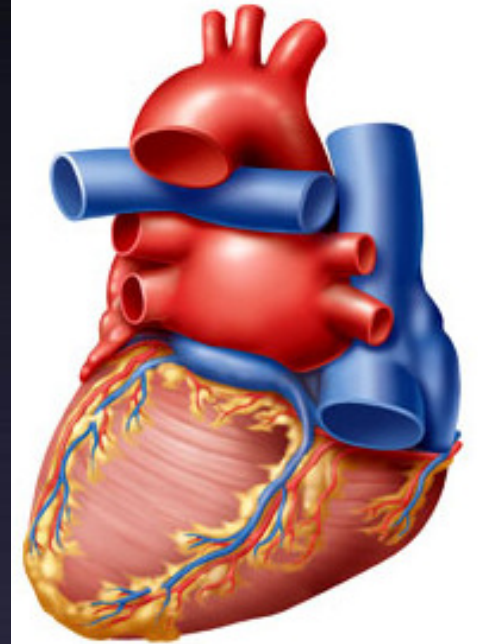
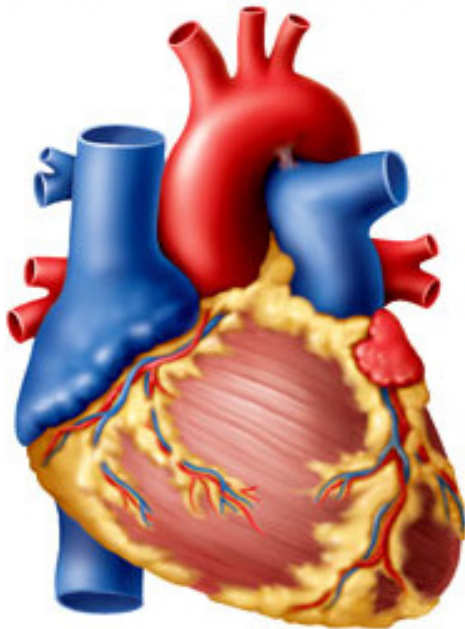




CARDIOVASCULAR SYSTEM

CARDIAC CYCLE



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Dept of Physiology
King Saud University

OBJECTIVES

❖ **At the end of the lecture you should be able to**

- 1. Enumerate the phases of cardiac cycle**
- 2. Explain the effect of heart rate on duration of systole and diastole**
- 3. Recognize the pressure, electrical, sound and volume changes during cardiac cycle**
- 4. Correlate different phases of cardiac cycle with various changes in events.**
- 5. Describe heart sounds and their clinical importance**
- 6. Explain regulation of heart pumping**



CARDIAC CYCLE

- **Definition:** Cardiac Cycle is the time duration comprising all the events from beginning of one heart contraction to the beginning of next heart contraction.
- At heart rate of 75 beats per minute duration of one Cardiac cycle is **0.8** second.

What are the Events?

