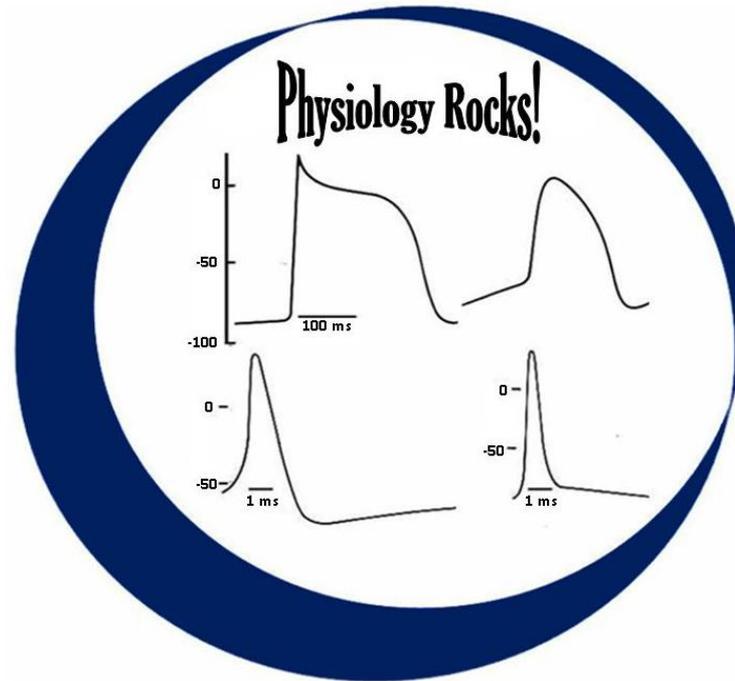


Lecture 8

Determinants of Cardiac Function

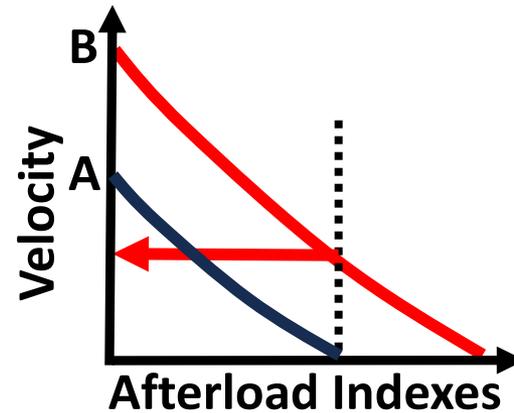
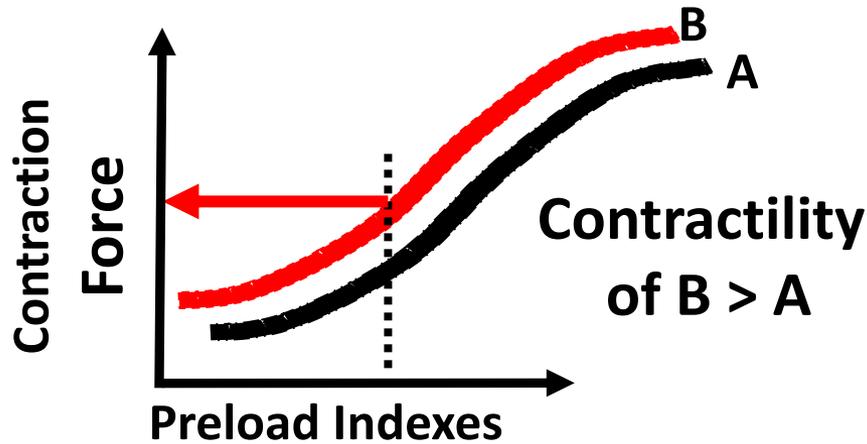


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Topics

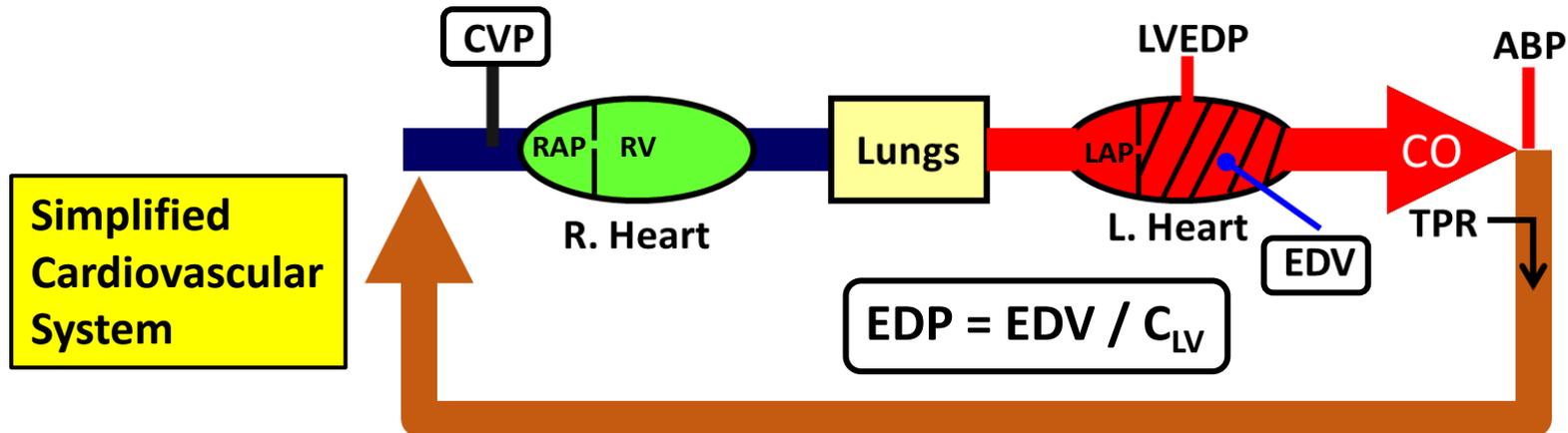
- Preload and afterload as determinants
- Myocardial wall stress as a factor
- Wall stress as afterload
- Determinants of cardiac output
- The cardiac cycle overview
- Stroke volume dependence on Frank-Starling process
- Contractility and the cardiac function curve
- Measures of myocardial energy demand
- Clinical correlation – E and A parameters
- Respiration-related dependencies
- Intramyocardial pressures as determinants
- Interactive multiple choice review questions

Preload and Afterload Determinants

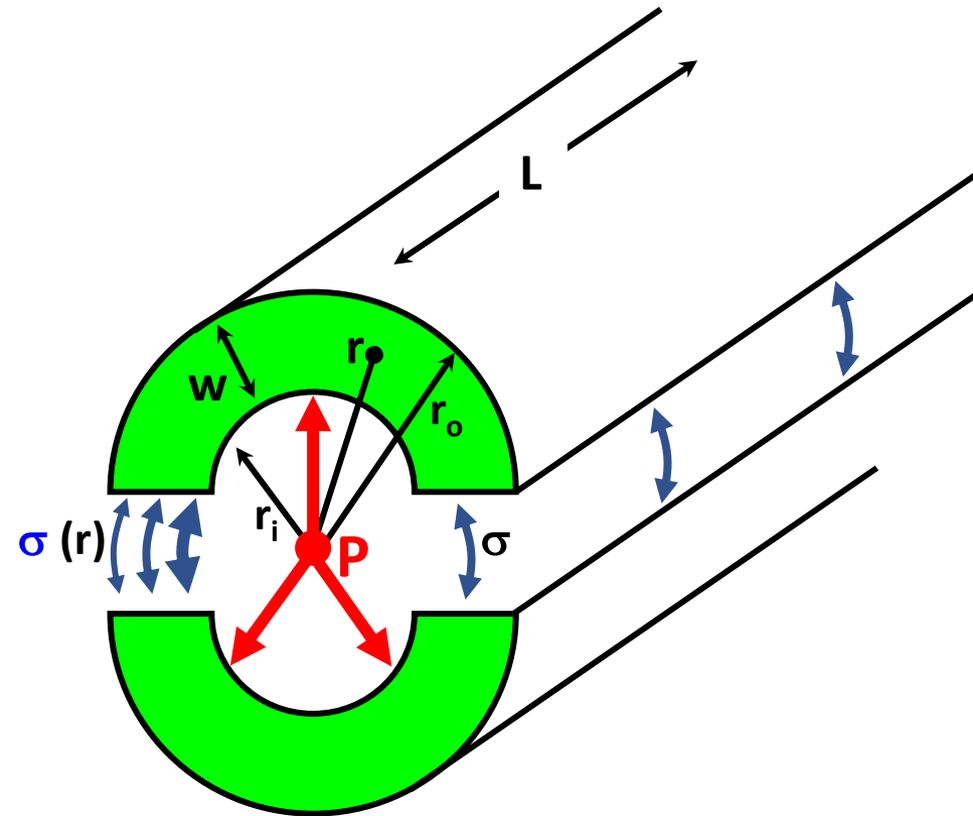


- **EDV**: LV volume at start of contraction-Best
- **EDP**: LV pressure at start of contraction -2nd
- **CVP**: Central venous pressure – ease measure

- **Wall Stress** ([next slide](#))
- **TPR**: $(MAP - CVP) / CO$
- **Aortic Blood Pressure**



