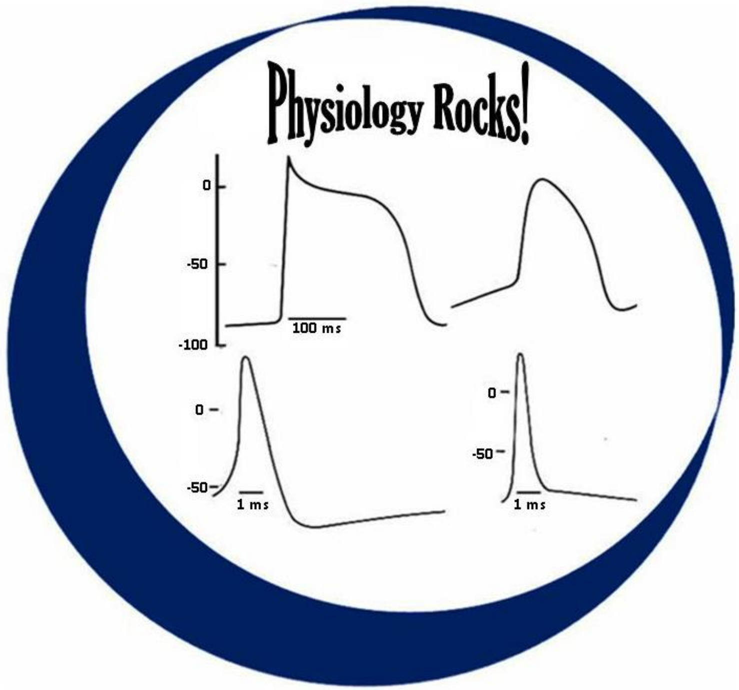
Lecture 8
Determinants of Cardiac Function



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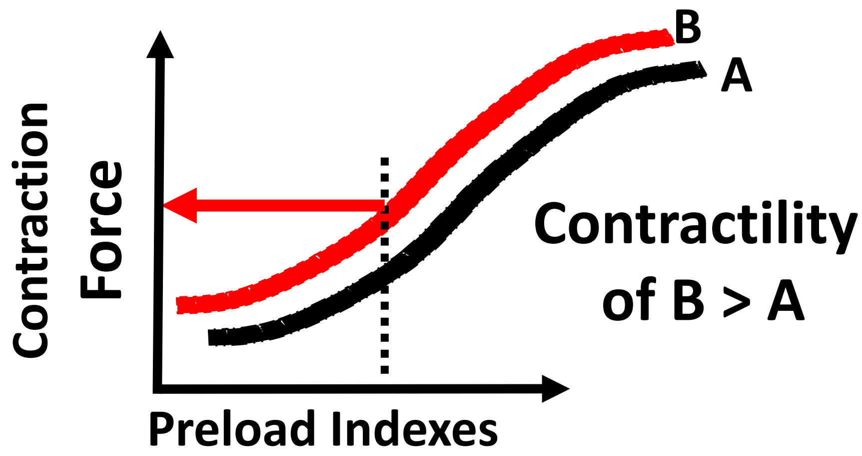
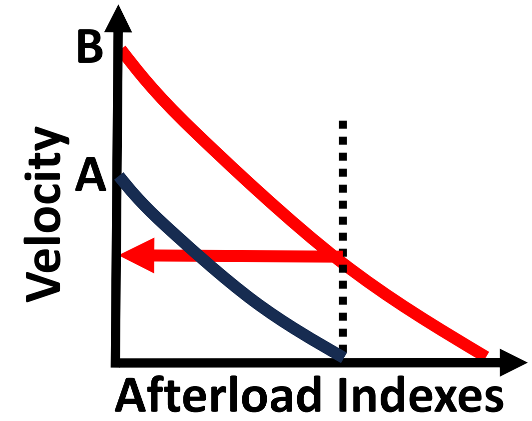
Topics

- 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

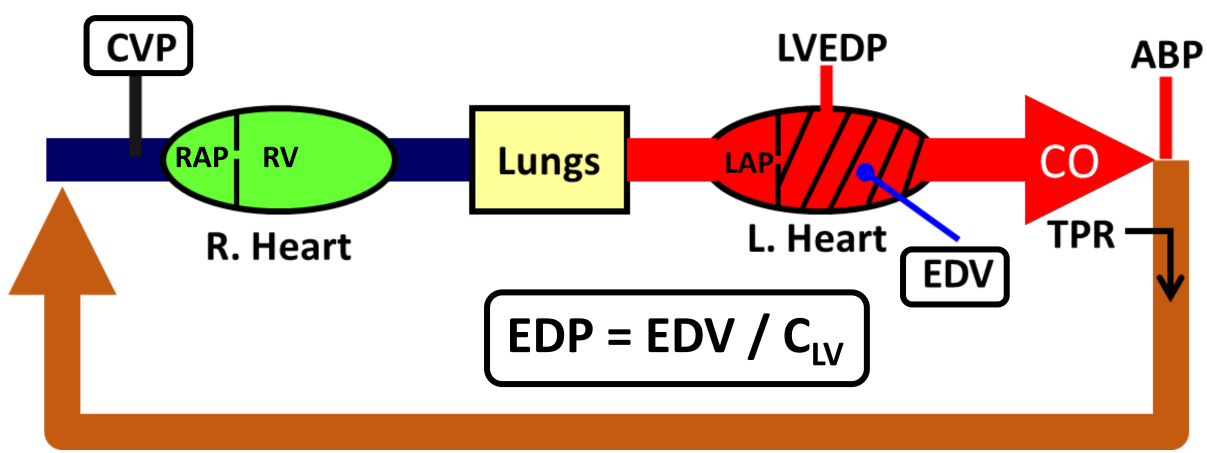




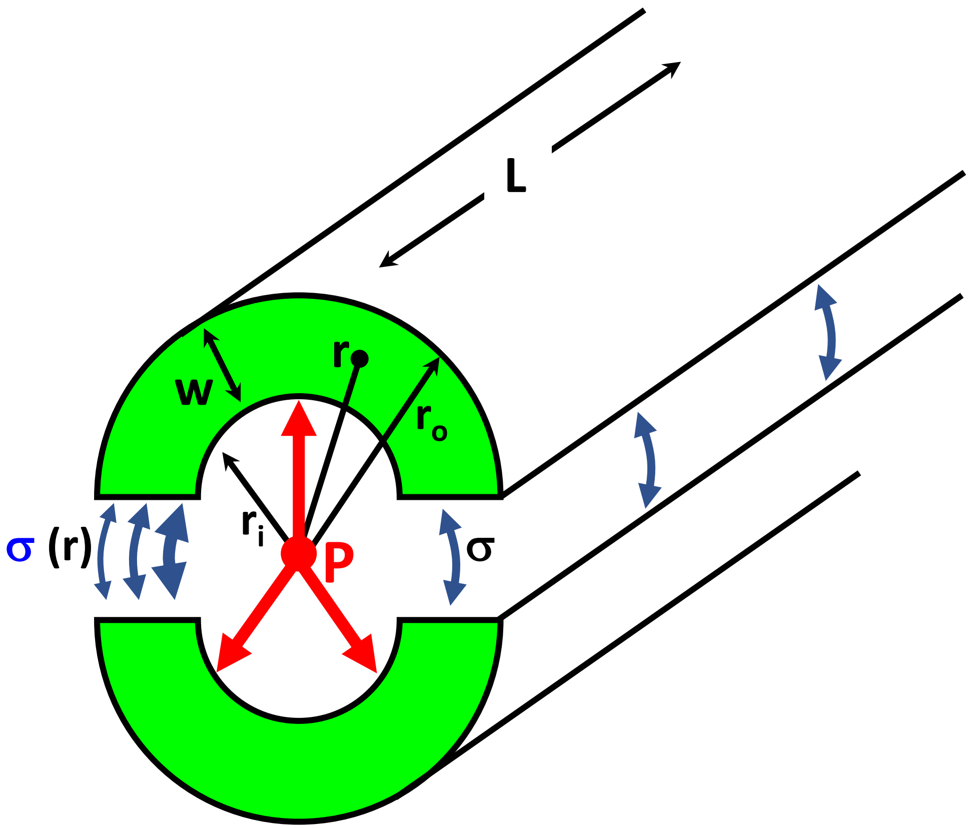
- ***EDV:*** ***LV volume at start of contraction-Best***
- ***EDP:*** ***LV pressure at start of contraction -2nd***
- ***CVP:*** ***Central venous pressure – ease measure***