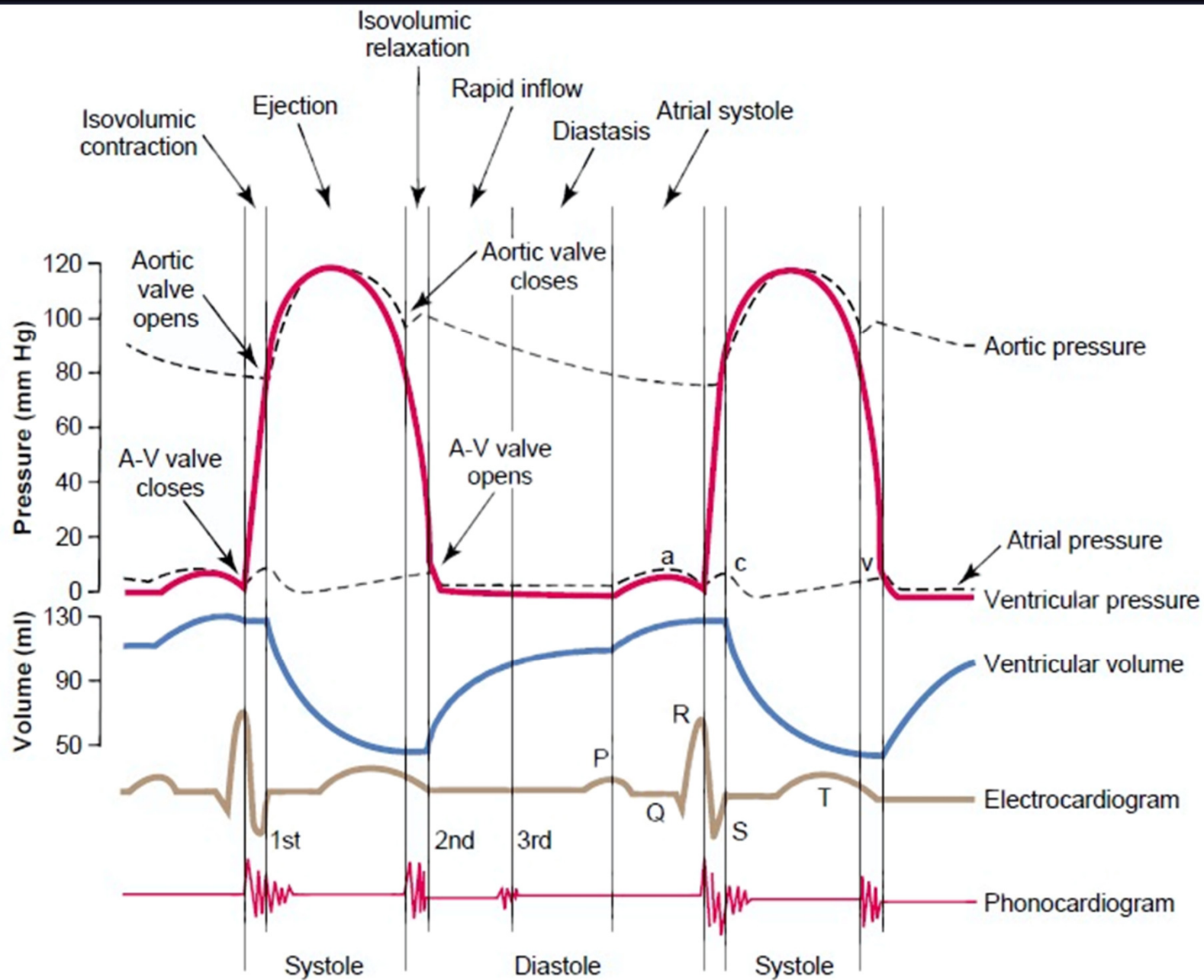
EVENTS OF CARDIAC CYCLE

Mechanical changes:

1. Phases of cardiac cycle
2. Heart sounds
3. Pressure changes during cardiac cycle
4. Volume changes during cardiac cycle