Wall Stress as a Factor



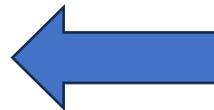
- Pressure (**P**) causes an outward force (**F**) tending to expand the vessel. The force acts over the length of the vessel (**L**).
- To hold the two halves together, there is a stress (σ) in the vessel wall acting in opposition to the distending force

$$\text{The distending force/L} = P \times \pi r_i^2$$

$$\text{The restoring force/L} = \sigma \times \pi r_i w$$

- Equate forces for an equilibrium: yielding $\sigma = (P \times r_i) / w = \text{modified Laplace's Law}$
 σ is the average stress in the wall

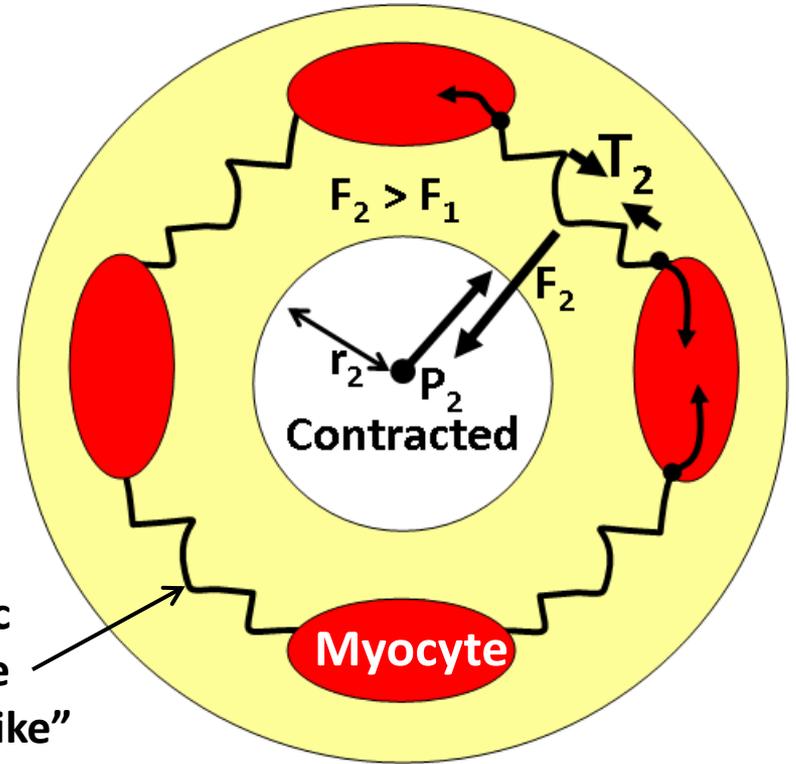
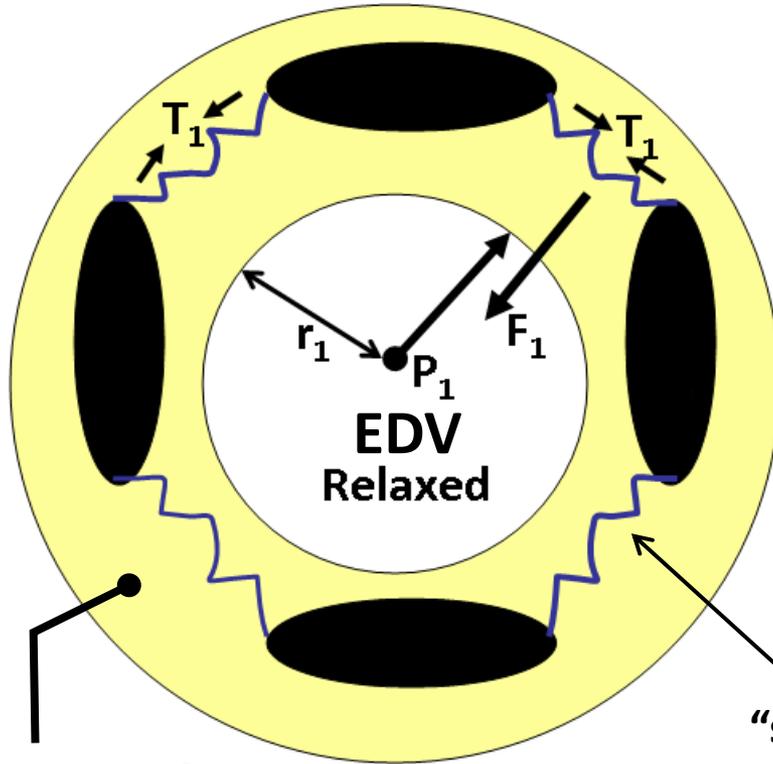
$$\sigma(r) = \frac{Pr_i^2 (1 + r_o^2/r^2)}{(r_o^2 - r_i^2)}$$



$$\sigma_{\text{avg}} = P r / w$$

- The radial distribution of stress $\sigma(r)$ is greatest at r_i and diminishes through the wall, becoming least at r_o

Wall Stress as the True Afterload



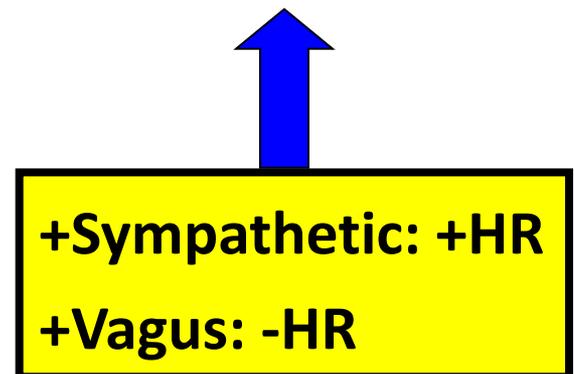
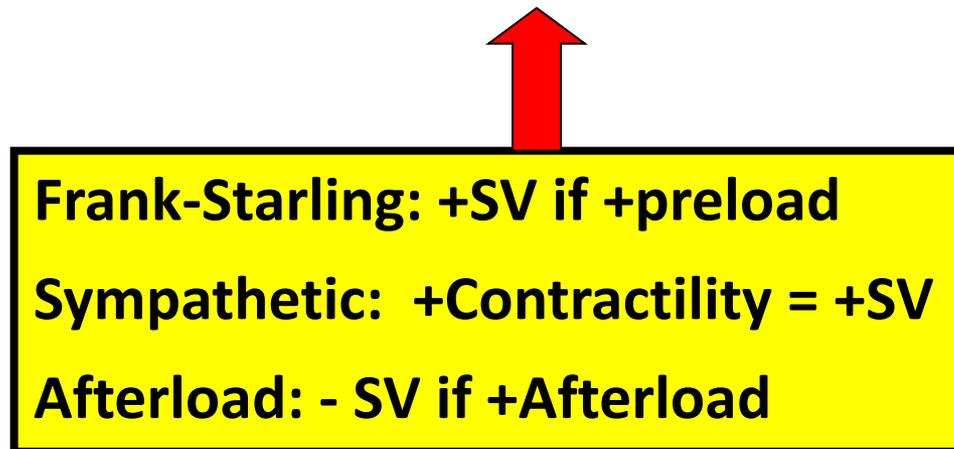
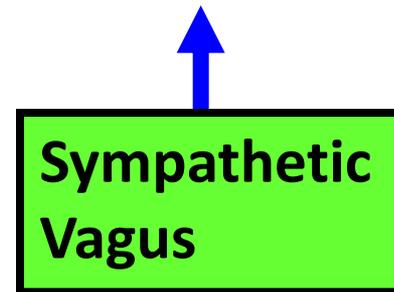
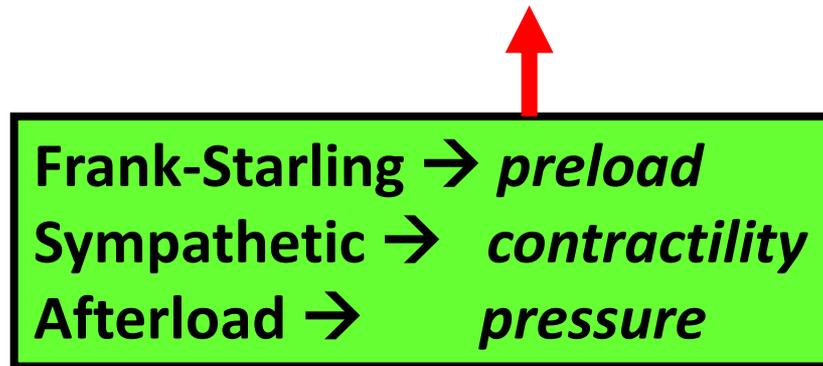
Myocardium

- Consider (T) the tension in the elastic wall elements
- Outward force/area due to P_1
- Inward force/area due to wall $T = F_1$
- Equal and opposite at equilibrium

