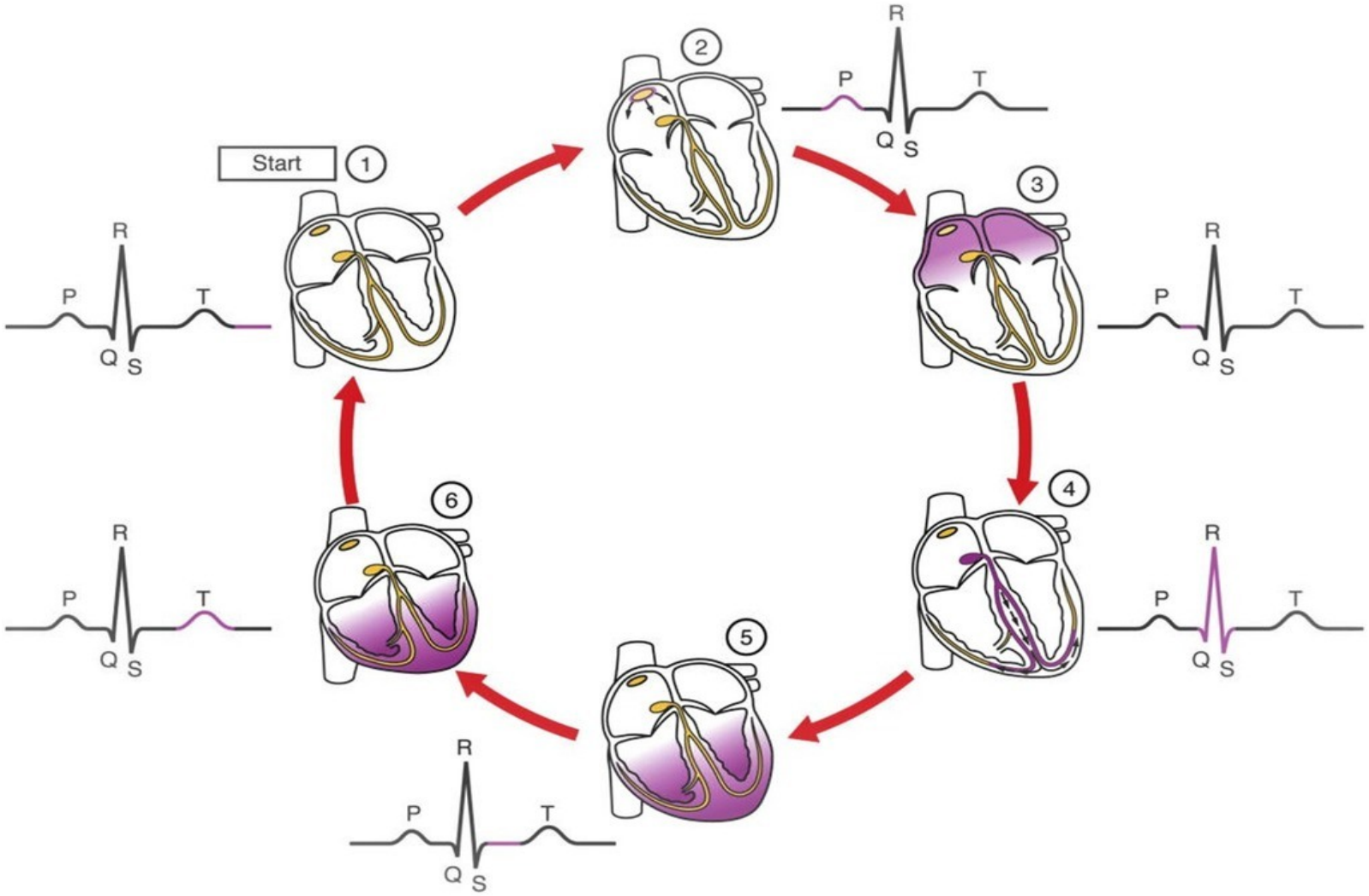
The Cardiac Cycle



Learning Objectives

- **Describe the Cardiac Cycle**

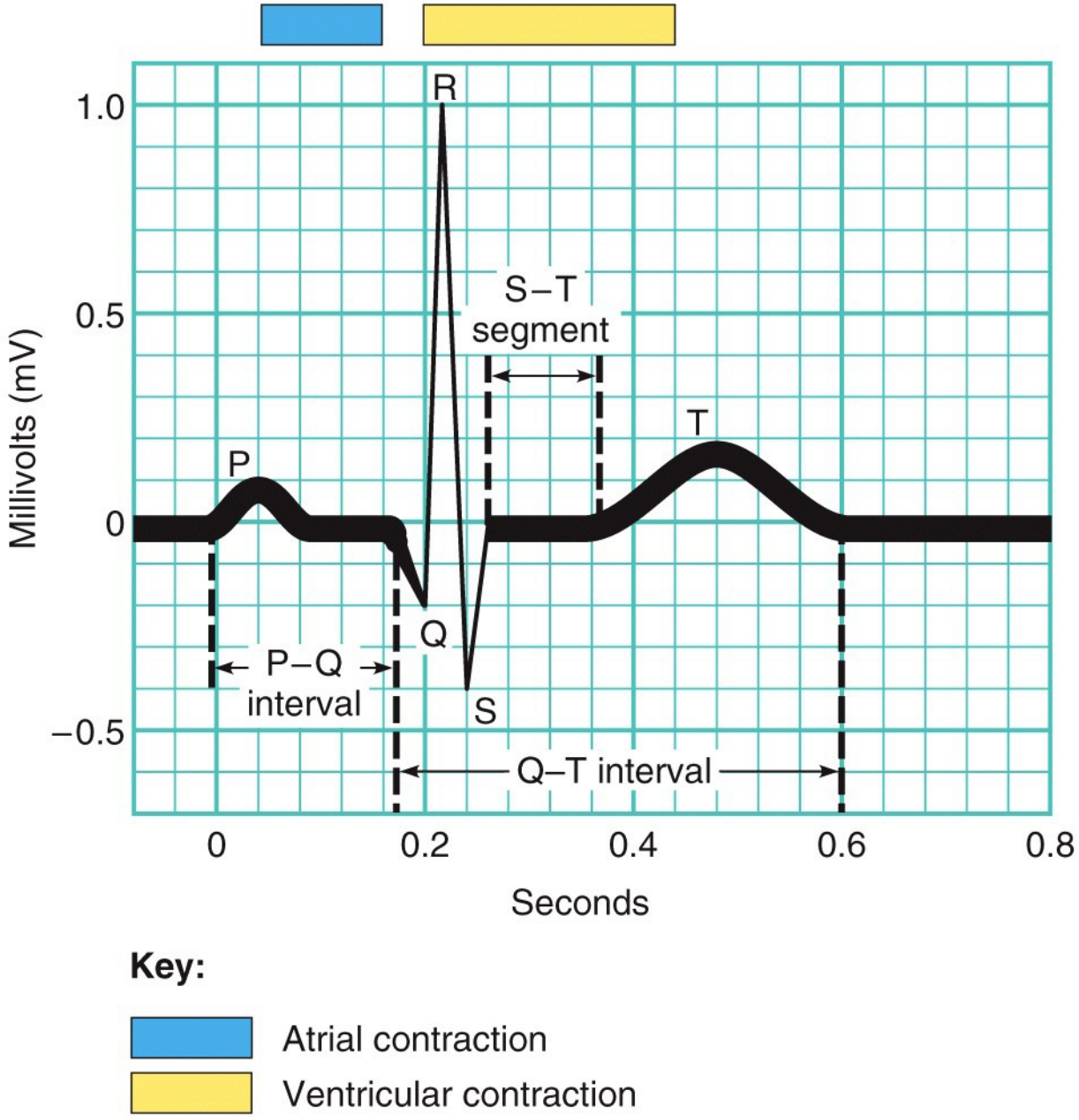
- **Describe events that occur during the systolic phase and diastolic phase of the cardiac cycle**

Cardiac Cycle

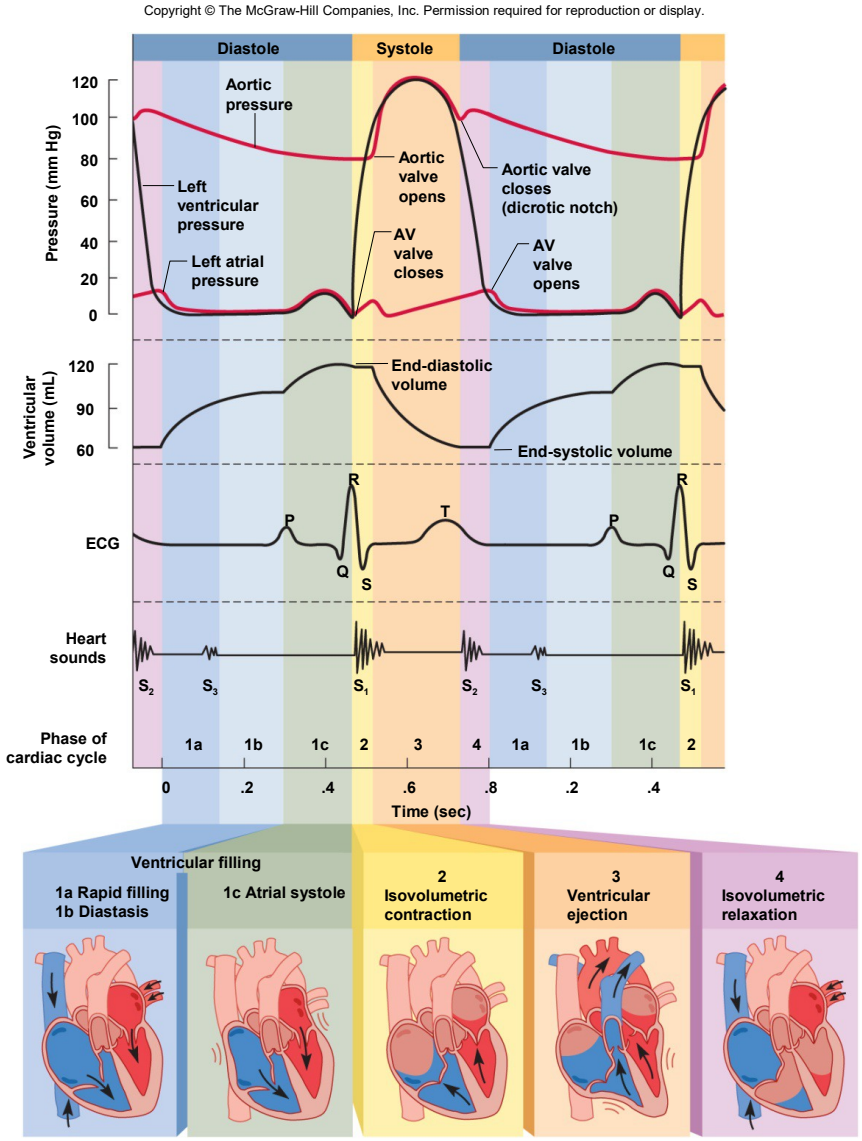
- Cardiac cycle - one complete contraction and relaxation of all four chambers of the heart (0.8 sec)
- Atrial systole (atrial contraction) occurs during ventricles diastole (ventricle relaxation)
- Ventricular systole (ventricle contraction) occurs during atrial diastole (atrial relaxation)
- Quiescent period occurs when all four chambers are relaxed at same time (diastolic phases)

Timing of Cardiac Cycle

- In a resting person
  - atrial systole last about 0.1 sec
  - ventricular systole about 0.3 sec
  - quiescent period, when all four chambers are in diastole, 0.4 sec
- Total duration of the cardiac cycle is therefore 0.8 sec // 75 bpm
- To analyze these events which occur in all four chambers, it is best to follow the events that occur in a single chamber.
- We will focus on events which occur in a ventricle.

