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

Cardiac Cycle

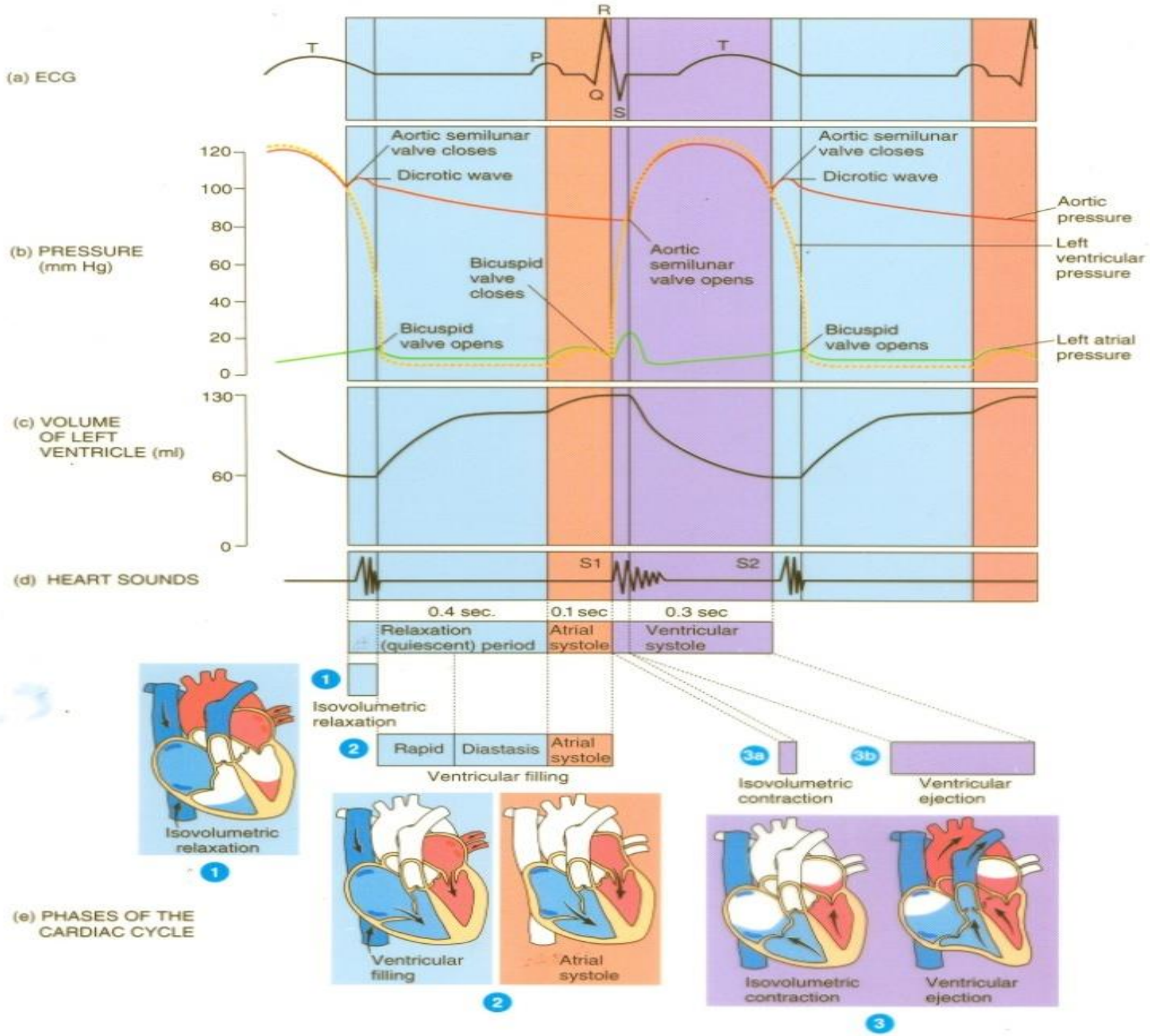
- Cardiac cycle refers to all events associated with blood flow through the heart
  - Systole – contraction of heart ventricles
  - Diastole – relaxation of heartventricles

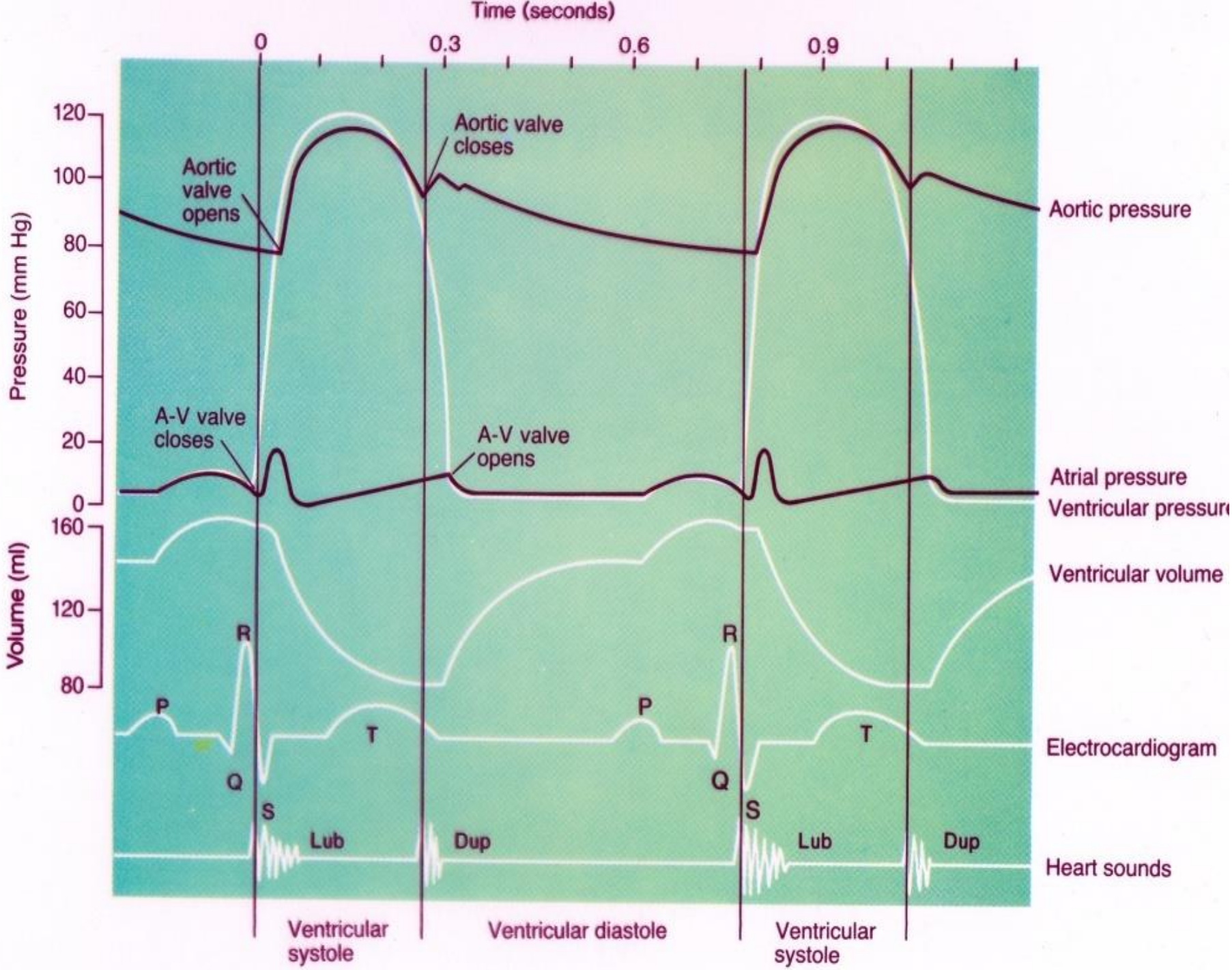
Cardiac Cycle

- 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

HR	Systole	Diastole
60	0.3	0.7
75	0.25	0.55
200	0.19	0.11

Each person has his own optimal maxHR.


(a) ECG
R
T
P
Q
S
T
(b) PRESSURE (mm Hg)
120
100
80
60
40
20
0
Aortic semilunar valve closes
Dicrotic wave
Bicuspid valve closes
Aortic semilunar valve opens
Bicuspid valve opens
Aortic semilunar valve closes
Dicrotic wave
Bicuspid valve opens
Aortic pressure
Left ventricular pressure
Left atrial pressure
(c) VOLUME OF LEFT VENTRICLE (ml)
130
60
0
(d) HEART SOUNDS
S1
S2
0.4 sec.
0.1 sec
0.3 sec
Relaxation (quiescent) period
Atrial systole
Ventricular systole
1
Isovolumetric relaxation
2
Rapid
Diastasis
Atrial systole
Ventricular filling
3a
Isovolumetric contraction
3b
Ventricular ejection
Isovolumetric relaxation
1
Ventricular filling
Atrial systole
2
Isovolumetric contraction
Ventricular ejection
3
(e) PHASES OF THE CARDIAC CYCLE


Time (seconds)
0
0.3
0.6
0.9
Pressure (mm Hg)
120
100
80
60
40
20
0
Aortic valve opens
Aortic valve closes
A-V valve closes
A-V valve opens
Aortic pressure
Atrial pressure
Ventricular pressure
Volume (ml)
160
120
80
Ventricular volume
R
P
Q
S
T
Lub
Dup
Electrocardiogram
Heart sounds
Ventricular systole
Ventricular diastole
Ventricular systole

Cardiac cycle …cont

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

Phases of the Cardiac Cycle

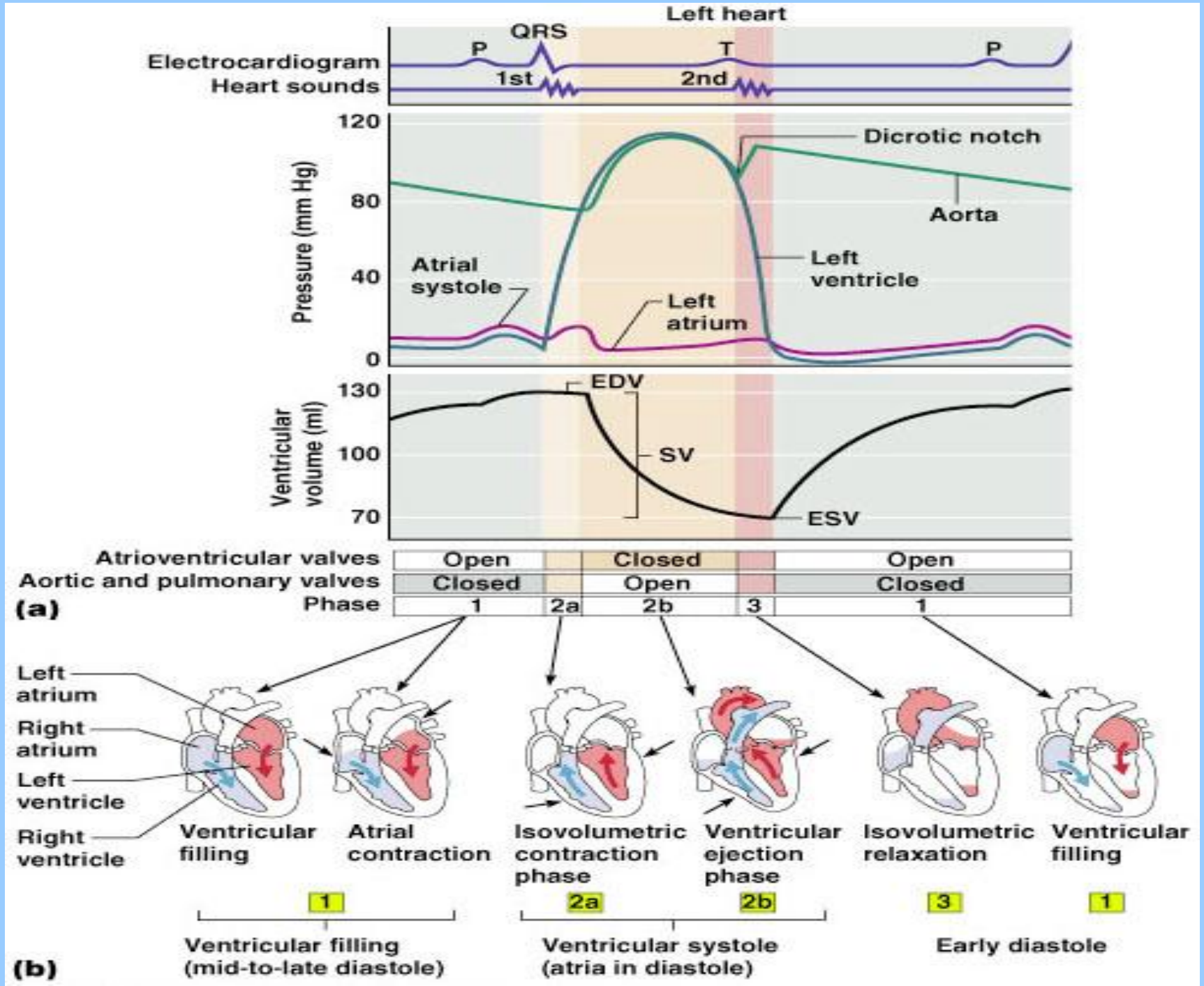
- Ventricular filling – mid-to-late diastole
  - Heart blood pressure is low as blood enters atria and flows into ventricles
  - AV valves are open, then atrial systole occurs