Electrical Changes

5. Electrocardiogram (ECG)



The Events of the Cardiac Cycle

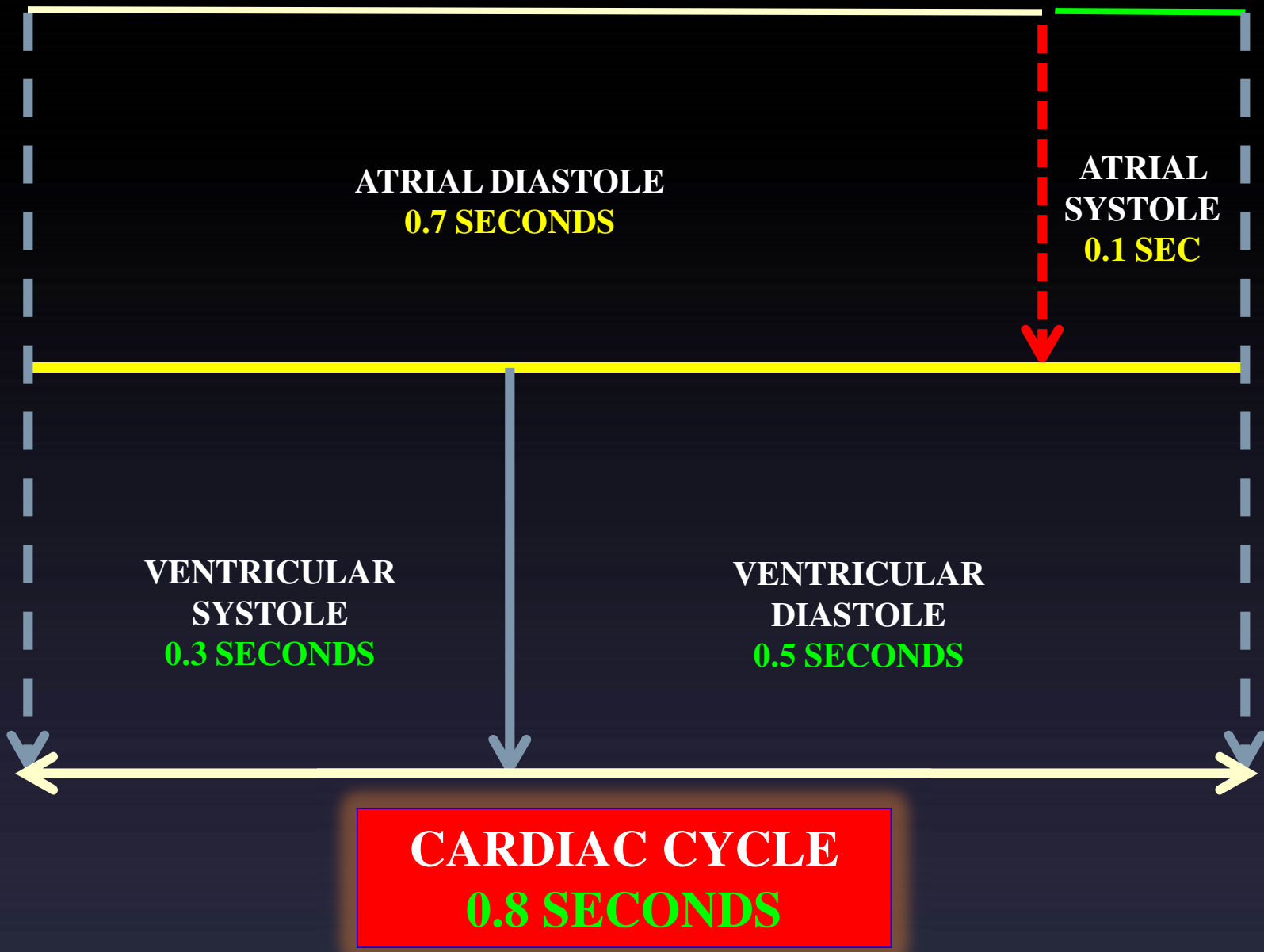
PHASES OF CARDIAC CYCLE

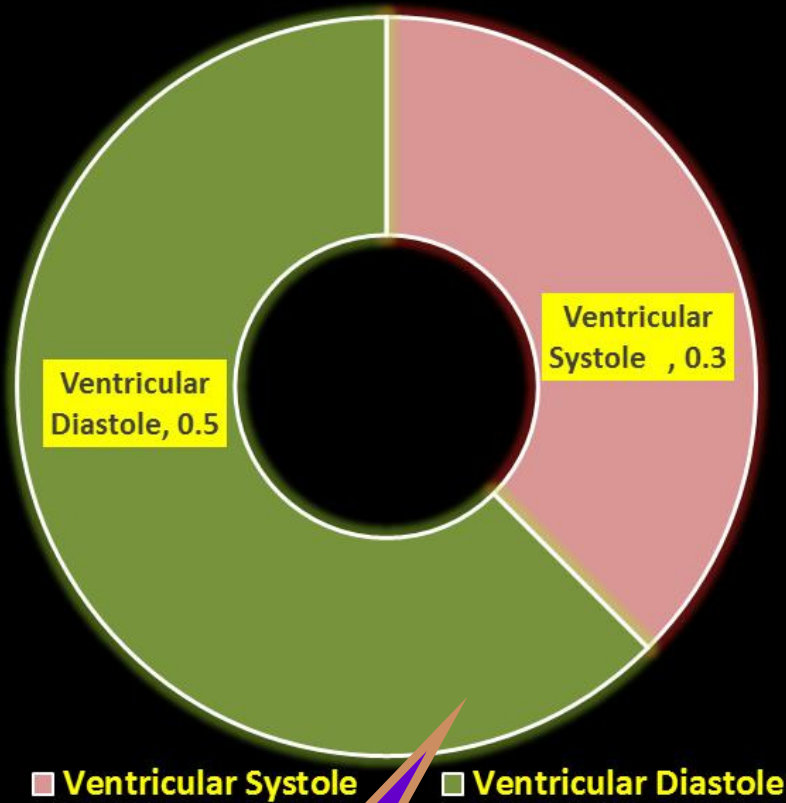
Atrial Events

