

Heart Pump and Cardiac Cycle

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Objectives

- To understand the volume, mechanical, pressure and electrical changes during the cardiac cycle
- To understand the inter-relationship between all these changes
- To describe the factors that regulate Cardiac output and Stroke volume.
- Resources: **Textbook of Medical Physiology By Guyton and Hall**

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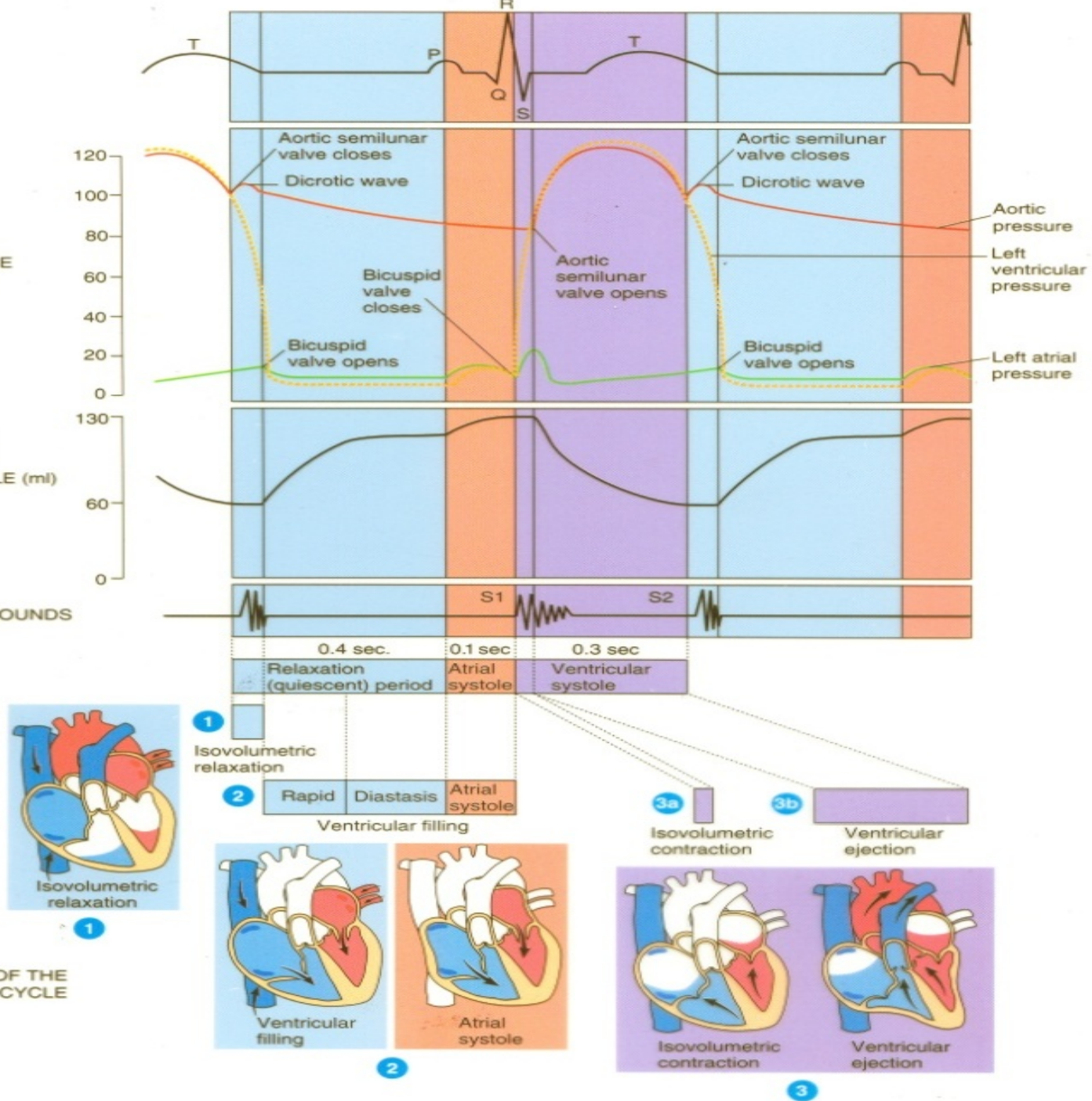
(a) ECG

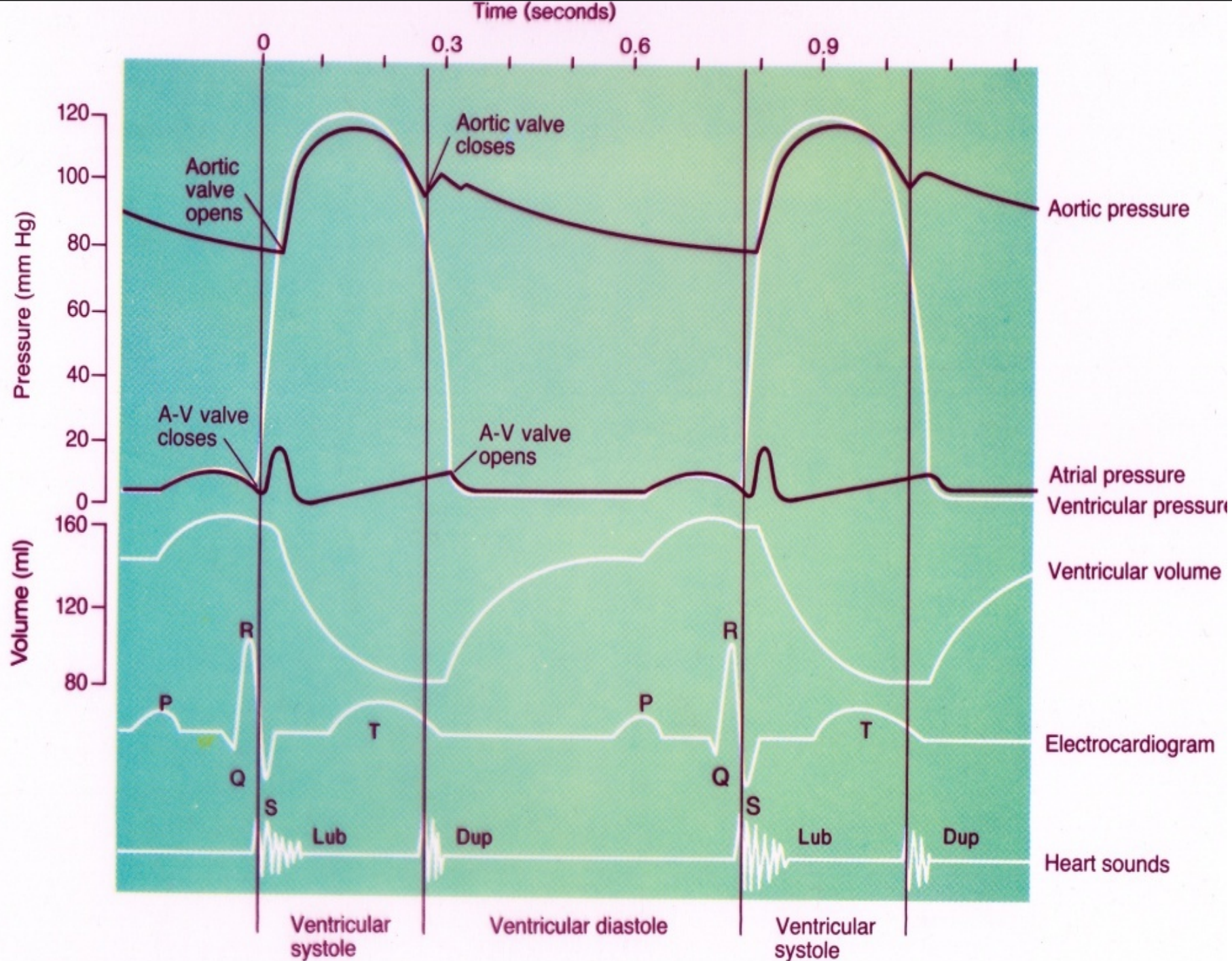
(b) PRESSURE (mm Hg)

(c) VOLUME OF LEFT VENTRICLE (ml)

(d) HEART SOUNDS

(e) PHASES OF THE CARDIAC CYCLE





Cardiac Cycle

- Cardiac cycle refers to all events associated with blood flow through the heart
 - Systole – contraction of heart muscle
 - Diastole – relaxation of heart muscle

Cardiac Cycle

- Atrial systole 0.1 second
- Atrial diastole 0.7 second
- Ventricular systole 0.3 second
 - Isovolumic contraction 0.01 seconds
 - Rapid ejection period
 - Slow ejection period
- Ventricular diastole 0.5 seconds
 - Isovolumic relaxation 0.02 seconds
 - Rapid filling
 - Slow filling (Diastasis)
 - Atrial contraction

Cardiac cycle ...cont

- End diastolic volume (EDV) – End systolic volume (ESV) = Stroke volume (SV)
- $SV \times \text{heart rate (HR)} = \text{cardiac output (CO)}$
- Ejection fraction = SV/EDV
- Inotropic vs. Chronotropic
- Autonomic control of cardiac cycle (pump)

Phases of the Cardiac Cycle

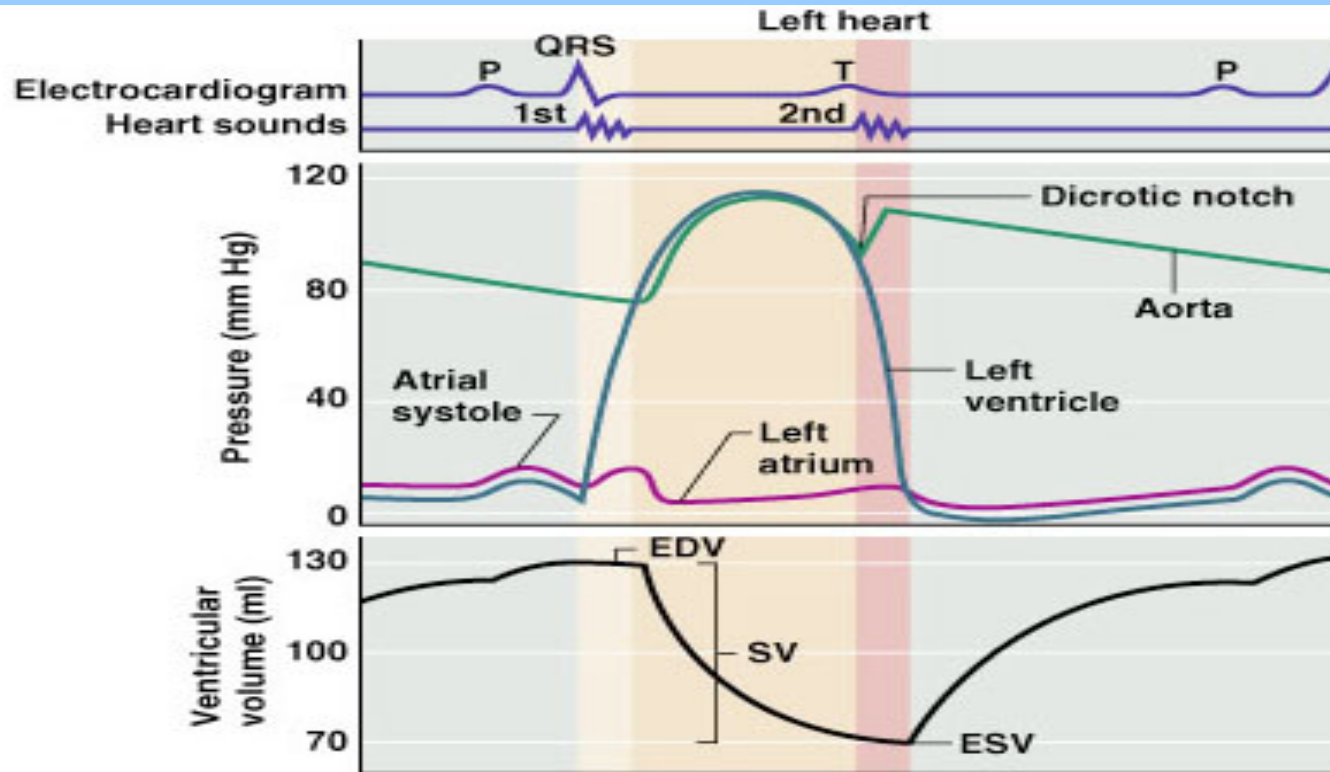
- Ventricular filling – mid-to-late diastole
 - Heart blood pressure is low as blood enters atria and flows into ventricles
 - AV valves are open, then atrial systole occurs