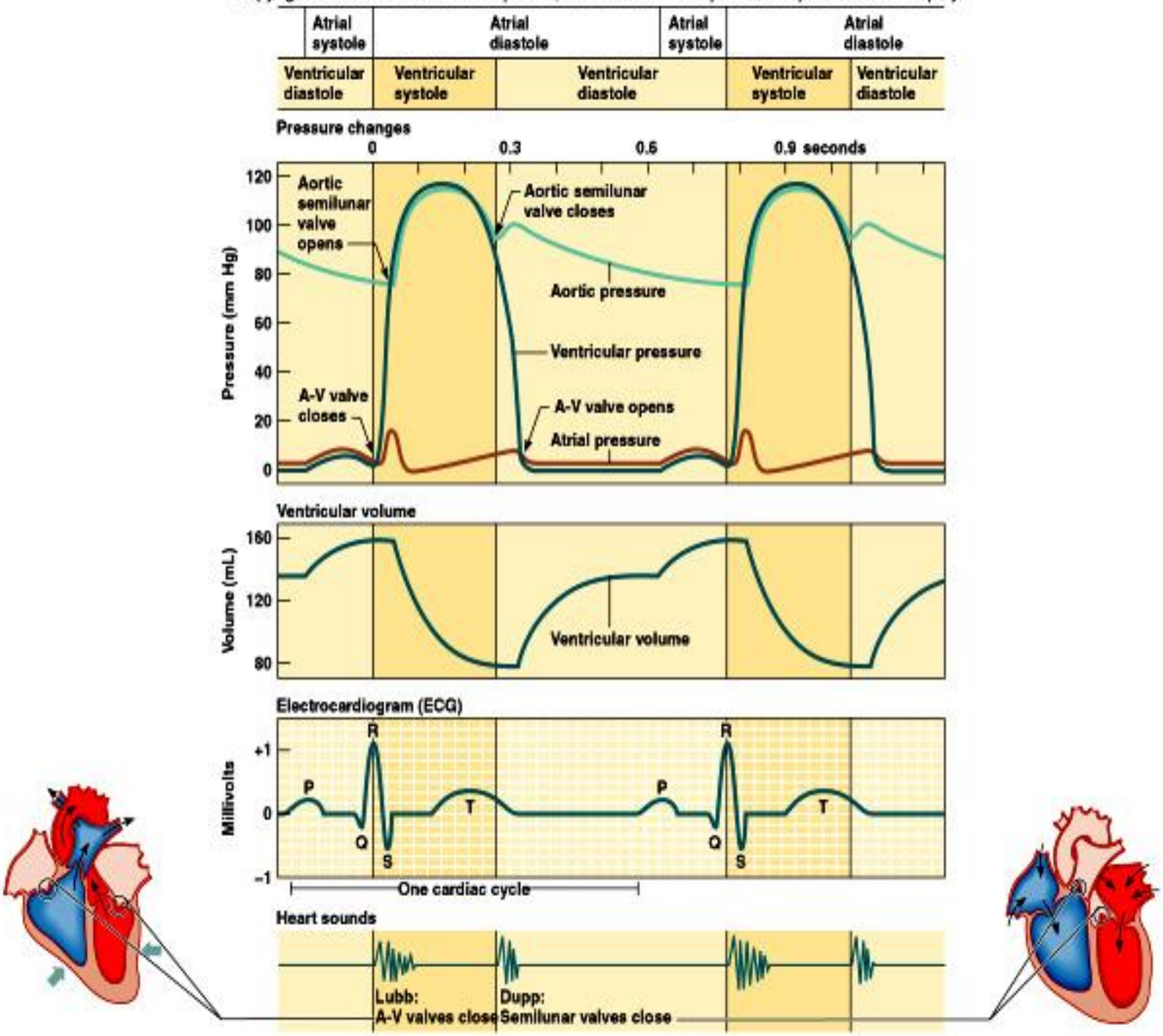
Phases of the Cardiac Cycle

- 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

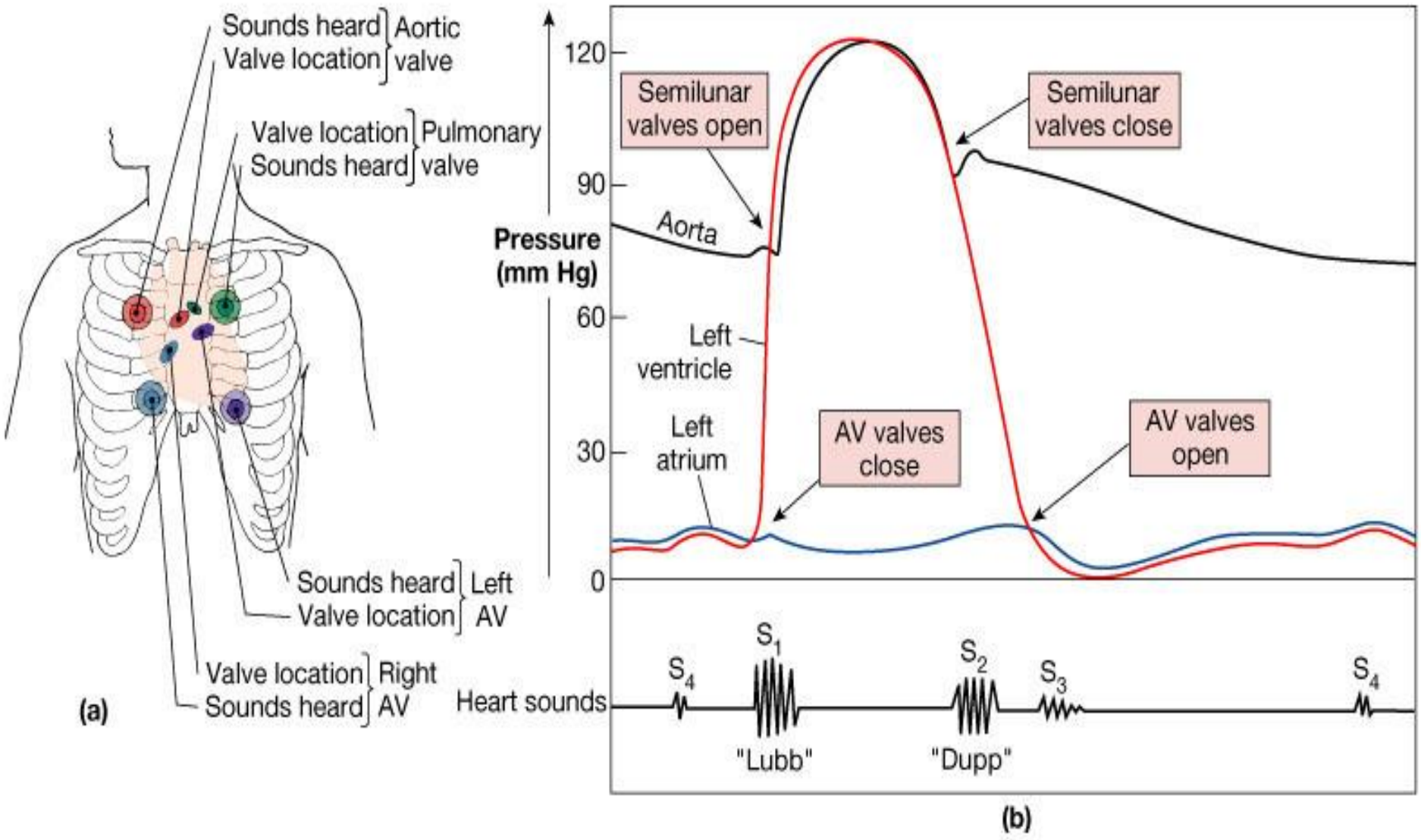

Left heart
Electrocardiogram
Heart sounds
QRS
P
T
P
1st
2nd
Pressure (mm Hg)
120
80
40
0
Dicrotic notch
Aorta
Atrial systole
Left ventricle
Left atrium
Ventricular volume (ml)
130
100
70
EDV
SV
ESV
Atrioventricular valves
Aortic and pulmonary valves
Phase
Open
Closed
Open
Closed
Open
Closed
1
2a
2b
3
1
(a)
Left atrium
Right atrium
Left ventricle
Right ventricle
Ventricular filling
Atrial contraction
Isovolumetric contraction phase
Ventricular ejection phase
Isovolumetric relaxation
Ventricular filling
1
2a
2b
3
1
Ventricular filling (mid-to-late diastole)
Ventricular systole (atria in diastole)
Early diastole
(b)



Changes during Cardiac cycle

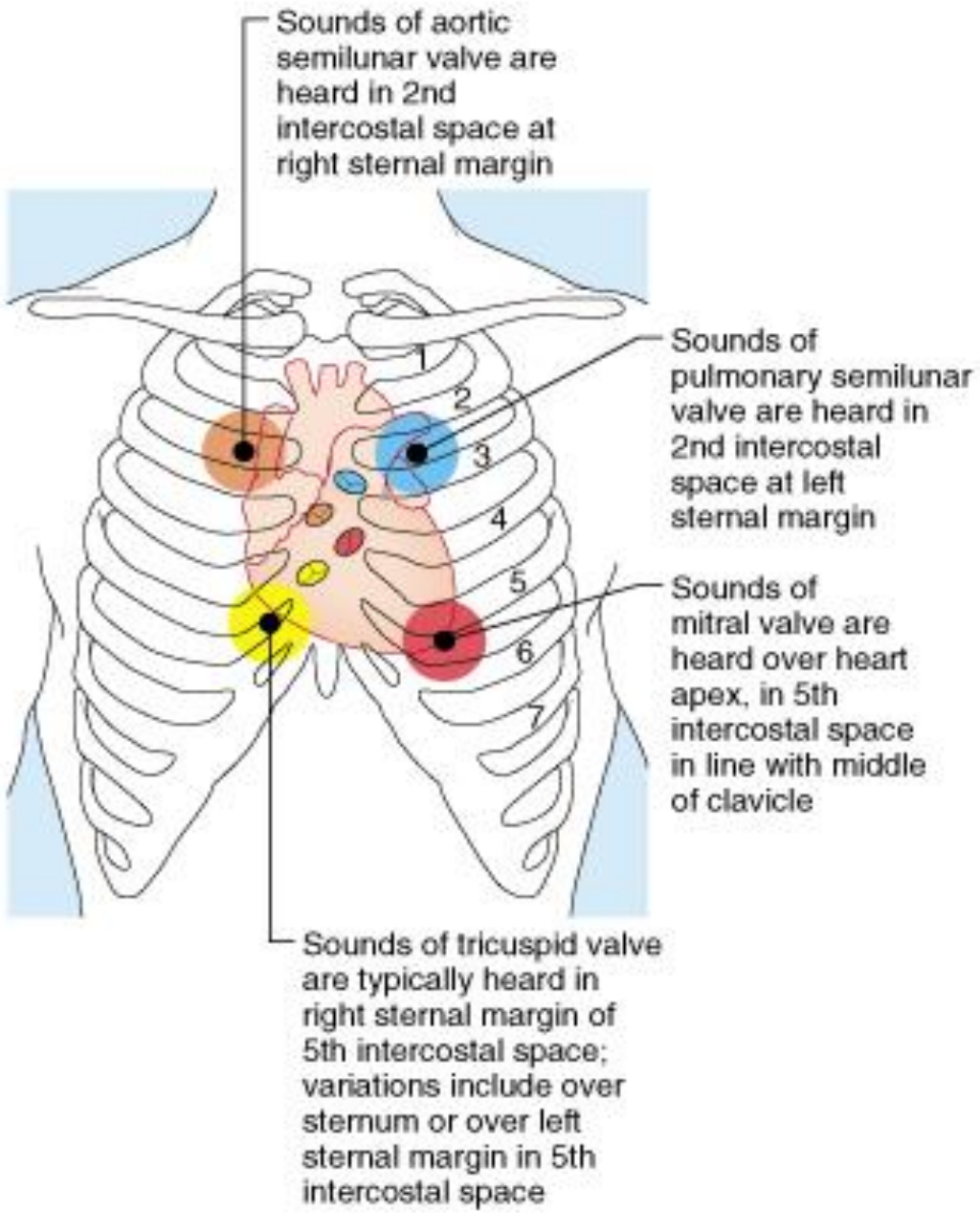
- Volume changes: End-diastolic volume, End-systolic volume, Stroke volume and Cardiac output.
- Aortic pressure: Diastolic pressure ~80 mmHg, Systolic pressure ~ 120 mmHg, most of systole ventricular pressure higher than aortic
- Ventricular pressure: Diastolic ~ 0, systolic Lt. ~120 Rt. ~ 25 mmHg.
- Atrial pressure: A wave =atrial systole, C wave= ventricular contraction (AV closure), V wave= ventricular diastole (Av opening). a-c-v waves. Bcs there is no valves between the Rt atrium and the SVC, the pressure waves in the Rt atrium are transmitted to the central veins which are distensible. They reflect a window inside the heart. a-wave at the end of the ventricular diastole “a” stands for atrial contraction, c-wave “c” stands for cusps, “v” stands for venous filling the atrium with blood.

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: Closure the valves make noise (S1 and S2). Opening of valves is silent
- S_1 = turbulence of blood around a closed AV valves, S_2 = turbulence of blood around a closed semilunar valves.
  - 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. During the rapid filling phase in adults and after exercise S3 might be heard (due to rapid venous return). This sound might be physiological or might be pathological.
  - S4 – another faint sound associated with atrial contraction. In stiffed ventricles, the hypertrophic atrium has to work harder and pump blood against a stiffed ventricle, atrial contraction phase might produce S4. This sound is always pathological.

S_3: Early diastole during rapid filling → turbulent flow→S3. It is normal in children where they have hyper dynamic state of blood flow. It is seen in mitral incompetence due to increase blood flow to the left ventricle. In heart failure it gives a characteristic **gallop rhythm (triple rhythm خبب)**, caused by ventricular dilation.

Ventricular filling is normally silent. This heart sound is normal in children; but is often pathological in adults and caused by ventricular dilation.