- ❖ Atrial systole: 0.1 second
- ❖ Atrial diastole : 0.7 seconds

Ventricular Events

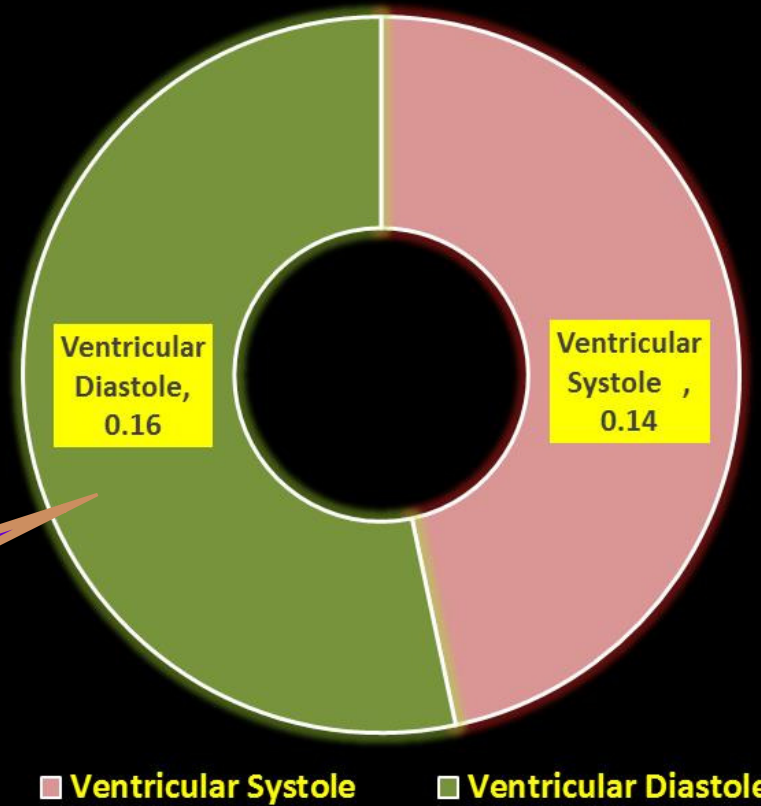
- ❖ Ventricular systole : 0.3 seconds
- ❖ Ventricular diastole : 0.5 seconds





