TEACH

TRAINING & EDUCATION IN ADVANCED CARDIOVASCULAR HEMODYNAMICS

Preload, Afterload, Contractility and Lusitropy

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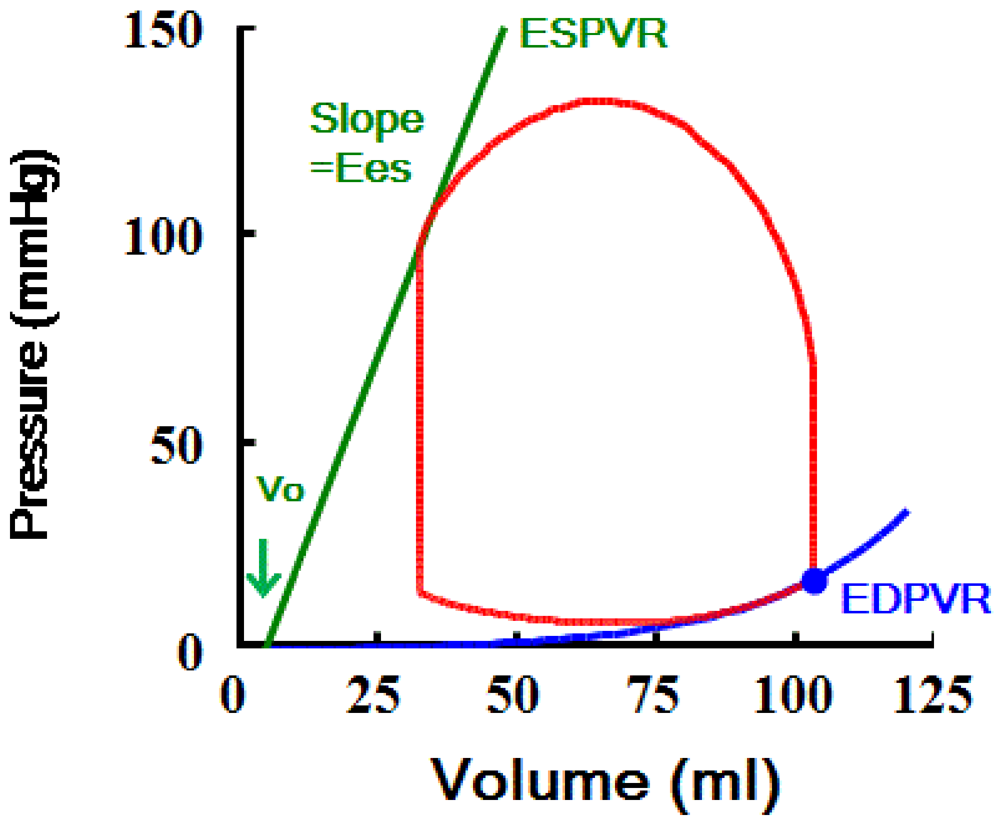
Cardiovascular Research Foundation

Objectives

- Review definitions and indexes of:
  - Preload
  - Afterload
  - Contractility
  - Lusitropy (diastolic properties)
- Understand how changes in these parameters are manifest on the pressure -volume diagram and their impact on cardiac performance:
  - blood pressure
  - cardiac output
  - CVP and PCWP

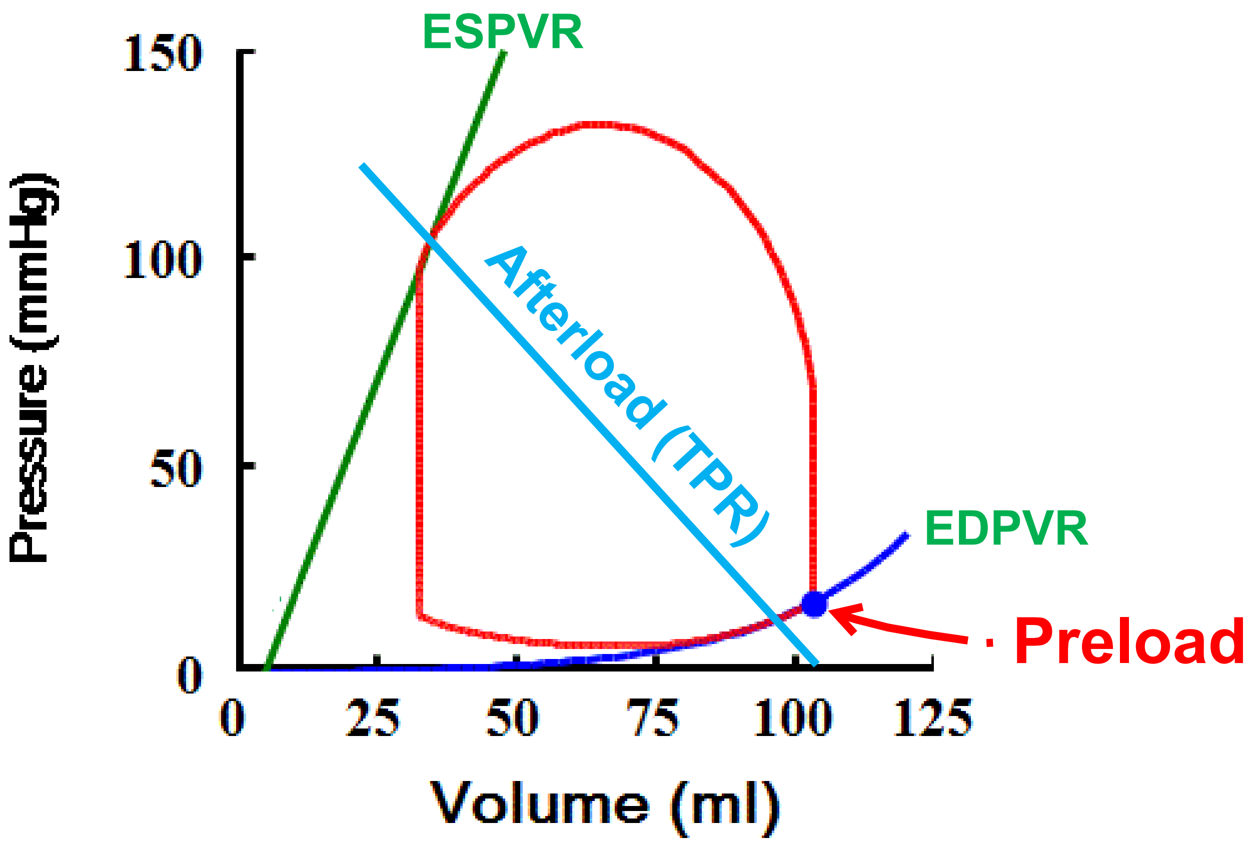
The Pressure-Volume Loop falls between the Boundaries set by the ESPVR and EDPVR



ESPVR: End-Systolic Pressure-Volume Relationship

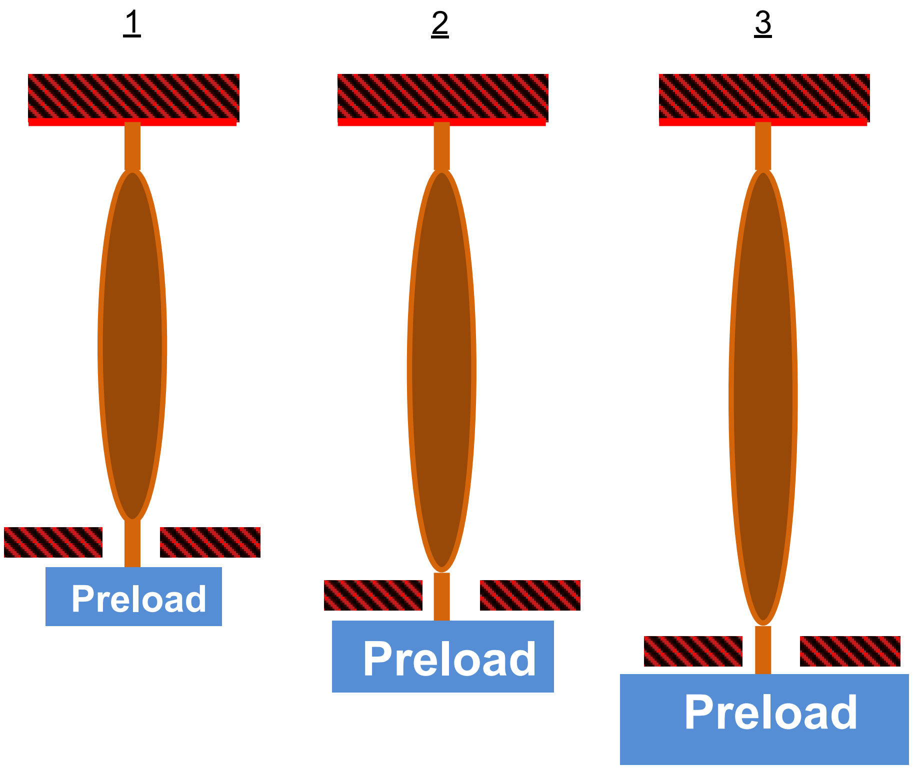
EDPVR: End-Diastolic Pressure-Volume Relationship

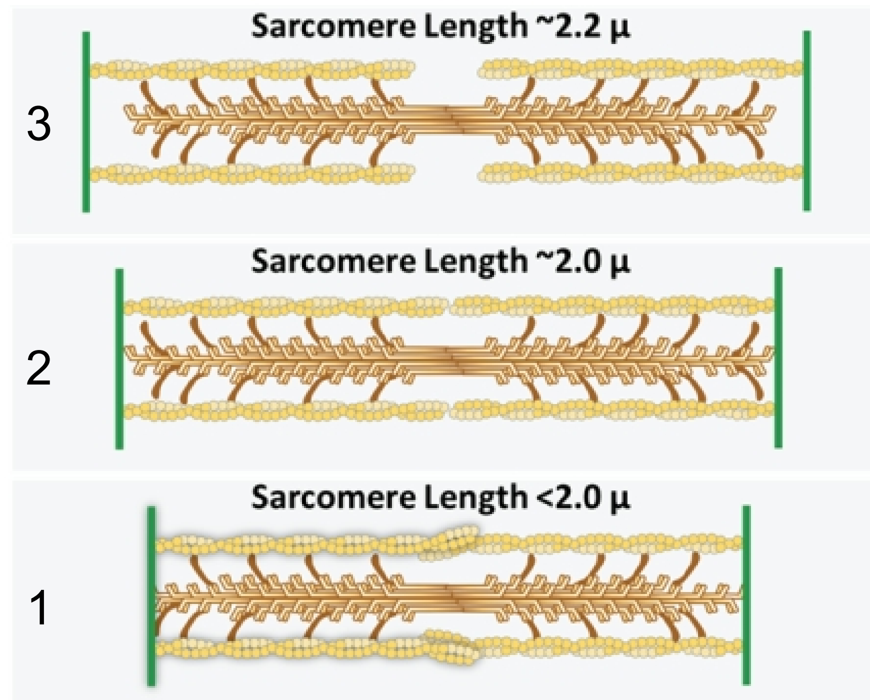
The Position of the PV Loop within the Boundaries of the ESPVR and EDPVR is determined by the Preload and Afterload



