PHYSIOLOGY

بسم الله الرحمن الرحيم

MID | Lecture 7

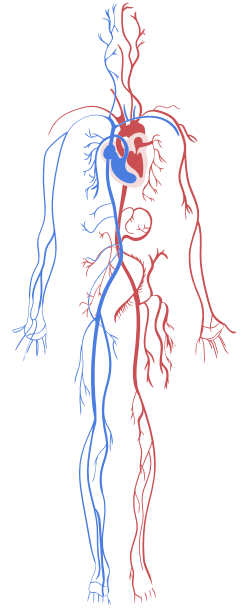
Heart Pump & Cardiac Cycle (Pt.1)

وَلَقَدْ خَلَقْنَا الْإِنسَانَ وَنَعْلَمُ مَا تُوَسْوِسُ بِهِ نَفْسُهُ ۖ وَنَحْنُ أَقْرَبُ إِلَيْهِ مِنْ حَبْلِ الْوَرِيدِ

اللهم إنّا نعوذ بك من شرور أنفسنا ومن سيئات أعمالنا

Written by: Omar Ibraheem, Zaid Bushnaq

Reviewed by: Mohammad Mahasneh



Heart Pump and Cardiac Cycle

Faisal I. Mohammed, MD, PhD

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

Electrical Events and what they Reflect Mechanically

1. P wave → Atrial systole

During the **P wave**, the **atria contract**.

- Atrial systole lasts **0.1 second**.
- It is immediately followed by **atrial diastole to allow the atria to be refilled**, which lasts **0.7 seconds**.

2. QRS complex → Ventricular systole

During the **QRS complex**, the **ventricles contract**.

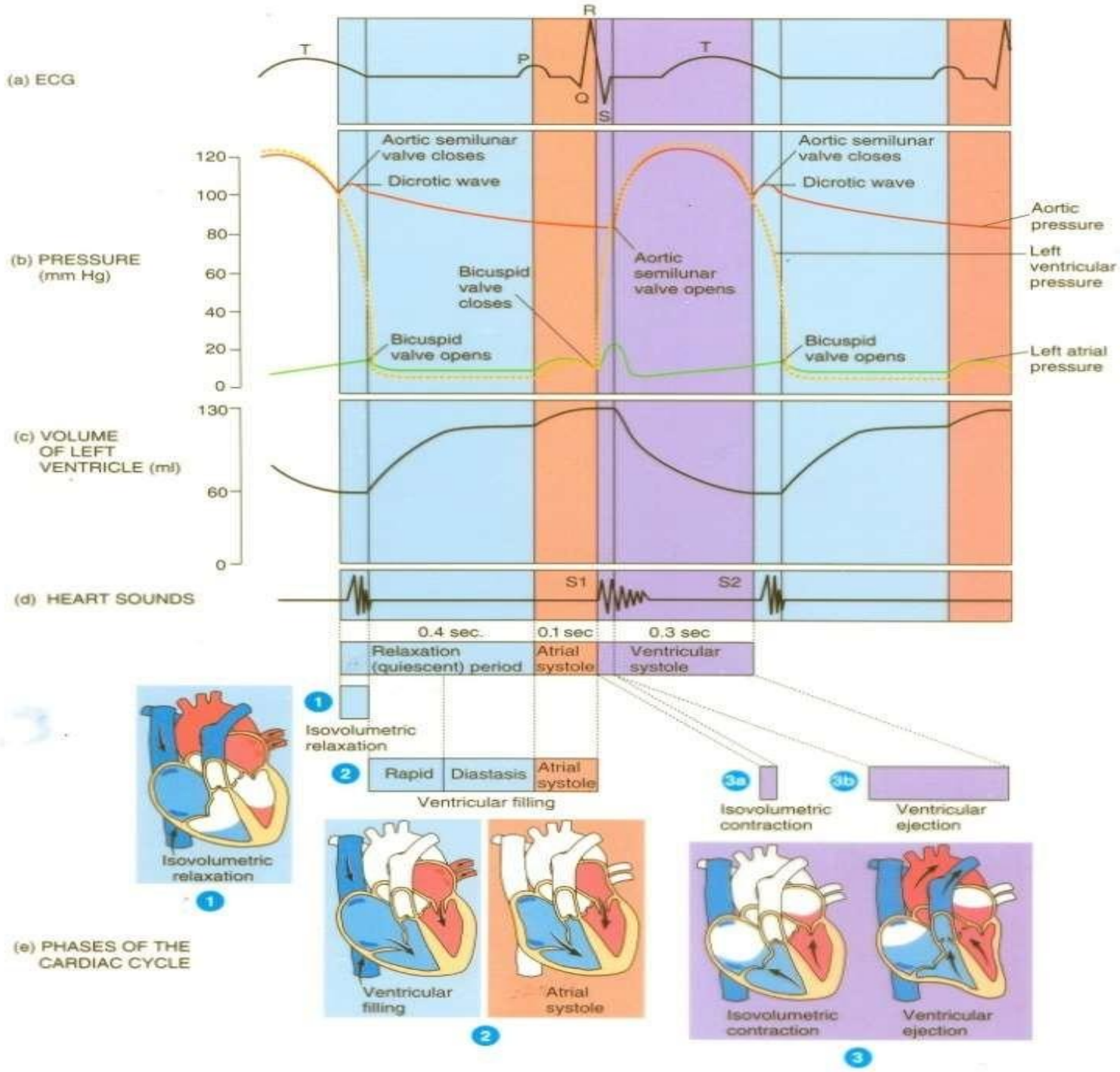
- Ventricular systole lasts **0.3 seconds**.

3. T wave → Ventricular diastole

During the **T wave**, the **ventricles relax**.

- Ventricular diastole lasts **0.5 seconds**.

- The **volume of blood** pumped by the right ventricle and the left ventricle is **the same**.
- However, the **pressure** generated by the **left ventricle** is **much higher** than that of the right ventricle, as the **left ventricle contracts much more strongly** to pump blood into the entire systemic circulation.



Types of Ventricular Filling

1. Passive Filling

- Happens **without any atrial contraction**.
- It is driven simply by the **gravity force** and the **pressure difference** between the atria and ventricles.
- This occurs during ventricular **diastole** when the AV valves are open.
- Most of the ventricular filling happens in this passive phase, **about 55-60 mL** of blood.

2. Active Filling (Atrial Kick):

- Occurs **at the end** of ventricular diastole **to empty the atria quickly before the ventricles start contracting.**
- This phase of filling is aided by the **atrial contraction**, which leads to pushing the remaining blood into the ventricles.
- This provides the **last ~25 mL** of blood to the ventricles.