At Rest

After Exercise



PHASES OF CARDIAC CYCLE

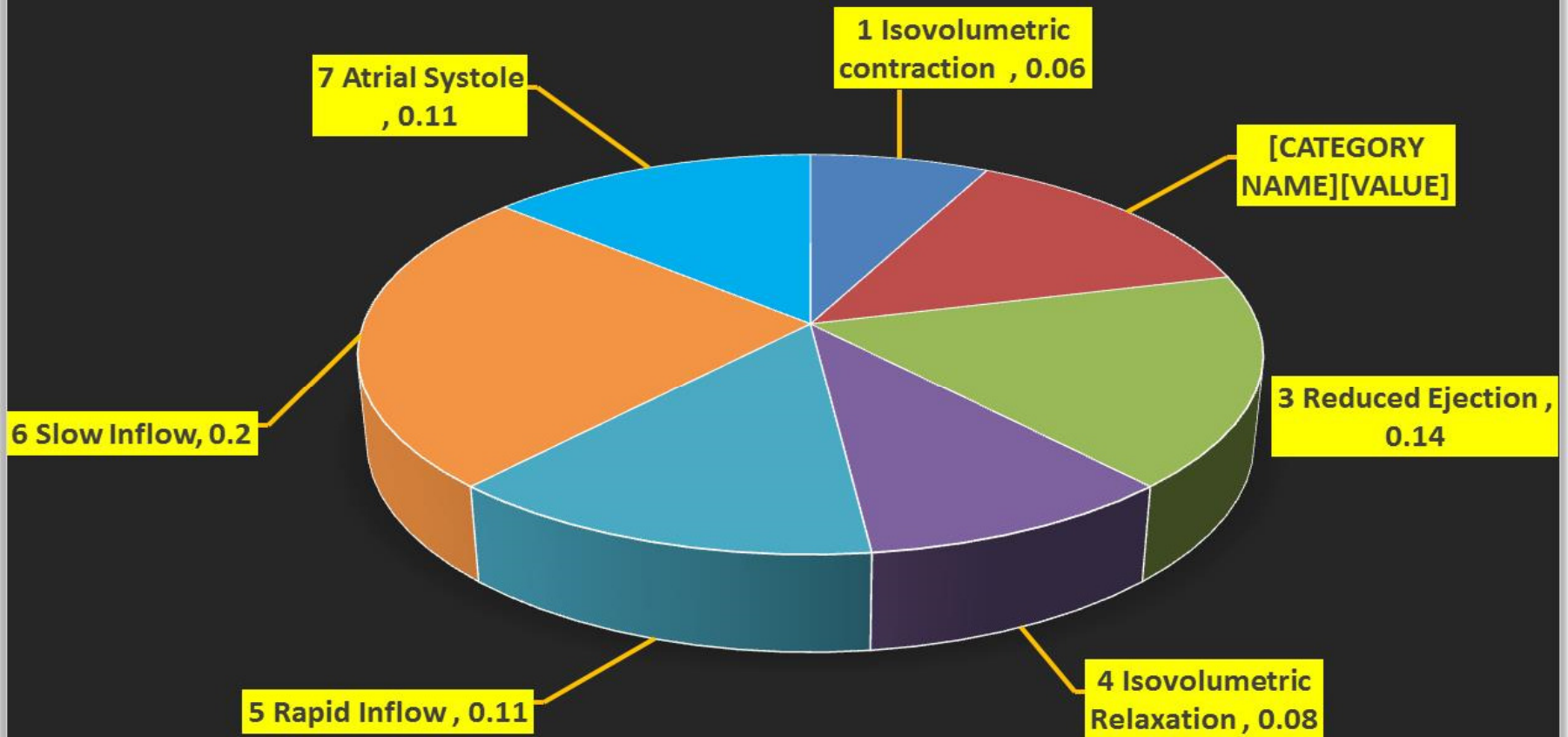


TABLE 30–1 Variation in length of action potential and associated phenomena with cardiac rate.^a

	Heart Rate 75/min	Heart Rate 200/min	Skeletal Muscle
Duration, each cardiac cycle	0.80	0.30	...
Duration of systole	0.27	0.16	...
Duration of action potential	0.25	0.15	0.007
Duration of absolute refractory period	0.20	0.13	0.004
Duration of relative refractory period	0.05	0.02	0.003
Duration of diastole	0.53	0.14	...

^aAll values are in seconds.

Courtesy of AC Barger and GS Richardson.

VENTRICULAR EVENTS

- **Ventricular systole**

1. Isovolumetric contraction

2. Ejection phase

- ✓ Rapid ejection

- ✓ Slow ejection

- **Ventricular Diastole**

1. Isovolumetric relaxation phase

2. Filling phase

- ✓ Rapid filling

- ✓ Slow filling (Continued filling)

- ✓ Last rapid filling (Atrial Systole)

VENTRICULAR SYSTOLE **0.31 sec**

(Peak of R wave of QRS complex to the end of T wave)

ISO-VOLUMETRIC CONTRACTION 0.06 sec

MAXIMUM EJECTION (2/3) 0.11 sec

REDUCED EJECTION (1/3) 0.14 sec

VENTRICULAR DIASTOLE **0.52 sec**

(End of T wave to the peak of R wave of QRS complex)