Phases of the Cardiac Cycle

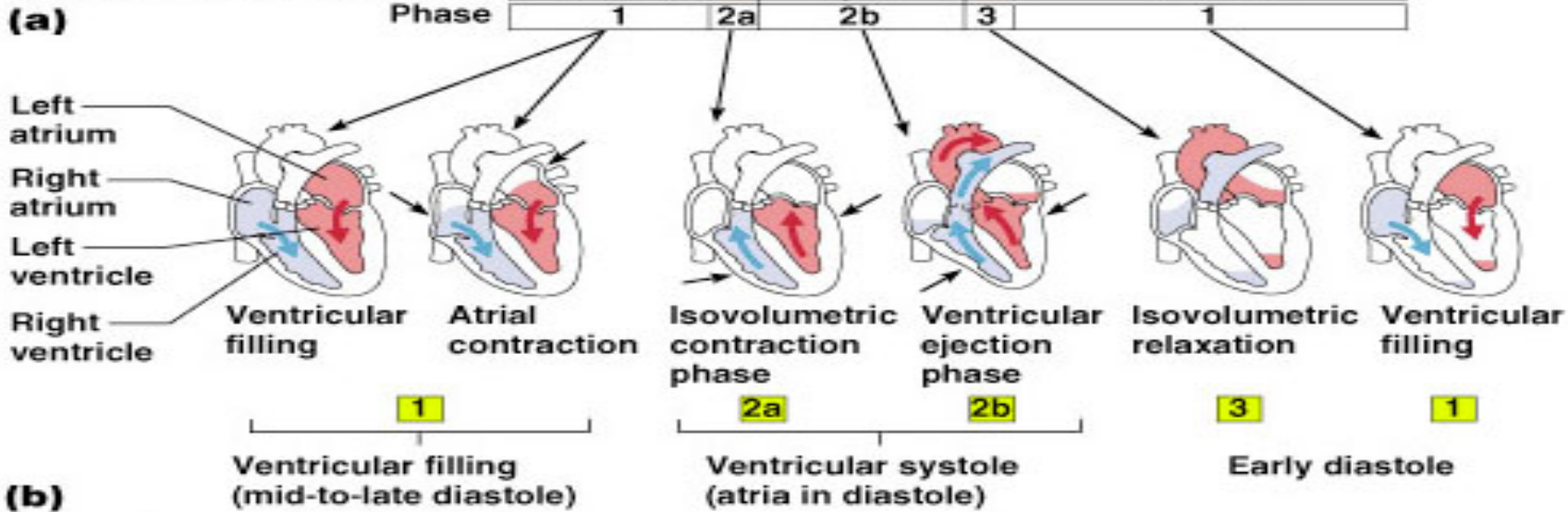
- Ventricular systole
 - Atria relax
 - Rising ventricular pressure results in closing of AV valves
 - Isovolumetric contraction phase
 - Ventricular ejection phase opens semilunar valves

Phases of the Cardiac Cycle

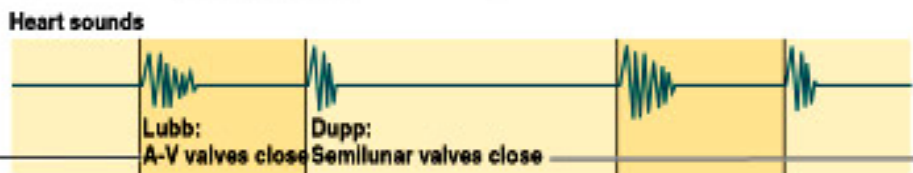
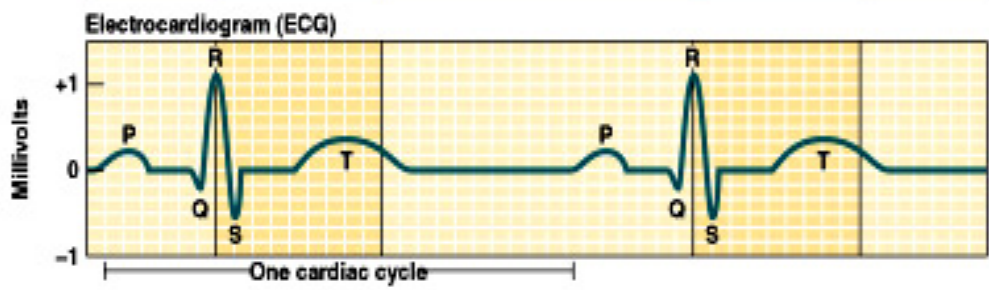
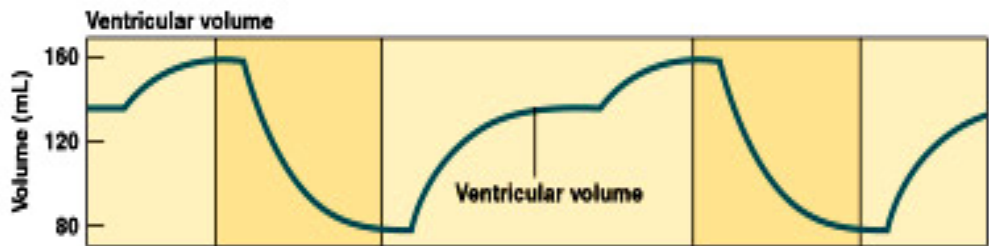
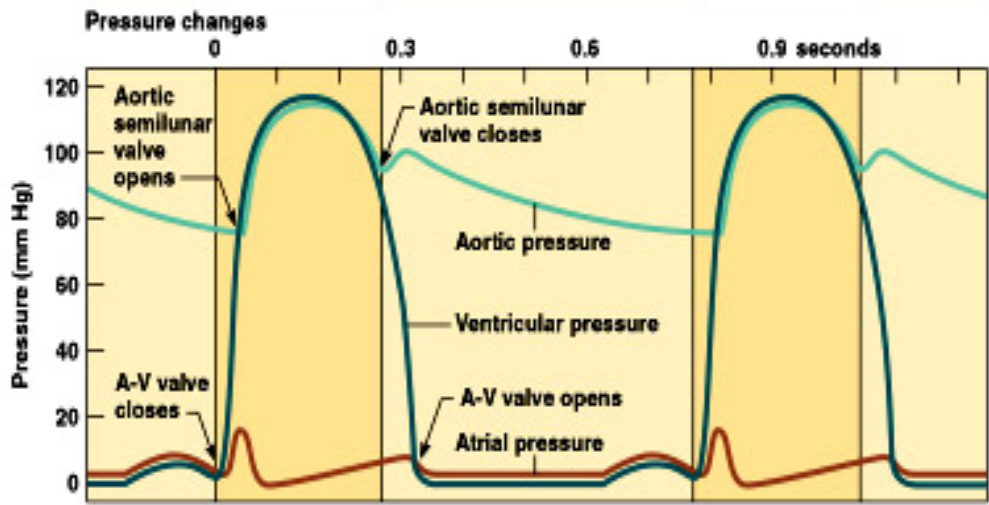
- Isovolumetric relaxation – early diastole
 - Ventricles relax
 - Backflow of blood in aorta and pulmonary trunk closes semilunar valves
- Dicrotic notch – brief rise in aortic pressure caused by backflow of blood rebounding off semilunar valves



Atrioventricular valves	Open	Closed	Closed	Open
Aortic and pulmonary valves	Closed	Open	Closed	Closed
Phase	1	2a	2b	3



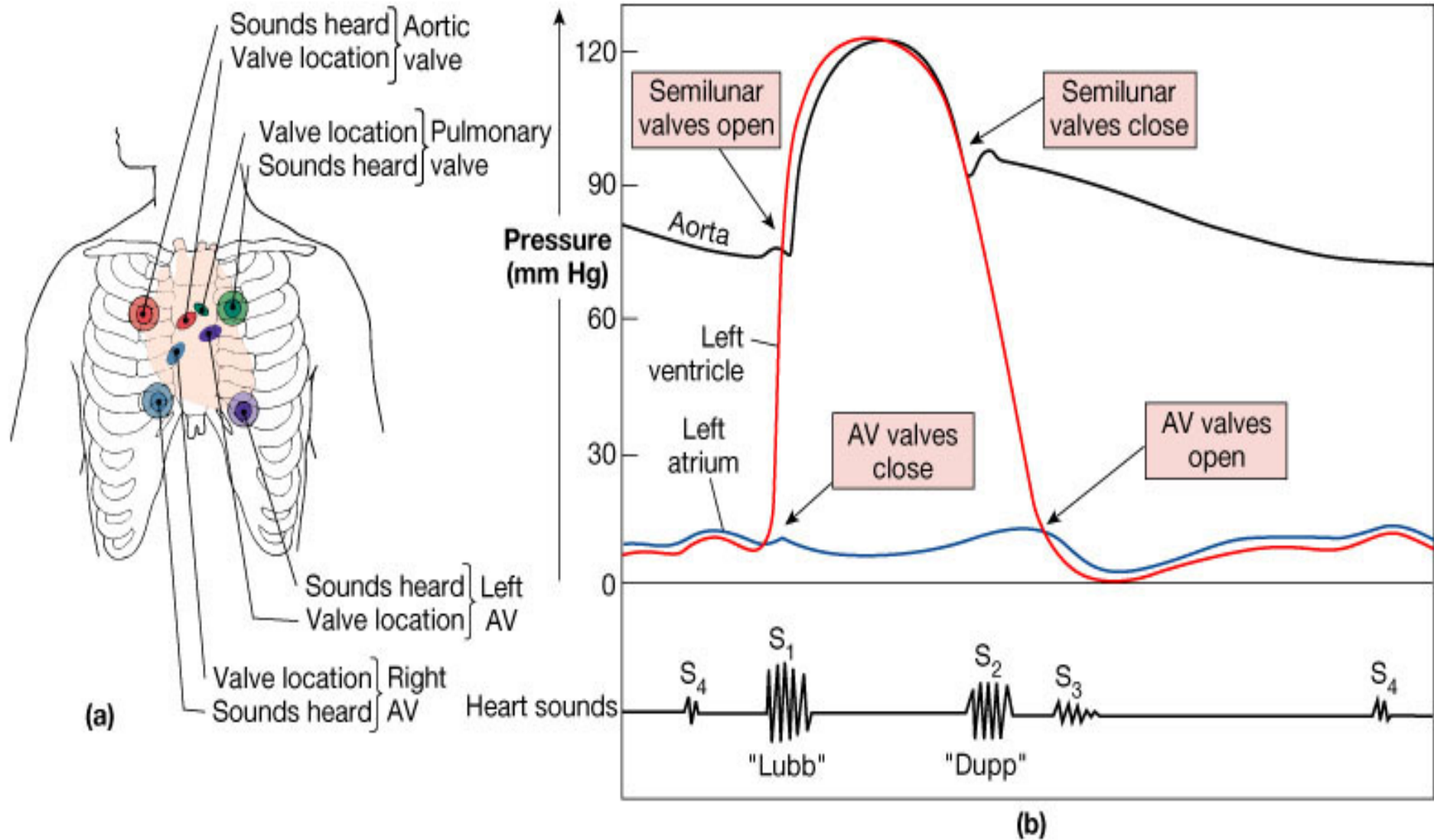
Atrial systole	Atrial diastole		Atrial systole	Atrial diastole	
Ventricular diastole	Ventricular systole	Ventricular diastole		Ventricular systole	Ventricular diastole