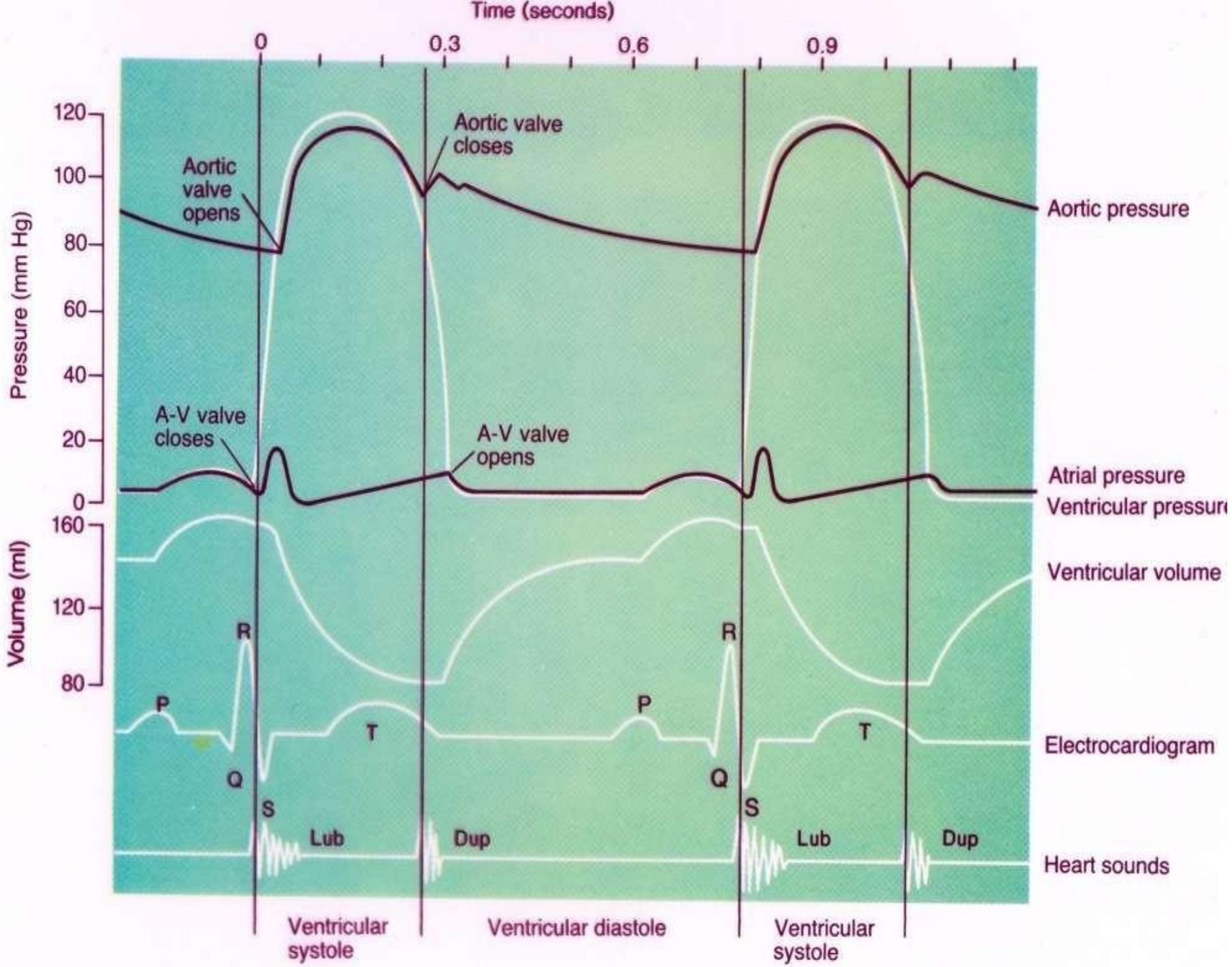
Effects of Atrial and Ventricular Fibrillation on the Heart Function

- **Atrial Fibrillation:**
  - With reference to what have been mentioned above, we can conclude that atrial fibrillation isn't fatal and is compatible with life, as the ventricles are approximately 80% filled by the passive filling without the need for atrial contraction.

  - Atrial fibrillation will cause some blood stasis in the atria due to the lack of the active filling phase; this could lead to a hypercoagulable state and to an increased risk of thrombosis. This could be simply managed by giving the patient anticoagulants.

- **Ventricular Fibrillation:**
  - On the other hand, ventricular fibrillation is considered fatal as most of the blood ejected towards the systemic circulation from the ventricles is dependent on the active ejection via the ventricular systole.

Next slides are going to discuss different events regarding each variable, make sure to look on the curve for better understanding



Mechanical Changes during the Cardiac Cycle

1. Passive Filling Phase

- Blood **passively fills the ventricles**.

2. Active Filling Phase

- Occurs **at the end of ventricular diastole**.
- The **atrium contracts** to push out the remaining blood quickly.

3. Start of Ventricular Systole → AV Valve Closure

- The ventricles begins to **contract; the ventricular pressure rises gradually as they contract.**
- As the ventricular pressure starts to rise, it becomes **higher than the atrial pressure**, which is near zero, this pressure difference forces the **AV valves to close, causing the formation of the isovolumic phase.**

Mechanical Changes during the Cardiac Cycle

4. Isovolumic Contraction Phase

- Both **AV valves** and **Semilunar valves** are **closed** (the semilunar valves are closed because the ventricular pressure hasn't yet exceeded the arterial pressure).
- The ventricles are contracting **without any change in volume** → ***isovolumic contraction***.
- Ventricular pressure continues to rise sharply during this phase.

5. Rapid Ejection Phase

- As contraction continues, the **ventricular pressure rises above the aortic pressure**.
- This pressure difference causes the **semilunar valves to open**.
- Once they open, the **rapid ejection** of blood from the ventricle begins.

Mechanical Changes during the Cardiac Cycle

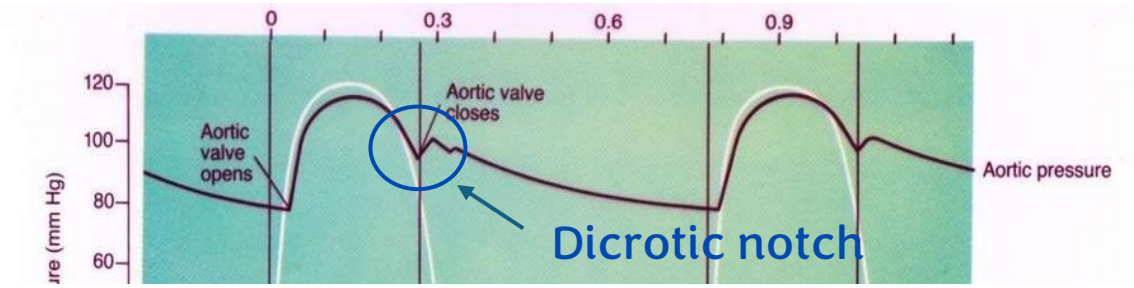
6. Continued Ejection Phase

- After the **rapid ejection**, the ventricle **continues ejecting blood**.
- This ejection continues **until the aortic pressure becomes higher** than the ventricular pressure, this happens because the ventricles are starting to relax leading to a drop in ventricular pressure.

