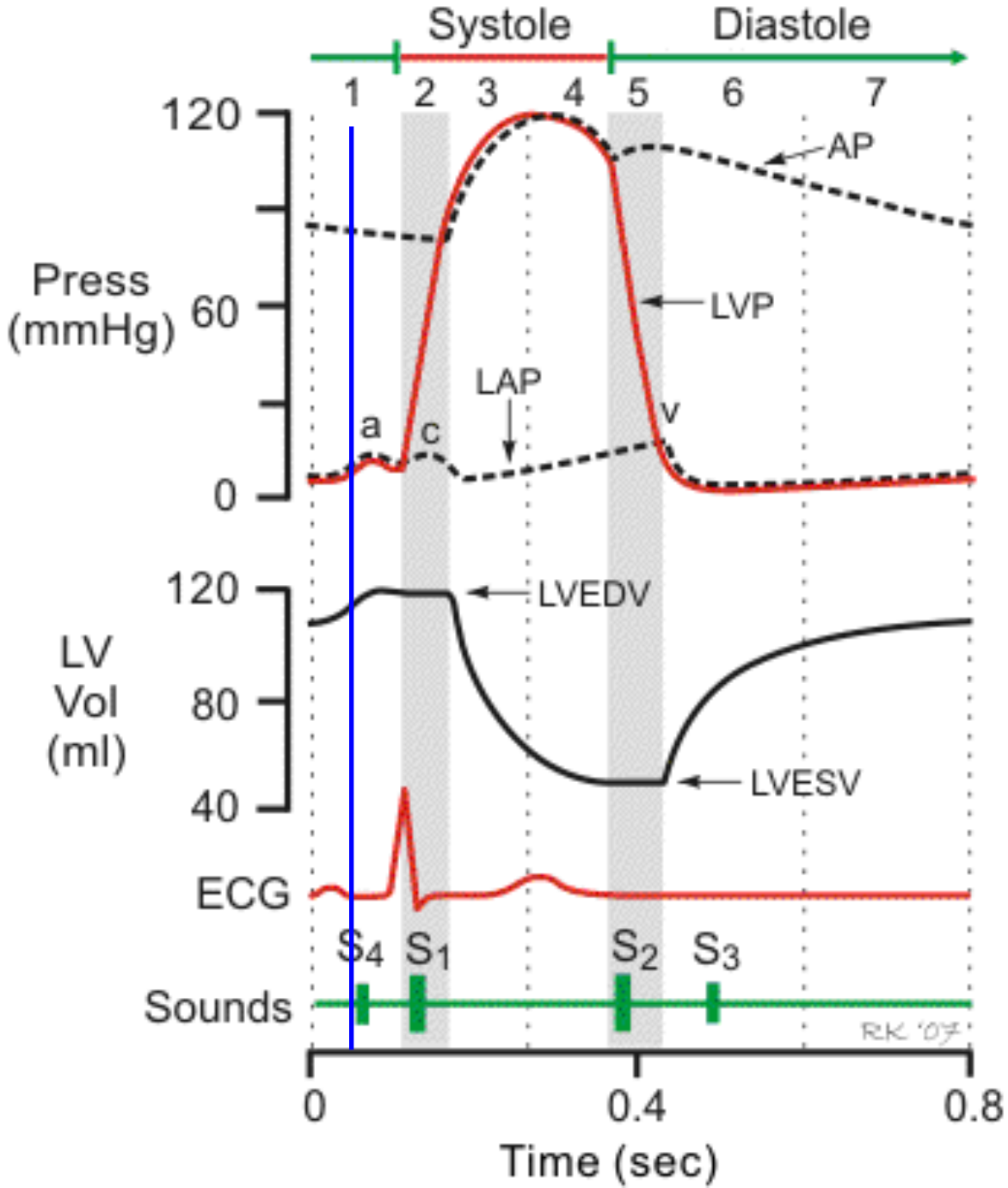
The Heart = pump

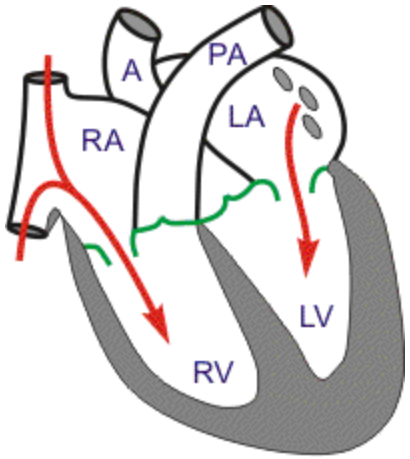
- Device pumping blood in cycles
- Filling (diastole) / ejecting (systole)
- pressure / volume work
- isotonic } contraction
  isometric } relaxation



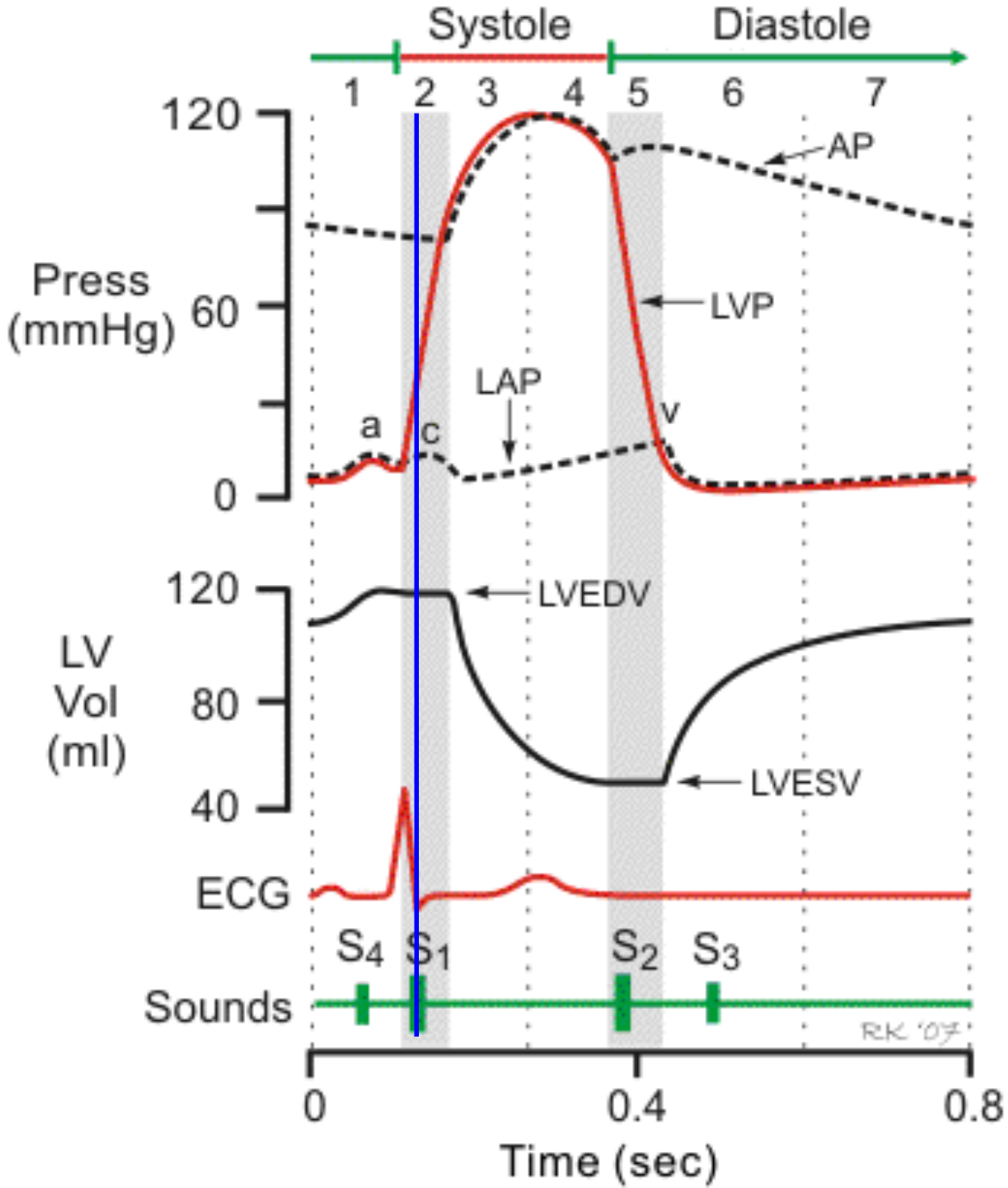
The Heart Cycle

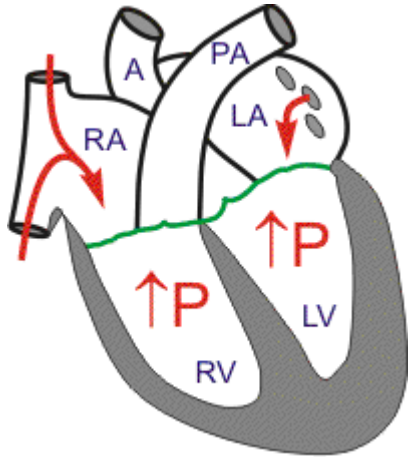
- Atrial contraction
- isovolumetric ventricular contraction
- Ejection
- Isovolumetric ventricular relaxation
- Passive ventricular filling