- ***Wall Stress (next slide)***
- ***TPR: (MAP – CVP)/CO***
- ***Aortic Blood Pressure***

Simplified Cardiovascular System



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

  The distending force/L = P x πr_i^2
  The restoring force/L = σ x $\pi r_i w$

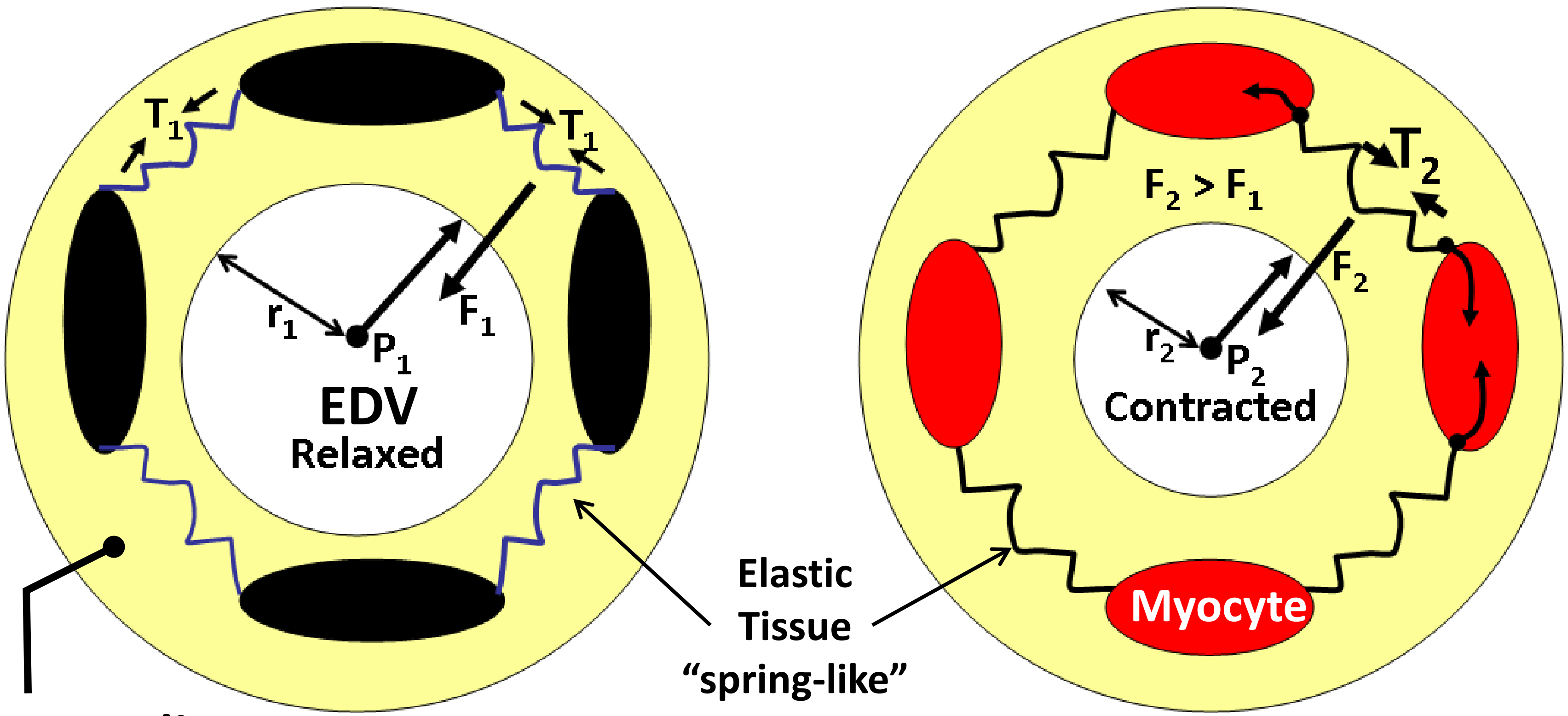
- Equate forces for an equilibrium: yielding $\sigma = (P \times r_i)/w$ = modified Laplace's Law
  σ is the average stress in the wall

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

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

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

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 x HR

SV:

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

HR:

- Sympathetic
- Vagus

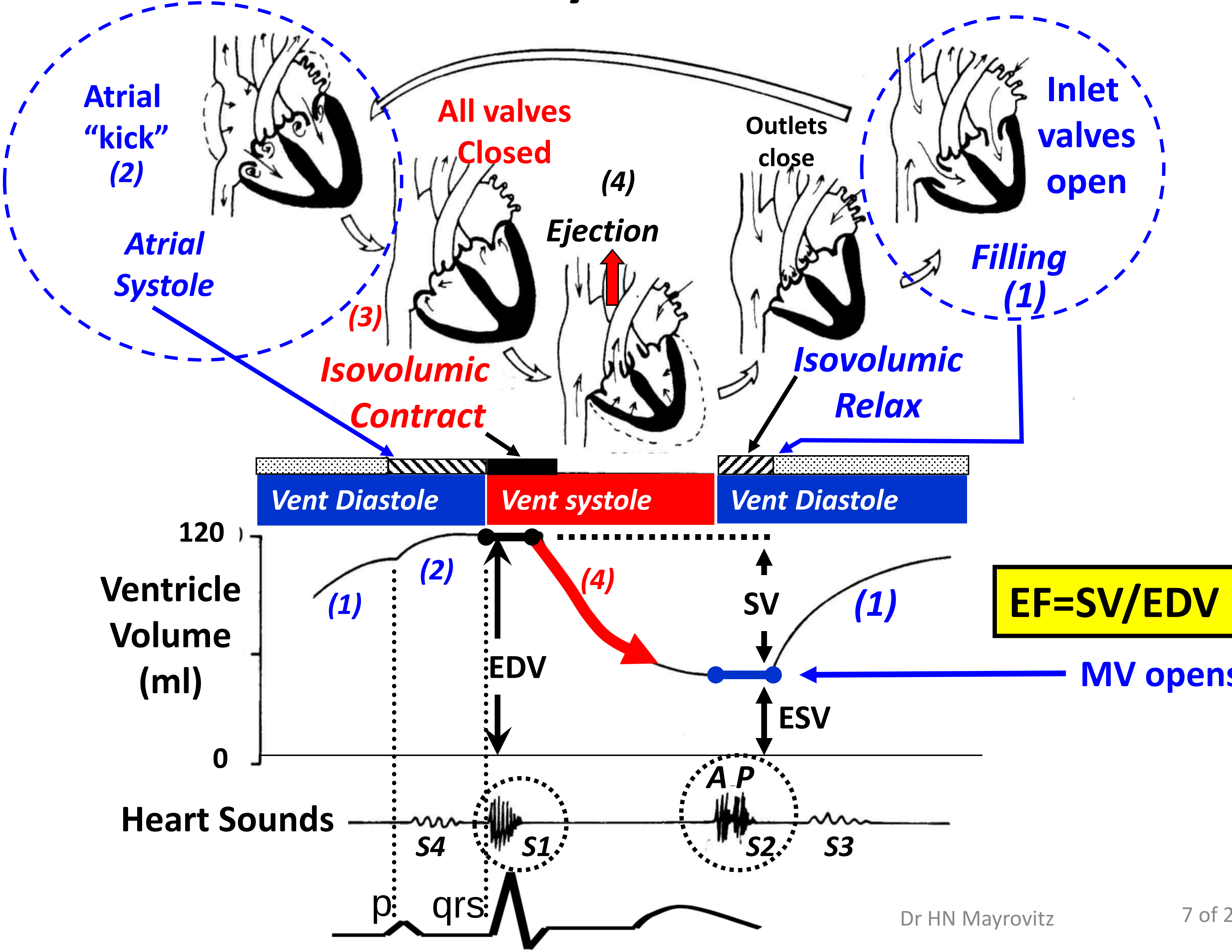
SV effects:

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

HR effects:

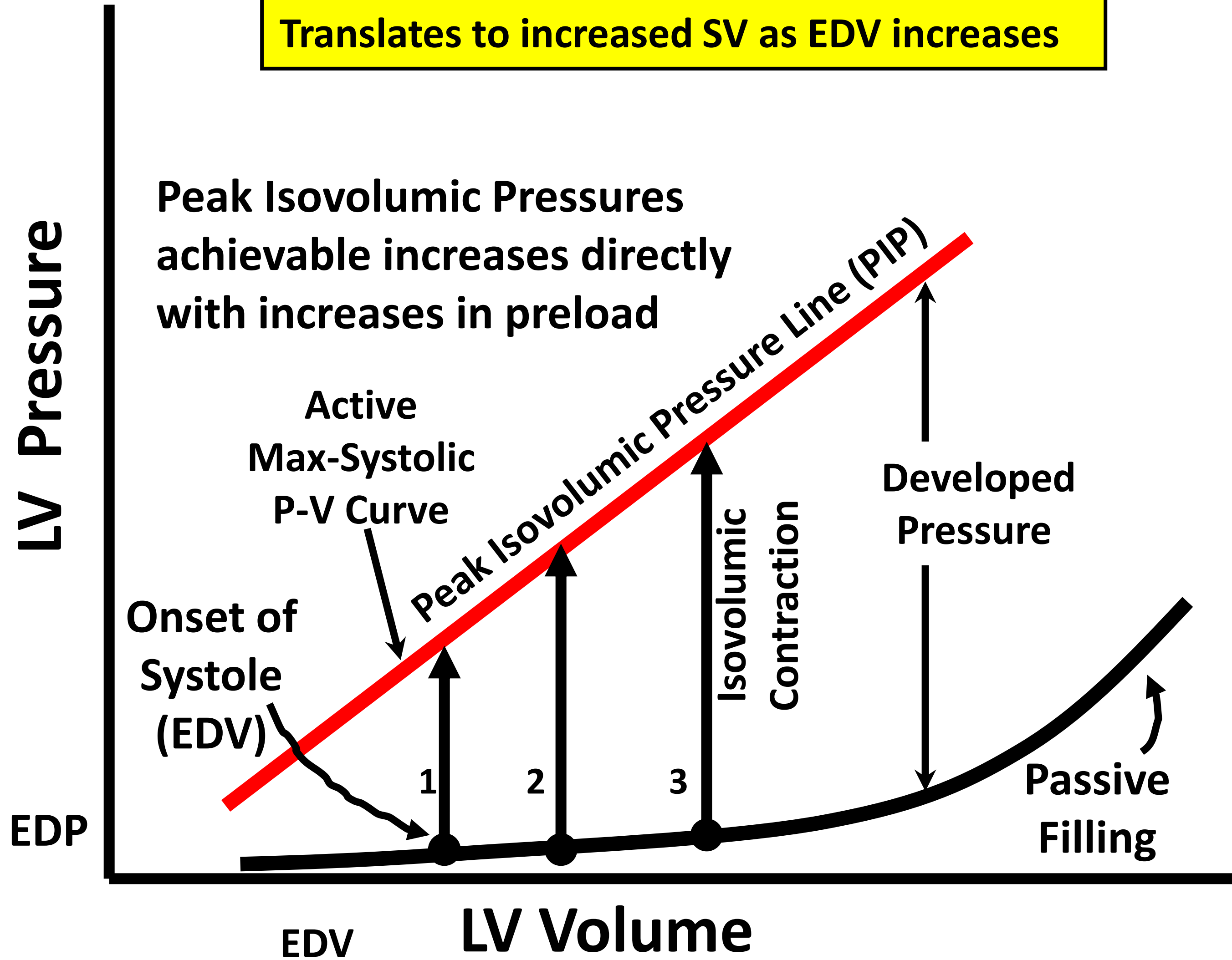
- +Sympathetic: +HR
- +Vagus: -HR

Cardiac Cycle Overview

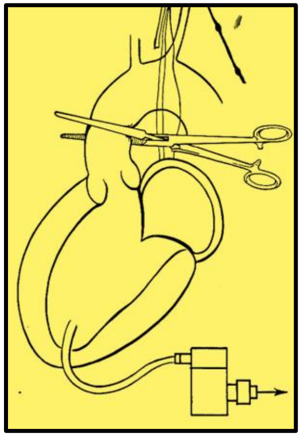


Frank-Starling "Law" of the Heart
Preload Dependent

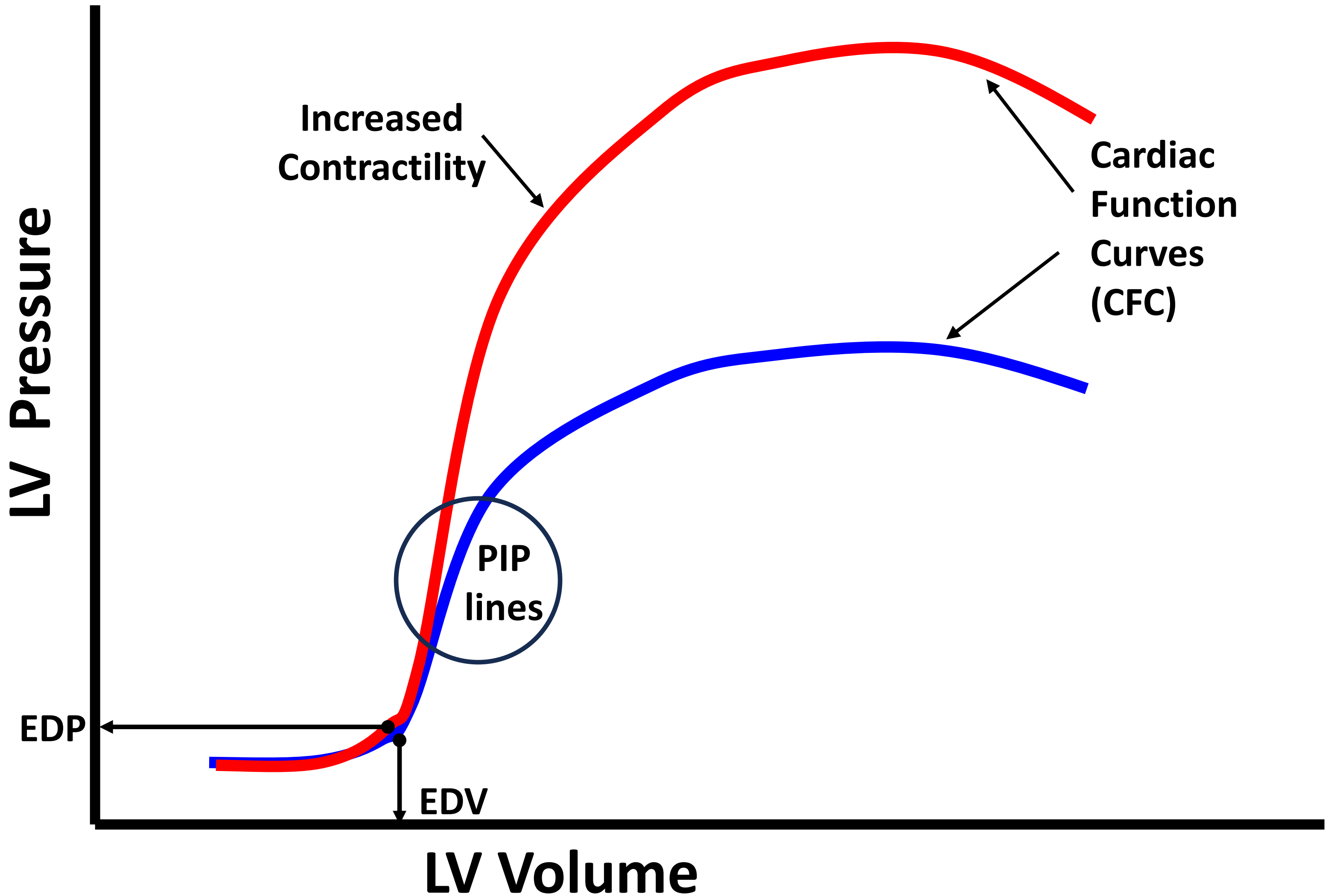
Contraction force increases as EDV increases
Translates to increased SV as EDV increases