Changes during Cardiac cycle

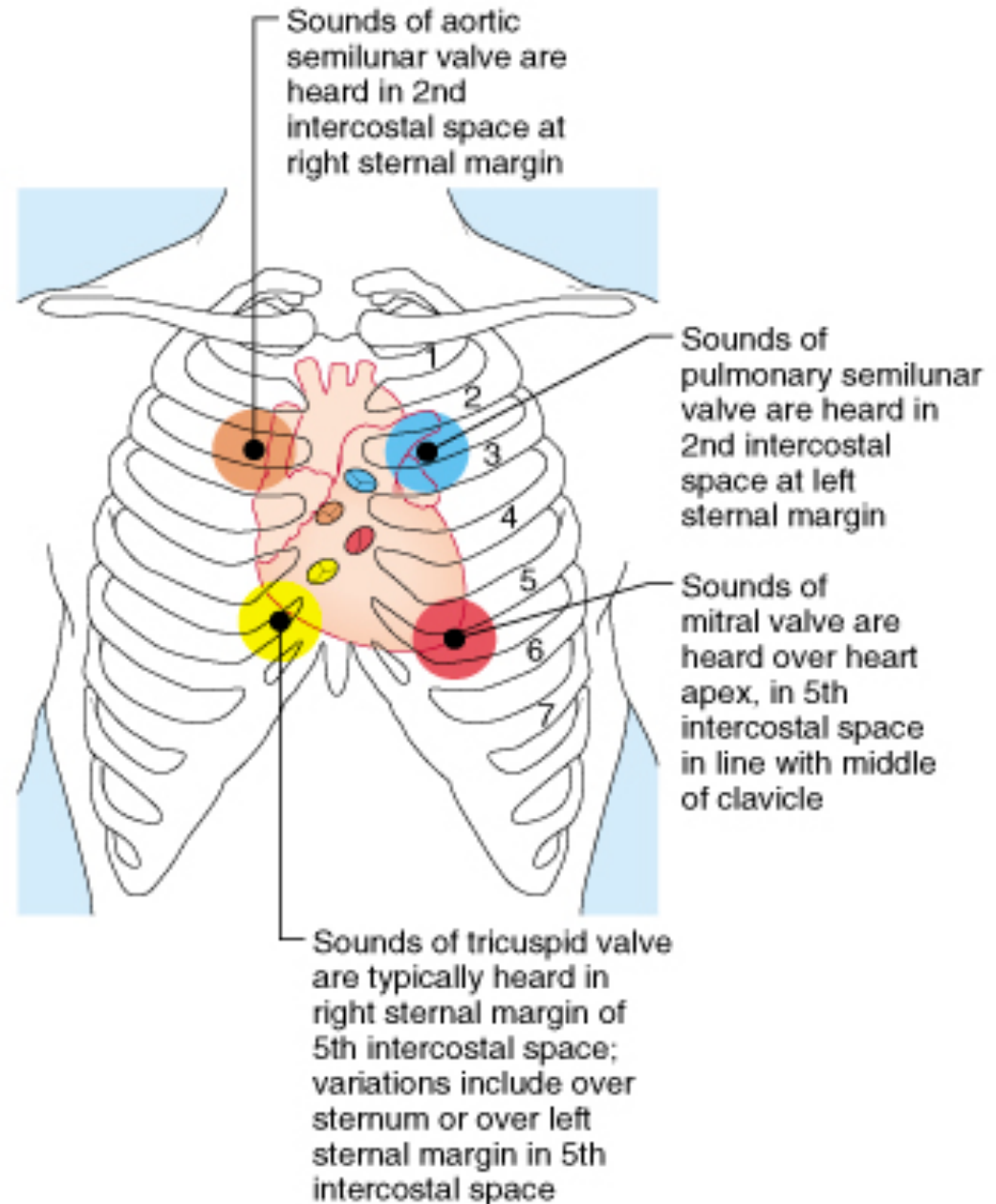
- Volume changes: End-diastolic volume, End-systolic volume, Stroke volume and Cardiac output.
- Aortic pressure: Diastolic pressure ~80 mmHg, Systolic pressure ~ 120 mmHg, most of systole ventricular pressure higher than aortic
- Ventricular pressure: Diastolic ~ 0, systolic Lt. ~120 Rt. ~ 25 mmHg.
- Atrial pressure: A wave =atrial systole, C wave= ventricular contraction (AV closure), V wave= ventricular diastole (Av opening)
- Heart sounds: S_1 = turbulence of blood around a closed AV valves, S_2 = turbulence of blood around a closed semilunar valves.

Heart Sounds



Heart Sounds

- Heart sounds (lub-dup) are associated with closing of heart valves



Heart sounds

- Auscultation – listening to heart sound via stethoscope
- Four heart sounds
 - S_1 – “lubb” caused by the closing of the AV valves
 - S_2 – “dupp” caused by the closing of the semilunar valves
 - S_3 – a faint sound associated with blood flowing into the ventricles
 - S_4 – another faint sound associated with atrial contraction

Cardiac Output (CO) and Reserve

- CO is the amount of blood pumped by each ventricle in one minute
- CO is the product of heart rate (HR) and stroke volume (SV)
- HR is the number of heart beats per minute
- SV is the amount of blood pumped out by a ventricle with each beat
- Cardiac reserve is the difference between resting and maximal CO

Cardiac Output: Example

- $CO \text{ (ml/min)} = HR \text{ (75 beats/min)} \times SV \text{ (70 ml/beat)}$
- $CO = 5250 \text{ ml/min (5.25 L/min)}$

Regulation of Stroke Volume

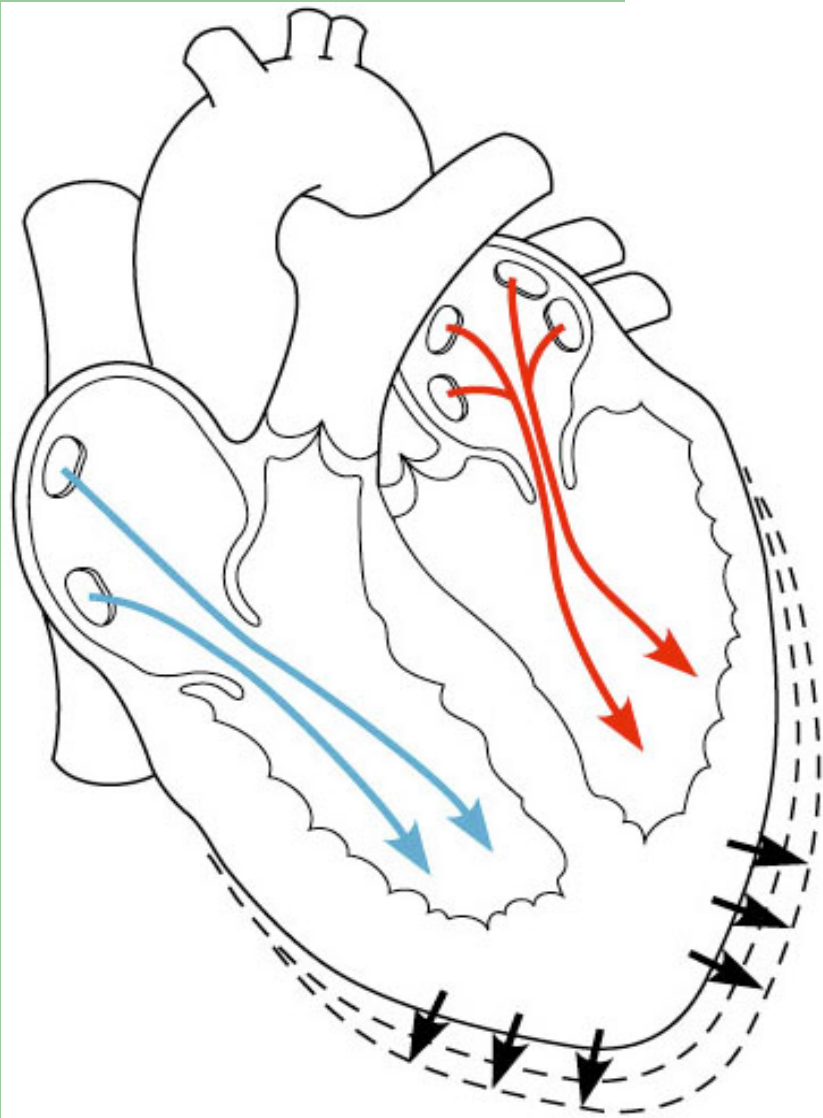
- $SV = \text{end diastolic volume (EDV)} - \text{end systolic volume (ESV)}$
- EDV = amount of blood collected in a ventricle during diastole
- ESV = amount of blood remaining in a ventricle after contraction

Factors Affecting Stroke Volume

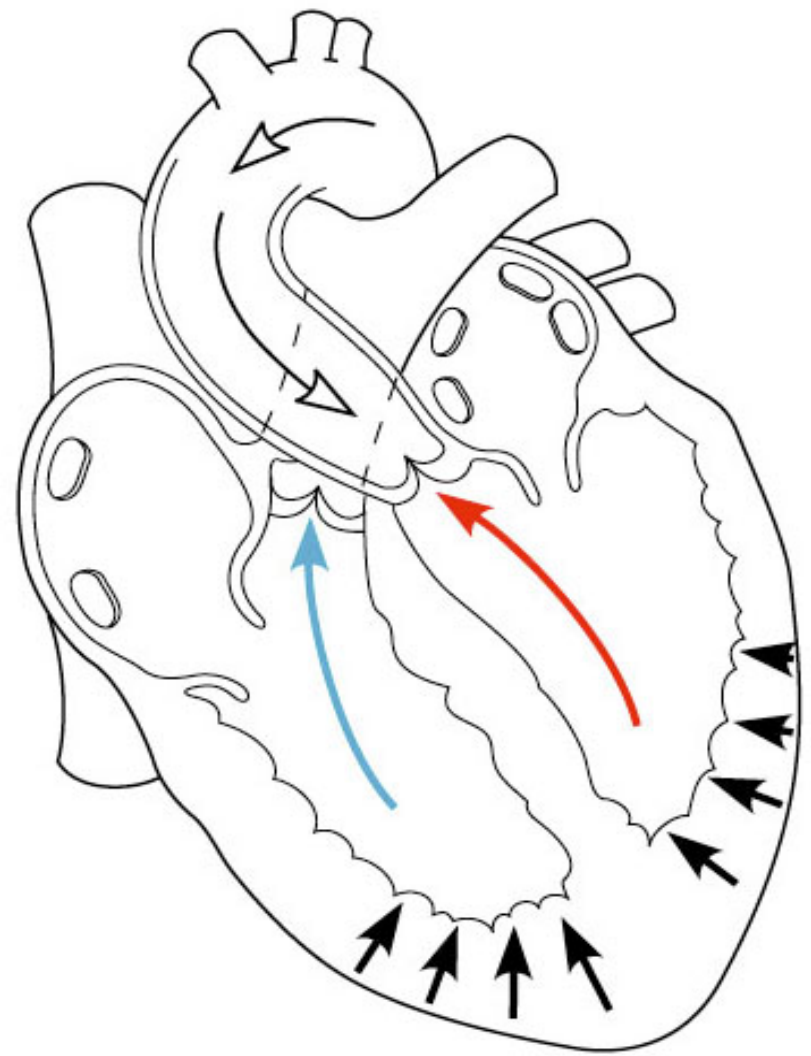
- Preload – amount ventricles are stretched by contained blood
- Contractility – cardiac cell contractile force due to factors other than EDV
- Afterload – back pressure exerted by blood in the large arteries leaving the heart

