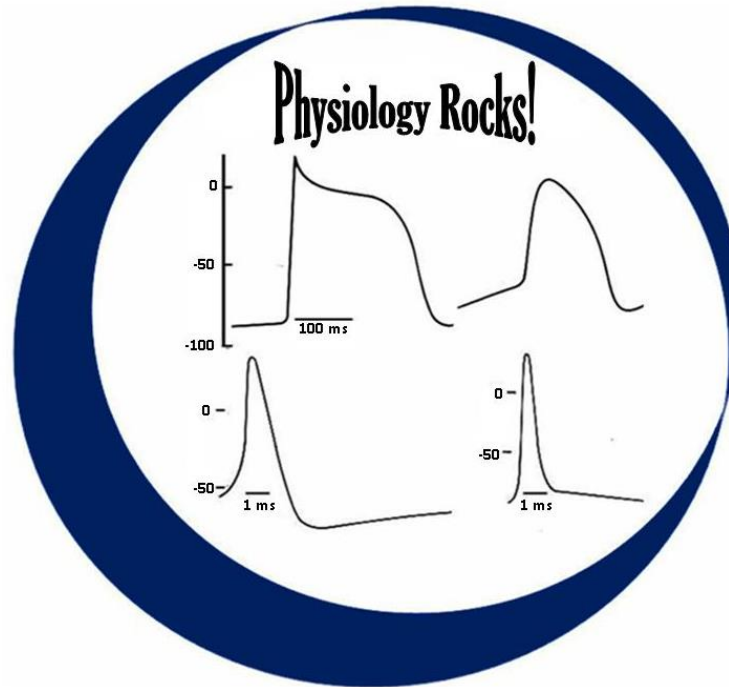


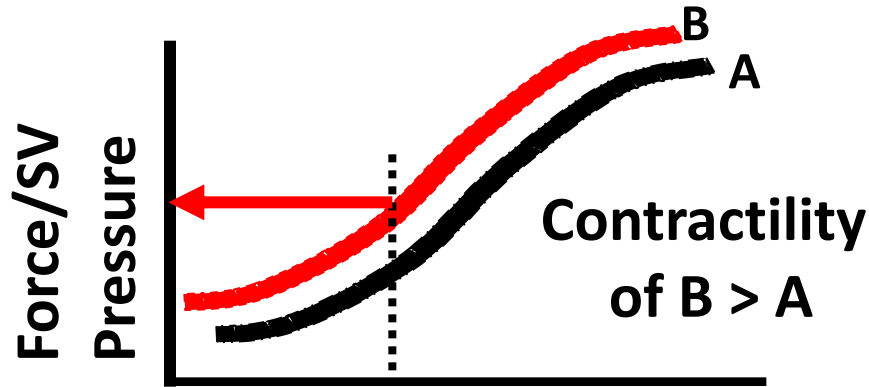
Lecture 23

Determinants of Cardiac Function



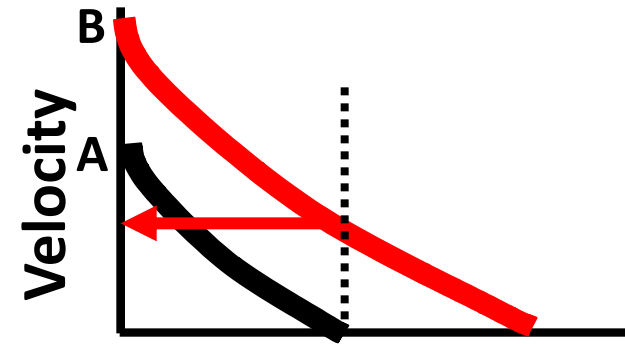
HN Mayrovitz PhD
mayrovit@nova.edu
drmayrovitz.com