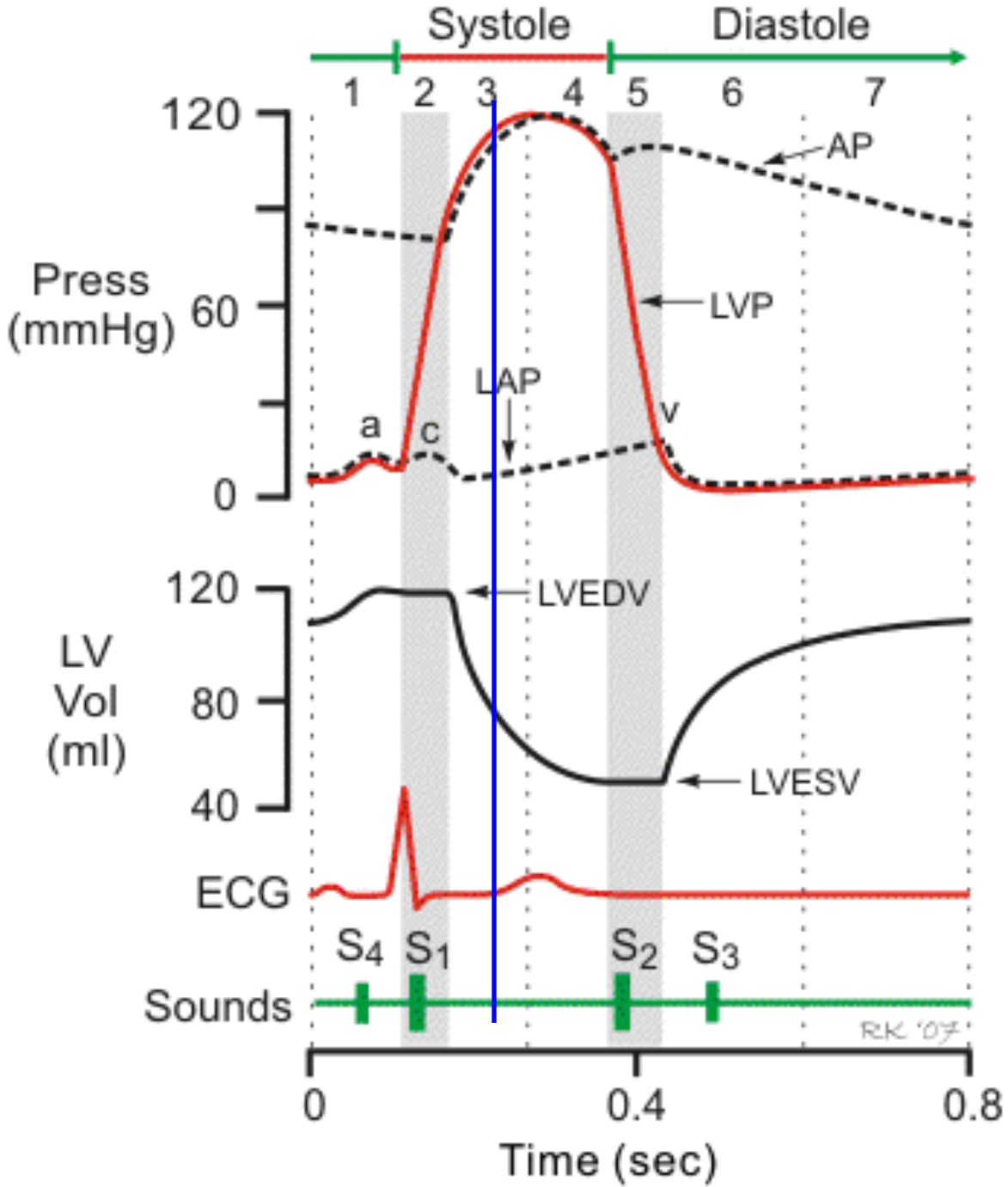


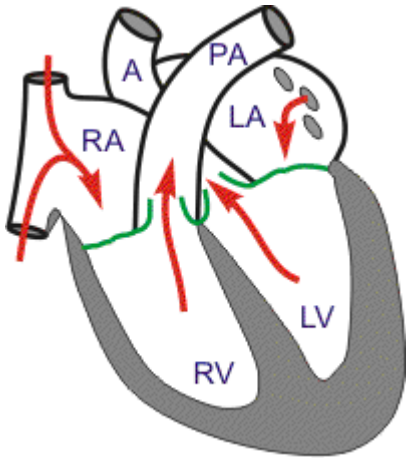
1. Atrial contraction



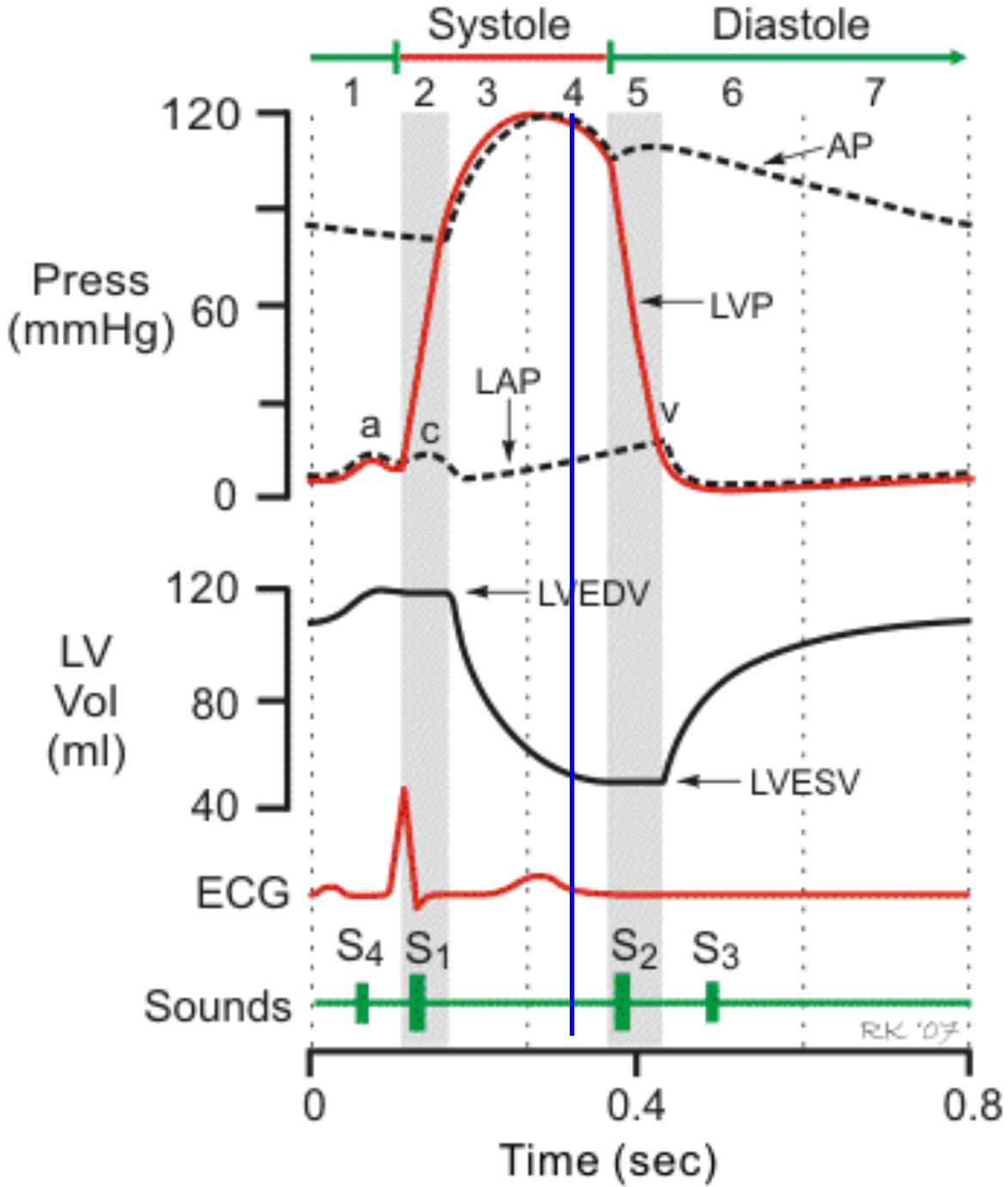


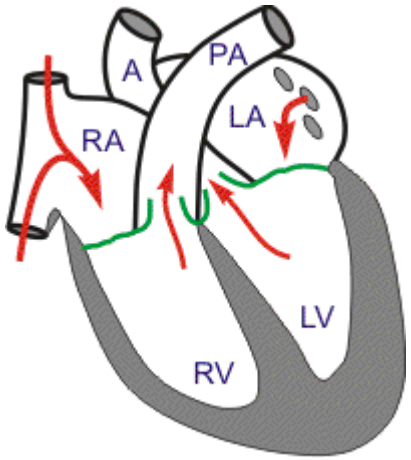
2. Isovolumetric contraction



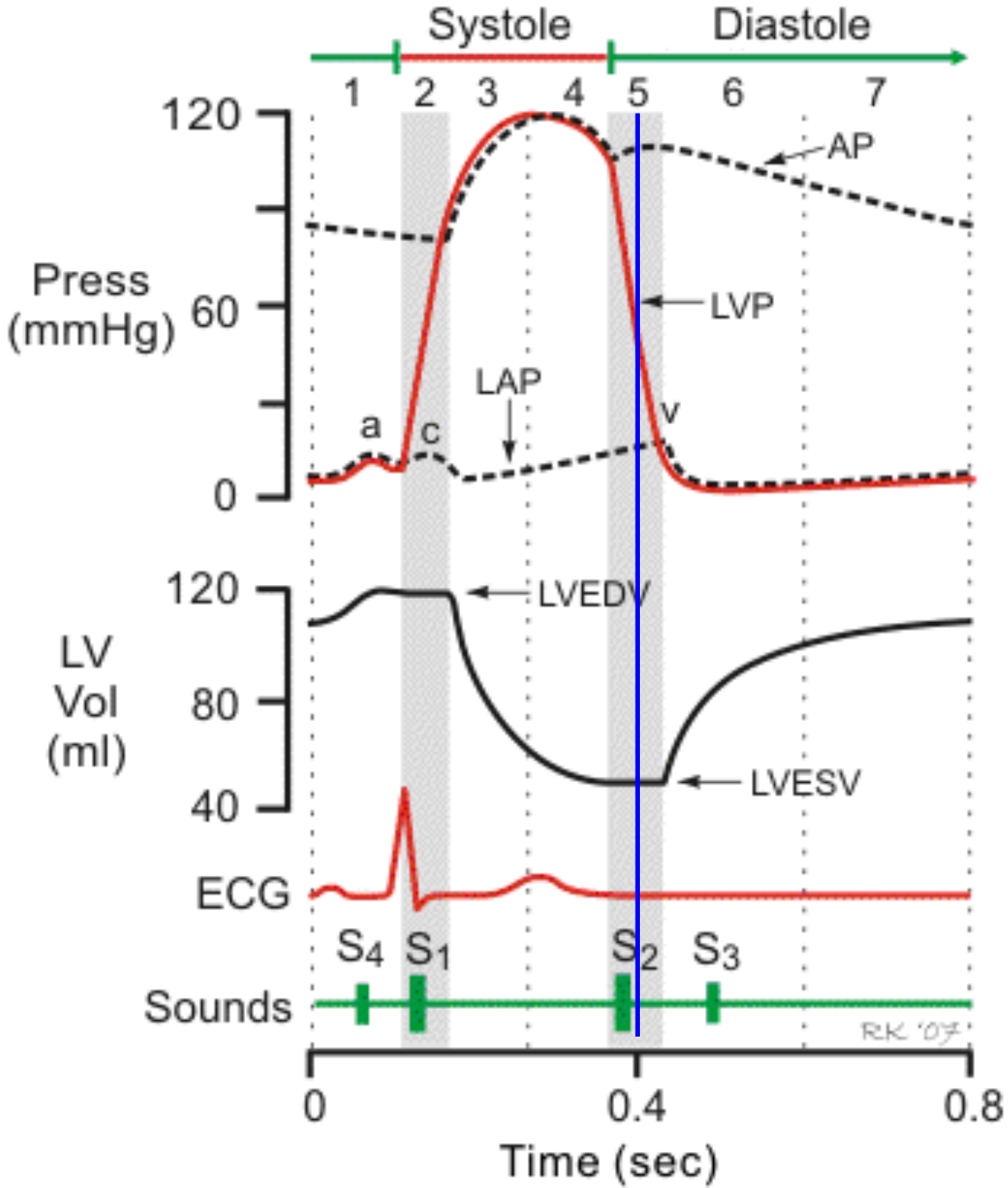


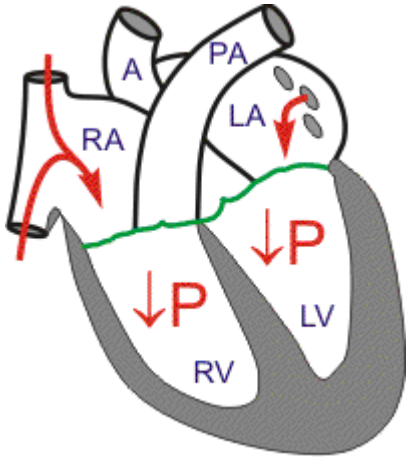
3. Fast ejection



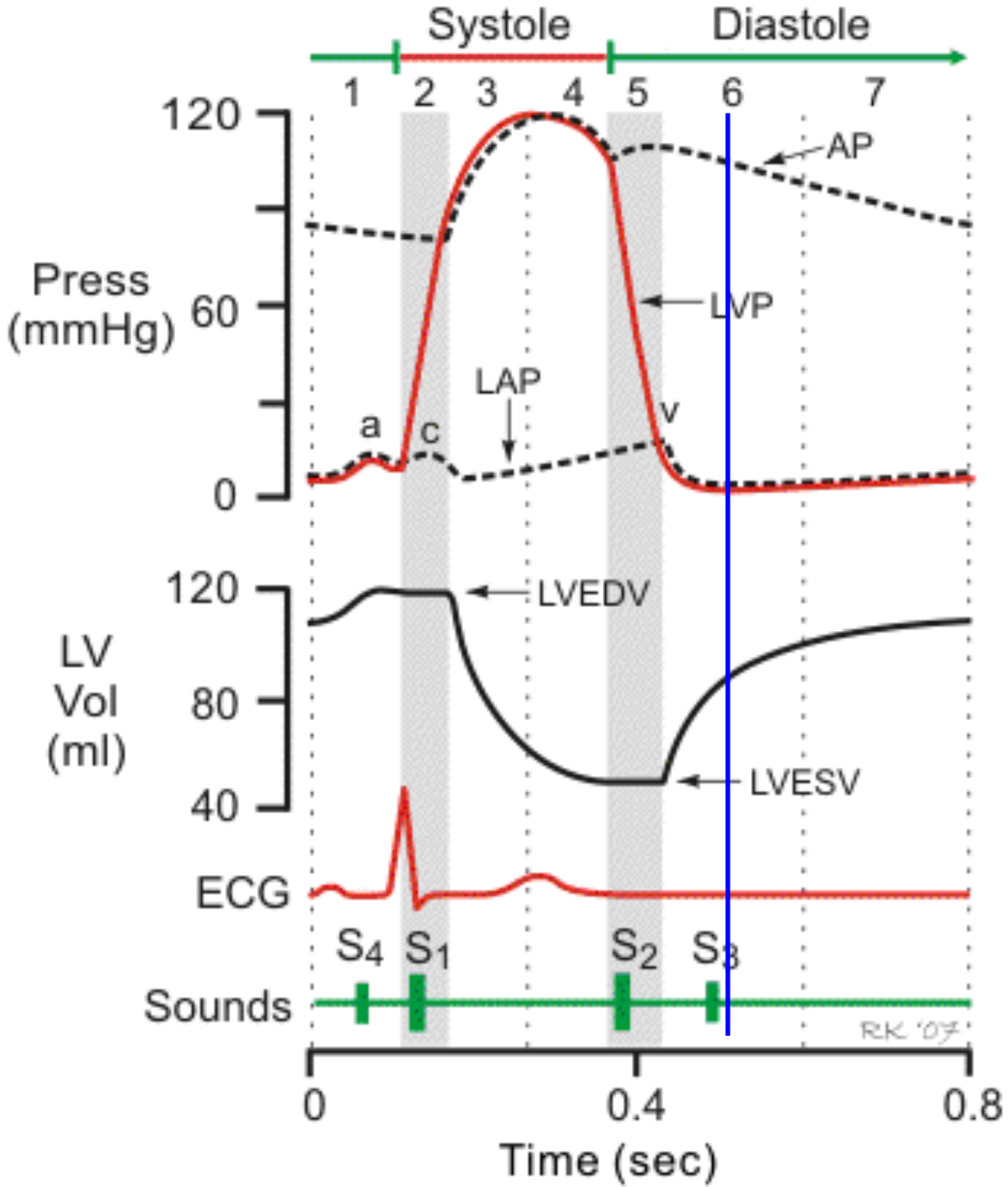


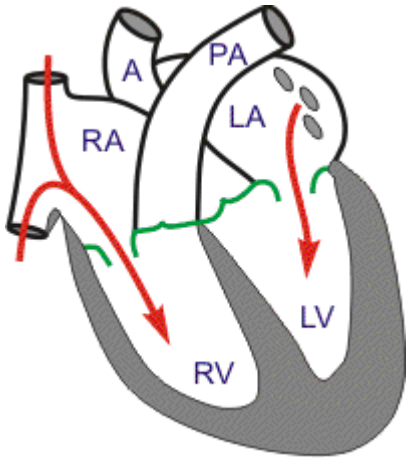
4. Slow ejection



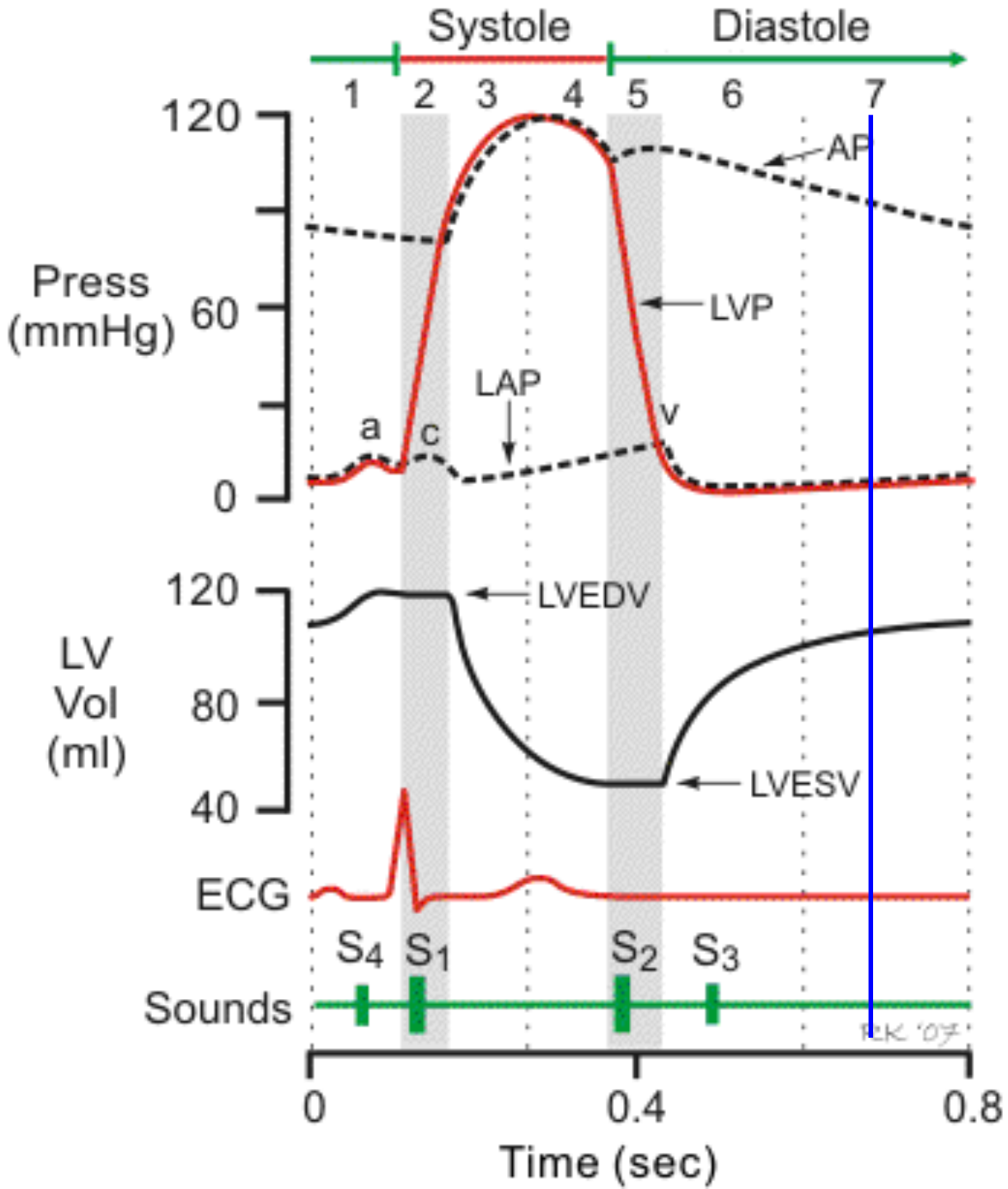


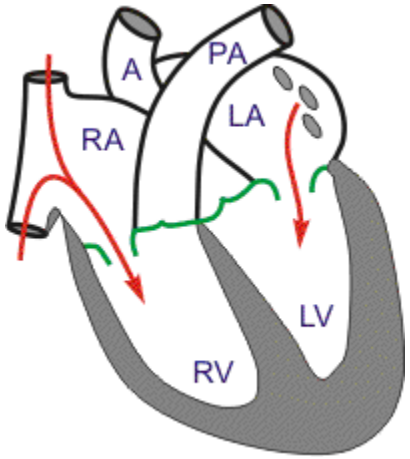
5. Isovolumetric relaxation





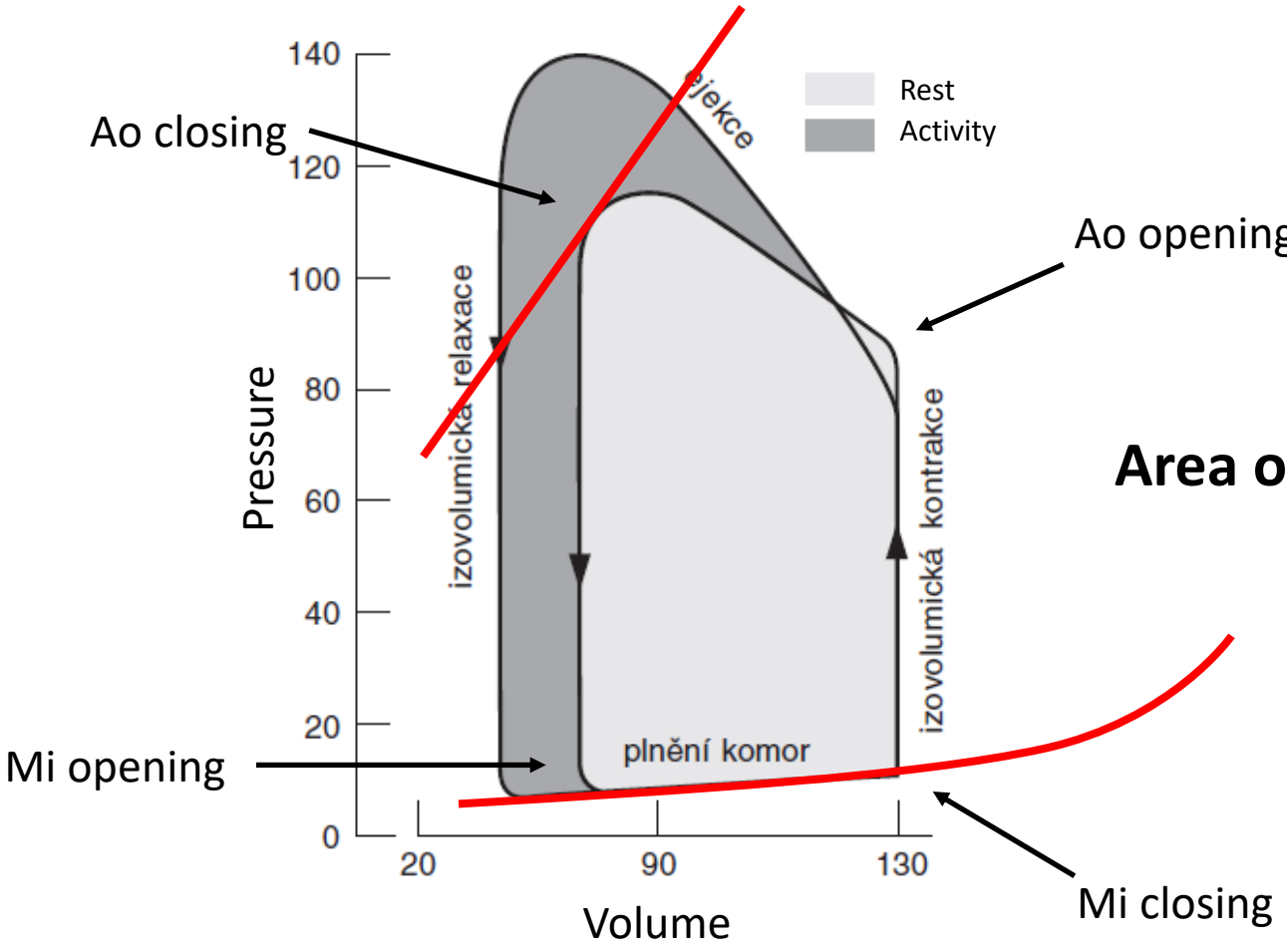
6. Fast filling





7. Slow filling

Pressure / Volume Relationship