Seen in 1. Childern, 2. HF, and 3. mitral incompetence

S_4: This can only be recorded by phonocardiogram. We cannot hear it even in pathological conditions. It is recorded at late diastolic phase duo atrial contraction. It is inaudible to the naked ear. Is caused by vibration of the ventricular wall during atrial contraction. Generally, it is noted when the ventricle compliance is reduced ("stiff" ventricle) as occurs in ventricular hypertrophy and in many older individuals

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 (ml/min) = HR (75 beats/min) x SV (70 ml/beat)
- CO = 5250 ml/min (5.25 L/min)

Regulation of Stroke Volume

- SV = end diastolic volume (EDV) minus end systolic volume (ESV)
- EDV = amount of blood collected in a ventricle during diastole
- ESV = amount of blood remaining in a ventricle after contraction. End systolic or end diastolic (volume or pressure) is time-point.

Cardiac reserve: The maximum percentage that the cardiac output can increase above normal. If cardiac output rises to 25 l/min, this corresponds to 400% cardiac reserve.

Diagnosis of Cardiac reserve: Exercise Test: Symptoms of HF such as 1. Dyspnea, 2. muscle fatigue, and 3. Excessive increase in HR because the nervous system is trying to compensate for the decrease Q.

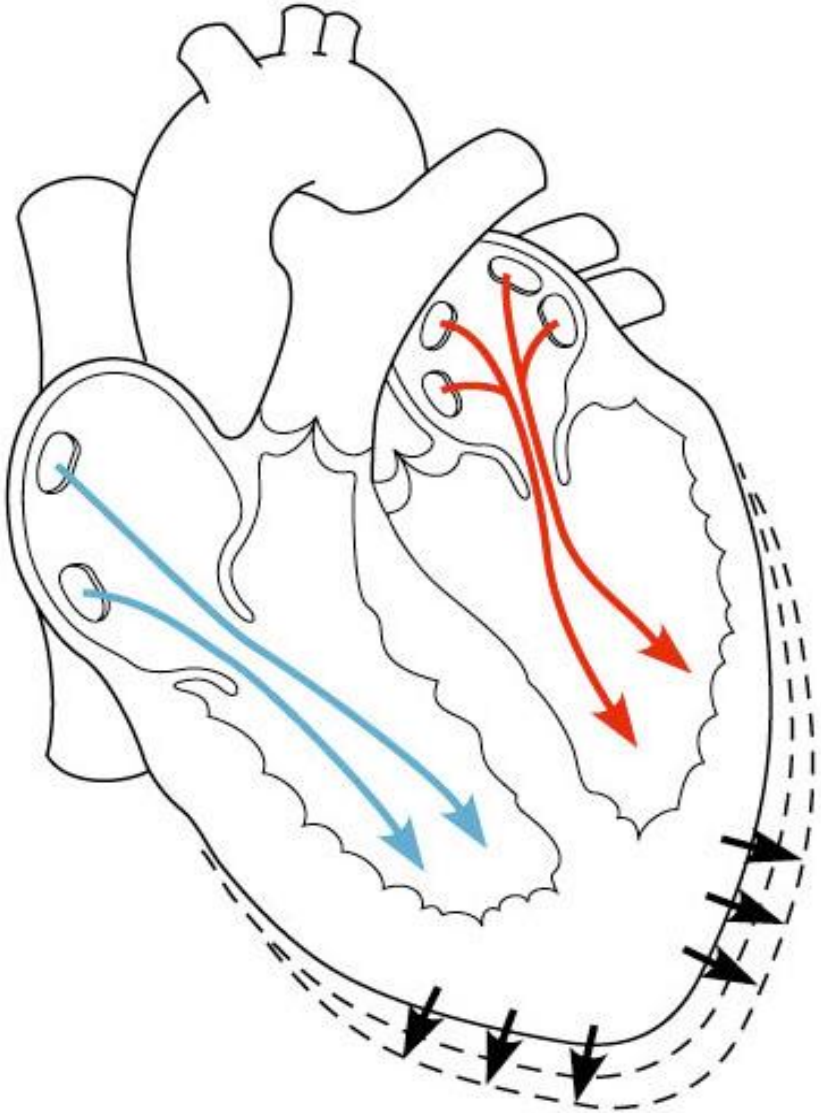
Cardiac Reserve	Normal	400%
Athlete		600%
Moderate coronary disease		200%
Severe coronary thrombosis		Zero%
Sever valvular disease		0%

Factors Affecting Stroke Volume

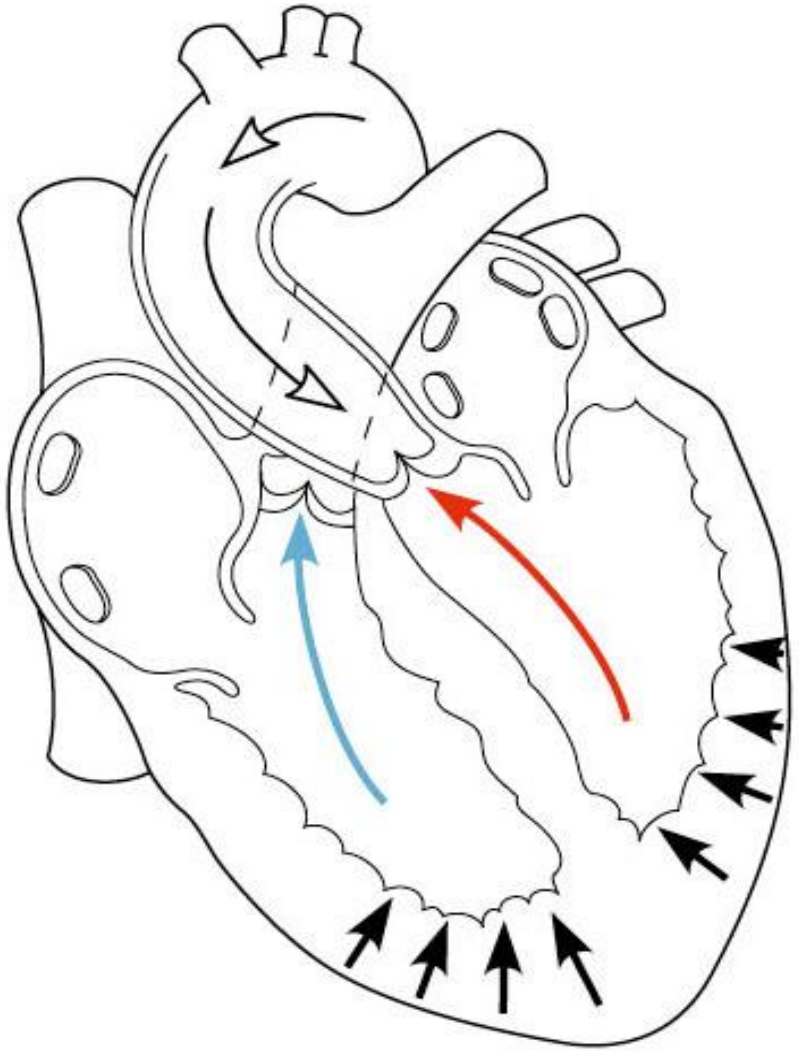
- Preload – amount ventricles are stretched by contained blood
- Contractility – cardiac cell contractile force due to factors other than EDV
- Afterload – back pressure exerted by blood in the large arteries leaving the heart

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

Increased end-diastolic volume (stretches the heart)

Positive inotropic agents such as increased sympathetic stimulation; catecholamines, glucagon, or thyroid hormones in the blood; increased Ca^{2+} in extracellular fluid

Decreased arterial blood pressure during diastole

Increased PRELOAD

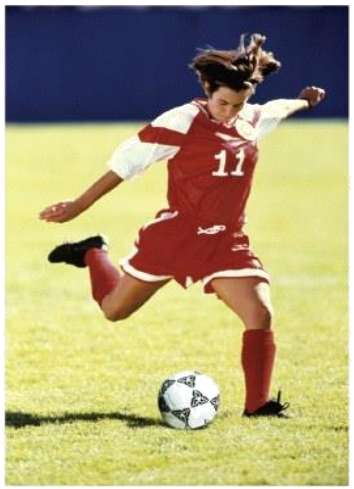
Increased CONTRACTILITY

Decreased AFTERLOAD

Within limits, cardiac muscle fibers contract more forcefully with stretching (Frank–Starling law of the heart)

Positive inotropic agents increase force of contraction at all physiological levels of stretch

Semilunar valves open sooner when blood pressure in aorta and pulmonary artery is lower



Increased STROKE VOLUME

Increased CARDIAC OUTPUT

Increased HEART RATE

Increased sympathetic stimulation and decreased parasympathetic stimulation

Catecholamine or thyroid hormones in the blood; moderate increase in extracellular Ca^{2+}

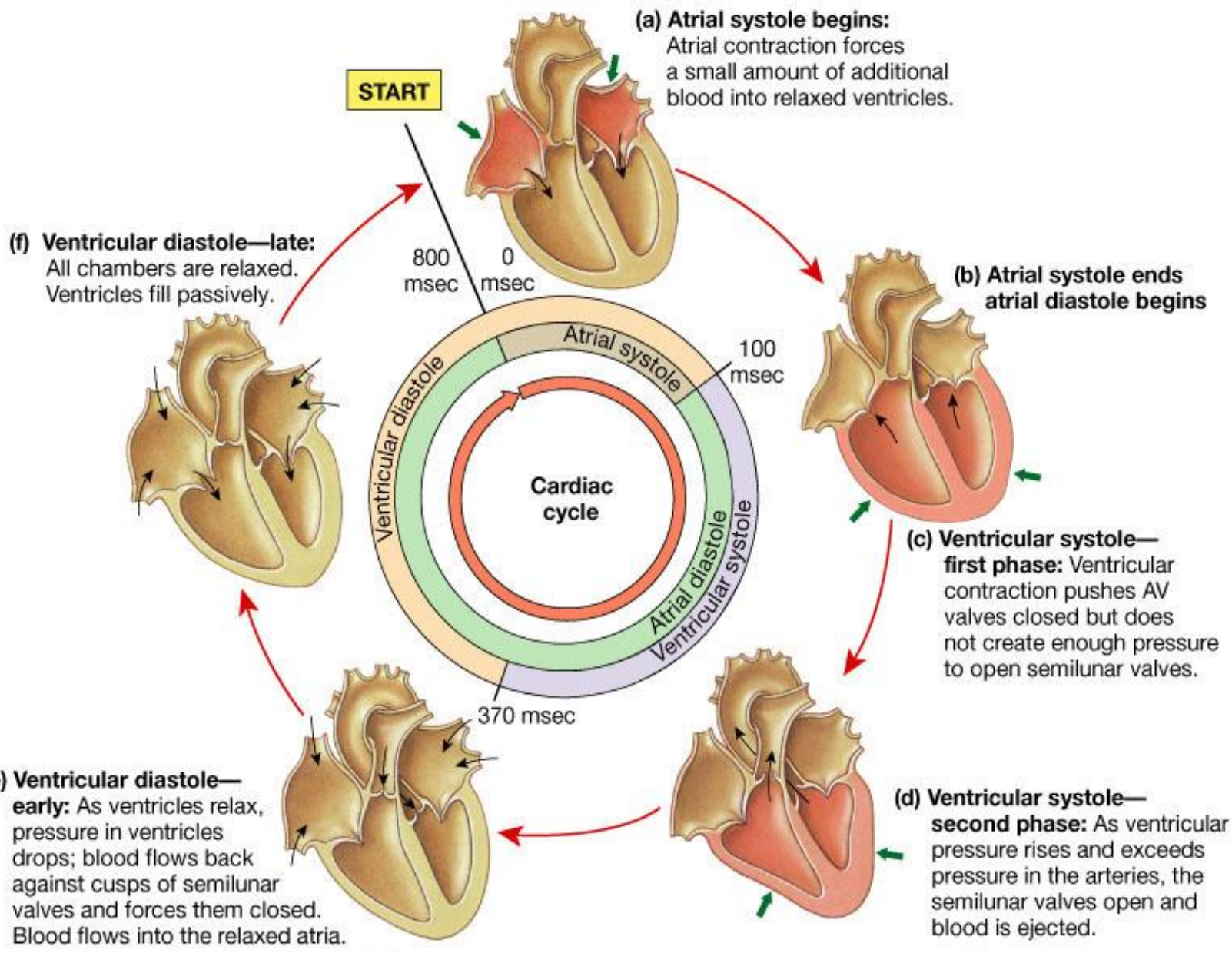
Infants and senior citizens, females, low physical fitness, increased body temperature

NERVOUS SYSTEM
Cardiovascular center in medulla oblongata receives input from cerebral cortex, limbic system, proprioceptors, baroreceptors, and chemoreceptors