7. Semilunar Valve Closure and the Formation of the Dicrotic Notch (or incisura).

- When the **aortic pressure exceeds ventricular pressure** → the **semilunar valve closes** to prevent further backflow.
- After the semilunar valve closes, a **small amount of blood briefly moves back** toward the valve.
- This regurgitation happens because of the **force of gravity**, which slightly raises the **aortic pressure**.
- This brief rise in aortic pressure at the beginning of ventricular diastole (just after ventricular systole) creates the **dicrotic notch** on the aortic pressure curve.

"An incisura occurs in the aortic pressure curve when the aortic valve closes. This is caused by a short period of backward flow of blood immediately before closure of the valve, followed by the sudden cessation of backflow." *Guyton and Hall Textbook of Medical Physiology* 14th ed.



Mechanical Changes during the Cardiac Cycle

8. Start of Ventricular Diastole

- After the semilunar valves close, the **ventricle begins to relax.**
- This marks the **start of ventricular diastole.**

9. Isovolumic Relaxation Phase

- During this early part of ventricular diastole, the **semilunar valves and the A-V valves are closed.**
- Because **both sets of valves are closed**, the ventricle relaxes **without any change in volume,** this creates the **isovolumic relaxation phase.**

*The four valves can never be open at the same time, but they can be **closed simultaneously** during **isovolumetric contraction** and **isovolumetric relaxation.***

Volumic Changes during the Heartbeat

- **Ventricular Filling From the Atria**
  - ✓ **Passive Filling:** Provides **about 50–55 mL** of blood.
  - ✓ **Active Filling (Atrial Systole):** Adds **about 25 mL** of blood.

  Total atrial delivery= **about 70 mL** of blood to the ventricle, about **50 mL** remain in the ventricle from the previous beat.

- **End-Diastolic Volume= 70 mL (new filling) + 50 mL (remnant) = 120 mL**

- **Ventricular Ejection**

  When the ventricle contracts, it ejects **only 70 mL** of blood before the semilunar valves closes, this volume is known as the stroke volume.

- **End-Systolic Volume**

  The **remaining 50 mL of blood** that isn't ejected and stays in the ventricle

Volumic Changes during the Heartbeat

- **Cardiac Output**

  If the **heart rate** is **70 beats/min** and the **Stroke volume is 70 mL**, **the cardiac output=** 70 mL/beat × 70 beats/min = 4900 mL/min, about the entire blood volume of the body is being circulated once per minute.

$$Q\ (Cardiac\ Output)\ \left[\frac{mL}{min}\right] = HR\ \left[\frac{beat}{min}\right] * SV\ (Stroke\ Volume)\ \left[\frac{mL}{beat}\right]$$

*Note that **both atria** pump the **same amount of blood** at the **same moment (≈ 70ml)**, and **both ventricles** pump the **same amount of blood** at the **same moment** too (≈ 70ml). = Equilibrium*

Atrial Pressure Waves (ACV Waves)

1. A-wave → Atrial contraction

- Happens during **atrial systole**.
- The atrium contracts → pressure inside the atrium **rises**.
- This produces the **A-wave**, the first atrial pressure bump.
- Occurs **just after the P wave** on ECG.

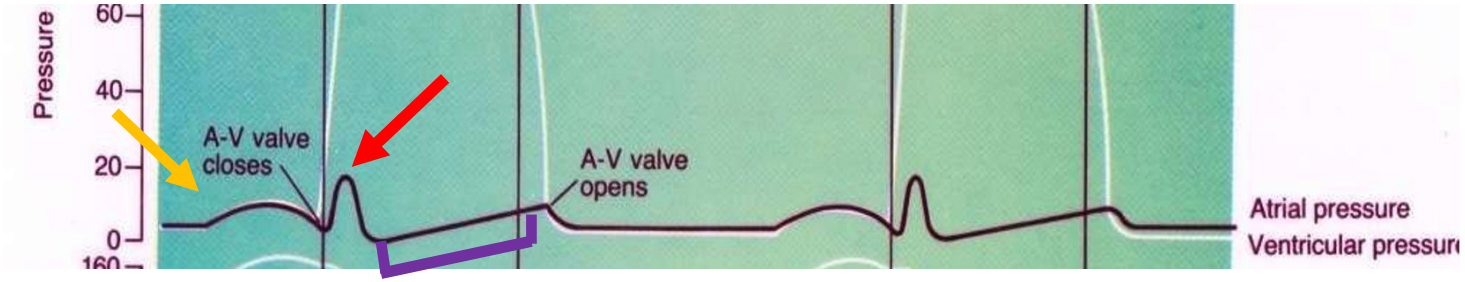
Note that these waves are not ECG waves, they are waves of atrial pressure change against time.

2. C-wave → Bulging of the AV valve during early ventricular systole

- When the ventricle starts contracting, the **closed AV valve bulges back upward** into the atrium.
- This causes a **brief upward rise in atrial pressure** → the **c-wave**.
- Occurs during **isovolumetric ventricular contraction**, right after the QRS wave.

3. V-wave → Venous filling of the atrium during ventricular systole

- While the ventricles are contracting and the **AV valves are closed**, blood continues to return from the veins into the atrium.
- This causes a **gradual rise in atrial pressure** → the **v-wave** ("venous filling wave").
- The pressure keeps rising until the **AV valve opens**, which causes:
  → the v-wave to **drop suddenly** as blood flows into the ventricle.



Heart sounds

- When the AV valves close, blood briefly pushes against them, creating **turbulence around the closed valves**, producing the **"Lub" (S1)** sound. It coincides with the **QRS complex** of the ECG, so the QRS complex occurs **just before S1. S1 occurs during isovolumetric contraction, *not* during rapid ejection.**

- The **"Dup" (S2)** sound is produced by turbulence around the **closed semilunar valves**.

- **The time** between the **first heart sound** and the **second heart sound** represents **the period** of **ventricular systole.**

- **The time** between **the second heart sound** and **the next first heart sound** is about **0.5 seconds**, which represents **ventricular diastole.**

Heart sounds

- During the rapid filling period of the ventricle, blood may **rush into** the ventricle, producing the **third heart sound, while d**uring atrial systole, blood is **pushed forcefully into** the ventricle, resulting in the **fourth heart sound**.

- We normally only hear the first and second heart sounds, and they are separated by a **short interval** that varies with heart rate.

- A murmur is an **abnormal sound**, such as that produced in **AV regurgitation**.

- The third and fourth heart sounds are usually **not easily audible**, so cardiologists typically record them using a **phonocardiogram**.

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 that includes the events of:
  - Isovolumic contraction 0.01 seconds
  - Rapid ejection period
  - Slow ejection period
- Ventricular diastole 0.5 seconds that includes the events of:
  - Isovolumic relaxation 0.02 seconds
  - Rapid filling
  - Slow filling (Diastasis)
  - Atrial contraction

The doctor emphasized on knowing the term **diastasis**, which is the slow phase of ventricular filling when atrial and ventricular pressures nearly equalize, resulting in minimal blood flow before atrial systole.