Preload

Preload: Sarcomere





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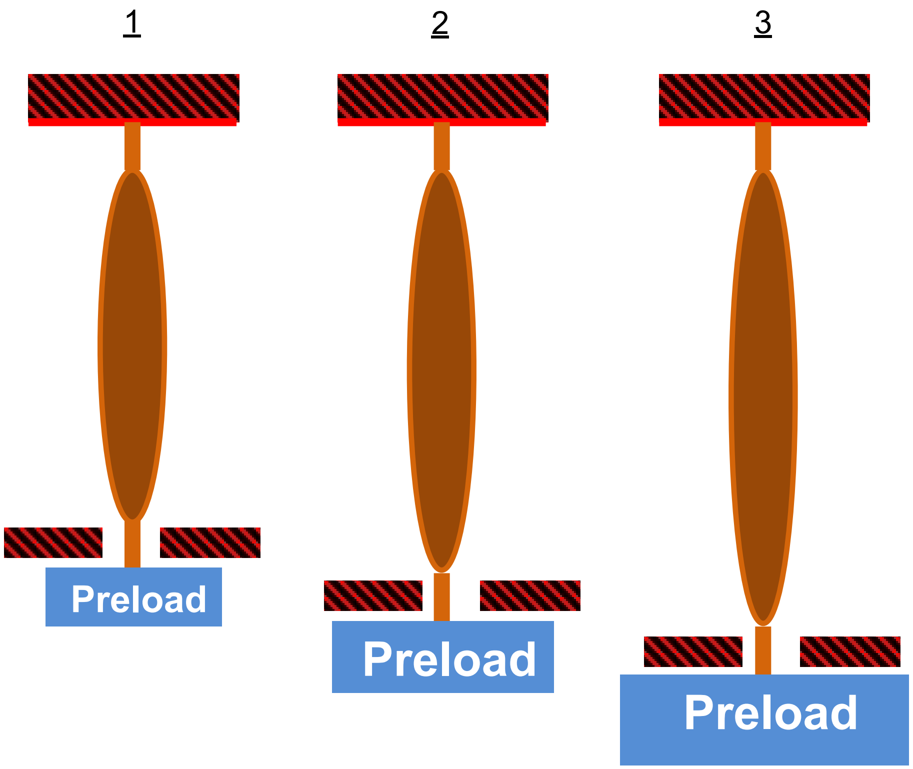
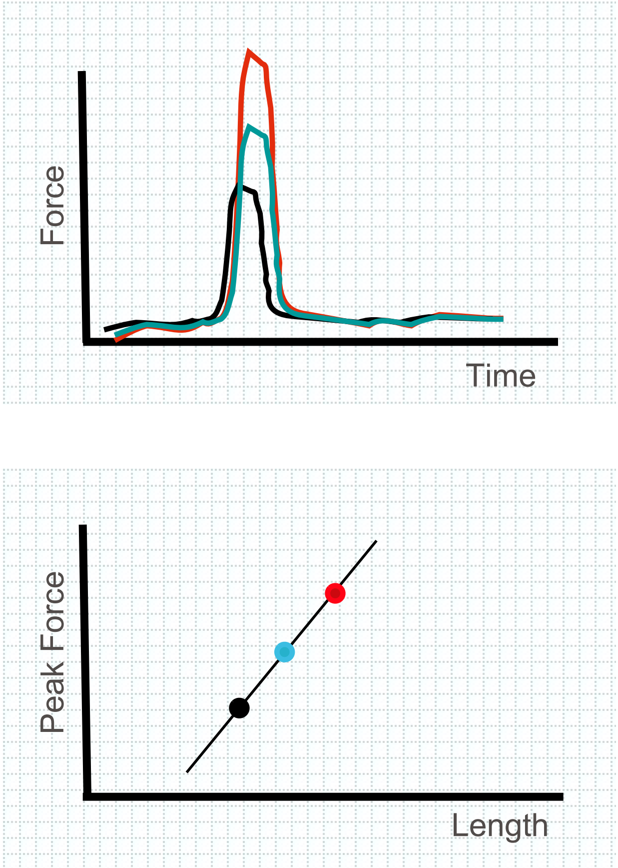
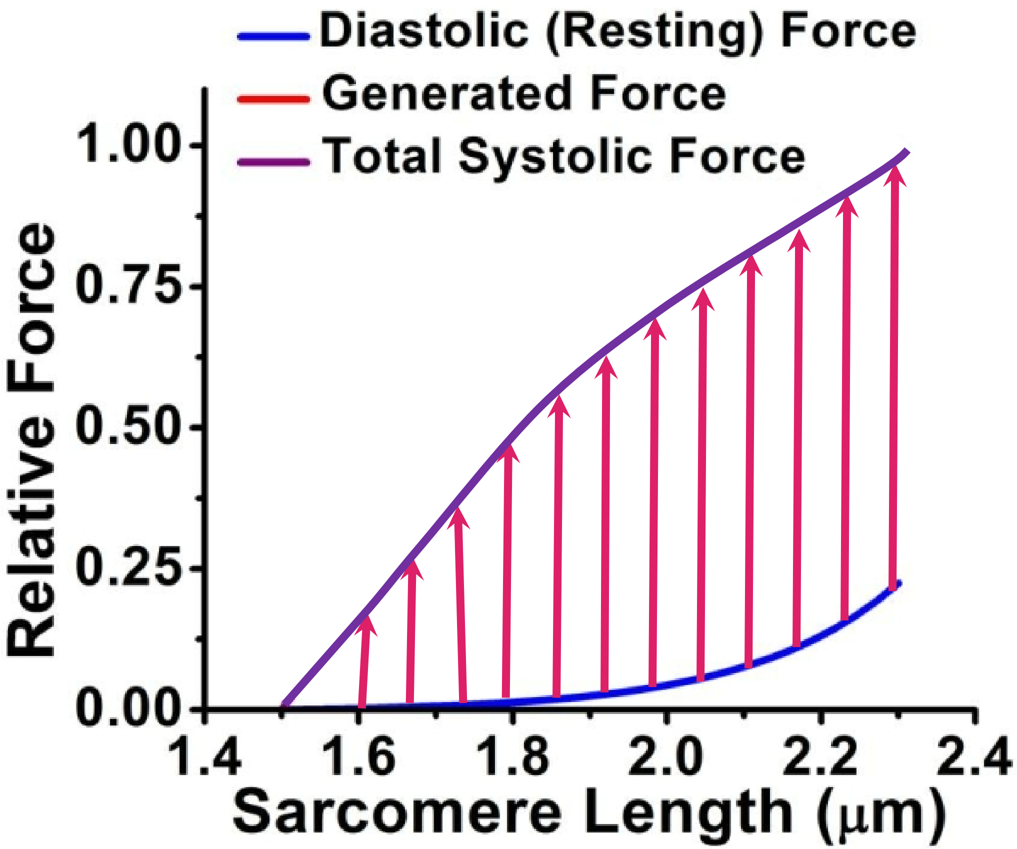




Sarcomere Isometric F-L Relation



Preload at the Ventricular Level

- Can't measure sarcomere length in the intact LV

- Most appropriate measures of preload in the intact LV are:
  - End-diastolic volume (EDV)
  - End-systolic volume (ESV)

Exercise 1: Preload

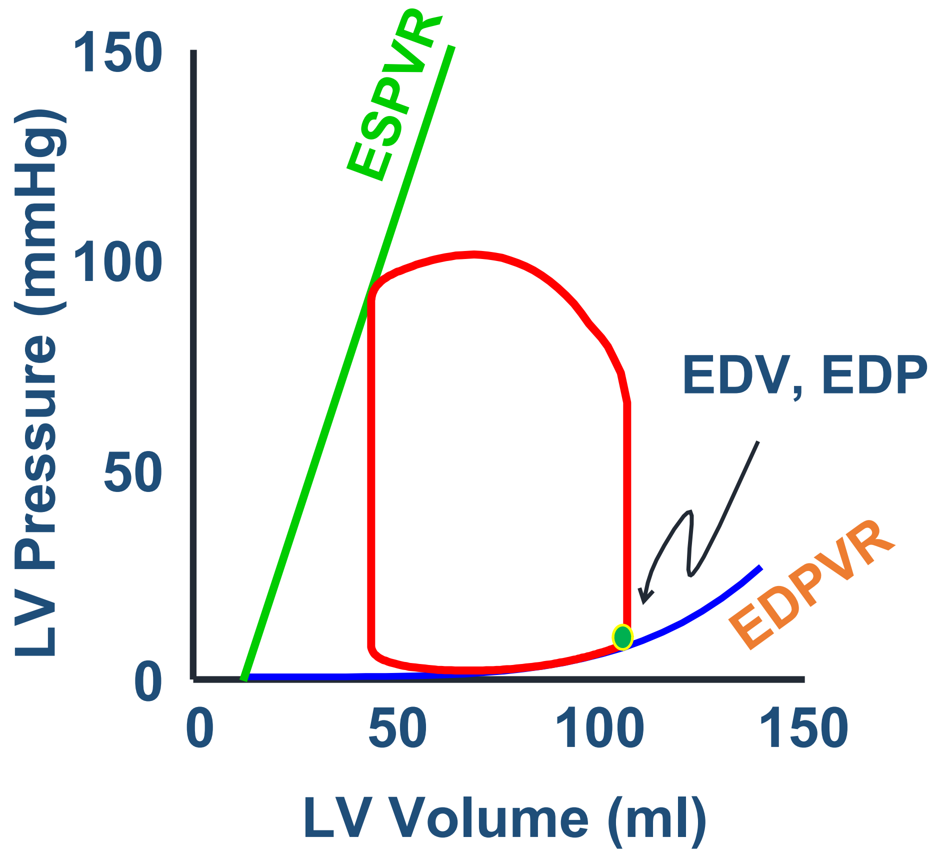
1. Open Preload *Try-it-Now*
2. Increase "*Stressed Blood Volume*"
3. Note what happens to the PV Loops
4. Focus on EDP, EDV, ESV, SV, Peak LV Pressure