Frank-Starling Law of the Heart

- Preload, or degree of stretch, of cardiac muscle cells before they contract is the critical factor controlling stroke volume
- Slow heartbeat and exercise increase venous return to the heart, increasing SV
- Blood loss and extremely rapid heartbeat decrease SV

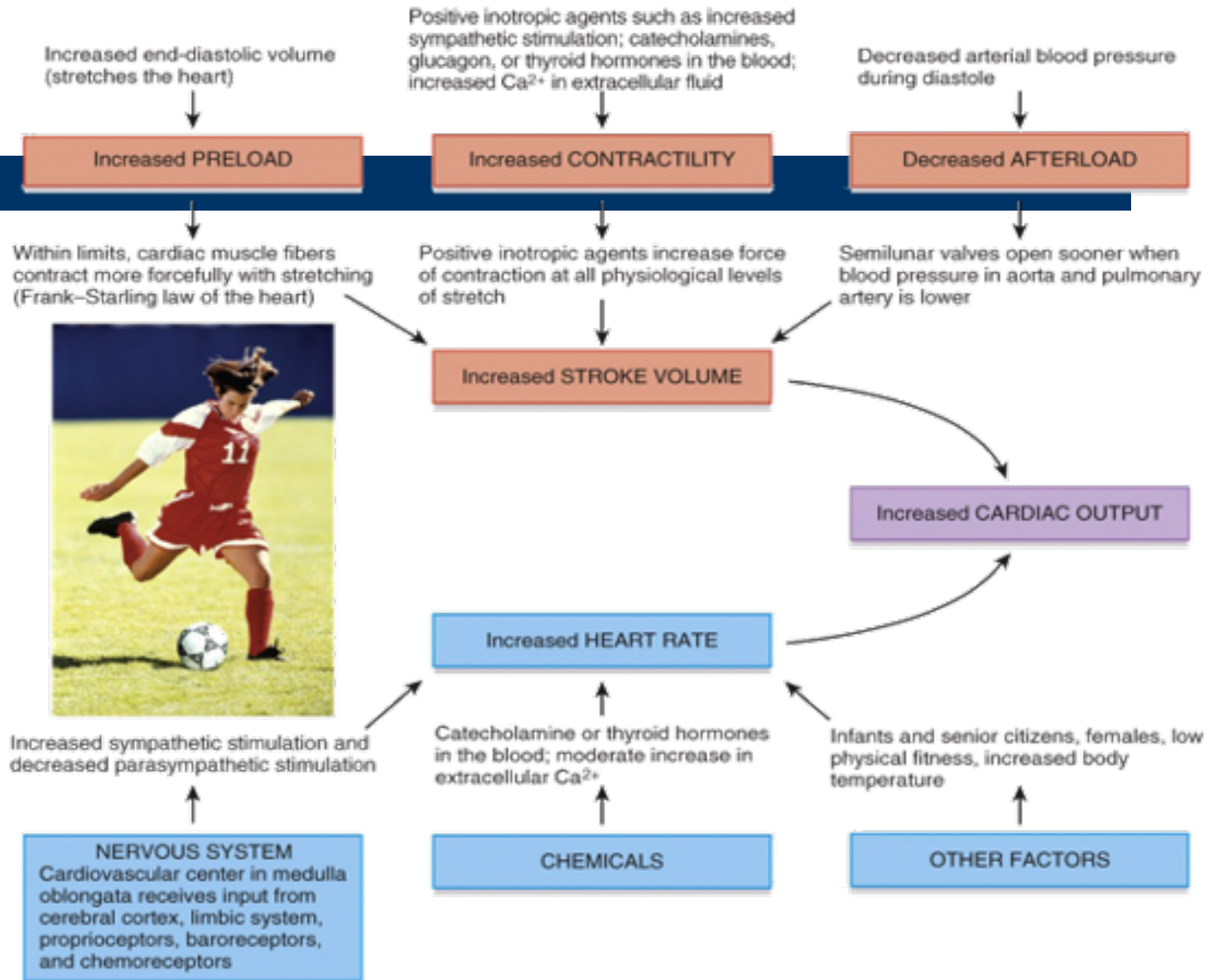


(a) Preload

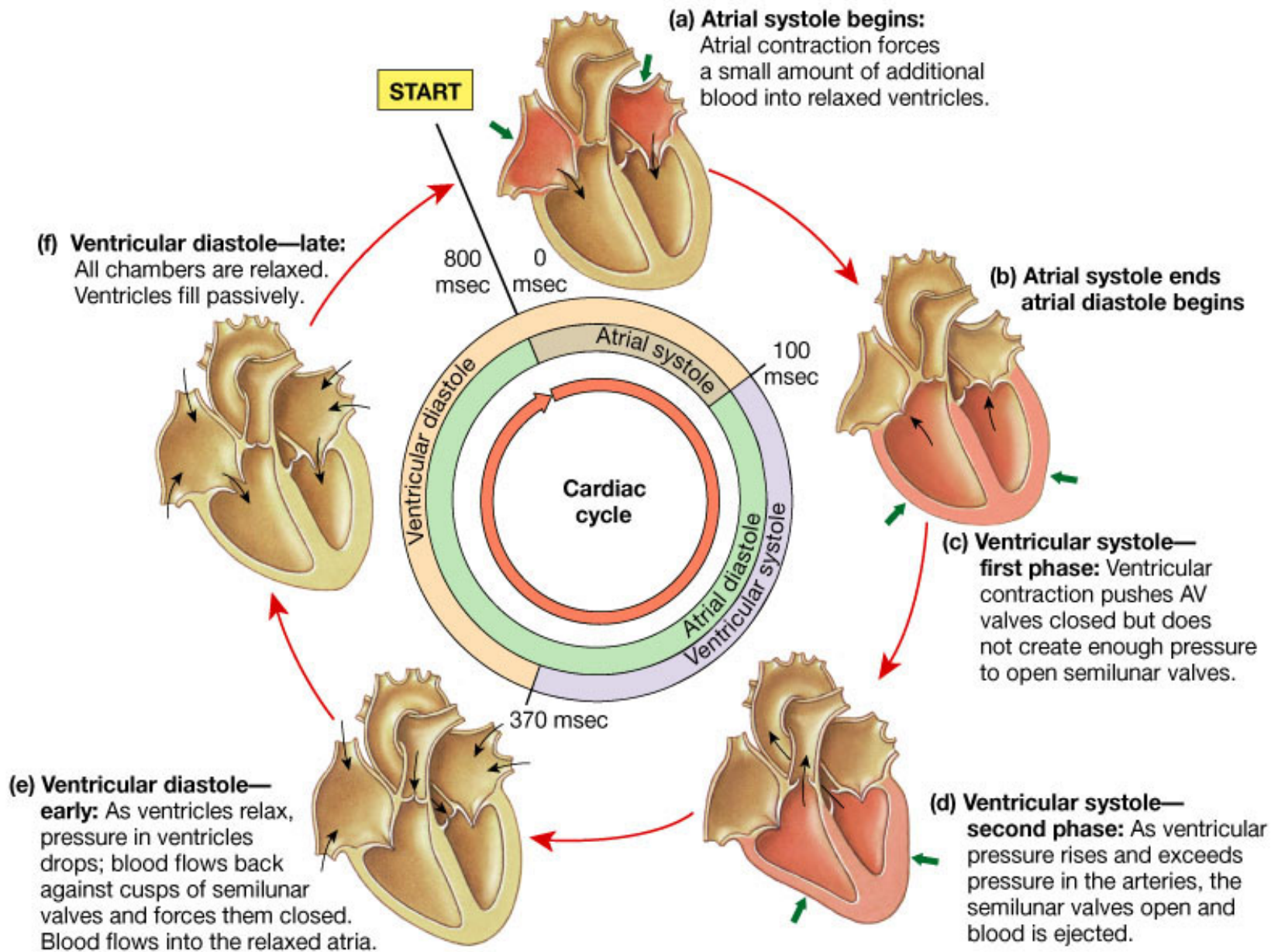


(b) Afterload

Cardiac Output



Phases of the Cardiac Cycle



Extrinsic Factors Influencing Stroke Volume

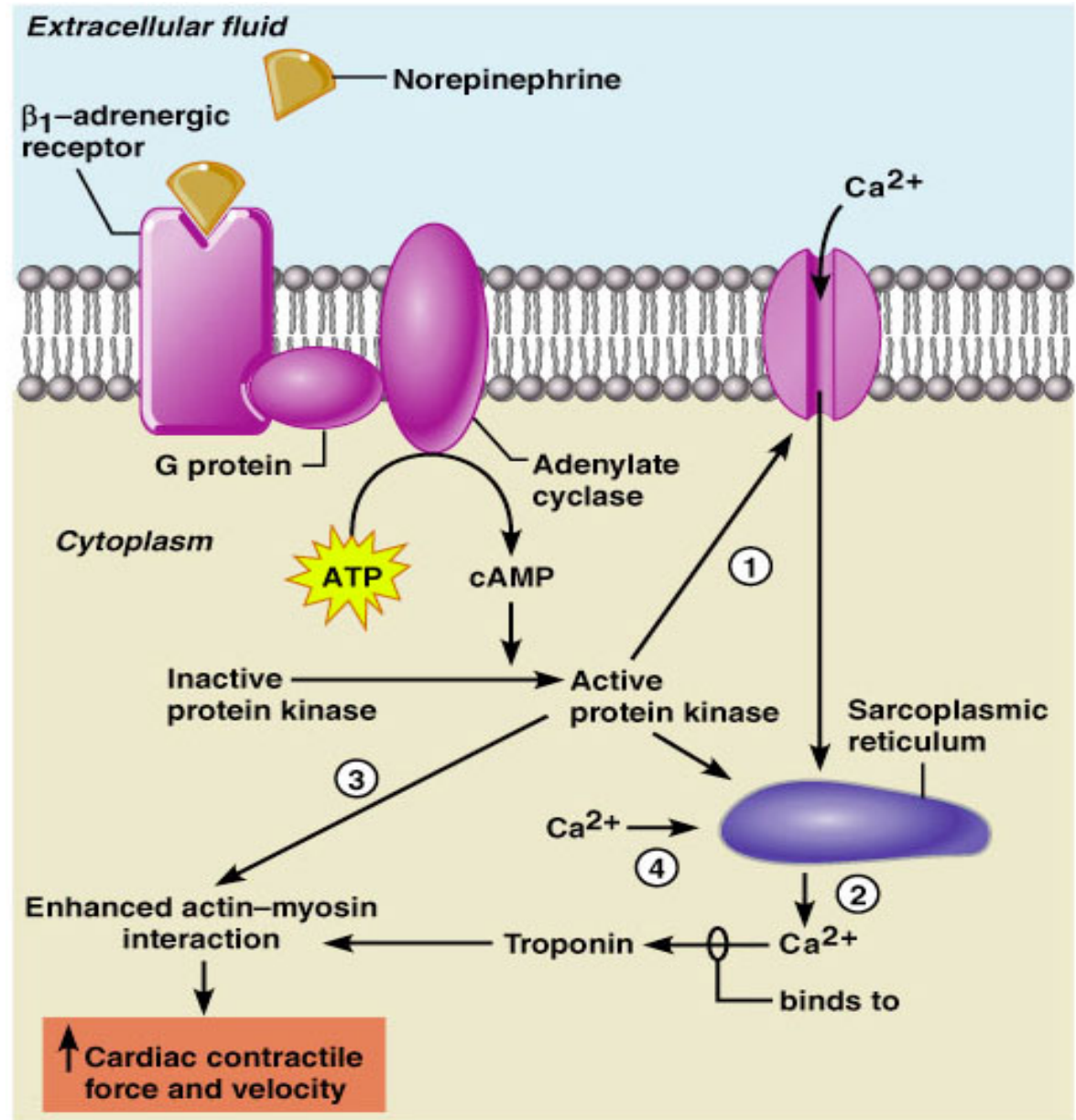
- Contractility is the increase in contractile strength, independent of stretch and EDV
- Increase in contractility comes from:
 - Increased sympathetic stimuli
 - Certain hormones
 - Ca^{2+} and some drugs