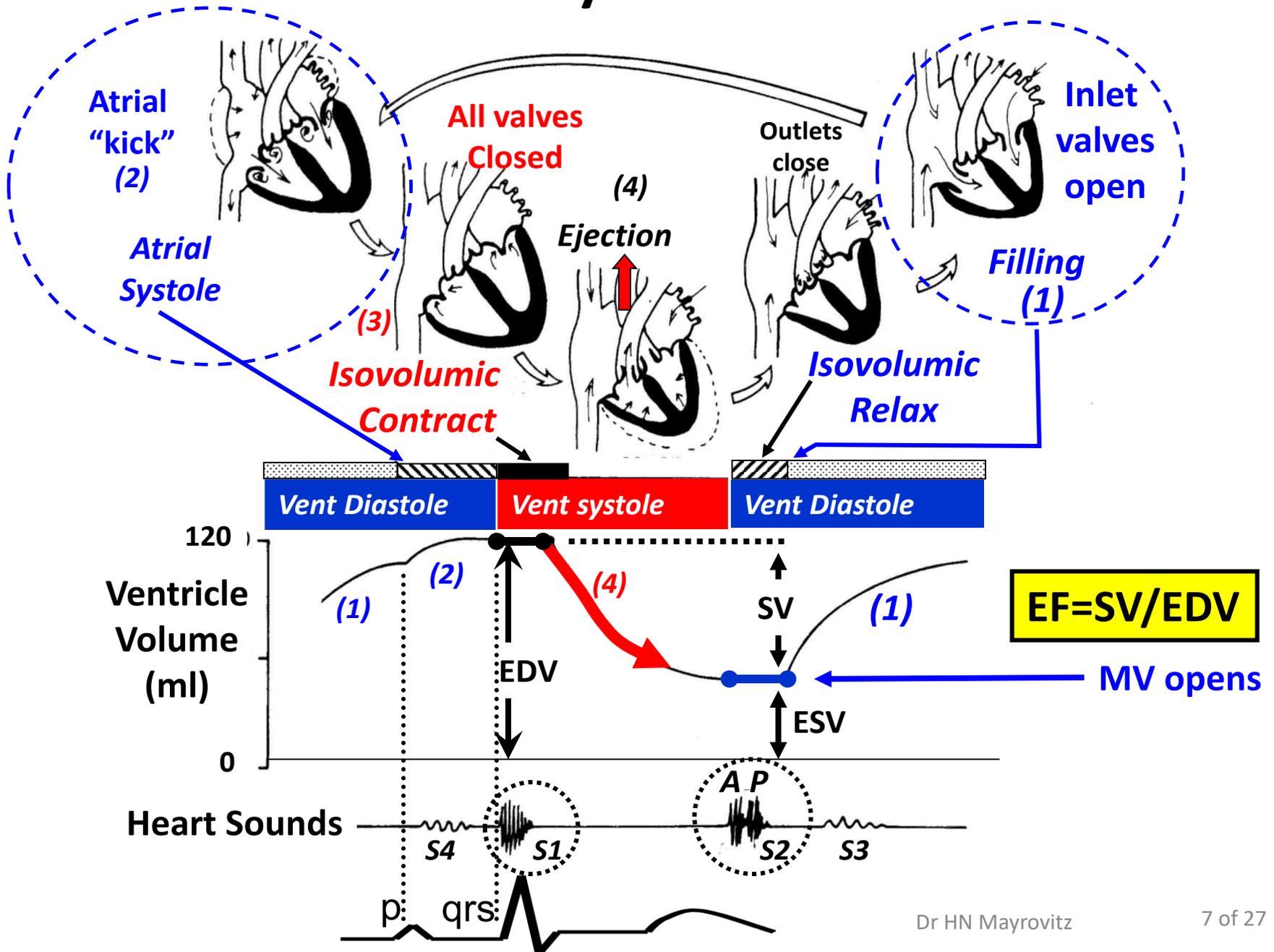
- Muscle contracts (systole)
- To shorten ... myocyte must overcome tension (Wall Stress)
AFTERLOAD
- Inward radial force increases (F_2)
- Chamber Radius decreases (r)
- LV Pressure increases
- Blood is ejected (Stroke Volume)