Question 1

As preload increases, ESV:

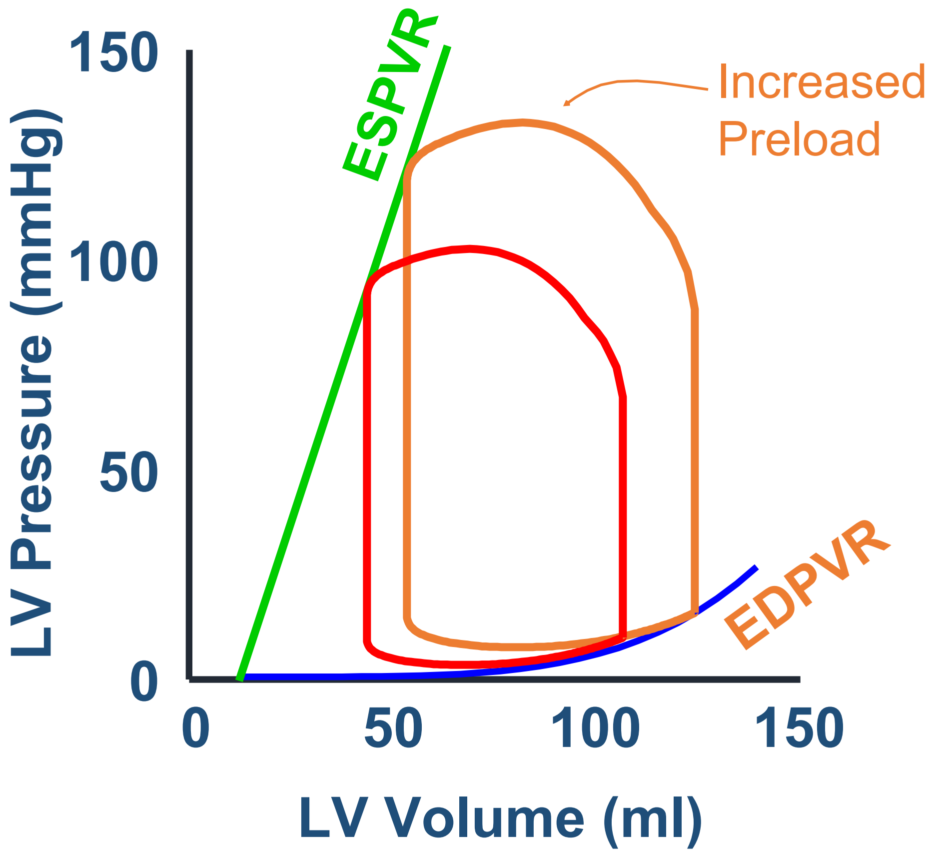
A. Increases
B. Decreases
C. Stays the same

Preload: Ventricular Level



Preload:
The load imposed on the ventricle at the end of diastole. The most common measures of preload include end-diastolic volume (EDV) and end-diastolic pressure (EDP).

Preload: Ventricular Level



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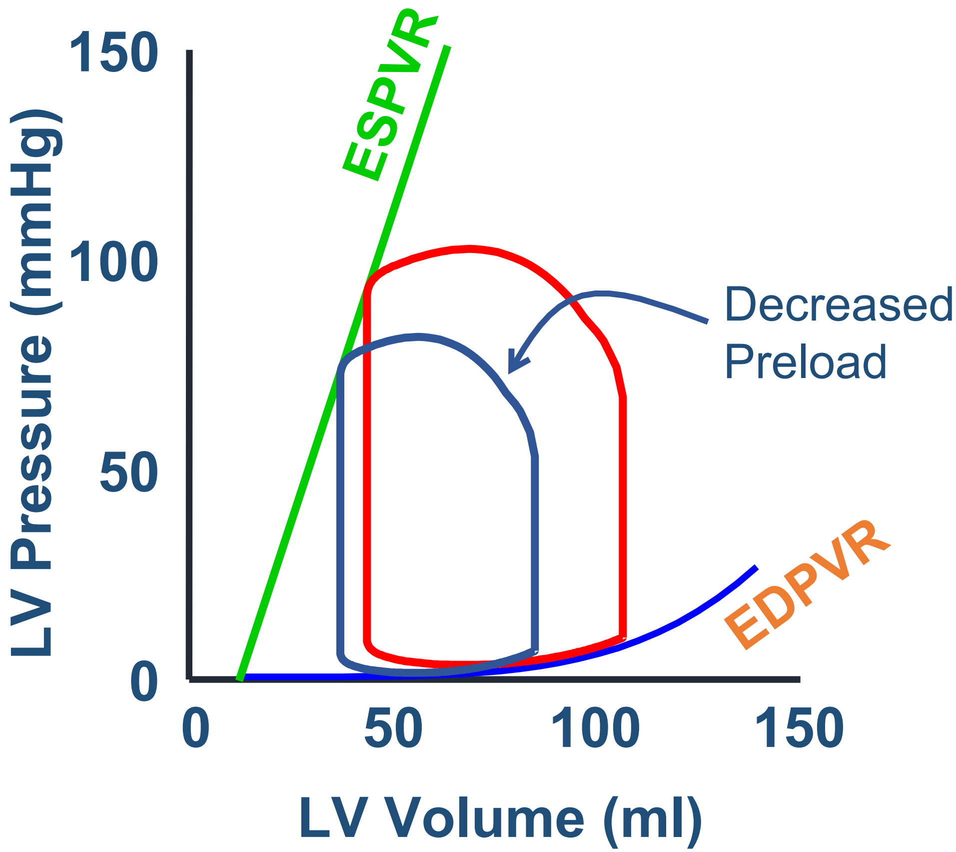

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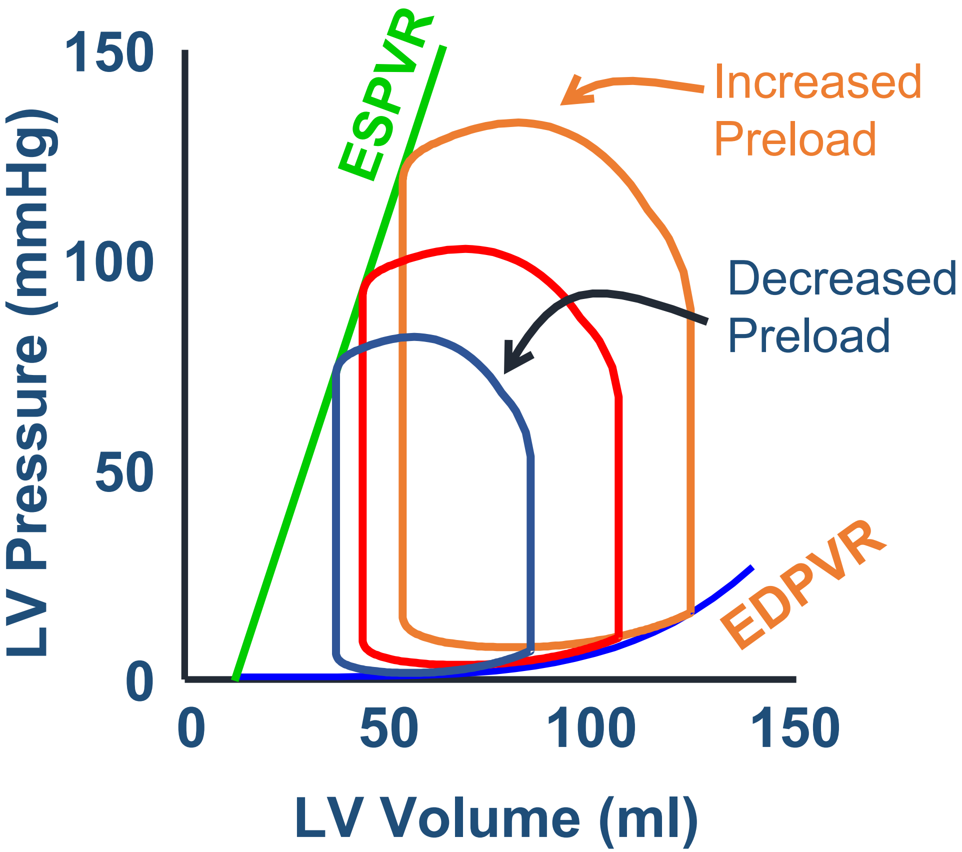
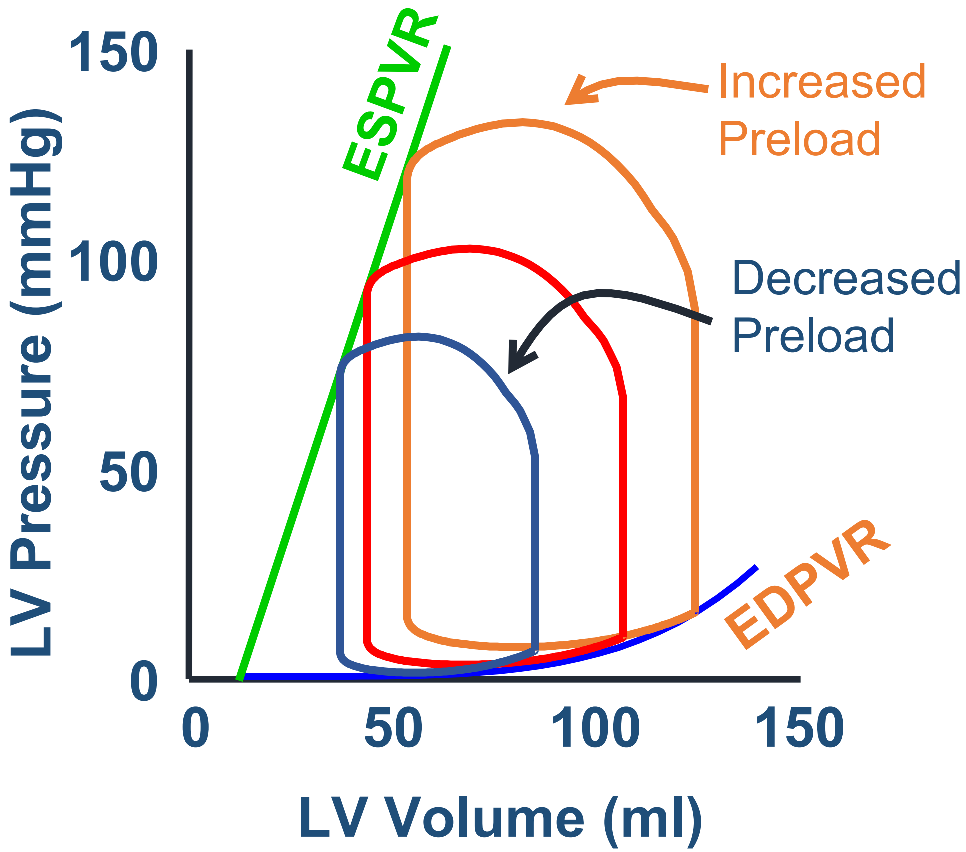


The different loops are obtained with different preloads, but constant *contractility* and *afterload resistance*.