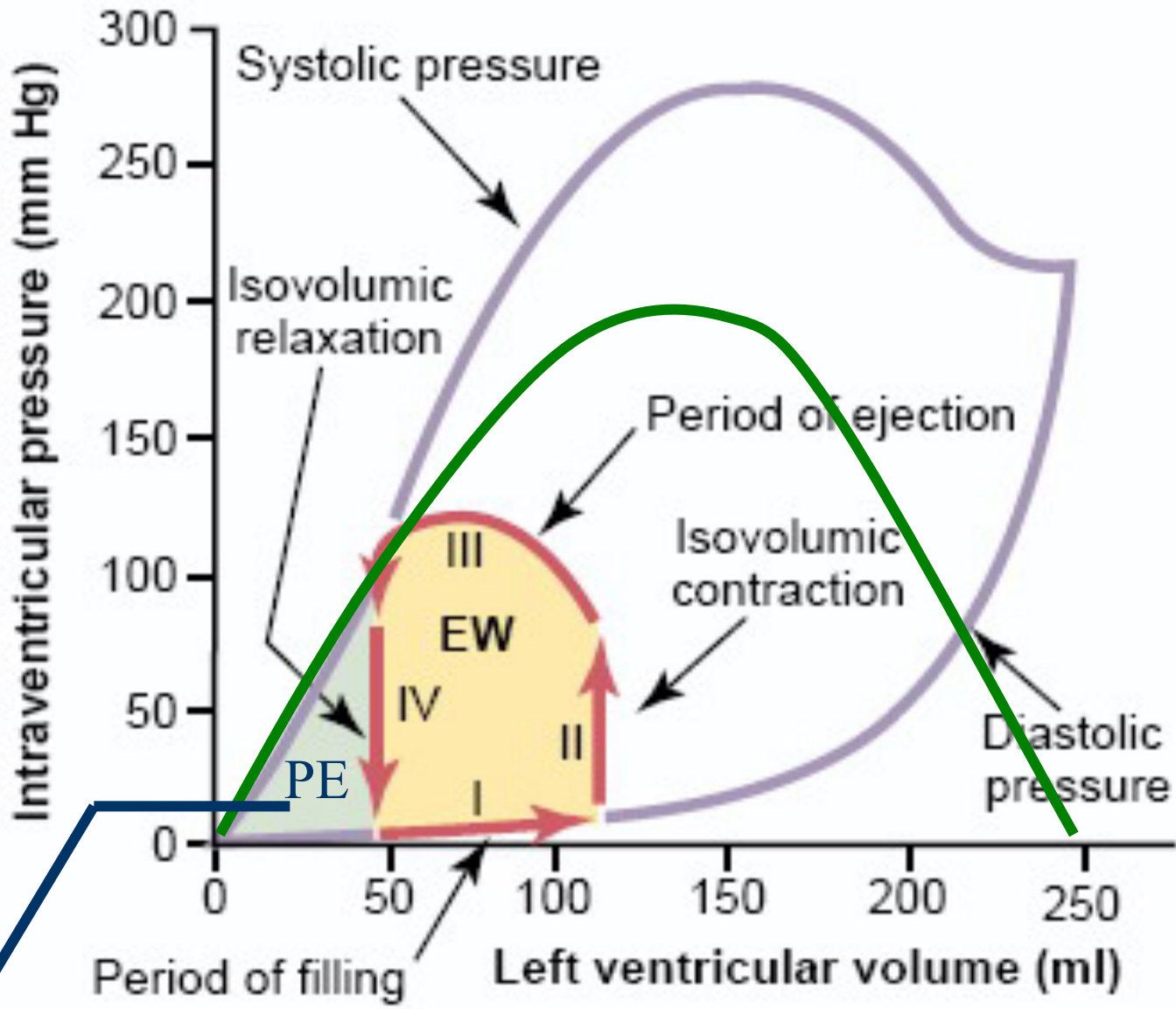
Extrinsic Factors Influencing Stroke Volume

- Agents/factors that decrease contractility include:
 - Acidosis
 - Increased extracellular K^+
 - Calcium channel blockers

Contractility and Norepinephrine

- Sympathetic stimulation releases norepinephrine and initiates a cyclic AMP second-messenger system



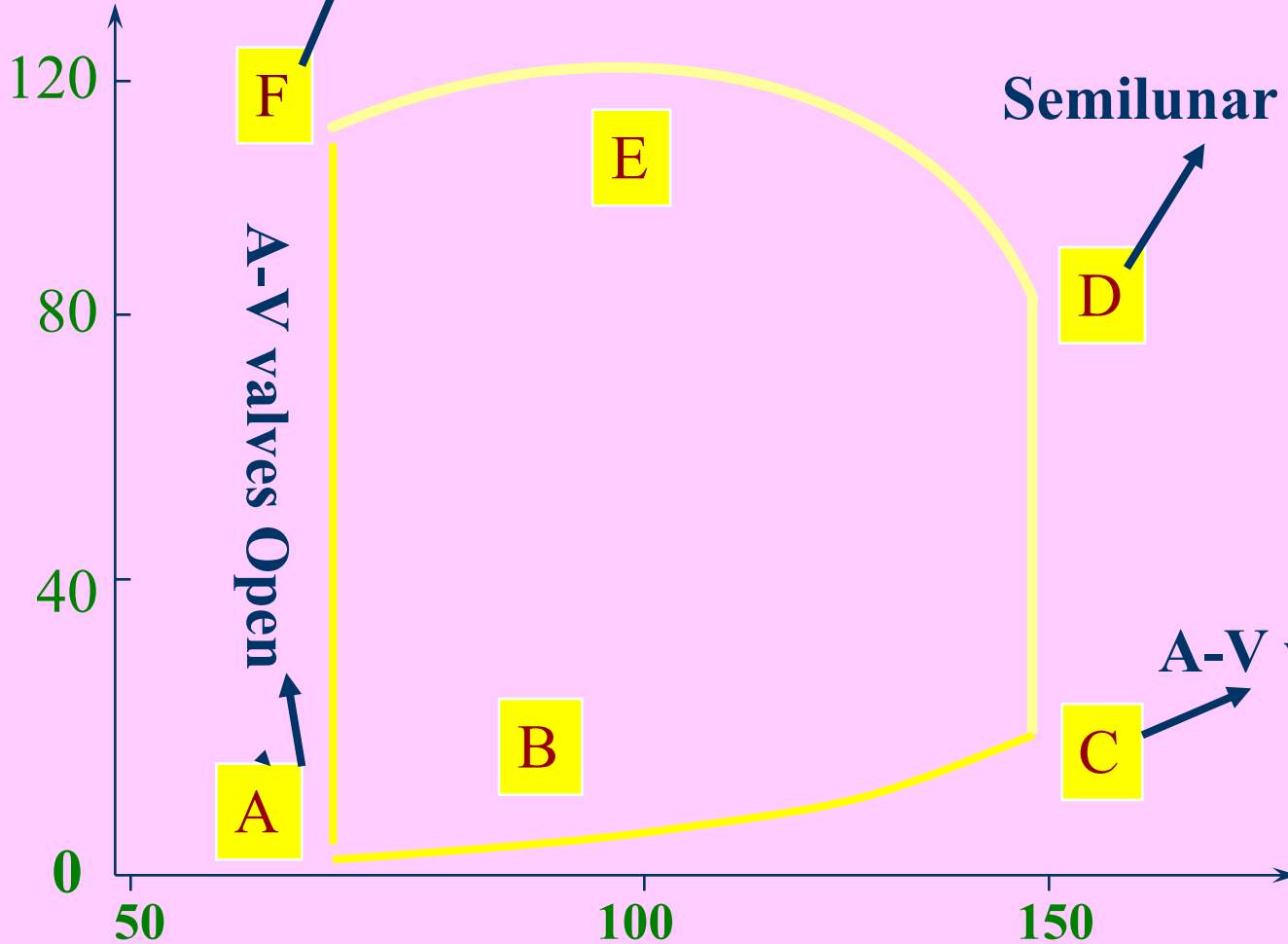


30 Potential Energy (PE)

LEFT VENTRICULAR PRESSURE/VOLUME P/V LOOP

LEFT VENTRICULAR PRESSURE (mmHg)

Semilunar Valves Close



LEFT VENTRICULAR VOLUME (ml)

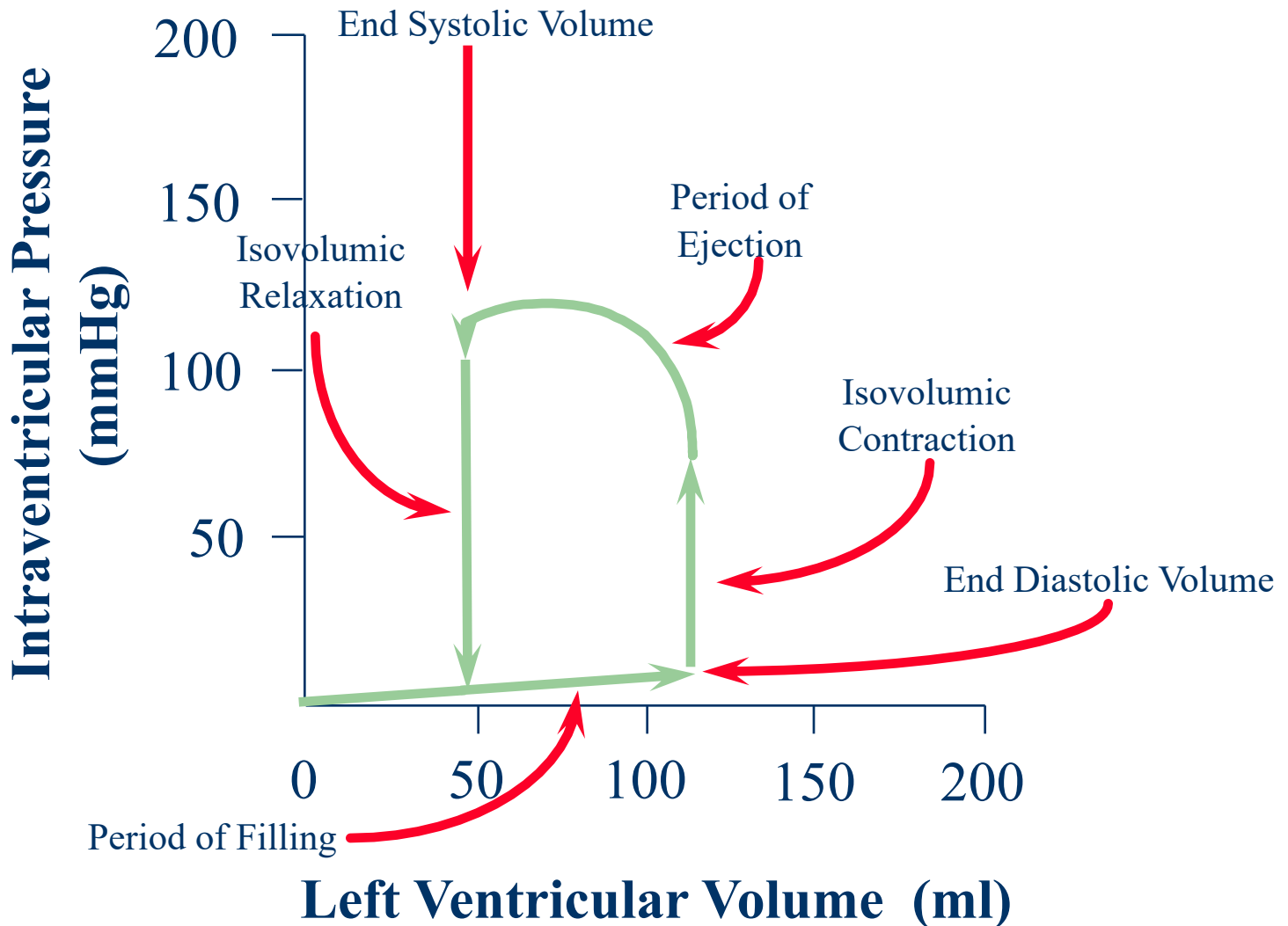
Valvular Function

- To prevent back-flow.
- Chordae tendineae are attached to A-V valves.
- Papillary muscle, attached to chordae tendineae, contract during systole and help prevent back-flow.
- Because of smaller opening, velocity through aortic and pulmonary valves exceed that through the A-V valves.