Determinants of Cardiac Output

$$CO = SV \times HR$$



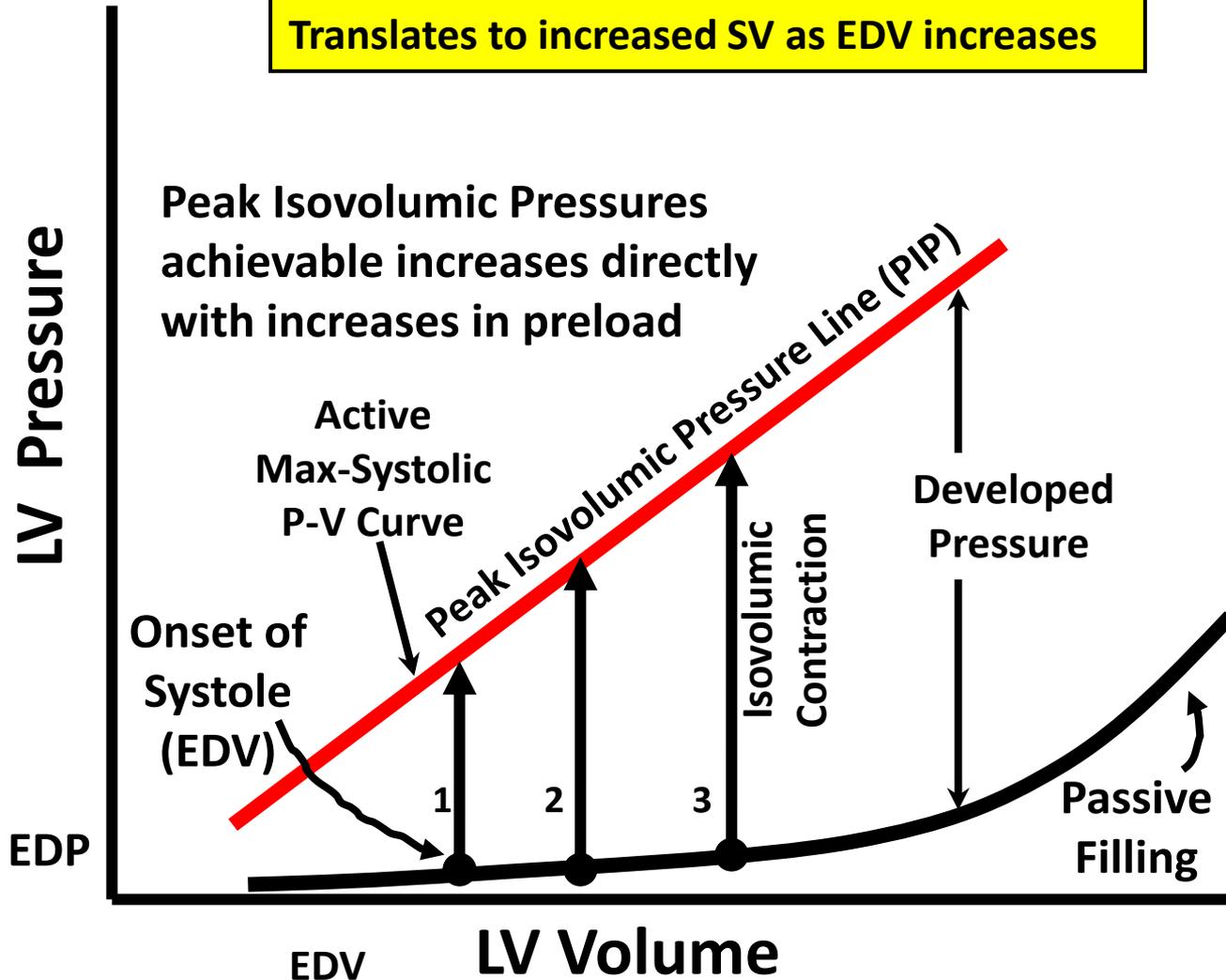
Cardiac Cycle Overview



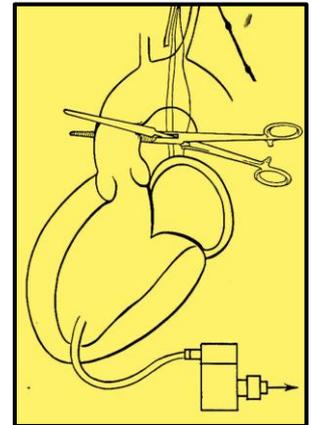
Frank-Starling "Law" of the Heart

Preload Dependent

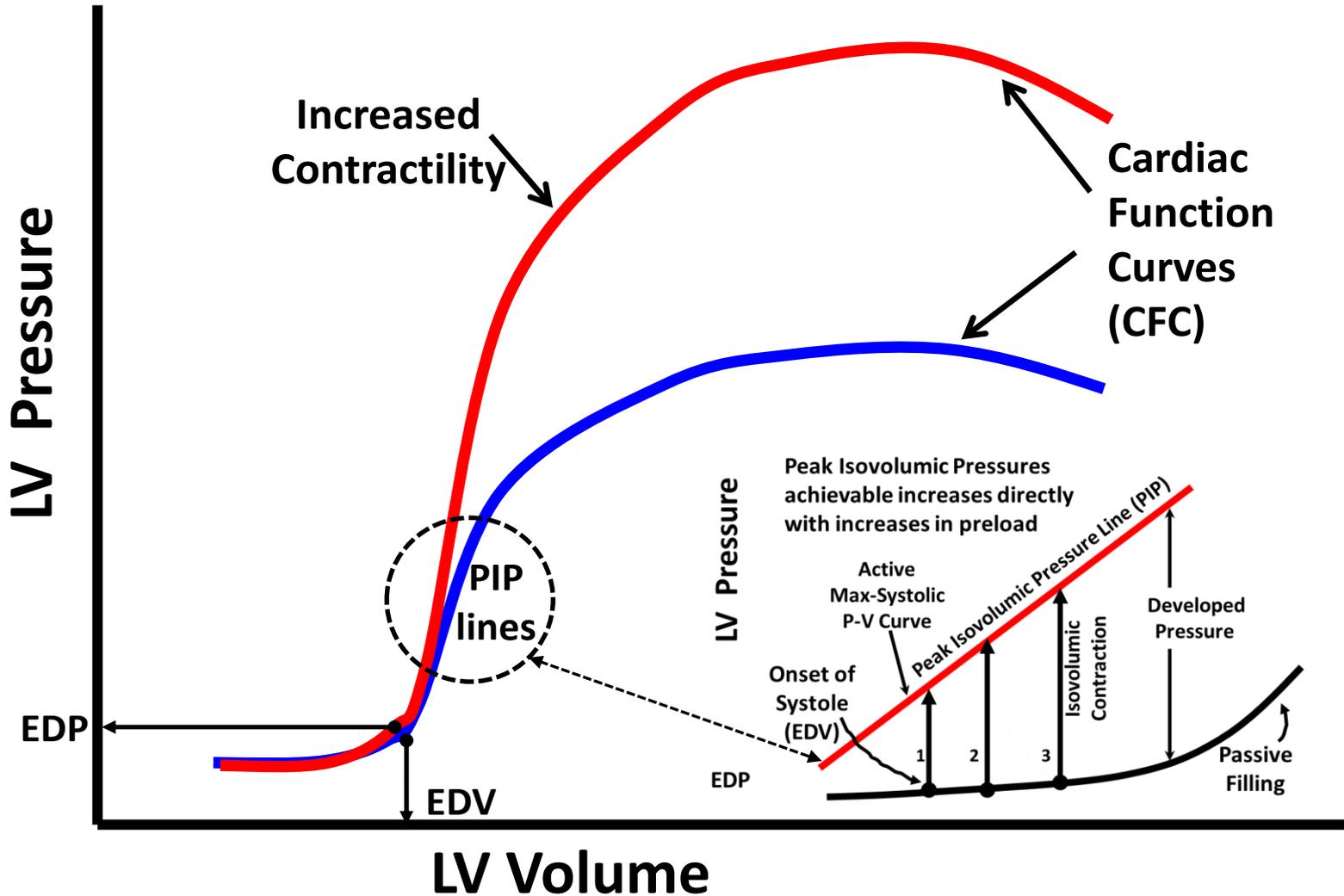
Contraction force increases as EDV increases
Translates to increased SV as EDV increases