Area of PV relathionship = heart work

Cardiac Output

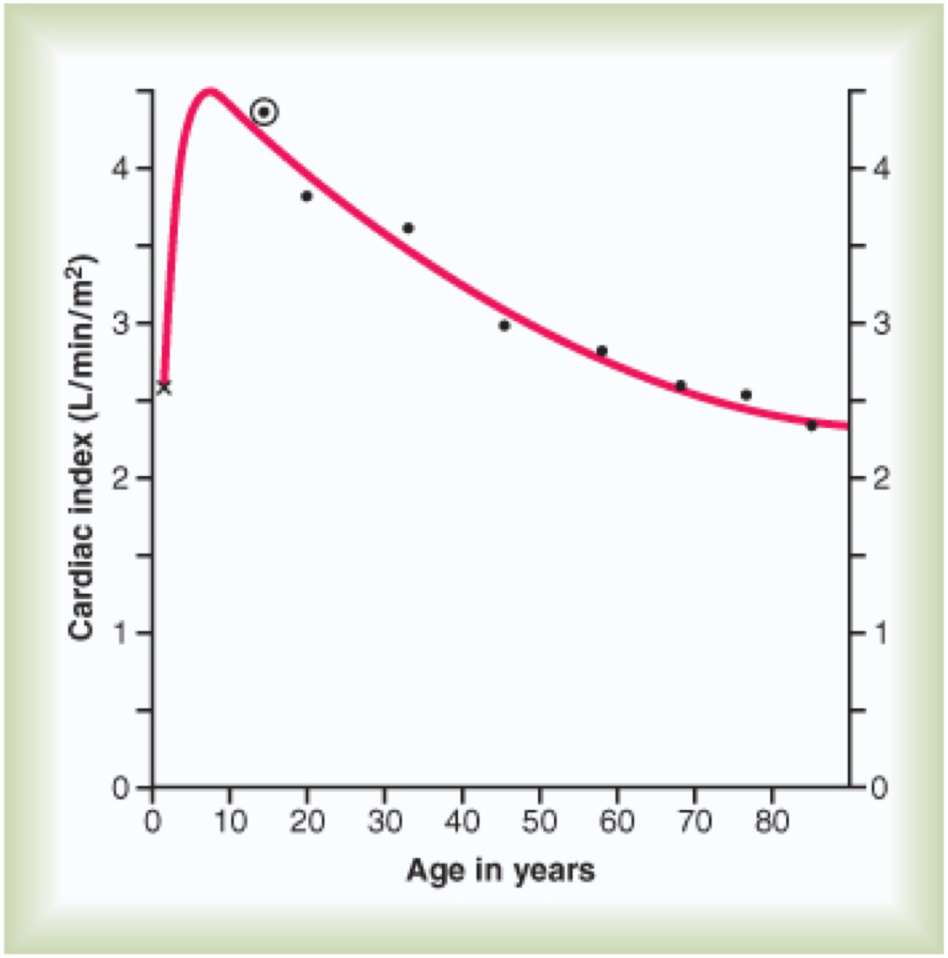
$$CO = SV \cdot HR$$

Cardiac Index (CI) = CO / BSA

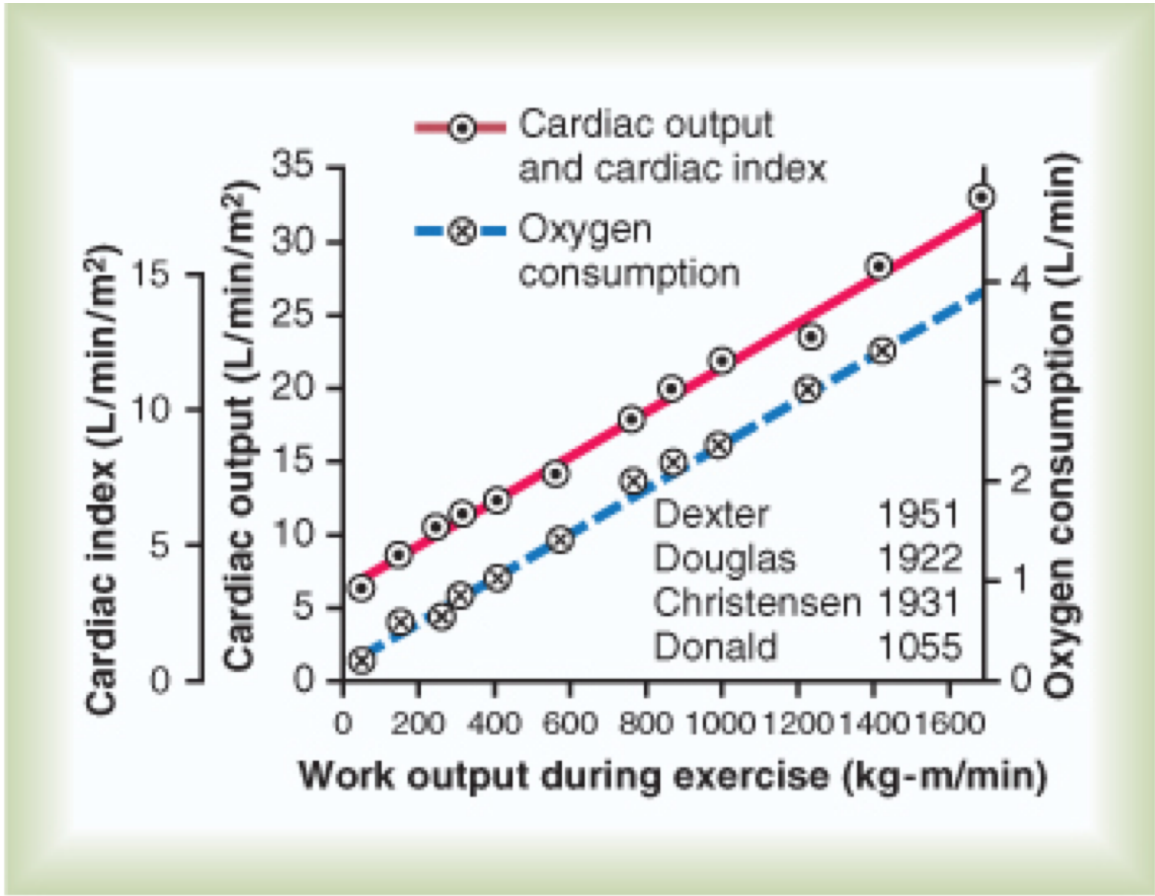
When the heart in unable to pump sufficiently to maintain blood flow to meet the body‘s needs = **the heart failure**

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

Relationship between CO and age



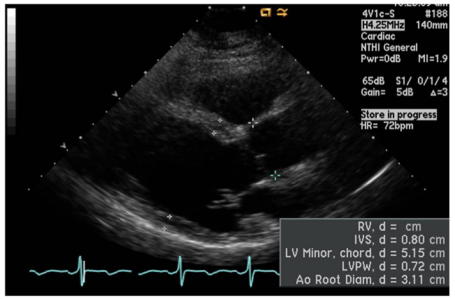
Relationship between CO and excercise



Measurement of CO

- Echo:

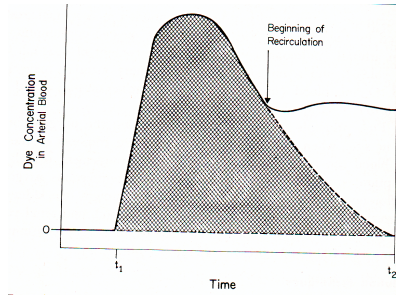
Diameter of LVOT= calc.of area of LVOT



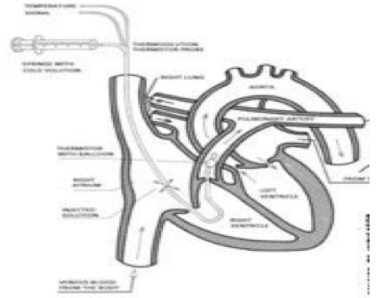
Velocity of flow in LVOT = VTI in LVOT

- Diluting methods:

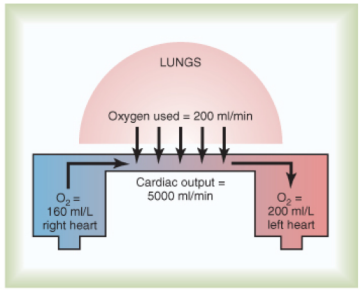
Dye dilution – experiments

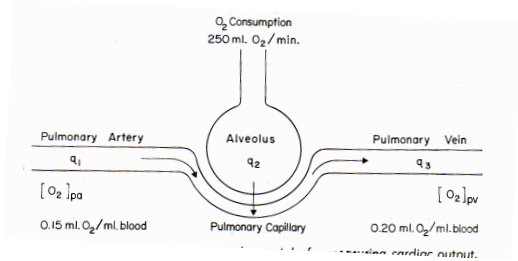


Thermodilution – common in critical ill patients



- Fick principle:

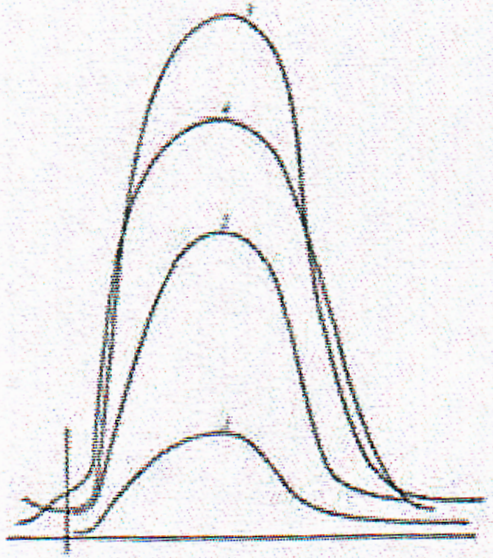




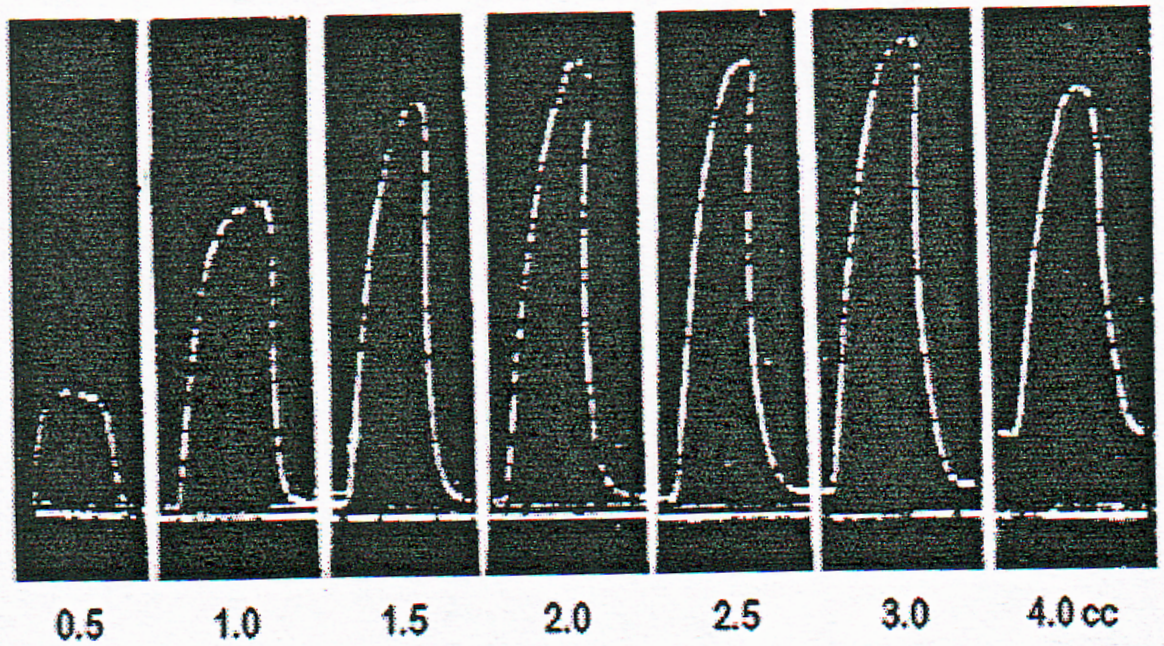
Preload

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

Frank-Starling



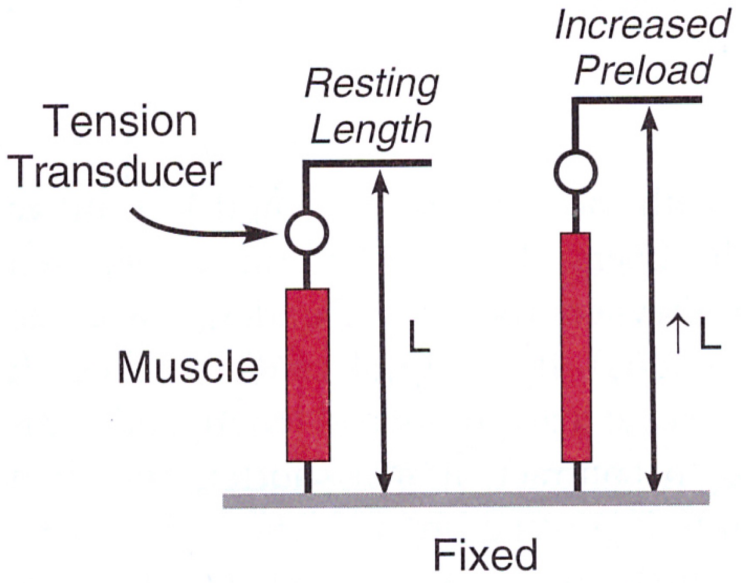
Frank (frog, 1895)



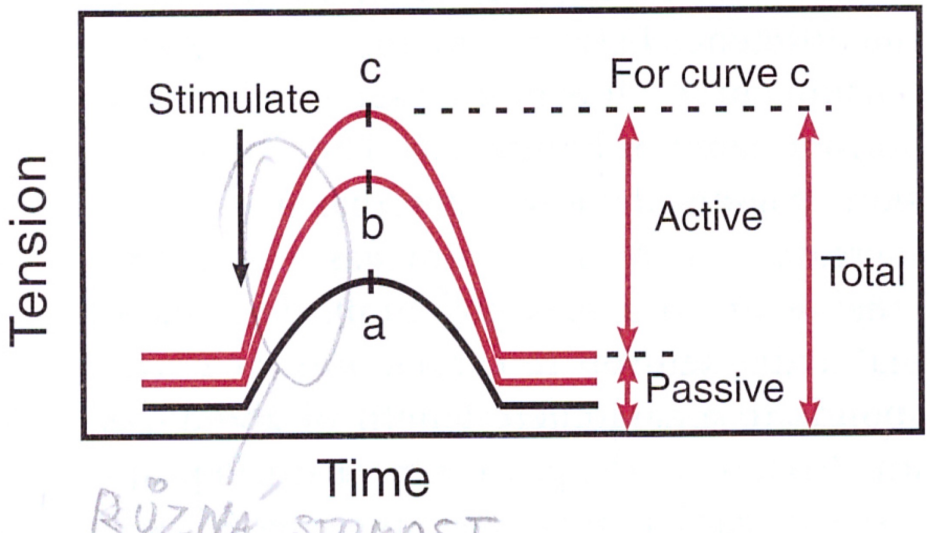
Starling (dog, 1914)

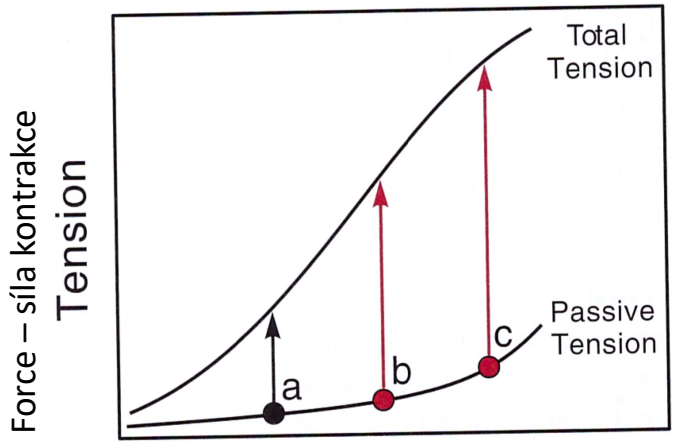
"Experiments carried out in this laboratory have shown that in an isolated heart [...] (within physiological limits) the larger the diastolic volume [...] the greater is the energy of its contraction."

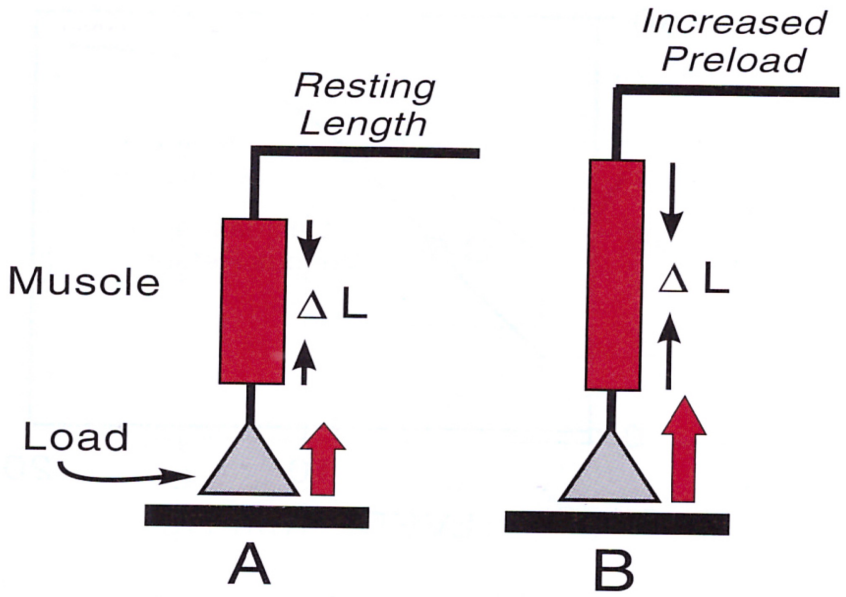
EH Starling & MB Visscher. The regulation of the energy output of the heart. *J Physiol* 1926/62:243-261.



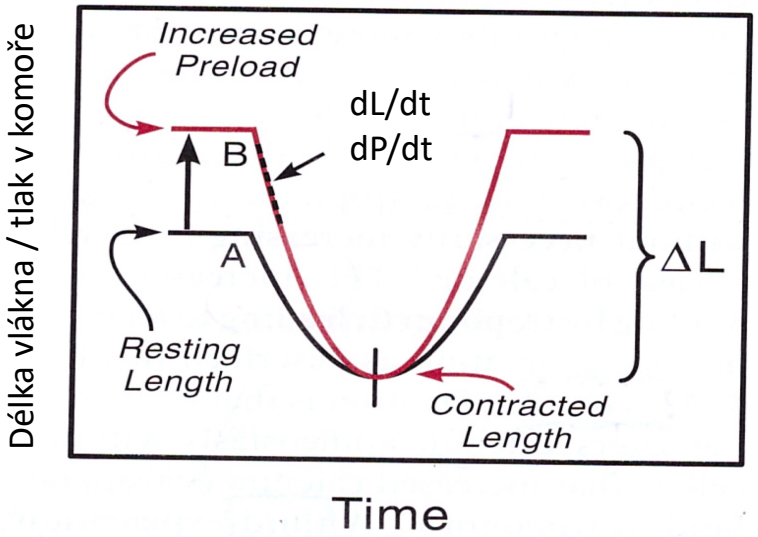
Isometric contraction

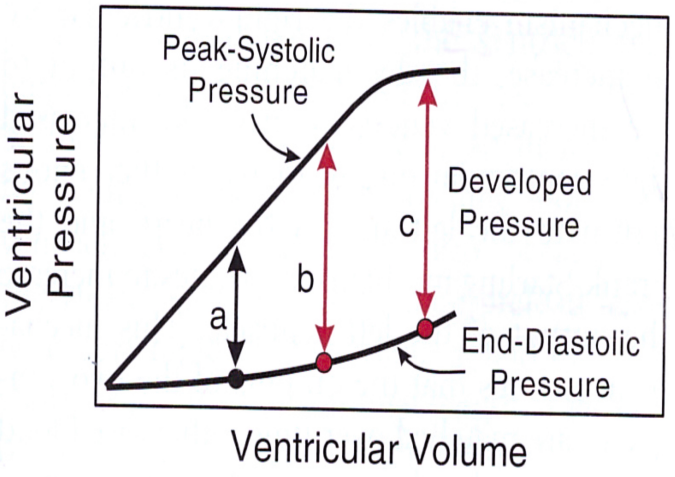




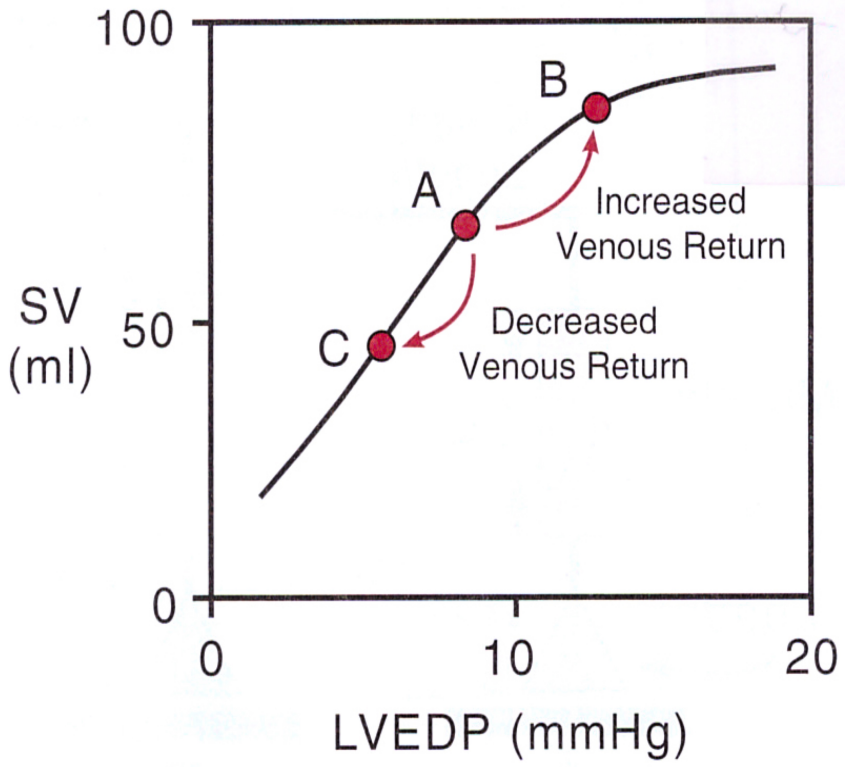


Isotonic contraction





Frank-Starling mechanism
(Heterometric regulation of contraction)