Calculating the Efficiency of the Heart

- The efficiency of the heart can be estimated using the **ejection fraction (EF)**, which represents the percentage of blood ejected from the ventricle relative to the total volume present before contraction. It is calculated as **stroke volume ÷ end-diastolic volume and** normally ranges from **60–75% (percentage, no unit).**

- Ejection fraction reflects **contractility**, the **inotropic** state of the heart.

- A **higher EF** indicates greater efficiency, while a **lower EF** indicates reduced efficiency.
  For example, if the end-diastolic volume is **125 ml** and the stroke volume is **70 ml**, the ejection fraction EF is= **70 ÷ 125 × 100 ≈ 56%**. If the stroke volume increases (e.g., to **80 ml**), the ejection fraction rises; if it decreases (e.g., to **60 ml**), the ejection fraction falls.

- A **positive inotropic effect** increases ejection fraction, whereas a **negative inotropic effect** decreases it.

- If cardiac output, heart rate, and end-systolic volume are known, the end-diastolic volume can be obtained by adding the **stroke volume** to the **end-systolic volume**, allowing the ejection fraction to be calculated.

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

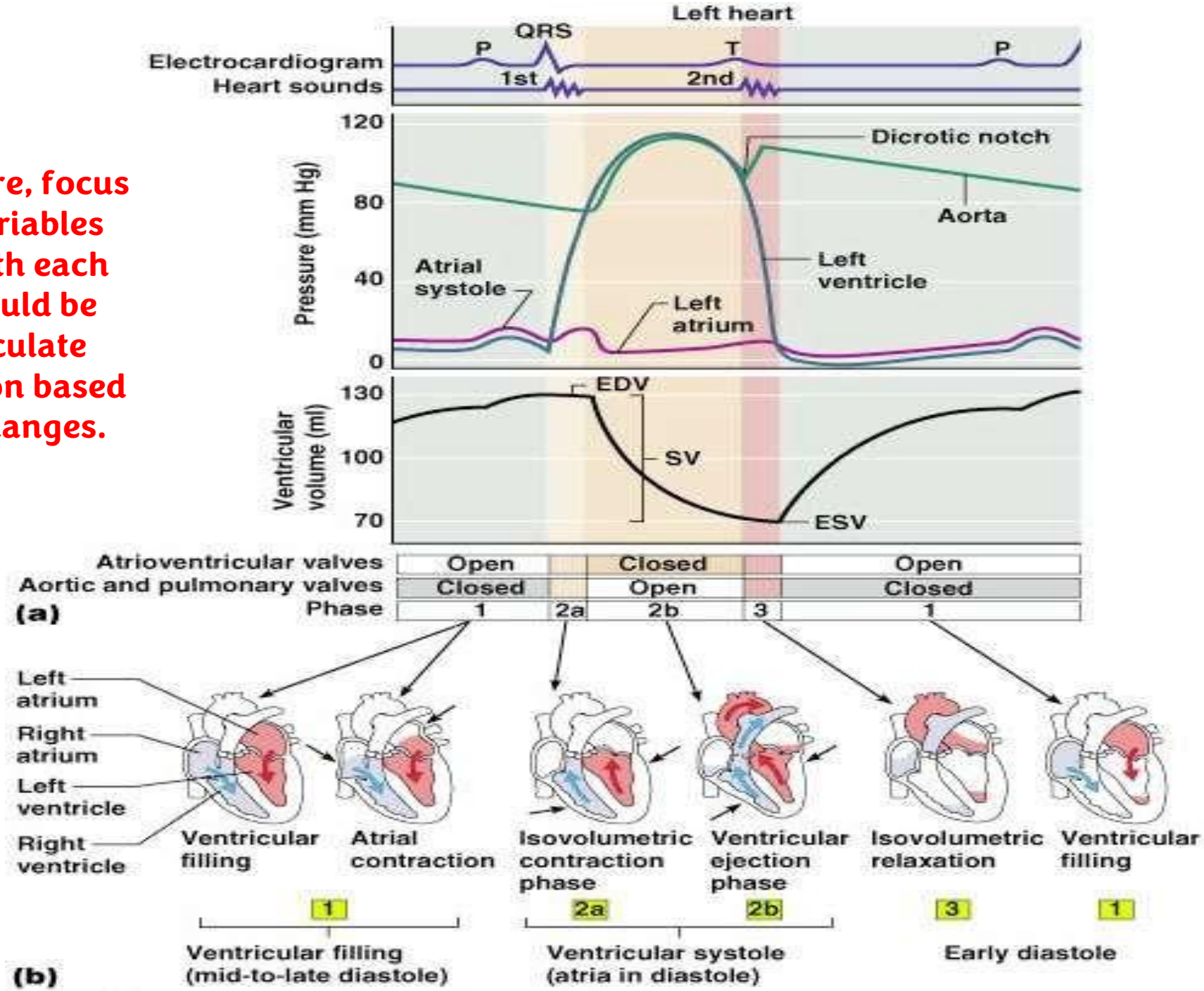
Phases of the Cardiac Cycle

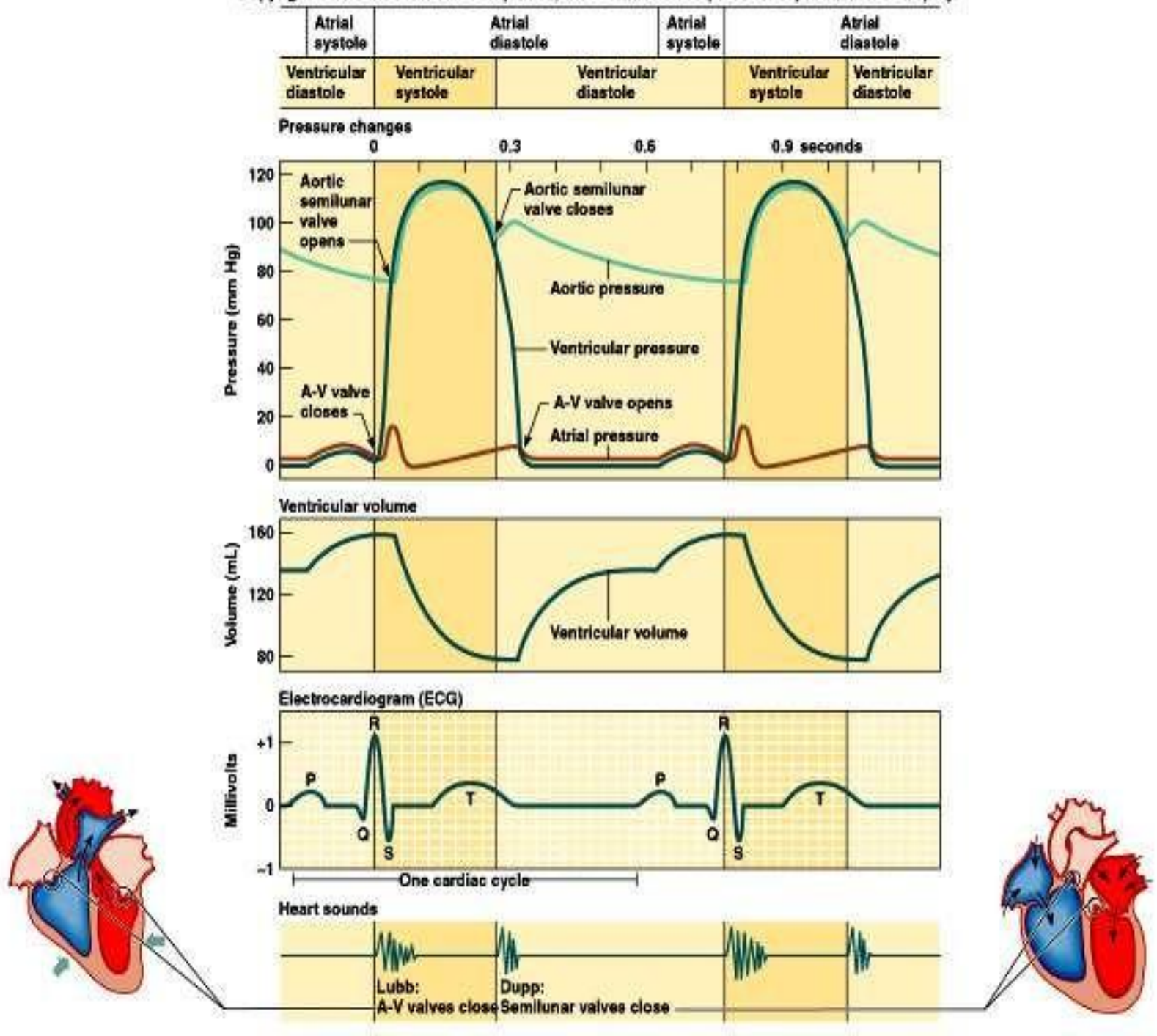
- Ventricular systole
  - Atria relax
  - Rising ventricular pressure results in closing of AV valves
  - Isovolumetric contraction phase
  - Ventricular ejection phase opens semilunar valves

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

Important figure, focus on how all variables interrelate with each other, you could be asked to calculate ejection fraction based on volume changes.

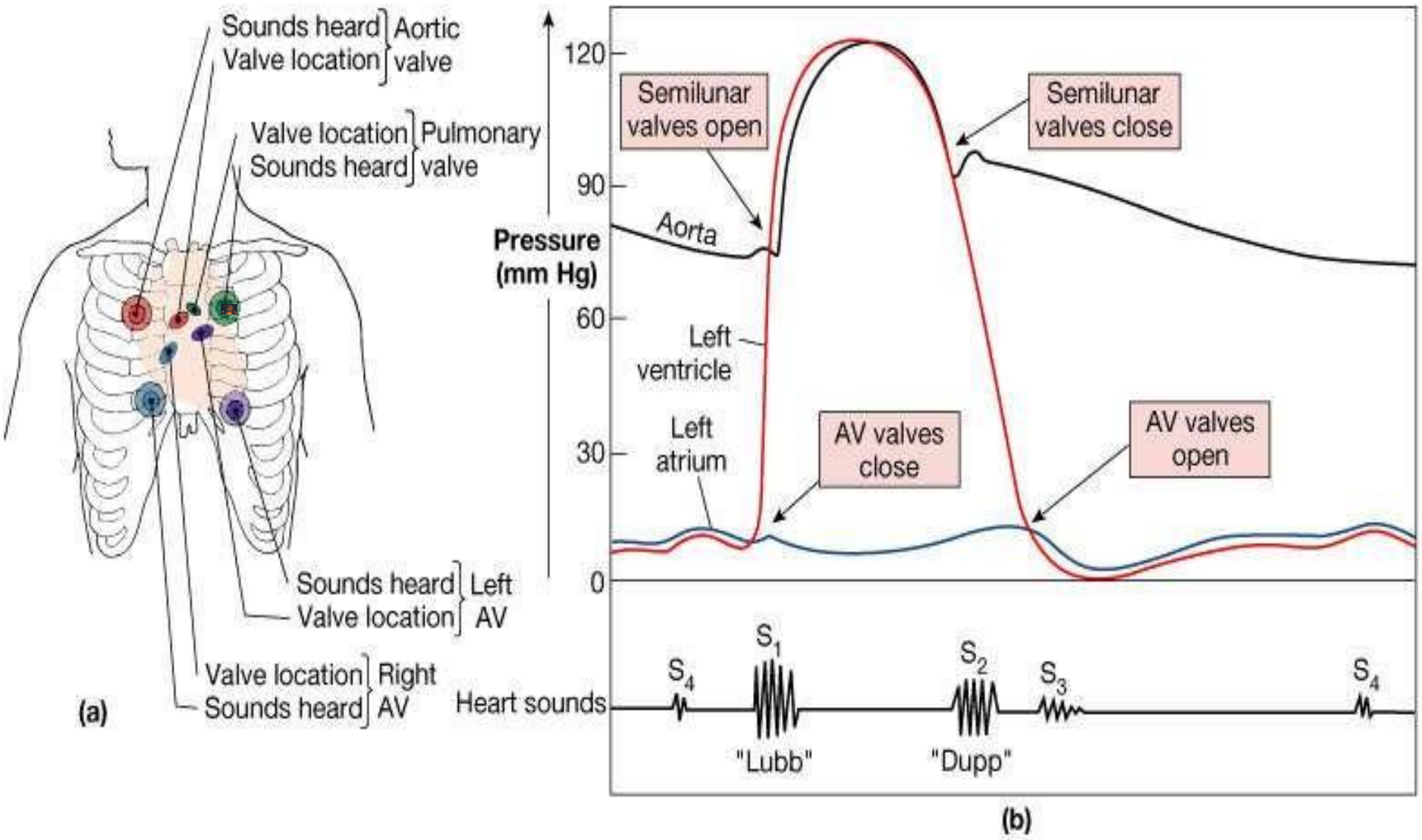




Changes during Cardiac cycle

- 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, systolic 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.