Muscle Fiber Mechanics → Cardiac Pump Function



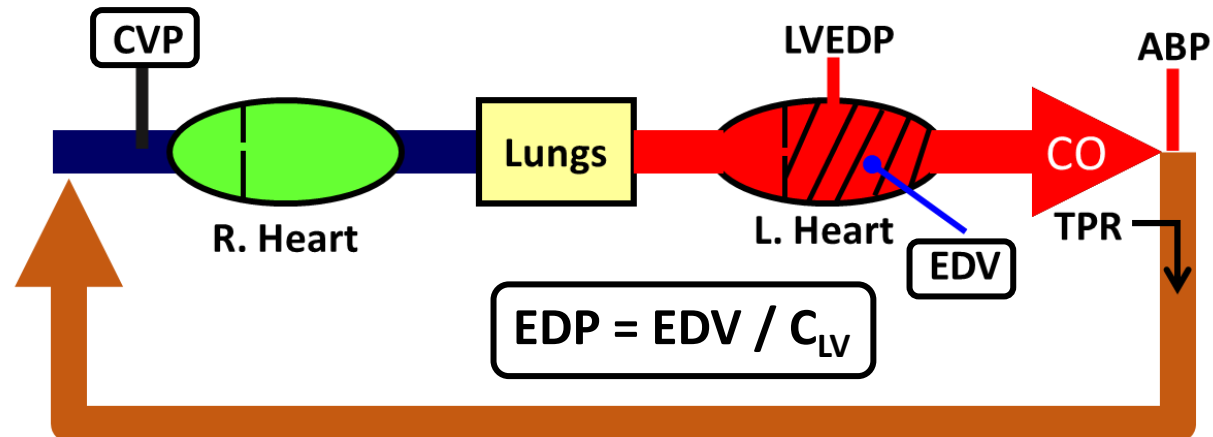
Preload

- EDV
- EDP
- CVP

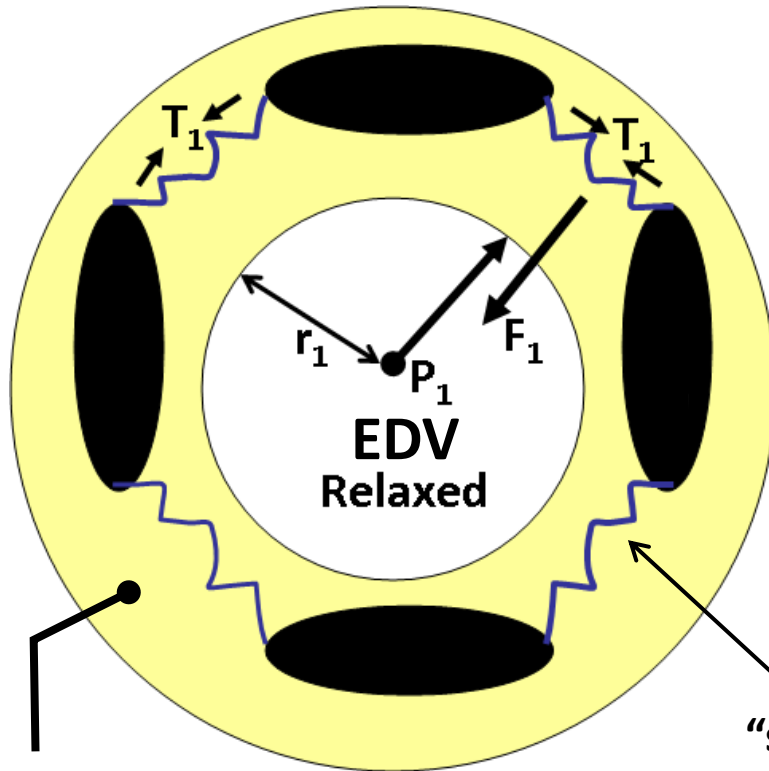


Afterload

- Wall Stress ([next slide](#))
- TPR
- Aortic/Ventricle Pressure

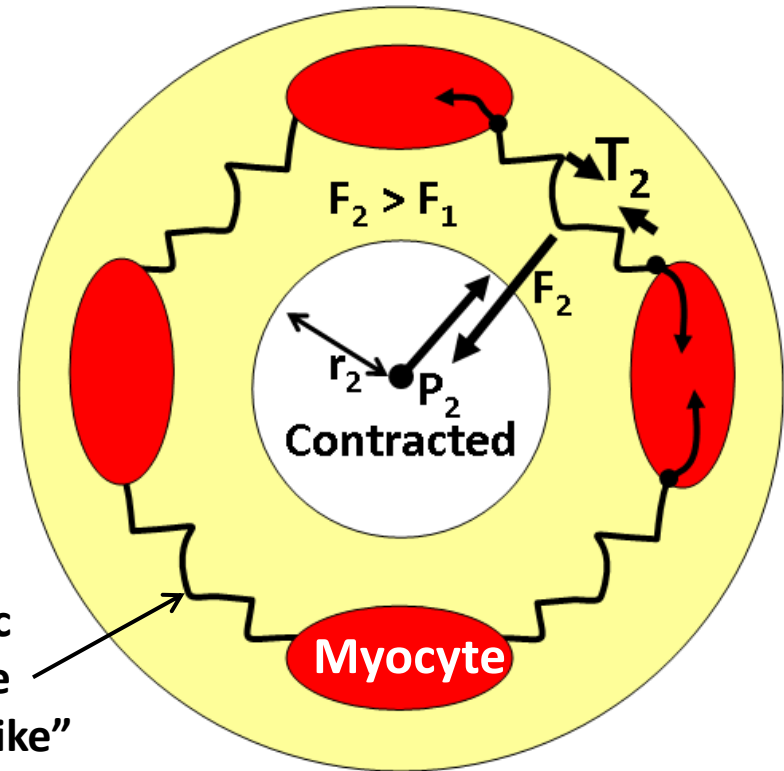


Cardiac Muscle : Stress as Afterload



Myocardium

- Outward force due to P_{TM}
- Inward force due to wall T
- 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

$$CO = SV \times HR$$

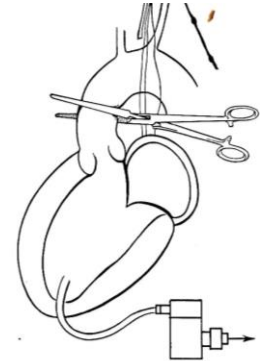
Frank-Starling ~ *preload*
Sympathetic ~ *contractility*
Afterload ~ *pressure*

Sympathetic
Vagus

Frank-Starling: +SV if +preload
Sympathetic: +Contractility = +SV
Afterload: - SV if +Afterload

+Sympathetic: +HR
+Vagus: -HR

Frank-Starling "Law" of the Heart



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

LV Pressure

Peak Isovolumic Pressures
achievable increases directly
with increases in preload

Active
Max-Systolic
P-V Curve

Onset of
systole

Peak Isovolumic Pressure Line

EDP

Isovolumic
Contraction

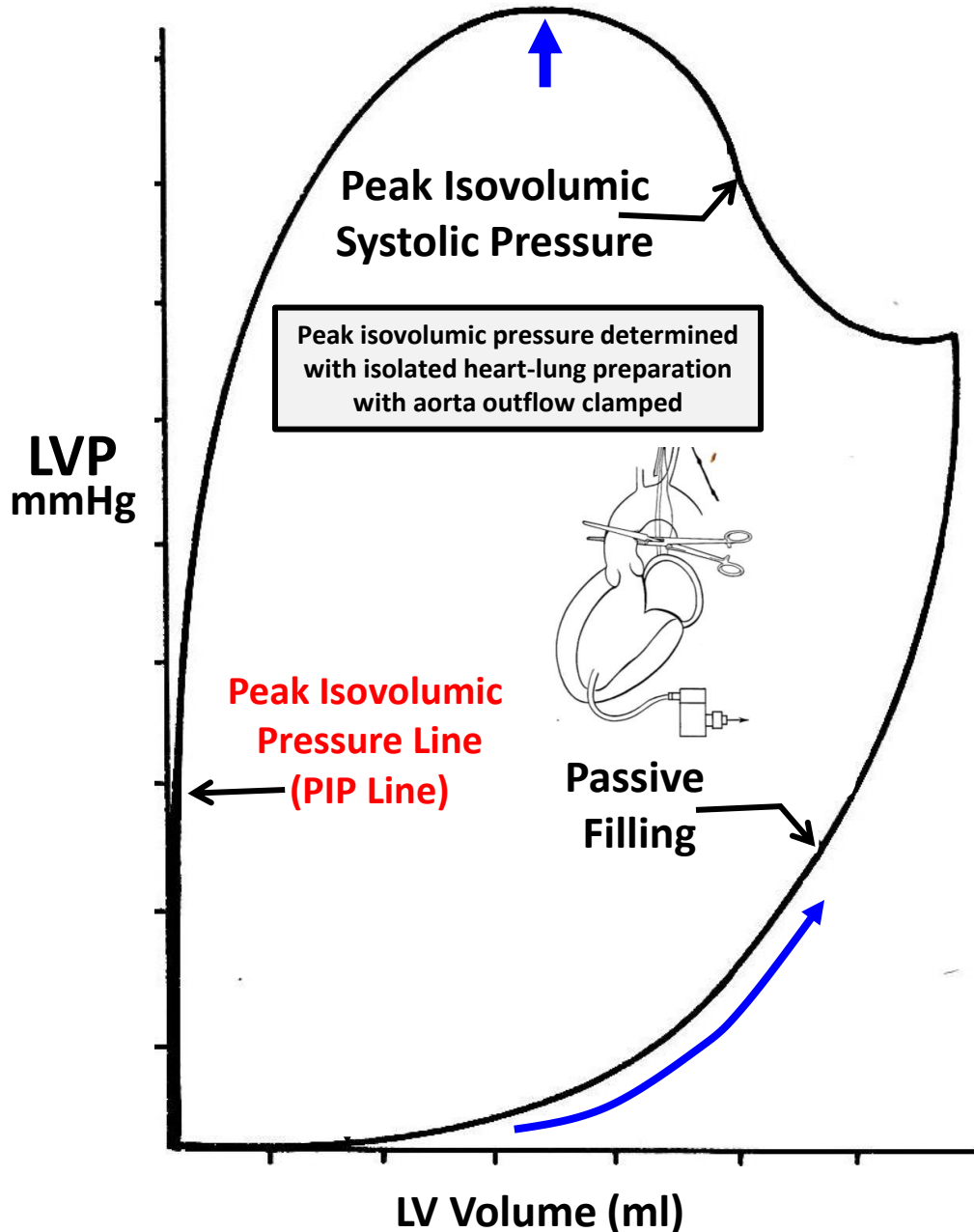
Developed
Pressure

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

Passive
Filling

LV Volume

Max Peak Isovolumic Left Ventricular Pressure (LVP)