- *Isolated Heart*
- *Ejection prevented*
- *Contractions are isovolumic*

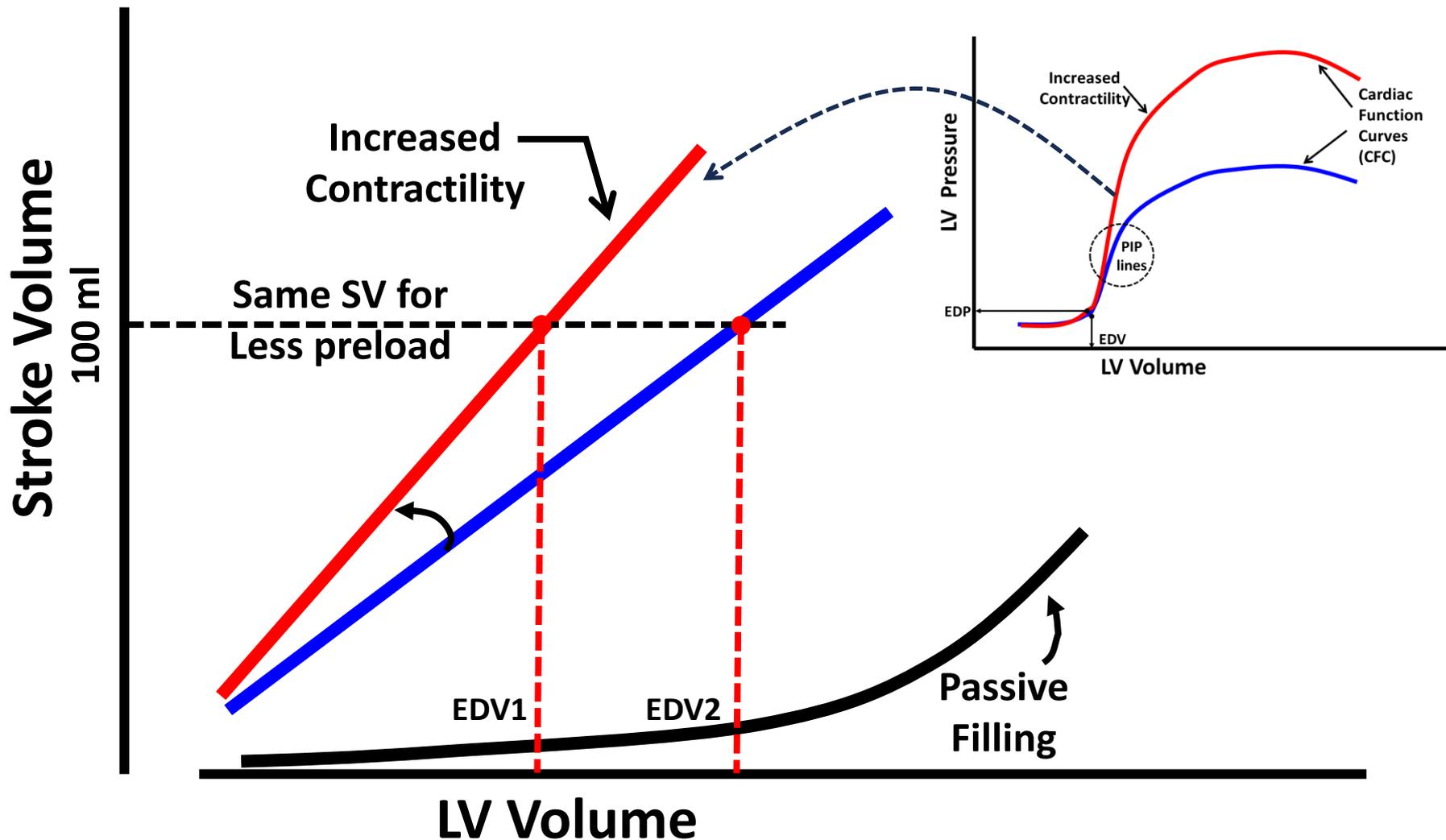


PIP Lines as part of the Cardiac Function Curves

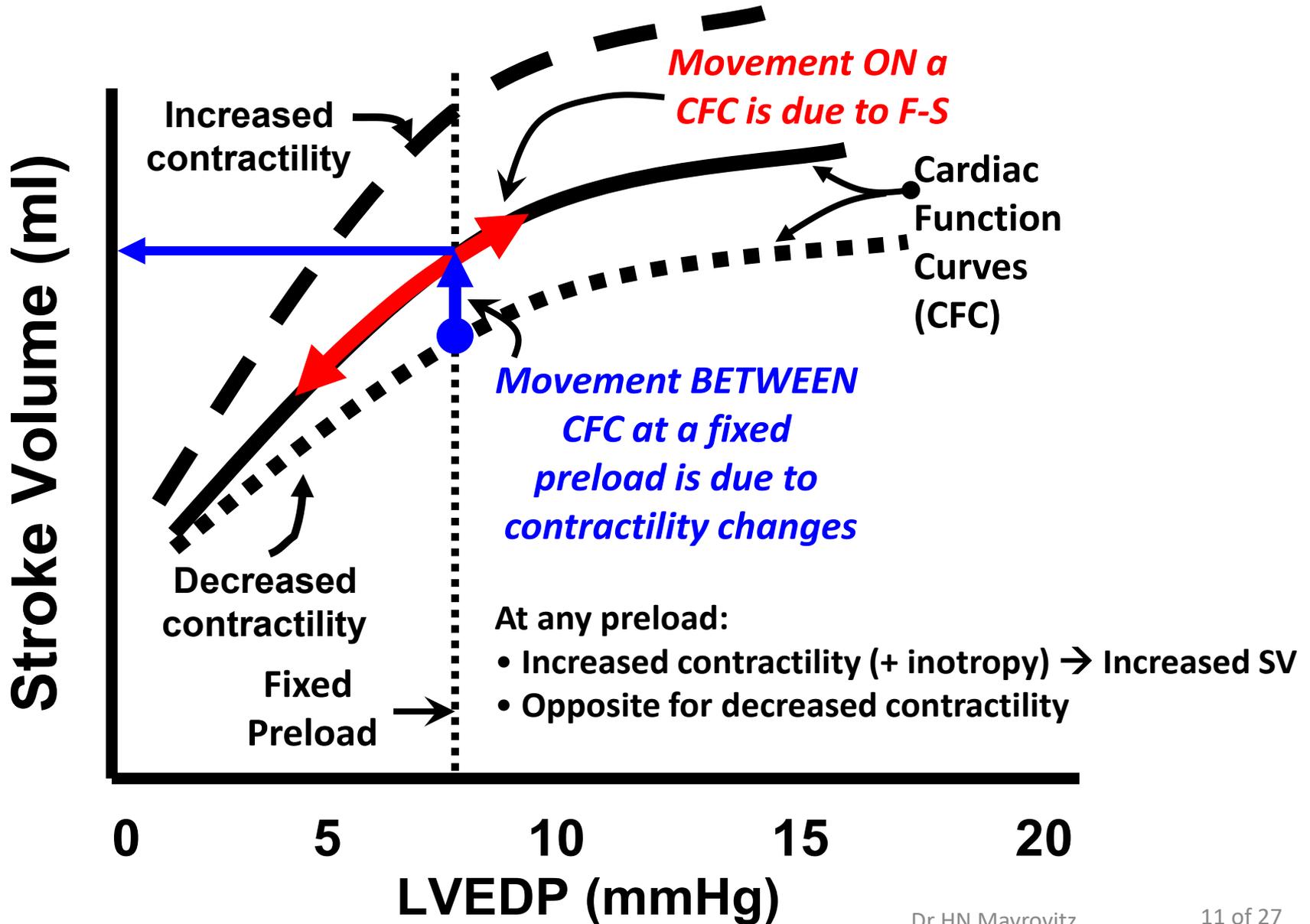


Effects of Contractility on PIP Line

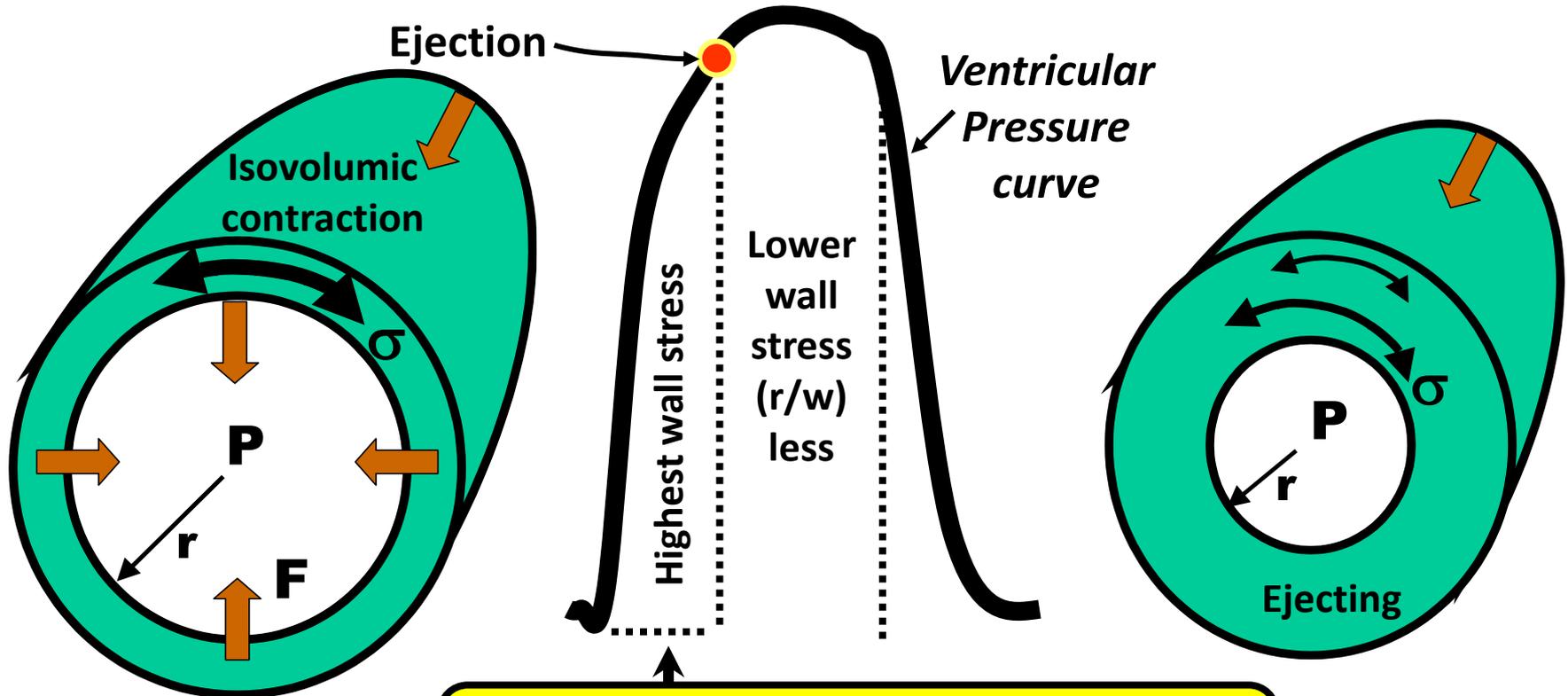
Increased contractility increases PIP line slope
Translates to increased SV as contractility increases



Frank-Starling vs. Contractility



Ventricular Muscle's Load and Energy Demand



Afterload \rightarrow Wall Stress $\sigma = P (r / w)$
Energy Need \sim Stress x Time

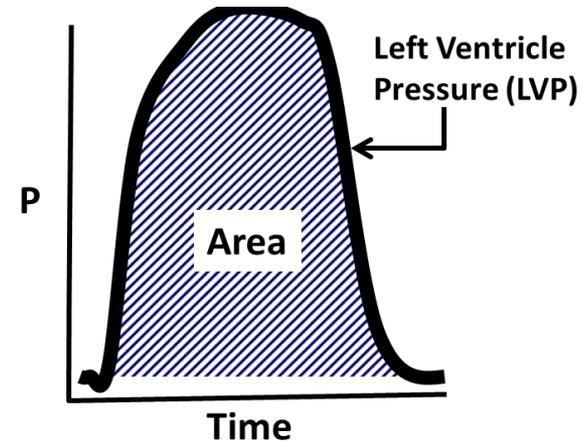
- Large O_2 demand during isovolumic contraction (large P and large r/w) = large σ
- Increased with elevated P (Aortic stenosis or HTN) and duration of high stress
- O_2 demand during ejection also increased in conditions with elevated P

Measures of Ventricle Energy Demand

- Area under the P – T curve

Increased P

Increased duration



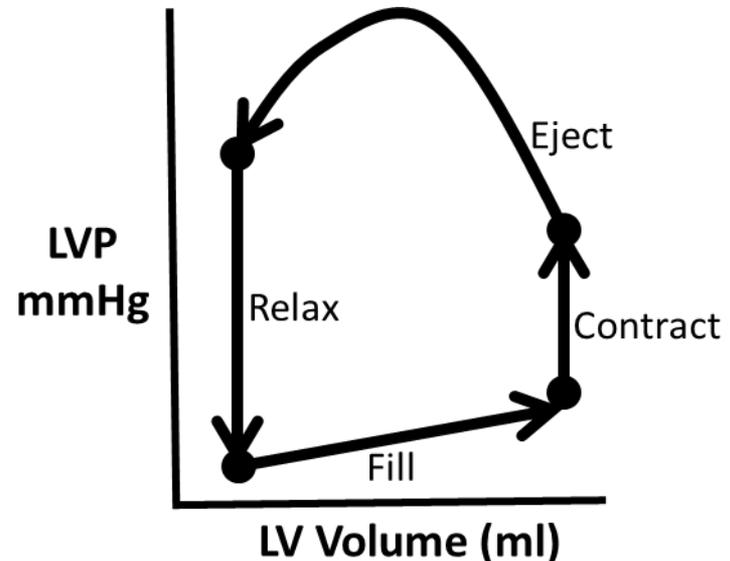
- Tension Time Integral (TTI)

$$\int \sigma(t) = \int [P(t) \times r(t) / w(t)]$$

- Double product (MAP X HR)