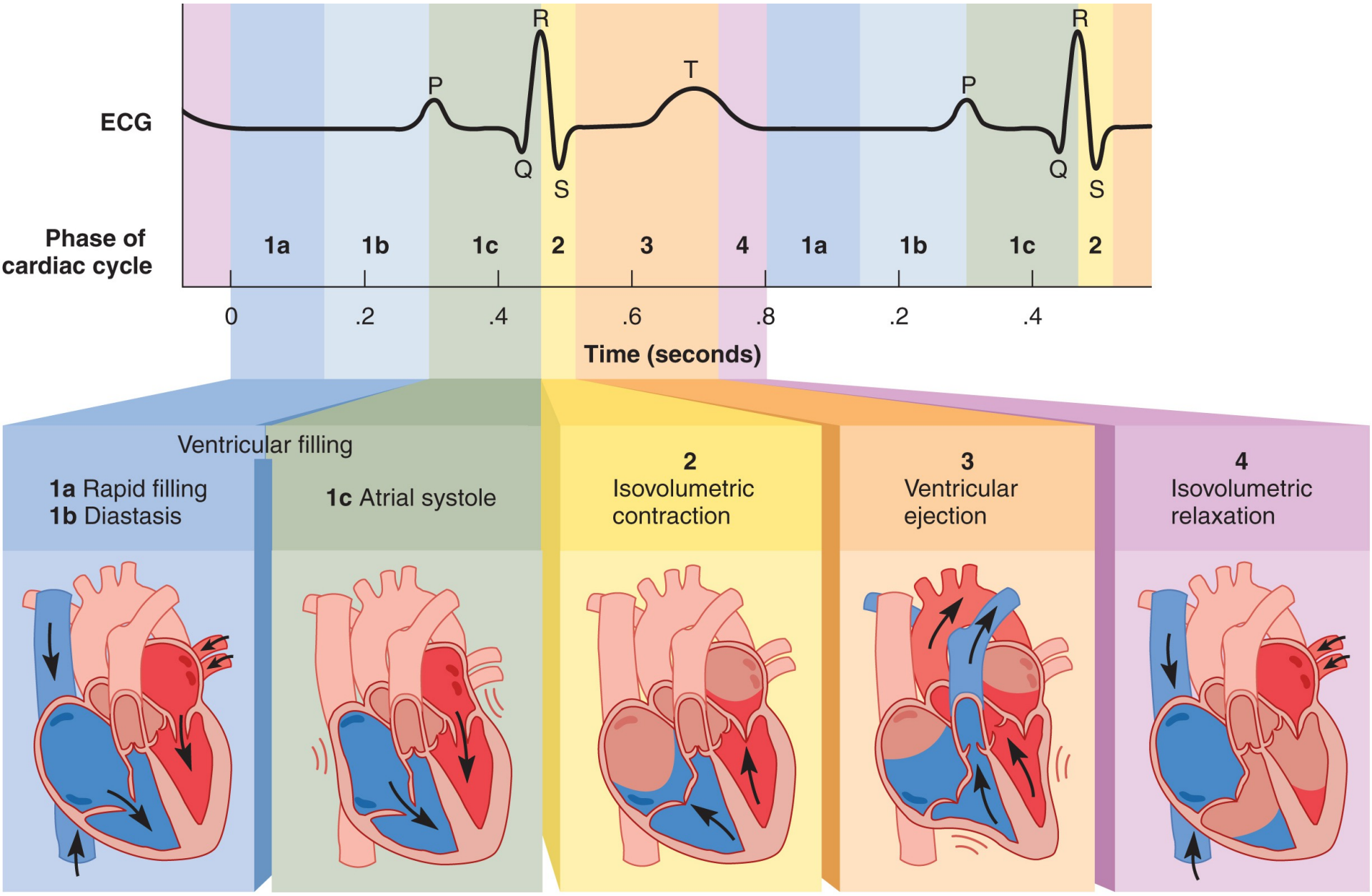

1.0
0.5
0
−0.5
Millivolts (mV)
R
S–T
segment
T
P
Q
S
P–Q
interval
Q–T interval
0
0.2
0.4
0.6
0.8
Seconds
Key:
Atrial contraction
Ventricular contraction

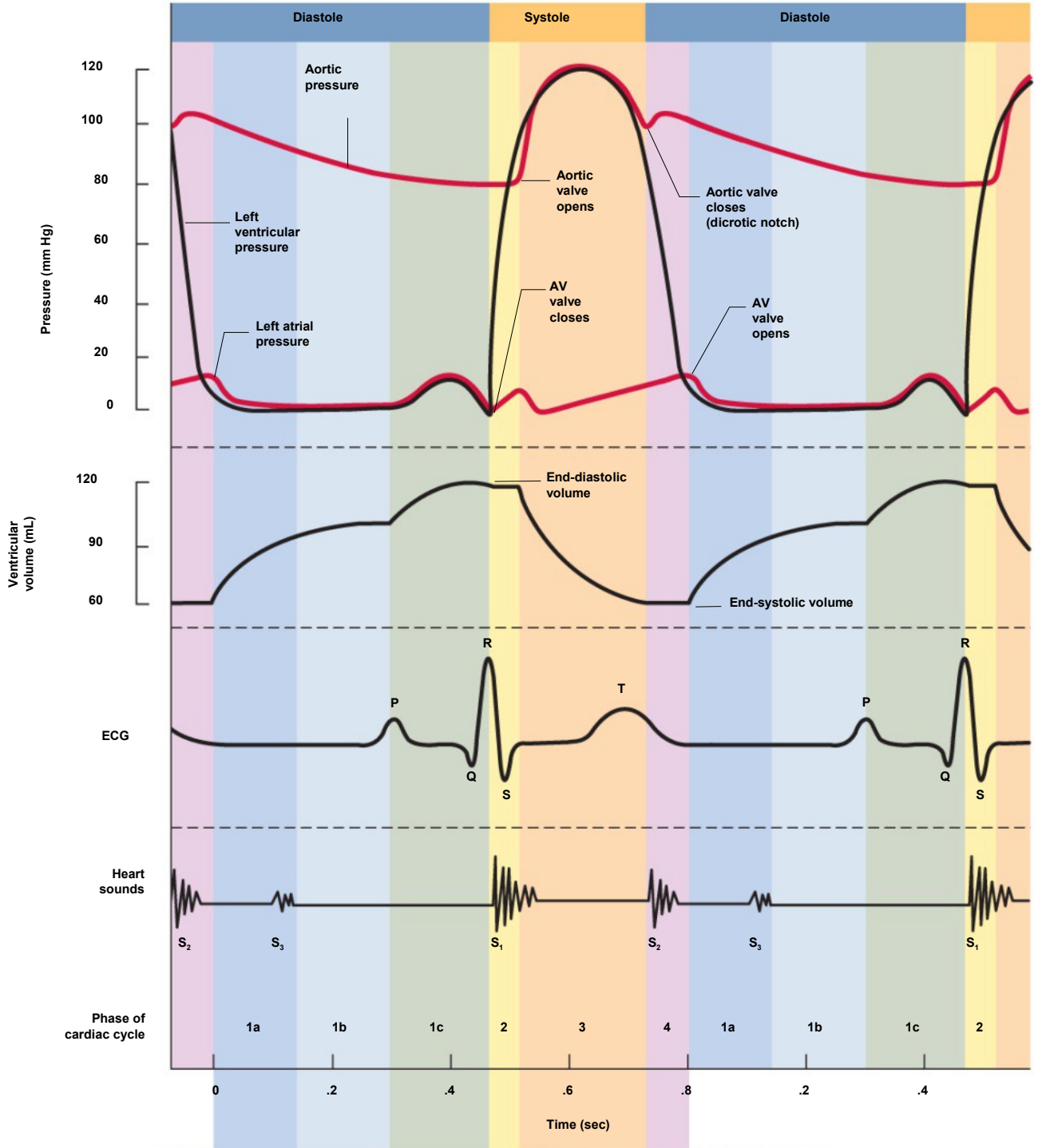
Four Phases of the Cardiac Cycle
(events occurring in one ventricle)



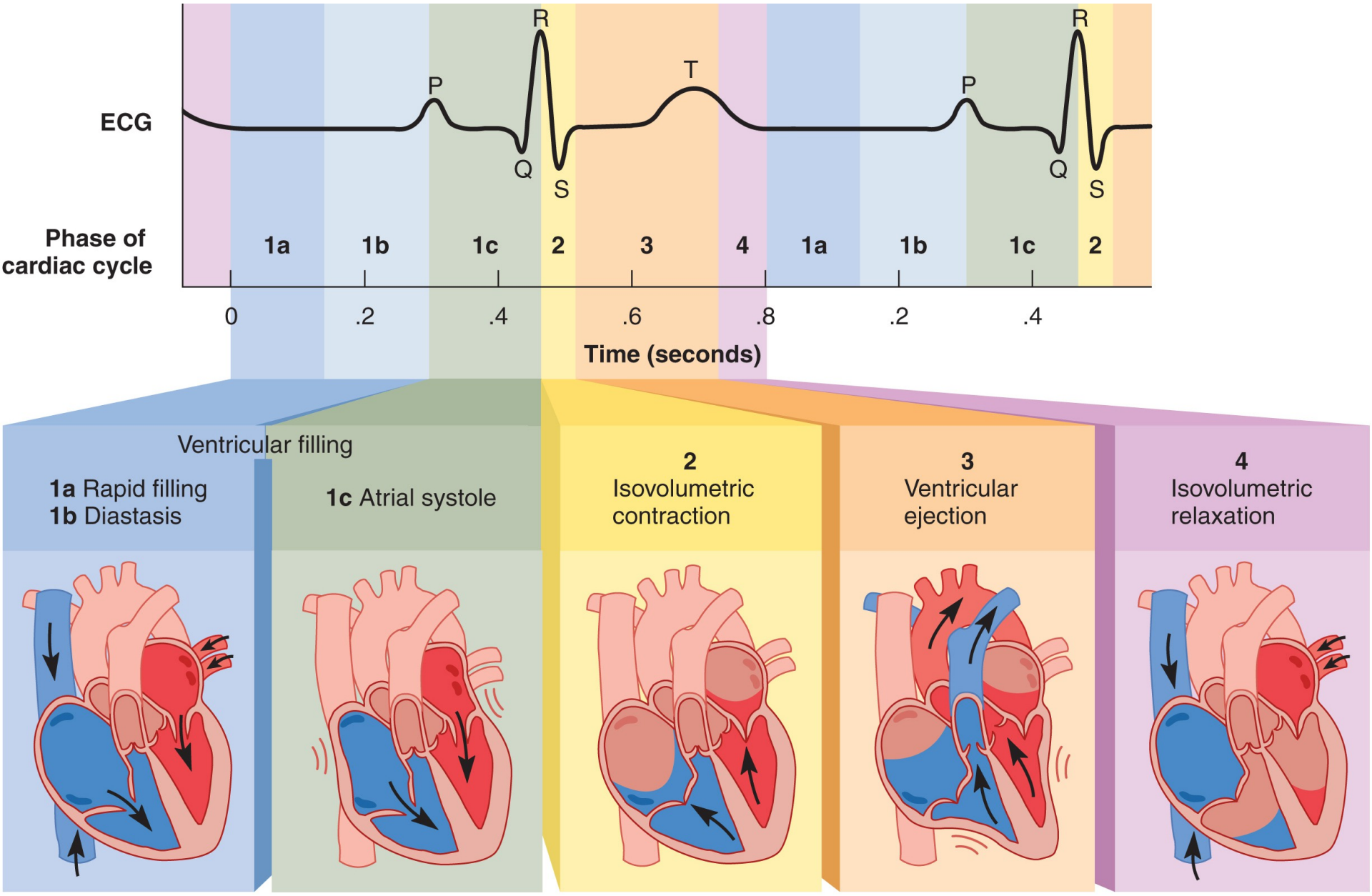
- ventricular filling
- isovolumetric contraction
- ventricular ejection
- isovolumetric relaxation
- The right and left ventricles eject the same volume of blood. Why?

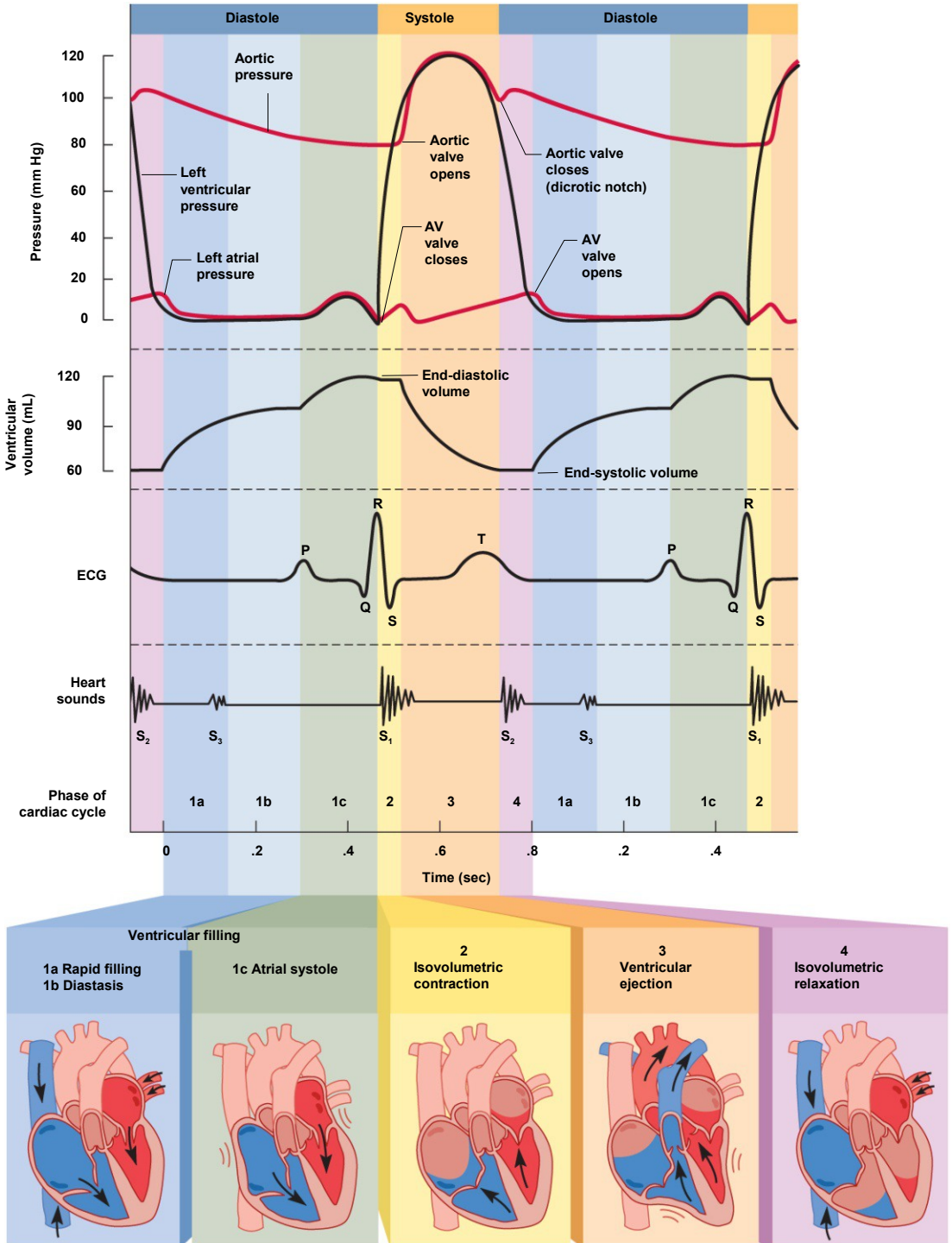
Phases of Cardiac Cycle




Diastole
Systole
Diastole
Pressure (mm Hg)
120
100
80
60
40
20
0
Aortic pressure
Aortic valve opens
Aortic valve closes (dicrotic notch)
Left ventricular pressure
AV valve closes
AV valve opens
Left atrial pressure
Ventricular volume (mL)
120
90
60
End-diastolic volume
End-systolic volume
ECG
P
R
Q
S
T
Heart sounds
S2
S3
S1
Phase of cardiac cycle
1a
1b
1c
2
3
4
0
.2
.4
.6
.8
Time (sec)