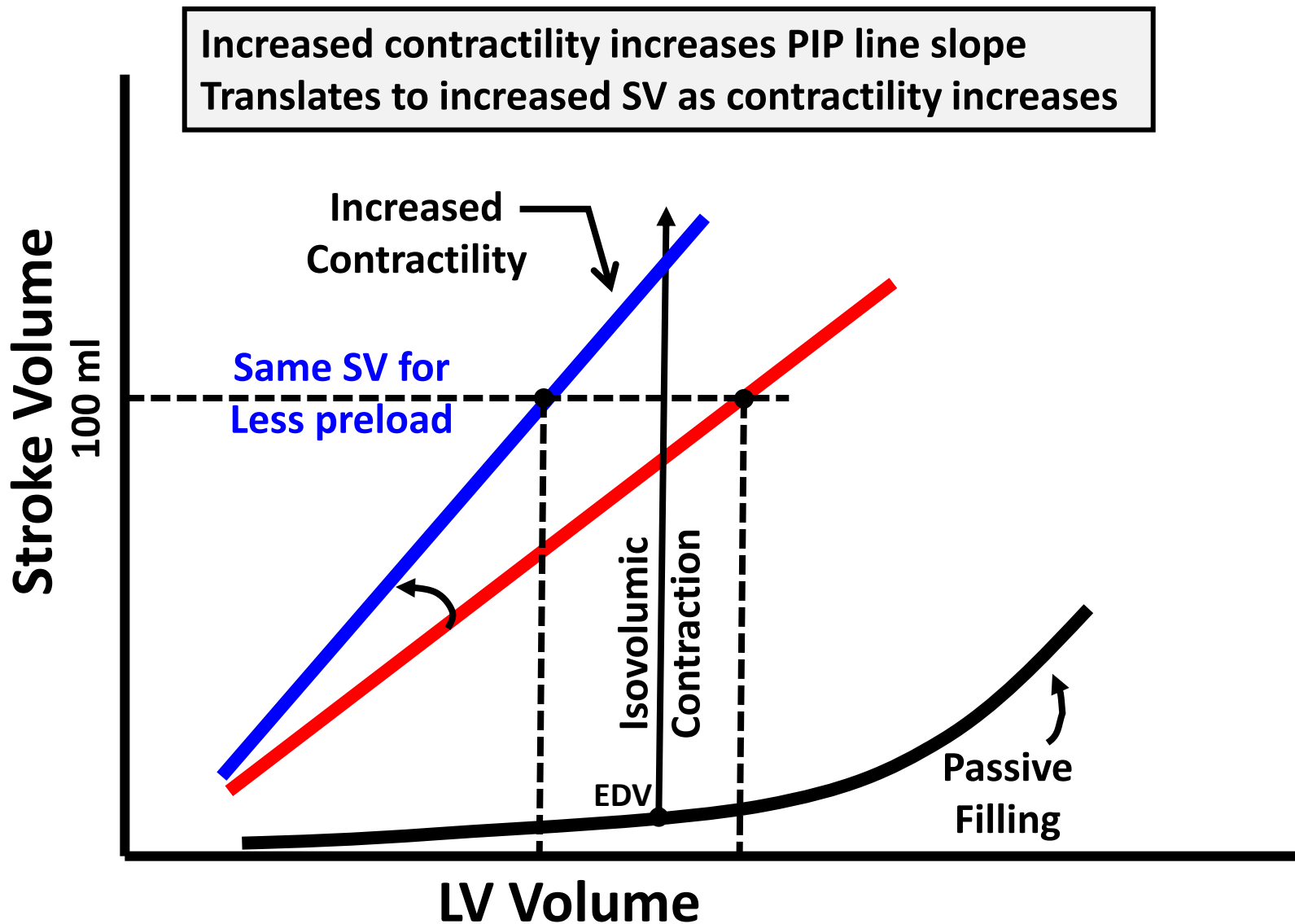
Clamped aorta with ventricle contracting

→ **isovolumic contraction**

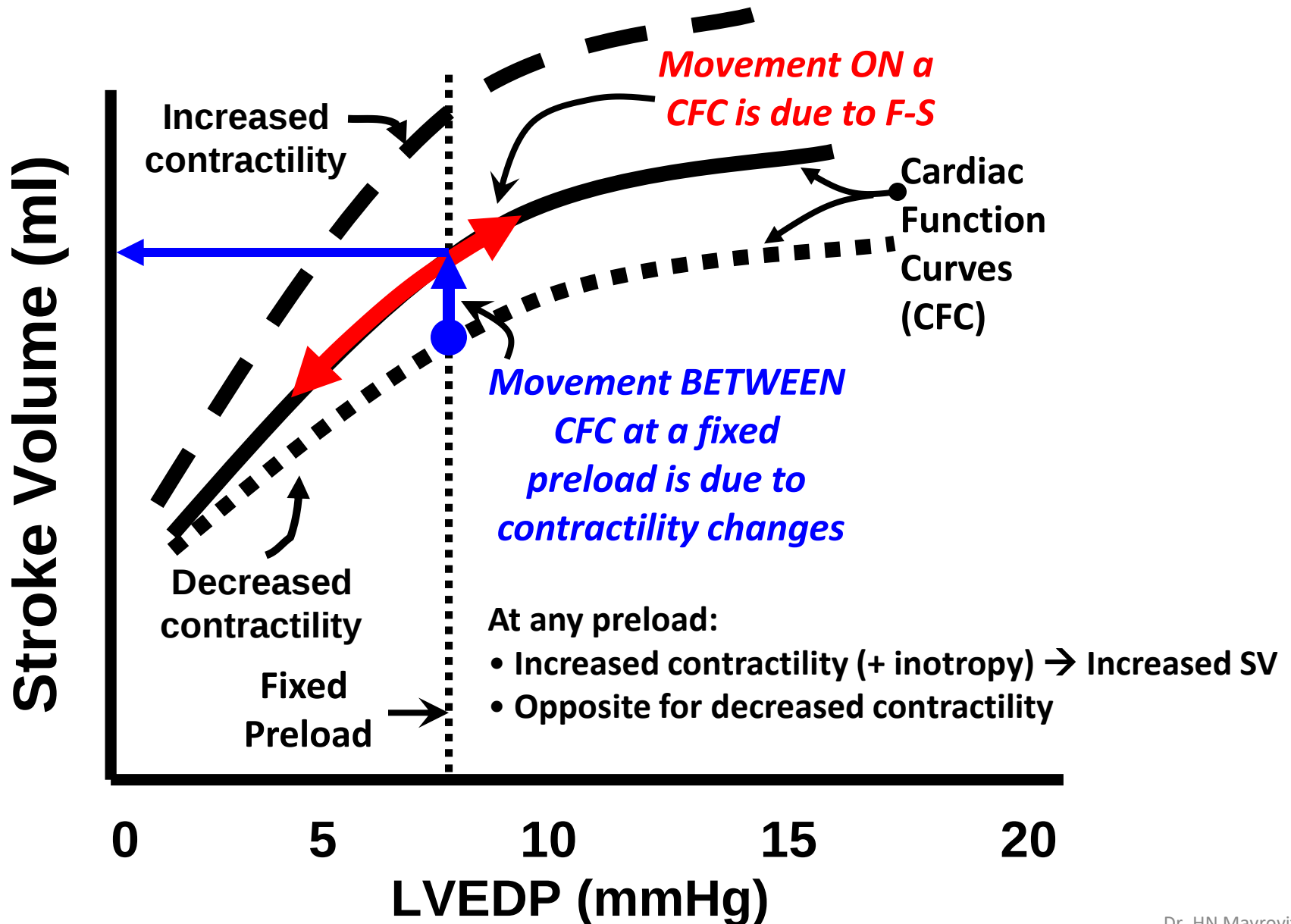
Developed ventricular pressure is maximum for its state of activation

