CARDIAC CYCLE

Learning objectives

- Cardiac cycle – phases
- Valve changes
- Pressure changes
- Volume changes

Cardiac cycle-

‘ The electrical and mechanical events that occur from the beginning of one heart beat to the beginning of next ’

Phases of cardiac cycle

Atrial cycle-

1. Atrial systole (0.1sec)
2. Atrial diastole (0.7sec)

Ventricular cycle-

1. Ventricular systole .(0.3sec)

I. Isovolumic (isometric) contraction phase(0.05 sec)

II. Ventricular Ejection phase

-Rapid ejection (0.1sec)

- slow ejection phase (0.15 sec)

2. <u>Ventricular Diastole </u>(0.5 sec)

I. Protodiastole (0.04 sec)

II. Isovolumic (isometric) relaxation (0.06 sec)

III. Ventricular filling phase

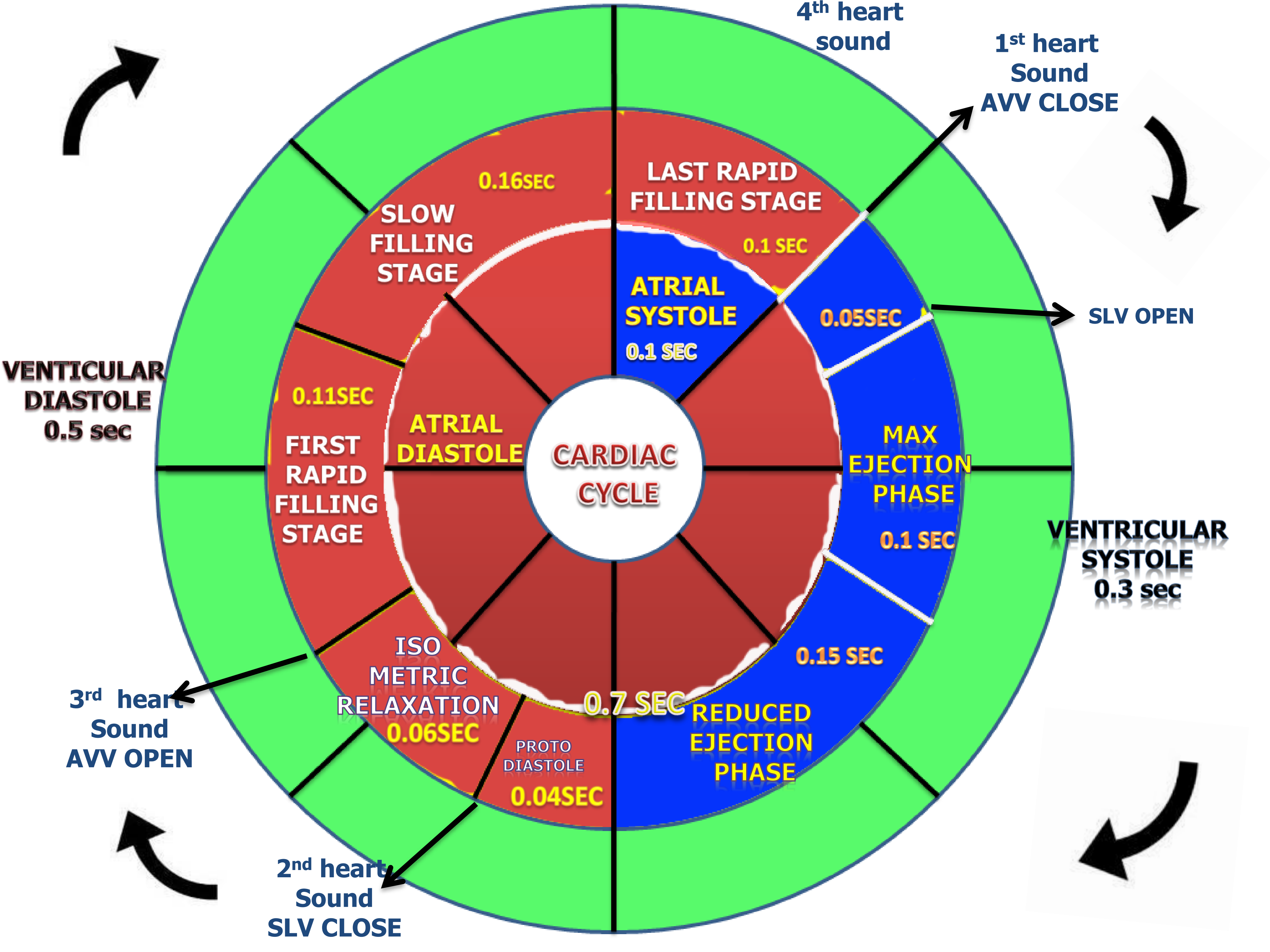
- rapid passive filling(0.11)
- reduced filling phase (0.19)
- last rapid filling phase (0.1sec)

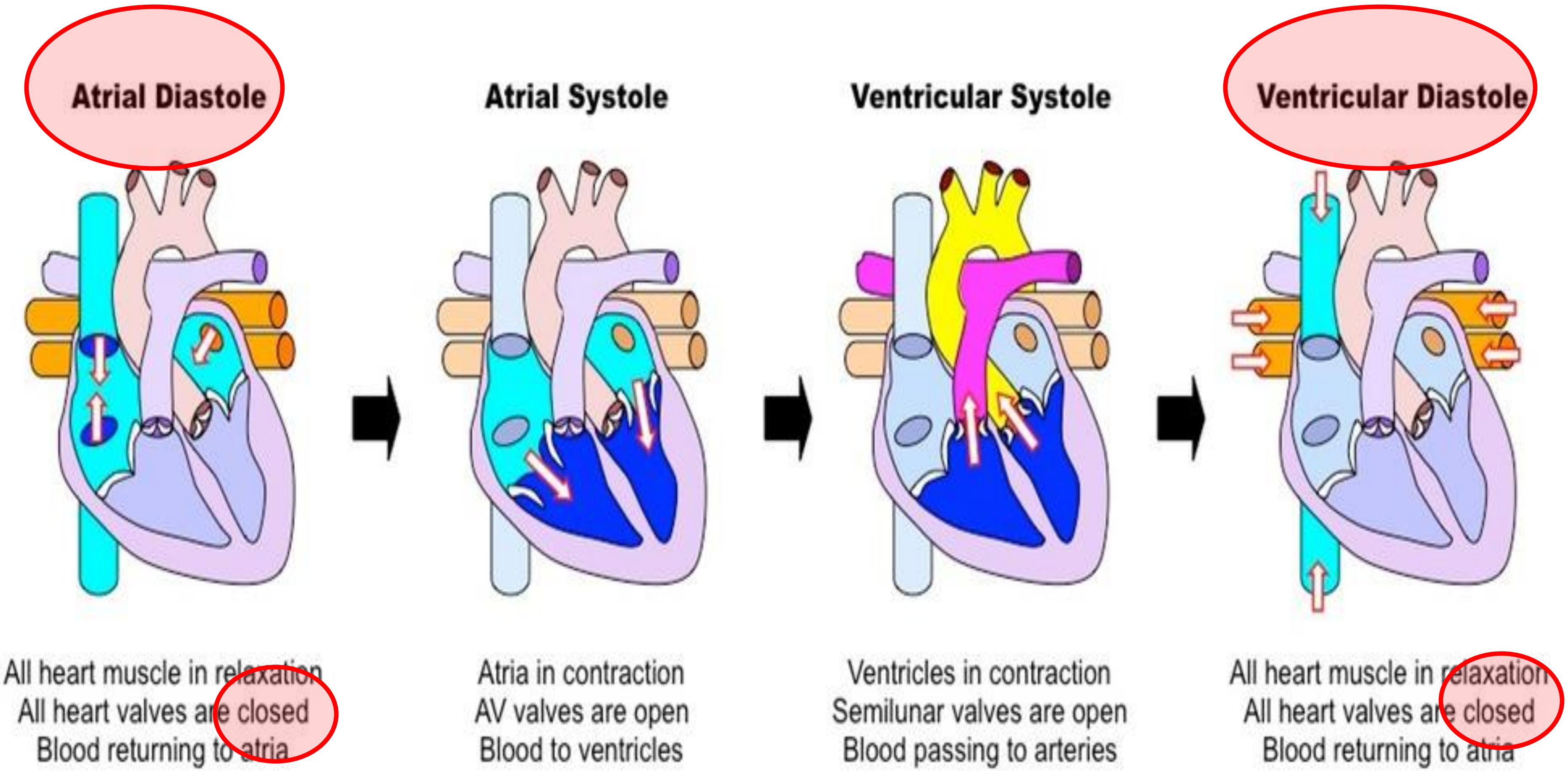
- **ATRIAL SYSTOLE:-----------------0.1 sec**
- **ATRIAL DIASTOLE:---------------0.7 sec**
- **VENTRICULAR SYSTOLE:--------0.3 sec**
- **VENTRICULAR DIASTOLE:-----0.5 sec**

Imagine 4 concentric circles inside each

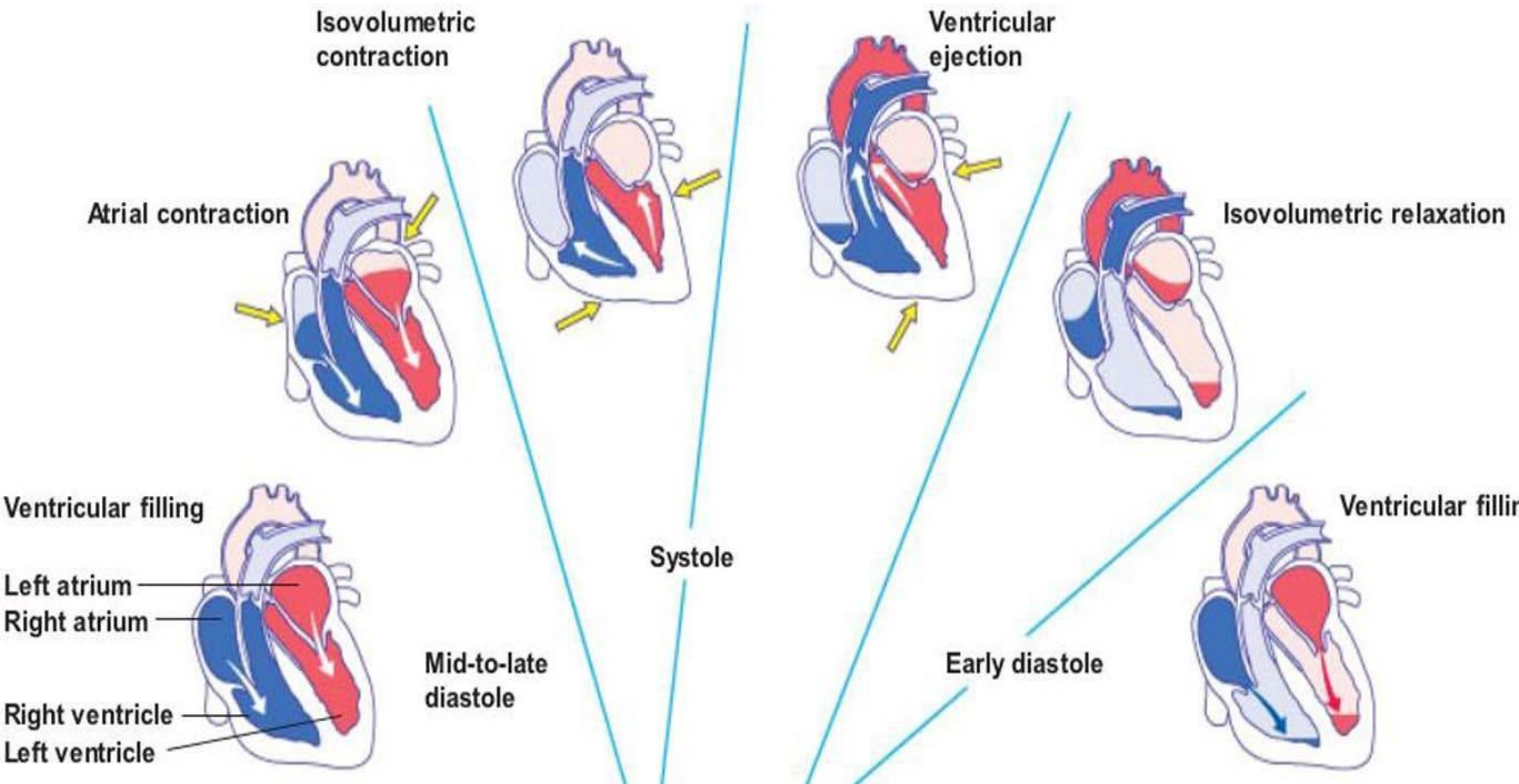
1. Title – cardiac cycle
2. Atrial cycle – systole + diastole
3. Ventricular cycle – systole + diastole
4. Heart sounds