Preload: Ventricular Level



The loops fall within the boundaries of the ESPVR and EDPVR and despite constant *afterload resistance* and *contractility:*

changes in *preload* induce changes in stroke volume (and CO) and arterial blood pressure

Afterload

Ventricular Afterload

- Ventricular *afterload* is the hemodynamic (mechanical) load against which the ventricle contracts and must overcome in order to eject blood
- Common measures of *afterload* include:
  - Wall stress
  - Arterial pressure
  - Vascular resistance (TPR = MAP/CO)
  - Effective arterial elastance (discussed later)
  - Vascular impedance

Ventricular Afterload

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- Common measures of *afterload* include:
  - Wall stress
    - depends on preload, contractility, vascular resistance
  - Arterial pressure
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- Common measures of *afterload* include:
  - Wall stress
    - depends on preload, contractility, vascular resistance
  - Arterial pressure
    - depends on preload, contractility, vascular resistance
  - Vascular resistance (TPR = MAP/CO)
    - Independent of other factors
  - Effective arterial elastance (discussed later)
    - TPR*HR
    - Can be represented on the PV diagram
  - Vascular impedance
    - Independent of other factors
    - Most comprehensive descriptor of vascular mechanical properties

Exercise 2: Afterload

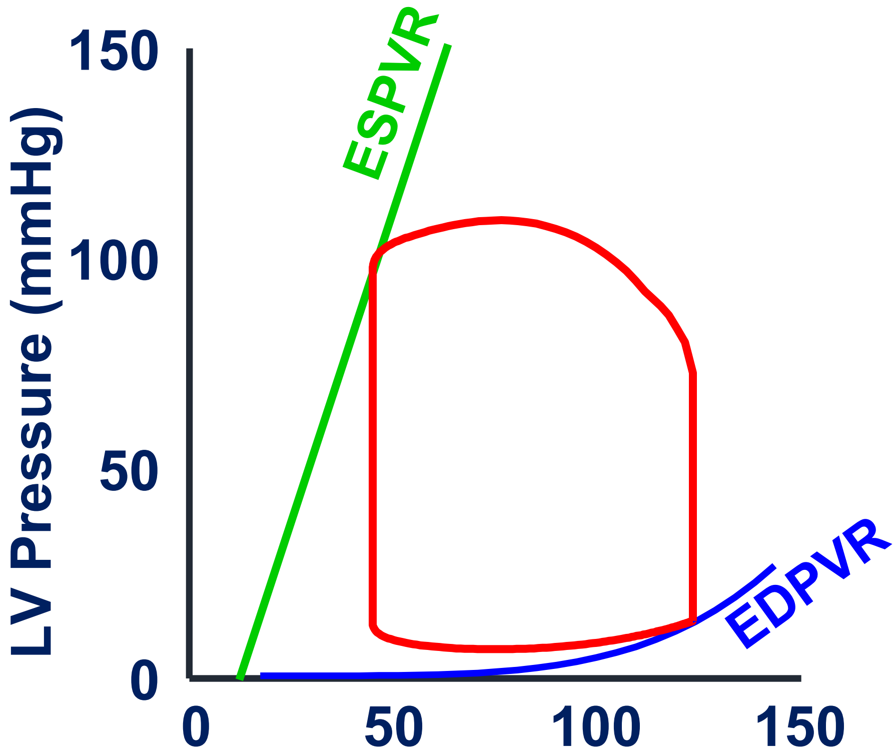
- Open the Afterload *Try-it-Now*
- Increase “Afterload Resistance” (Ra)
- Notice the changes in peak pressure, ESV, EDV, SV and Ejection Fraction

QUESTION 2:

In response to an increase in arterial resistance (Ra), which of the following DECREASES?

A. Contractility
B. End-systolic Volume
C. Ejection Fraction
D. All of the above decrease

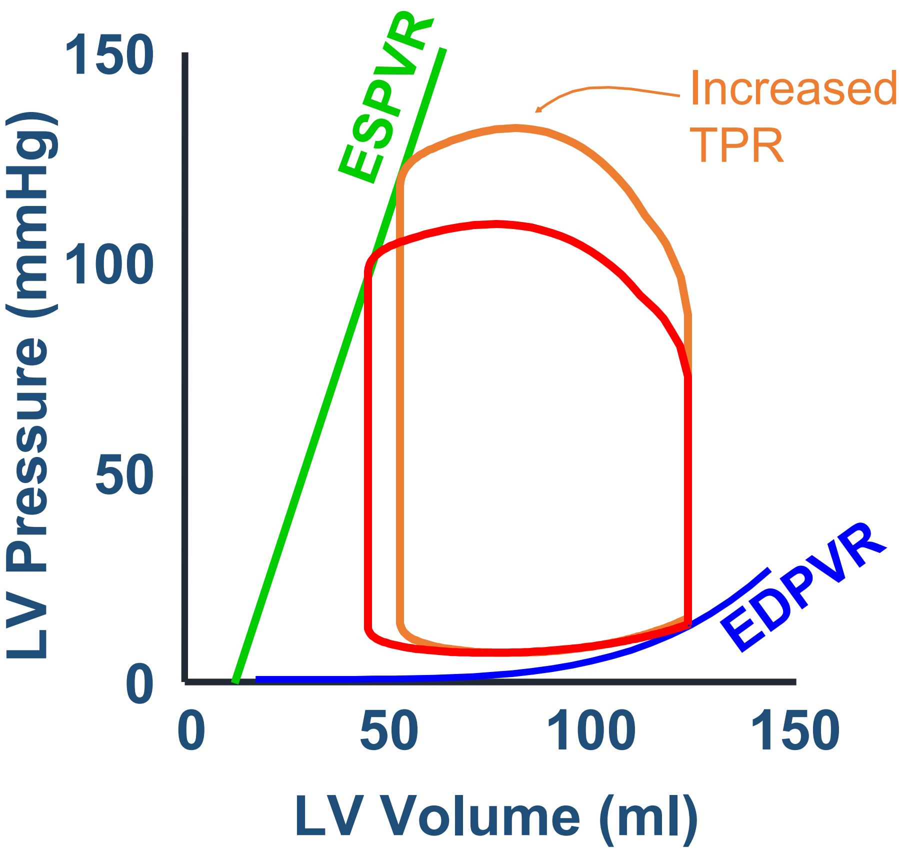
Afterload: Impact on LV Performance



Afterload: The mechanical load on the ventricle that must be overcome in order to eject blood. Under normal physiological conditions, this is determined by the arterial system. The most common index of afterload is arterial resistance, or total peripheral resis-tance (TPR):

TPR = (MAP-CVP)/CO

Afterload: Impact on LV Performance

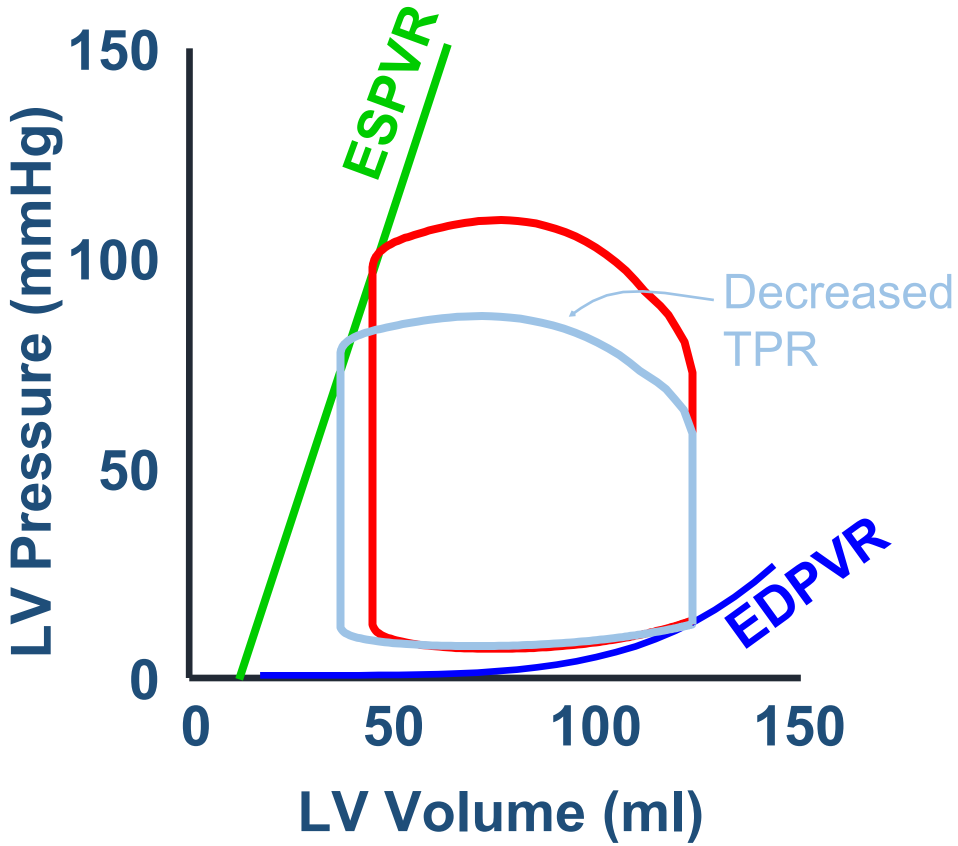


Despite constant preload and contractility:

Increased TPR

- Increases pressure
- Decreases SV

Afterload: Impact on LV Performance



Despite constant preload and contractility:

Increased TPR

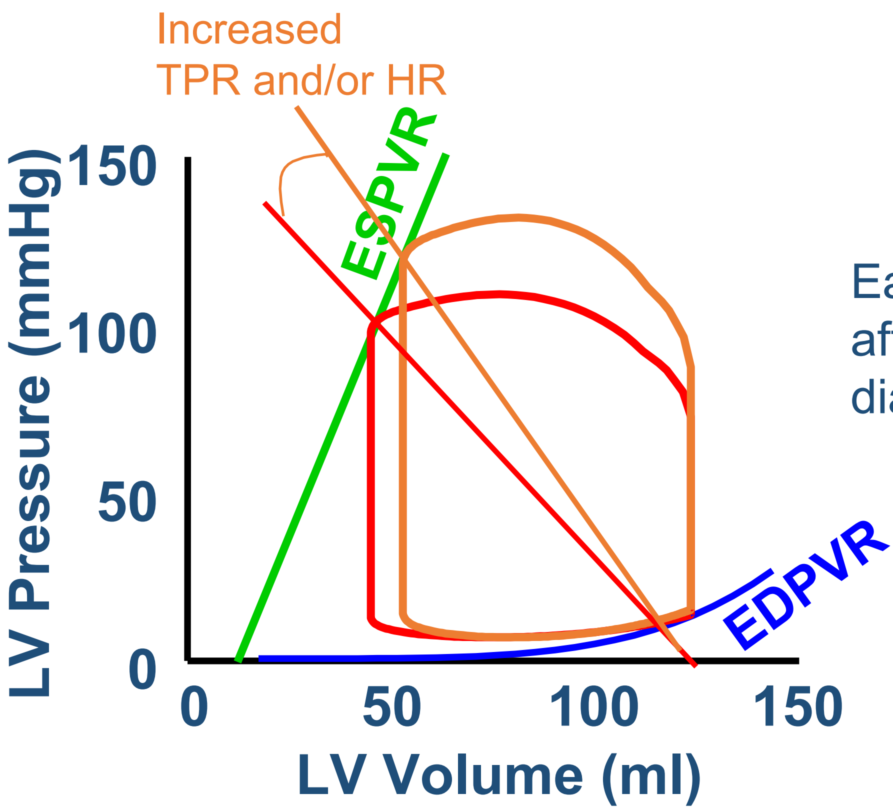
- Increases pressure
- Decreases SV

Decreased TPR

- Decreases SV
- Increases pressure

Afterload: Impact on LV Performance

Ea = Effective Arterial Elastance
= TPR·HR
= Pes/SV



Ea: a means of displaying afterload resistance on the PV diagram

Contractility

Contractility

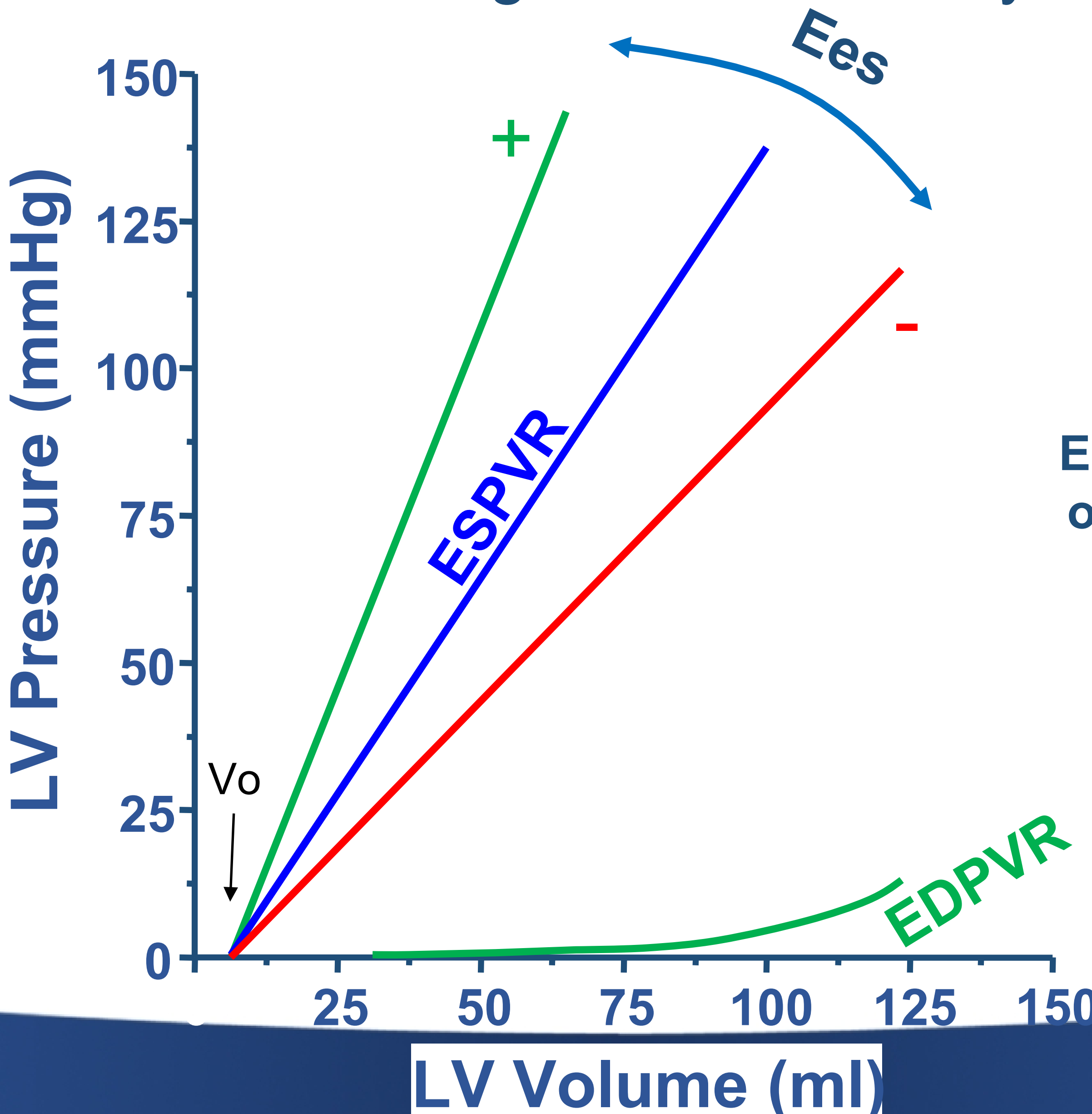
- There is no precise definition of *contractility*
- Conceptually, *contractility* refers to the intrinsic strength of the ventricle, <u>independent</u> of the phenomenon whereby changes in loading conditions (preload or afterload) result in changes in pressure and flow generation

Contractility
Muscle vs Ventricle

Ventricular contractility affected by:

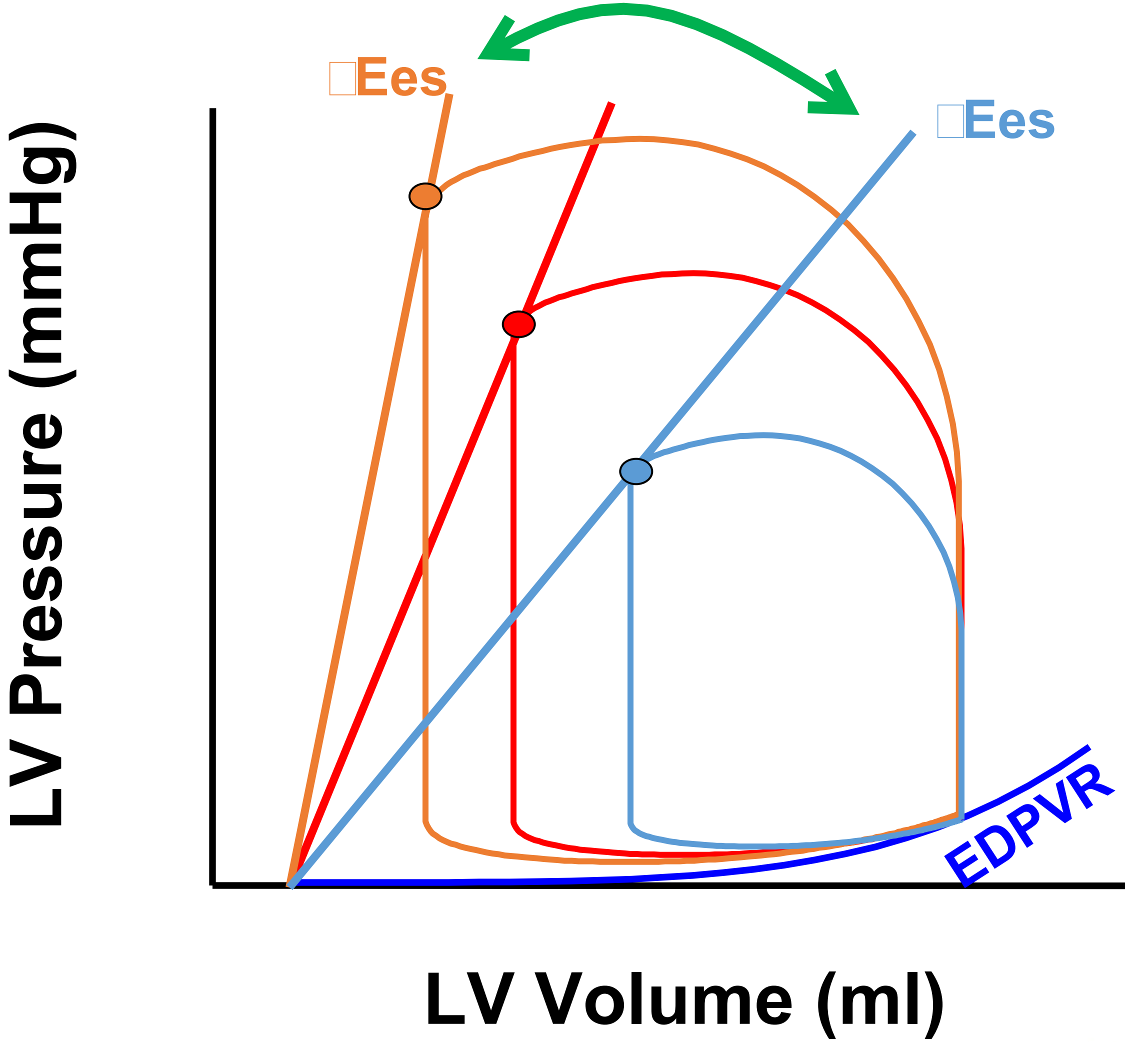
- All of the things that affect cellular contractility (calcium, energy supply, ischemia, pH, temp, cytoskeleton, etc)
- Myocardial Mass
- Synchrony of contraction
- Regional differences in mechanical properties (as in ischemia/infarct)