- Heart contracts at different amounts of LV filling volume
- Peak LVP increases with EDV
- At a certain EDV peak and developed LVP starts to decline
- Occurs due to actin-myosin overlap less efficient at this stretch amount
- Ascending part of the peak isovolumic curve is a consequence of the **Frank-Starling mechanism**

Effects of Contractility on PIP Line



Frank-Starling vs. Contractility

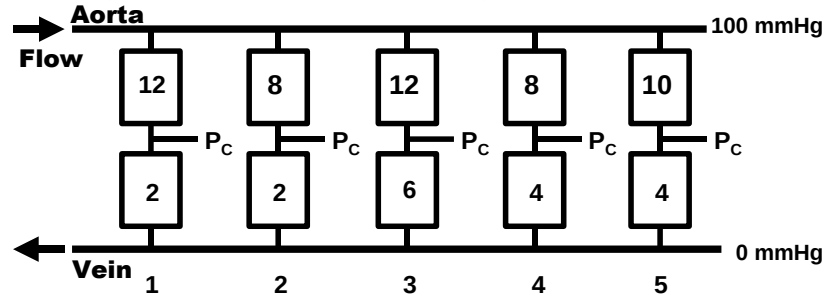


Interactive “Review” Questions



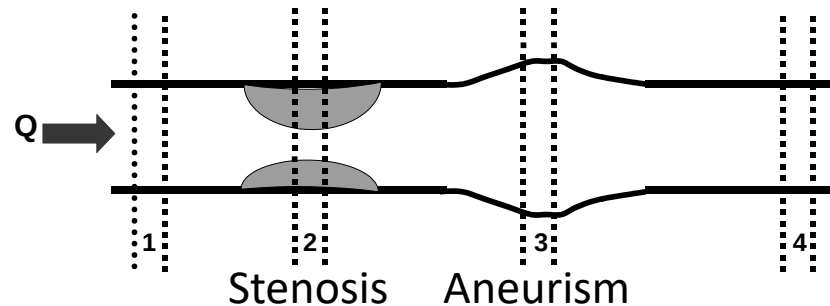
In which organ is capillary pressure 20 mmHg?

- A. 1
- B. 2
- C. 3
- D. 4
- E. 5

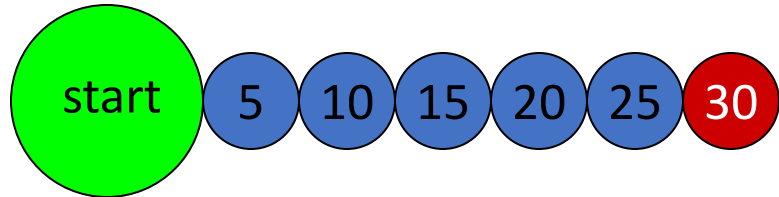


For the figure below, which one of the following statements is true?

- A. Blood flow (Q) is greater in section 1 than in section 2
- B. Average blood velocity is greater in section 1 than in section 3
- C. Reynolds number is greater in section 1 than in section 2
- D. Pressure loss across section 4 is greater than across section 1
- E. Resistance of section 3 is greater than that of section 2



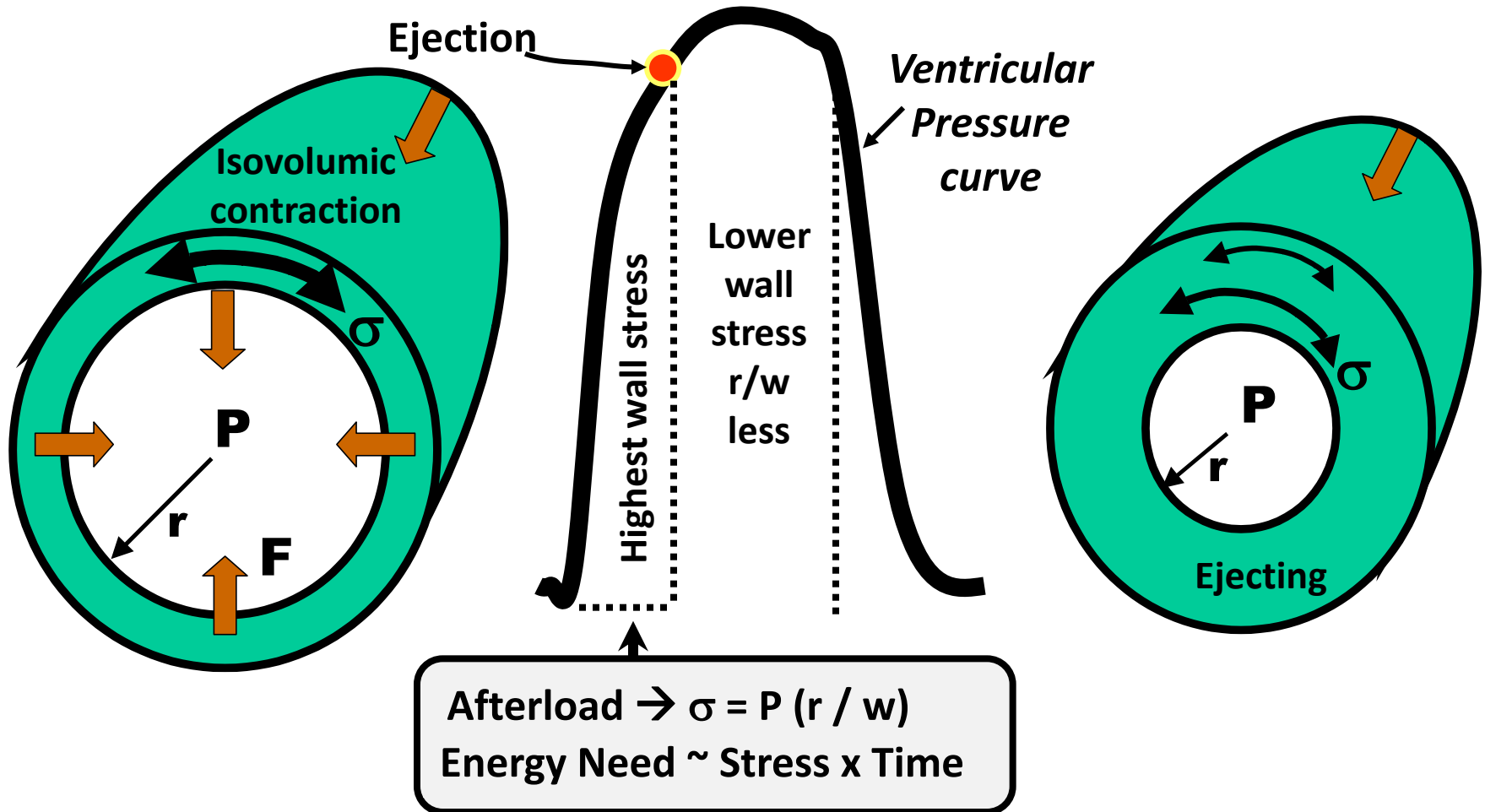
Interactive “Review” Question



Maximum velocity of shortening ($V_{\max}$) of myocardial fibers is mainly affected by changes in:

- A. Preload
- B. Afterload
- C. End diastolic volume
- D. Contractility
- E. Diastolic blood pressure