Then,
See the diagram in the next slide


4th heart sound
1st heart Sound AVV CLOSE
SLV OPEN
VENTICULAR DIASTOLE 0.5 sec
VENTRICULAR SYSTOLE 0.3 sec
3rd heart Sound AVV OPEN
2nd heart Sound SLV CLOSE
0.16SEC
SLOW FILLING STAGE
LAST RAPID FILLING STAGE
0.1 SEC
ATRIAL SYSTOLE
0.1 SEC
0.05SEC
0.11SEC
FIRST RAPID FILLING STAGE
ATRIAL DIASTOLE
CARDIAC CYCLE
MAX EJECTION PHASE
0.1 SEC
ISO METRIC RELAXATION
0.06SEC
PROTO DIASTOLE
0.04SEC
0.7 SEC
0.15 SEC
REDUCED EJECTION PHASE



Valves open below, only when systole occurs above
Normally remain closed all the time, blood leaks down during diastole



Phase	1	2	3	4	1
Atrioventricular valves	Open		Closed		Open
Aortic and pulmonary (semilunar) valves	Closed		Open		Closed

Follow the white areas and shaded areas in table

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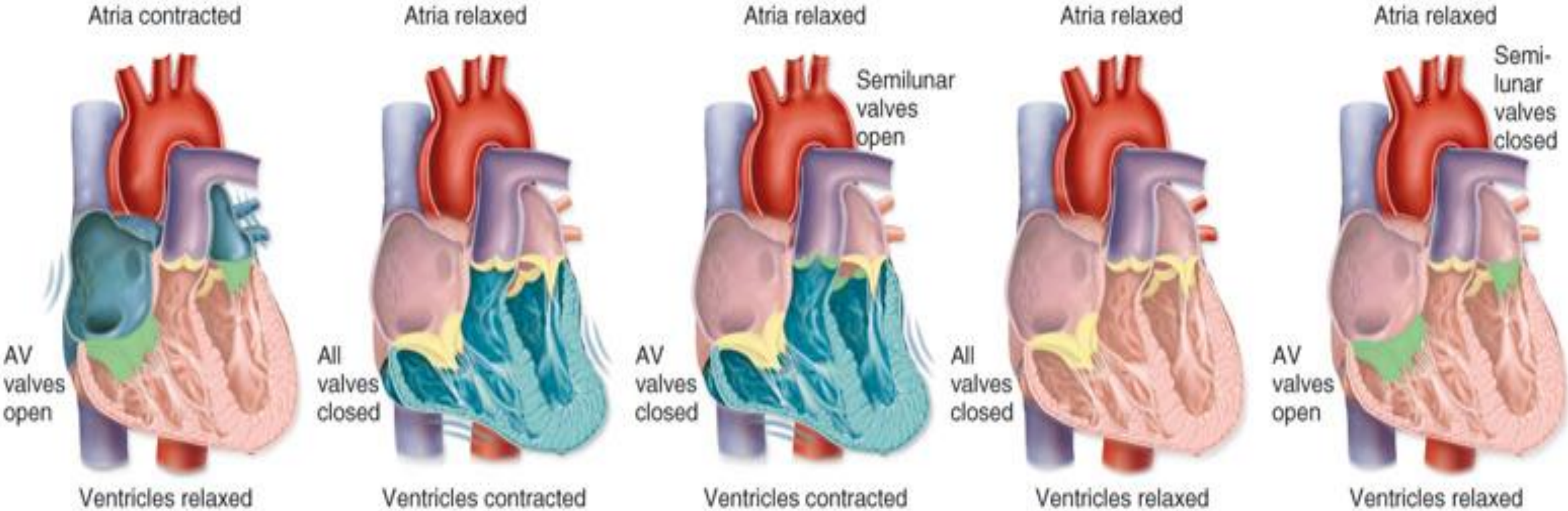
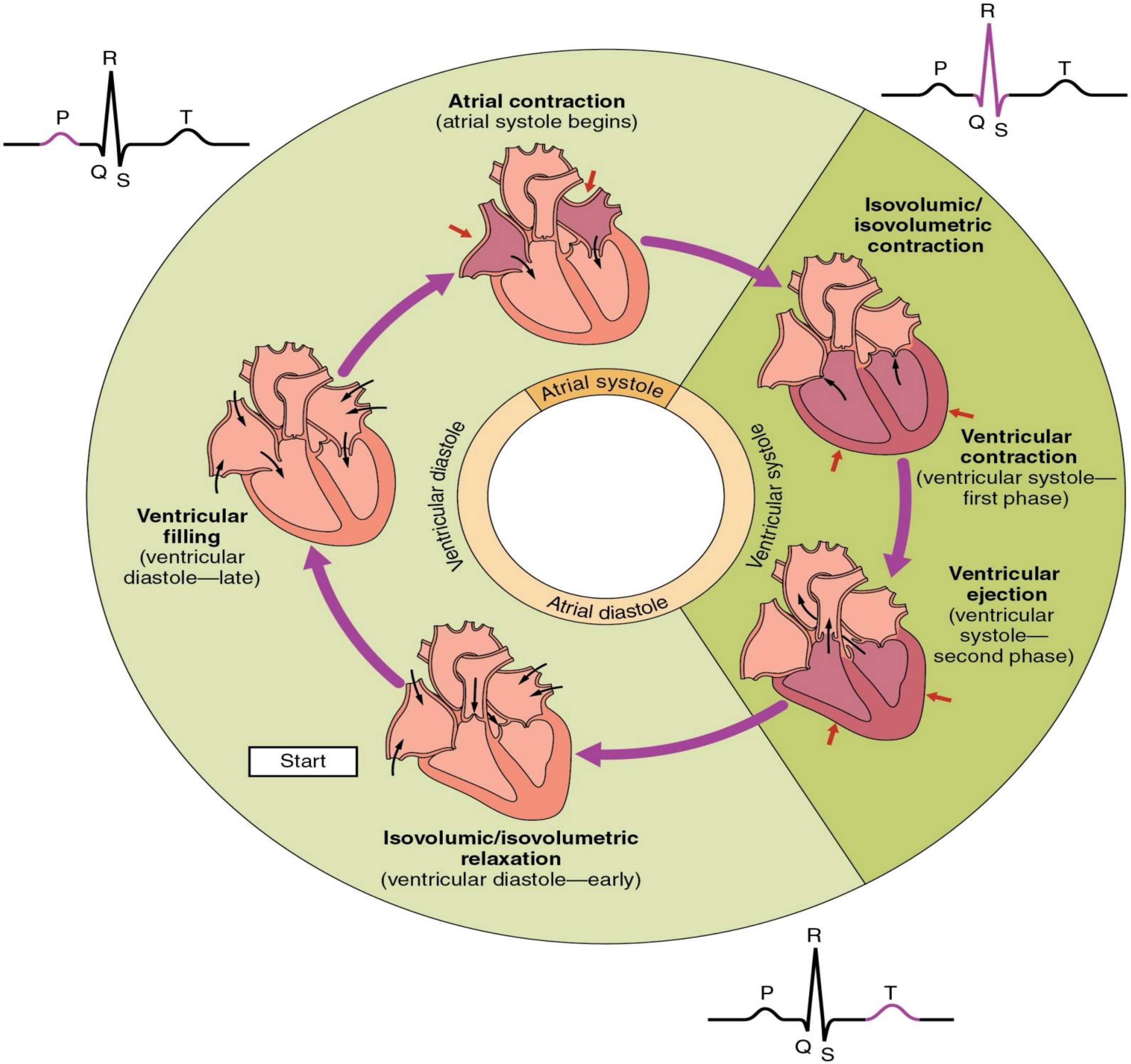
Structure \ Phase	Atrial systole	Early ventricular systole ⟷	Late ventricular systole	Early ventricular diastole ⟷	Late ventricular diastole
Atria	Contract	Relax		Relax	
Ventricles	Relax	Contract		Relax	
AV valves	Open	Closed		Open	
Semilunar valves	Closed	Open		Closed	



1. **Atrial systole** Atria contract, AV valves open, semilunar valves closed
2. **Early ventricular systole** Atria relax, ventricles contract, AV valves forced closed, semilunar valves still closed
3. **Late ventricular systole** Atria relax, ventricles contract, AV valves remain closed, semilunar valves forced open
4. **Early ventricular diastole** Atria and ventricles relax, AV valves and semilunar valves closed, atria begin passively filling with blood
5. **Late ventricular diastole** Atria and ventricles relax, atria passively fill with blood as AV valves open, semilunar valves closed


R
P
T
Q
S
Atrial contraction
(atrial systole begins)
Isovolumic/
isovolumetric
contraction
Atrial systole
Ventricular diastole
Ventricular systole
Ventricular
contraction
(ventricular systole—
first phase)
Ventricular
filling
(ventricular
diastole—late)
Ventricular
ejection
(ventricular
systole—
second phase)
Atrial diastole
Start
Isovolumic/isovolumetric
relaxation
(ventricular diastole—early)