Phases of Cardiac Cycle




Diastole
Systole
Diastole
Pressure (mm Hg)
120
100
80
60
40
20
0
Aortic pressure
Left ventricular pressure
Left atrial pressure
Aortic valve opens
AV valve closes
Aortic valve closes (dicrotic notch)
AV valve opens
Ventricular volume (mL)
120
90
60
End-diastolic volume
End-systolic volume
ECG
P
R
Q
S
T
Heart sounds
S2
S3
S1
Phase of cardiac cycle
1a
1b
1c
2
3
4
0
.2
.4
.6
.8
Time (sec)
Ventricular filling
1a Rapid filling
1b Diastasis
1c Atrial systole
2
Isovolumetric contraction
3
Ventricular ejection
4
Isovolumetric relaxation

Events of Ventricular Filling (1 of 3)

- Occurs during ventricular diastole
  - Ventricles relax and expand
  - Ventricular pressure drops below pressure in atria
  - AV valves open and blood flows into the ventricles

Events of Ventricular Filling (2 of 3)

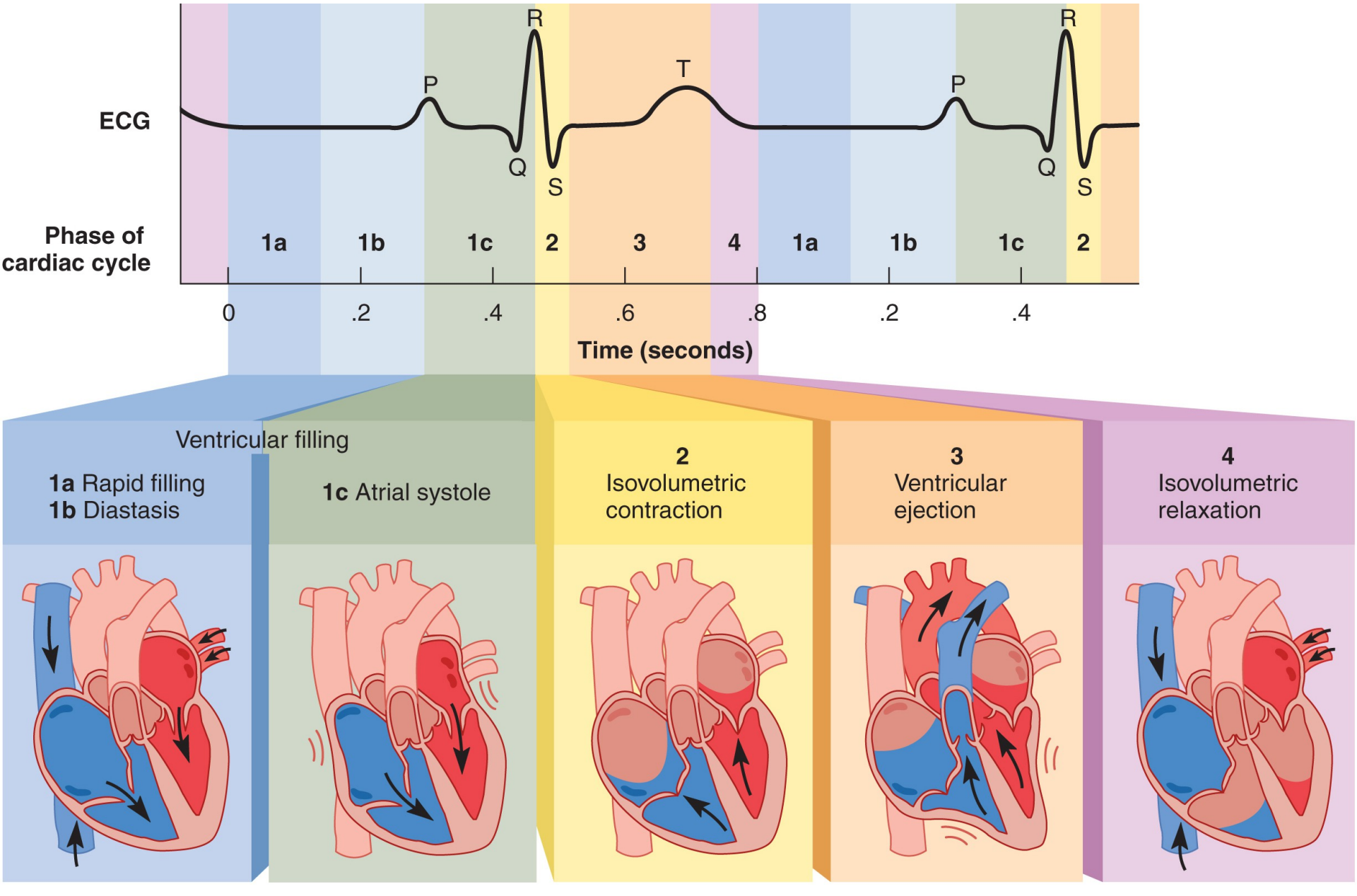
- Ventricular filling occurs in three phases:
  - rapid ventricular filling - first one-third
    - blood enters very quickly / passive and occurs before atrial systole begins
  - diastole continues in atria - second one-third
    - marked by slower filling
    - P wave occurs (depolarization occurs)at the end of diastasis
  - atrial systole - final one-third occurs with atria contraction

> What is the clinical significance for a patient with with atrial fibrillation (Afib)?
> Ventricular fibrillation?

Events of Ventricular Filling (3 of 3)

- End-diastolic volume occurs when ventricles stop filling with blood (EDV)
  - this is the amount of blood contained in ventricles at the end of ventricular filling
  - same volume will be in the right and left ventricle // must never be different
  - 130 mL of blood in each ventricle at end of ventricular diastole in resting state

Events of Ventricular Filling



Events of Iso-volumetric Contraction (1 of 2)

- Iso-volumetric describes condition in ventricles when both semilunar valves and atrial-ventricular valves are both closed

- During isovolumetric contraction the atria repolarize and they are in diastole // remain in diastole for the rest of the cardiac cycle

- Now ventricles depolarize (enter systole - contraction)
  - this initiates the QRS complex
  - depolarization followed by the contraction
  - pressure in ventricles start to increase

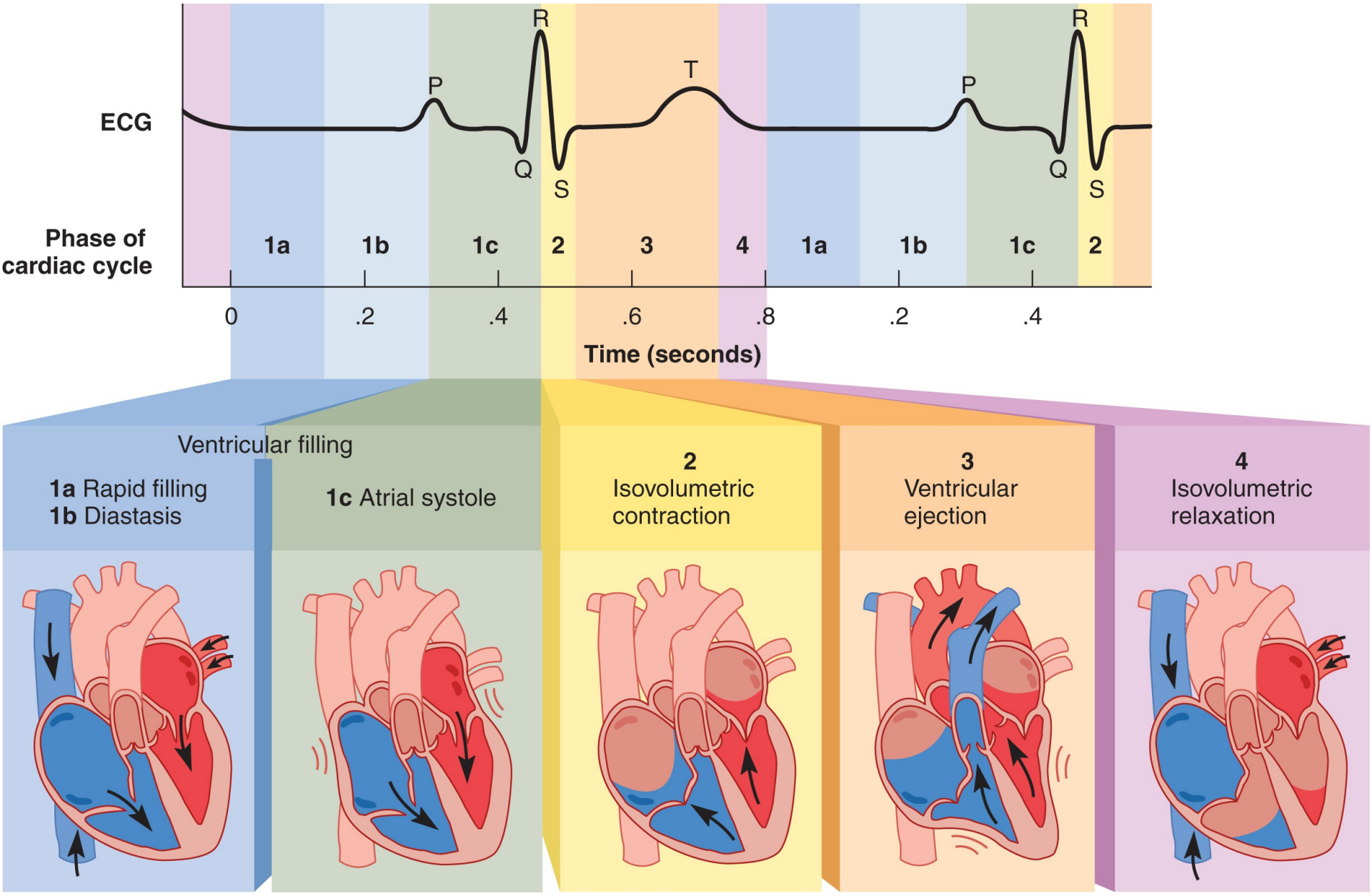
- AV valves close as ventricular blood pressure increases // forcing blood to surge back against the AV cusps /// heart sound S_1 occurs at the beginning of this phase // closing of AV valves

- Semilunar valves are still closed from previous cycle /// therefore – both AV valves and semilunar valves are both closed!

Events of Iso-volumetric Contraction (1 of 2)

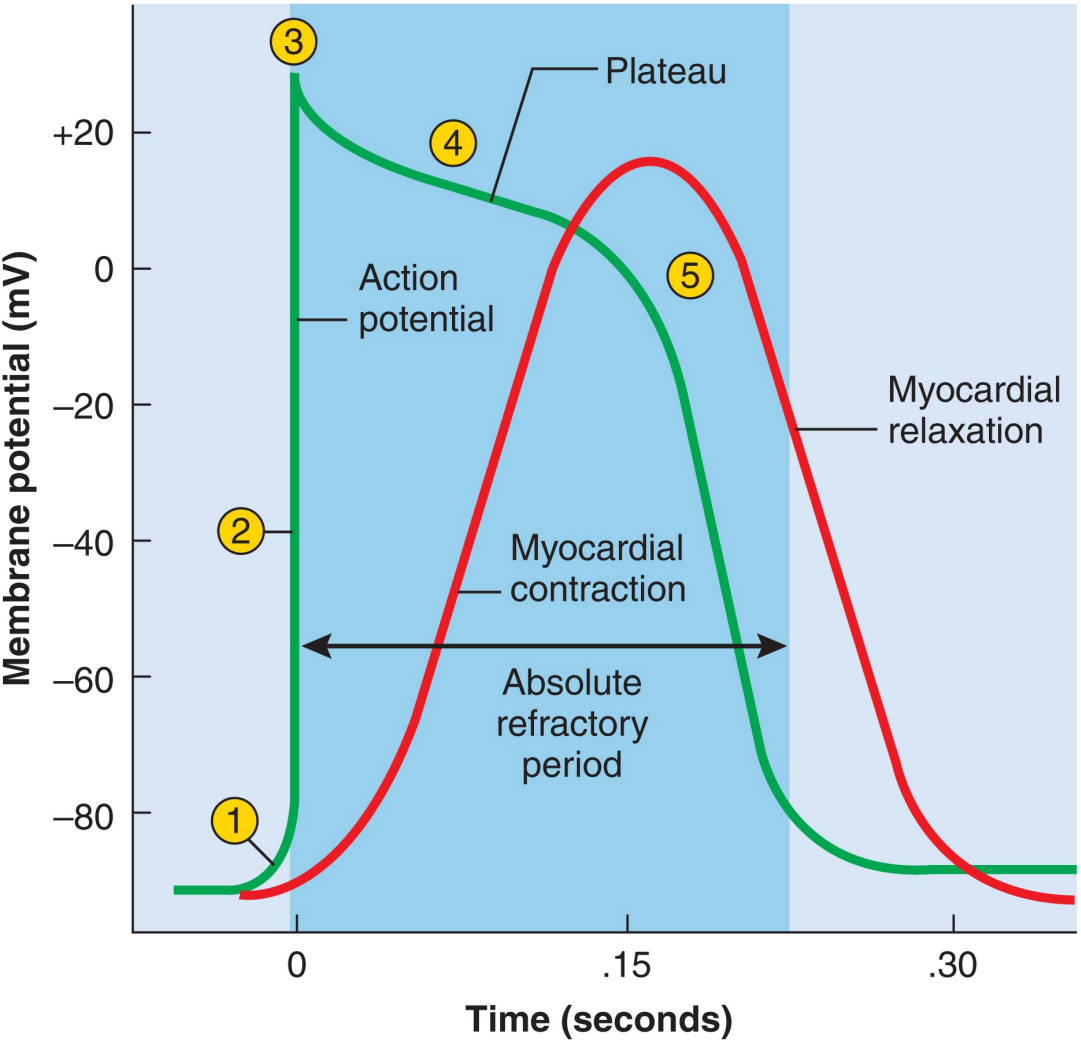
- Now entering the 'isovolumetric' contraction phase
- Ventricles contracting but they do not eject blood
- Why? --- both AV and semilunar values are BOTH STILL CLOSED
  - Semilunar valves are closed because pressure in the aorta (80 mm Hg) and in pulmonary trunk (10 mm Hg) is still greater than in the pressure in the two ventricles
- Cardiocytes exert force, but with all four valves closed, the blood cannot go anywhere // This results in rapid increase in ventricular pressure