Ventricular Muscle's Load and Energy Demand



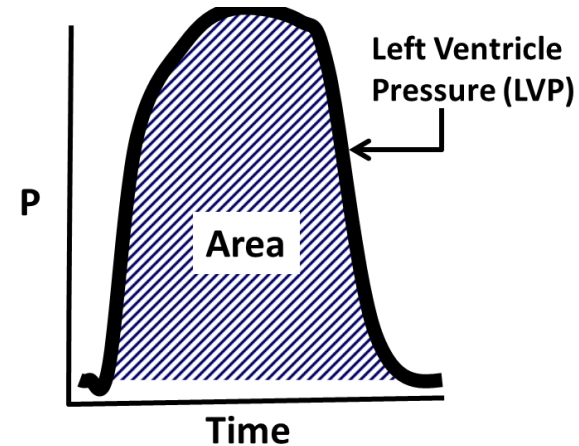
- Large O_2 demand during isovolumic contraction (large P and large r)
- Increased in conditions with elevated P (Aortic stenosis or Hypertension)
- O_2 demand during ejection also increased in conditions with elevated P

Measures of Ventricle Energy Demand

- Area under the P – T curve

Increased P

Increased duration



- Tension Time Integral (TTI)

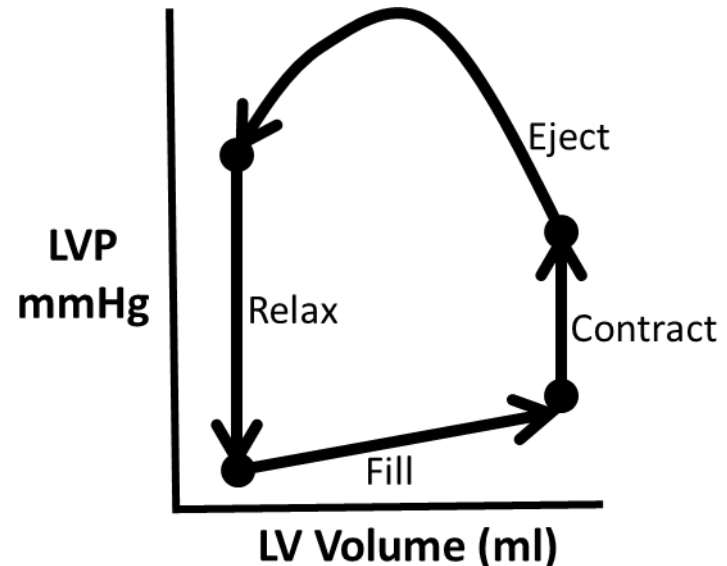
$$\int \sigma(t) = \int [P(t) \times r(t) / w(t)]$$

- Double product (MAP X HR)

Clinically Measurable

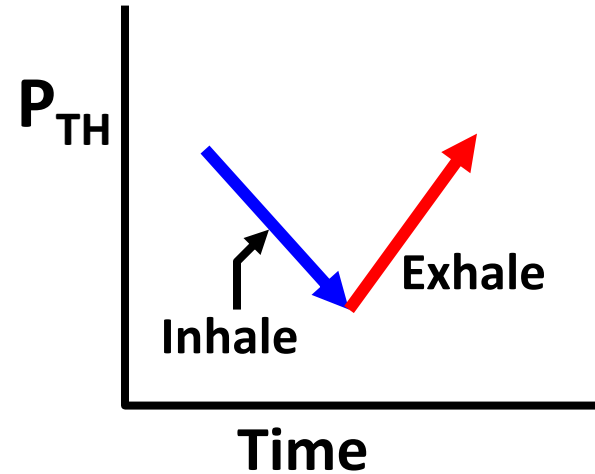
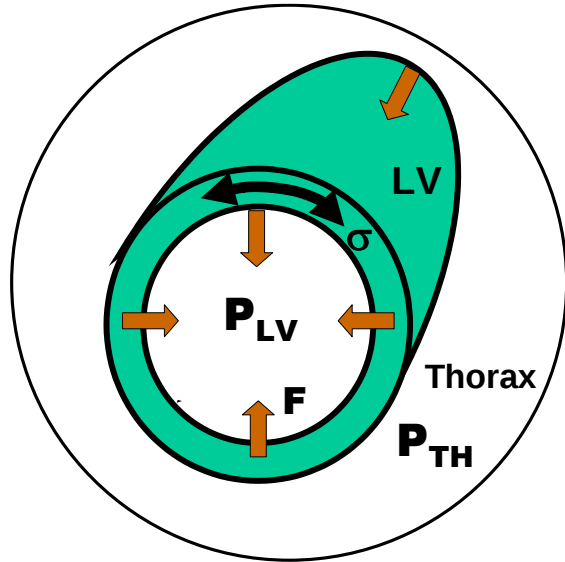
Clinically Useful

Does not include SV component



$$\text{Stroke Work} = \text{loop area} = \int P dV$$

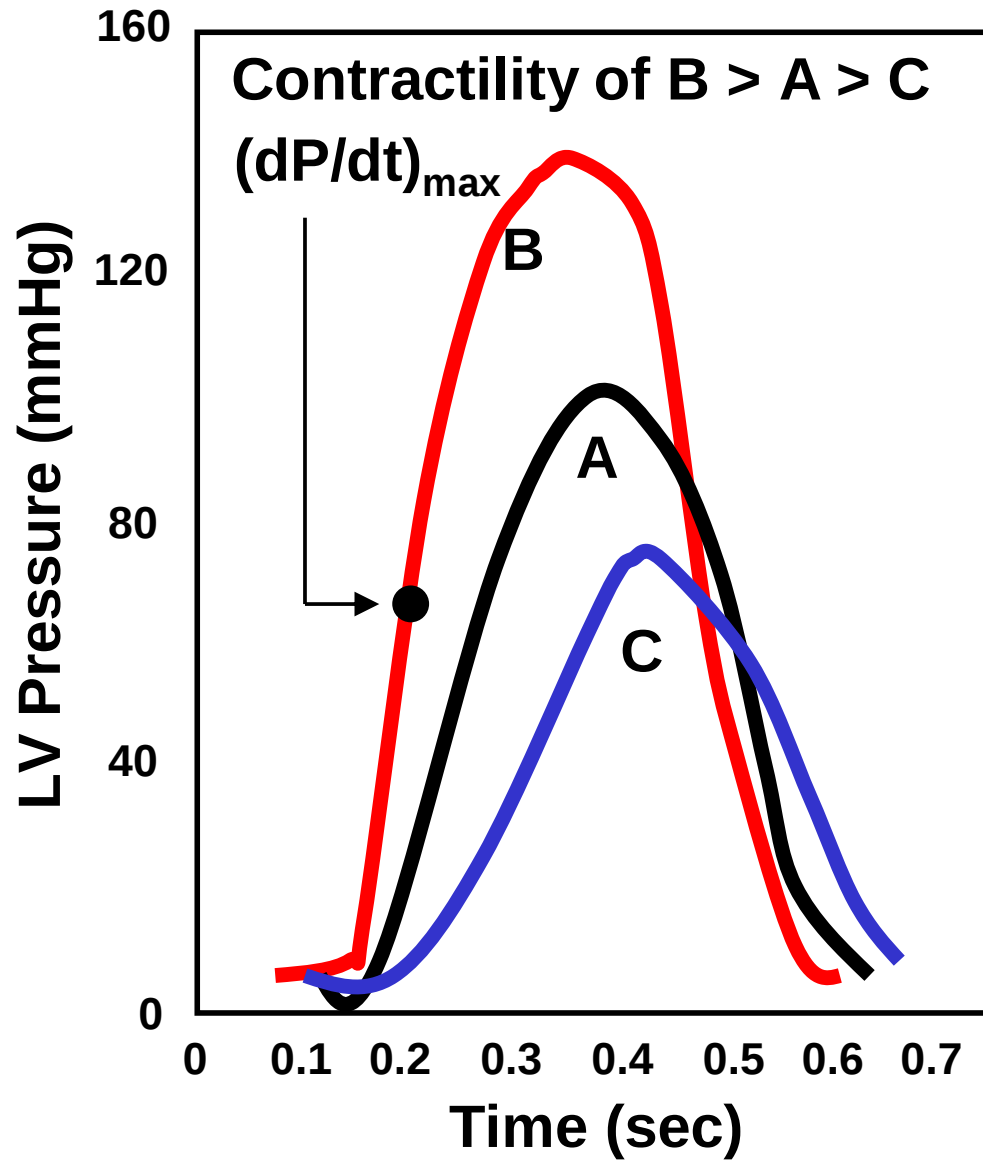
+ Intra-thoracic Pressure → + 'Afterload' Effect