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

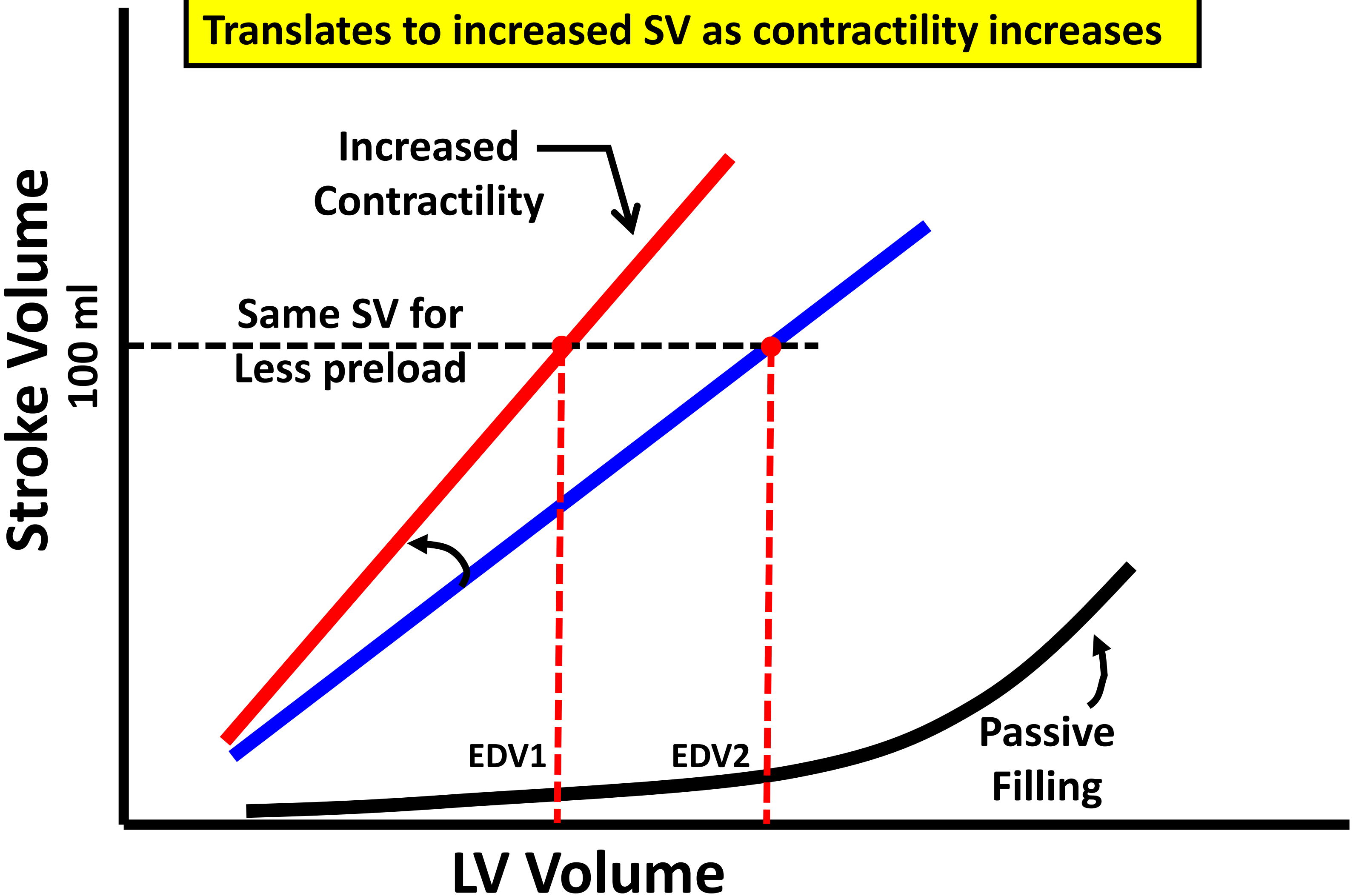


PIP Lines as part of the Cardiac Function Curves

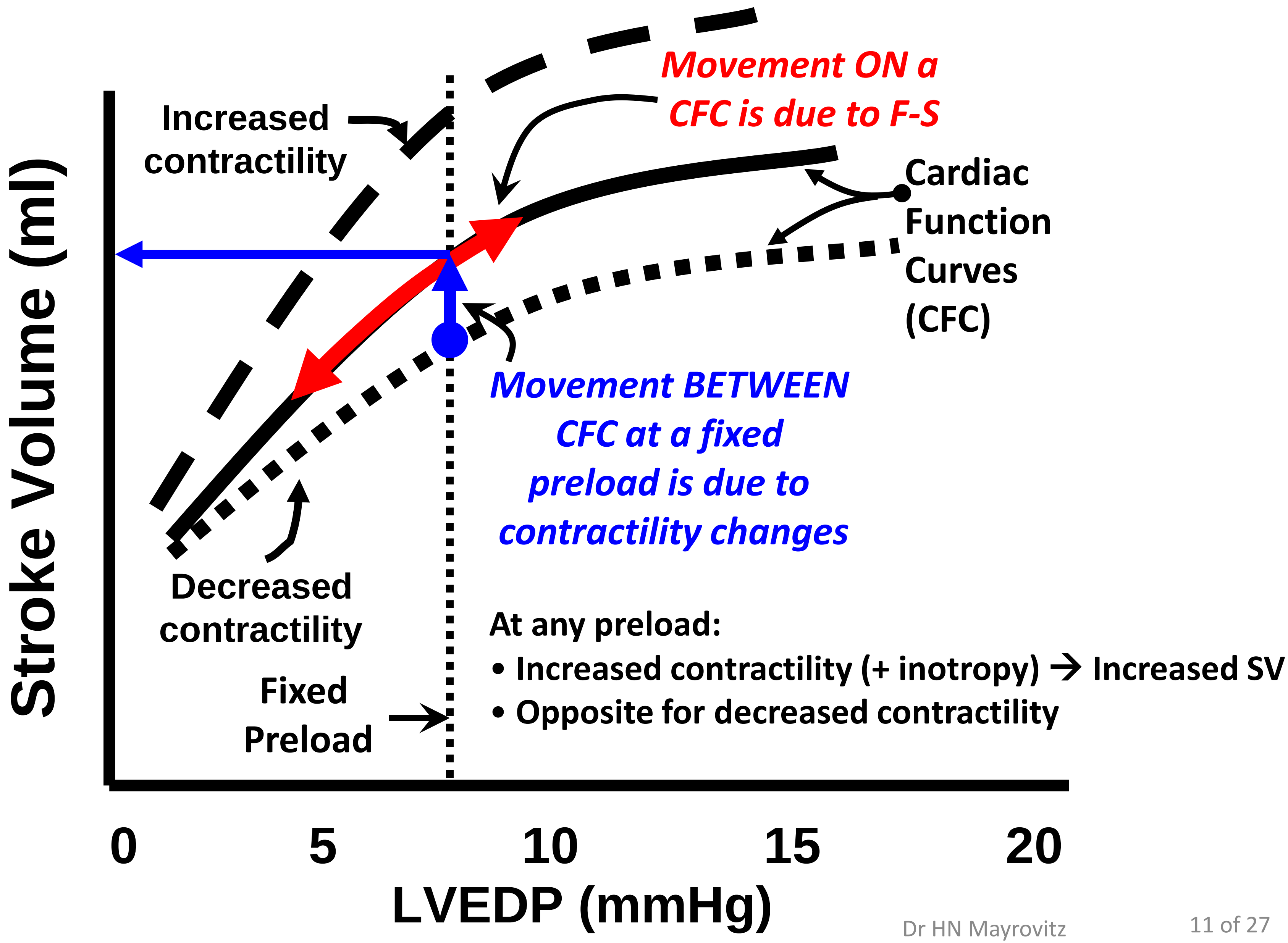


Effects of Contractility on PIP Line

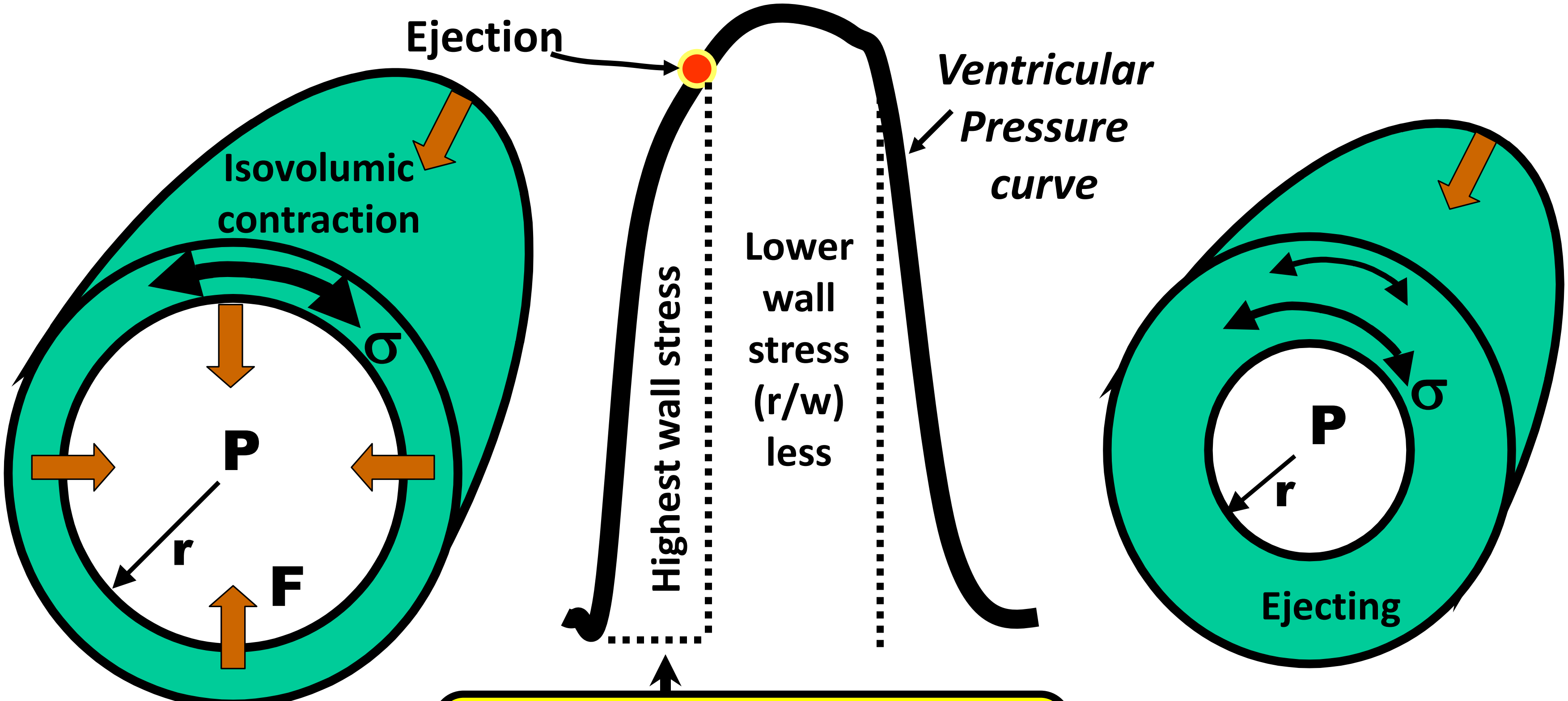
Increased contractility increases PIP line slope
Translates to increased SV as contractility increases



Frank-Starling vs. Contractility



Ventricular Muscle's Load and Energy Demand



Afterload → σ = P (r / w)
Energy Need ~ Stress x Time

- Large O_2 demand during isovolumic contraction (large P and large r) = large σ
- 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)**