Valvular Function (cont'd)

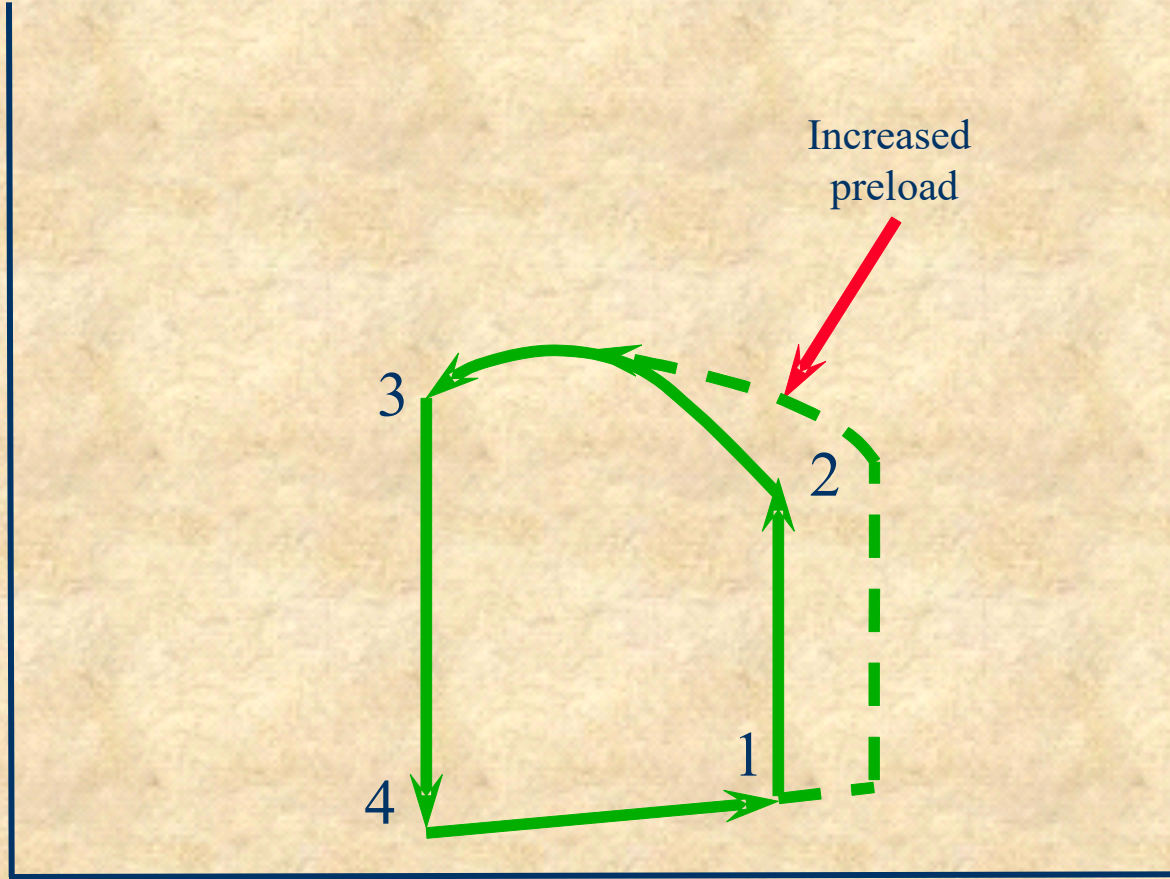
- ☀ Most work is external work or pressure-volume work.
- ☀ A small amount of work is required to impart kinetic energy to the heart ($1/2 mV^2$).
- ☀ What is stroke-volume in previous figure?
- ☀ External work is area of Pressure-Volume curve.
- ☀ Work output is affected by “preload” (end-diastolic pressure) and “afterload” (aortic pressure).