Slope of the ESPVR (Ees) changes with changes of contractility



Ees is an index of contractility

Changes in Contractility



Exercise 3: Contractility

- Open Contractility *Try-it-Now*
- Decrease Contractility such that SV reduces by 30%
- Notice what happens to EF and EDP

Question 3:

As a result of myocardial ischemia affecting 30% of the LV, which of the following increases substantially?EF

A. EDP
B. SV
C. ESV

Question 4: *Contractility:*

As preload increases, *Contractility:*

A. Increases

B. Decreases

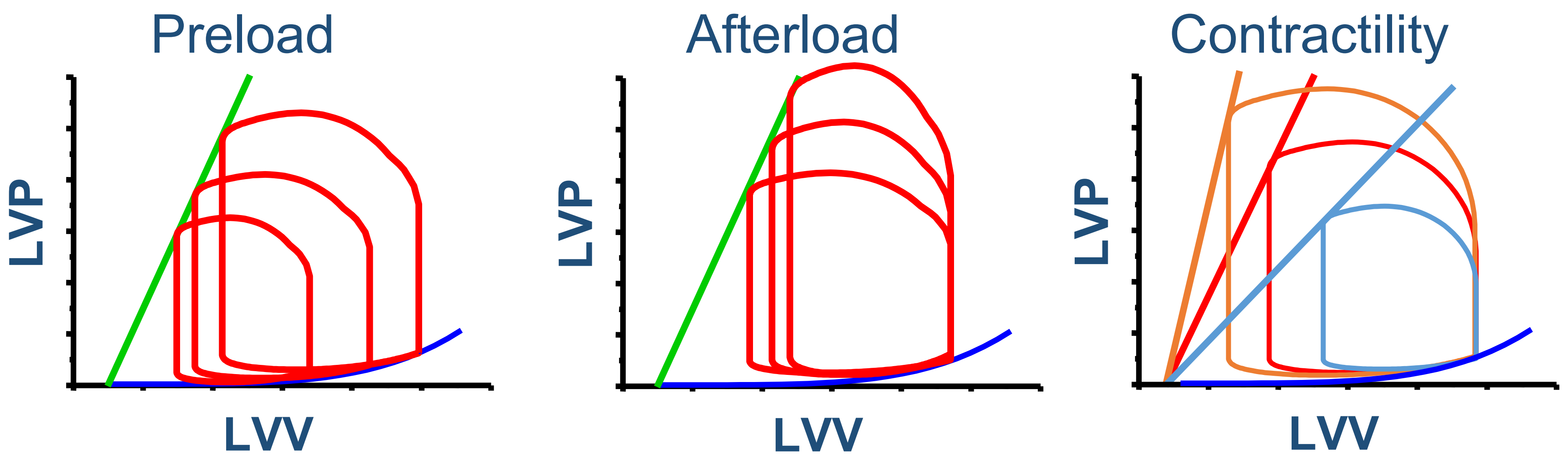
C. Stays the same

Ejection Fraction (SV/EDV)

The most commonly used index of *contractility*

Three important points:

1) EF varies with contractility
2) There are minor effects of preload
3) Afterload can impact significantly on EF



Lusitropy / Diastole

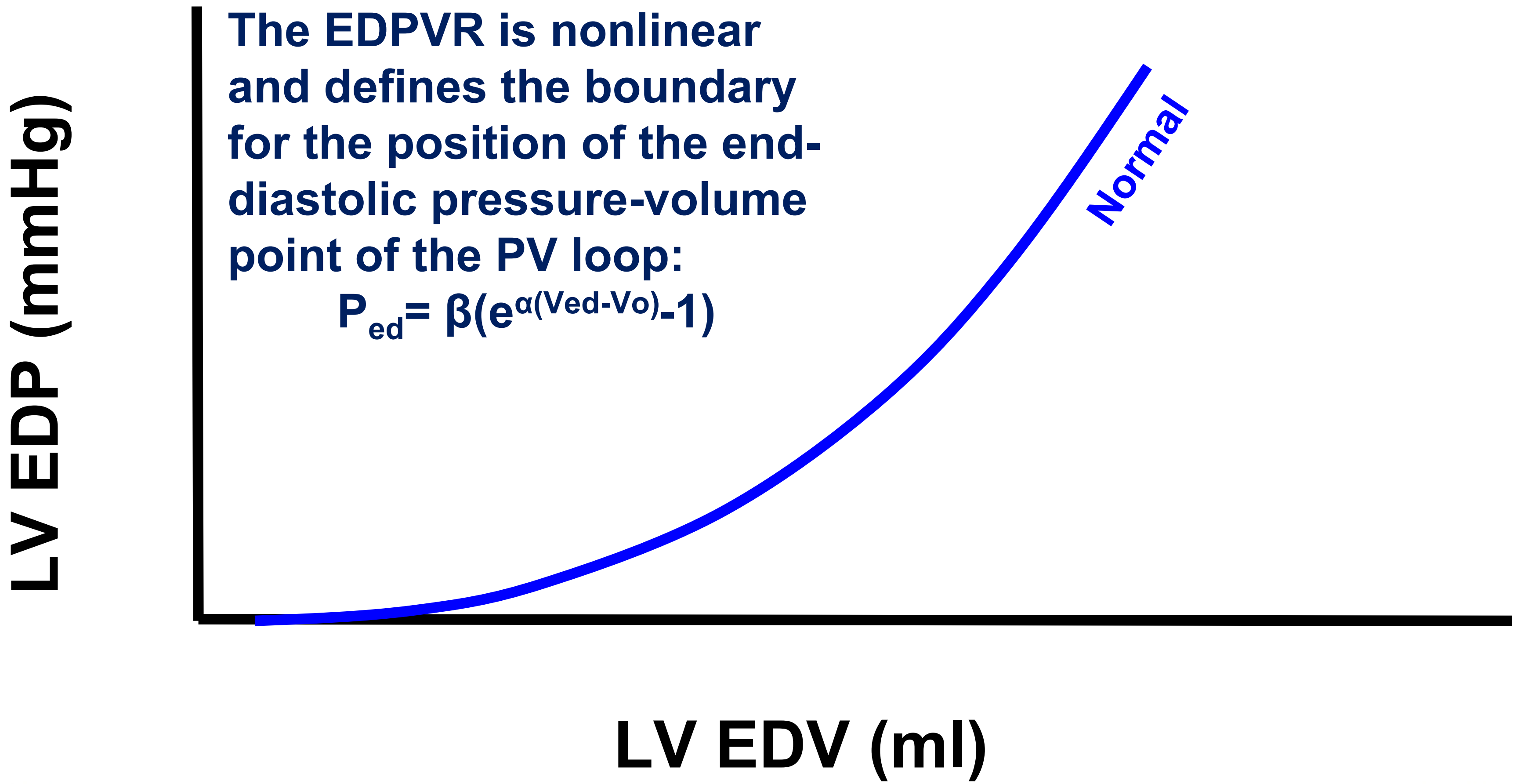
Lusitropy

"Lusitropy" refers to mechanical properties of the ventricle during relaxation and at the point of complete relaxation.

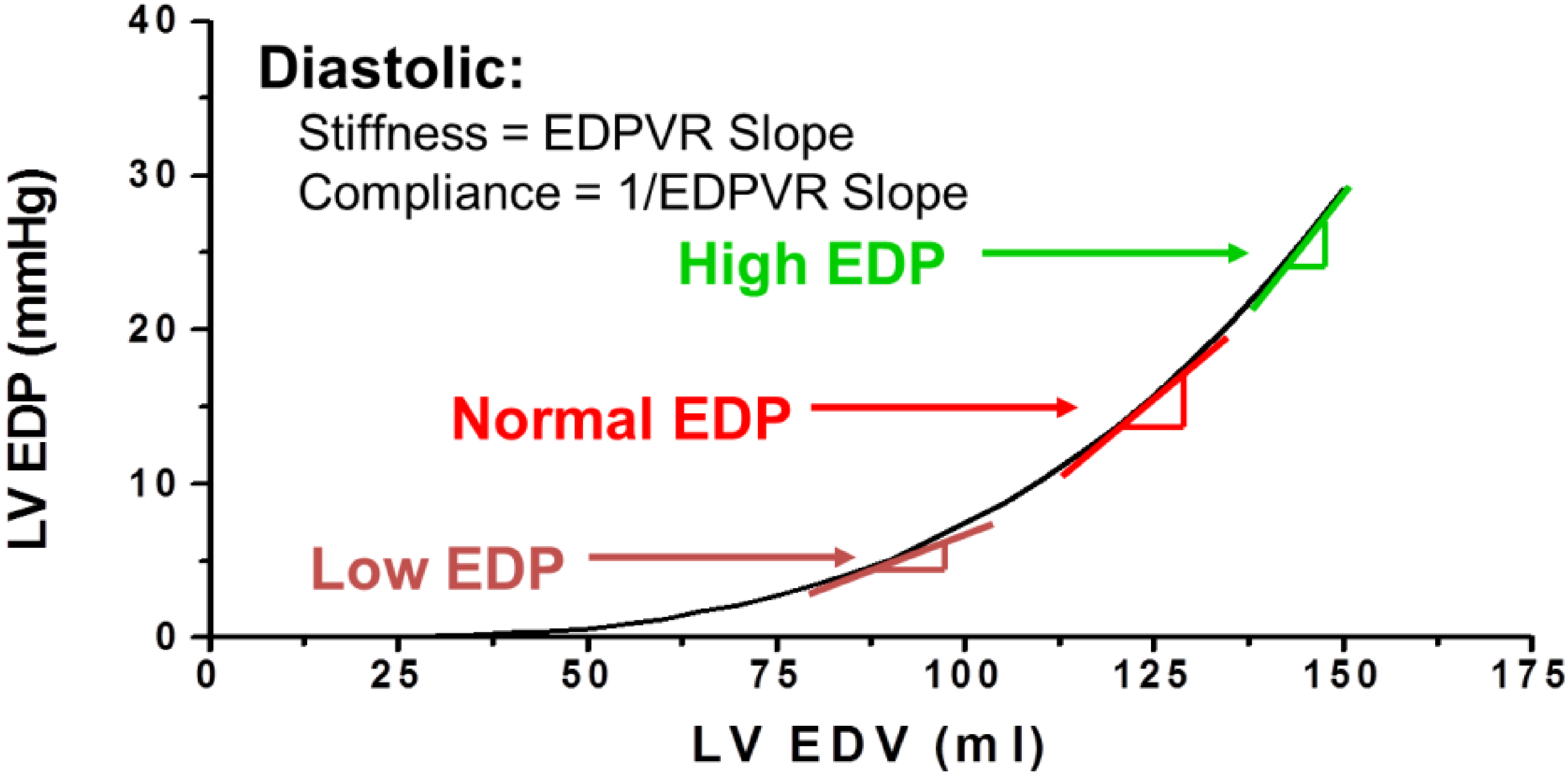
There are two distinct aspects of lusitropy:

- Active relaxation: the **rate of relaxation**
- Passive diastolic properties: **the extent of relaxation**
  - Compliance
  - Stiffness
  - Capacitance

Lusitropy: Passive Properties



Lusitropy: Passive Properties



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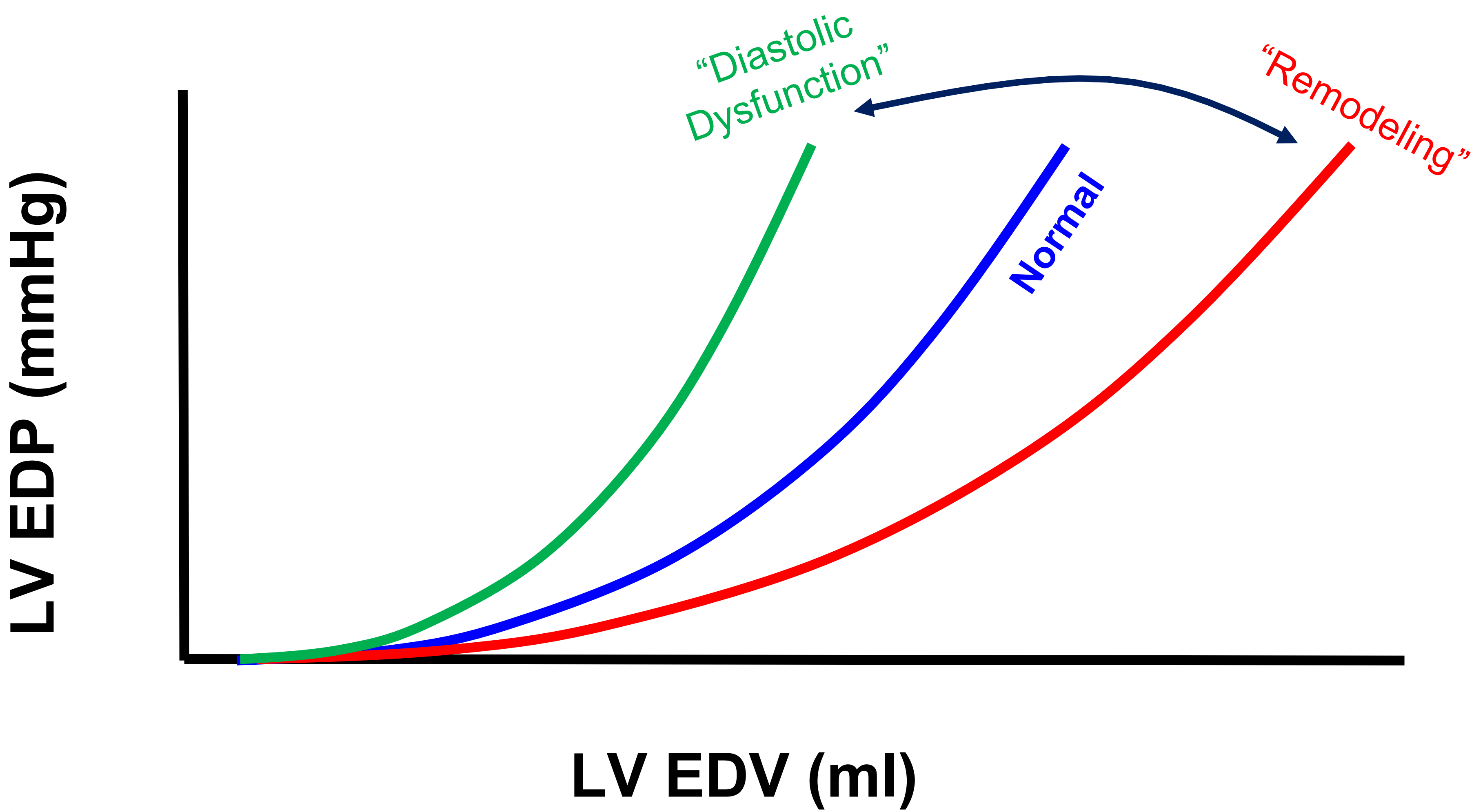

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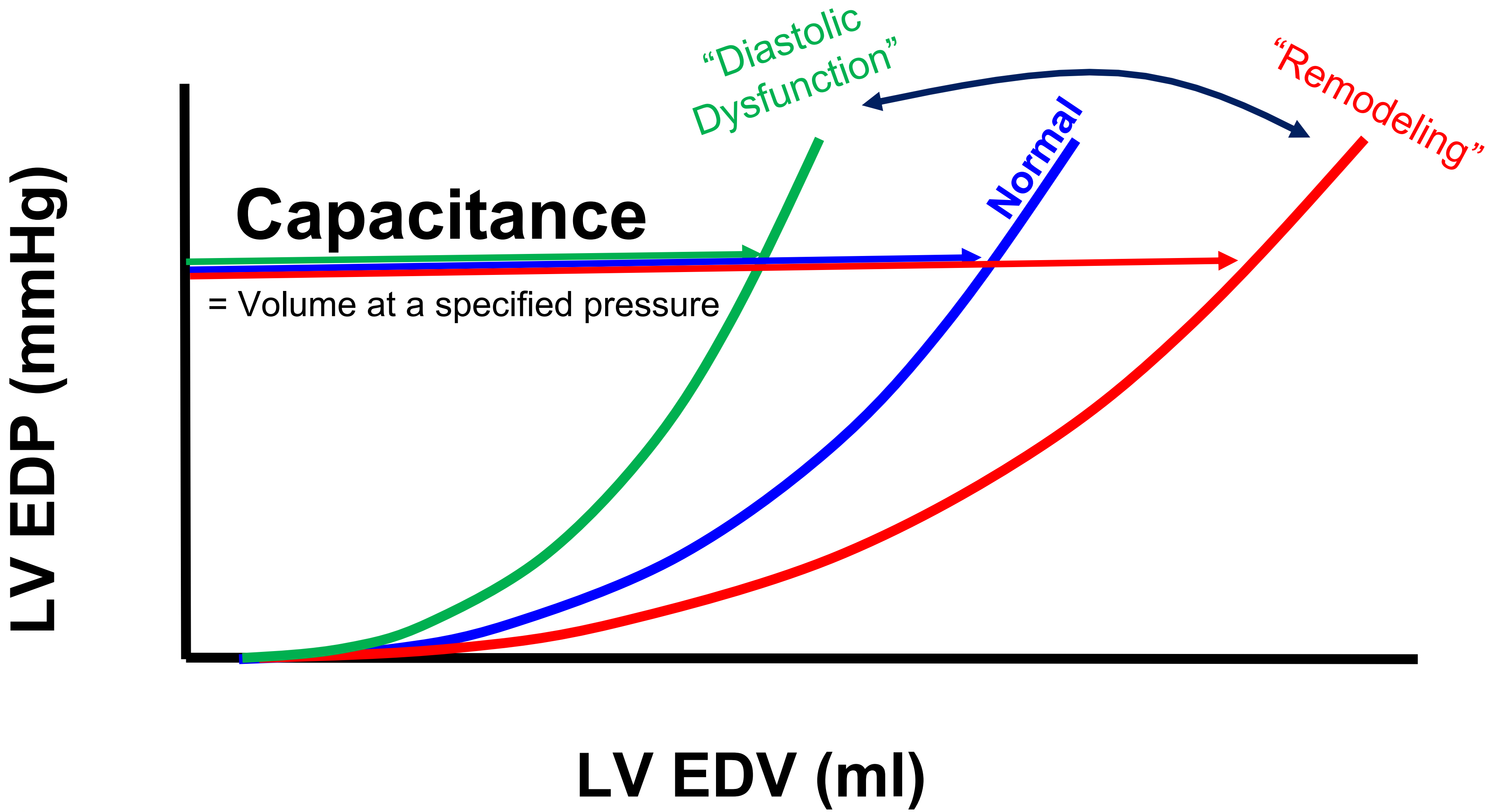
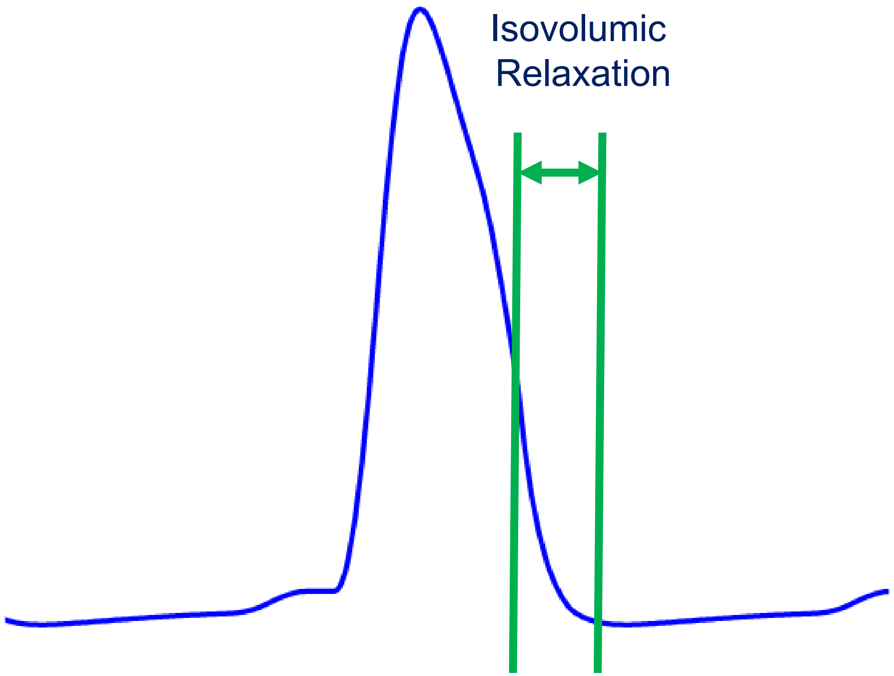