Frank-Starling mechanism
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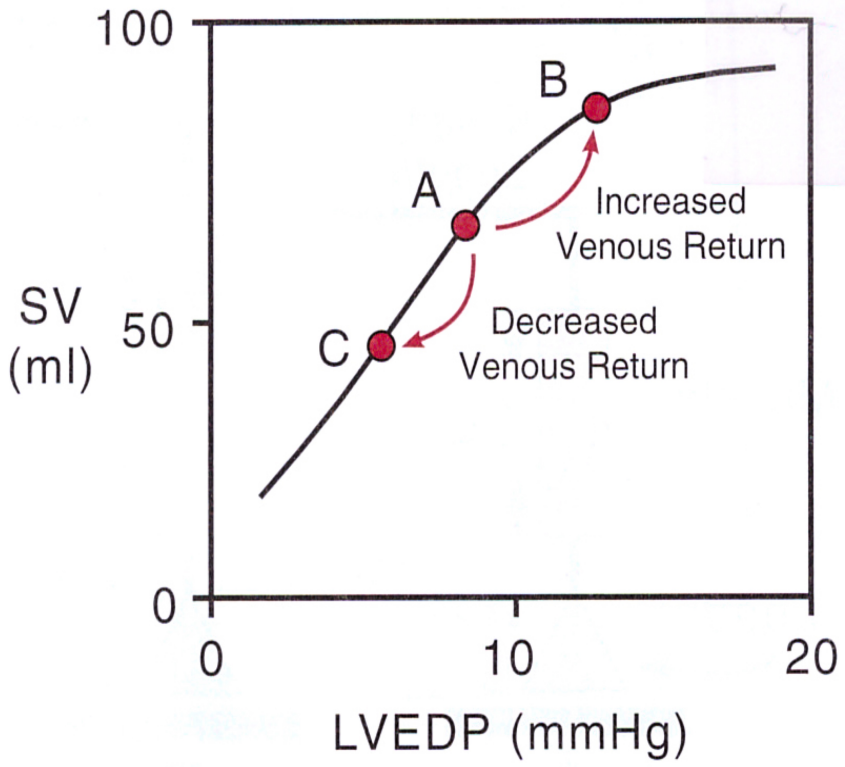
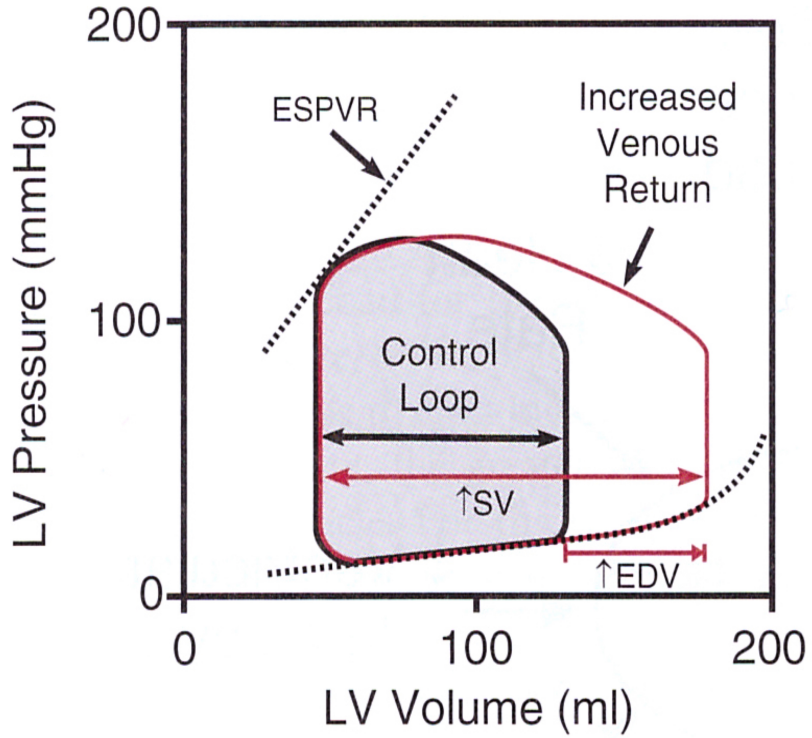




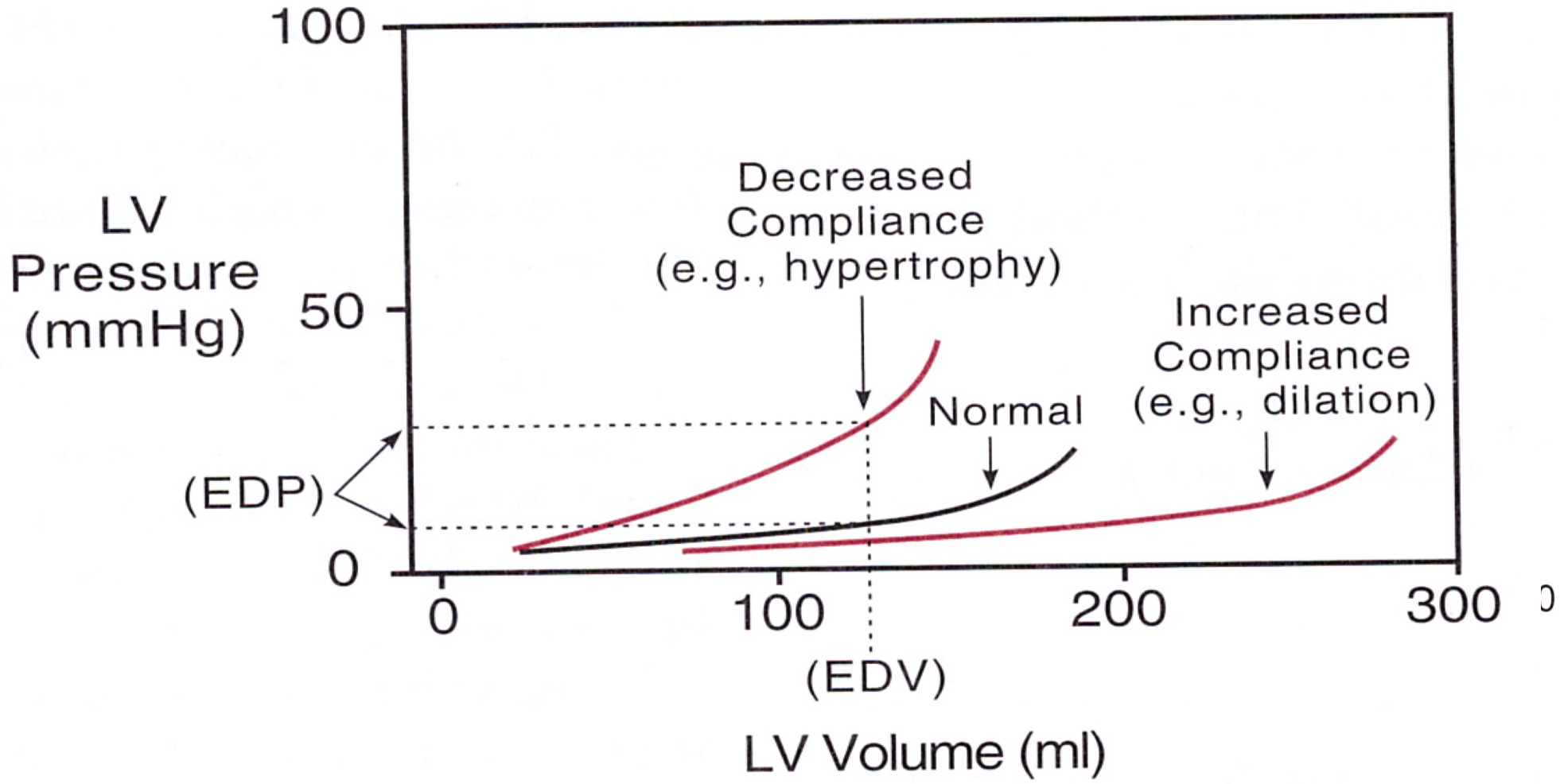
Frank-Starling mechanism

Increased filling – faster and stronger contraction, why?

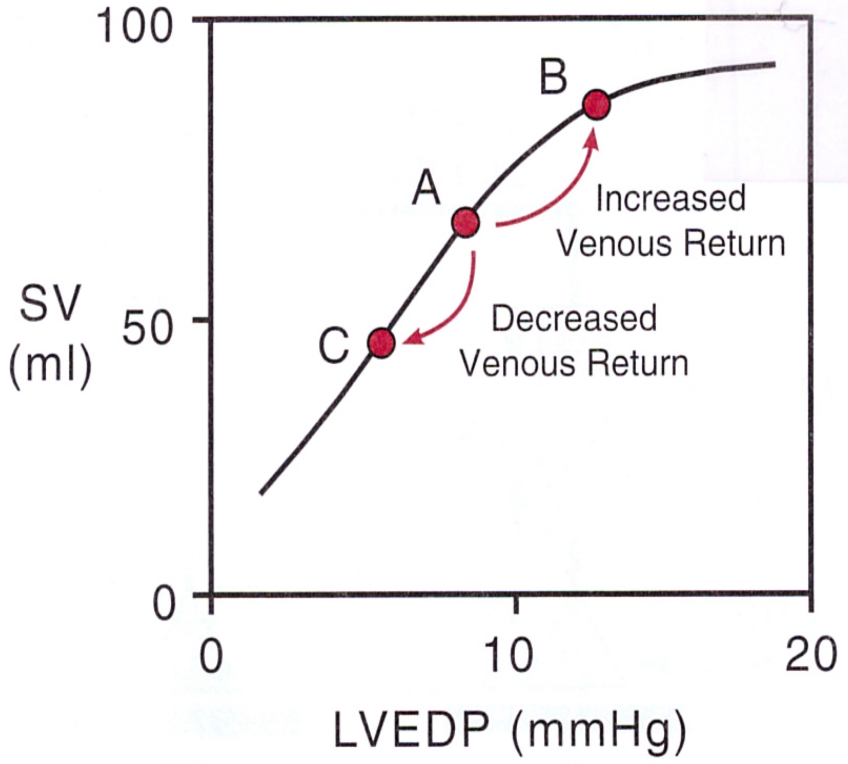
- <u>Prolonged sarcomere:</u>
    - More actin-myosin interactions – more ATP, more energy
    - Increased sensitivity of troponin C to Ca^{2+}
    - Increased intracelular [Ca^{2+}]
    - Decreased diameter of muscle fiber – actin+myozin closer together

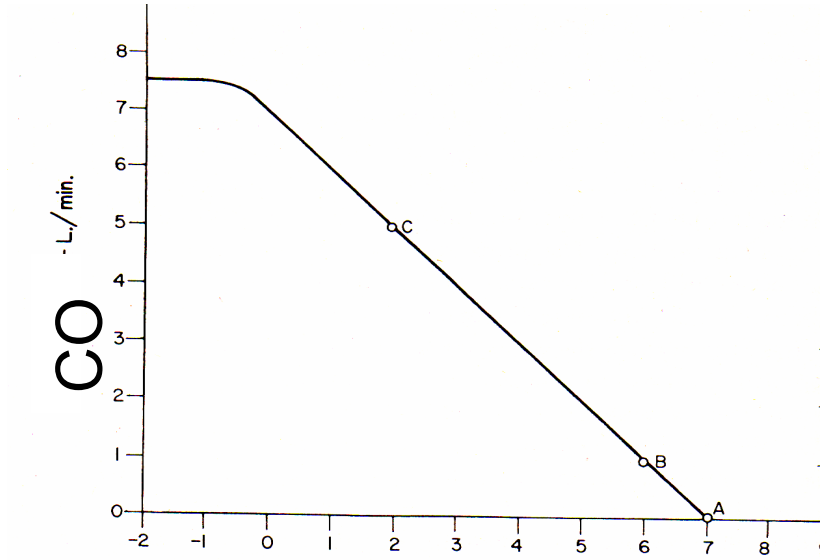
Frank-Starling mechanism – ventricular compliance

(Heterometric regulation of contraction)