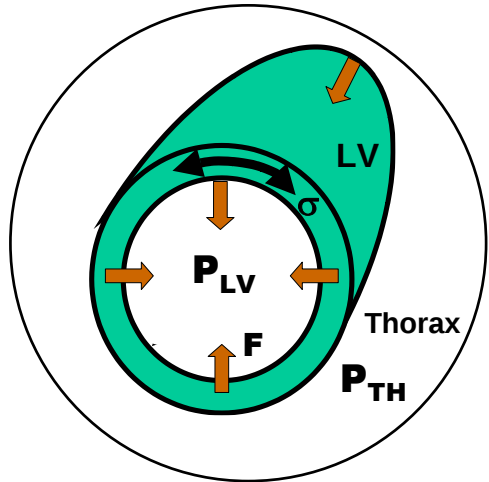
$$\text{Afterload} \rightarrow \sigma = P r / w = (P_{LV} - P_{TH}) (r/w)$$

During a deep inspiration against a closed glottis P_{TH} can decrease substantially causing a substantial increase in the LV transmural pressure. This increases the effective afterload and may reduce SV

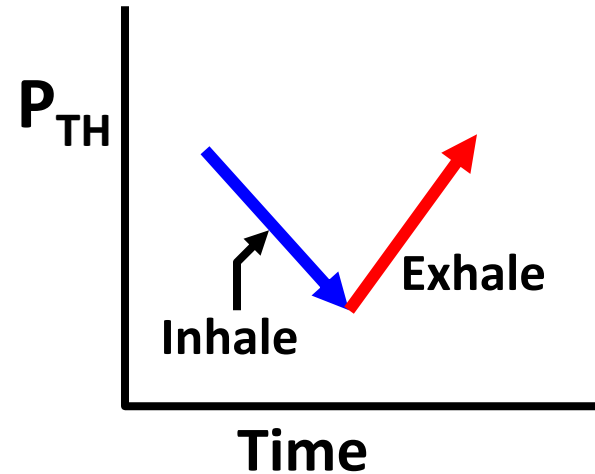
Contractility Effect on LVP



If Large decrease in intrathoracic Pressure → + 'Afterload' Effect



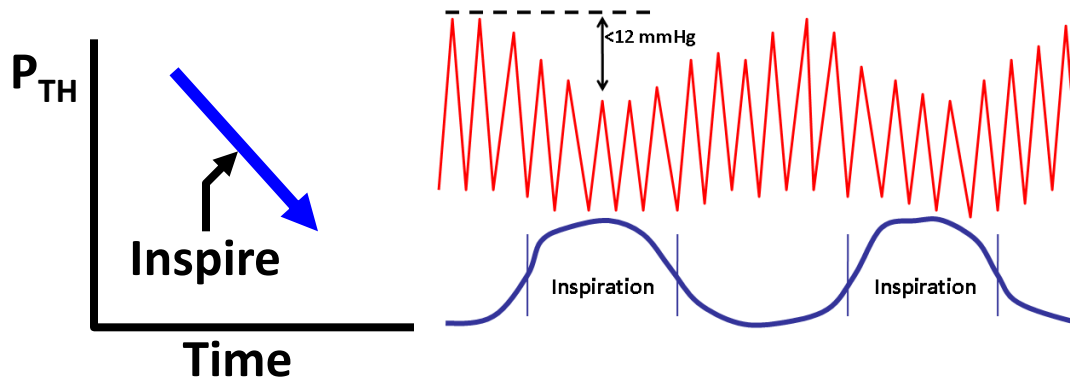
$$P_{TM} = P_{LV} - P_{TH}$$



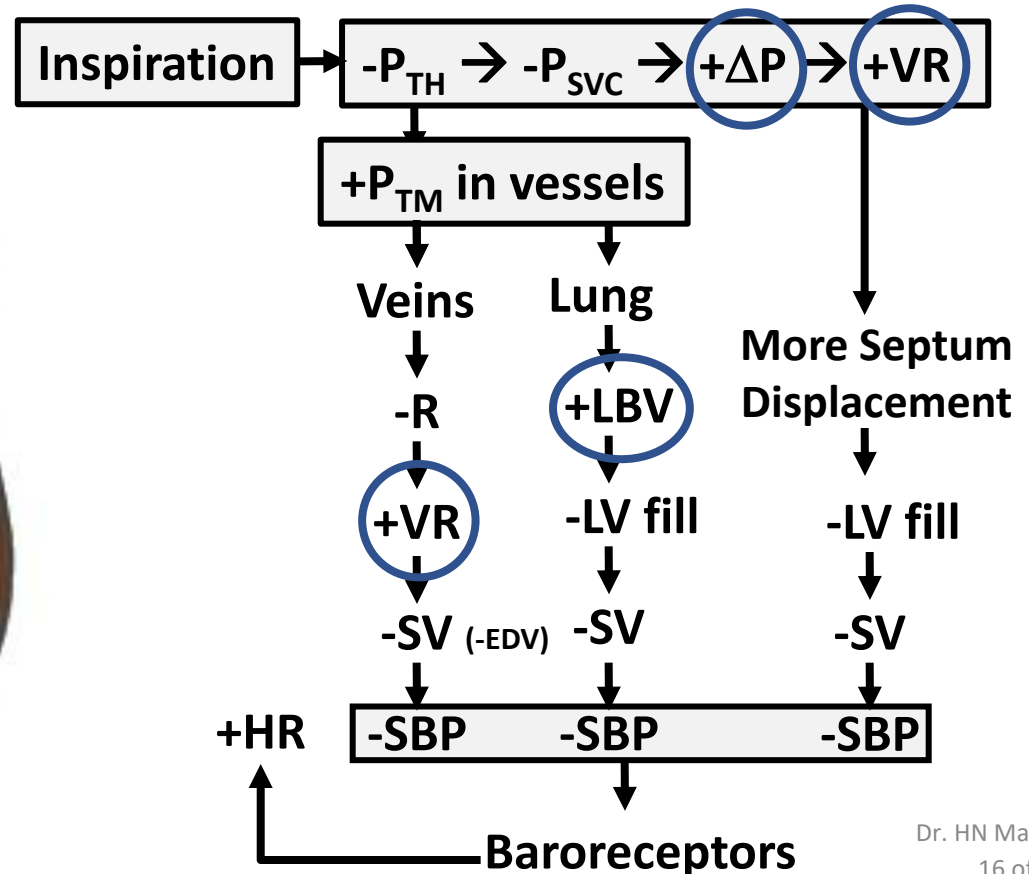
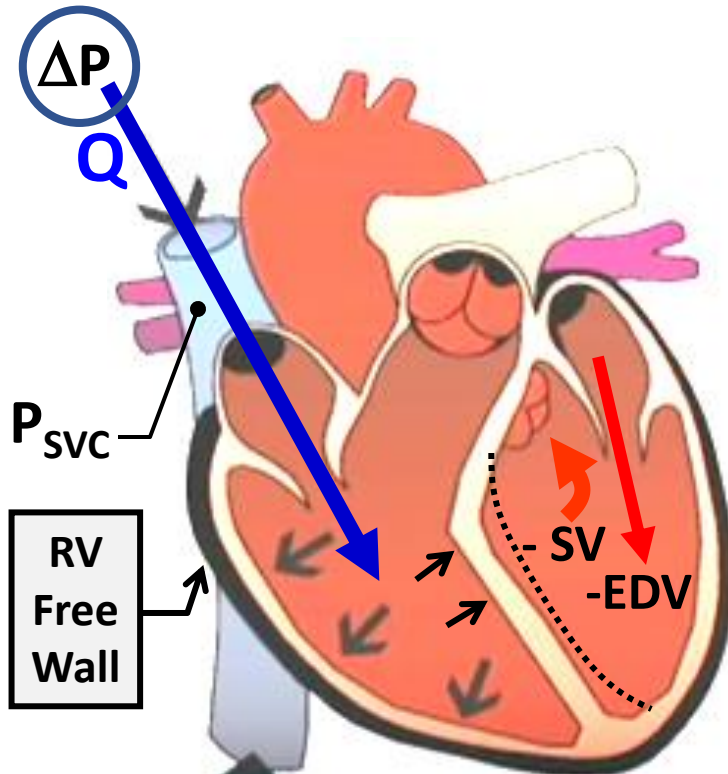
During a deep inspiration against a closed glottis P_{TH} can decrease substantially causing a substantial increase in the LV transmural pressure. This increases the effective afterload and may reduce SV