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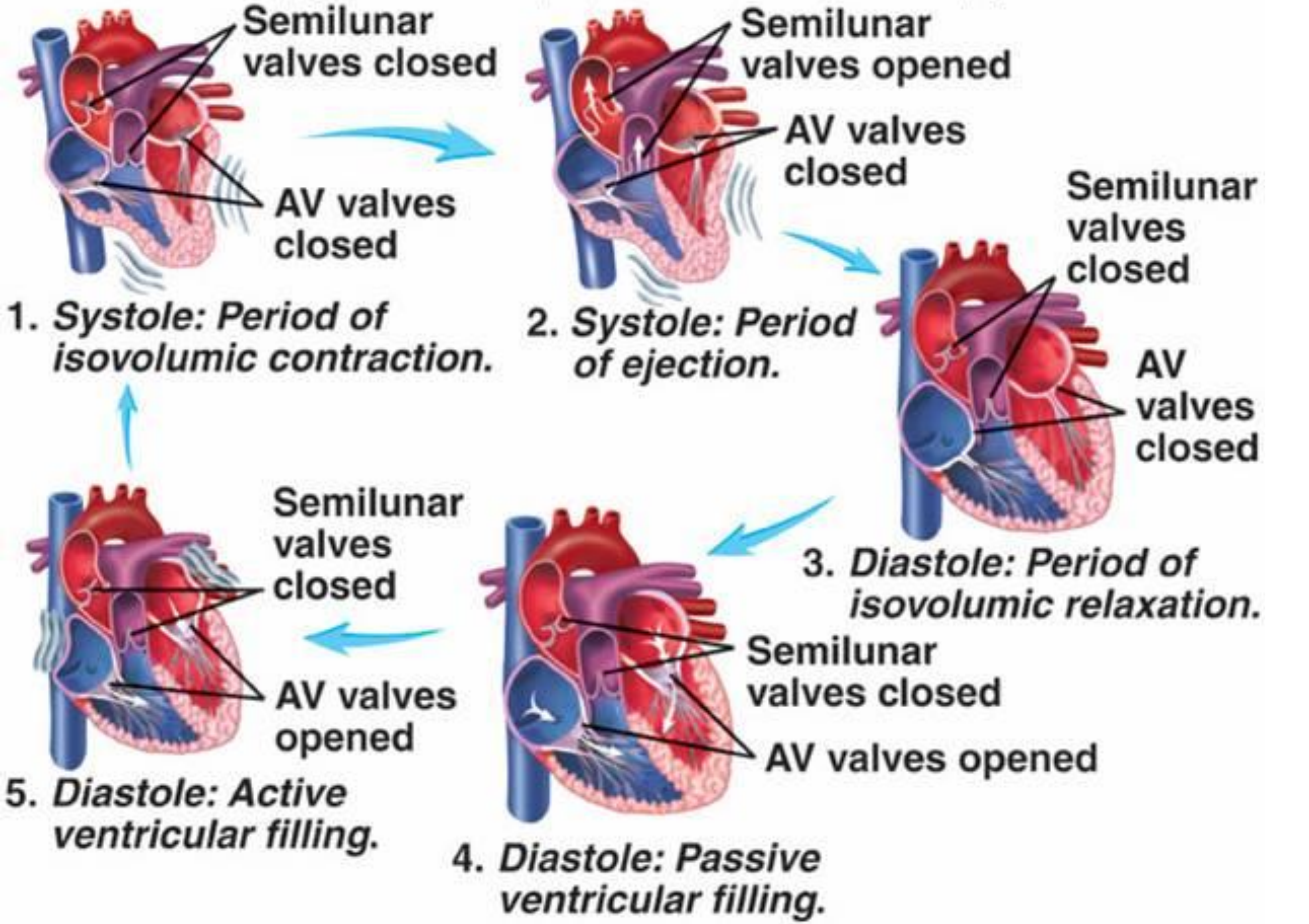
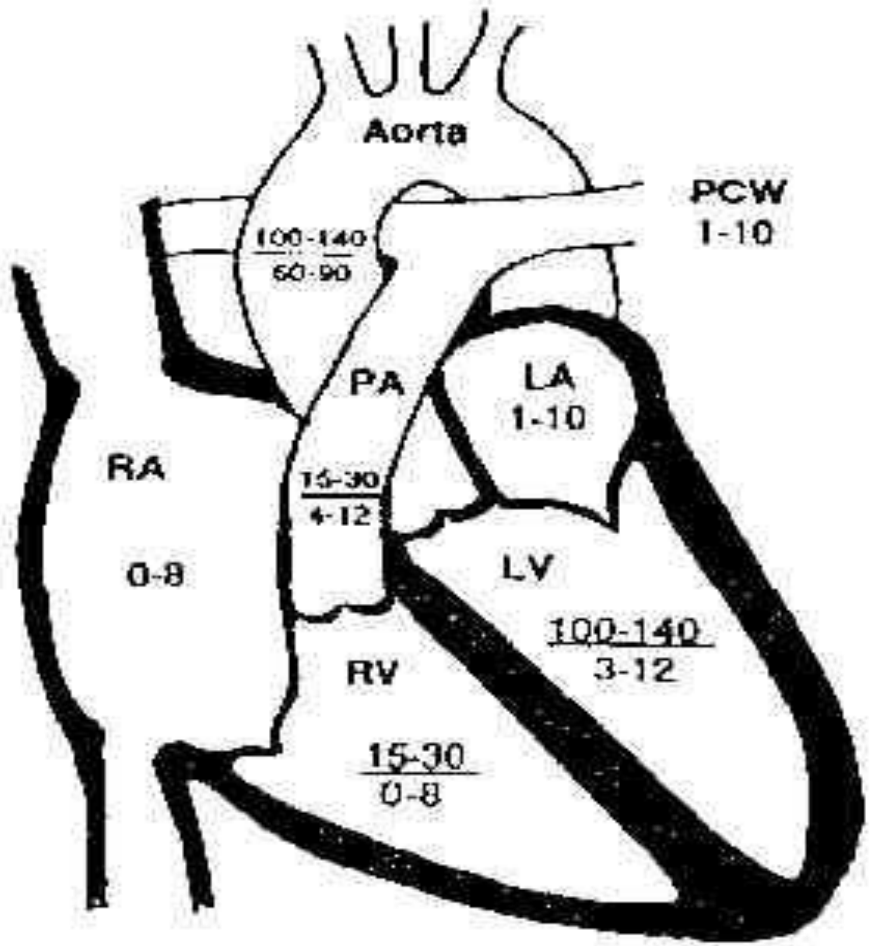
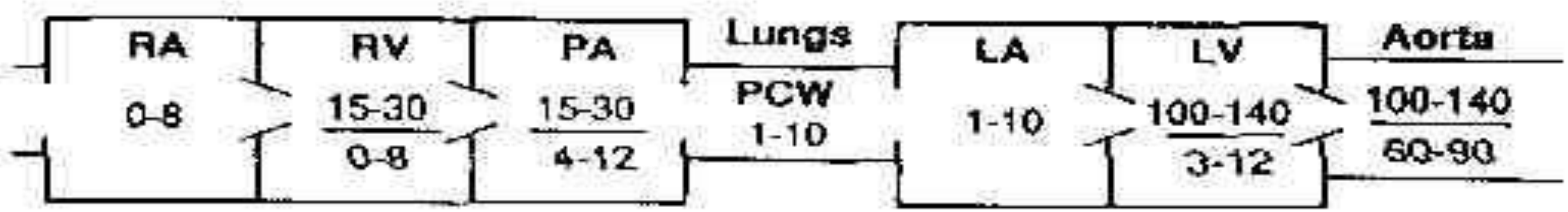



Aorta
PCW
1-10
100-140
60-90
PA
LA
1-10
RA
15-30
4-12
0-8
LV
100-140
3-12
RV
15-30
0-8


RA
0-8
RV
15-30
0-8
PA
15-30
4-12
Lungs
PCW
1-10
LA
1-10
LV
100-140
3-12
Aorta
100-140
60-90

Aortic pressure is measured by inserting a pressure-measuring catheter into the aorta from a peripheral artery,

Left ventricular pressure is obtained by placing a catheter inside the left ventricle and measuring changes
in intraventricular pressure as the heart beats.

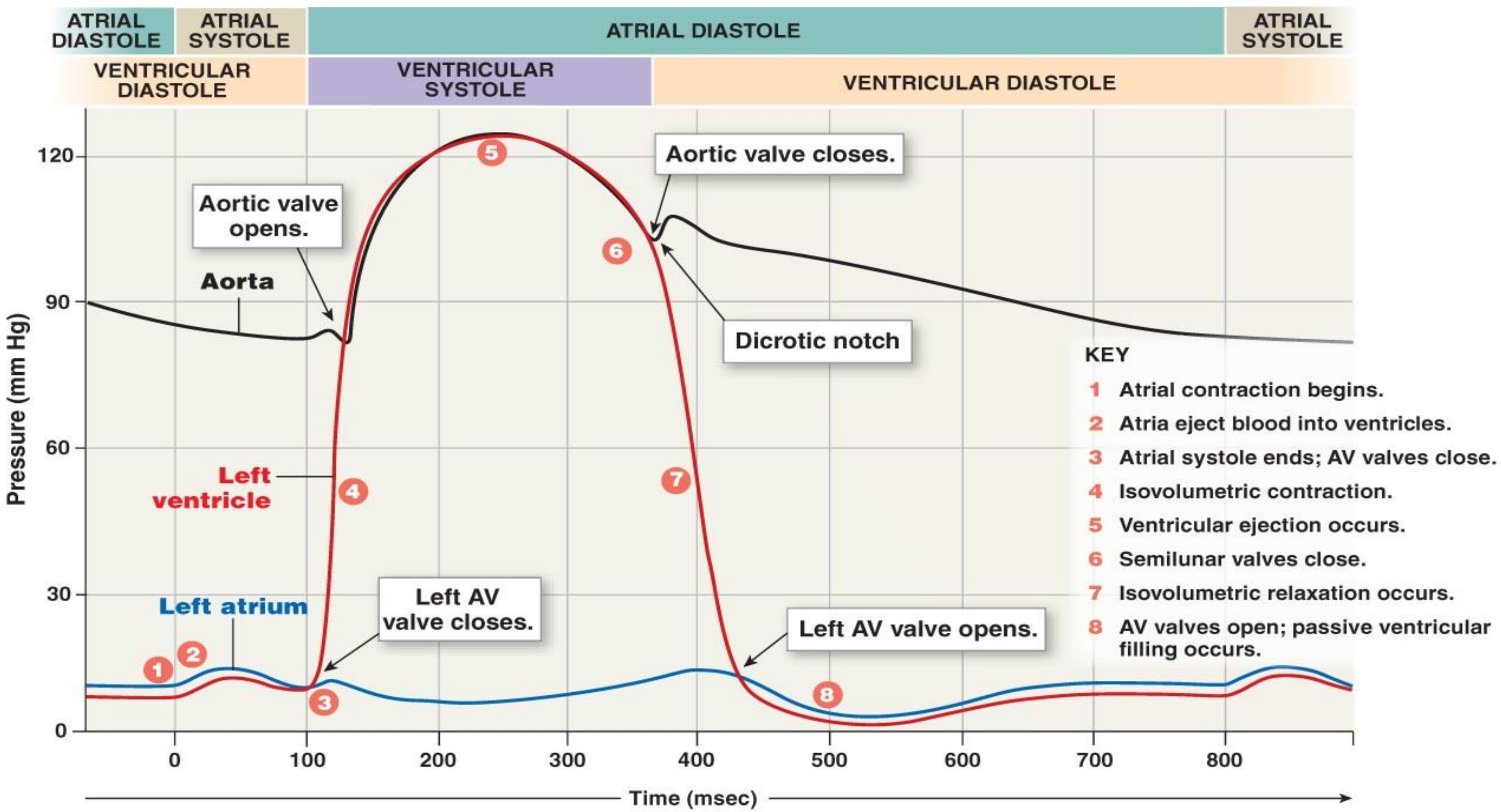
Left atrial pressure is not usually measured directly, except in investigational procedures; BUT, can be estimated by recording the pulmonary capillary wedge pressure.