CO and Venous Return

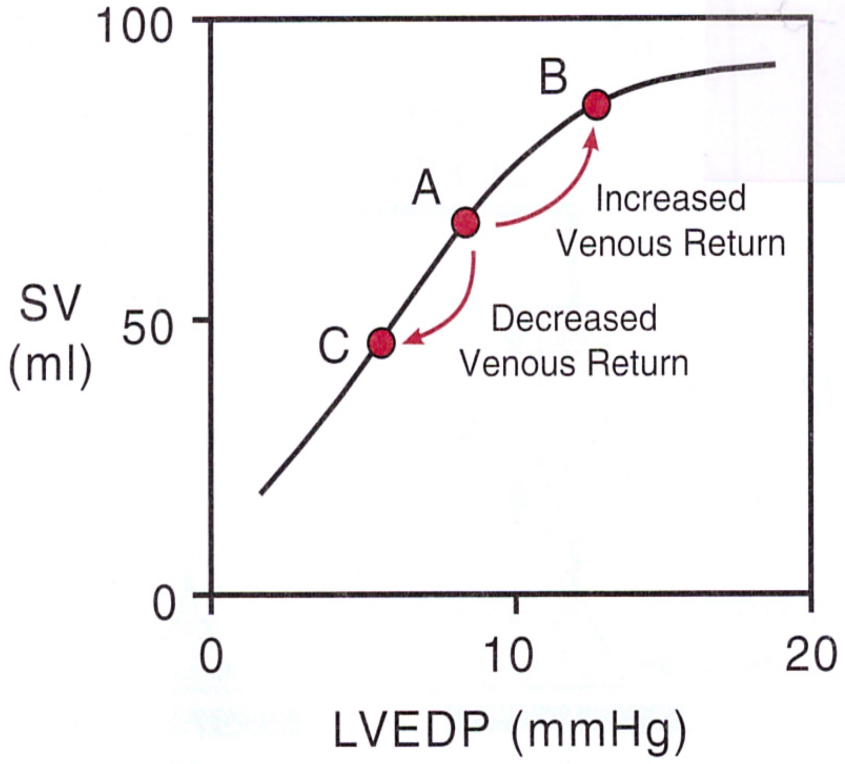


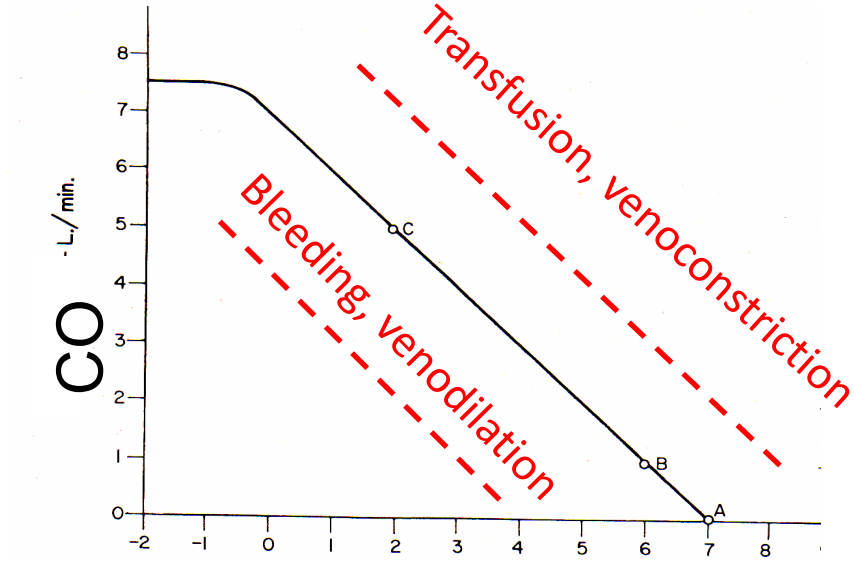


Venous return (CVP)

Střední oběhový tlak

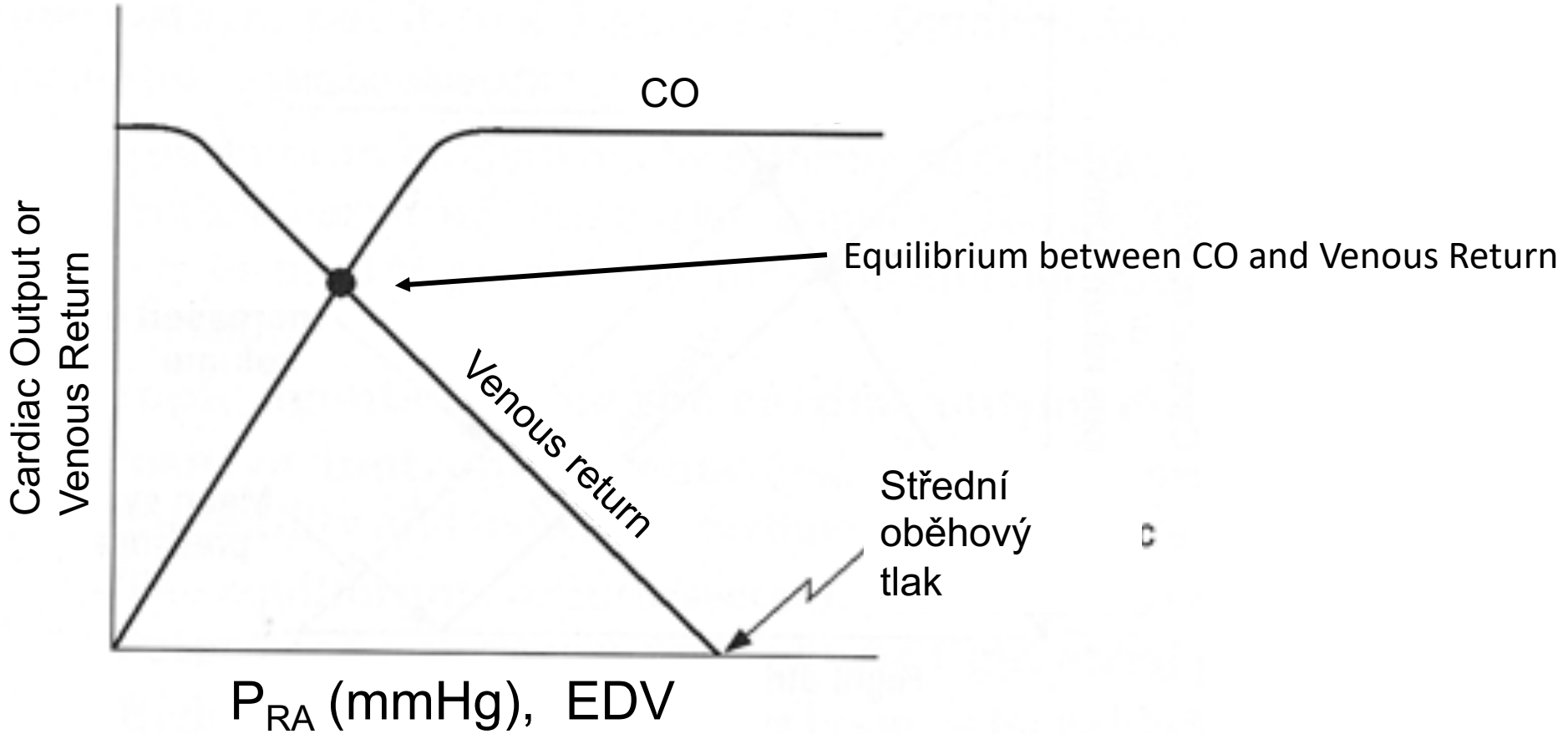
CO and Venous Return





Střední oběhový tlak

Relationship between CO and Venous Return



Factors Determining Ventricular Preload

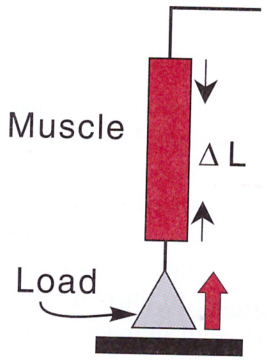
- Venous Pressure, venous return, CVP
- Ventricular compliance
- Heart Rate – filling time, time of diastole
- Atrial contraction – more important in tachycardia, atrial fibrillation
- Inflow resistance – Tri stenosis, Mi stenosis
- Outflow resistance – PAP, Pu stenosis, hypertension, Ao stenosis
- Ventricular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

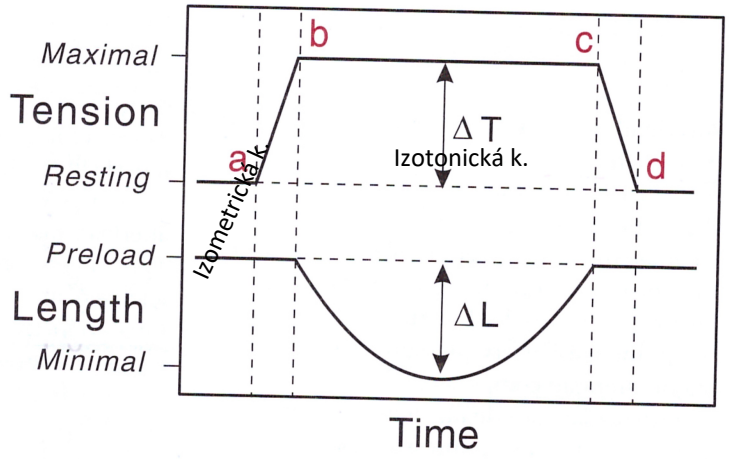
Afterload

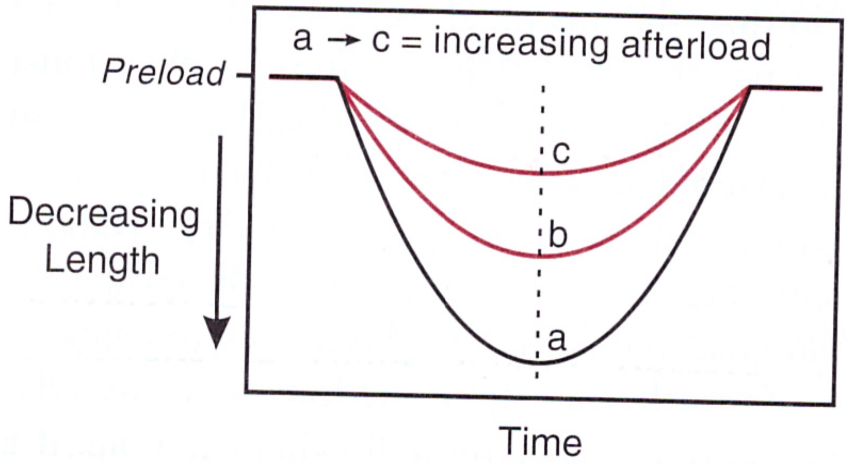
- Load against which the heart must contract to eject blood
- Characterized by SVR (small arteries, arterioles)
- Can be measured like wall stress:

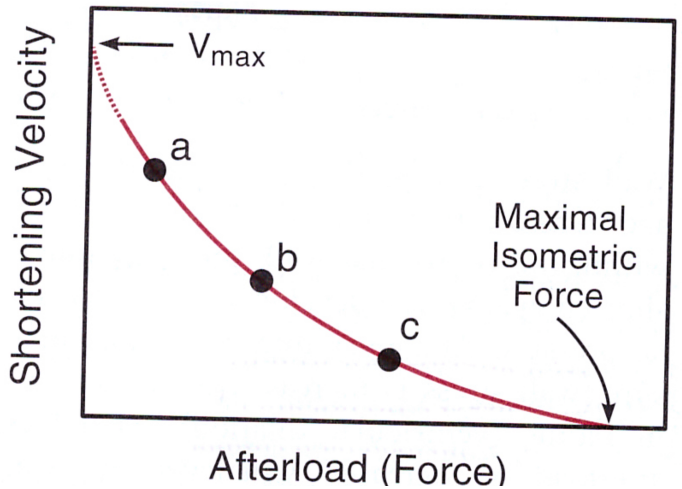
$$\sigma \propto \frac{P \cdot r}{h}$$

Afterload: force / velocity relationship

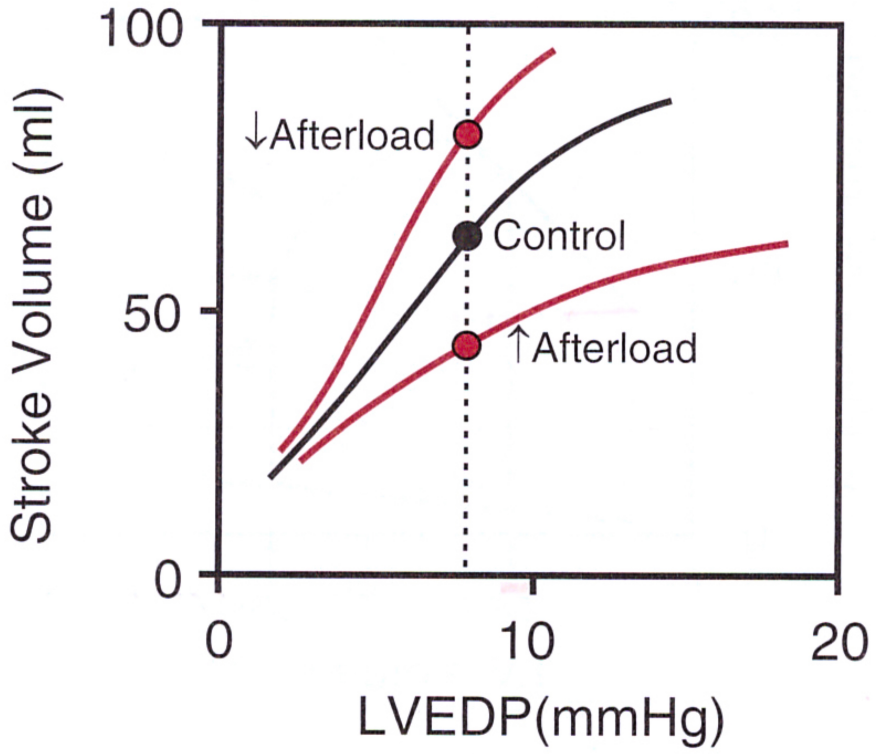


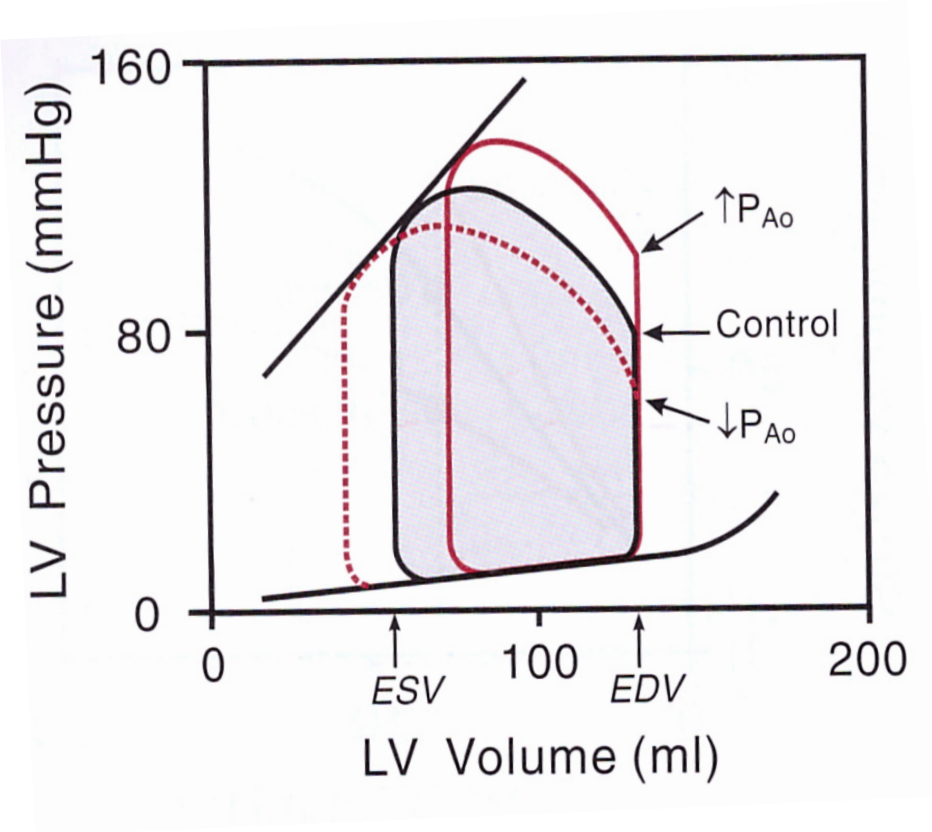






Afterload



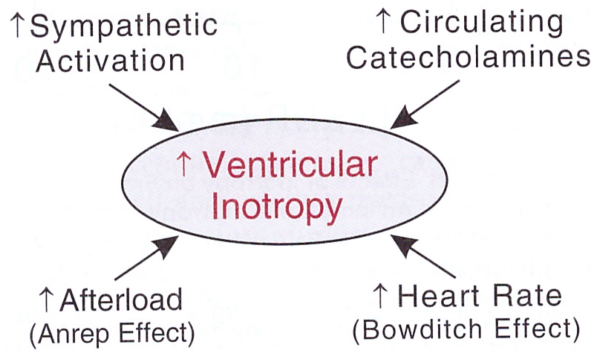


↓CO

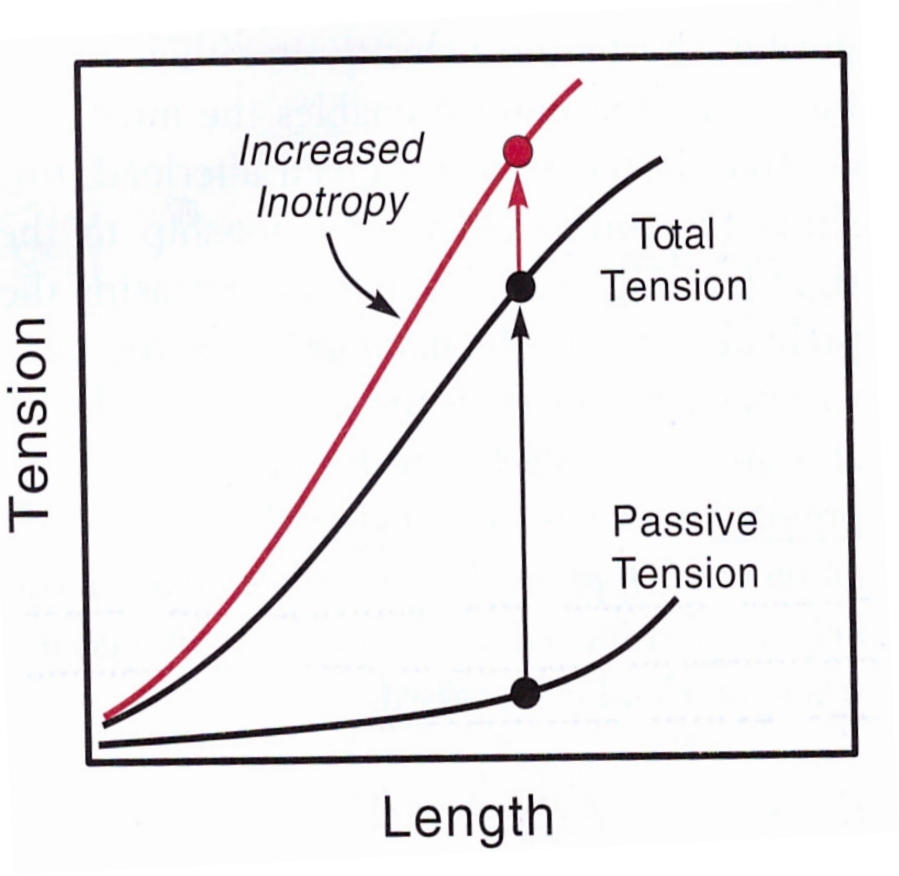
↑ESV

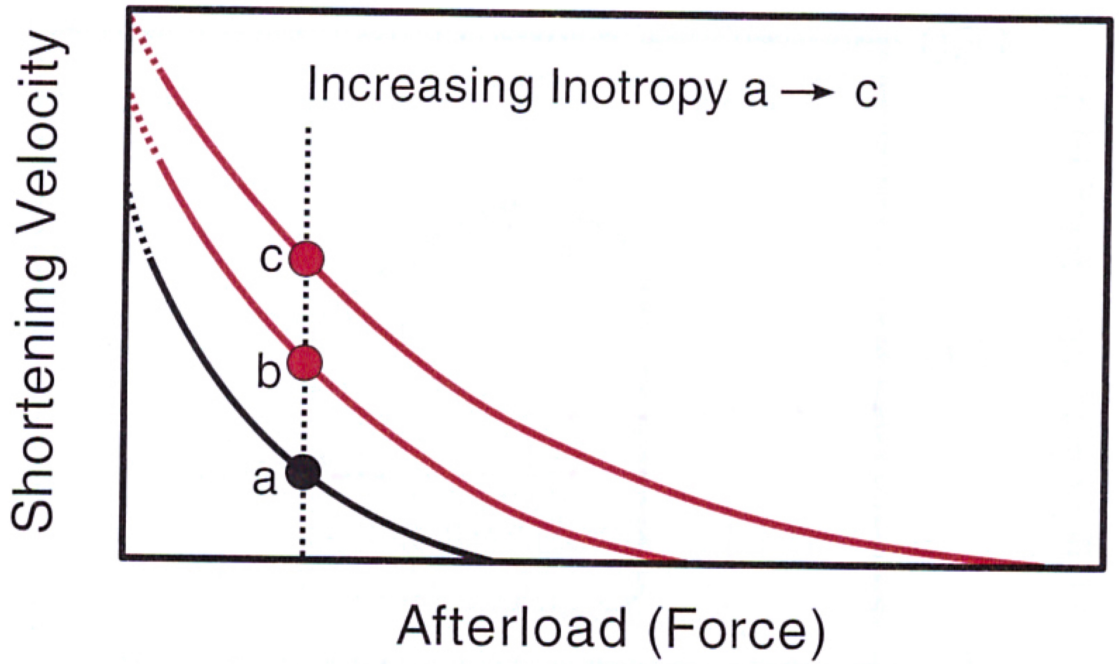
Contractility - inotropy

- Contractility (inotropy) = the force of contraction
- Sarcomere lenght is changing by different mechanism than 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 lenght of sarcomere = HOMEOMETRIC REGULATION OF CONTRACTION
- Factors influencing inotropy:

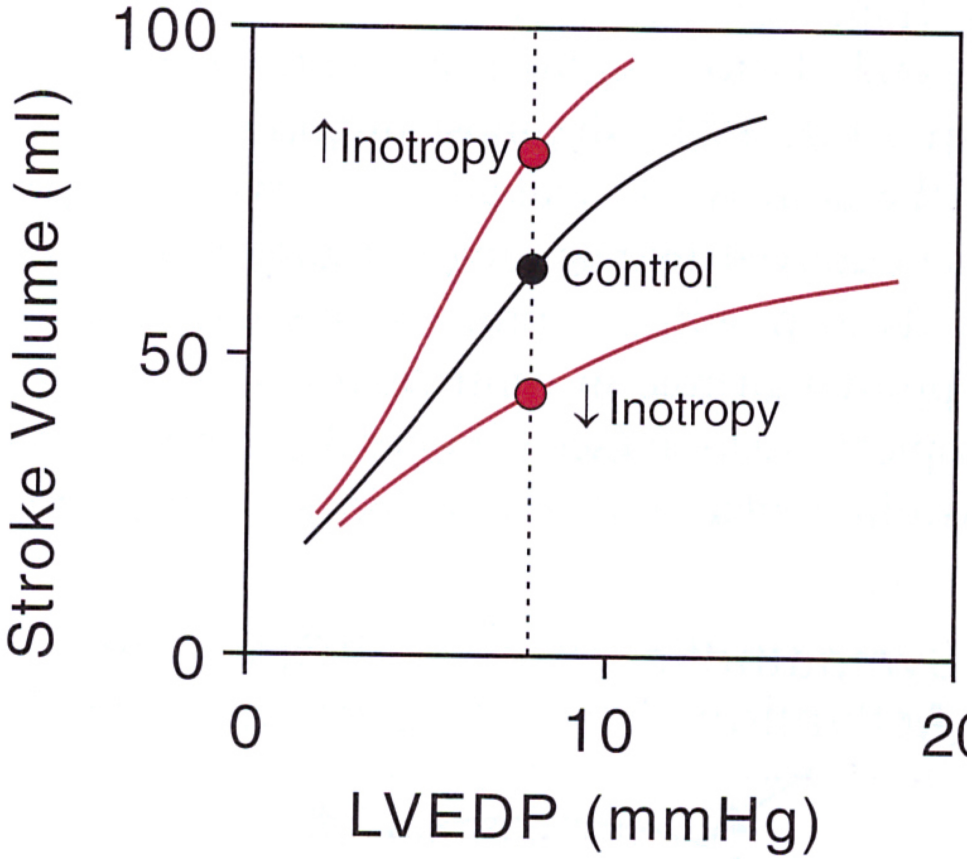


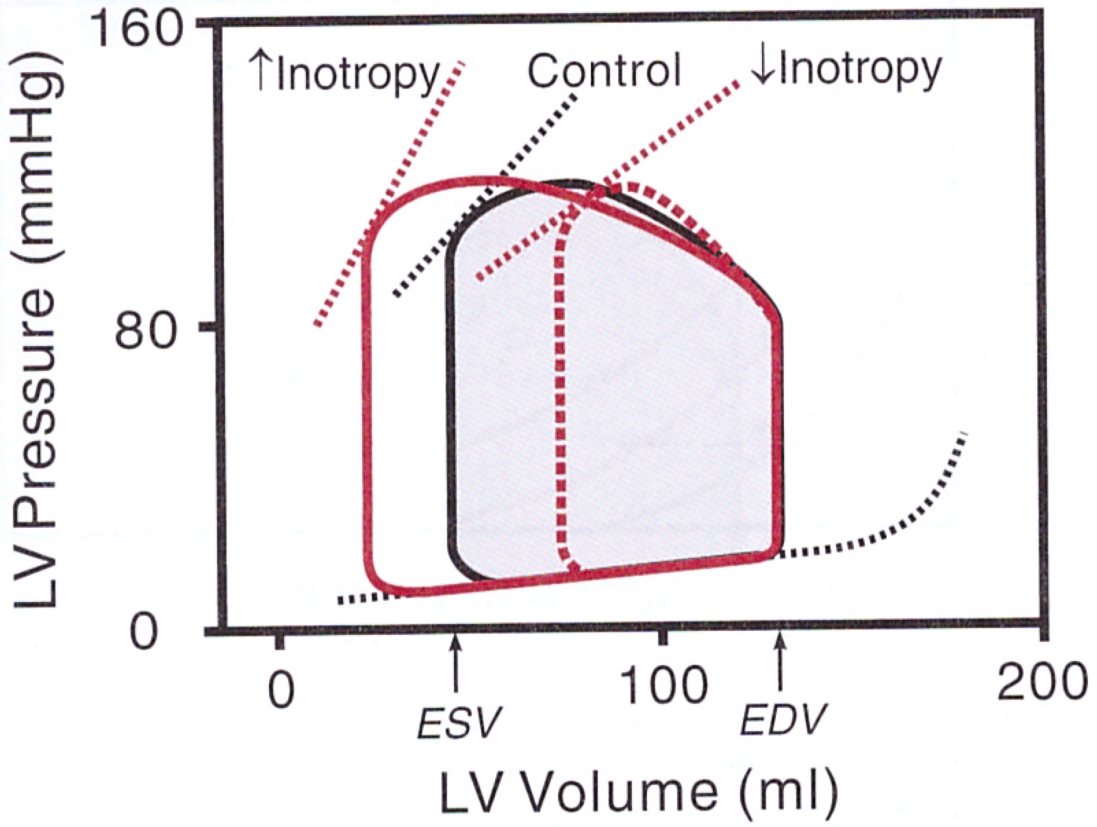
Inotropy


Increased
Inotropy
Total
Tension
Passive
Tension
Tension
Length


Increasing Inotropy a → c
c
b
a
Shortening Velocity
Afterload (Force)