Pulsus Paradoxus → Abnormally decreased systolic pressure (peripheral pulse) during inspiration

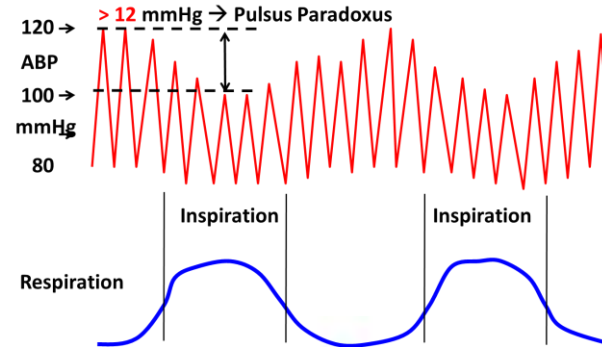
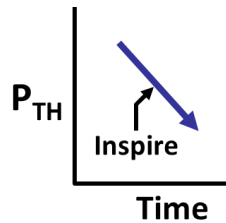
Normal Variation is Systolic BP with Respiration



- During inspiration
 - small septum shift & + Lung BV (LBV)
 - small Left Ventricle SV decrease
- Causes small decrease in systolic BP (<12 mmHg)
- Increased venous return (VR) to RH can cause similar effects
- If > 12 mmHg then **Pulsus Paradoxus**

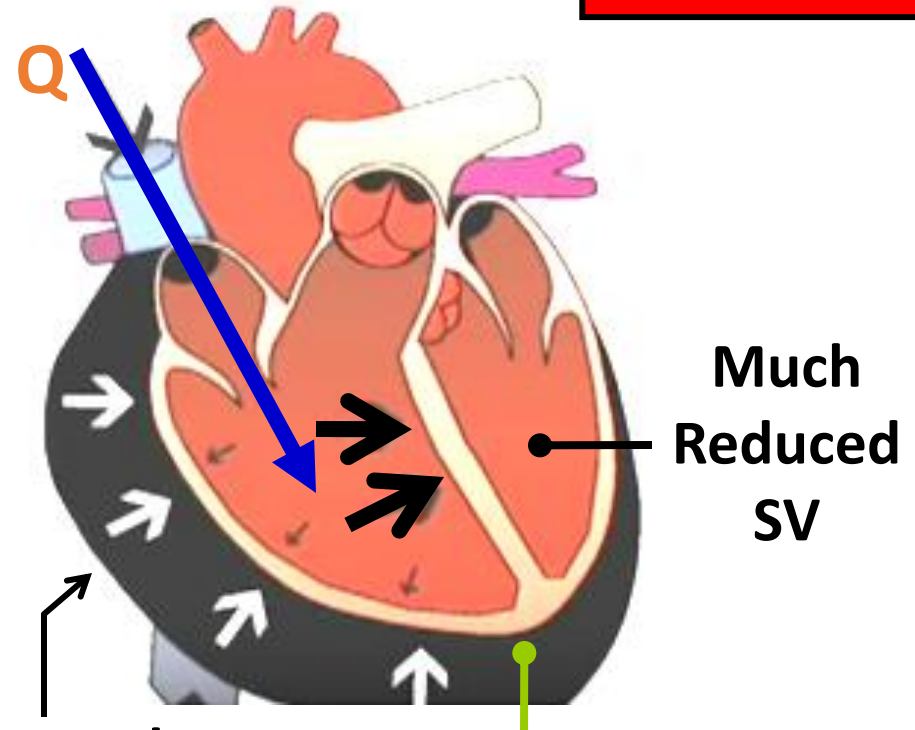
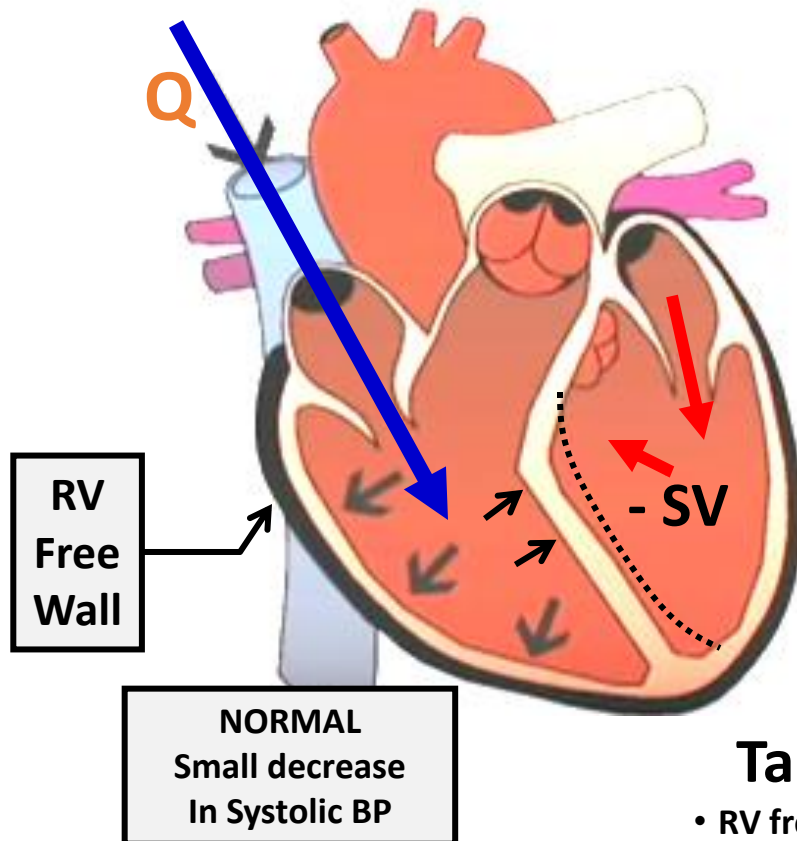