Ventricular volume changes can be assessed in real time using echocardiography

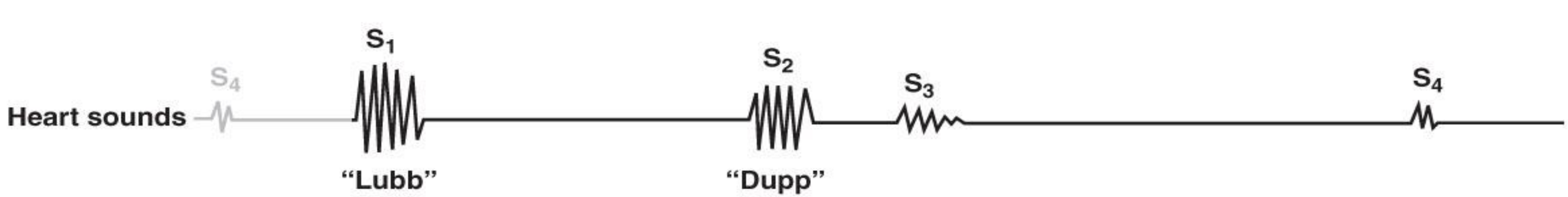
Pressures in Pulmonary Circulation		Pressures in Systemic Circulation	
Right ventricle	25/0 mm Hg	Left ventricle	120/0 mm Hg
Pulmonary artery	25/8 mm Hg	Aorta	120/80 mm Hg
Mean pulmonary arterial pressure	15 mm Hg	Mean arterial blood pressure	93 mm Hg
Capillary	7–9 mm Hg	Capillary: skeletal renal glomerular	30 mm Hg 45–50 mm Hg
Pulmonary venous	5 mm Hg	Peripheral veins	15 mm Hg
Left atrium	5–10 mm Hg	Right atrium (central venous)	0 mm Hg
Pressure gradient	15 – 5 = 10 mm Hg	Pressure gradient	93 – 0 = 93 mm Hg

Gradient = difference

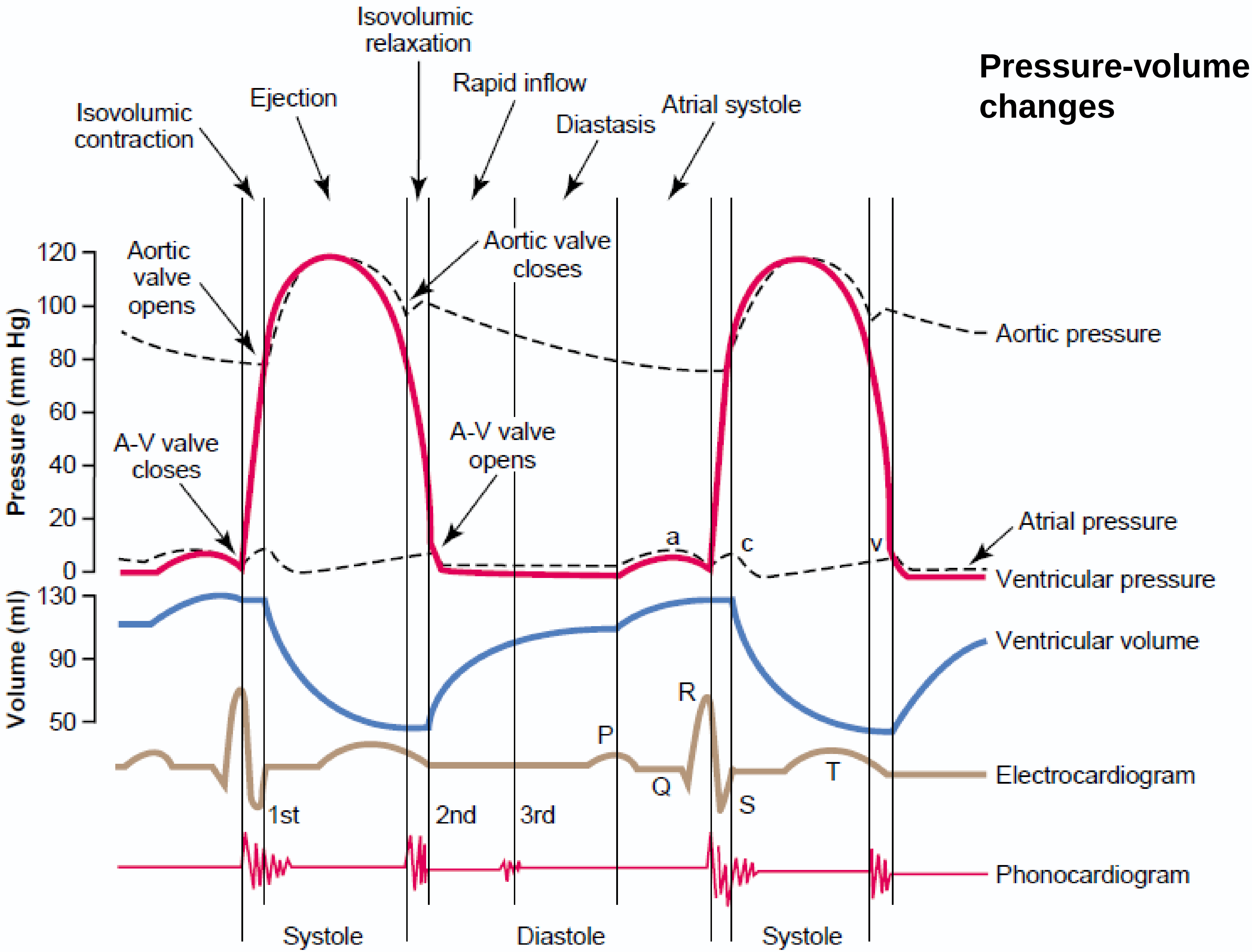
The pressure changes within the aorta, left atrium, and left ventricle during the cardiac cycle