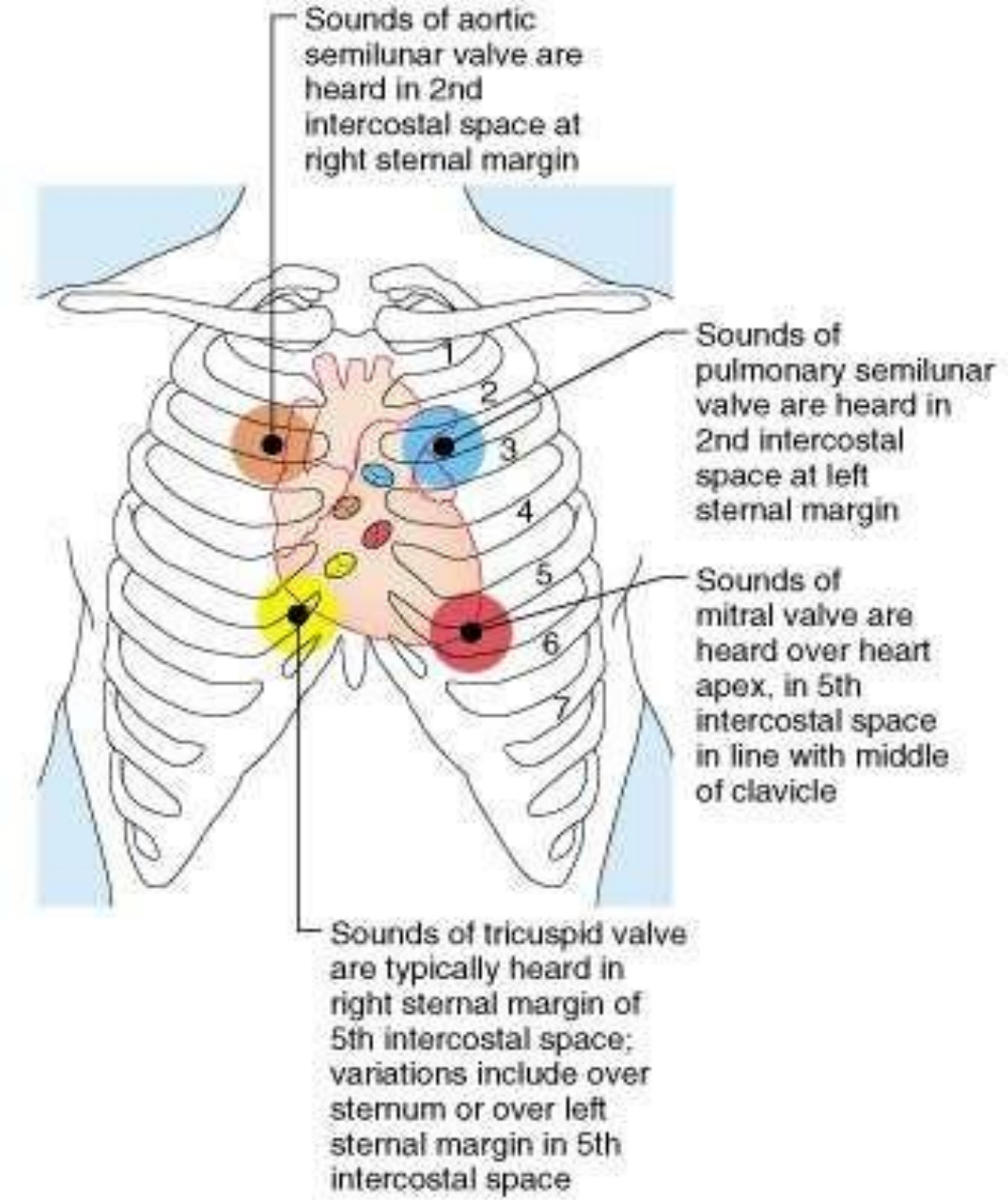
Heart Sounds



You won't be asked about 3rd and 4th 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 (Tricuspid and Bicuspid valves)
  - S_2 – “dupp” caused by the closing of the semilunar valves (Aortic and pulmonary) Normally they close together but in children sometimes the 2^{nd} heart sound might be split.
  - 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 (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

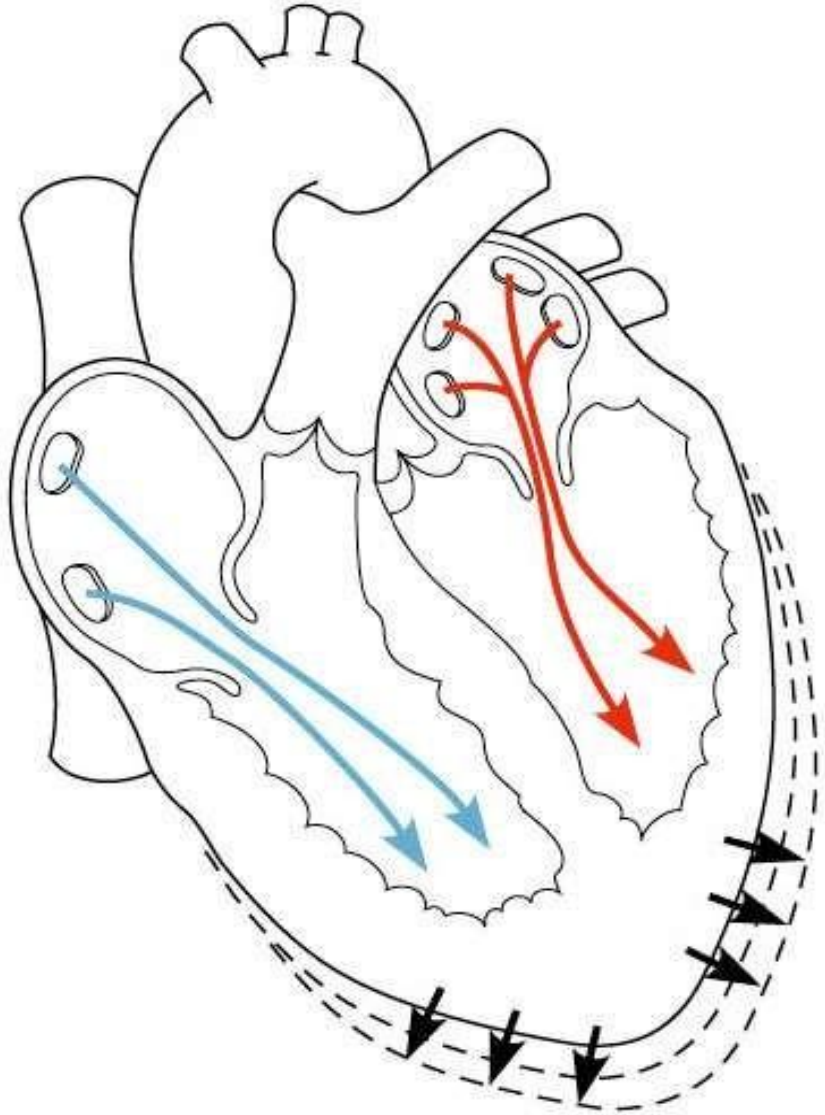
Factors Affecting Stroke Volume

- Preload –is the amount the ventricles are stretched by contained blood before it contracts, (passive tension according to Frank-Starling law) and is reflected as end diastolic volume.
- Contractility – cardiac cell contractile force due to factors independent of end-diastolic volume, and is commonly assessed using the **ejection fraction**.
- Afterload – back pressure exerted by blood in the large arteries leaving the heart, it is the **resistance** the ventricle must overcome to eject blood. It is influenced by **arterial pressure**, which is often approximated by **aortic diastolic pressure**, but it is **not equal to a fixed value such as 80 mmHg**. It represents the **force the ventricle must generate to open the semilunar valve**. A higher afterload requires **more energy** to maintain the same stroke volume.**The more the afterload the more energy to keep the stroke volume the same.**
- This explains the treatment of **hypertension**: increased afterload forces the heart to work harder, raising oxygen demand. In patients with reduced coronary flow, this can lead to **angina and ischemia**. Reducing afterload **decreases cardiac workload**, which is why antihypertensive therapy protects the heart.