CHEMICALS

OTHER FACTORS

Phases of the Cardiac Cycle



Extrinsic Factors Influencing Stroke Volume

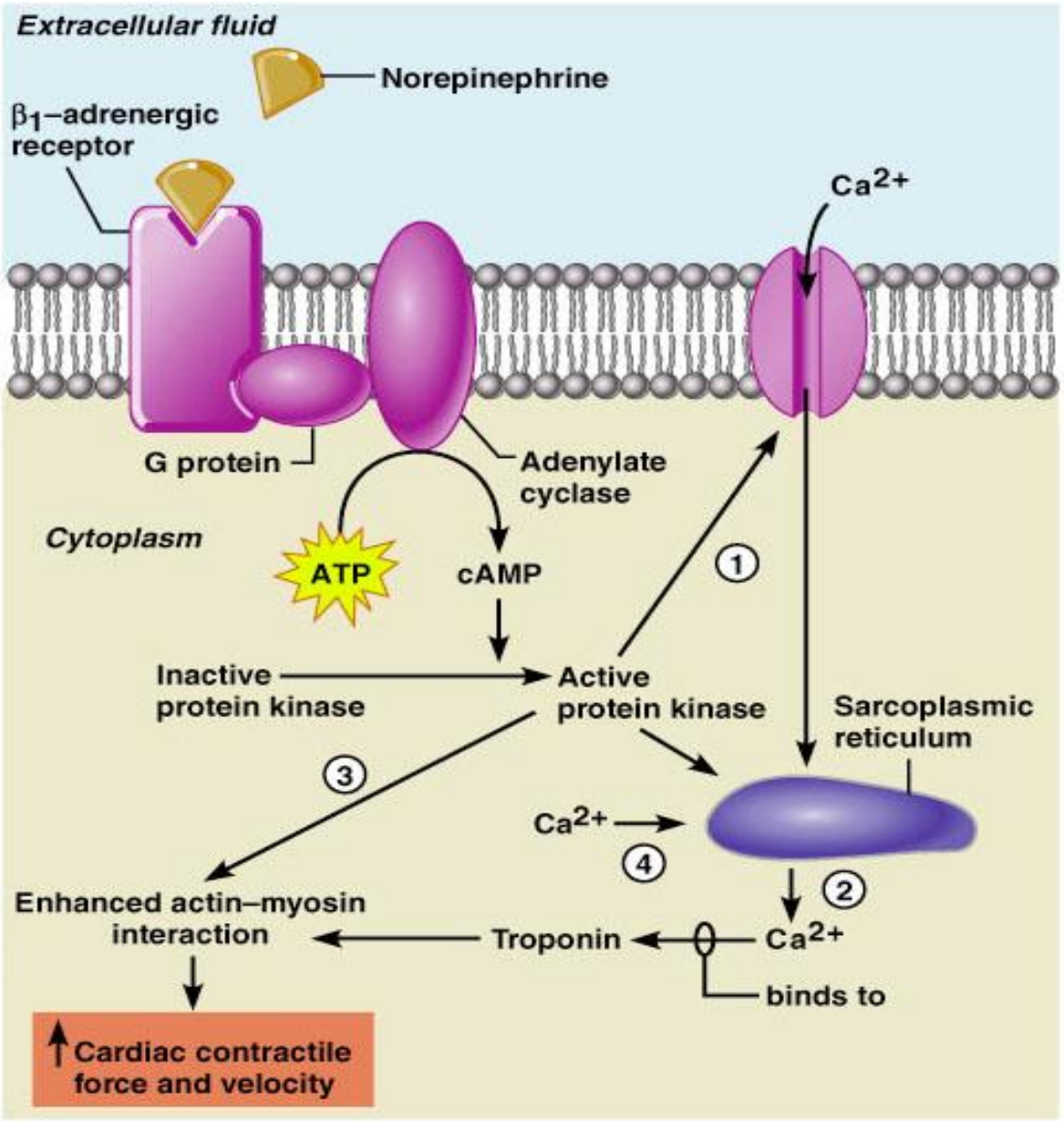
- Contractility is the increase in contractile strength, independent of stretch and EDV
- Increase in contractility comes from:
  - Increased sympathetic stimuli
  - Certain hormones
  - Ca^{2+} and some drugs

Extrinsic Factors Influencing Stroke Volume

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

Contractility and Norepinephrine

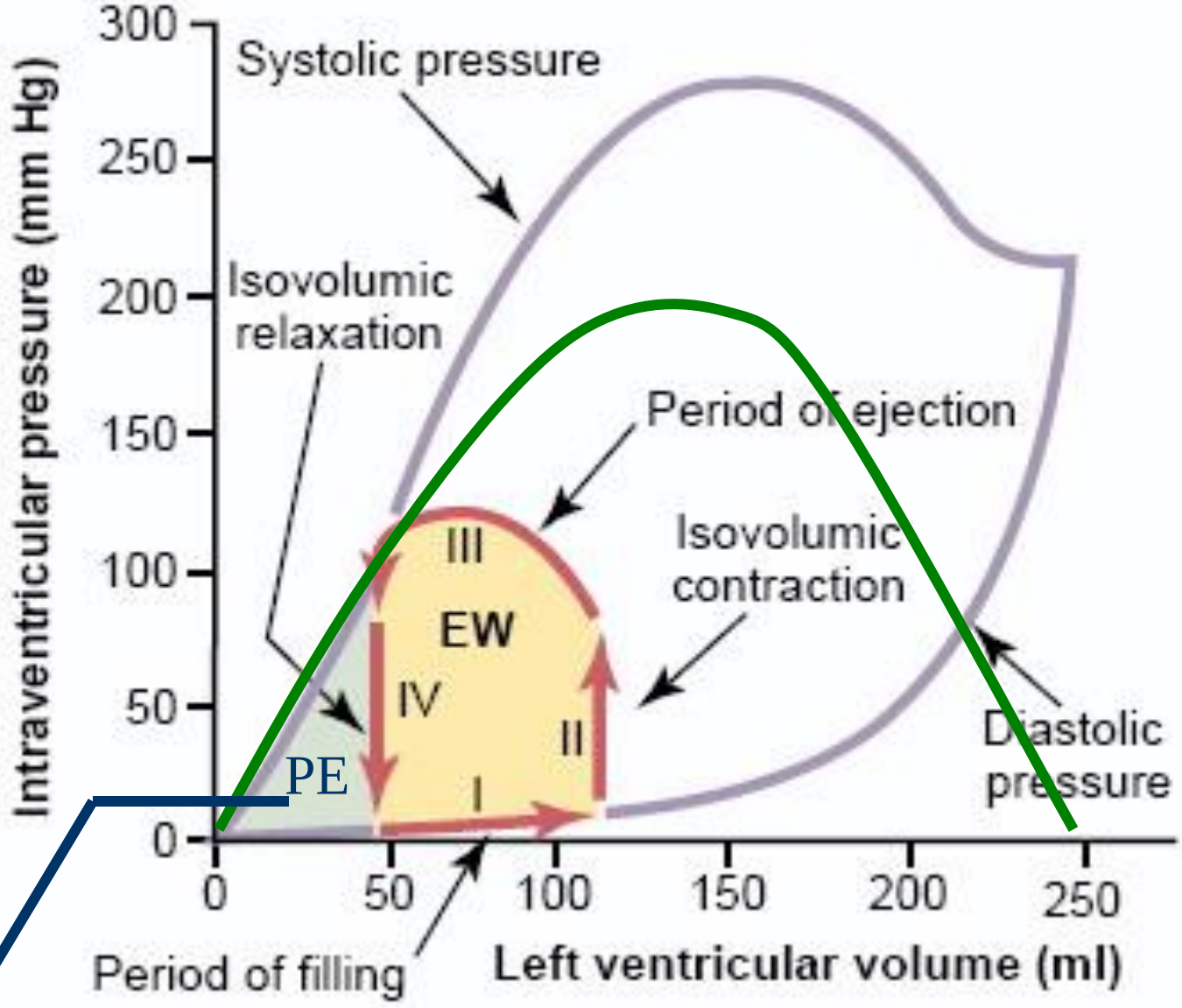
- Sympathetic stimulation releases norepinephrine and initiates a cyclic AMP second-messenger system



Tissue	Blood flow (ml/g/min)	A-V difference Vol%
Heart	0.8	11
Brain	0.5	6.2
Sk muscles	0.03	6
Liver	0.6	3.4
Kidney	4.2	1.4
Carotid bodies	20	0.5

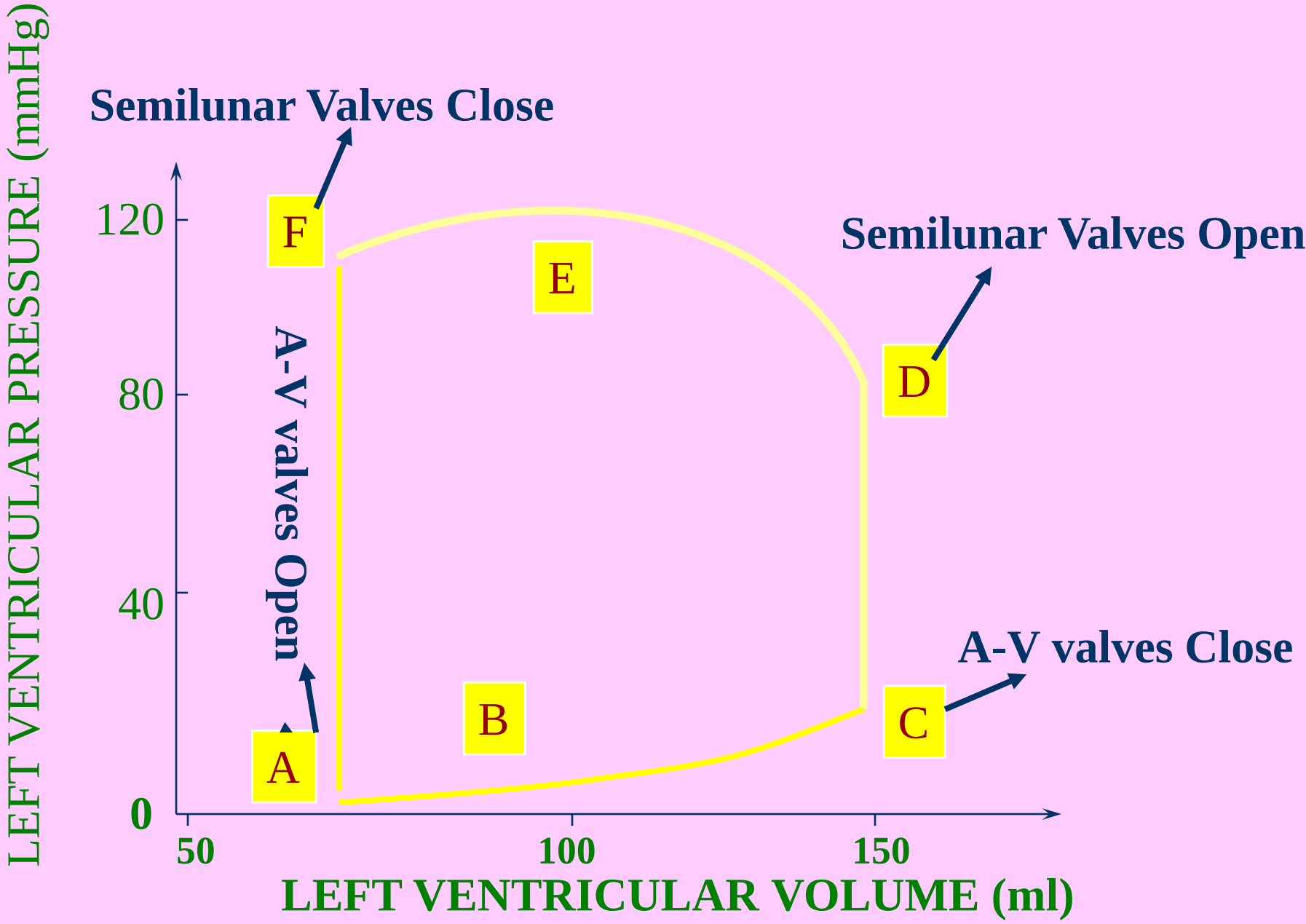
Carotid blood flow (ml/g/min)

Tissue	Blood flow (ml/g/min)	A-V difference (Vol %)	Flow ml/min	O_2 consumption ml/min
Heart	0.8	11	250	27
Brain	0.5	6.2 (25-30% Extraction)	750-900	
Skeletal Muscle	0.03	6	1200	70
Liver	0.6	3.4 Reconditioner organ		
SKIN	0.1			
Kidney	4.2	1.4	1250	18
Carotid bodies	20	0.5	0.6	



Potential Energy (PE)

LEFT VENTRICULAR PRESSURE/VOLUME P/V LOOP:
It is time independent diagram



Valvular Function

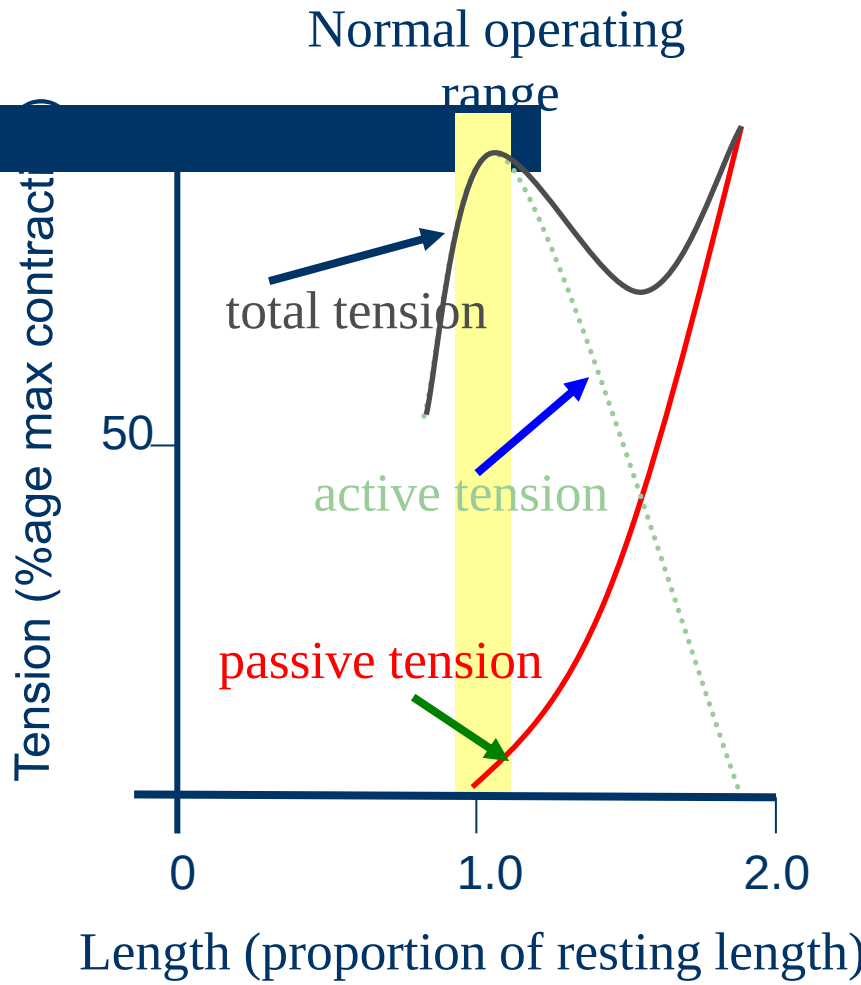
- To prevent back-flow.
- Chordae tendineae are attached to A-V valves.
- Papillary muscle, attached to chordae tendineae, contract during systole and help prevent back-flow.
- Because of smaller opening, velocity through aortic and pulmonary valves exceed that through the A-V valves.