The correspondence of the heart sounds with events during the cardiac cycle



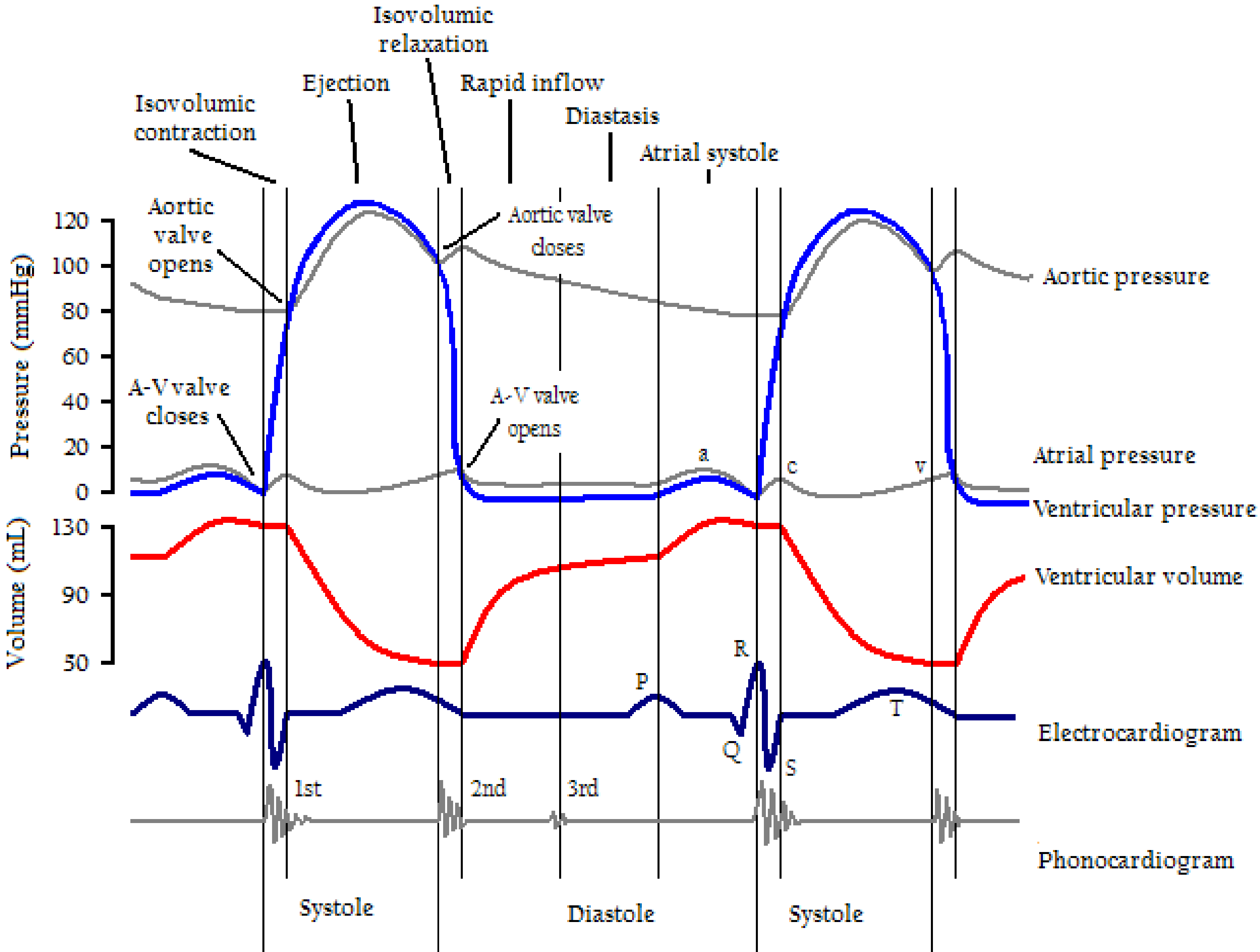
Pressure-volume changes



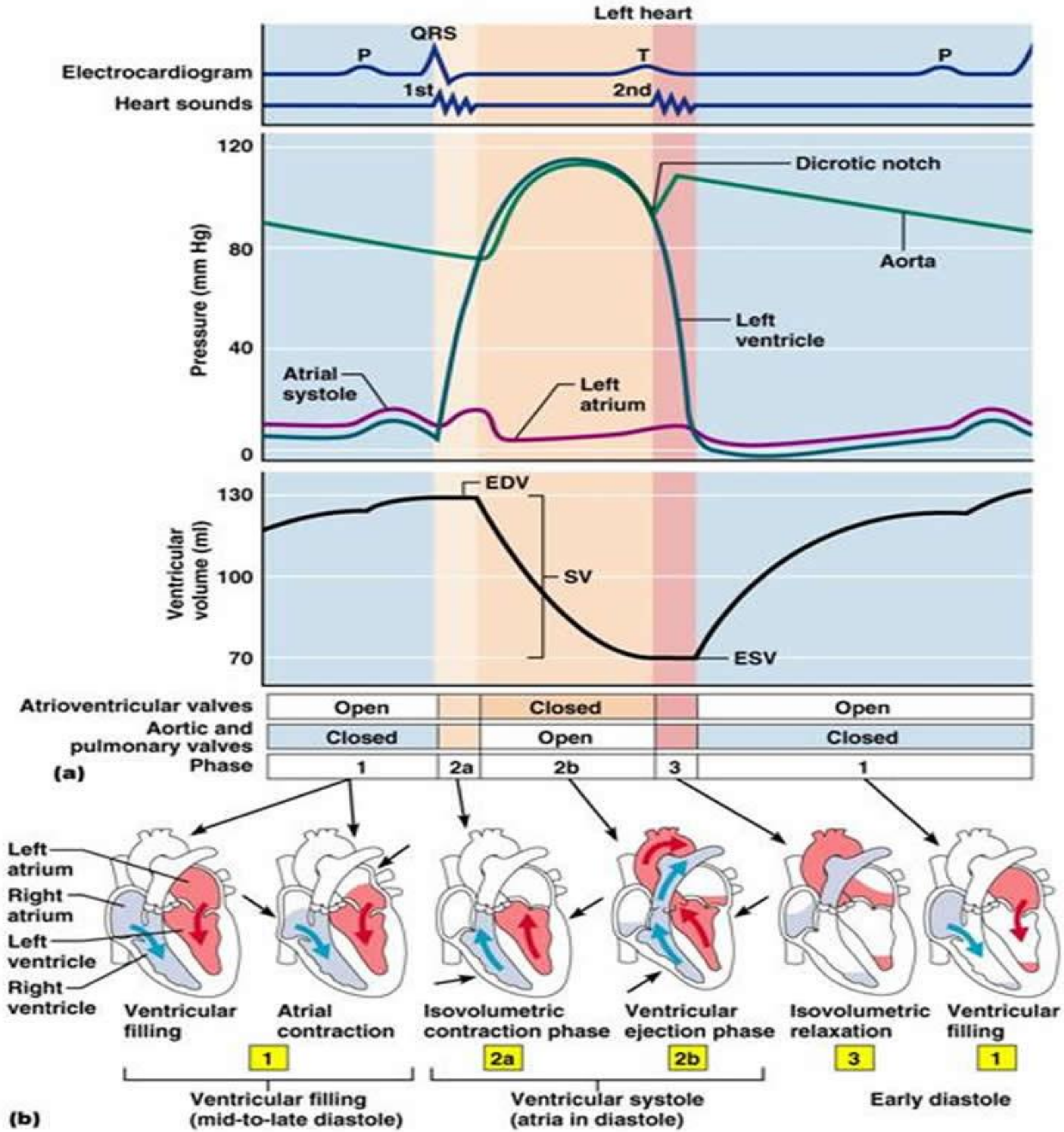
In the graph…….

Ventricular pressure graph = 0 to 120 mm
Ventricular volume graph = 120 to 60 ml

Atrial pressure = at the base line
Aorta pressure = at the apex

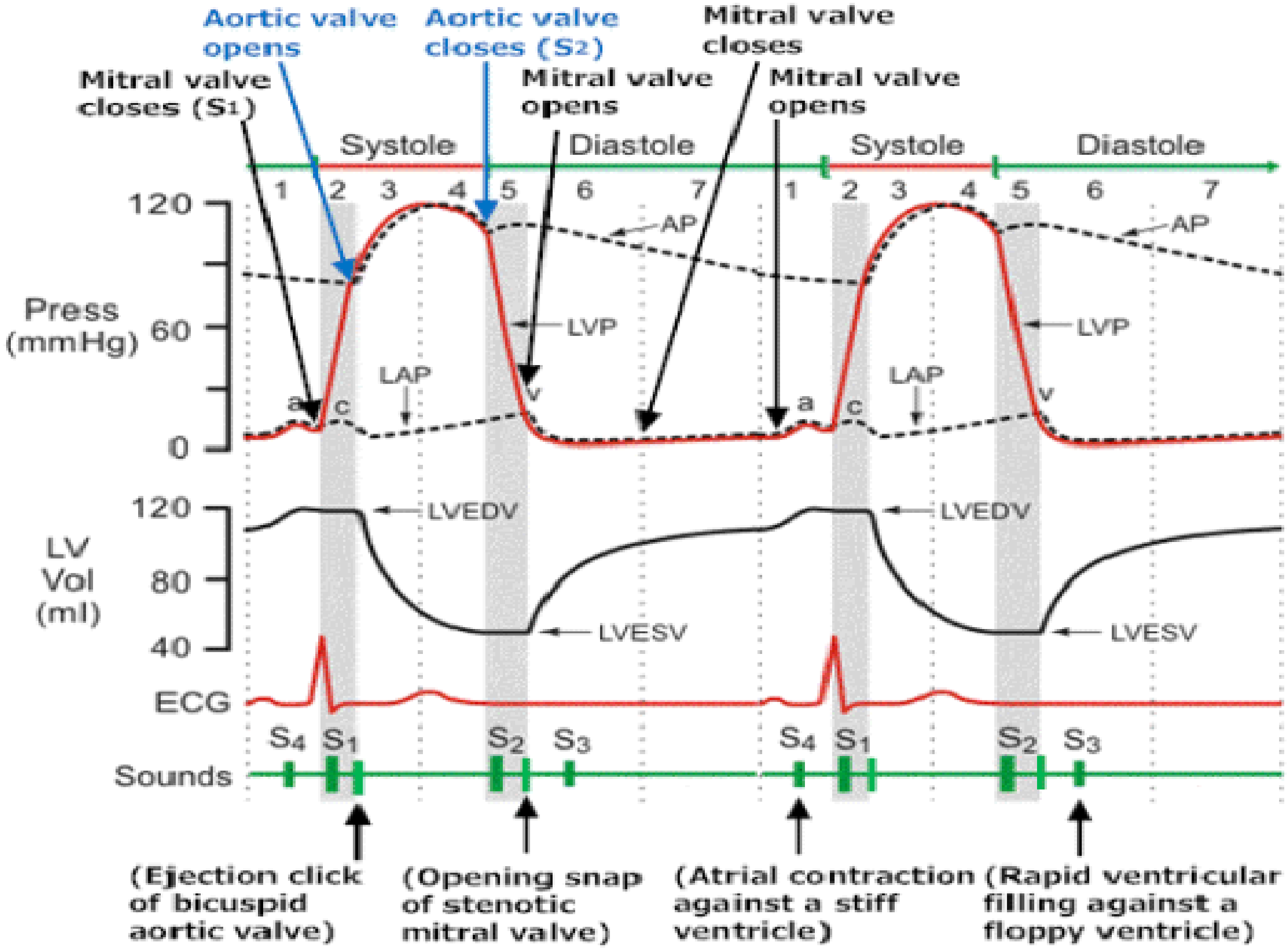


Isovolumic relaxation
Ejection
Rapid inflow
Isovolumic contraction
Diastasis
Atrial systole
Aortic valve opens
Aortic valve closes
A-V valve closes
A-V valve opens
Pressure (mmHg)
120
100
80
60
40
20
0
Volume (mL)
130
90
50
a
c
v
Aortic pressure
Atrial pressure
Ventricular pressure
Ventricular volume
R
P
Q
S
T
Electrocardiogram
1st
2nd
3rd
Phonocardiogram
Systole
Diastole
Systole

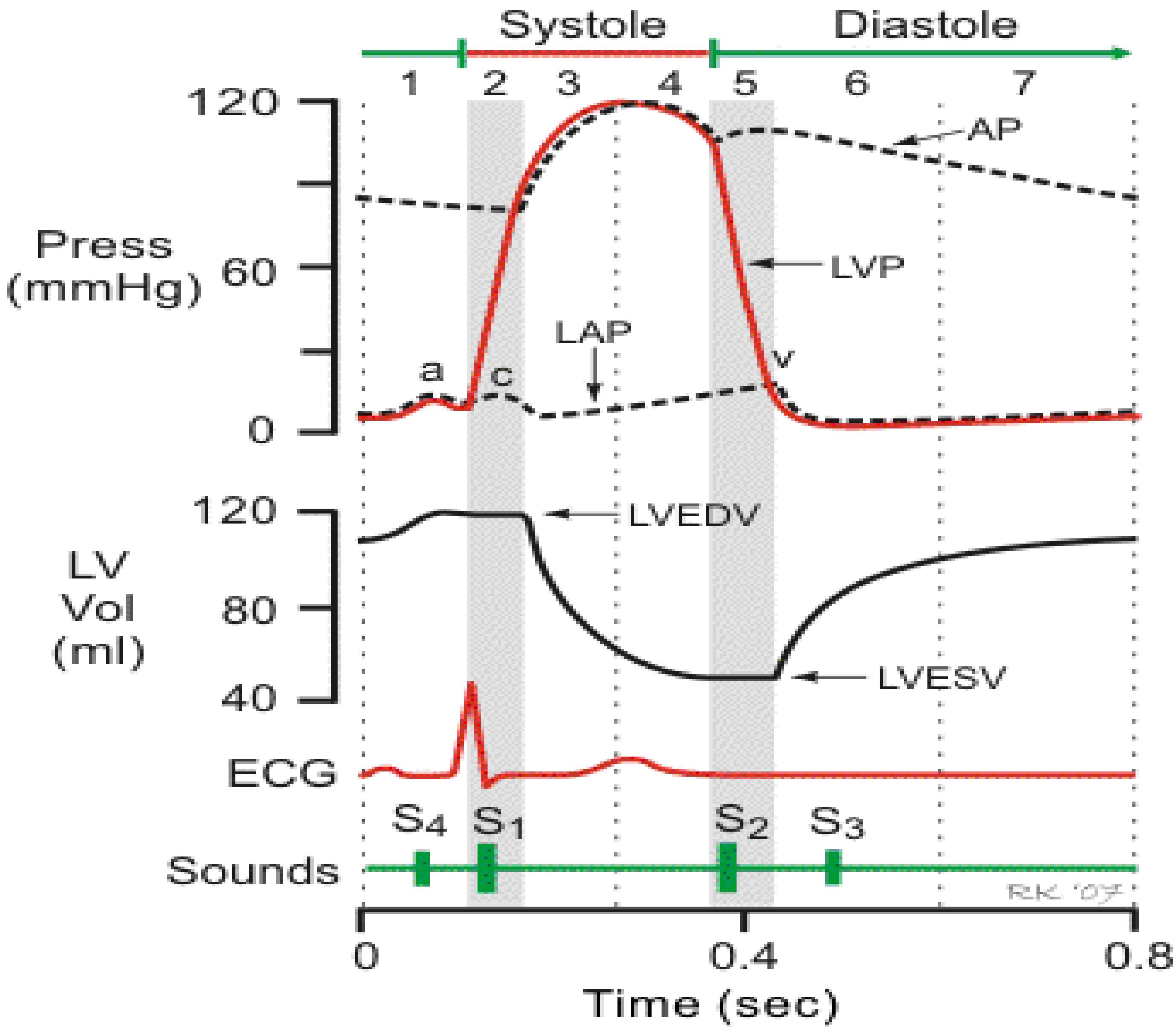


Left heart
QRS
P
T
P
Electrocardiogram
1st
2nd
Heart sounds
120
80
40
0
Pressure (mm Hg)
Dicrotic notch
Aorta
Left ventricle
Atrial systole
Left atrium
EDV
130
100
70
Ventricular volume (ml)
SV
ESV
Atrioventricular valves
Open
Closed
Open
Aortic and pulmonary valves
Closed
Open
Closed
Phase
1
2a
2b
3
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)