Events of Iso-volumetric Contraction



Events of Ventricular Ejection (1 of 2)

- Ejection of blood begins when the ventricular pressure exceeds afterload in pulmonary trunk and aorta and forces semilunar valves open
  - pressure peaks in left ventricle at about 120 mm Hg and 25 mm Hg in the right
- Blood ejected out of each ventricle rapidly at first – rapid ejection
- Followed by slower rate of blood ejection because reduced pressure – reduced ejection
- Ventricular ejections last about 200 – 250 msec // this corresponds to the plateau phase of the cardiocyte action potential

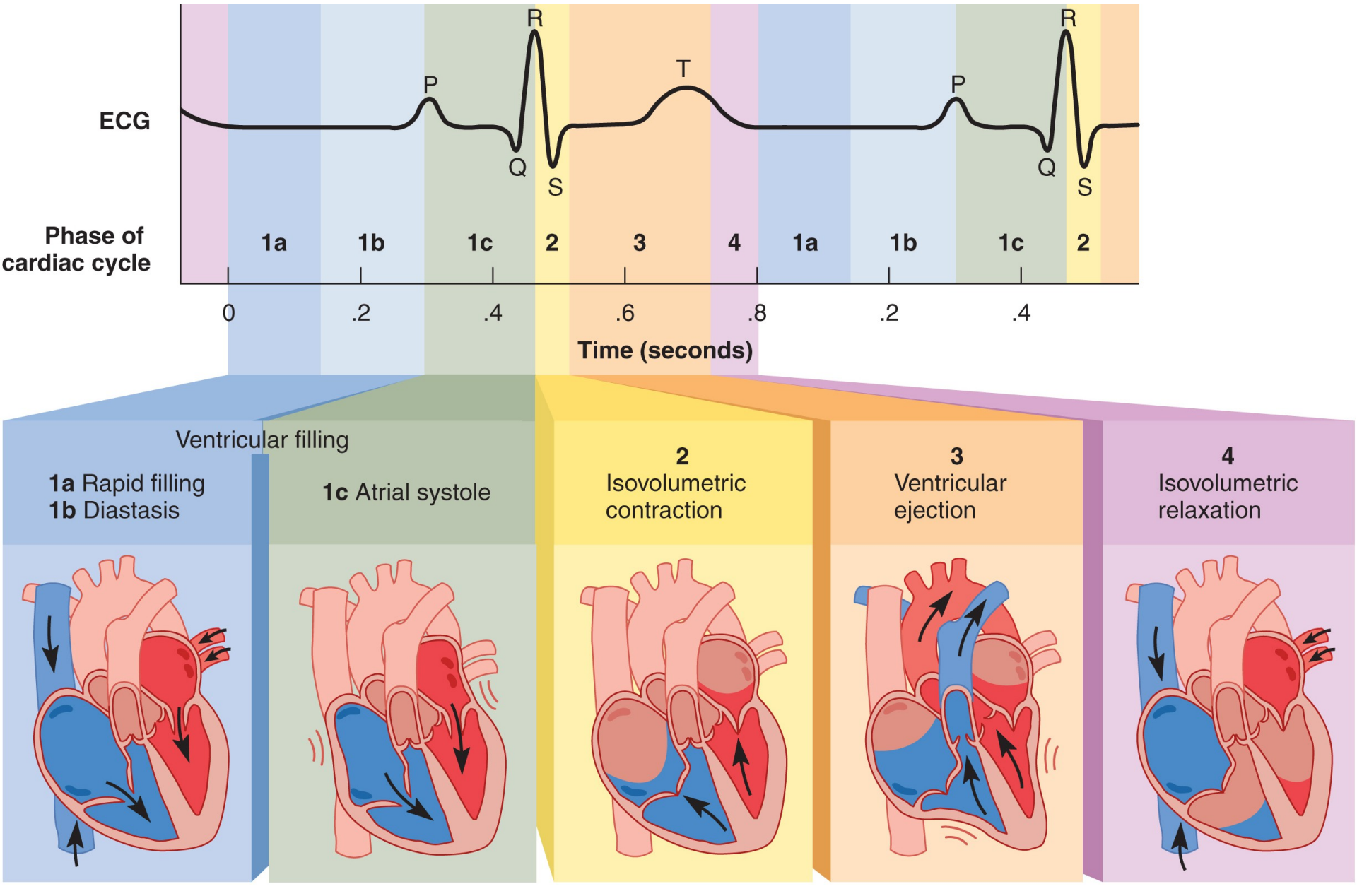


1. Voltage-gated Na^+ channels open.
2. Na^+ inflow depolarizes the membrane and triggers the opening of still more Na^+ channels, creating a positive feedback cycle and a rapidly rising membrane voltage.
3. Na^+ channels close when the cell depolarizes, and the voltage peaks at nearly +30 mV.
4. Ca^{2+} entering through slow Ca^{2+} channels prolongs depolarization of membrane, creating a plateau. Plateau falls slightly because of some K^+ leakage, but most K^+ channels remain closed until end of plateau.
5. Ca^{2+} channels close and Ca^{2+} is transported out of cell. K^+ channels open, and rapid K^+ outflow returns membrane to its resting potential.

Events of Ventricular Ejection (2 of 2)

- Stroke volume (SV) = 70 mL of blood from the 130 mL of blood in each ventricle (resting state volumes)
  - ejection fraction of about 54%
  - as high as 90% in vigorous exercise
  - long prolonged contraction of ventricles associated with slow calcium channels // the plateau of the myocardiocyte action potential
- End-systolic volume (ESV) // 60 mL of blood left behind
- T wave occurs at the end of this phase as ventricles repolarize and then enter diastole

Events of Ventricular Ejection



Isovolumetric Relaxation

- Occurs during early **ventricular diastole ///** when **T wave ends** and **the ventricles enter diastole (muscle relaxes)**

- Diastole of ventricles cause pressure to drop in ventricles

  - due to elastic recoil from blood inside the aorta and pulmonary truck- now the blood in these vessels starts to **flow towards the ventricles**

  - **this reverse flow of blood fill the "cusps" of the semilunar valves** and **closes the semilunar valves**

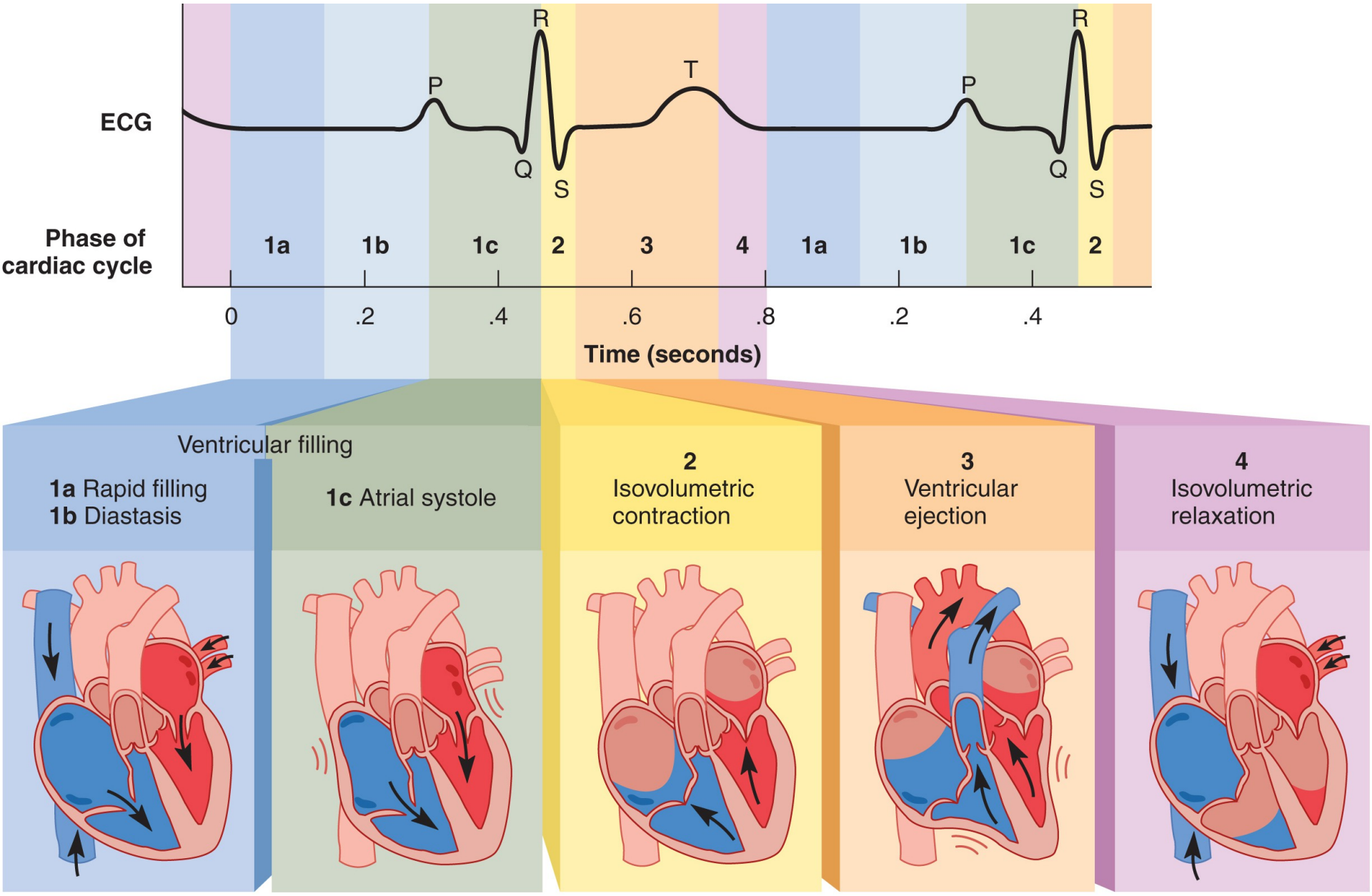
  - creates a **slight pressure rebound** that appears as the **dicrotic notch** of the aortic pressure curve

  - AV valves are still closed because pressure in ventricles are still greater than the pressure in the atria – isovolumetric relaxation!

Isovolumetric Relaxation

- heart sound S_2 occurs as blood closes semilunar valves
- isovolumetric because semilunar valves are closed and AV still closed
- ventricles are unable to receive blood
- when pressure in atria exceed ventricluar pressure then AV valves open again to renew ventricular filling