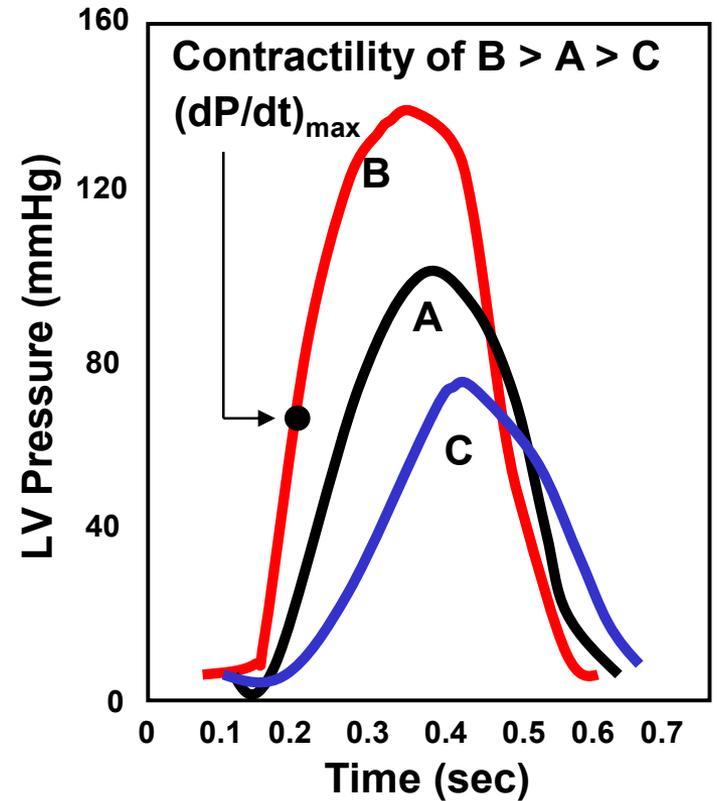
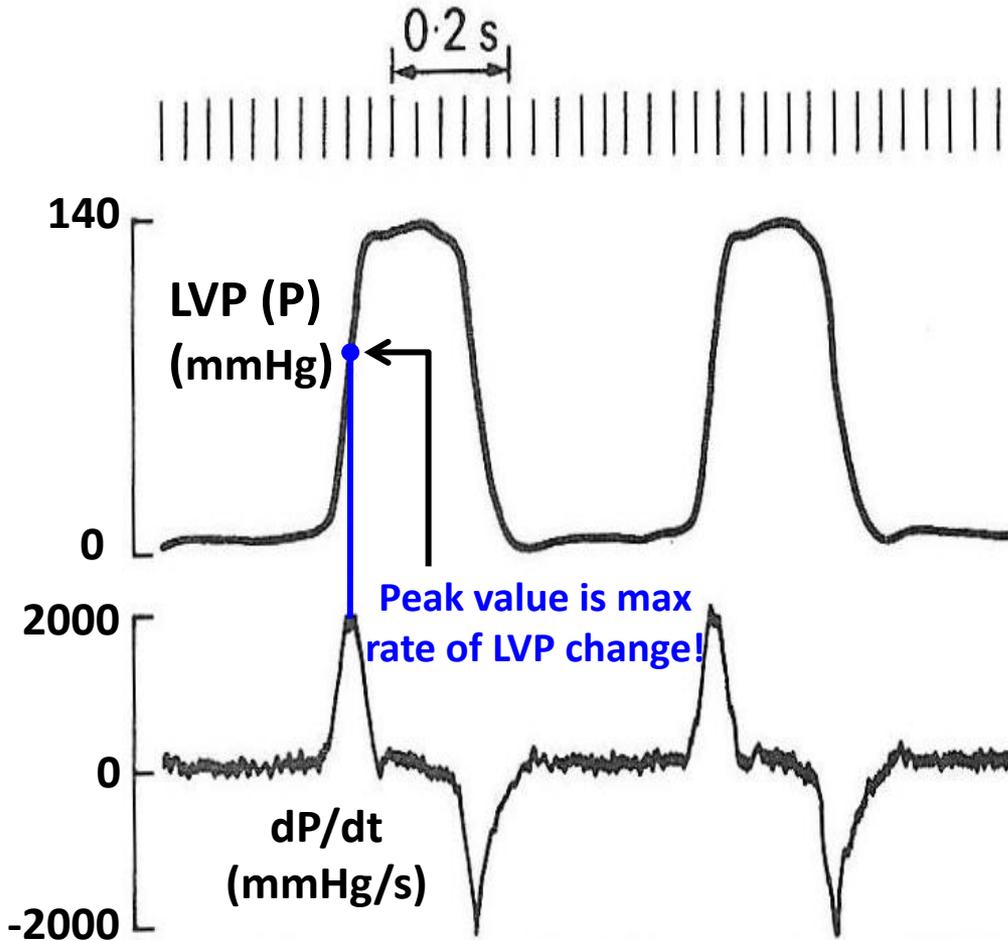
Clinically Measurable

Clinically Useful

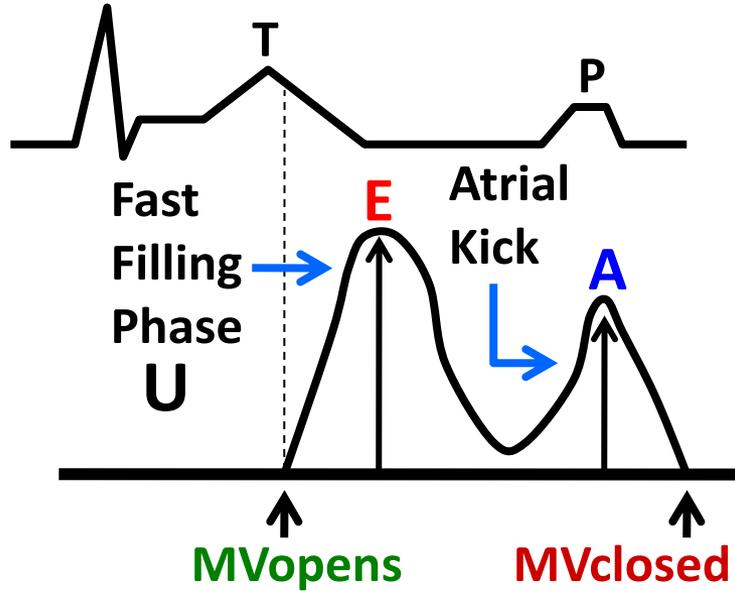
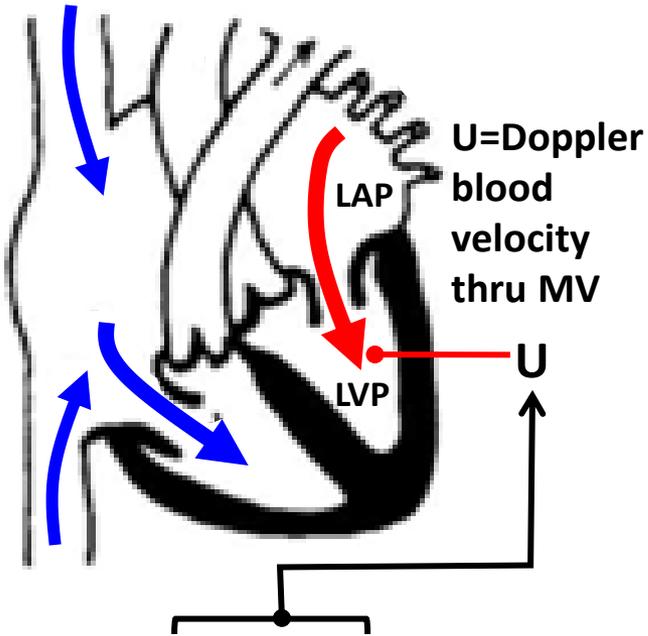


$$\text{Stroke Work} = \text{loop area} = \int PdV$$

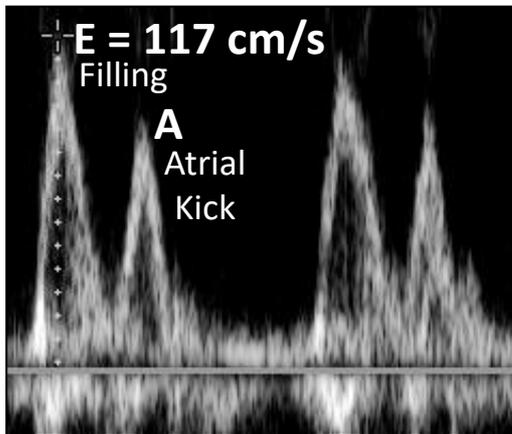
Measure of Pump Function (LV dP/dt)



Clinical Correlation: LV Diastolic Filling Parameter

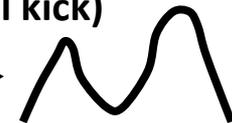


- E is peak U during early fast filling
- A is peak U during atrial contraction
- E depends on
 - LAP-LVP gradient
 - LVP Stiffness
- E/A is age dependent
21-40 (1.5), >60 (1.0)