The Cardiac Cycle



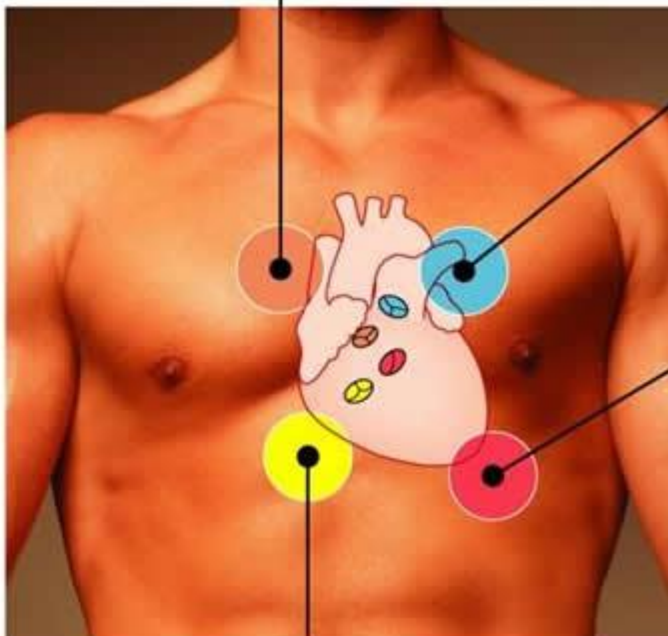
1 -> Atrial Contraction --> Mitral Valve Opens
2 -> Isovolumic Ventricular Contraction --> Mitral Valve Closes
---> Aortic Valve Opens
3 -> Rapid Ejection
4 -> Reduced Ejection
5 -> Isovolumic Ventricular Relaxation --> Aortic Valve Closes
---> Mitral Valve Opens
6 -> Rapid Ventricular Filling
7 -> Diastasis ---> Mitral Valve Closes
1 -> Atrial Contraction --> Mitral Valve Opens



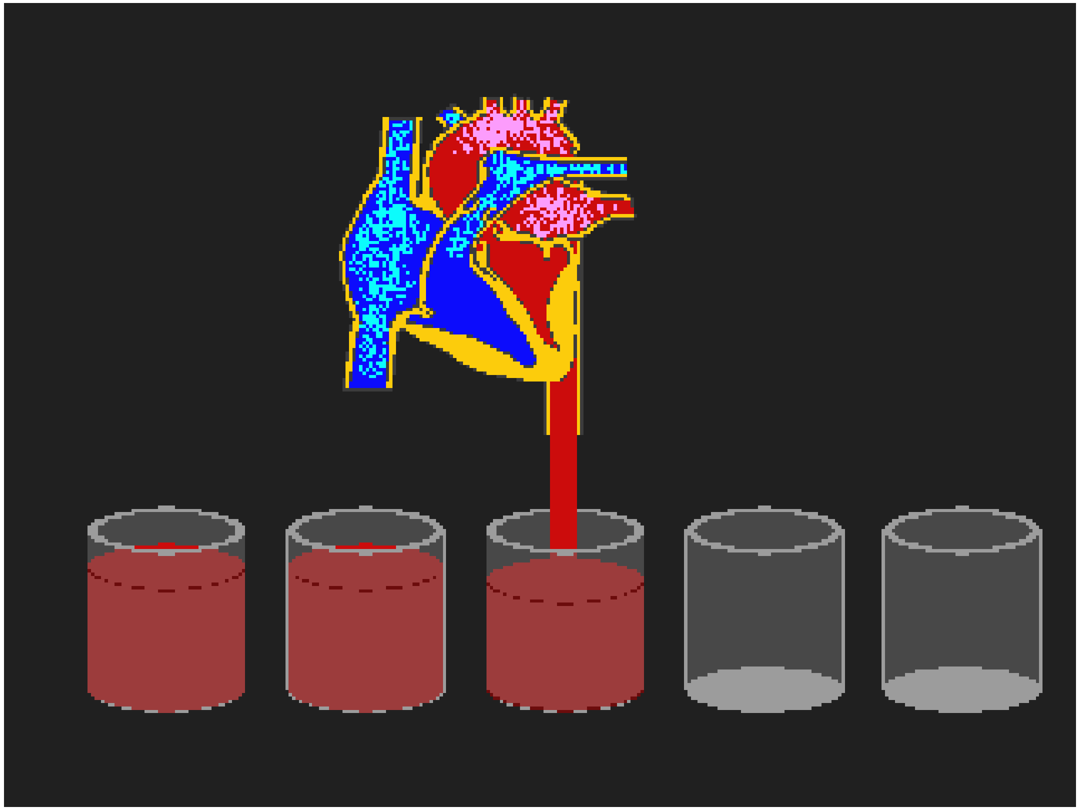
Systole
Diastole
1
2
3
4
5
6
7
120
60
0
Press (mmHg)
AP
LVP
LAP
a
c
v
120
80
40
LV Vol (ml)
LVEDV
LVESV
ECG
S_4
S_1
S_2
S_3
Sounds
0
0.4
0.8
Time (sec)

Pressure – Volume relationship in Left ventricle

IVC = volume is constant, but pressure rises
IVR = volume is constant, but pressure decreases


Sounds of aortic valve are heard in 2nd intercostal space at right sternal margin.
Sounds of pulmonary valve are heard in 2nd intercostal space at left sternal margin.
Sounds of mitral valve are heard over heart apex, in 5th intercostal space in line with middle of clavicle.
Sounds of tricuspid valve are typically heard in right sternal margin of 5th intercostal space; variations include over sternum or over left sternal margin in 5th intercostal space.

AP
TM



Cardiac Output

Cardiac Output

- **CO** = volume of blood ejected from left (or right) ventricle into aorta (or pulmonary trunk) <u>each minute</u>
- <u>CO = stroke volume (SV) x heart rate (HR)</u>
- In typical resting male

  <u>5.25L/min</u> = 70mL/beat x 75 beats/min
- <u>Entire blood volume flows</u> through pulmonary and systemic circuits <u>each minute</u>
- Cardiac reserve – difference between maximum CO and CO at rest
  - Average cardiac reserve 4-5 times resting value

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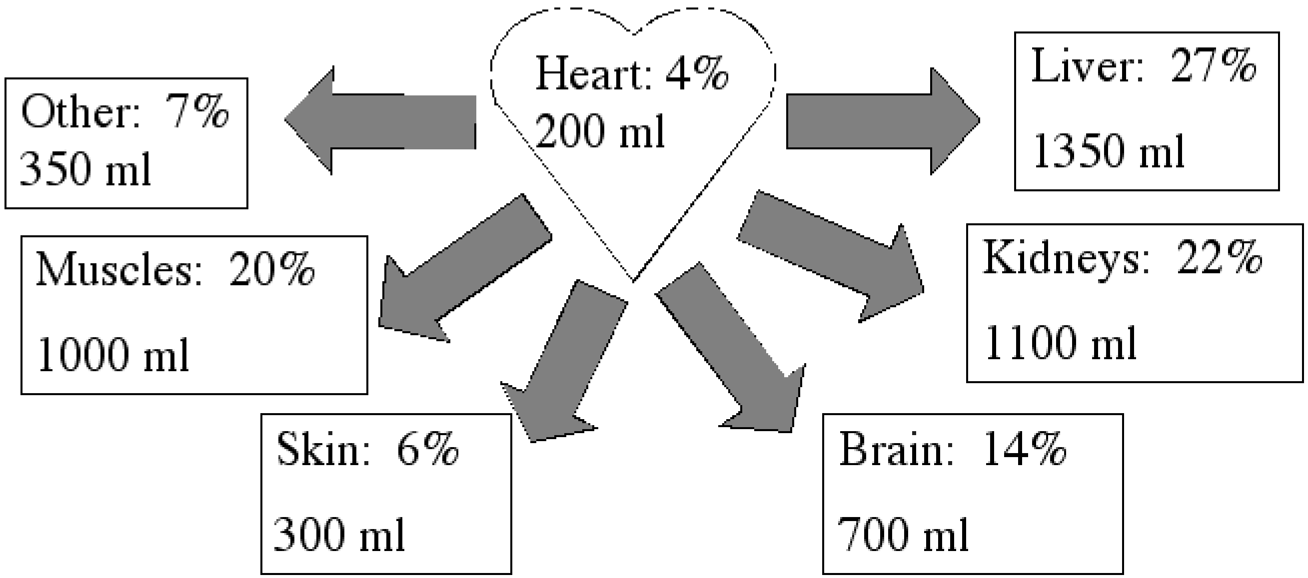
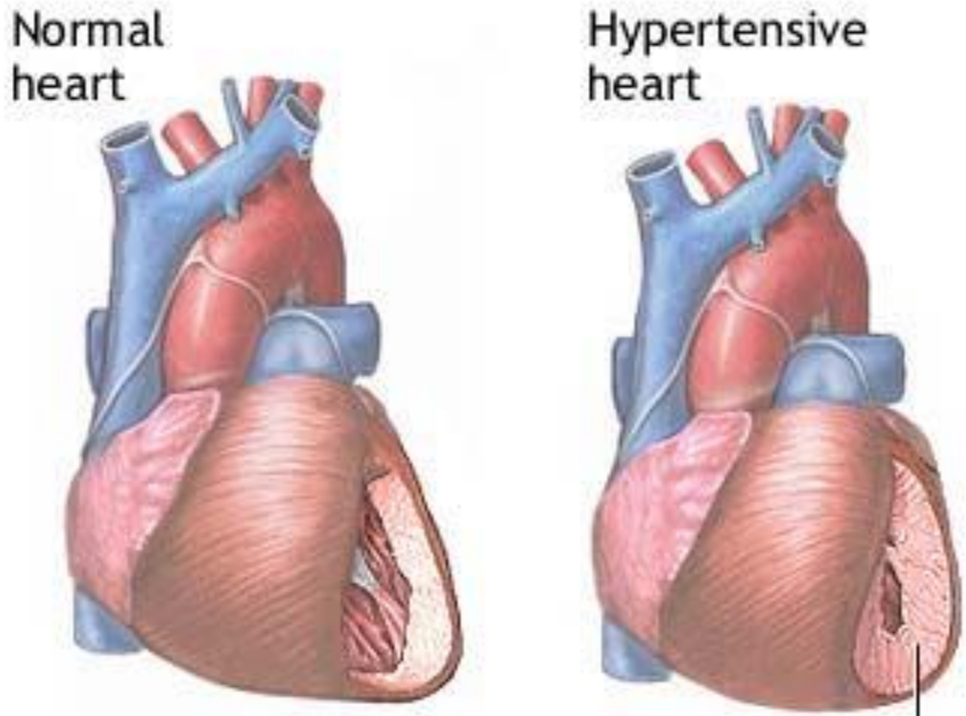
Terminology