Isovolumetric Relaxation

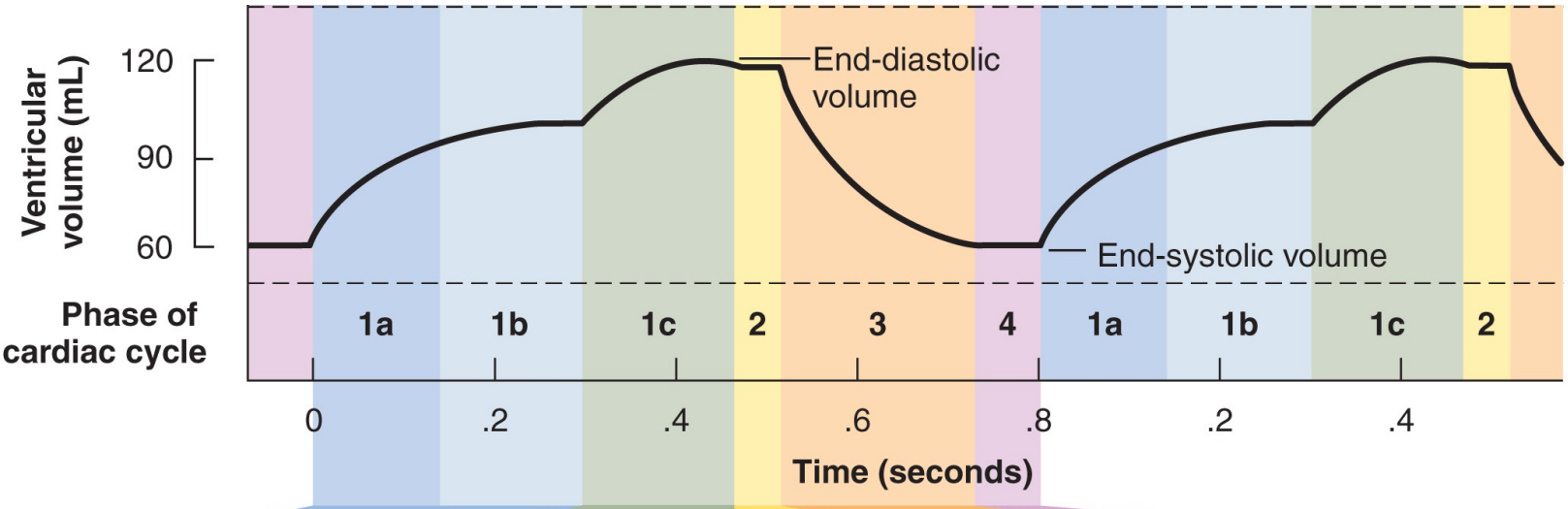


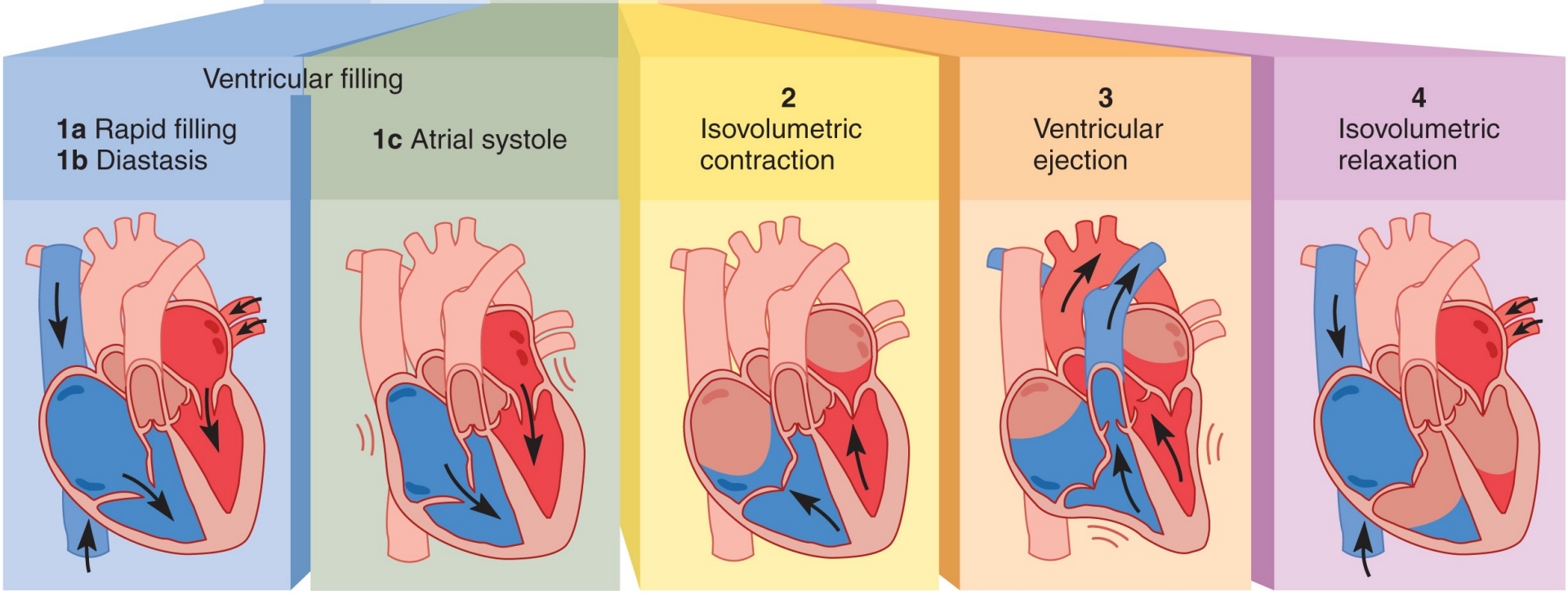
Overview of Volume Changes

end-systolic volume (ESV)	60 ml
- passively added to ESV during atrial diastole	30 ml
- added to ESV by atrial systole	40 ml
Total end-diastolic volume (EDV)	130 ml
stroke volume (SV) / blood ejected ejected by ventricular systole	-70 ml
end-systolic volume (ESV)	60 ml

Note: both ventricles must eject same amount of blood

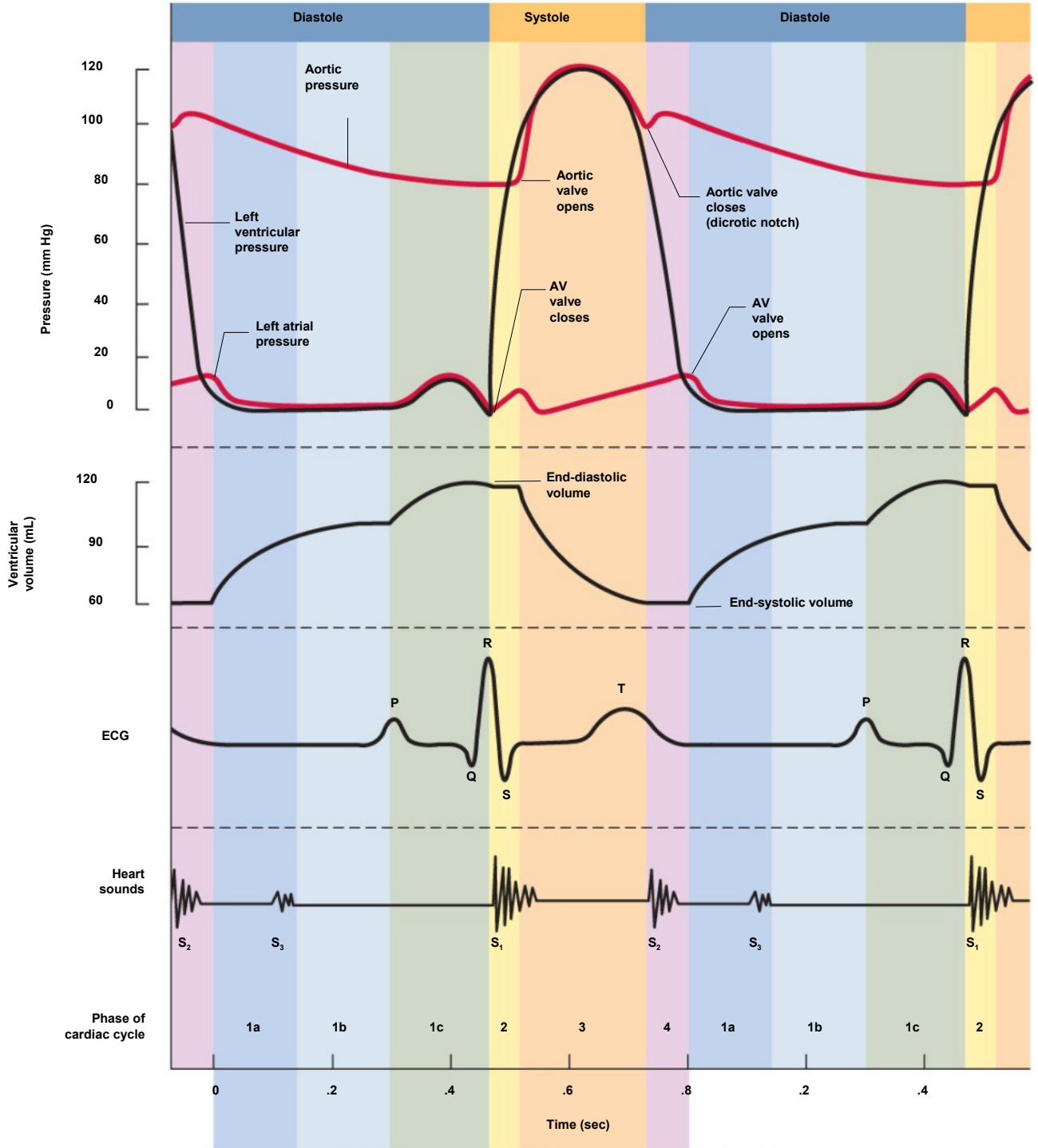
Overview of Volume Changes





Heart Sounds

- auscultation - listening to sounds made by body
- first heart sound (S_1), louder and longer “lubb”, occurs with closure of AV valves, turbulence in the bloodstream, and movements of the heart wall
- second heart sound (S_2), softer and sharper “dupp” occurs with closure of semilunar valves, turbulence in the bloodstream, and movements of the heart wall
- S_3 - rarely heard in people over 30


Diastole
Systole
Diastole
Pressure (mm Hg)
120
100
80
60
40
20
0
Aortic pressure
Aortic valve opens
Aortic valve closes (dicrotic notch)
Left ventricular pressure
AV valve closes
AV valve opens
Left atrial pressure
Ventricular volume (mL)
120
90
60
End-diastolic volume
End-systolic volume
ECG
P
R
Q
S
T
Heart sounds
S2
S3
S1
Phase of cardiac cycle
1a
1b
1c
2
3
4
0
.2
.4
.6
.8
Time (sec)

Pressure Gradients / Blood Flow / Valve Function

- Fluid flows only if it is subject to a pressure gradient /// fluid flows down a pressure gradient from high pressure to low pressure

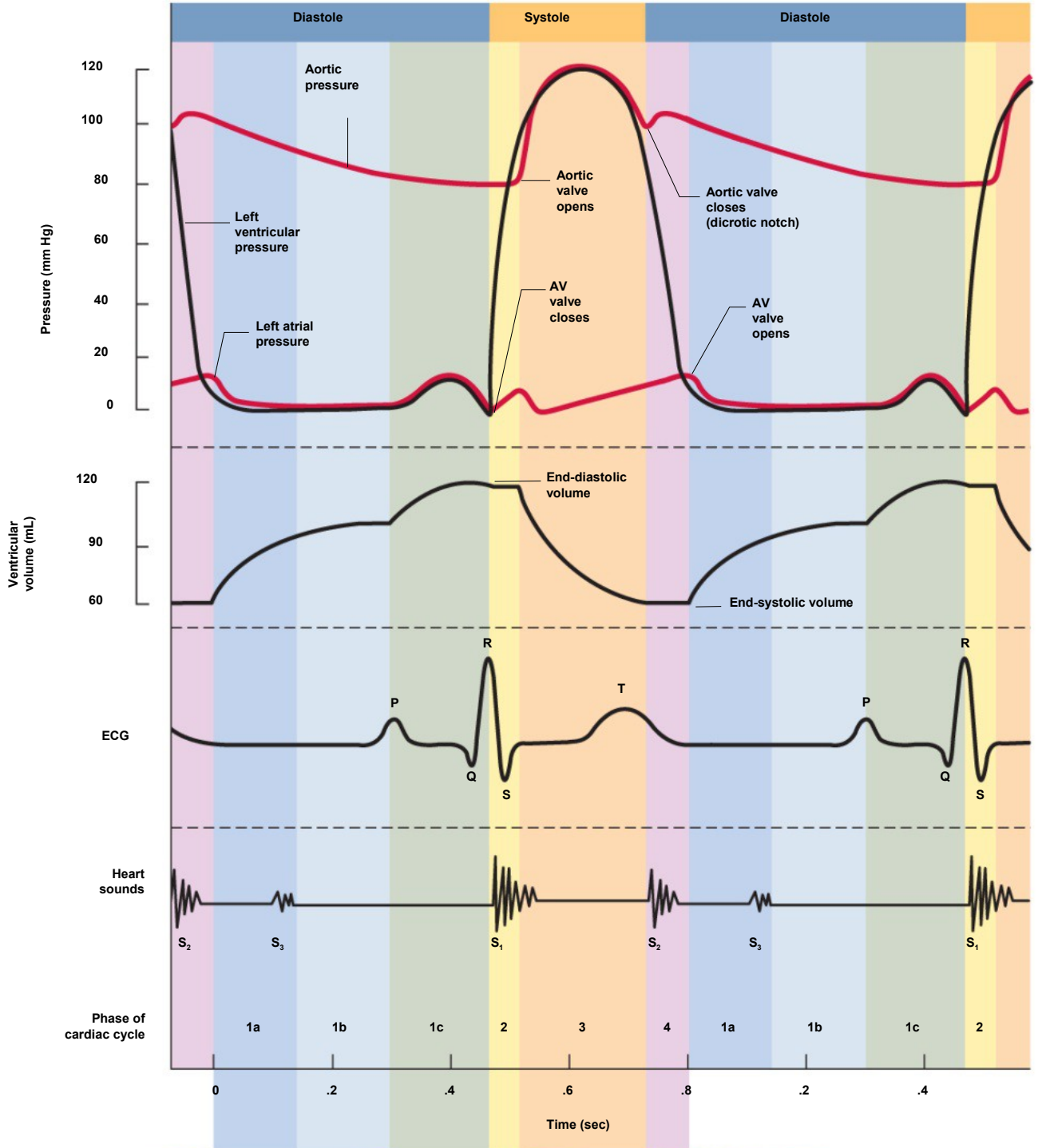
- Follow events that occur on left side of heart (note: similar events occur on the right side of the heart but with lower blood pressure / left and right heart functions must occur simultaneously)

  - when ventricle relaxes and expands (this allows ventricles to fill with blood (this is pre-load in ventricle) // occurs as ventricle internal pressure falls