ISO-VOLUMETRIC RELAXATION 0.06 sec

RAPID INFLOW 0.11 sec

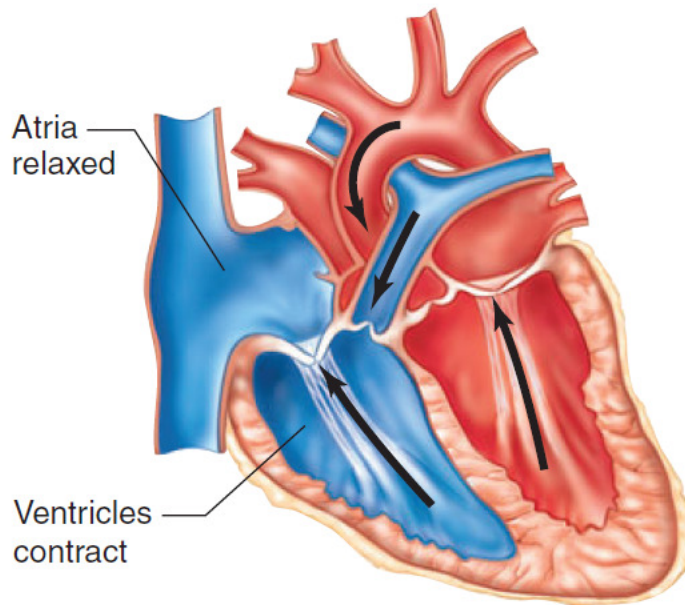
SLOW INFLOW / DIASTASIS 0.2 sec

ATRIAL SYSTOLE (after P wave) 0.11 sec

7 Phases of CARDIAC CYCLE 0.8 sec

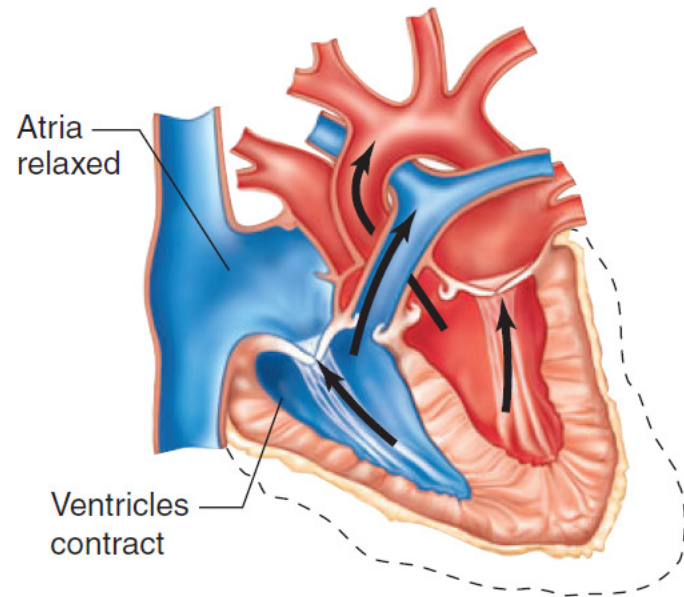
A Systole

Isovolumetric ventricular contraction



Ventricular ejection

Blood flows out of ventricle



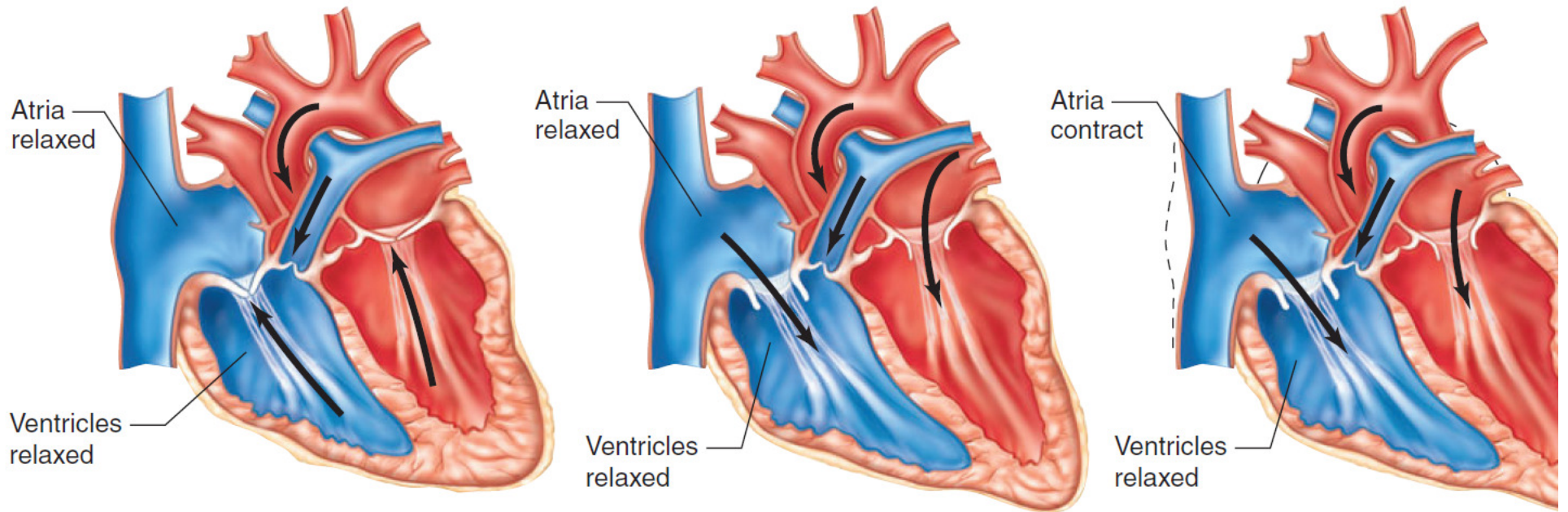
AV valves:	Closed	Closed
Aortic and pulmonary valves:	Closed	Open