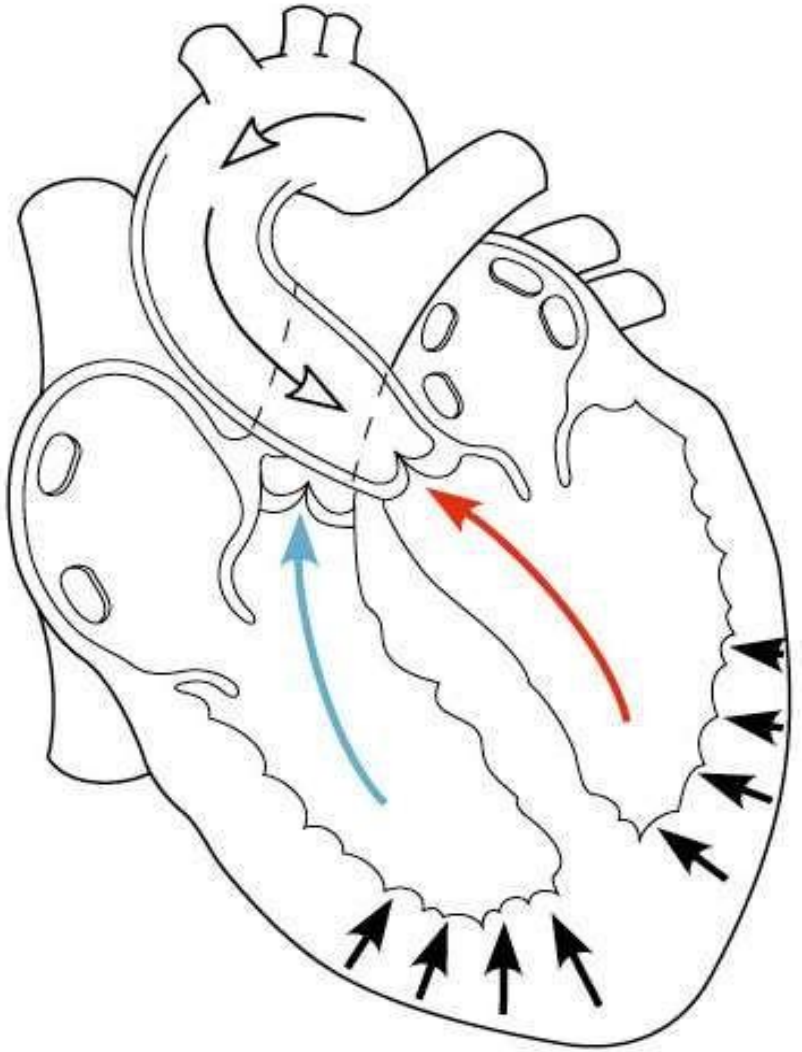
Frank-Starling Law of the Heart

- Preload, or degree of stretch, of cardiac muscle cells before they contract (the passive tension) 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

Preload and Afterload



(a) Preload



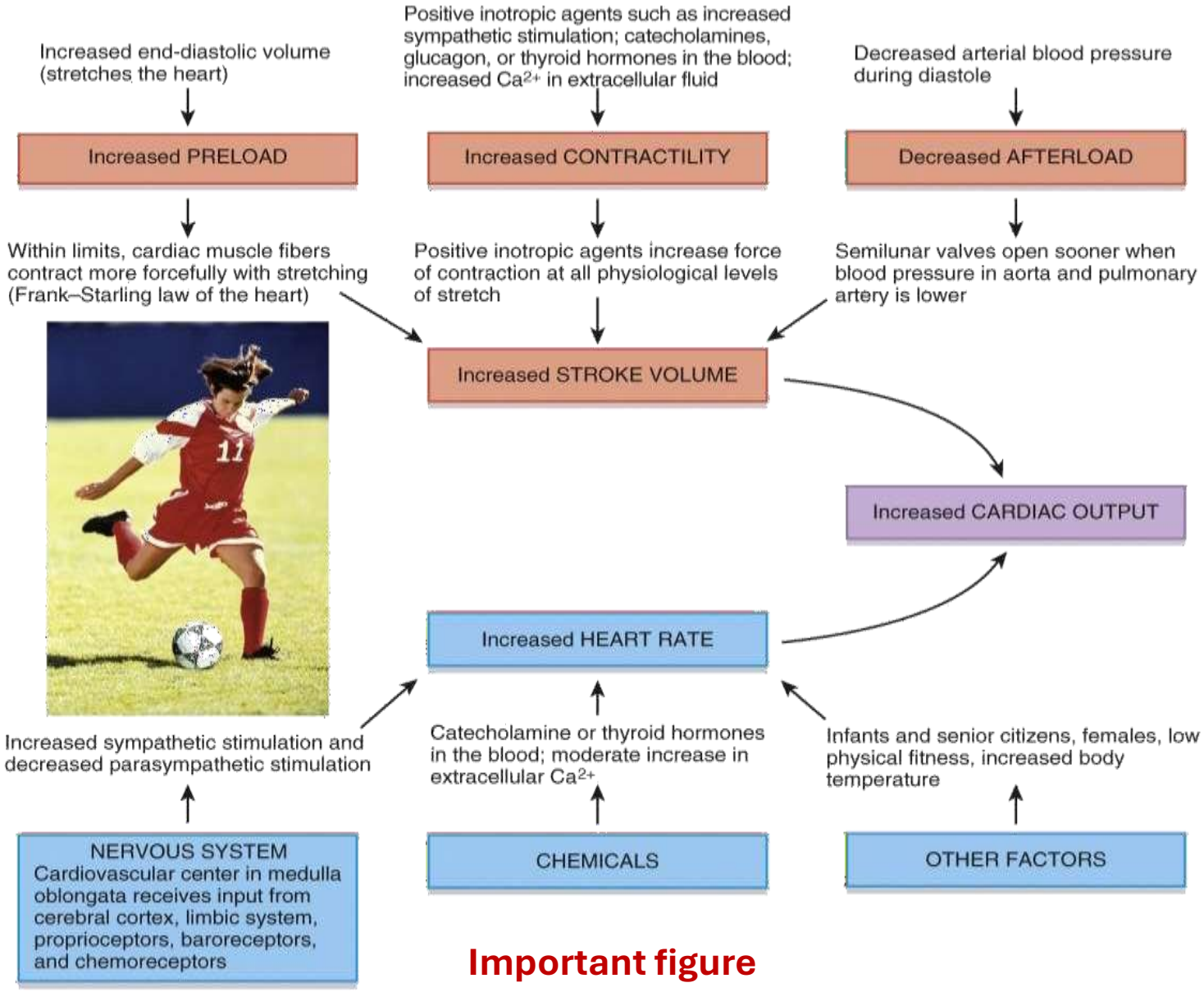
(b) Afterload

Cardiac Output

To increase the cardiac output, we either increase the stroke volume or the heart rate or both.