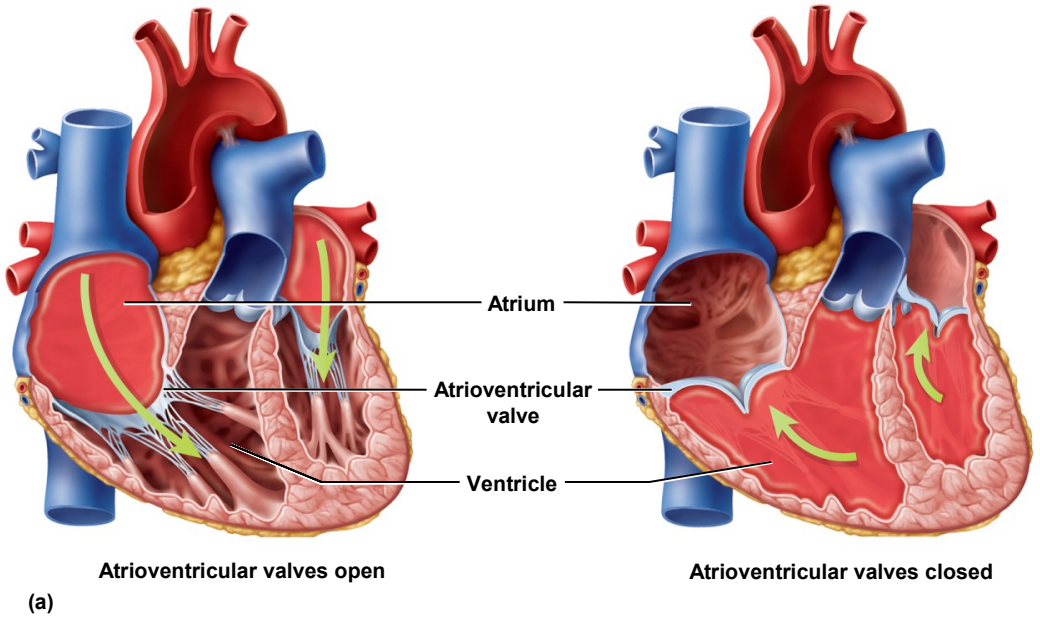
  - if bicuspid valve is open, blood flows into left ventricle

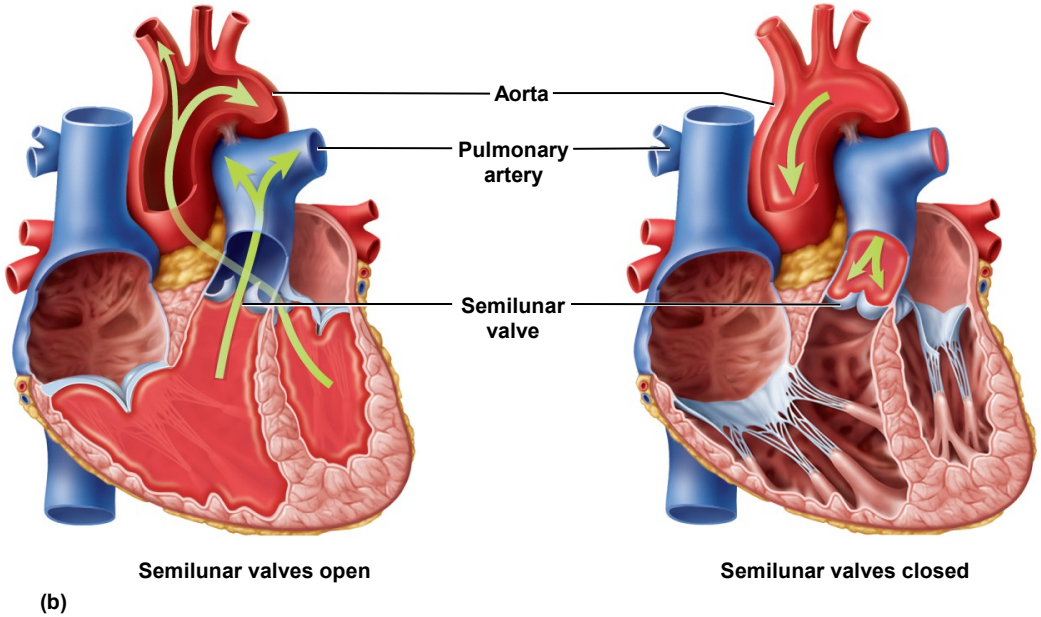
  - when ventricle starts to contract, pressure increases - blood flow towards atria / this closes AV valves (What prevents prolapse?)

  - after AV valves closes, pressure in ventricle continues to rise // the aortic valve is now pushed open (overcoming after-load = the pressure above semilunar valves in plumonary truck and aorta) /// when afterload pressure is exceeded in ventricles, blood is ejected blood flows into aorta


Diastole
Systole
Diastole
Pressure (mm Hg)
120
100
80
60
40
20
0
Aortic pressure
Aortic valve opens
Aortic valve closes (dicrotic notch)
Left ventricular pressure
AV valve closes
AV valve opens
Left atrial pressure
Ventricular volume (mL)
120
90
60
End-diastolic volume
End-systolic volume
ECG
P
R
Q
S
T
Heart sounds
S2
S3
S1
Phase of cardiac cycle
1a
1b
1c
2
3
4
0
.2
.4
.6
.8
Time (sec)

Operation of Heart Valves





Cardiac Output = stroke volume X heart rate

- CO about 4 to 6 L/min at rest (test figure 5.25 L per min)
  - This means a RBC leaving the left ventricle will arrive back at the left ventricle in 1 minute (approximately 5 L of blood in circulates)
  - vigorous exercise increases CO during event
    - Fit person up to 21 L/min
    - World class athlete up to 35 L/min
- Cardiac reserve – the difference between a person's maximum and resting Cardiac Output

Cardiac Output Changed By

cardiac output = stroke volume x heart rate

Volume of blood ejected by ventricle in 1 minute

CO = 70 ml / Beat x 75 Beat / Minutes = 5.25 L / Min

Cardiac Output May Be Changed By

chronotropic effects (time // related to the heart rate)

inotropic effects (related to contraction force called contractility // increase contractility will increase stroke volume)

Heart Rate and Cardiac Output

- Heart rate varies throughout life
  - infants have HR of 120 bpm or more
  - young adult females avg. 72 - 80 bpm
  - young adult males avg. 64 to 72 bpm
  - heart rate rises again in the elderly

- Positive chronotropic agents – factors that raise the heart rate
- Negative chronotropic agents – factors that lower heart rate
- Positive inotropic agents – factors that increase force of contraction
- Negative inotropic agents – facors that decrease force of contraction

cardiac output = stroke volume x heart rate

- *Key Idea:*

  When at rest, CO is "regulated" so CO = 5.25 L / min /// Why?

- *Therefore if stroke volume increases due to conditioning then HR will fall*

- *Stoke volume increases with exercise because ventricle hypertrophy*

- *This means the heart is not working as hard /// therefore it may "last longer"!!!!*

  - SV increases with fitness /// SV decreases with disease and aging

  - to keep cardiac output constant as we increase in age, the heart rate increases as the stroke volume decreases

The other factor that influence cardiac output

- Three variables govern stroke volume:
  - Preload (more preload = more SV = more blood ejected!)
  - After load (blood pressure in aorta which resist ejection of blood from heart) /// if afterload increases there is more resistance to eject blood / result in less SV)
  - Contractility = inotropic influence = as force of myocardiocyte contraction increases results in more SV
- Net result:
  - increased preload or increasing the contractility increases stroke volume
  - increased after load causes decrease stroke volume

Preload and Stroke Volume

- **Preload** – the amount of tension (caused by filling of the ventricles) in myocardium immediately before it begins to contract
  - as you Increase preload you increase force of contraction
  - exercise increases venous return, preload, and stretches myocardium
  - cardiocytes generate more tension (not like skeletal muscle / no tension length relationship)
  - increased cardiac output matched to increased venous return
- **Frank-Starling Law of the Heart** - SV$\propto$ EDV
  - stroke volume is proportional to the end diastolic volume
  - ventricles eject as much blood as they receive
  - the more they are stretched, the harder they contract // not like skeletal muscle

Afterload and Stroke Volume

- After load – the blood pressure in the aorta and pulmonary trunk immediately distal to their semilunar valves (ie the pressure above these valves)
  - opposes the opening of aortic and pulmonary semilunar valves
  - limits stroke volume
- *Hypertension increases after load and opposes ventricular ejection // overtime cause hypertrophy of heart // enlarged heart is very bad!*

What causes cor pulmonale?

- Anything that *impedes circulation* in either the systemic or pulmonary circuit may also increase after load (pressure above semilunar valve)

- E.g. // lung diseases will restrict blood flow into pulmonary circulation // blood "backs up" /// pressure "builds up" above the pulmonary semilunar valve

- Cor pulmonale – results in right ventricular failure due to *obstructed pulmonary circulation*

  - These diseases *obstruct pulmonary circulation* through lungs: emphysema, chronic bronchitis, and black lung disease

Inotropic VS Chronotropic

- Positive inotropic agents that increase contractility
  - hypercalcemia can cause strong, prolonged contractions and even cardiac arrest in systole
  - catecholamines increase calcium levels
  - glucagon stimulates cAMP production
  - digitalis raises intracellular calcium levels and contraction strength

Inotropic VS Chronotropic

- Negative inotropic agents reduce contractility
  - hypocalcemia can cause weak, irregular heartbeat and cardiac arrest in diastole
  - hyperkalemia reduces strength of myocardial action potentials and the release of Ca^{2+} into the sarcoplasm
  - vagus nerve has an effect on atria (the nodes) which reduces heart rate
  - However…..few vagus nerves innervate myocardiocytes in ventricles /// therefore vagus has no significant negative inotropic effect

Chronotropic Effects of the Autonomic Nervous System

- Autonomic nervous system does not initiate the heartbeat,
  - ANS modulates the rhythm and force
- Cardiostimulatory effect - some neurons of the cardiac center transmit signals to the heart by way of sympathetic pathways
- Cardioinhibitory effect - others transmit parasympathetic signals by way of the vagus nerve

Chronotropic Effects of the Autonomic Nervous System

- Sympathetic postganglionic fibers are adrenergic
  - they release norepinephrine // binds to β-adrenergic receptors in the heart
  - activates c-AMP second-messenger system in *cardiocytes (and nodal cells) --- result in 3 important events*
    - leads to the opening of slow Ca^{2+} channels in plasma membrane / fibers contract more quickly
    - opens calcium channels in sarcoplasmic reticulum / fibers contract more quickly
    - cAMP accelerates the uptake of Ca^{2+} by the sarcoplasmic reticulum // fibers relax more quickly
    - net result is ability to accelerate heart rate up to 240 bpm!

Chronotropic Effects of the Autonomic Nervous System

- Parasympathetic (vagus nerves) are cholinergic fibers // inhibitory effects on the SA and AV nodes
  - acetylcholine (ACh) binds to muscarinic receptors (cAMP mediated)
  - opens K^+ gates in the nodal cells
  - as K^+ leaves the cells, they become hyperpolarized and fire less frequently
  - heart slows down
  - parasympathetic effect on the heart is faster than sympathetic effect

Chronotropic Effects of the Autonomic Nervous System

- Vagal Tone (parasympathetic tone)
  - the heart has a intrinsic "natural" firing rate of 100 bpm
  - this means if both sympathetic and parasympathetic fibers are cut to the SA node the heart rate is faster!!!! why?
  - vagal tone – holds down this natural heart rate to 70 – 80 bpm at rest /// caused by steady background firing rate of the vagus nerves

Chronotropic Chemicals

- Chemicals may effect heart rate // in addition to the neurotransmitters from cardiac nerves
  - blood born adrenal catecholamines (NE and epinephrine) are potent cardiac stimulants

- Drugs that stimulate the heart
  - nicotine stimulates catecholamine secretion from adrenal gland
  - thyroid hormone increases number adrenergic receptors on heart so more responsive to sympathetic stimulation
  - caffeine inhibits cAMP breakdown /// therefore can prolong the adrenergic effect

Chronotropic Effects of Electrolytes

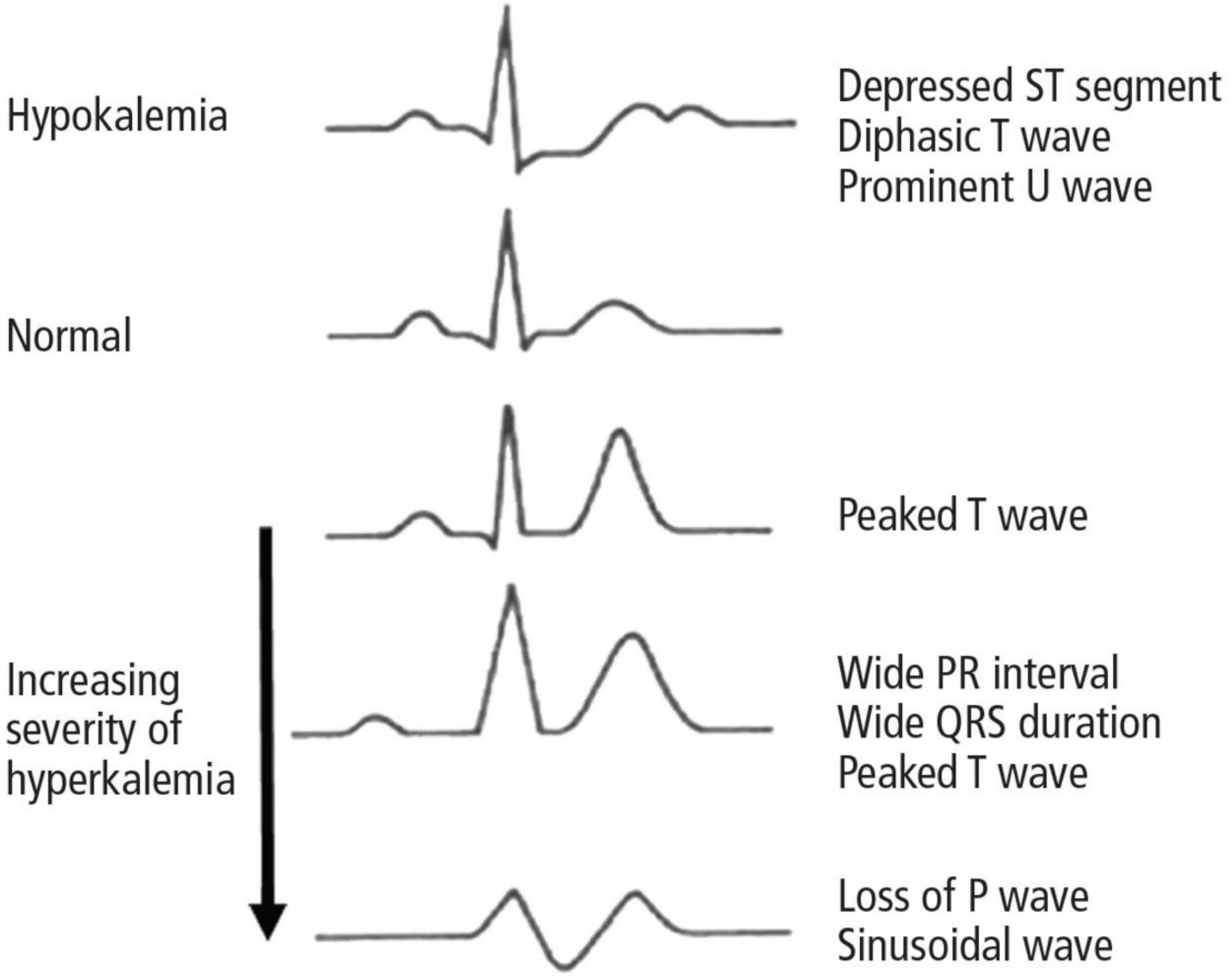
- Electrolyte : K^+ has greatest chronotropic effect

  - hyperkalemia (higher than normal concentration in blood)
    - Result / to much K^+ diffuses into cardiocytes / non excess K^+ cytoplasm
    - Membrane voltage elevated // now resting potential closer to threshold
    - As K concentration increases sodium leaking channels down regulated
    - Repolarization harder to achieve
    - Myocardium becomes less excitable
    - heart rate slows and becomes irregular
    - May arrest in diastolic phase

  - hypokalemia (lower than normal concentration in blood)
    - K^+ diffuses out of the cardiocytes
    - cells hyperpolarized / membrane potential more negative
    - require increased stimulation to reach threshold / harder to stimulate heart

- Potassium imbalances are very dangerous and require emergency medical treatment!