B Diastole

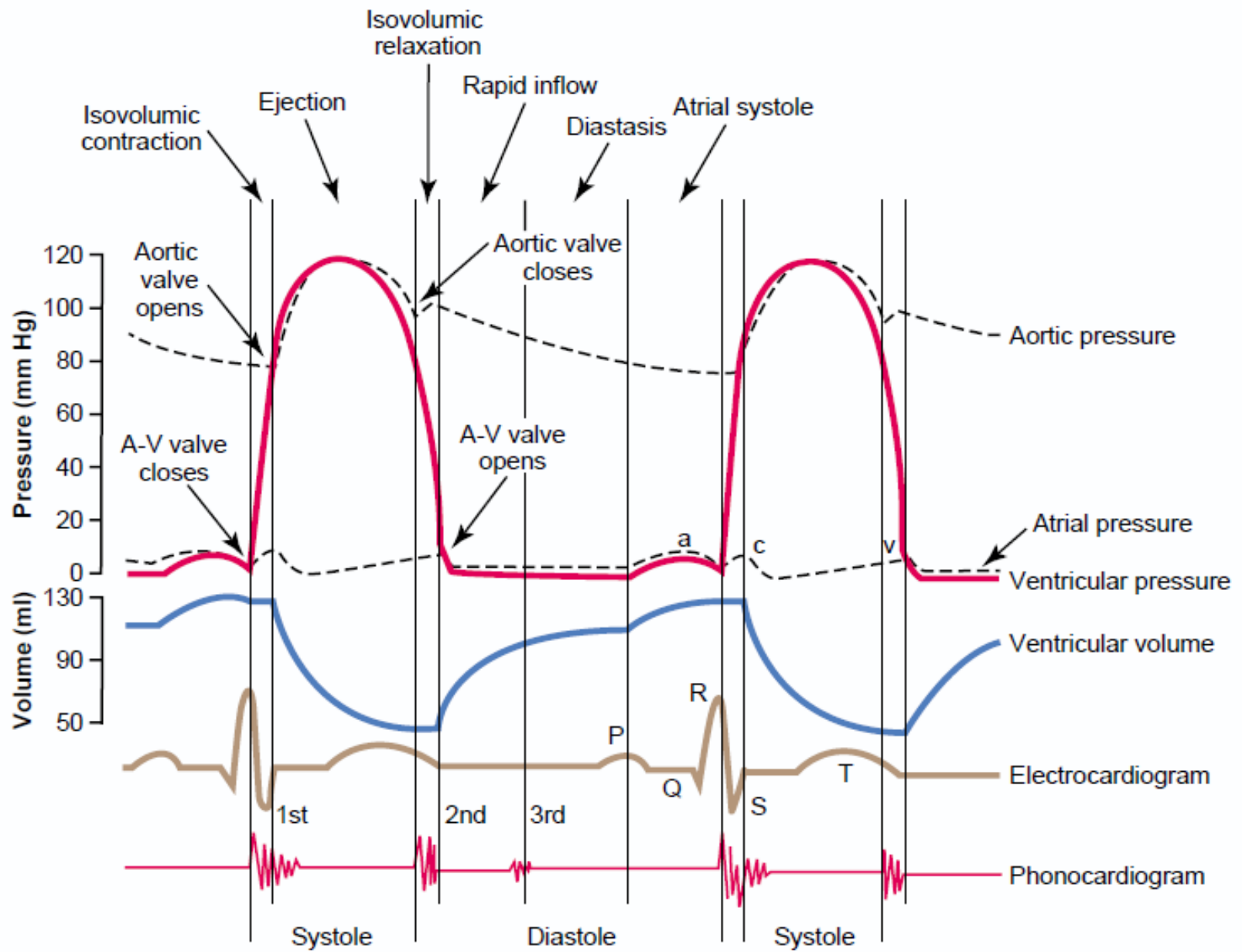
Isovolumetric ventricular relaxation

Ventricular filling Blood flows into ventricles

Atrial contraction



AV valves:	Closed	Open	Open
Aortic and pulmonary valves:	Closed	Closed	Closed