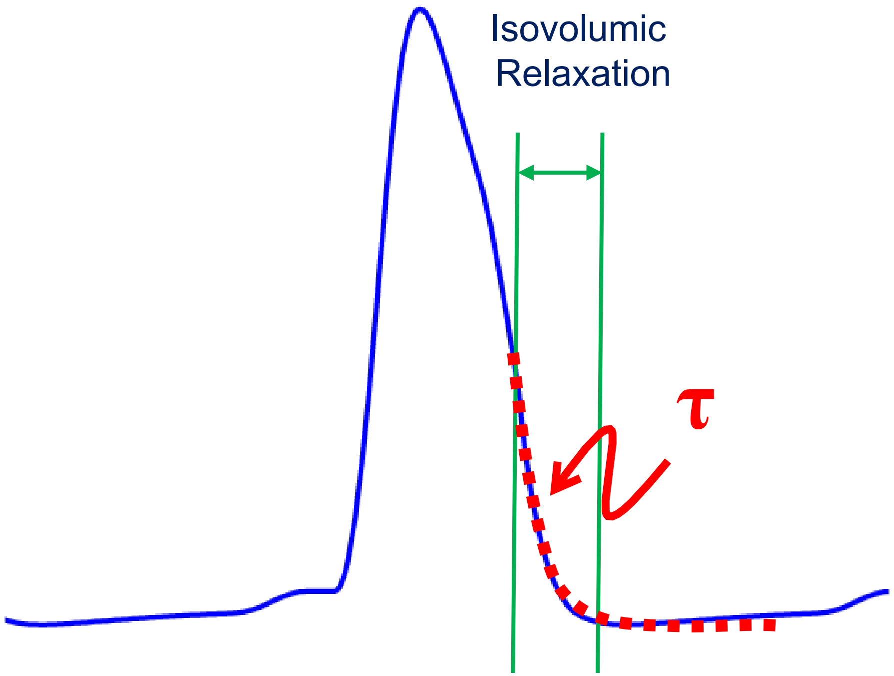
Lusitropy: Active Relaxation



The decay of pressure during the isovolumic relaxation phase of diastole follows a roughly exponential time course.

This can therefore be characterized by τ, the time constant of relaxation.

Lusitropy: Active Relaxation



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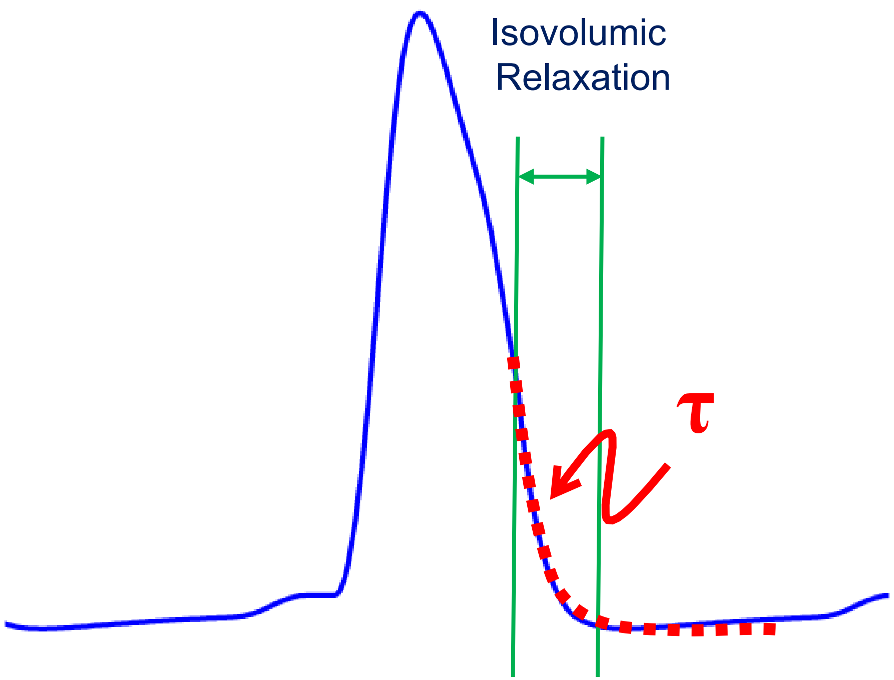


τ depends on rate of X-bridge uncoupling:

- Is ATP-dependent
- Rate increase = τ decrease
- τ decreases as HR increases
- τ decreases with β-agonists
- τ increases with ischemia

Lusitropy:
The Rate of Relaxation

Changes in τ can have profound impact on cardiac performance especially at high heart rates and high values of τ

Exercise: Lusitropy

1. Open the Diastole-Active *Try-it-Now*
2. Increase HR from 60 to 120 and notice changes in PV loops
3. Return HR back to 60. Increase τ from 25 to 75 and notice changes in PV loops
4. With τ at 75 ms, increase HR from 60 to 120.

Question 5:

With τ set at 75 ms and a HR of 120 bmp, how did the position of the PV loops change?

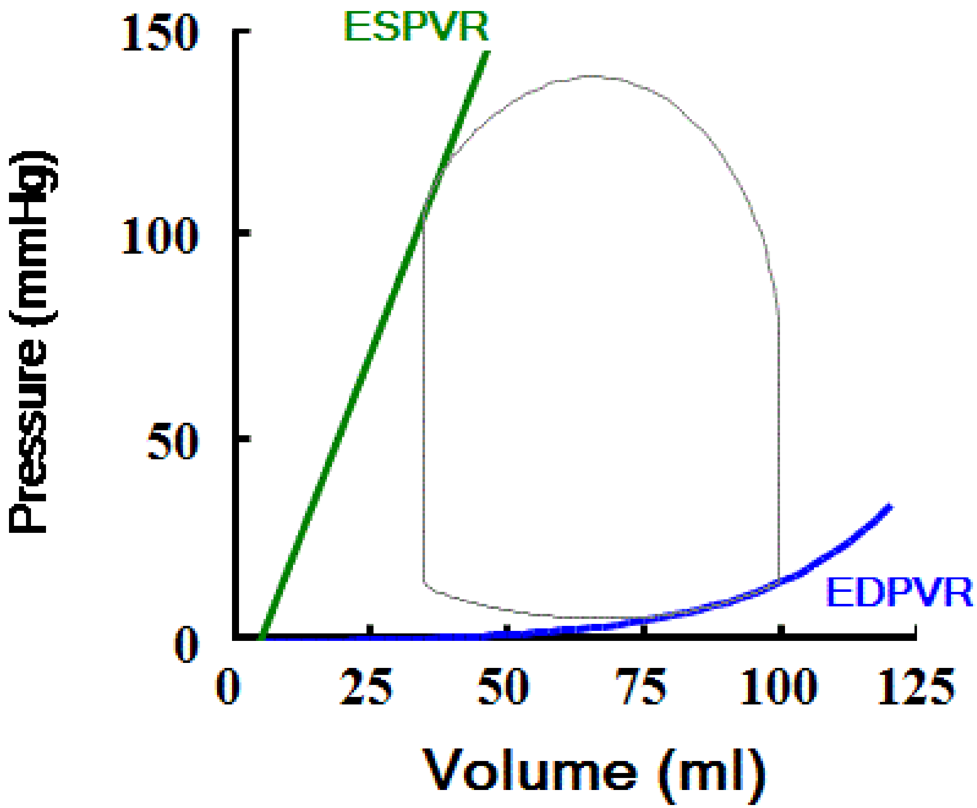
A. The PV loops falls on the EDPVR as usual

B. The PV loop breaches the ESPVR so that the end-systolic point is shifted upwards and leftwards compared to the ESPVR

C. The PV loop fails to reach the EDPVR at end-diastole. Instead, the end-diastolic pressure-volume point is shifted upwards compared to the EDPVR

D. The EDPVR, reflecting the true passive LV diastolic properties, shifts upwards indicating an intrinsically stiffer heart

Incomplete Relaxation

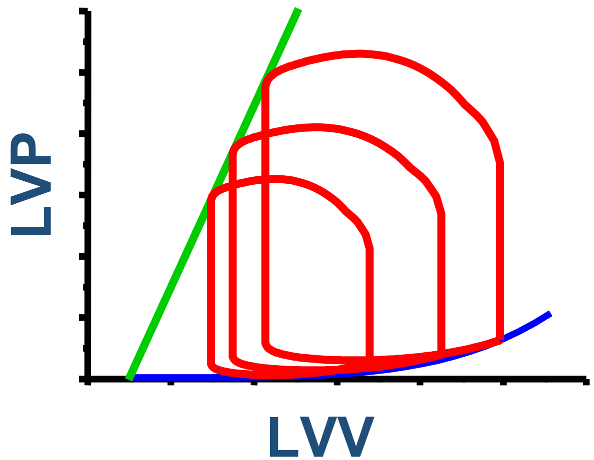


Occurs with:

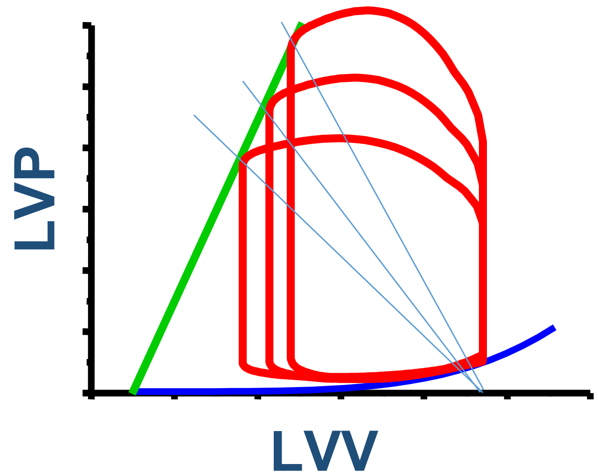
- increased τ
- increased HR

SUMMARY

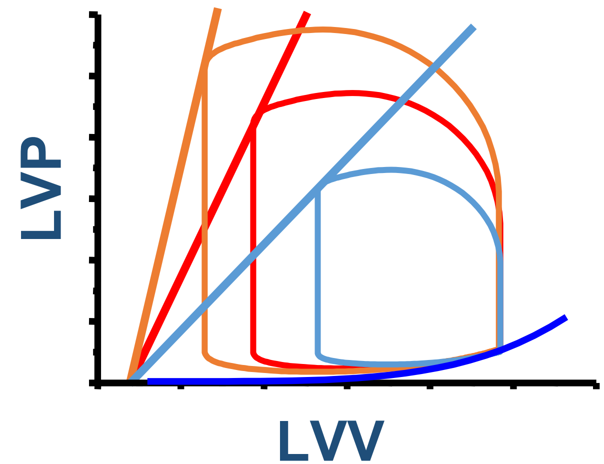
Preload



Afterload



Contractility



Lusitropy/Diastole