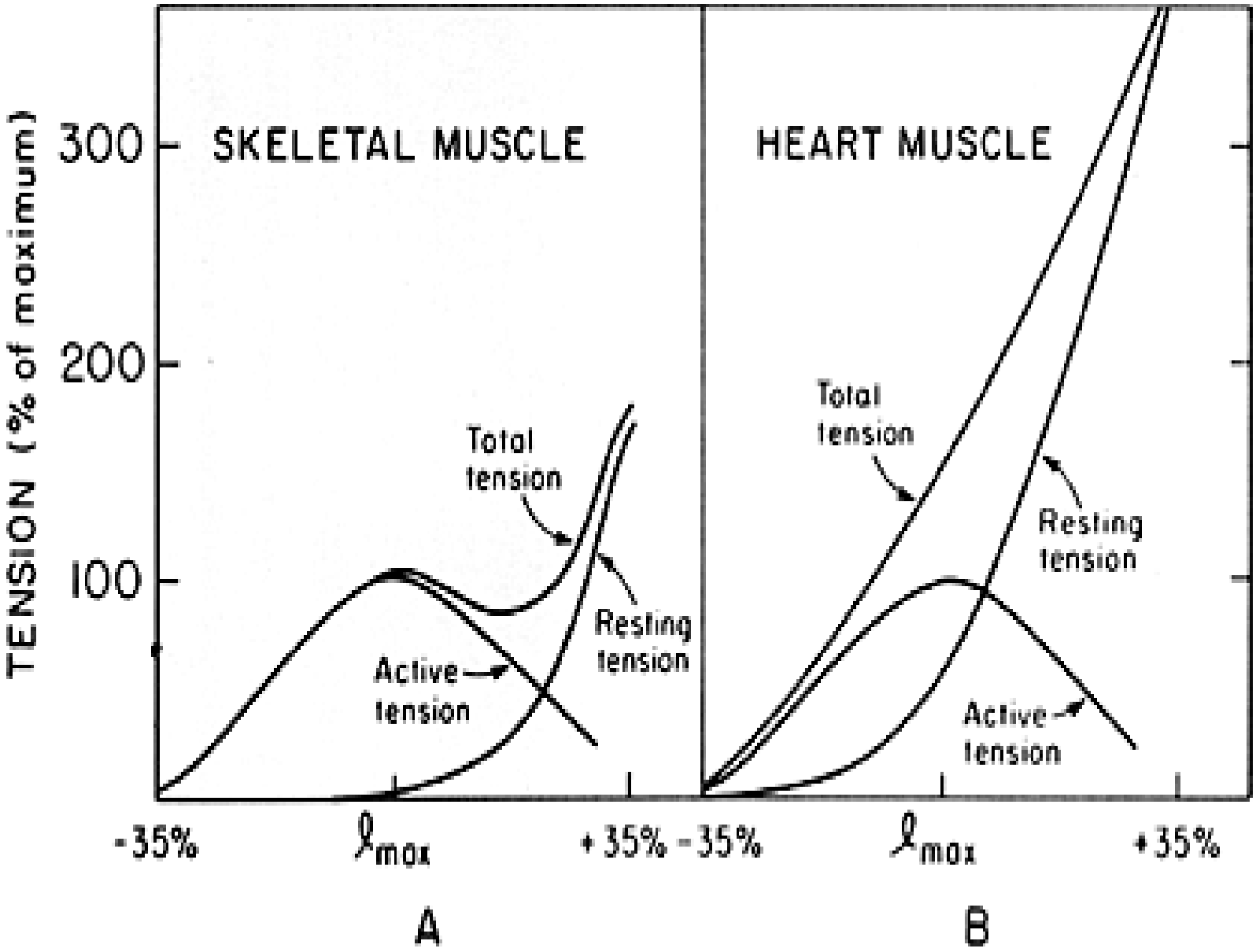
Valvular Function (cont'd)

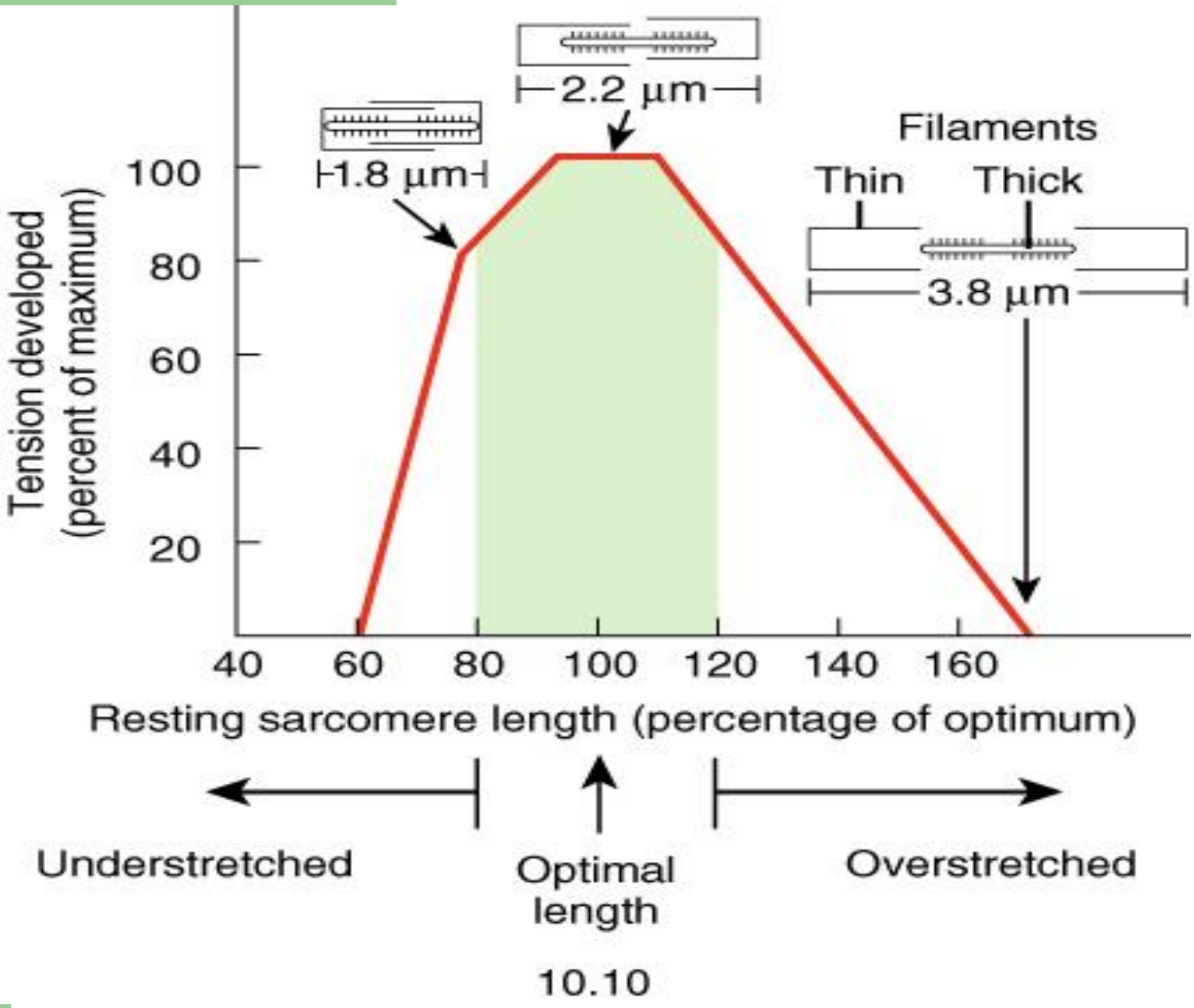
- Most work is external work or pressure-volume work.
- A small amount of work is required to impart kinetic energy to the heart ($1/2\ mV^2$).
- What is stroke-volume in previous figure?
- External work is area of Pressure-Volume curve.
- Work output is affected by "preload" (end-diastolic pressure)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

Length-Tension Relation for Skeletal Muscle

- Active tension cannot be
- What can be measured?
  - (1) passive tension - *tension required to extend a resting muscle*
  - (2) total tension - *active tension and passive combined*
- Active is calculated from 1 & 2
- (AT = TT – PT)
- Note that active tension falls away linearly with increasing length

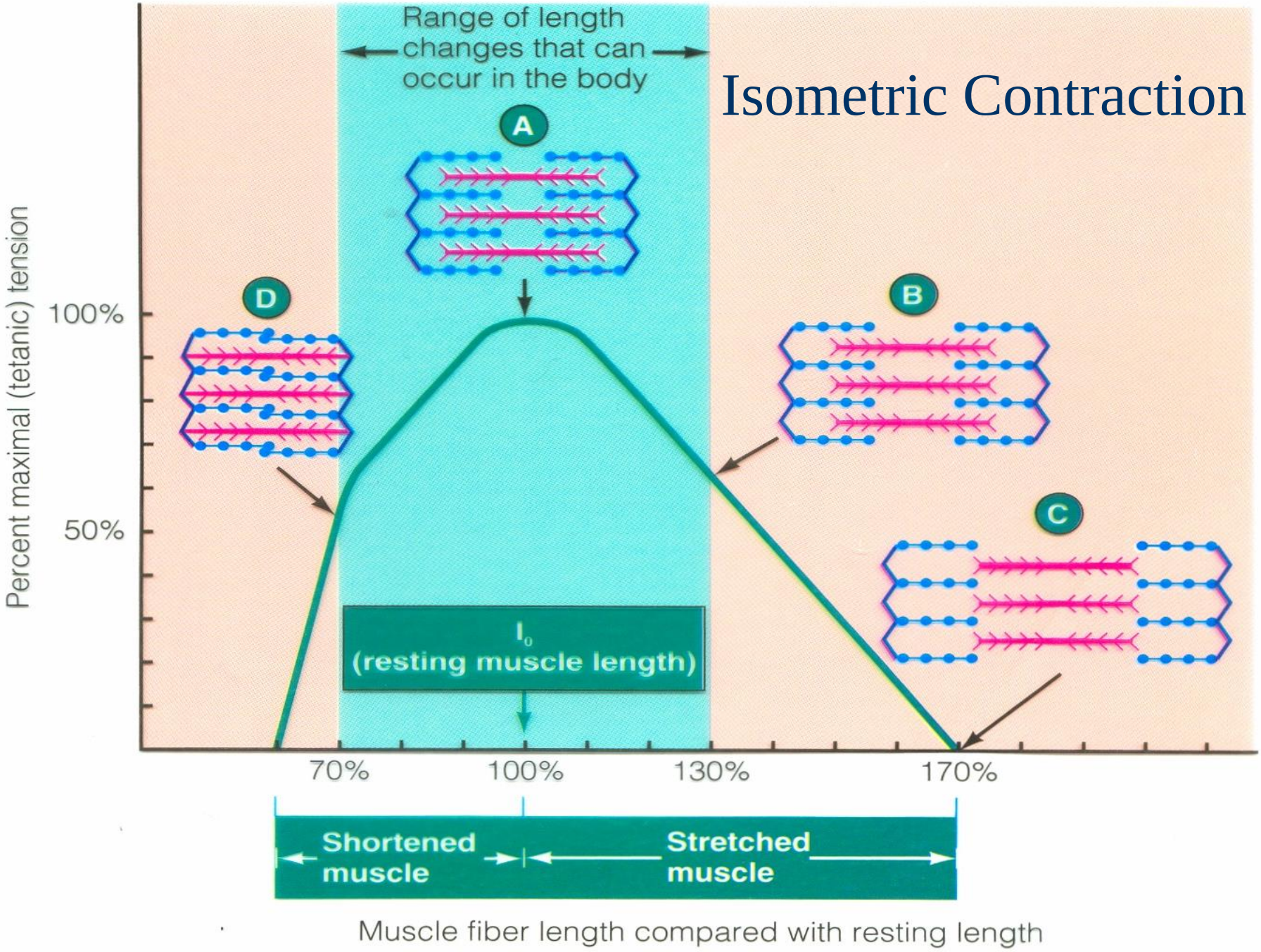



TENSION (% of maximum)
300
200
100
SKELETAL MUSCLE
HEART MUSCLE
Total tension
Resting tension
Active tension
Total tension
Resting tension
Active tension
-35%
l_{max}
+35%
-35%
l_{max}
+35%
A
B

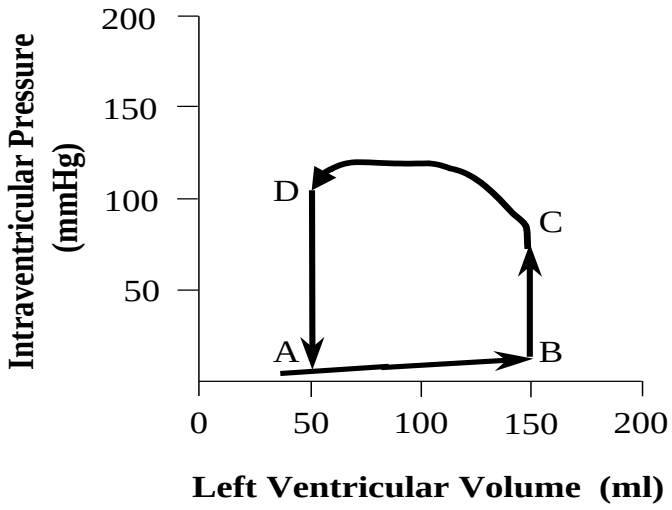

2.2 µm
1.8 µm
Filaments
Thin
Thick
3.8 µm
Tension developed (percent of maximum)
100
80
60
40
20
40
60
80
100
120
140
160
Resting sarcomere length (percentage of optimum)
Understretched
Optimal length
Overstretched