Work Output of the Heart



A

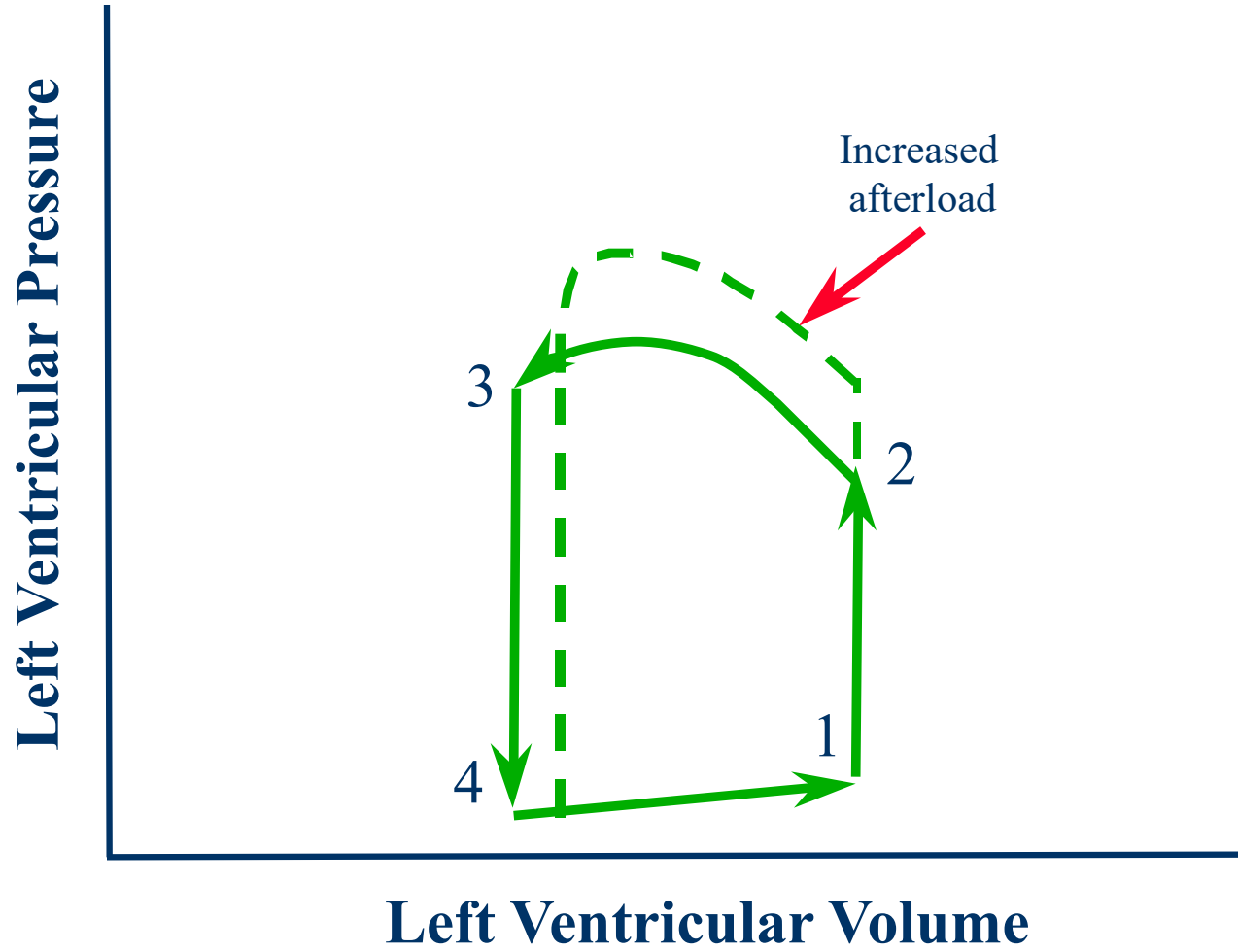
Left Ventricular Pressure



**Increased
preload**

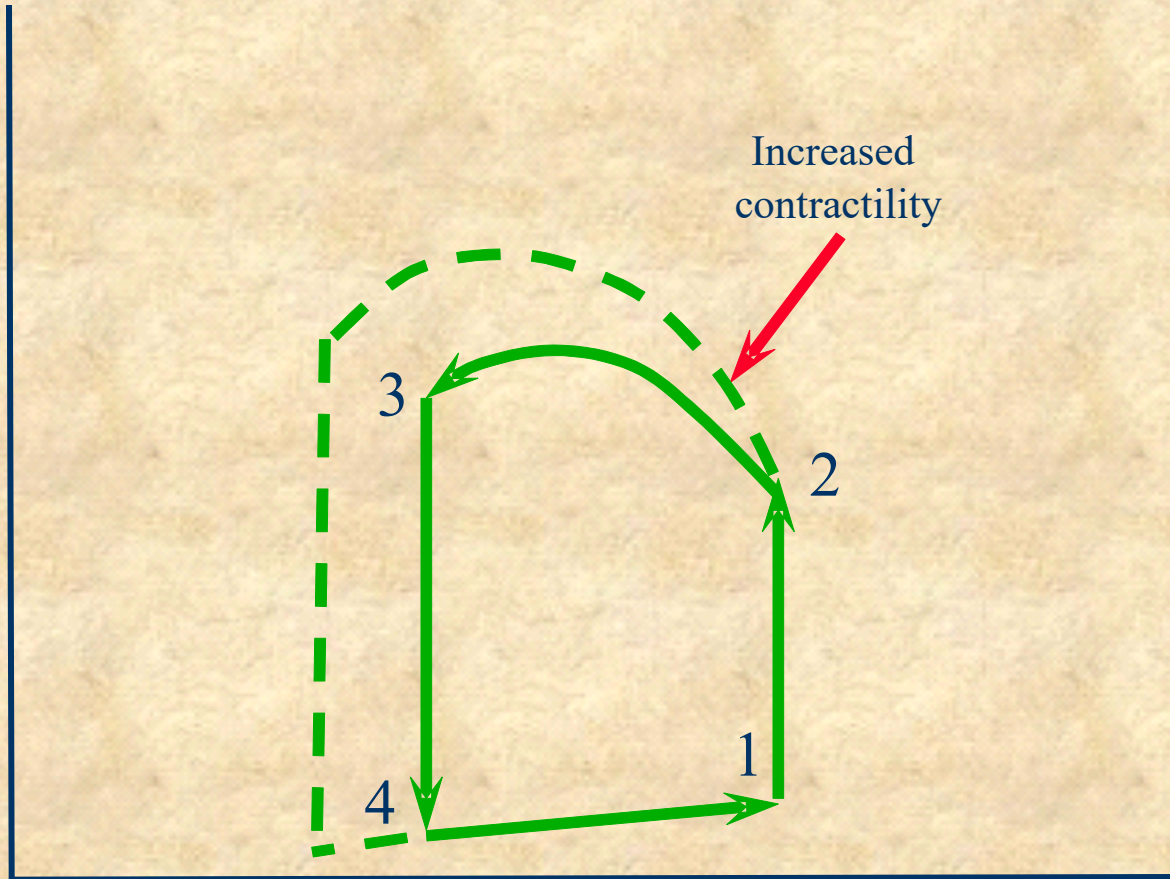
Left Ventricular Volume

B



C

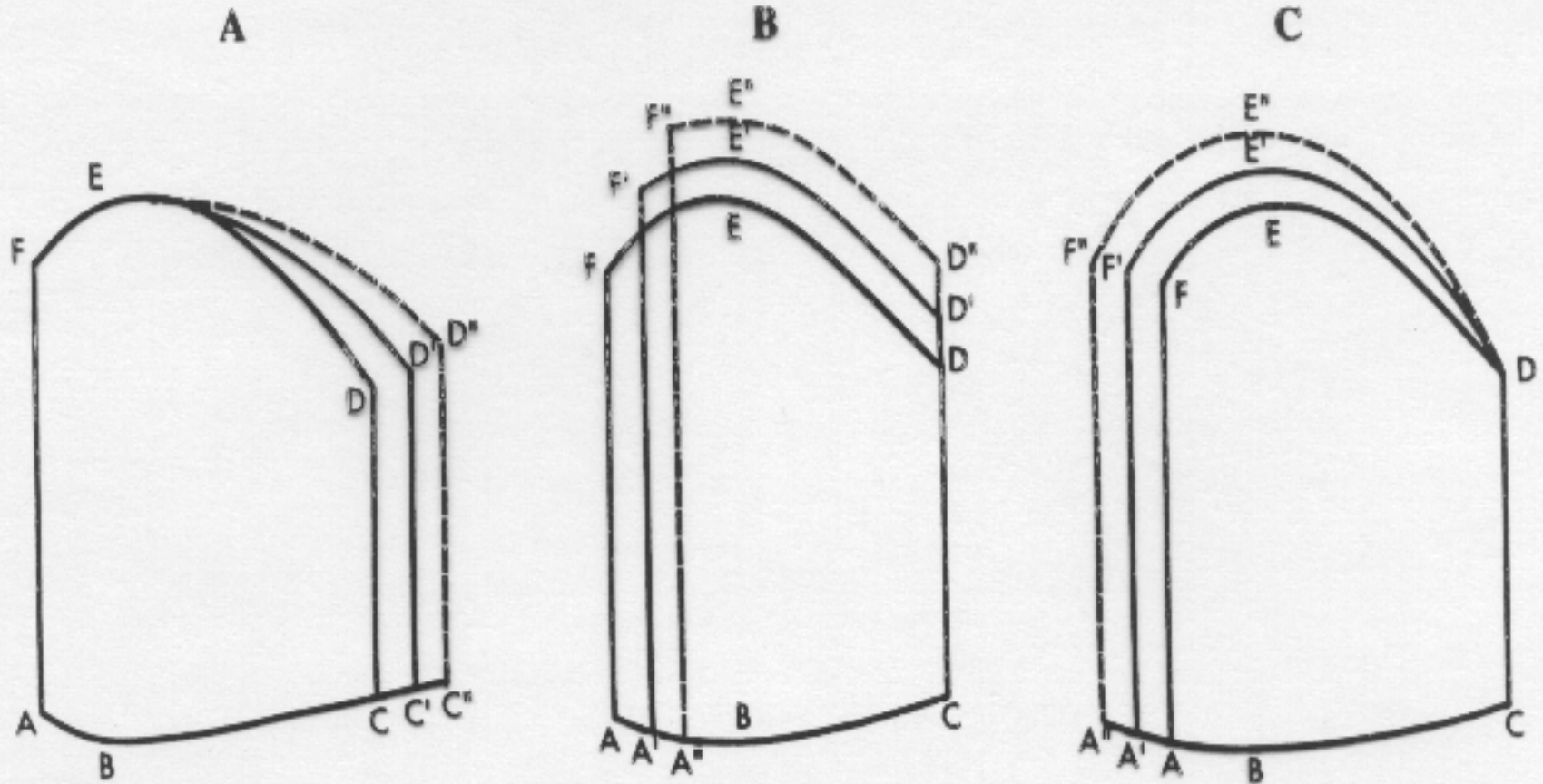
Left Ventricular Pressure



Increased contractility

Left Ventricular Volume

PRESSURE/VOLUME RELATIONSHIPS UNDER DIFFERENT CONDITIONS



PRELOAD

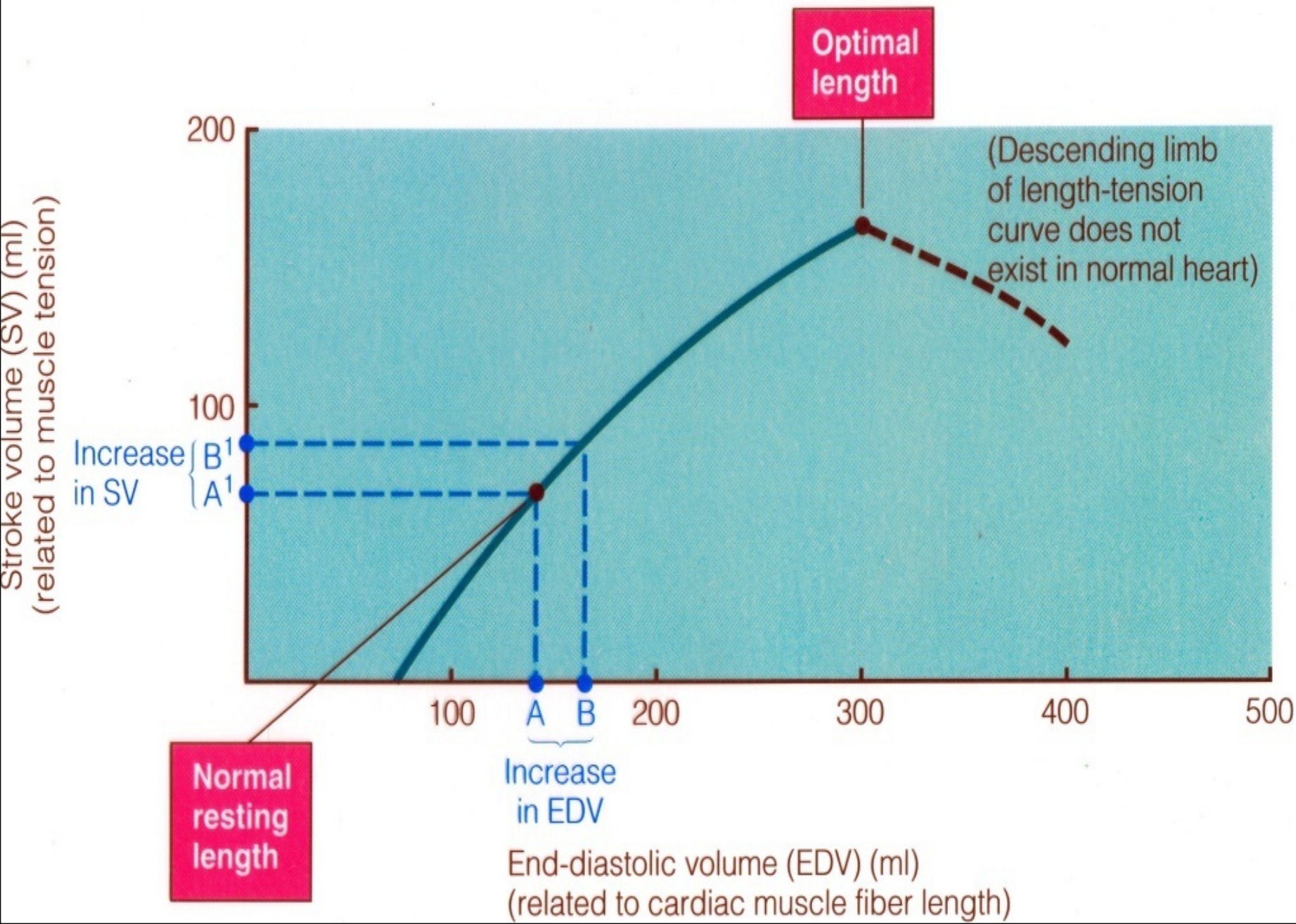


AFTERLOAD



CONTRACTILITY

Intrinsic Control of Stroke Volume (Frank-Starling Curve)



Regulation of Heart Rate

- Positive chronotropic factors increase heart rate
- Negative chronotropic factors decrease heart rate

Regulation of Heart Rate: Autonomic Nervous System

- Sympathetic nervous system (SNS) stimulation is activated by stress, anxiety, excitement, or exercise
- Parasympathetic nervous system (PNS) stimulation is mediated by acetylcholine and opposes the SNS
- PNS dominates the autonomic stimulation, slowing heart rate and causing vagal tone

Atrial (Bainbridge) Reflex

- Atrial (Bainbridge) reflex – a sympathetic reflex initiated by increased blood in the atria
 - Causes stimulation of the SA node
 - Stimulates baroreceptors in the atria, causing increased SNS stimulation

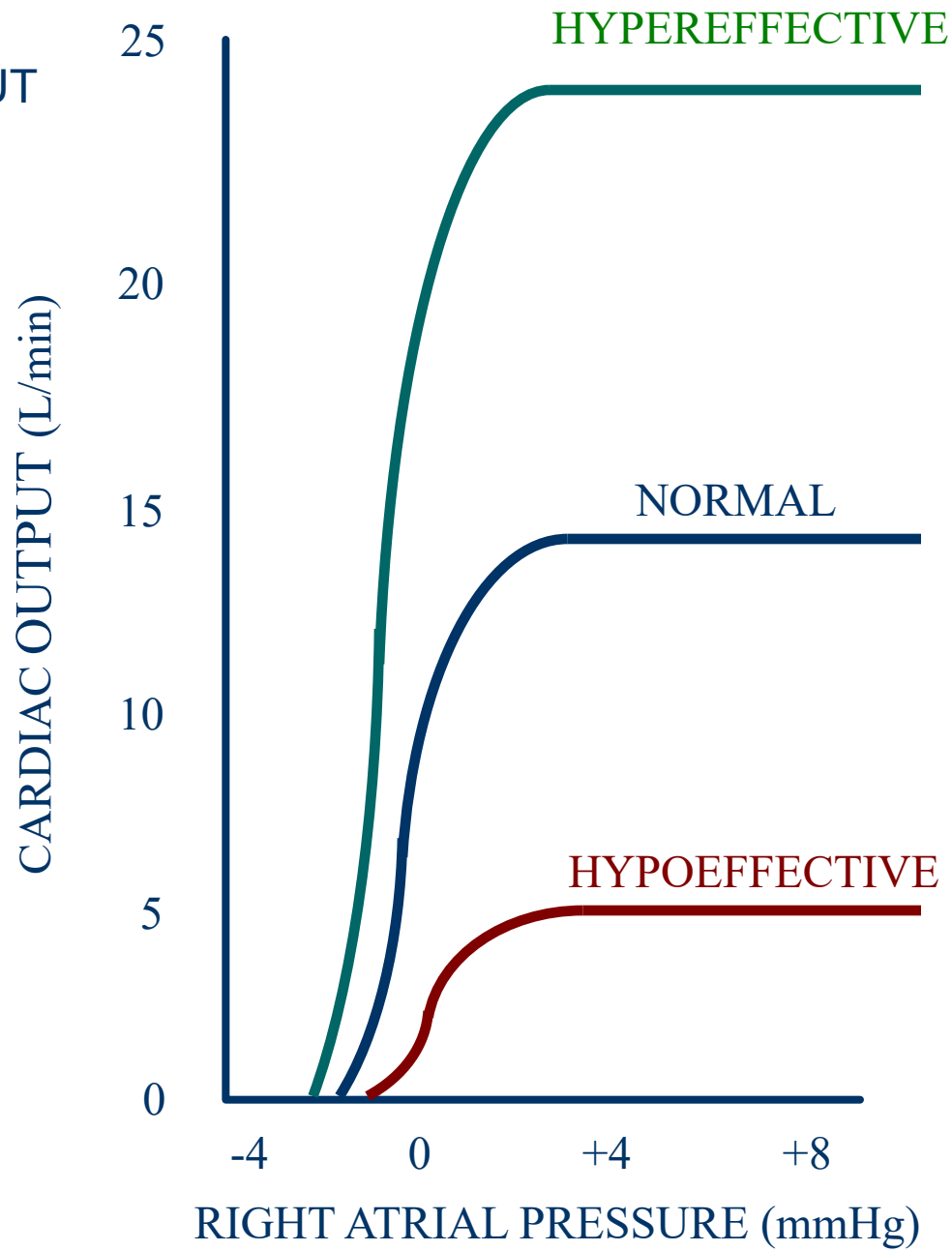
Chemical Regulation of the Heart

- The hormones epinephrine and thyroxine increase heart rate
- Intra- and extracellular ion concentrations must be maintained for normal heart function