- What may happen after a crush injury to the arm?


Hypokalemia
Depressed ST segment
Diphasic T wave
Prominent U wave
Normal
Peaked T wave
Increasing
severity of
hyperkalemia
Wide PR interval
Wide QRS duration
Peaked T wave
Loss of P wave
Sinusoidal wave

Chronotropic Effects of Electrolytes

- Electrolyte : Ca^{2+} also affect heart rate (greater effect on contraction strength)
  - **hypercalcemia** – excess of Ca^{2+}
    - decreases heart rate and contraction strength
    - slow heart rate
  - **hypocalcemia** – deficiency of Ca^{2+}
    - increases heart rate
    - rare condition
    - *Greater effect is on nerve fibers causing action potential in somatic nerve fibers going to skeletal muscles (like diaphragm) / death from respiratory arrest!*

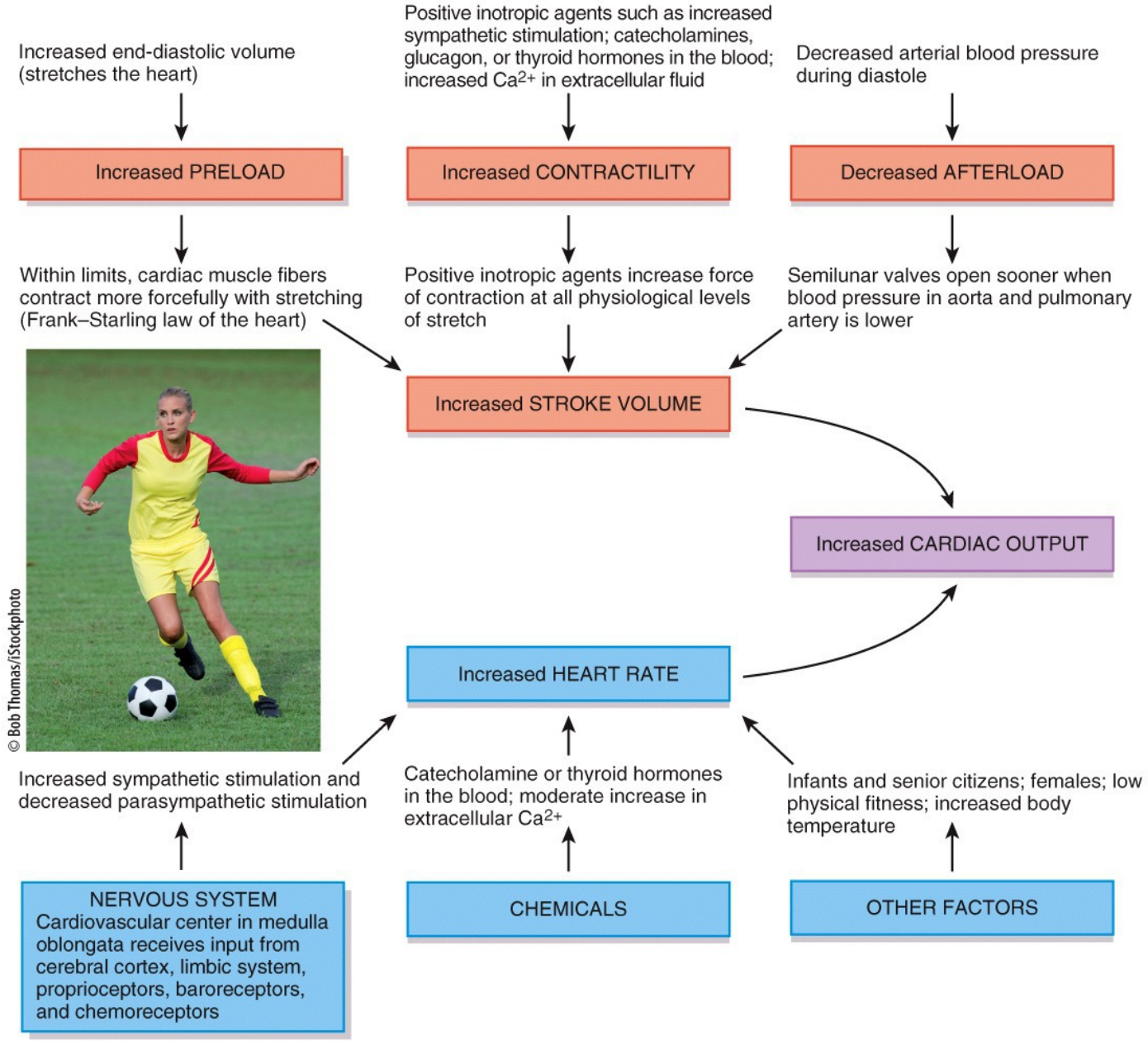
Why May Extreme Chronotropic Effects Reduce Stroke Volume?

- By accelerating the rate of contraction (how fast calcium is added to sarcoplasm) and then accelerating the reuptake of calcium into sarcoplasmic reticulum to increase rate of relaxation --- heart rate is increased!

- Sympathetic NS (norepinephrine) able to increase the heart rate as high as 240 bpm

  - At 240 bpm both stroke volume and cardiac output are reduced

  - Why? Note: at these high heart rates / diastole becomes too brief for complete filling of the ventricles!!!!



Great slide! Study this to learn key events.

Heart Function Terms

- Pulse pressure – surge of pressure produced by each heart beat that can be felt by palpating a superficial artery with the fingertips

- Tachycardia - resting adult heart rate above 100 bpm
  - stress, anxiety, drugs, heart disease, or fever
  - loss of blood or damage to myocardium

- Bradycardia - resting adult heart rate of less than 60 bpm
  - in sleep, low body temperature, and endurance trained athletes

Exercise and Cardiac Output

- *Exercise improves heart functon*
- *Exercise will strengthen the heart resulting in an increase stroke volume*
- *Then heart rate can be slower and still reach target cardiac output (5.25 L/min)*
- *This will now increase cardiac reserve*
- Exercise stimulate proprioceptors in skeletal muscles that send signal to cardiac center
  - at beginning of exercise, signals from joints and muscles reach the cardiac center
  - sympathetic output from cardiac center increases cardiac output
  - increased muscular activity /// increases venous return // increases preload /// results in an increase cardiac output
  - *increases in heart rate and stroke volume will cause an increase in cardiac output*

Exercise and Cardiac Output

- Exercise will cause moderate ventricular hypertrophy
  - Result = increased stroke volume /// will allow heart to beat more slowly while at rest
  - this increases cardiac reserve in the athlete so they can tolerate more exertion during performance than a sedentary person
  - *Note: a condition that causes a "pathologic enlarged heart" will reduce total ventricular volume and stroke volume then is decreased*
  - *therefore heart rate will need to increase to keep CO at 5.25 L/min // more stress on heart*

Valvular Insufficiency

- Valvular insufficiency (incompetence) // any failure of a valve to prevent reflux (regurgitation) the backward flow of blood

- Valvular stenosis – cusps are stiffened and opening is constricted by scar tissue

    - May result of **rheumatic fever** /// autoimmune attack on the mitral and aortic valves

    - heart now overworked and may become enlarged

Valvular Insufficiency

- Heart murmur – abnormal heart sound produced by regurgitation of blood through incompetent valves

- Mitral valve prolapse – insufficiency in which one or both mitral valve cusps bulge into atria during ventricular contraction

    - hereditary in 1 out of 40 people
    - may cause chest pain and shortness of breath

Congestive Heart Failure

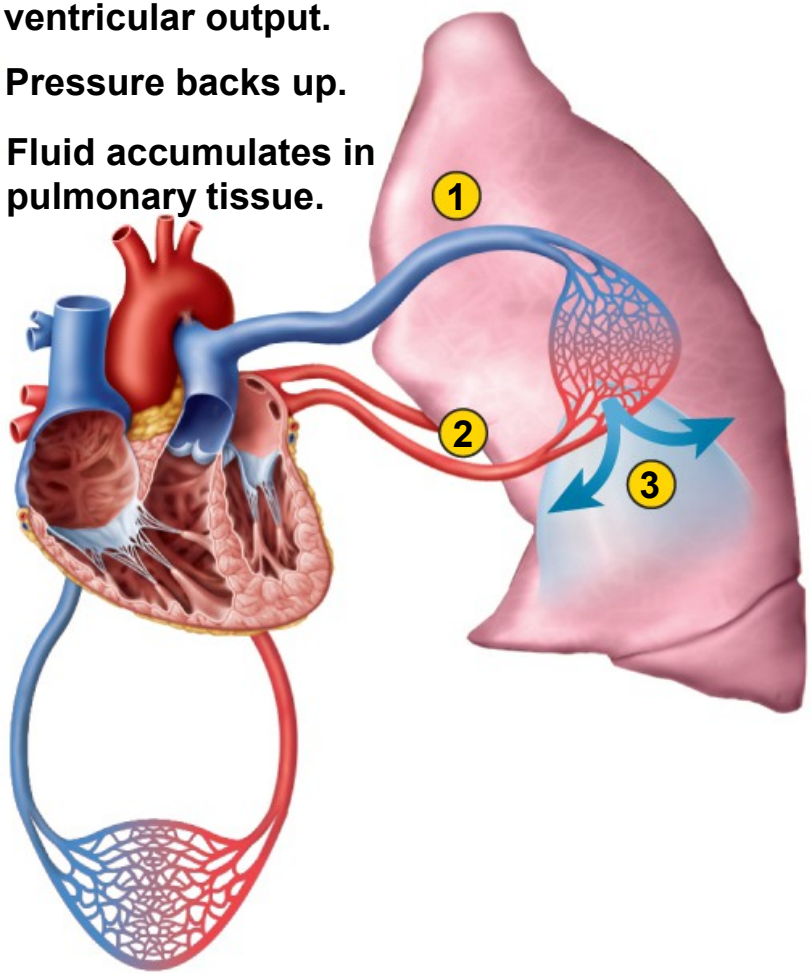
- The circulatory system is a closed system. The amount of blood ejected should be the same volume as blood returning.
- CHF occurs when one either ventricle ejects less than the normal volume of blood
- <u>One ventricle ejects proper amount of blood while the other ventricle ejects less blood</u>
- The ventricle which ejects less blood is the failing ventricle
- Usually due to a heart weakened by
  - myocardial infarction
  - chronic hypertension
  - valvular insufficiency
  - congenital defects in heart structure.

Congestive Heart Failure By Left Ventricular Failure

- Left ventricle ejects less blood (e.g. Rt V ejects 70 ml and Lt V ejects 50 ml)
- Rt. Ventricle is ejecting 20 ml more blood than Lt. ventricle during each cardiac cycle
- Extra “20 ml” most go somewhere // it accumulates in the lung interstitial space // pulmonary edema
- shortness of breath and/or sense of suffocation

Unbalanced Left Ventricular Output

1. Right ventricular output exceeds left ventricular output.
2. Pressure backs up.
3. Fluid accumulates in pulmonary tissue.