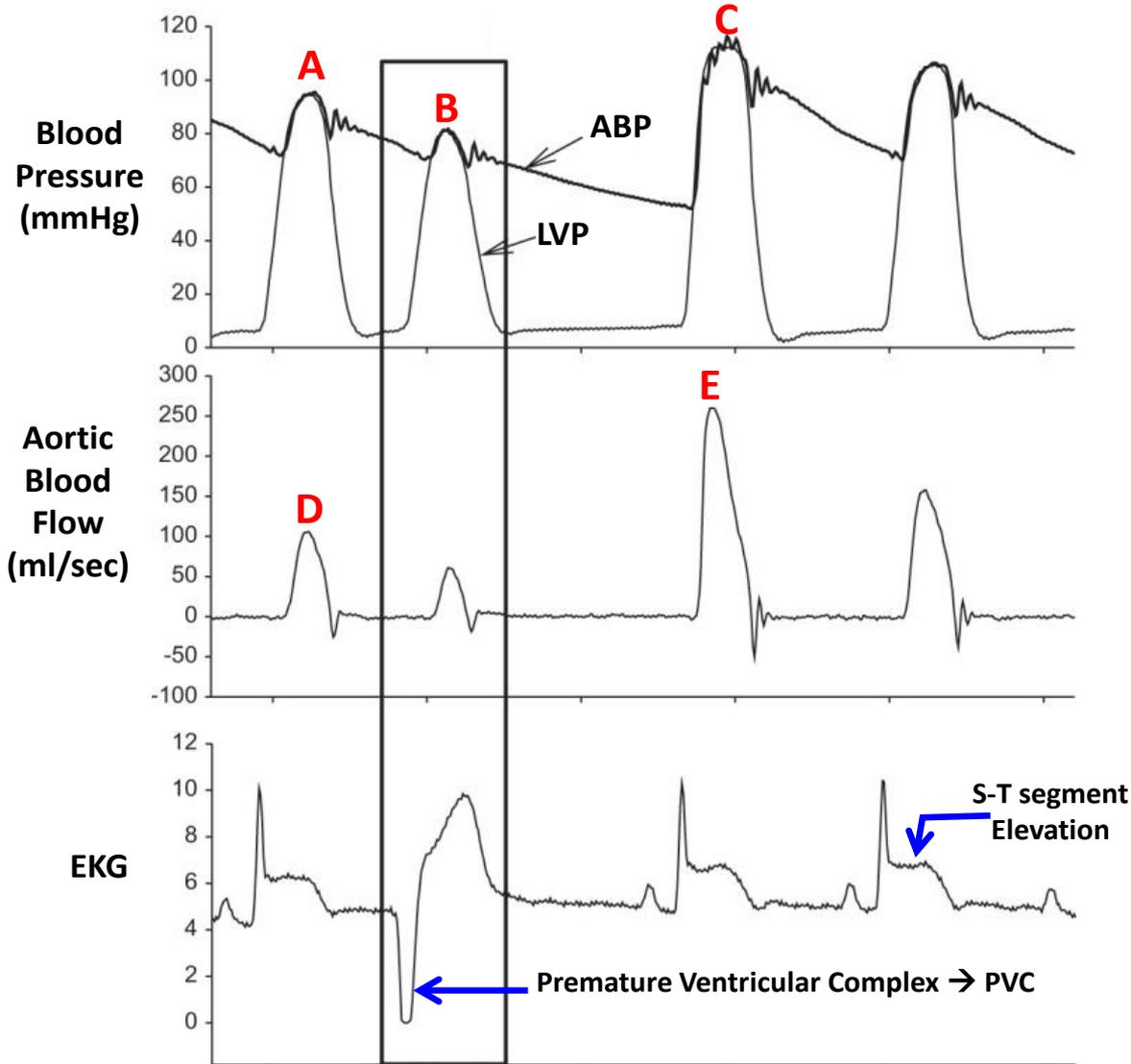


- Stiffness can be functional or structural
 - Functional: Impaired relaxation (Grade I)
 - Structural: Fibrosis or LVH (thick-walled)

- Increased Stiffness → decreased C → +LVP
 - E decreases (LAP-LVP decreased → early filling reduced)
 - A increases (more LA volume left for atrial kick)
 - E/A decreases usually < 1

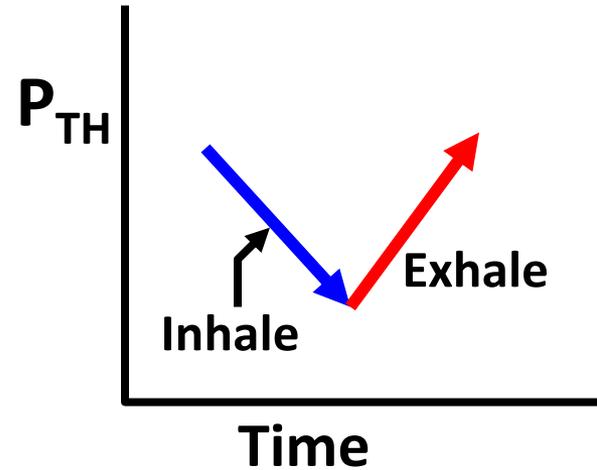
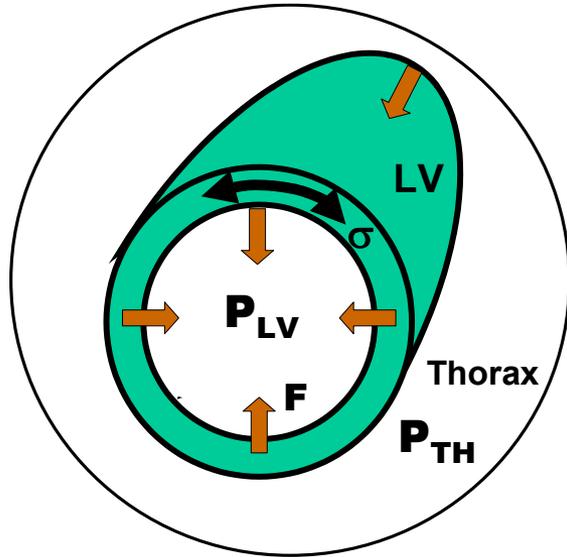


Clinical correlation: Patient with MI and PVC



1. Why is ABP less at B than A?
2. Why is ABP greater at C than A?
3. Why is flow greater at E than D?

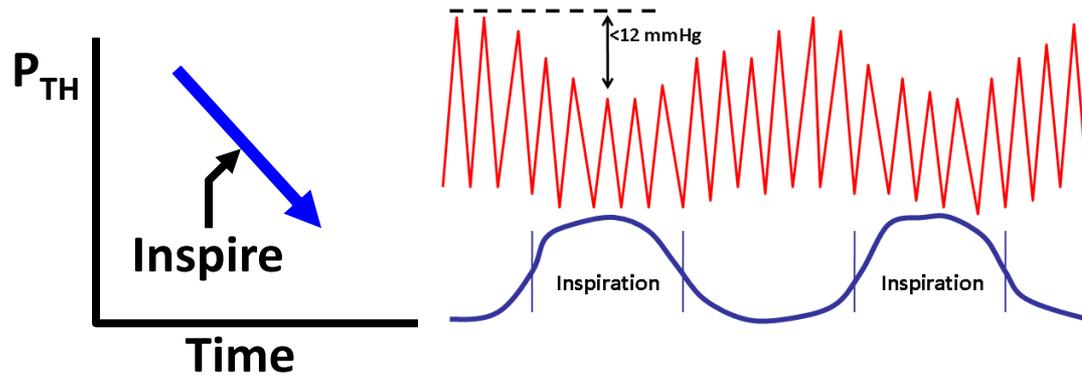
+ Intra-thoracic Pressure → + 'Afterload' Effect



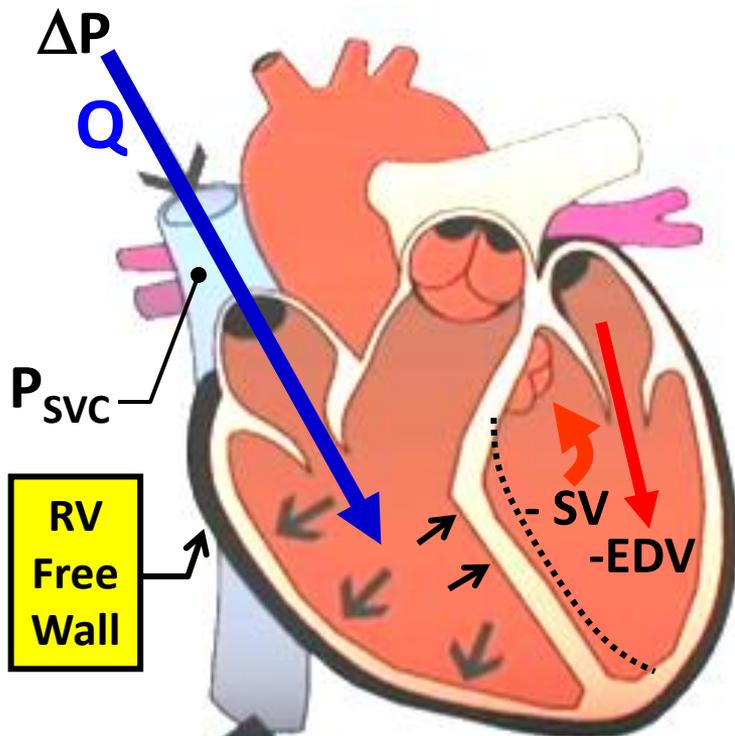
$$\text{Afterload} \rightarrow \sigma = P r / w = (P_{LV} - P_{TH}) (r/w)$$

- A deep inspiration against a closed glottis decreases P_{TH} substantially
- LV transmural pressure increases substantially
- Effective afterload Increases
- Stroke volume reduced

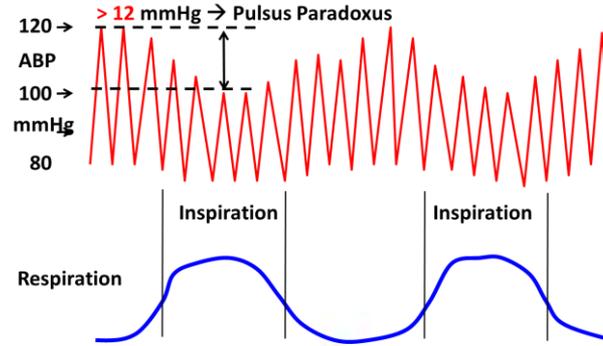
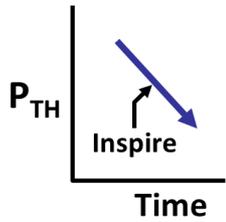
Normal Variation in Blood Pressure with Respiration



- During inspiration blood returning to the RV increases RV pressure causing a slight displacement of the septum into the LV
- Diastolic filling of the LV is thus slightly reduced as evidenced by a decreased EDV
- This preload reduction is associated with a decrease in SV (Frank-Starling)
- The decreased SV is associated with a decrease in systolic pressure
- Pressure decreases not greater than about 12 mmHg are considered within the normal range
- Pressure changes > 12 mmHg are defined as Pulsus Paradoxus
- Occurs when limitation in free wall expansion