Inotropy

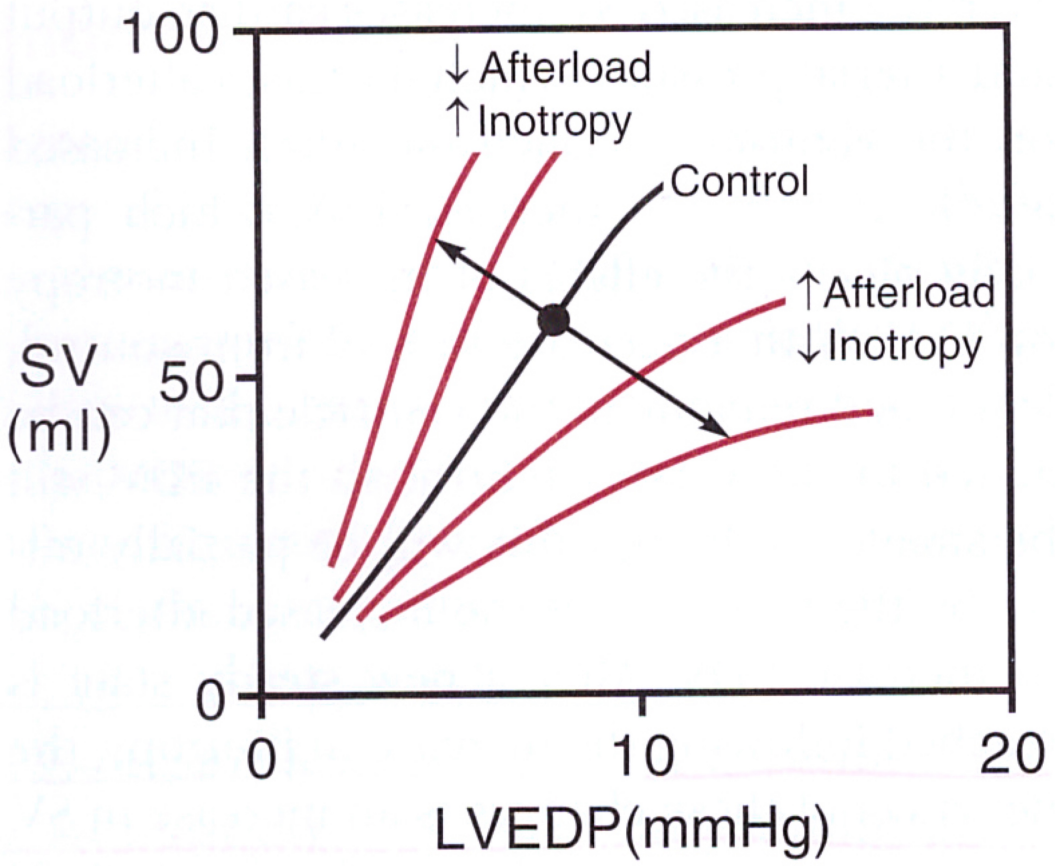




↑CO

↓ESV

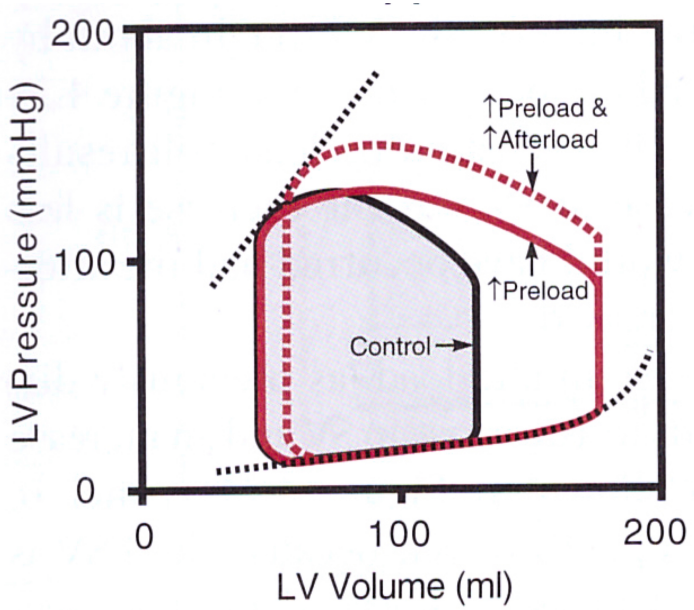
Inotropy vs. afterload



Interaction: preload, afterload, inotropy

Increased preload:

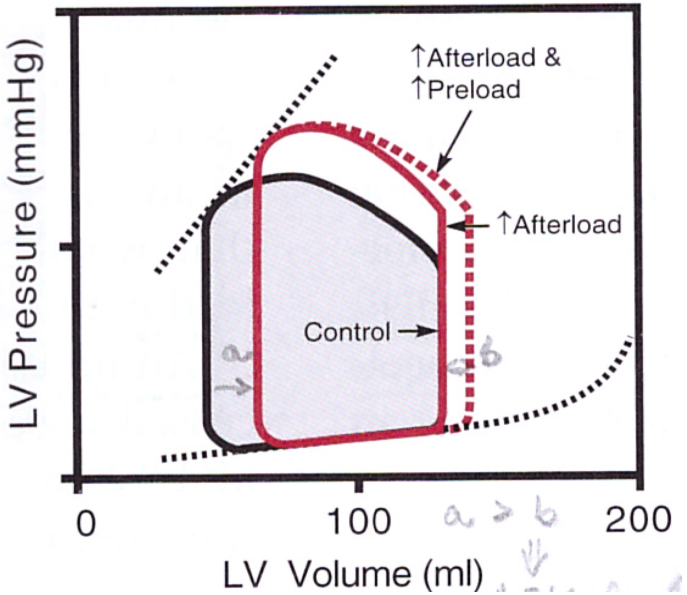
- increased SV and CO
- SVR not changed
- Primal vascular tone caused to relative increase of afterload:



Interaction: preload, afterload, inotropy

Increased afterload:

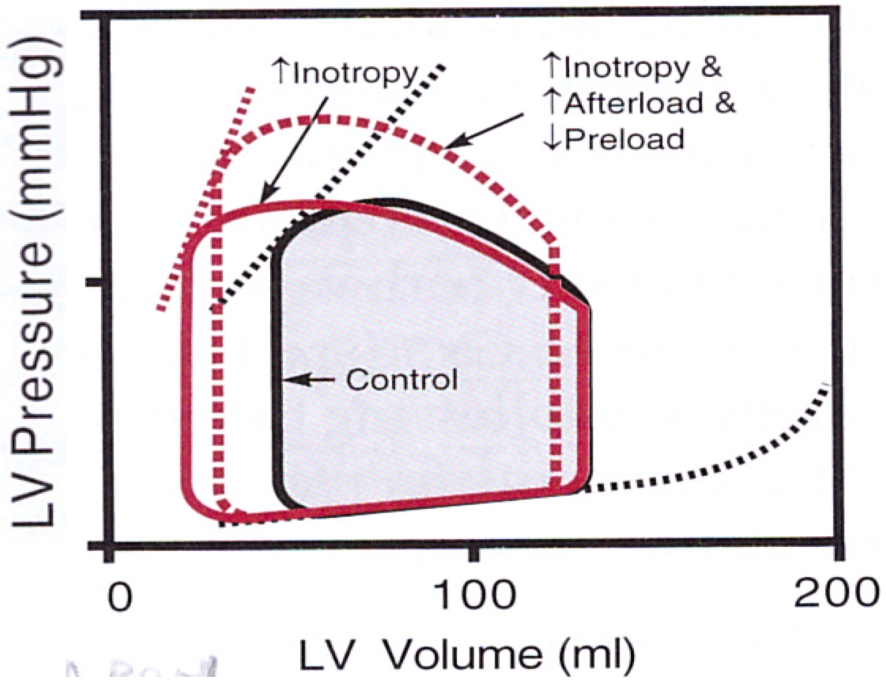
- decreased SV and CO
- decreased CO is not able pumps whole Venous Return
- Increased Venous Return leads to increase preload



Interaction: preload, afterload, inotropy

Increased inotropy:

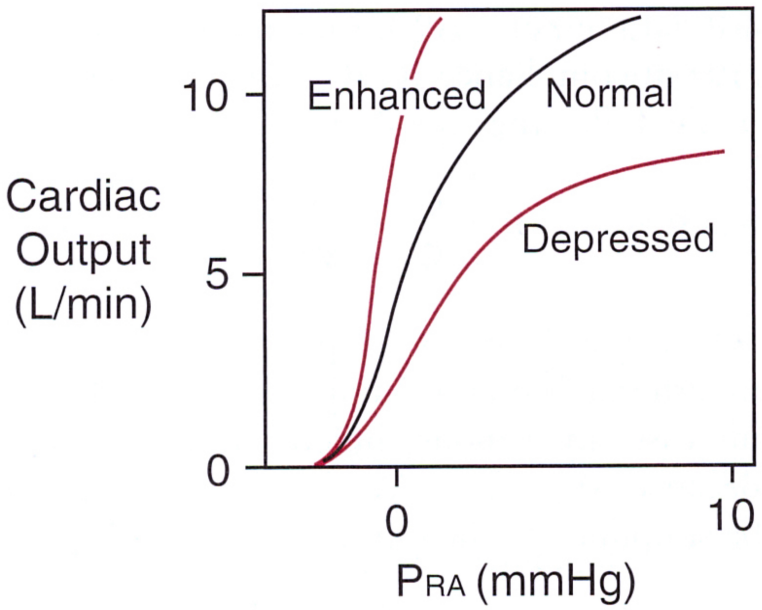
- Increased CO and decreased ESV
- SVR not changed
- Relative increased afterload
- Increased afterload caused decreased contractility
- Decreased contractility caused decreased preload



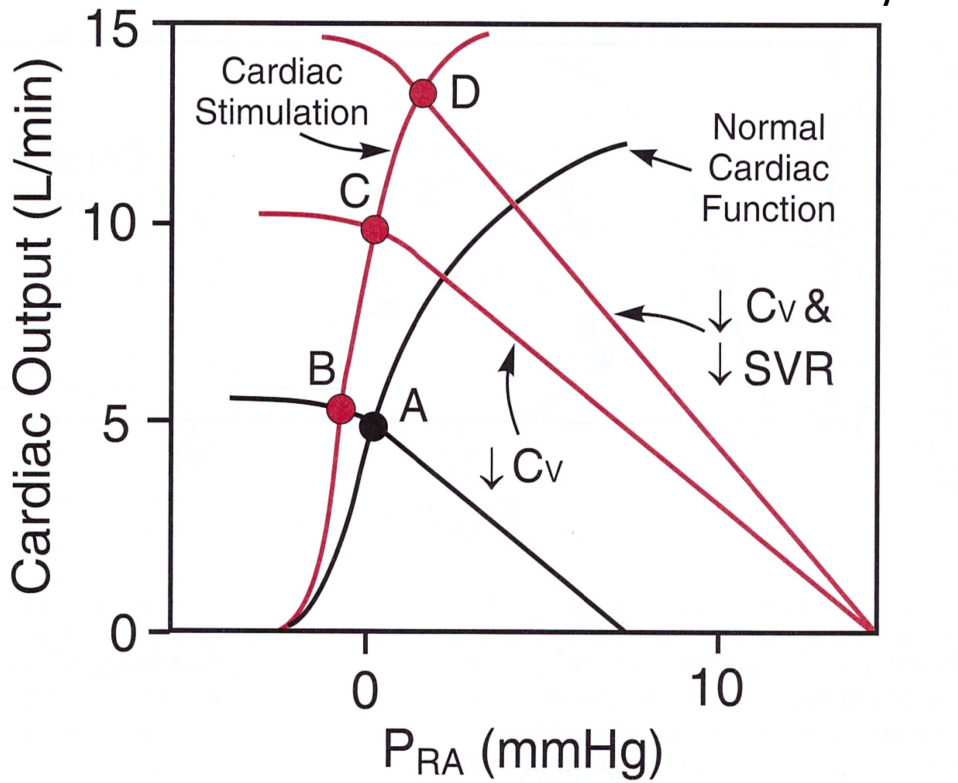
Increased sympathetic activation

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

Srdeční funkční křivka



Kombinace srdeční a cévní funkční křivky

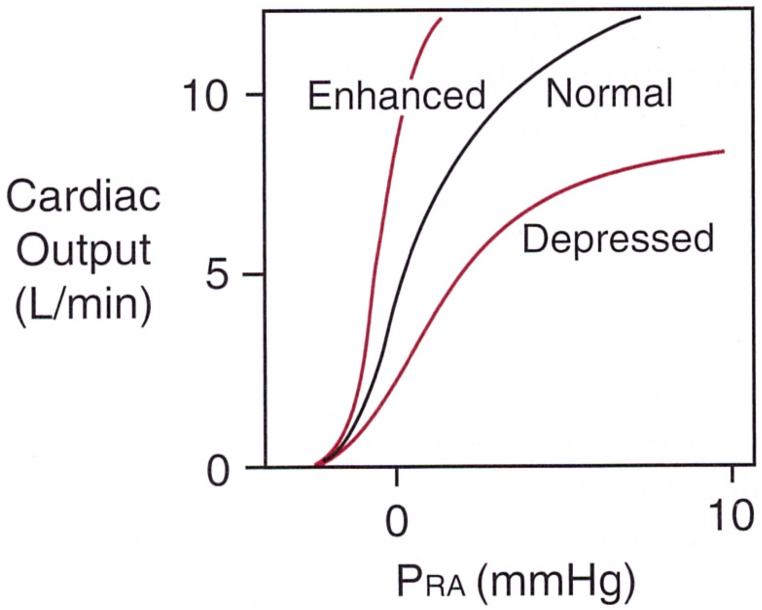


A – equilibrium between CO anad Venous Return

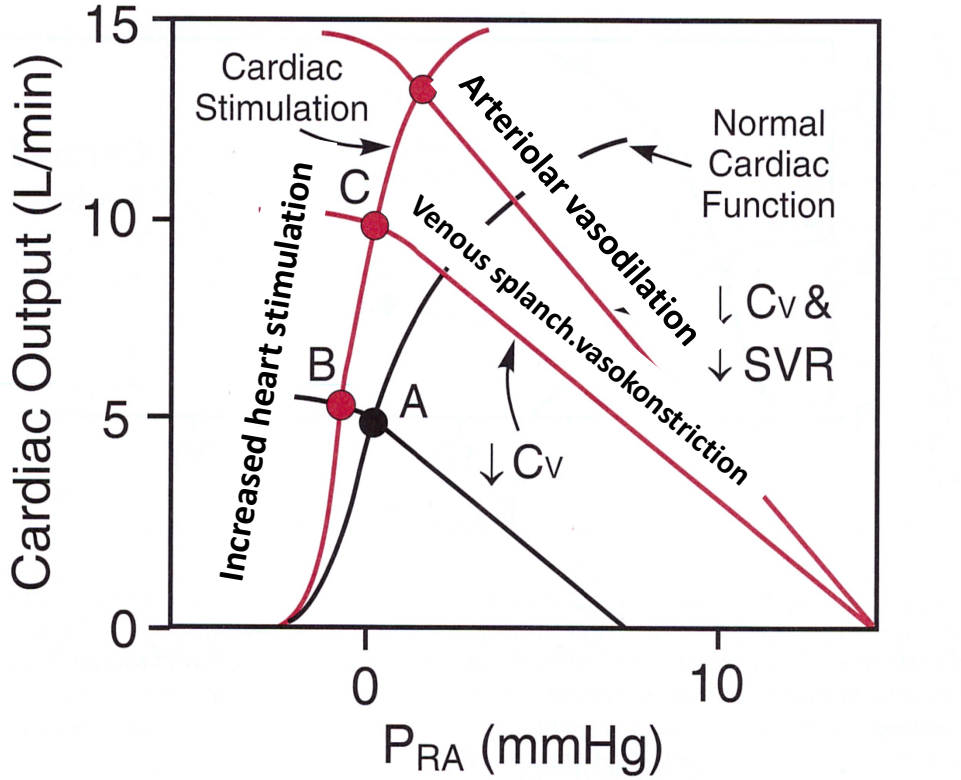
Increased sympathetic activation

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

The heart curve



the heart and the vascular curve interaction



A – equilibrium between CO anad Venous Return

Heart failure