Abnormal Variation is Systolic BP with Respiration



Pulsus Paradoxus

**ABNORMAL
LARGE decrease
In Systolic BP**

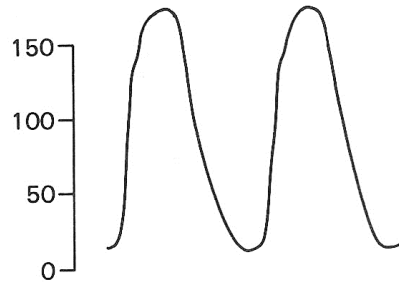


Tamponade **Fluid in pericardial sac**

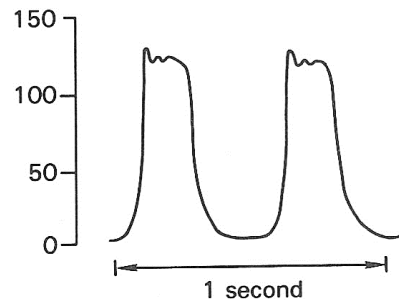
- RV free wall can't easily expand during filling
- RV pressure rises and further displaces (bows) the septum inward

Intra-Myocardial Pressures

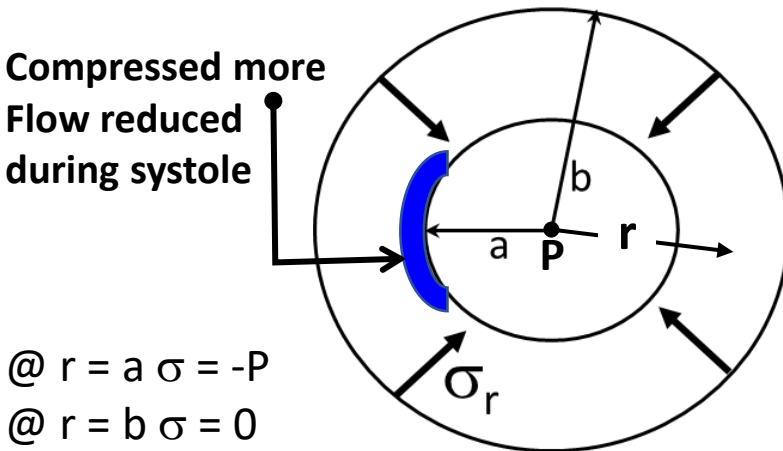
Intramyocardial Pressure (mmHg)



Left Ventricle Pressure (mmHg)



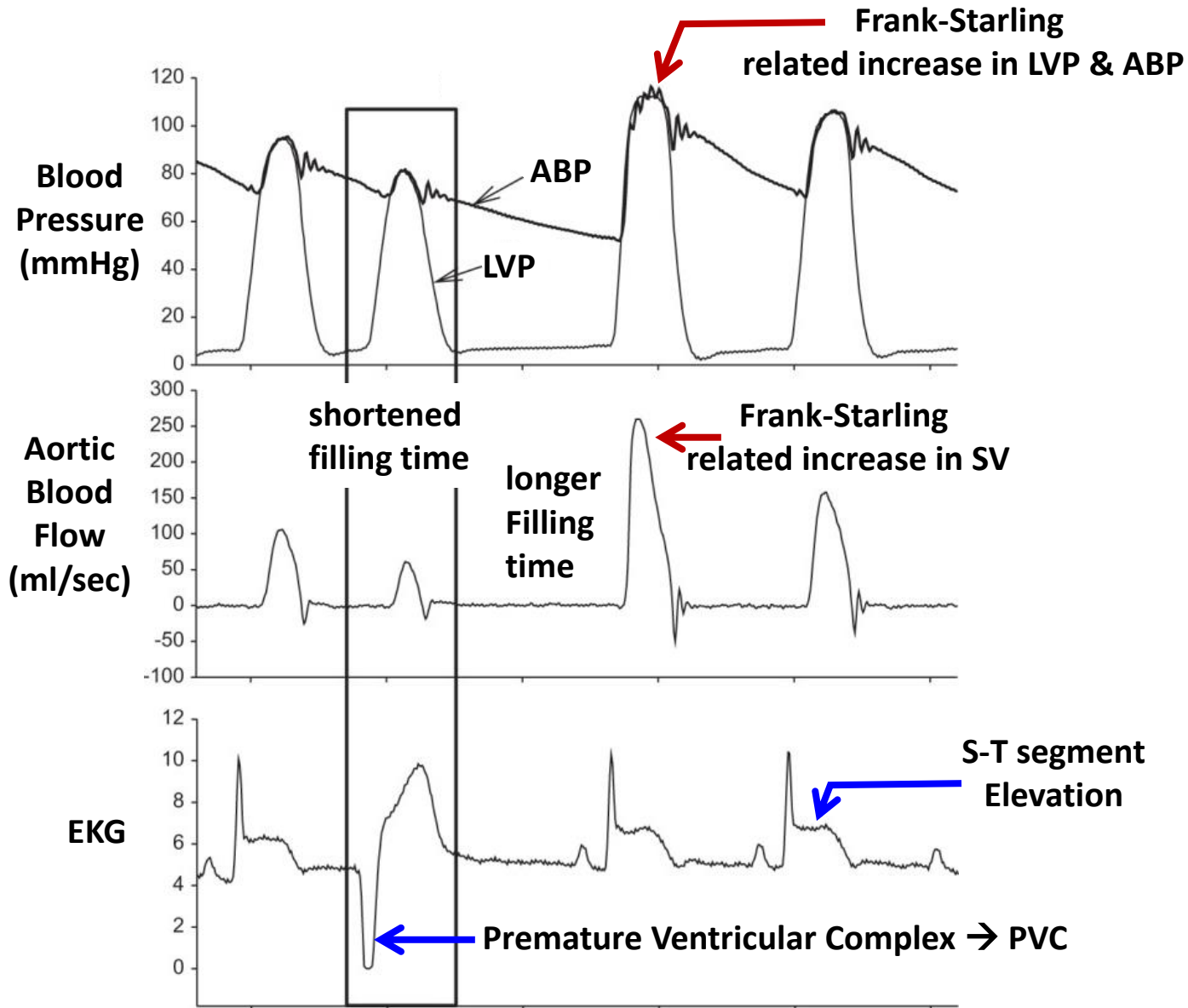
Compressed more
Flow reduced
during systole



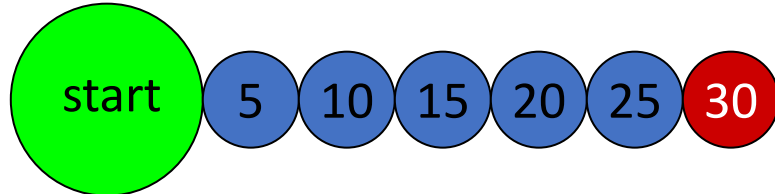
$$\sigma_r = [a^2 P / (b^2 - a^2)] \times (1 - b^2 / r^2)$$

- 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**

Clinical Correlation: Patient with MI and PVC



Interactive Question



Which of the following, if increased, would most likely increase blood pressure If the change was the only change?

Note on this question there can be more than one choice!

- a) arteriolar diameters
- b) vascular resistance ←
- c) stroke volume ←
- d) cardiac output ←
- e) blood viscosity ←
- f) blood volume ←

End Lecture 21
MCQs as time permits