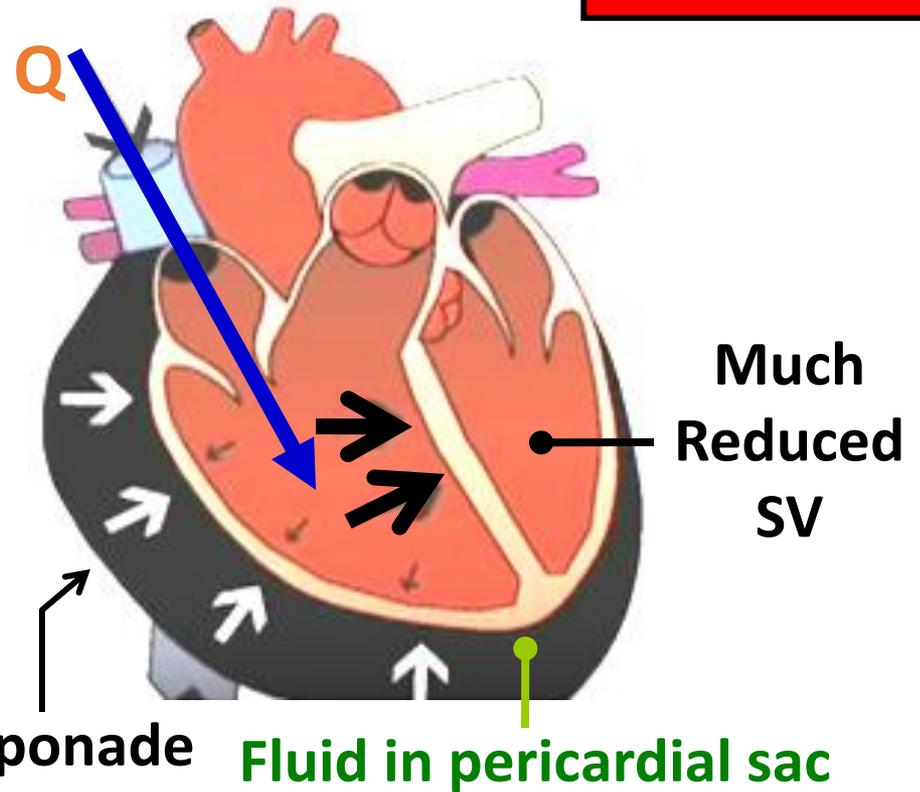
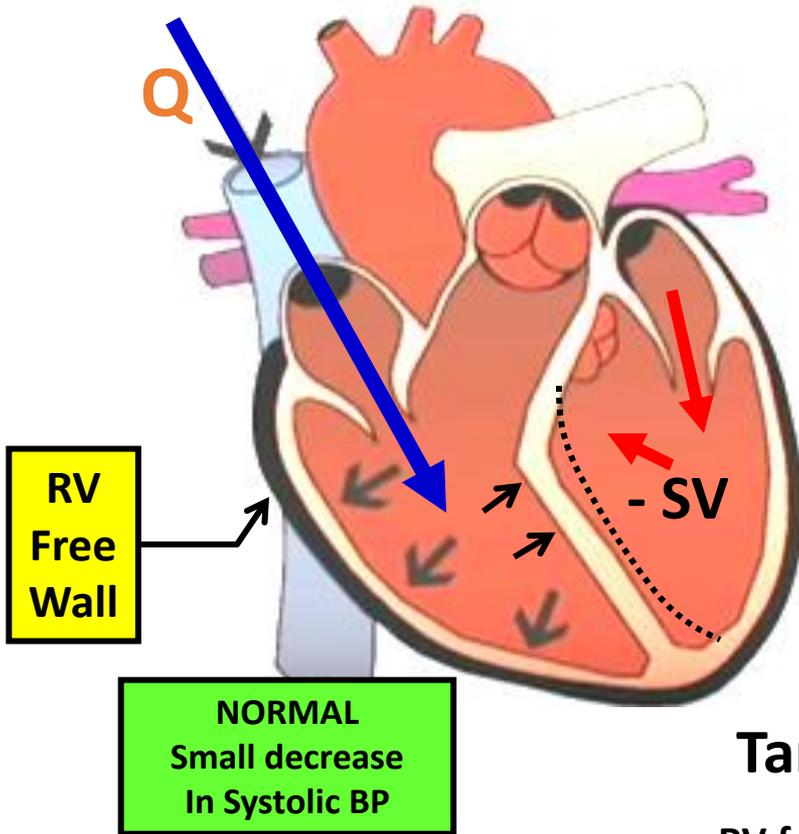


Abnormal Variation is Systolic BP with Respiration



Pulsus Paradoxus

**ABNORMAL
LARGE decrease
In Systolic BP**

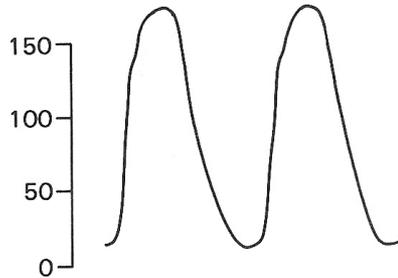


Tamponade

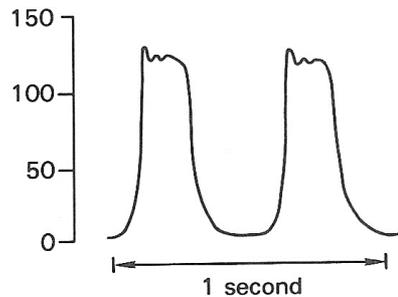
- RV free wall can't easily expand during filling
- RV pressure rises and further displaces (bows) septum inward

Intra-Myocardial Pressures

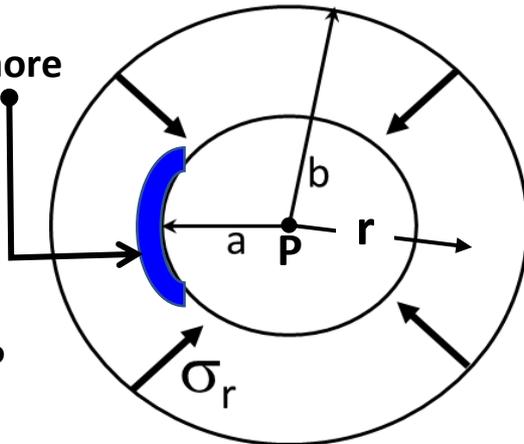
Intramyocardial Pressure (mmHg)



Left Ventricle Pressure (mmHg)



Compressed more
Flow reduced
during systole



@ $r = a$ $\sigma = -P$

@ $r = b$ $\sigma = 0$

$$\sigma_r = [a^2P/(b^2-a^2)] \times (1 - b^2/r^2)$$

- Contraction increases ventricular pressure and intra-myocardial pressure
- Stress in myocardial wall (radial and tangential) with contraction is **greater towards endocardial** vs. epicardial surfaces
- Consequence is that during contraction the blood vessels toward the endocardial surface (subendocardial) are **compressed more** and blood flow is compromised more
- This contributes to the increased **vulnerability** of the endocardial part of the ventricle wall to ischemia and injury when perfusion pressure is reduced
- Also explains why **most of subendocardial blood flow occurs during diastole**

After: Pfluegers Arch 1963;278:181 cp196

Interactive Question



Which change is a normal but usually minor cardiovascular effect of inspiration?

- A. Intrathoracic pressure increases
- B. Ventricular afterload increases
- C. Ventricular transmural pressure decreases
- D. Aortic systolic pressure increases
- E. Aortic pulse pressure increases

Interactive Question



Which of the following is correct regarding the cardiac mean electrical axis (MEA)?

- A. if at an angle of -50 degrees it indicates right axis deviation
- B. its direction tends to shift away from an area of hypertrophied myocardium
- C. a direction that would be considered normal would include +120 degrees
- D. its direction tends to shift in the direction of the side of a bundle branch block
- E. its direction is mainly determined from direction of septal depolarization

Interactive Question



With no sympathetic or parasympathetic impulses to the heart its intrinsic HR in bpm is closest to:

- A. 70
- B. 80
- C. 90
- D. 100
- E. 110

Interactive Question



Which cardiovascular system change would tend to increase systolic blood pressure?

- A. Thinning of arterial walls
- B. Peripheral vascular vasodilation
- C. Reduced sympathetic impulses to the arterioles
- D. Increased compliance of the aorta
- E. Increased elastic modulus of large arteries

Interactive Question



What is a main feature of blood flow (Q) in collapsible vessels such as central veins?

- A. Q depends on the proximal minus the distal pressure
- B. Q is more likely to be turbulent
- C. Q is increased when transmural pressure decreases
- D. Q depends on the proximal minus extravascular pressure
- E. Q is independent of proximal pressure



Interactive Question



Which tends to increase the viscosity of blood flowing in a small vein?

- A. An increase in average blood velocity within the vein
- B. An increase in average shear rate within the vein
- C. An increase in the velocity gradient within the vein
- D. An increase in both the blood's temperature and the average shear rate
- E. An increase in hematocrit

Interactive Question



An ectopic impulse triggers an AP in ventricular myocytes during the myocyte's relative refractory period. Which is a definite result?

- A. A reentrant tachyarrhythmia
- B. An AP that has a rate of rise that is slower than normal
- C. A conducted AP with a slower than normal conduction speed
- D. A conducted AP with an amplitude larger than normal
- E. An AP that is non-conducted or blocked

End CV Physiology Lecture 8