10.10

Isometric Contraction

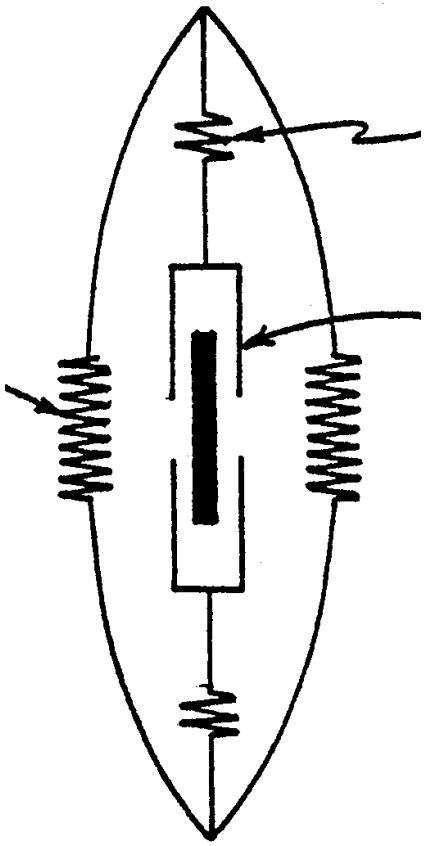


The PV diagram. From A-B (0.35 sec: filling phase), from B-C (0.05 sec isovlumetric contraction phase), from C-D (0.25 sec emptying phase=ejection), from D-A (0.15 sec isovolumetric relaxation phase). Total duration is 0.8 sec



PARALLEL ELASTIC ELEMENTS

(PASSIVE TENSION)



SERIES ELASTIC ELEMENTS

CONTRACTILE COMPONENT

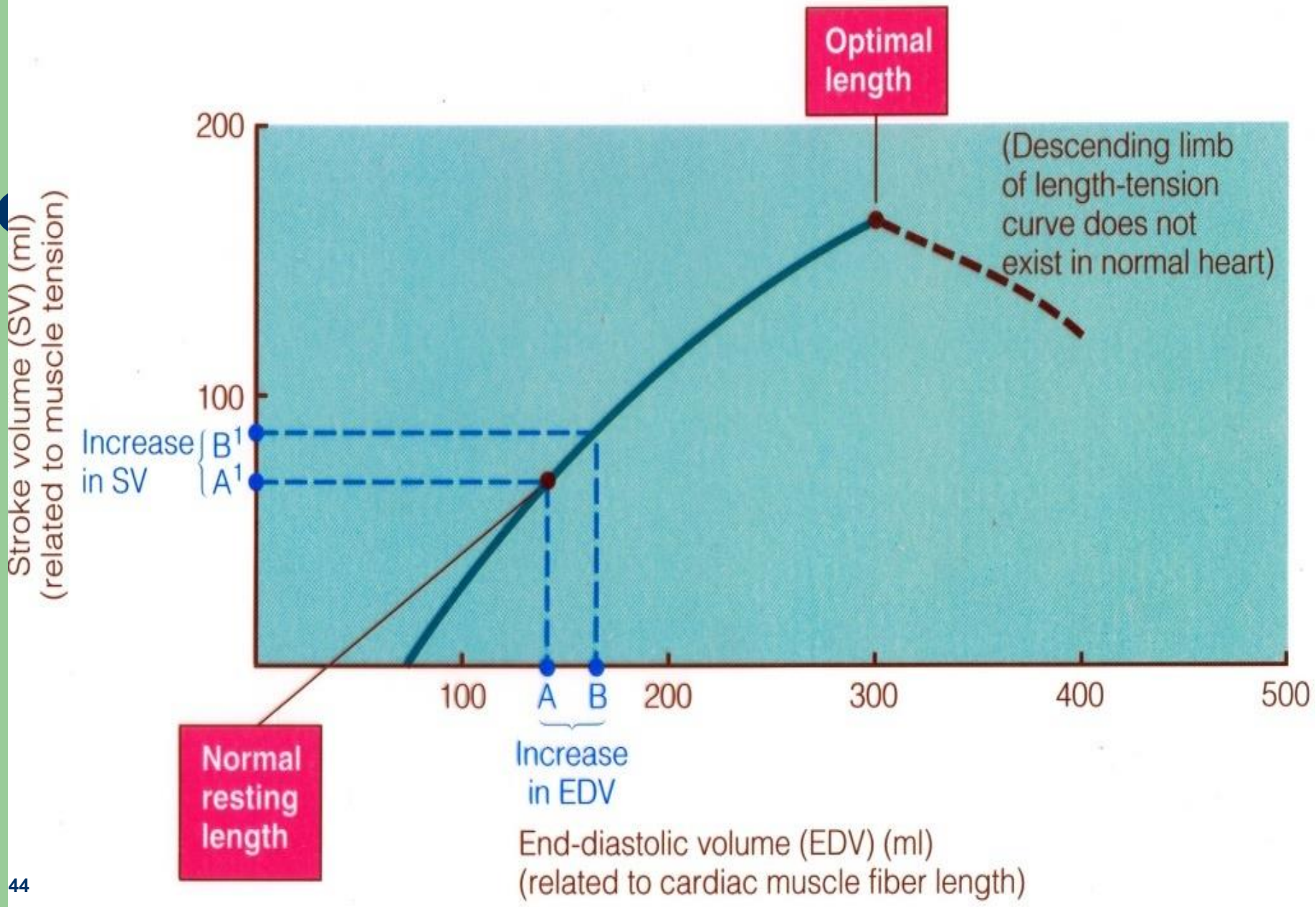
(ACTIVE TENSION)

TOTAL TENSION

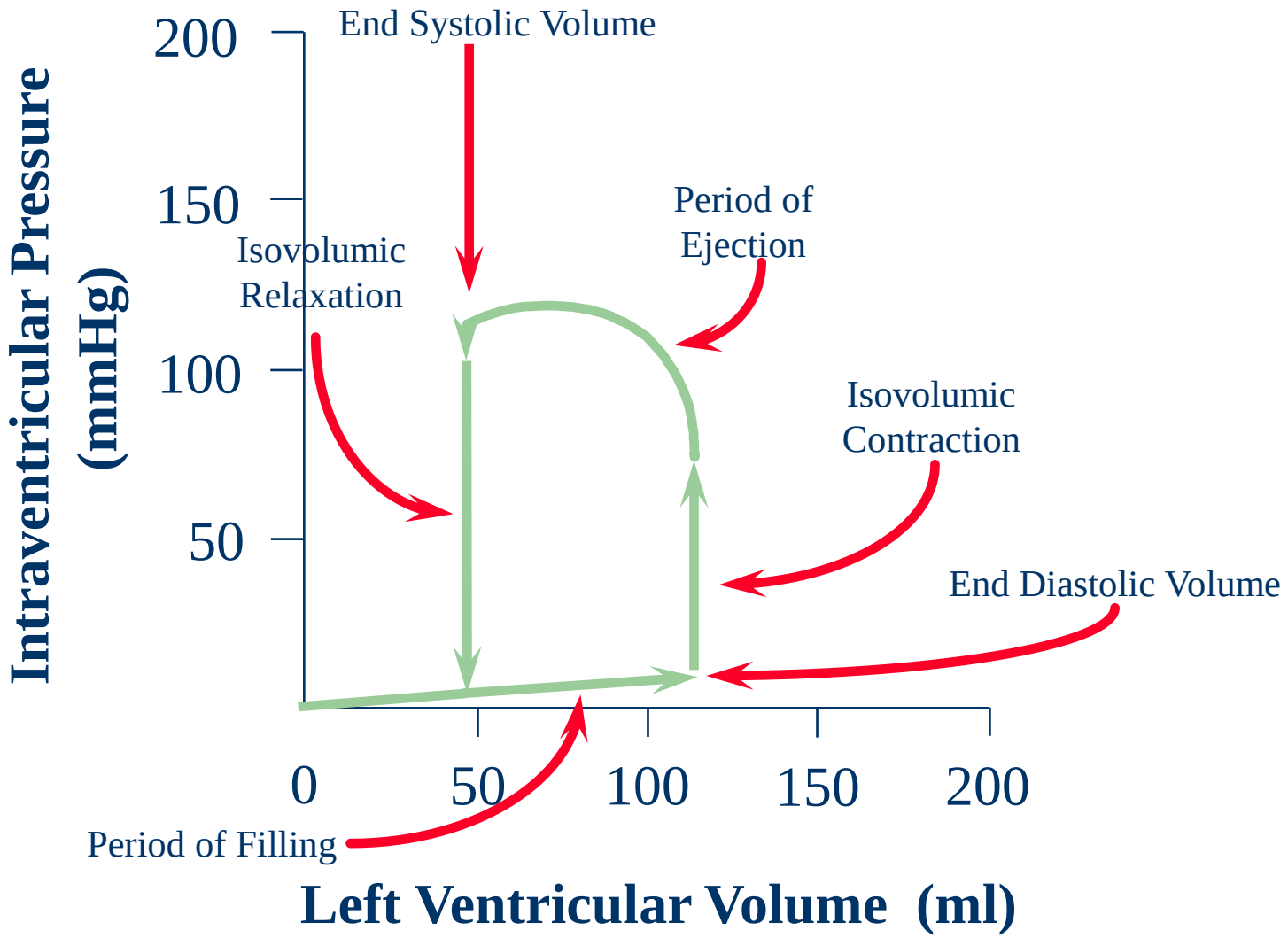
Cardiac Muscle length-tension relationship

- Cardiac muscle works at much less than its maximum length in contrast to skeletal
- Total, Active and Passive length-tension relationship differ
- Frank-Starling law of the heart

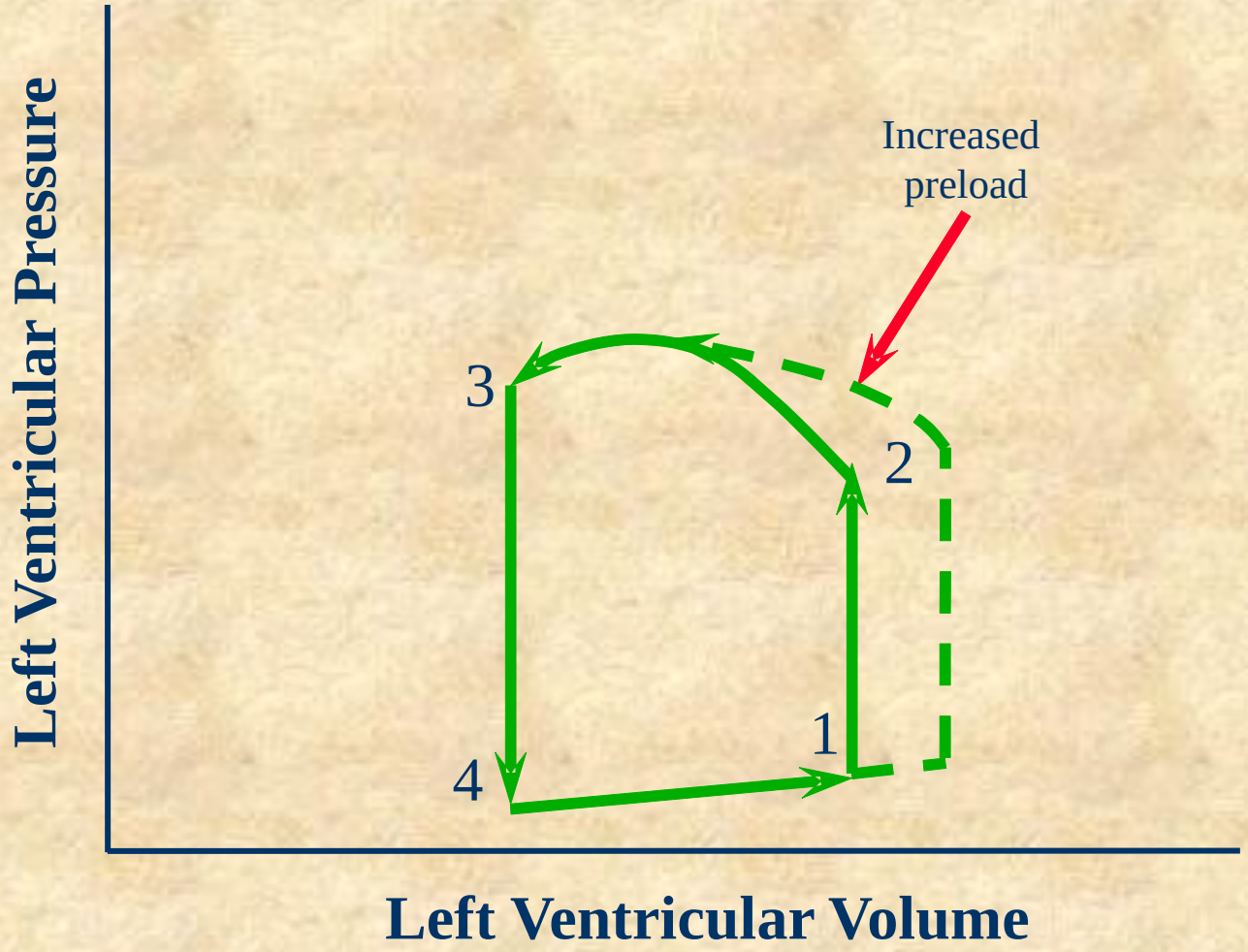
Intrinsic Control of Stroke Volume (Frank-Starling Curve)