Frank-Starling mechanism within physiological limits, **cardiac muscle contracts more forcefully when it is stretched.**

Thyroid hormones are positive ionotropic agents; glucagon is also a positive ionotropic agent but doesn't increase the heart rate. Contrary to what the doctor said, glucagon is a positive inotropic and chronotropic agent, so it does raise heart rate.



Important figure

Heat can **raise heart rate**, which may be **more problematic in children.**

Impact of Faster Heart Rates on Diastolic Filling and Cardiac Output

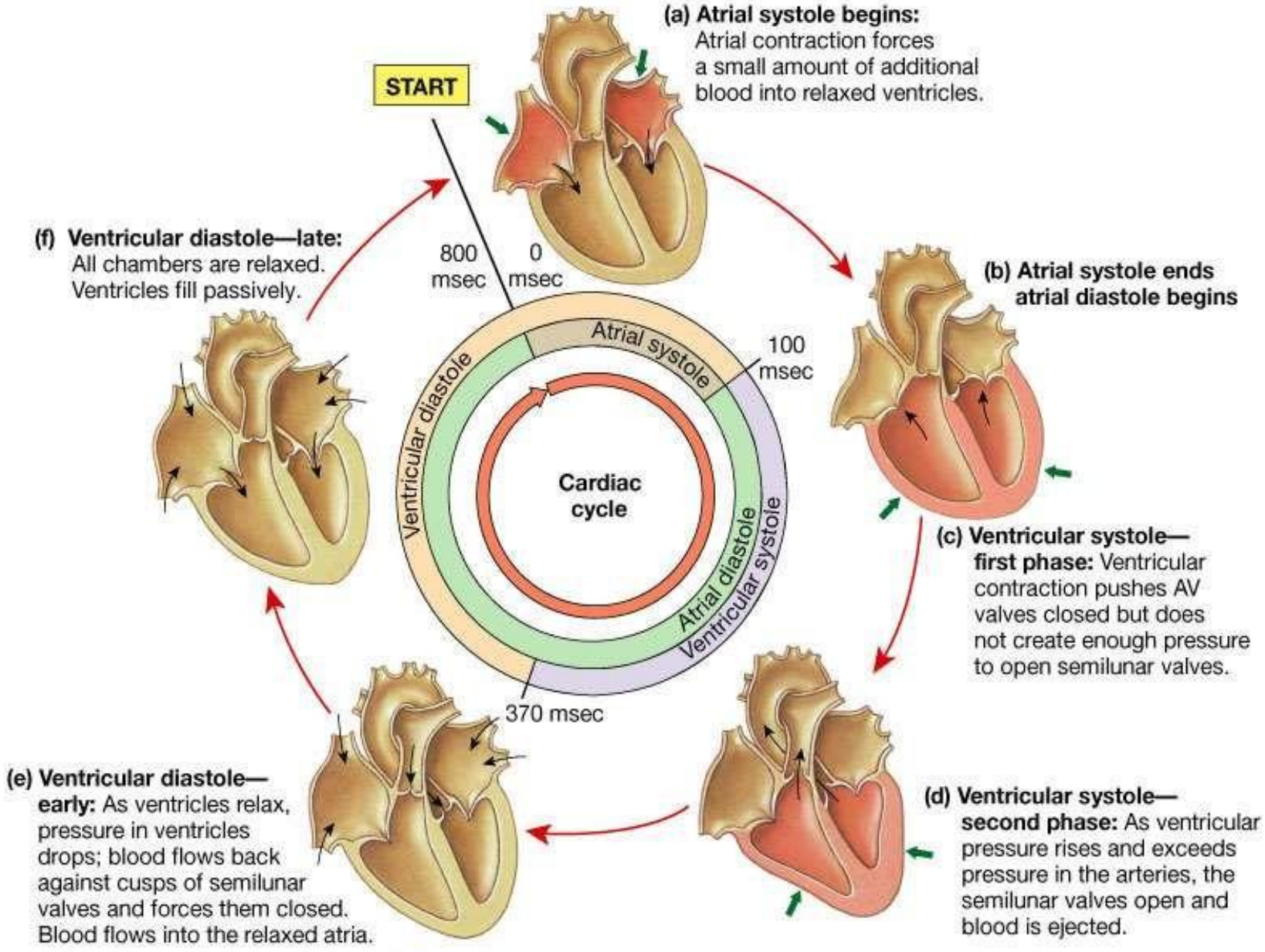
- At a heart rate of **75 bpm**, one cardiac cycle lasts **0.8 s** (about **0.3 s** for systole and **0.5 s** for diastole).

- If the heart rate increases to **100 bpm**, the cardiac cycle shortens to **0.6 s**, so systole becomes **0.29 s** and diastole **0.31 s**. This reduces filling time → less filling → less blood ejected → a lower stroke volume.

- Within the normal range, the increase in heart rate can compensate for the fall in stroke volume, so **cardiac output still rises**.

- Heart rate should not exceed **80–90%** of the **maximum heart rate that is (220 bpm – age)**, above this level, diastole becomes too short, stroke volume falls further, and the higher heart rate can no longer compensate. As a result, **cardiac output decreases**, which may lead to ischemia.

Phases of the Cardiac Cycle



PHYSIOLOGY QUIZ

LECTURE 7

External Resources

رسالة من الفريق العلمي

- *Guyton and Hall Textbook of Medical Physiology* 14th ed.

- *Comprehensive File 2:* https://doctor2023.jumedicine.com/wp-content/uploads/sites/15/2024/04/Conducting-L2-1.pdf

"Fever is not a sign of ceftriaxone deficiency"
-Unknown

Scan the QR code or click it for FEEDBACK

Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1	14; bottom figure 39; Quiz	Wrong arrow alignments Link no working	Corrected arrow alignments Fixed (in fact it was fixed and uploaded before this V1 issue)
V1 → V2	10; Point 7 (subpoint 4)	This brief rise in aortic pressure after ventricular diastole creates the **dicrotic notch** on the aortic pressure curve.	This brief rise in aortic pressure at the beginning of ventricular diastole (just after ventricular systole) creates the **dicrotic notch** on the aortic pressure curve.