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

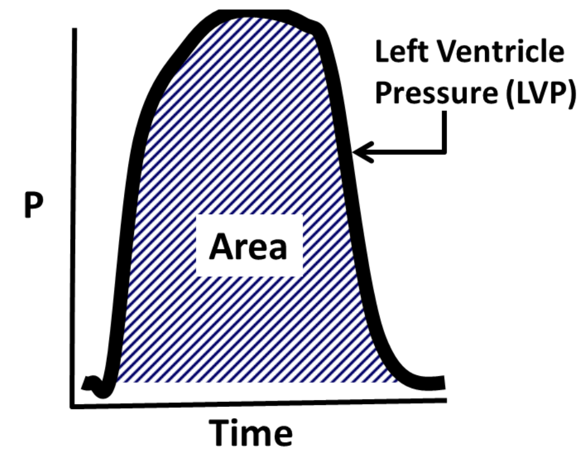
- **Double product (MAP X HR)**

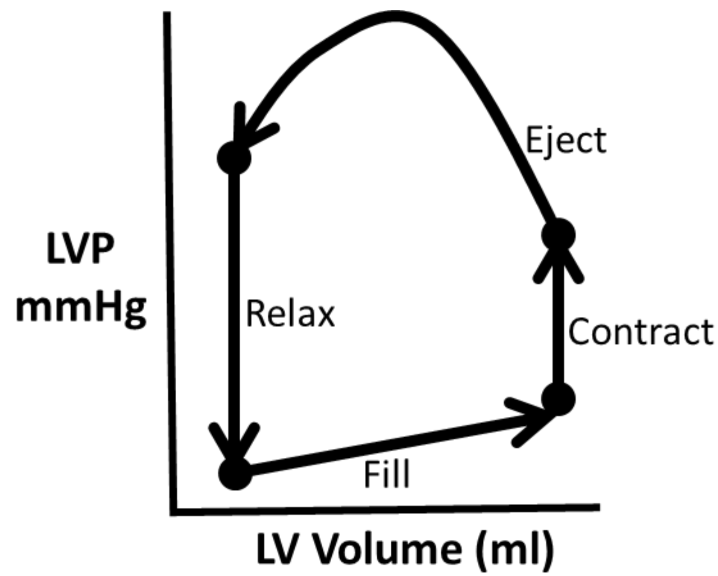
  Clinically Measurable

  Clinically Useful

  Does not include SV component

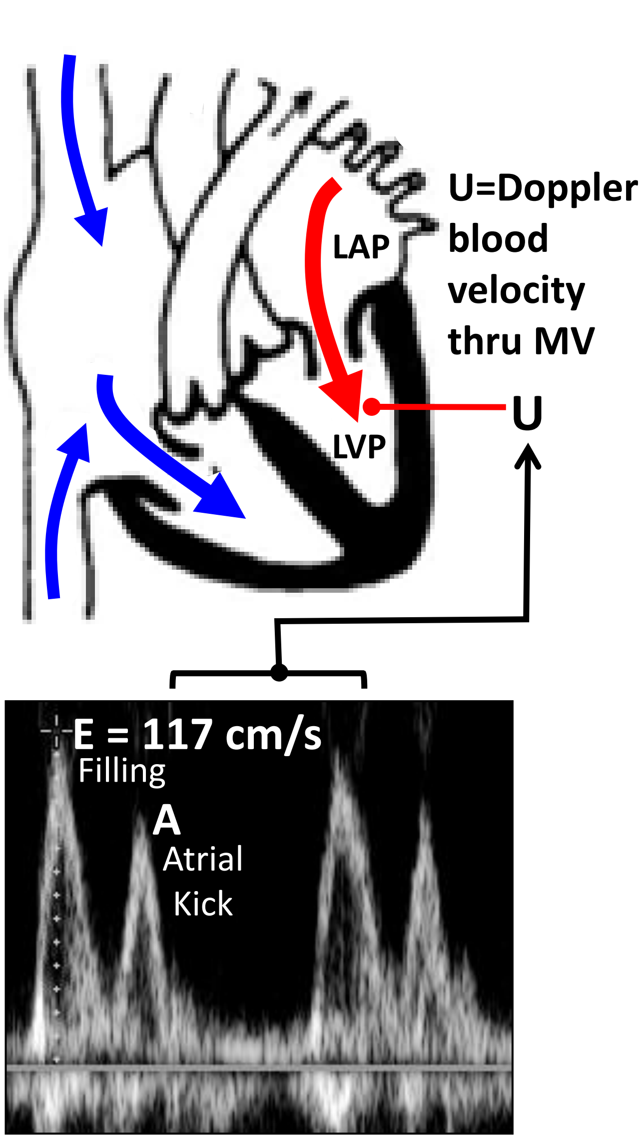
  (Generally small error)