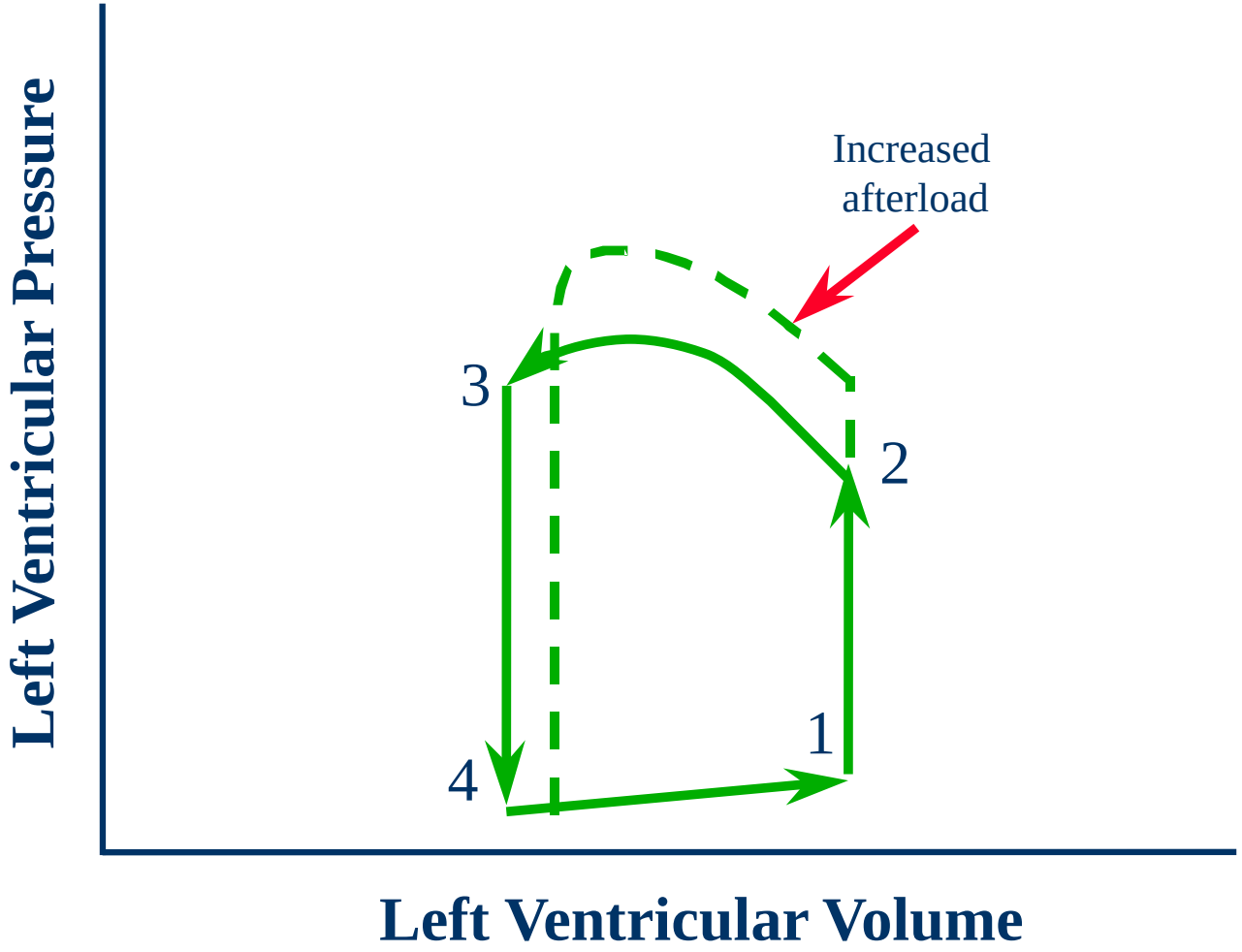
Work Output of the Heart



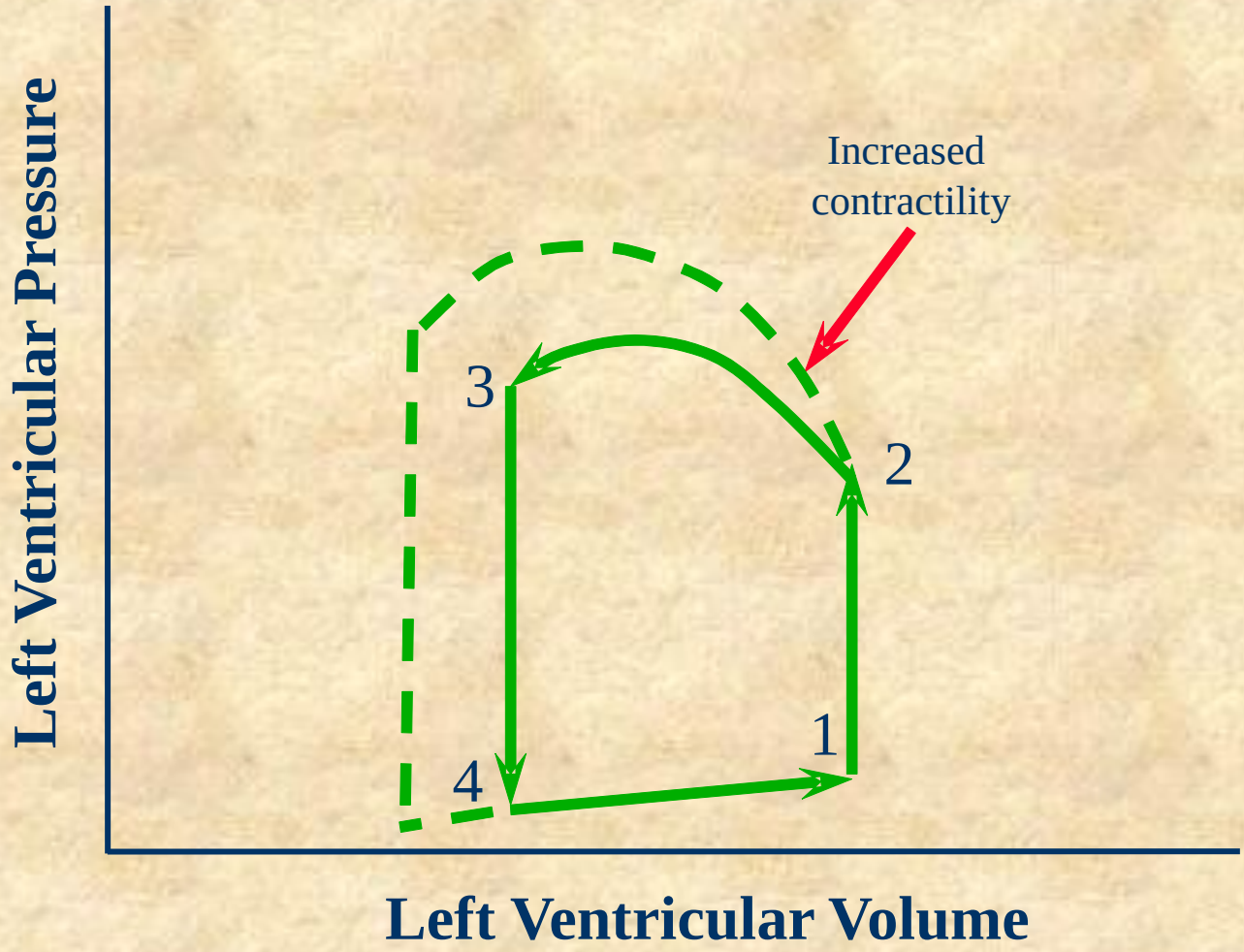
A


Left Ventricular Pressure
Increased
preload
3
2
1
4
Left Ventricular Volume

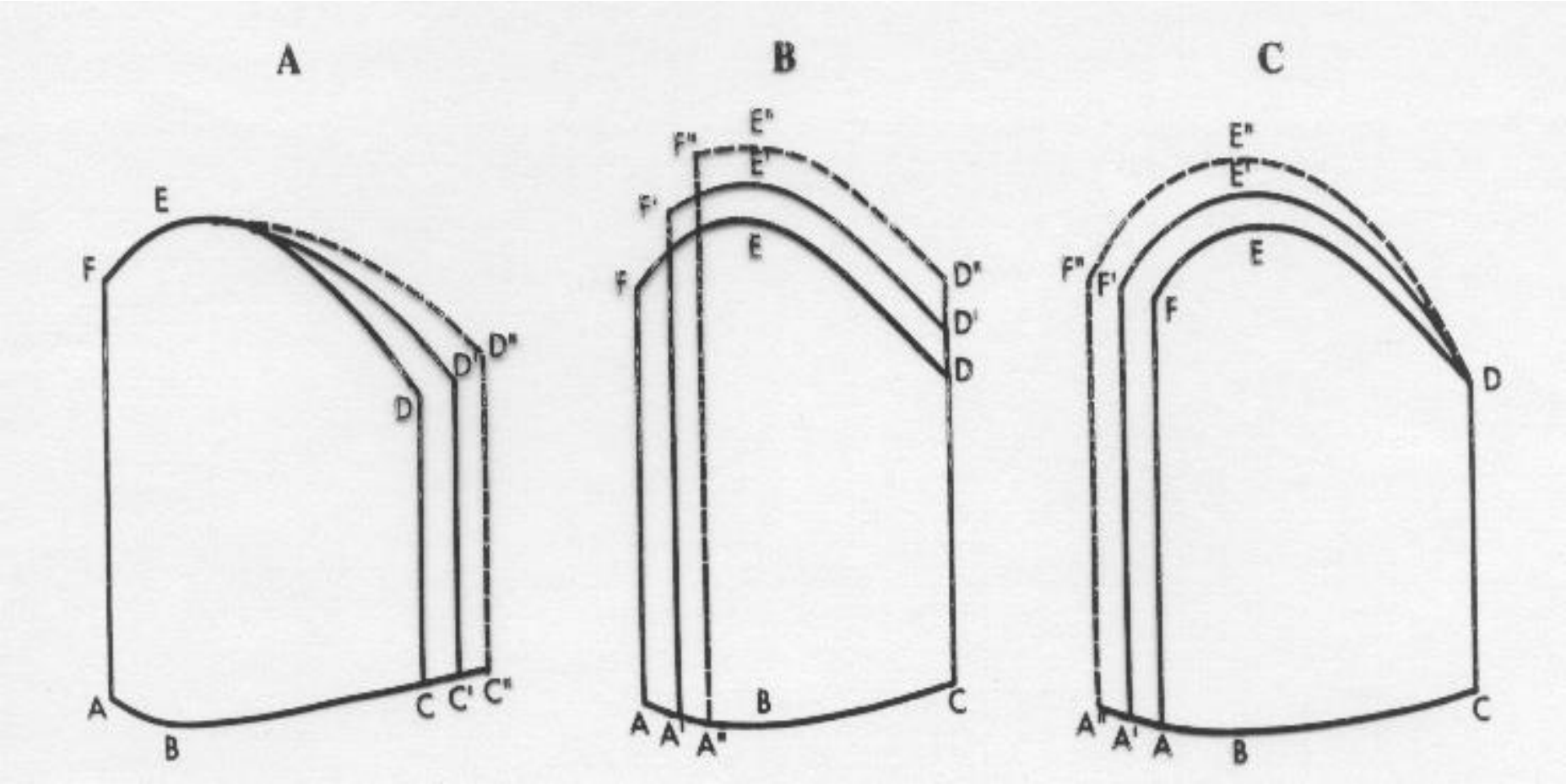
B


Left Ventricular Pressure
Increased
afterload
3
2
1
4
Left Ventricular Volume

C


Left Ventricular Pressure
Increased
contractility
3
2
1
4
Left Ventricular Volume