- **EDV** – amount of blood remain at the end of diastole – 130 ml
- **ESV** - amount of blood remain at the end of systole – 50 ml
- **SV** – amount of blood ejected by each ventricle per beat (EDV – ESV) – 80 ml
- **CO** - amount of blood ejected by each ventricle per minute – SV x HR – 5 lit.
- **EF** - the % proportion of blood in the heart pumped out of the heart during a single contraction – SV/EDV X 100 = 60 %

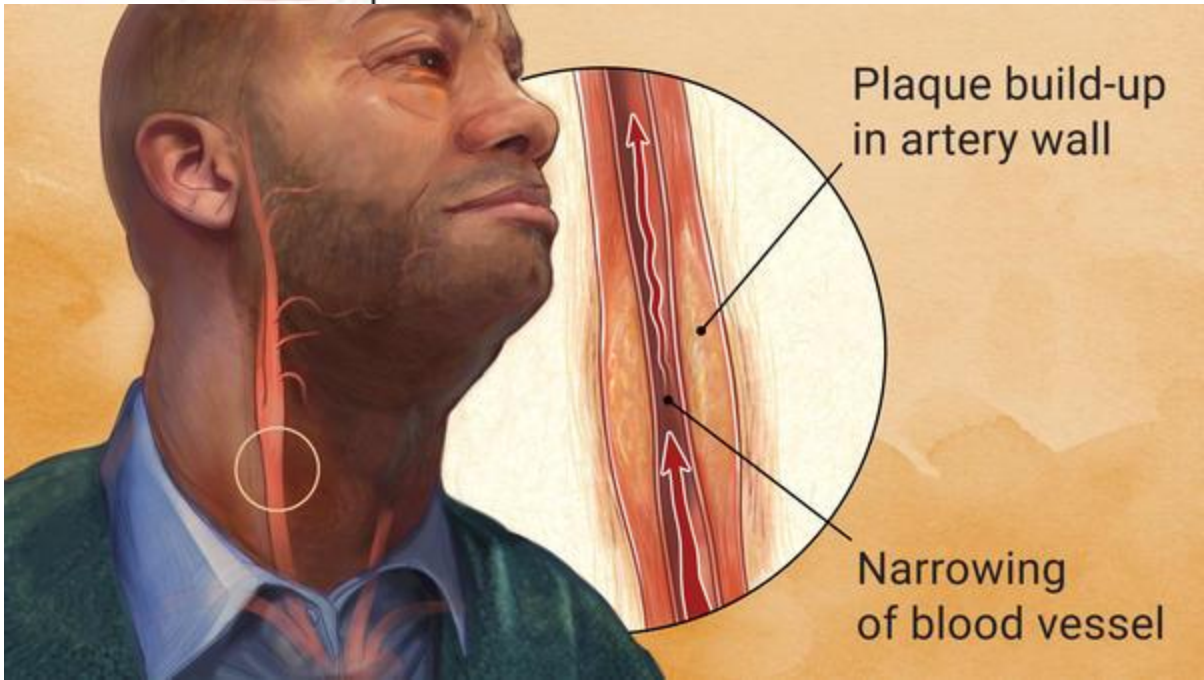
Cardiac Output at Rest = 5 L/min





Hypertension
> 140/90

Atherosclerosis



Factors

- EDV - affected by
  – Venous return - vol. of blood returning to heart
  – Preload – amount ventricles are stretched by blood (=EDV)
- ESV - affected by
  – Contractility – myocardial contractile force due to factors other than EDV
  – Afterload – back pressure exerted by blood in the large arteries (Aorta) leaving the heart

Extrinsic Factors Influencing Stroke Volume

- Contractility is the increase in contractile strength, independent of stretch and EDV
- Referred to as extrinsic since the influencing factor is from some *external source outside of heart*
- Increase in contractility comes from:
  - Increased sympathetic stimuli
  - Certain hormones
  - Ca^{2+} and some drugs
- Agents/factors that decrease contractility include:
  - Acidosis H+
  - Increased extracellular K^+
  - Calcium channel blockers – Diltiazime, Verapamil

Effects of Autonomic Activity on Contractility

- Sympathetic stimulation
  - Release norepinephrine from symp. postganglionic fiber
  - Also, EP and NE from adrenal medulla
  - Have positive ionotropic effect
  - Ventricles contract more forcefully, increasing SV, increasing ejection fraction and decreasing ESV
- Parasympathetic stimulation via Vagus Nerve –CN-X
  - Releases ACh
  - Has a negative inotropic effect
    - Hyperpolarization and inhibition
  - Force of contractions is reduced, ejection fraction decreased

Contractility and Norepinephrine

- <u>Sympathetic stimulation</u> releases <u>norepinephrine</u> and initiates a <u>cyclic AMP</u> 2nd-messenger system