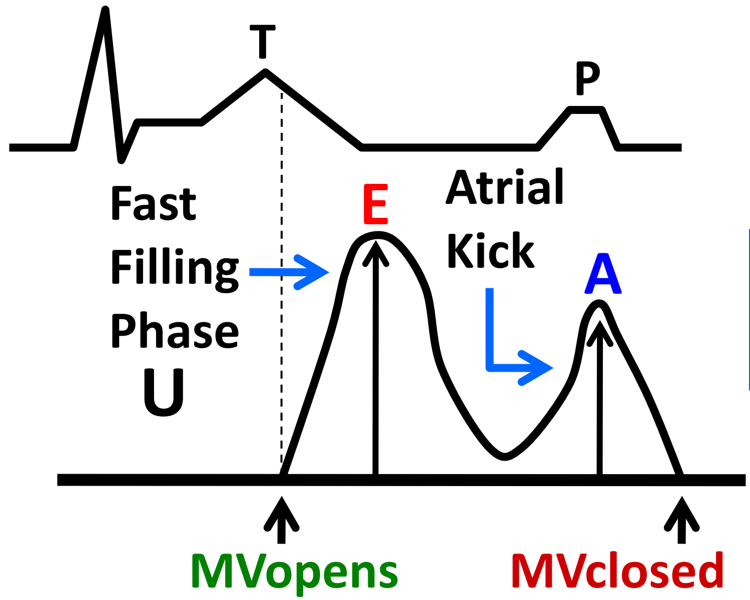




Stroke Work = loop area = $\int PdV$

Clinical Correlation: LV Diastolic Filling Parameter

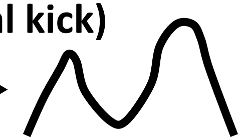




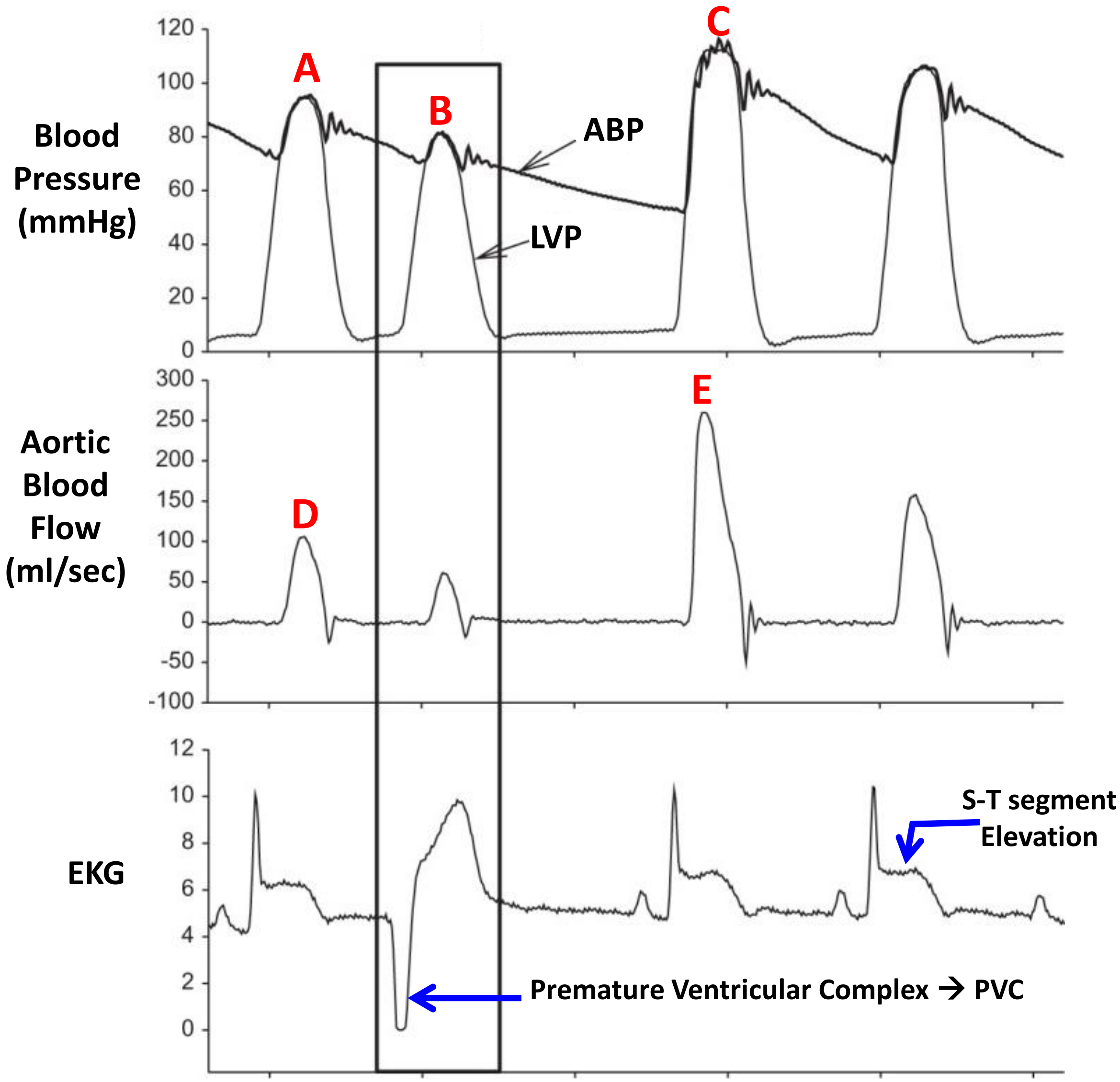
- E is peak U during early fast filling
- A is peak U during atrial contraction
- **E depends on**
  - → LAP-LVP gradient
  - → LVP Stiffness
- E/A is age dependent 21-40 (1.5), >60 (1.0)

- 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



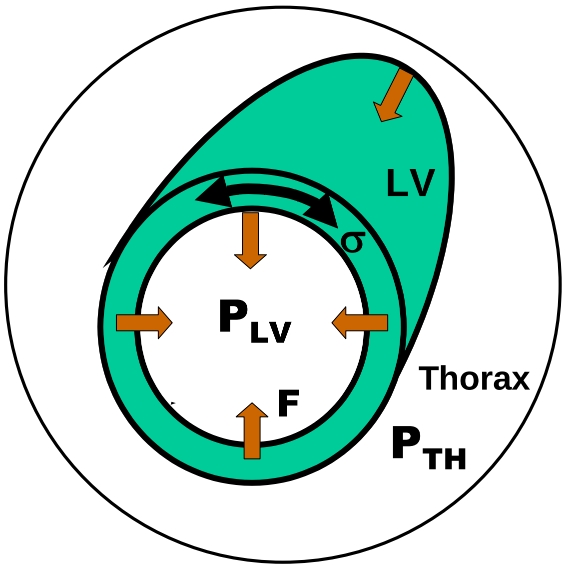
1. Why is ABP less at B than A?

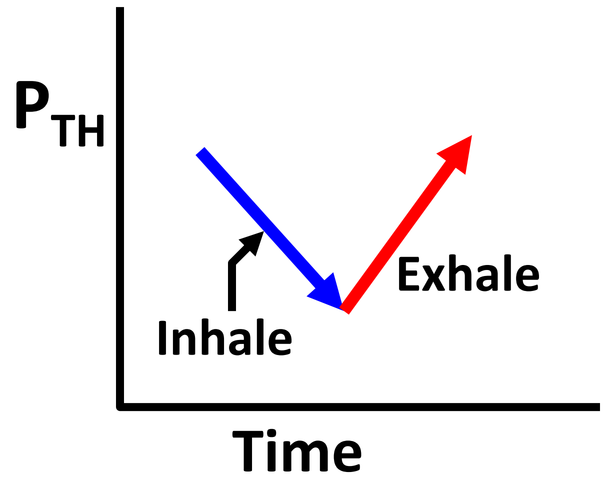
2. Why is ABP greater at C than A?

3. Why is flow greater at E than D?

Respiration Related Processes

+ Intra-thoracic Pressure → + 'Afterload' Effect

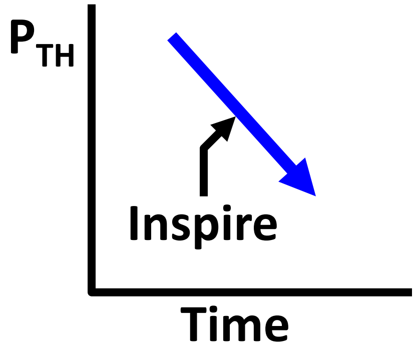


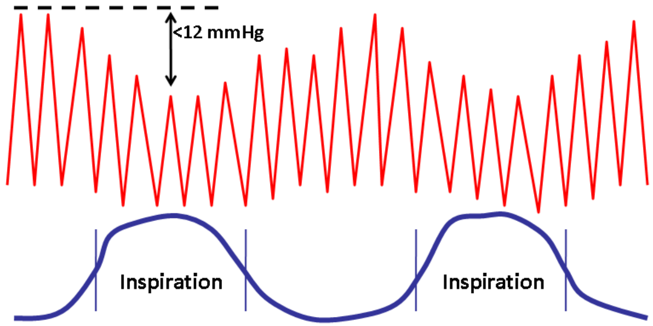


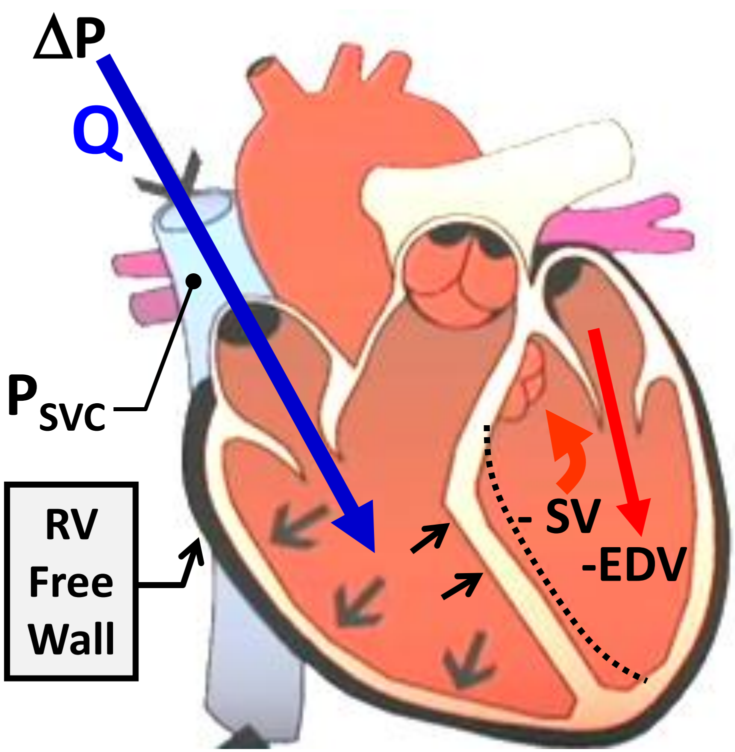
Afterload → $\sigma = P\ r / w = (P_{LV} - P_{TH})\ (r/w)$