Important Concepts About Cardiac Output (CO) Control

- Cardiac Output is the sum of all tissue flows and is affected by their regulation (CO = 5L/min, cardiac index = 3L/min/m² (surface area in m²)).
- CO is proportional to tissue O₂ use.
- CO is proportional to 1/TPR when AP is constant.
- **$CO = (MAP - RAP) / TPR$**

CARDIAC OUTPUT CURVES



Ventricular Pressure and Volume Curves (cont'd)

- During the latter part of the ejection phase how can blood still leave the ventricle if pressure is higher in the aorta? Momentum of blood flow
- Total energy of blood = $P + mV^2/2$
= pressure + kinetic energy
- Total energy of blood leaving ventricle is greater than in aorta.

Ejection Fraction

- 👉 End diastolic volume = 125 ml
- 👉 End systolic volume = 55 ml
- 👉 Ejection volume (stroke volume) = 70 ml
- 👉 Ejection fraction = $70\text{ml}/125\text{ml} = 56\%$
(normally 60%)
- 👉 If heart rate (HR) is 70 beats/minute, what is cardiac output?
- 👉 Cardiac output = HR * stroke volume
= 70/min. * 70 ml
= 4900ml/min.

Ejection Fraction (cont'd)

- If HR = 100, end diastolic volume = 180 ml, end systolic vol. = 20 ml, what is cardiac output?
- C.O. = 100/min. * 160 ml = 16,000 ml/min.
- Ejection fraction = $160/180\% \approx 90\%$

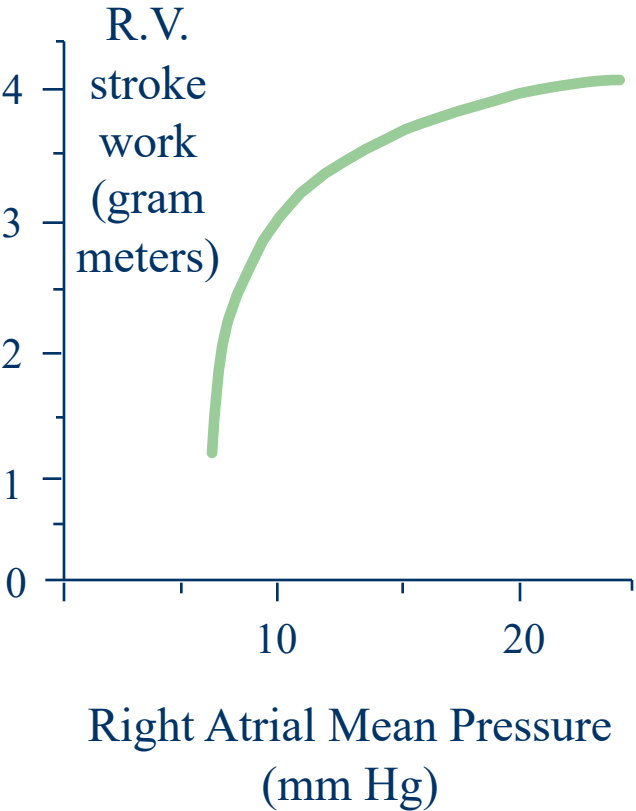
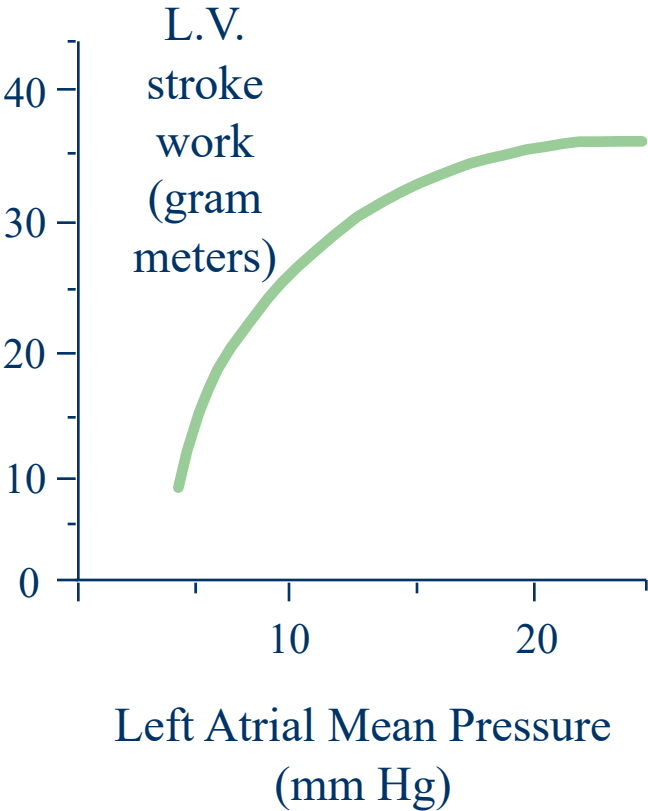
Aortic Pressure Curve

- ❖ Aortic pressure starts increasing during systole after the aortic valve opens.
- ❖ Aortic pressure decreases toward the end of the ejection phase.
- ❖ After the aortic valve closes, an *incisura* occurs because of sudden cessation of back-flow toward left ventricle.
- ❖ Aortic pressure decreases slowly during diastole because of the elasticity of the aorta.

Frank-Starling Mechanism

- Within physiological limits the heart pumps all the blood that comes to it without excessive damming in the veins.
- Extra stretch on cardiac myocytes makes actin and myosin filaments interdigitate to a more optimal degree for force generation.

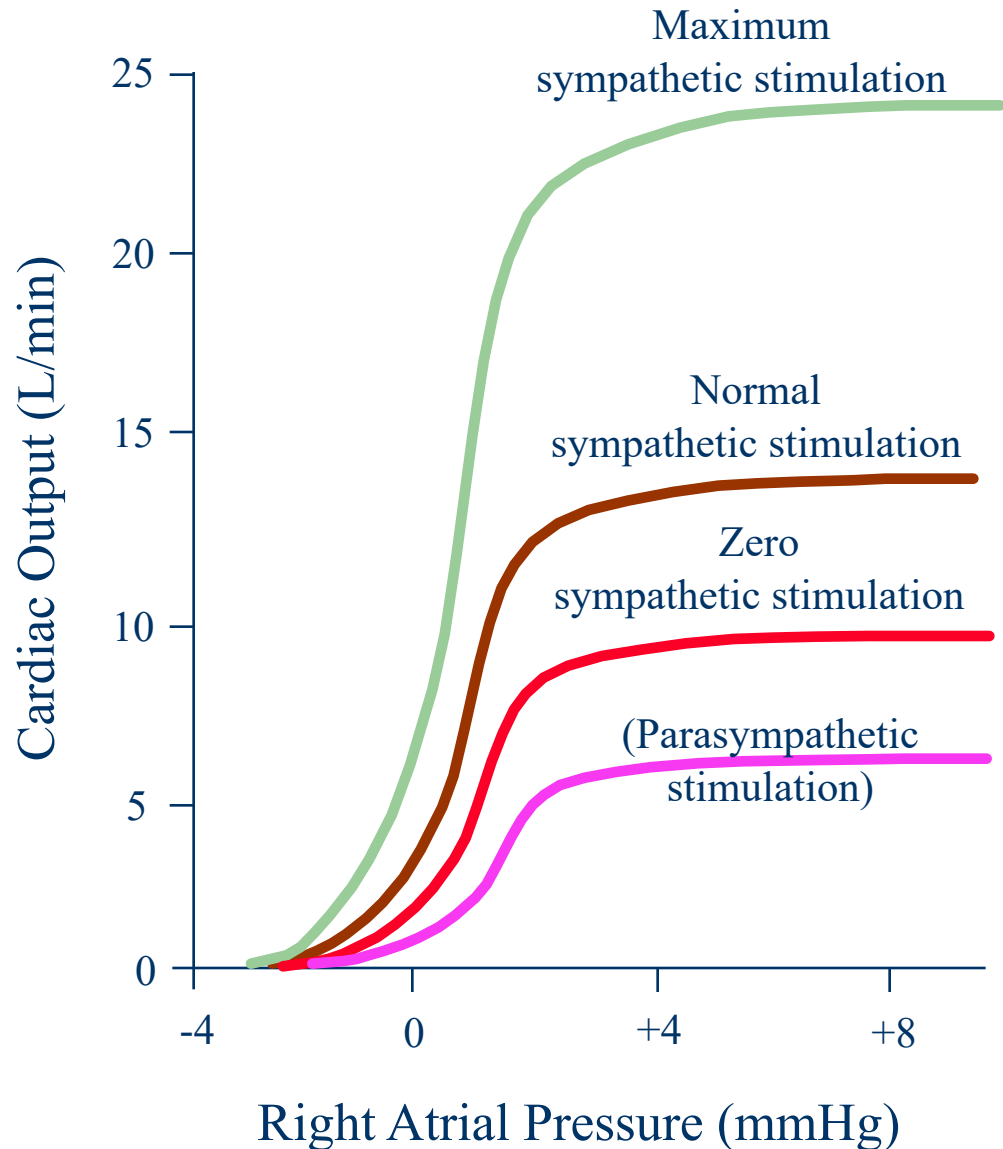
Ventricular Stroke Work Output



Autonomic Effects on Heart

- ❑ Sympathetic stimulation causes increased HR and increased contractility with HR = 180-200 and C.O. = 15-20 L/min.
- ❑ Parasympathetic stimulation decreases HR markedly and decreases cardiac contractility slightly. Vagal fibers go mainly to atria.
- ❑ Fast heart rate (tachycardia) can decrease C.O. because there is not enough time for heart to fill during diastole.

Effect of Sympathetic and Parasympathetic Stimulation on Cardiac Output



Cardiac Contractility

- Best is to measure the C.O. curve, but this is nearly impossible in humans.
- dP/dt is not an accurate measure because this increases with increasing preload and afterload.
- $(dP/dt)/P_{\text{ventricle}}$ is better. $P_{\text{ventricle}}$ is instantaneous ventricular pressure.
- Excess K^+ decreases contractility.
- Excess Ca^{++} causes spastic contraction, and low Ca^{++} causes cardiac dilation.

Thank You