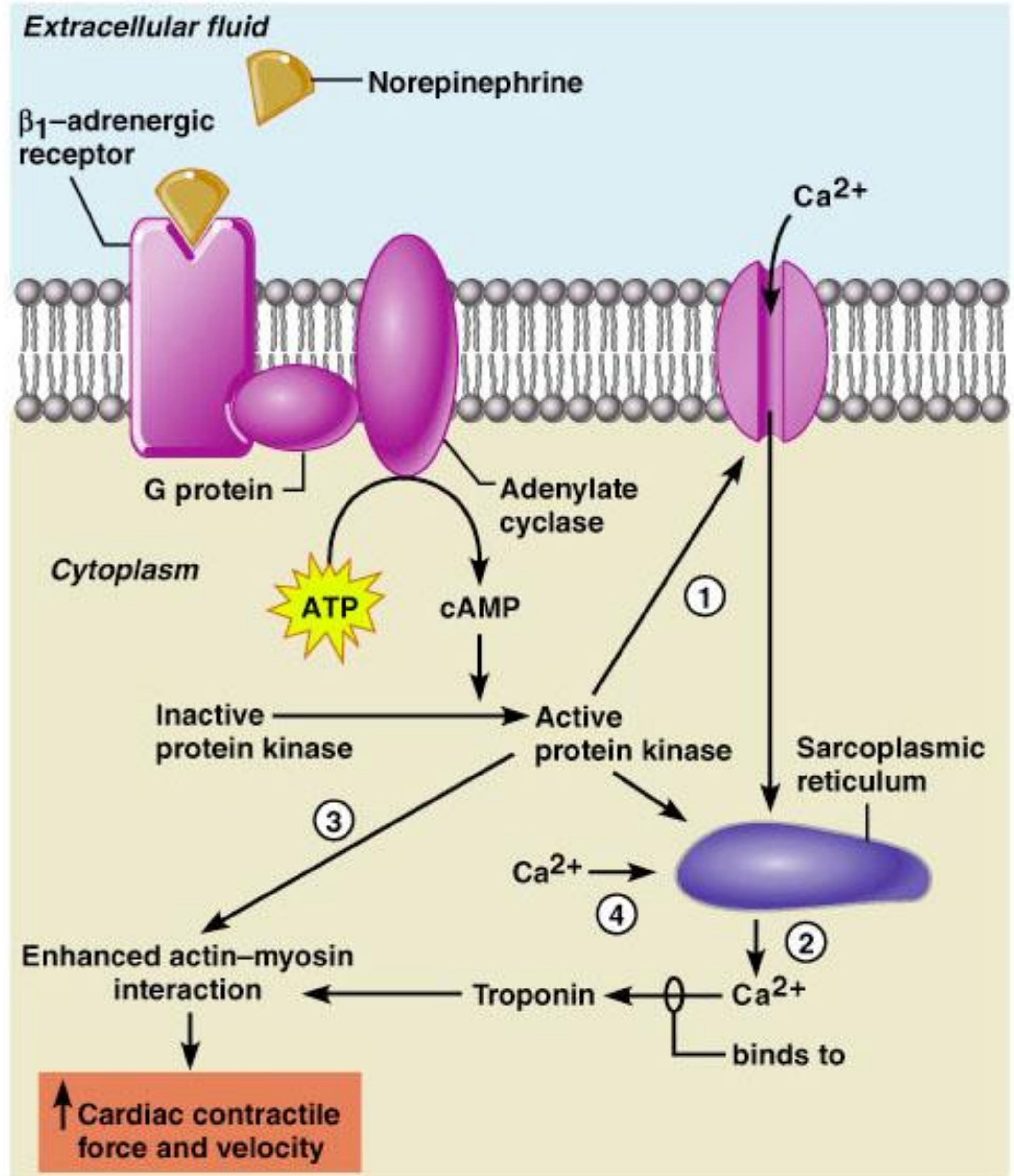


Figure 18.22

Preload and Afterload direction

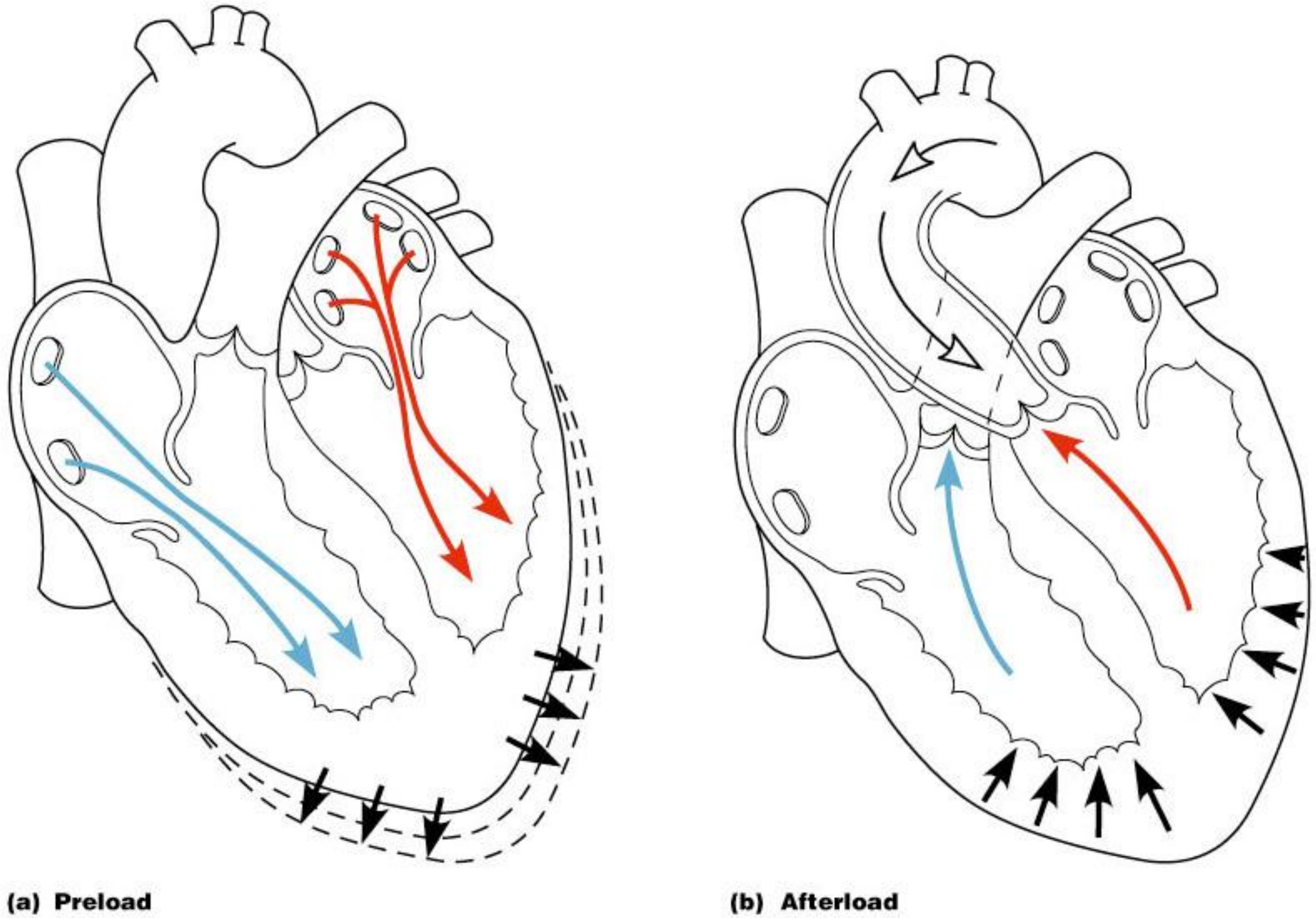


Figure 18.21

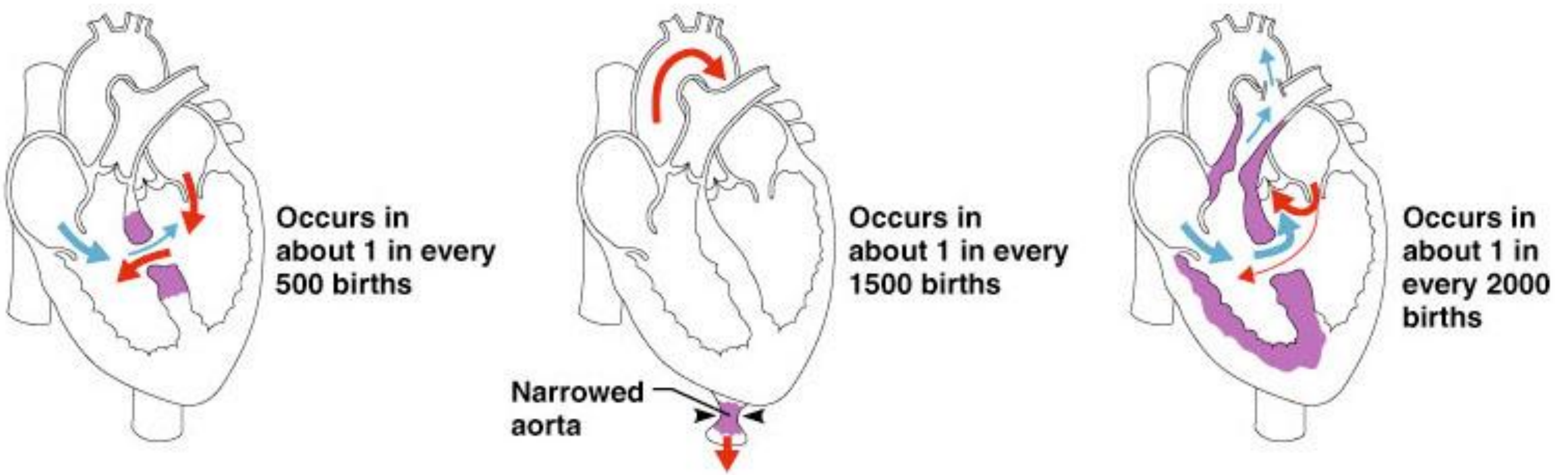
Effects of Hormones on Contractility

- Epi, NE, and Thyroxine all have positive ionotropic effects and thus ↑contractility
- Digitalis elevates intracellular Ca^{++} concentrations by interfering with its removal from sarcoplasm of cardiac cells= contractility
- Hence given in heart failure/CCF
- Beta-blockers (*propanolol, timolol*) block beta-receptors and prevent sympathetic stimulation of heart (neg. chronotropic effect) reduce BP

Exercise and Cardiac Output

- Proprioceptors
  - HR ↑ at beginning of exercise due to signals from joints, muscles
- Venous return
  - muscular activity ↑ venous return causes ↑ SV
- ↑ HR and ↑ SV cause ↑CO
- Exercise <u>produces ventricular hypertrophy</u>
  - ↑ SV allows heart to beat more <u>slowly at rest</u>
  - <u>↑ cardiac reserve</u>

Examples of Congenital Heart Defects in new born babies



(a) Ventricular septal defect. The superior part of the inter-ventricular septum fails to form; thus, blood mixes between the two ventricles, but because the left ventricle is stronger, more blood is shunted from left to right.

(b) Coarctation of the aorta. A part of the aorta is narrowed, increasing the workload on the left ventricle.

(c) Tetralogy of Fallot. Multiple defects (*tetra* = four): Pulmonary trunk too narrow and pulmonary valve stenosed, resulting in a hypertrophied right ventricle; ventricular septal defect; aorta opens from both ventricles; wall of right ventricle thickened from overwork.

Figure 18.25

- https://www.youtube.com/watch?v=kcWNjt77uHc
- https://www.youtube.com/watch?v=MKRGgX5rYbY

Heart Rate (HR)

- In normal conditions **pulse rate represents HR**.
- As SA Node is the primary pacemaker, heart rate is the rate of discharge of SA Node.
- Normal heart rate = **60- 100 / min**.
- < 60 = **Bradycardia**
- > 100 = **Tachycardia**.

Physiological variation of HR

i. **Age** – HR more in infants and children
ii. **Gender** – Females > males.
iii. **Diurnal variation** – HR more in day than night
iv. **Respiration** – HR more during Inspiration & less during Expiration (**Sinus Arrhythmia**).
v. **Body temp. –**
vi. **Food intake**
vii. **Posture-**
viii. **Exercise** -

Regulation of Heart Beat

– Cardiac output depends on heart rate and stroke volume
– Adjustments in heart rate important in short-term control of cardiac output and blood pressure
– Autonomic nervous system and epinephrine/ norepinephrine most important

•Chemical regulation of heart rate

- Hormones
  - (Epinephrine and norepinephrine increase heart rate and contractility)
  - Thyroid hormones also increase heart rate and contractility
- Cations
  - Ionic imbalance can compromise pumping effectiveness
  - Relative concentration of K^+, Ca^{2+} and Na^+ important.
  - Low potassium levels - stable arrhythmias, while high potassium levels - lethal arrhythmias- death
  - Treatment - correcting the imbalance, preventing blood clots and treating of symptoms.

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- hypoxia ↑es HR which is partly mediated by release of catecholamines from adrenal medulla.
- Hypercapnia & acidosis ↓es HR.
- LINKS
- https://www.youtube.com/watch?v=RHxfP5rm3Ks
- https://www.youtube.com/watch?v=CI_BZuFSkyk

Increased end diastolic volume (stretches the heart)

Increased PRELOAD

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

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

Increased CONTRACTILITY

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

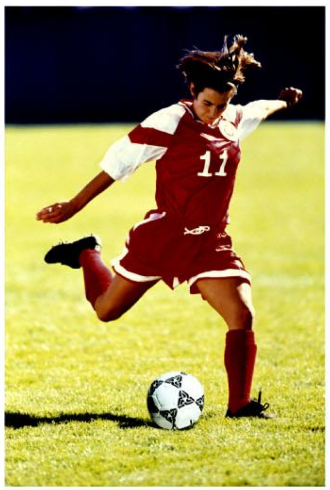
Decreased arterial blood pressure during diastole

Decreased AFTERLOAD

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

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

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

CHEMICALS

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

OTHER FACTORS

Figure 20.17 Tortora - PAP 12/e

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Examples of Congenital Heart Defects in new born babies

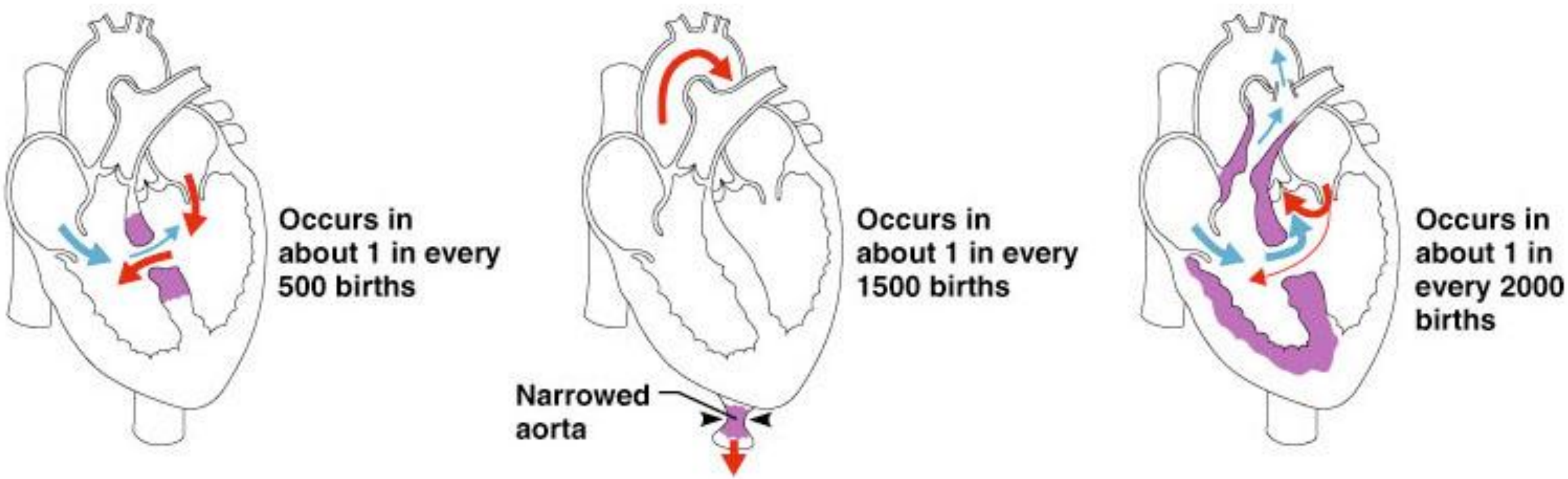

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Figure 18.25

Critical concepts

- Frank starling law. Define preload
- HR regulation - factors
- Cardiac cycle – events list
- Cardiac contractility is increased under the influence of hormone Norepinephrine. Elucidate.

- https://www.youtube.com/watch?v=vFRkSB46bl8
- https://www.youtube.com/watch?v=5tUWOF6wEnk
- https://www.youtube.com/watch?v=QllguanpKic