PRESSURE/VOLUME RELATIONSHIPS UNDER DIFFERENT CONDITIONS

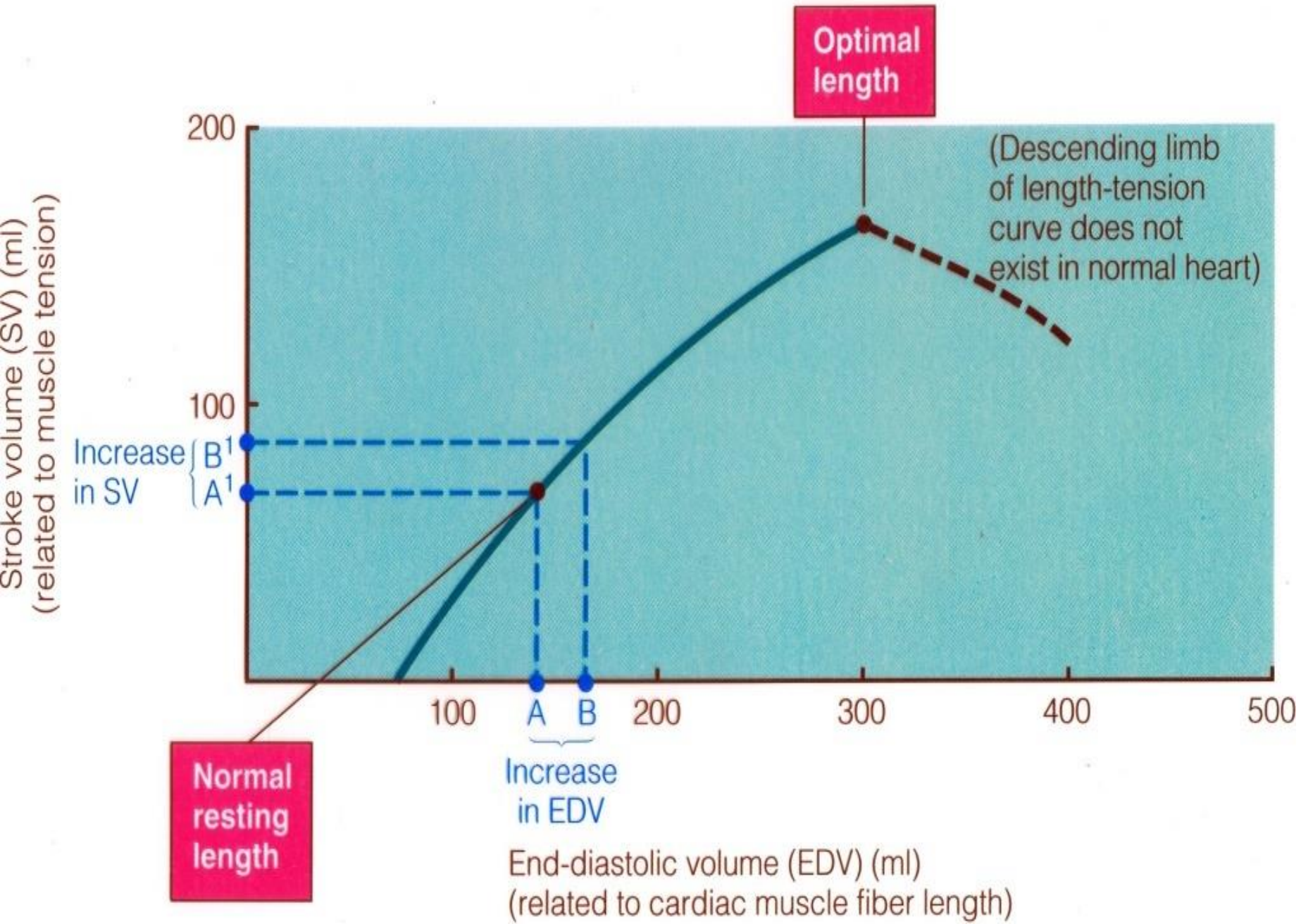


PRELOAD

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

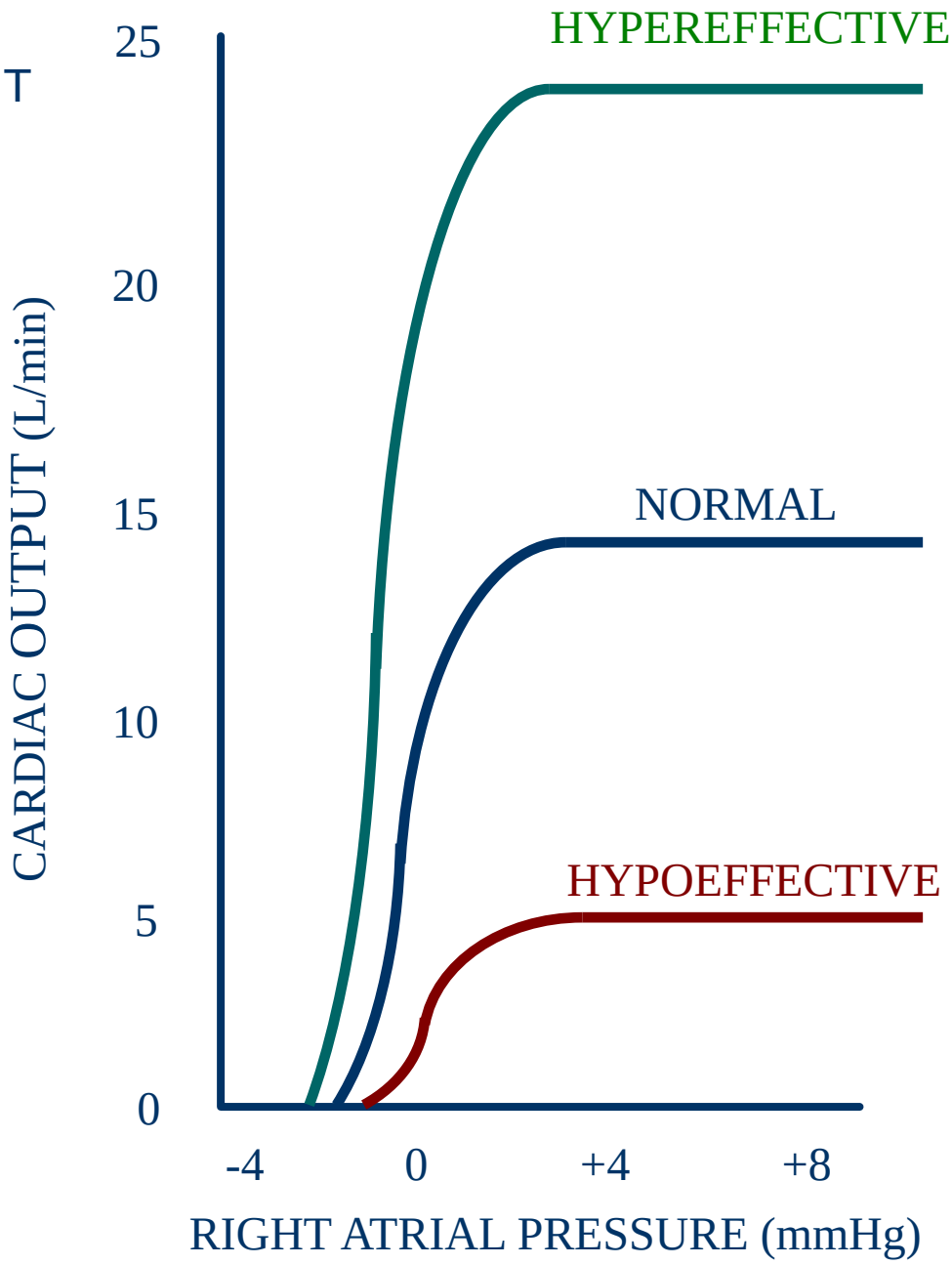
Chemical Regulation of the Heart

- 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/m^2 (surface area in m^2).
- CO is proportional to tissue O_2 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 = 70ml/125ml = 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 as a tool to calssify heart failure:
Depending on the percentage measured, and other tests, heart failure may be classed as the following:

- heart failure with preserved ejection fraction (HFpEF) - >50%)
- heart failure with mildly reduced ejection fraction (>40% - 49%)
- heart failure with reduced ejection fraction (<40%)

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%=~ 90%

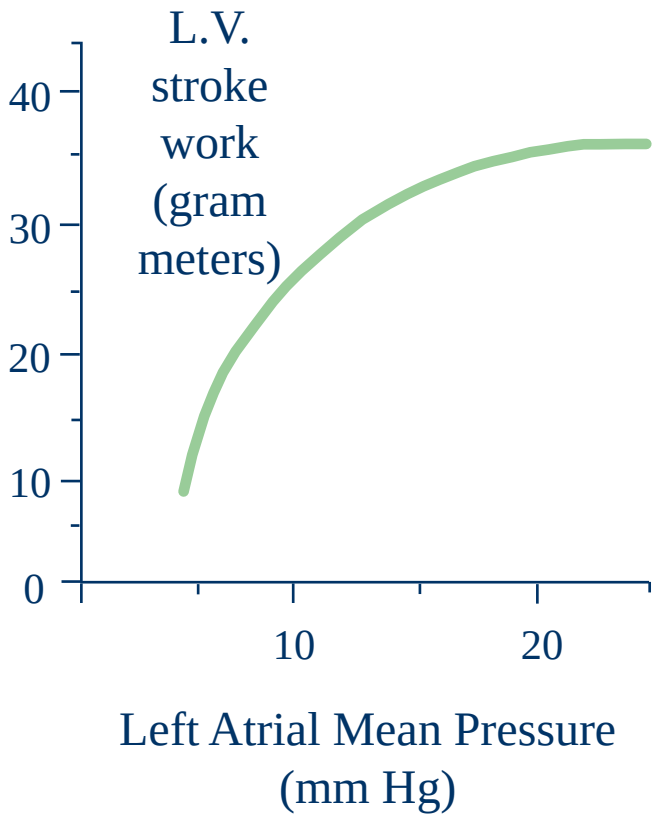
Aortic Pressure Curve

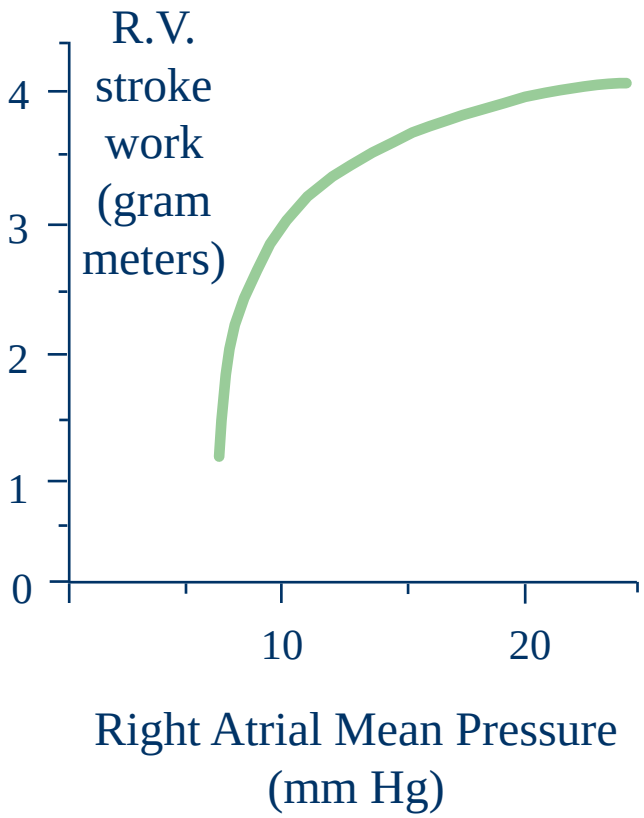
- Aortic pressure starts increasing during systole after the aortic valve opens.
- Aortic pressure decreases toward the end of the ejection phase.
- After the aortic valve closes, an *incisura* occurs because of sudden cessation of back-flow toward left ventricle.
- Aortic pressure decreases slowly during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

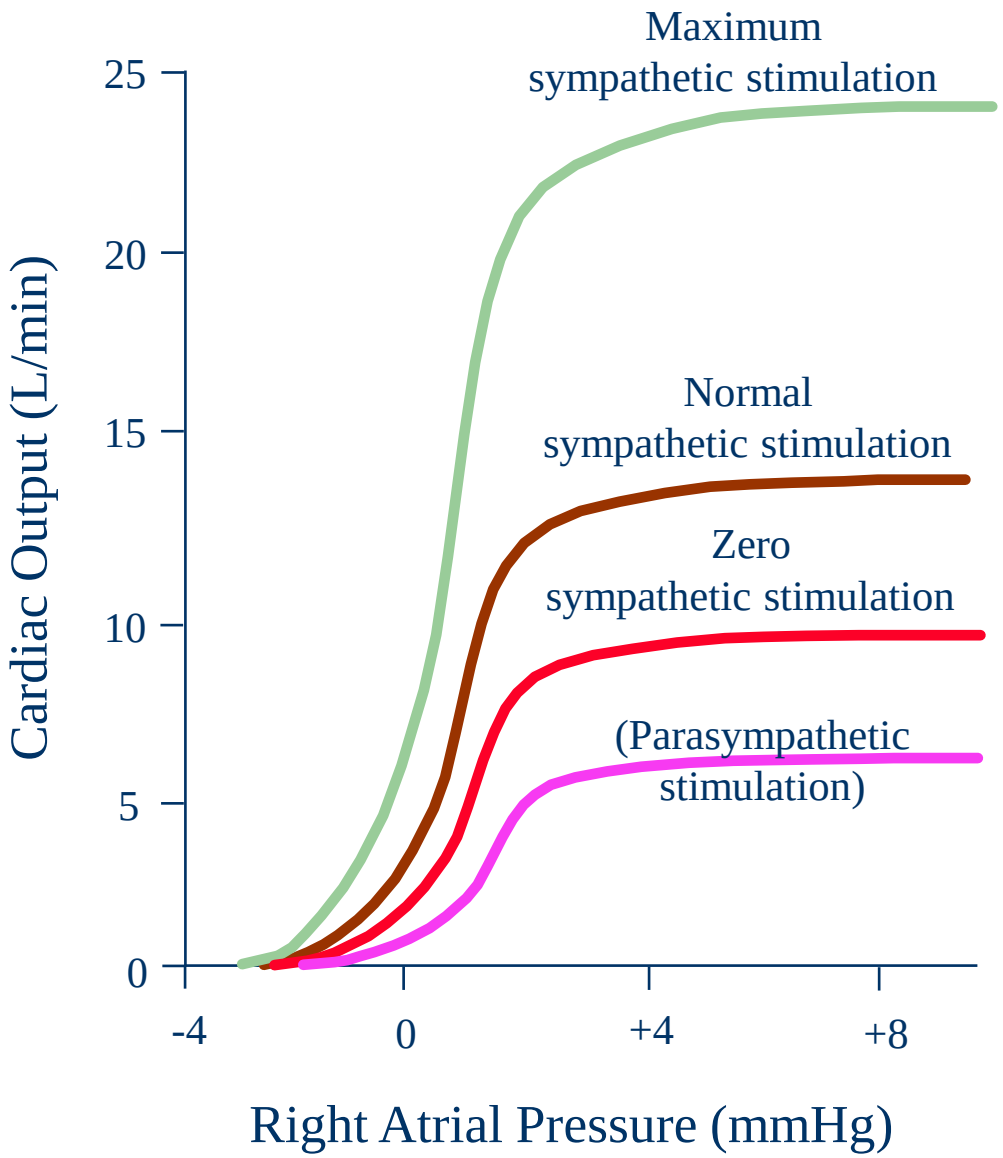




Autonomic Effects on Heart

- 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.
- $(dP/dt)/P_{ventricle}$ is better. $P_{ventricle}$ is instantaneous ventricular pressure.
- Excess K^+ decreases contractility.
- Excess Ca^{++} causes spastic contraction, and low Ca^{++} causes cardiac dilation.

Thank You