- **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

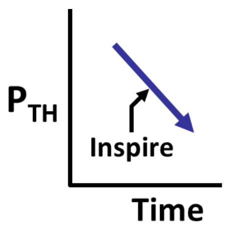


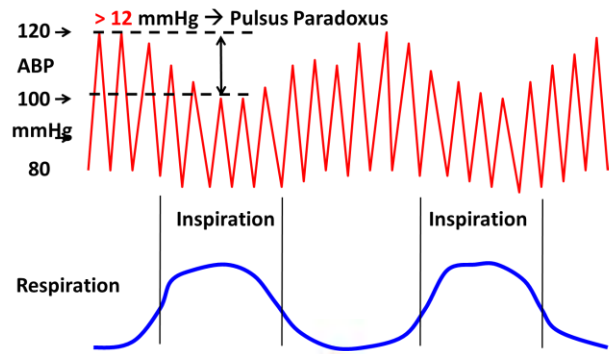




- During inspiration blood returning to the RV increases RV pressure causing a slight displacement of the septum into the LV
- Diastolic filling of the LV is thus slightly reduced as evidenced by a decreased EDV
- This preload reduction is associated with a decrease in SV (Frank-Starling)
- The decreased 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

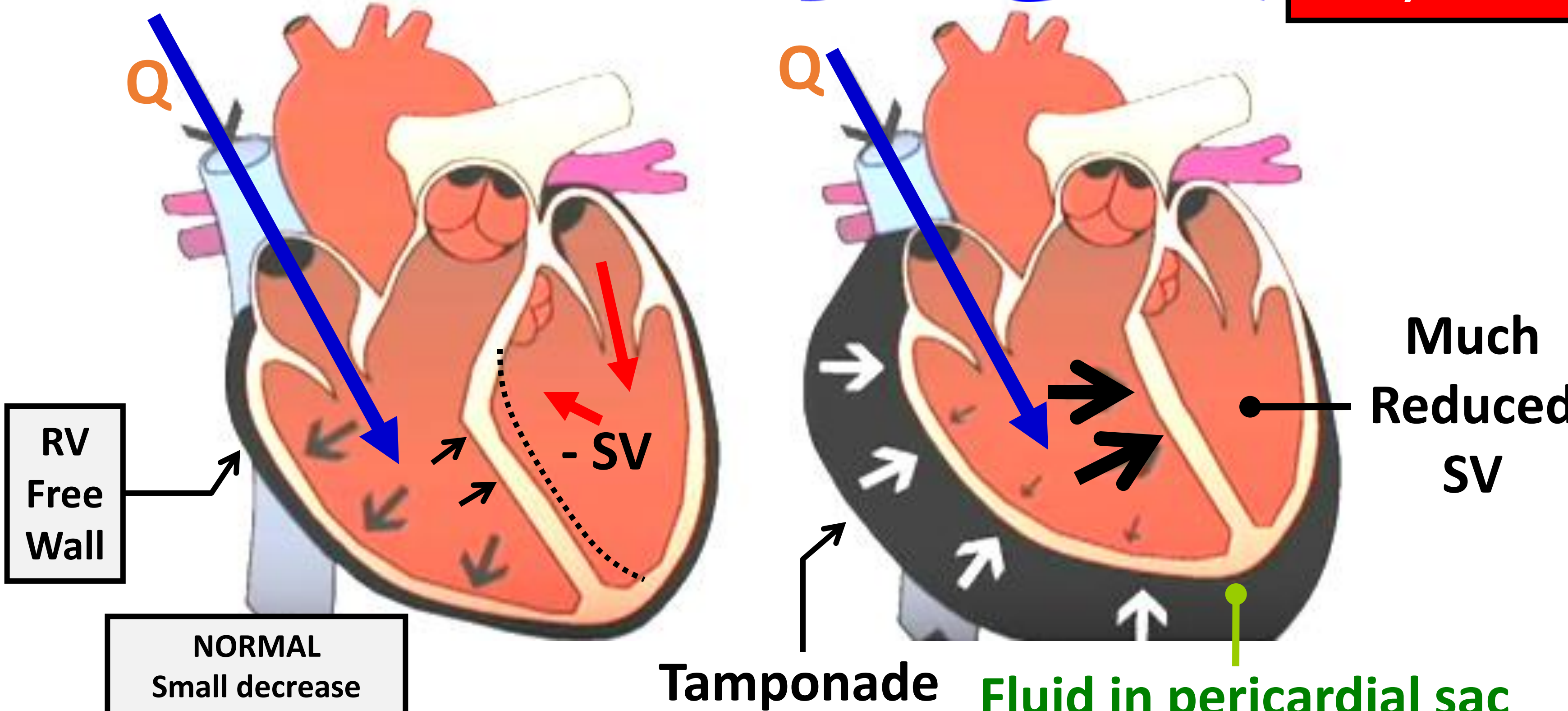
Abnormal Variation is Systolic BP with Respiration





Pulsus Paradoxus

ABNORMAL LARGE decrease In Systolic BP



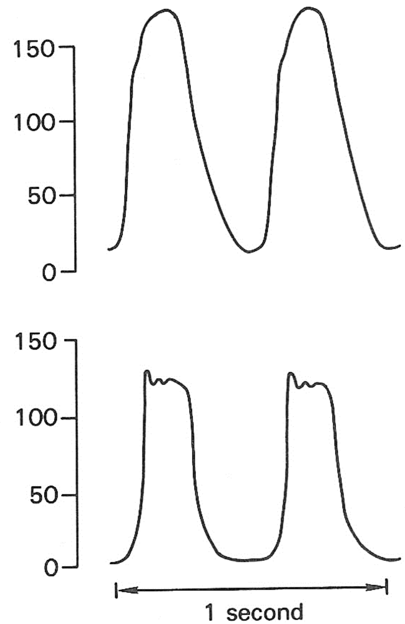
NORMAL
Small decrease
In Systolic BP

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

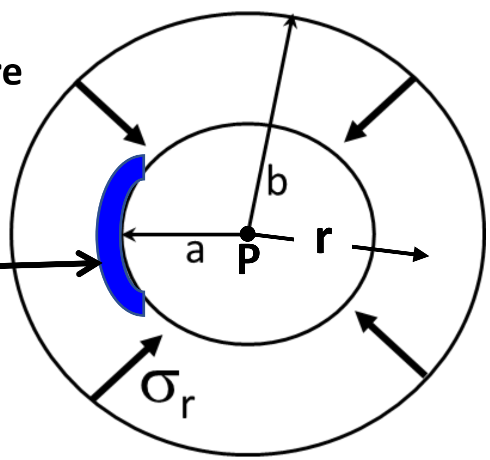
Intra-Myocardial Pressures

Intramyocardial Pressure (mmHg)

Left Ventricle Pressure (mmHg)



Compressed more
Flow reduced
during systole



@ r = a σ = -P

@ r = b σ = 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

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

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

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

Which cardiovascular system change would tend to increase arterial 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 and small arteries

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

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

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