(a) Pulmonary edema

Left ventricular failure results in pulmonary edema

Note:

Cor pulmonale will also results in pulmonary edema

Due to lung emphysema and other disease states which cause restriction (fibrosis) in lung tissue

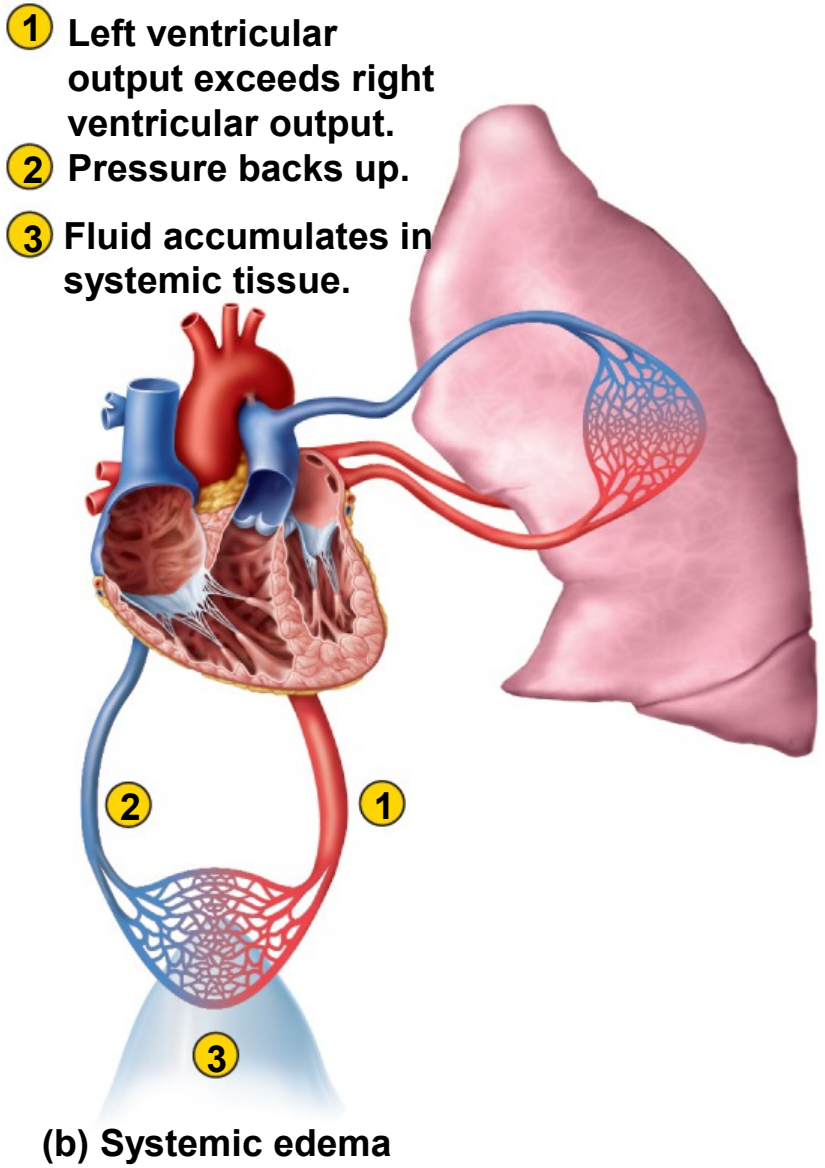
Enlarged right heart // these condition will contribute to right heart failure

Congestive Heart Failure By Right Ventricular Failure

- Right ventricle ejects less blood (e.g. Lt V ejects 70 ml and Rt V ejects 50 ml)
- Left ventricle ejects extra 20 ml of blood per cardiac cycle
- Rt ventricle can not receive the total volume so extra 20 ml filters into the systemic interstitial space // systemic edema - seen primarily in the legs
- enlargement of the liver, ascites (pooling of fluid in abdominal cavity), distension of jugular veins, swelling of the fingers, ankles, and feet

- *Note: Either condition will lead eventually to total heart failure*

Unbalanced Right Ventricular Output



(b) Systemic edema

Right ventricular failure results in systemic edema

Fluid accumulates in legs.

Shock – Loss of Blood Pressure

Hypovolumic
- Hemorrhage
- Loss of blood
- Loss of blood pressure

Cardiac Shock
- Lower heart rate
- Falling blood pressure

Neurogenic Shock
- Decrease in sympathetic tone to arterioles
- Arteriole dilation
- Falling blood pressure
- Anaphylactic shock – antigen causes systemic basophile degranulation

Shock

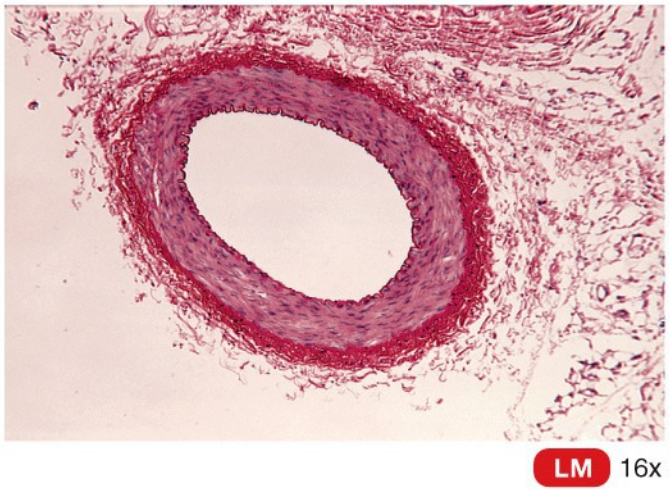
Compensated Shock
- Increase blood pressure
- Increase respiration
- Constriction of arterioles
- Pale cool skin

Decompensated Shock
- Low blood pressure
- Lack of perfusion
- Falling blood pressure

Irreversible Shock
- Perfusion to organs can not be restored
- Cell damage / death
- Organ damage / death

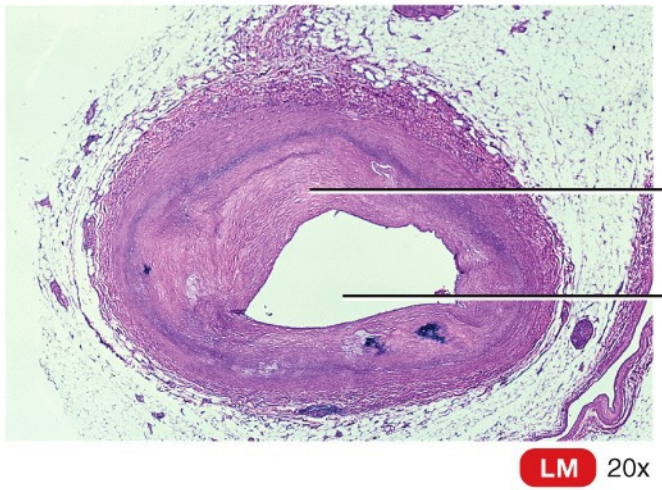
Pathology in Heart's Arteries

Chuck Brown/Photo Researchers, Inc.



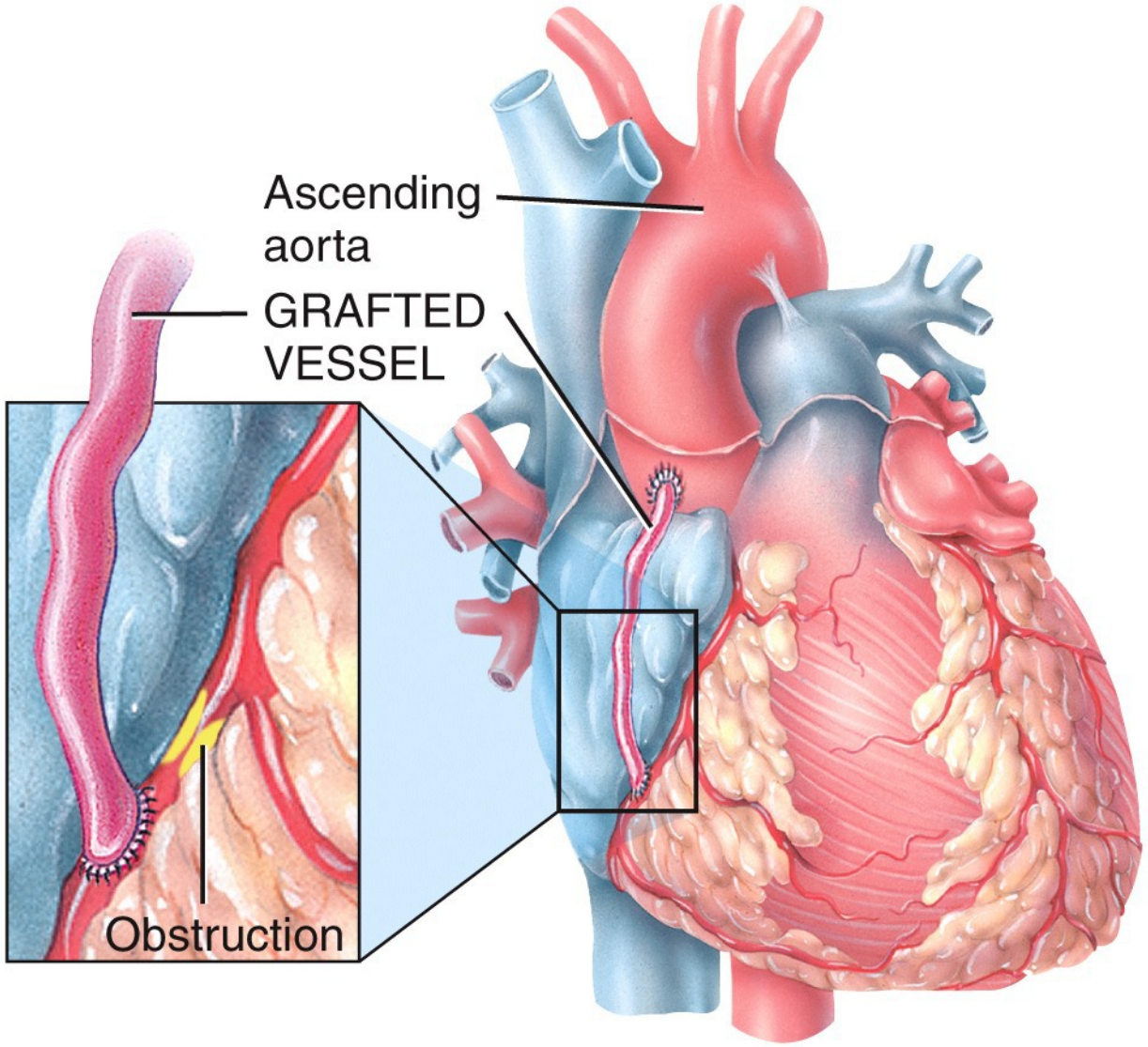
Normal artery

Carolina Biological Supply Company/Phototake

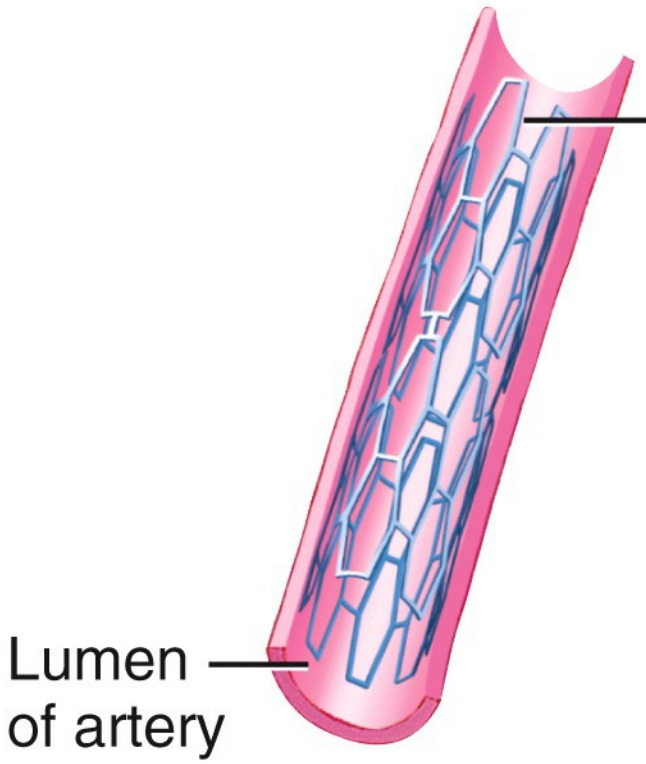


Obstructed artery

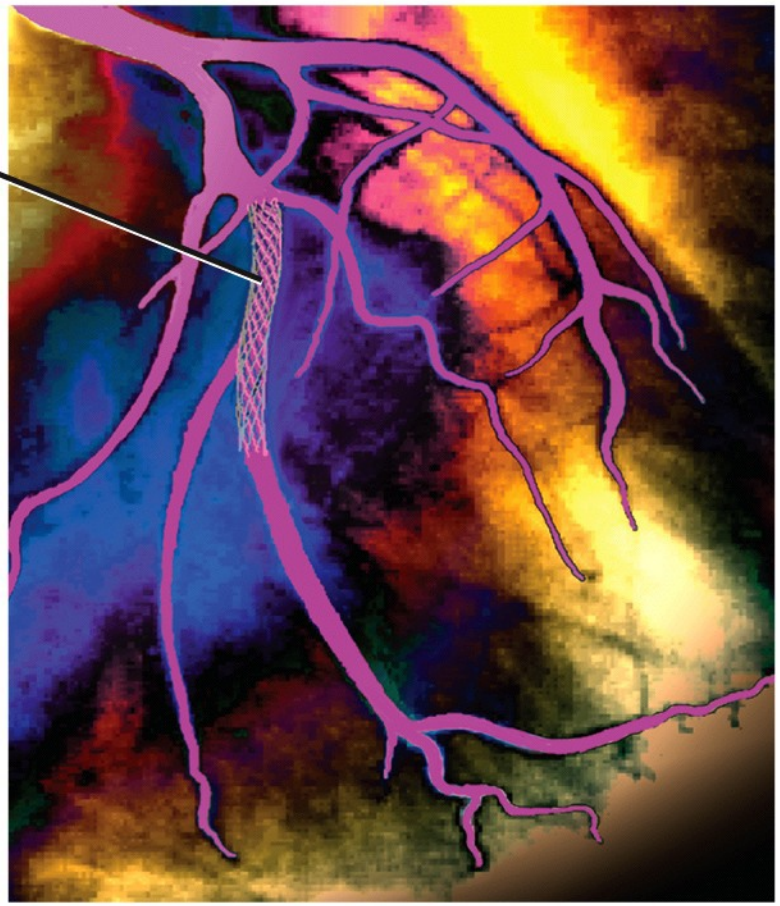
What is arteriosclerosis?



(a) Coronary artery bypass grafting (CABG)



(c) Stent in an artery



(d) Angiogram showing a stent in the circumflex artery