ATRIAL SYSTOLE

Atrial Depolarization



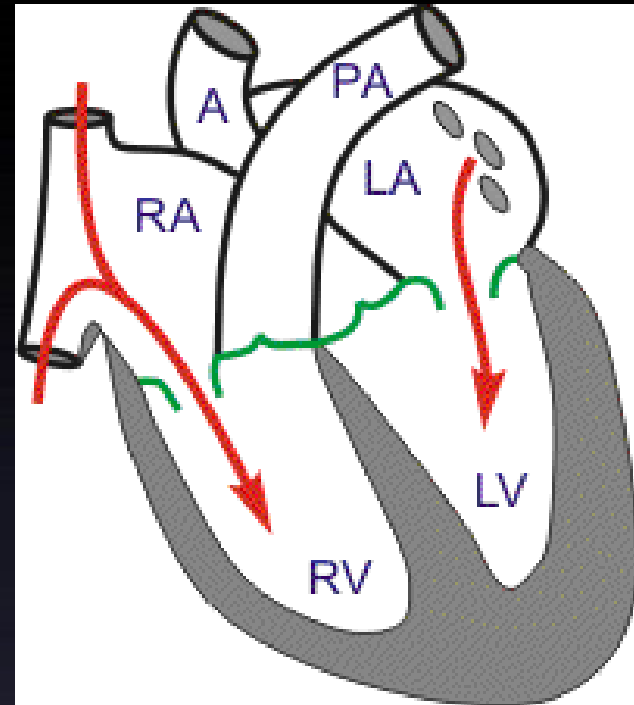
Atrial contraction



Atrial pressures rise



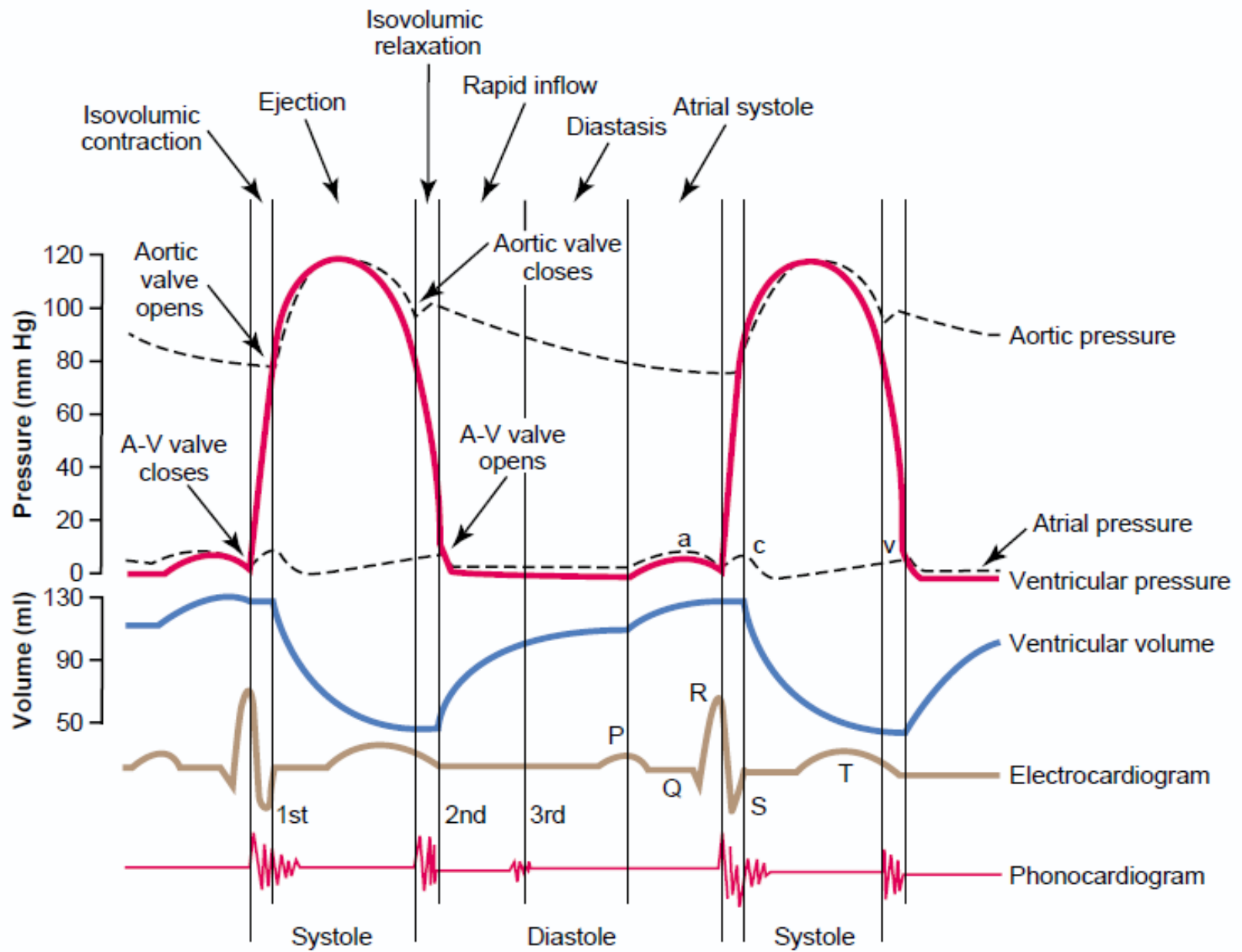
Blood flows across AV valves

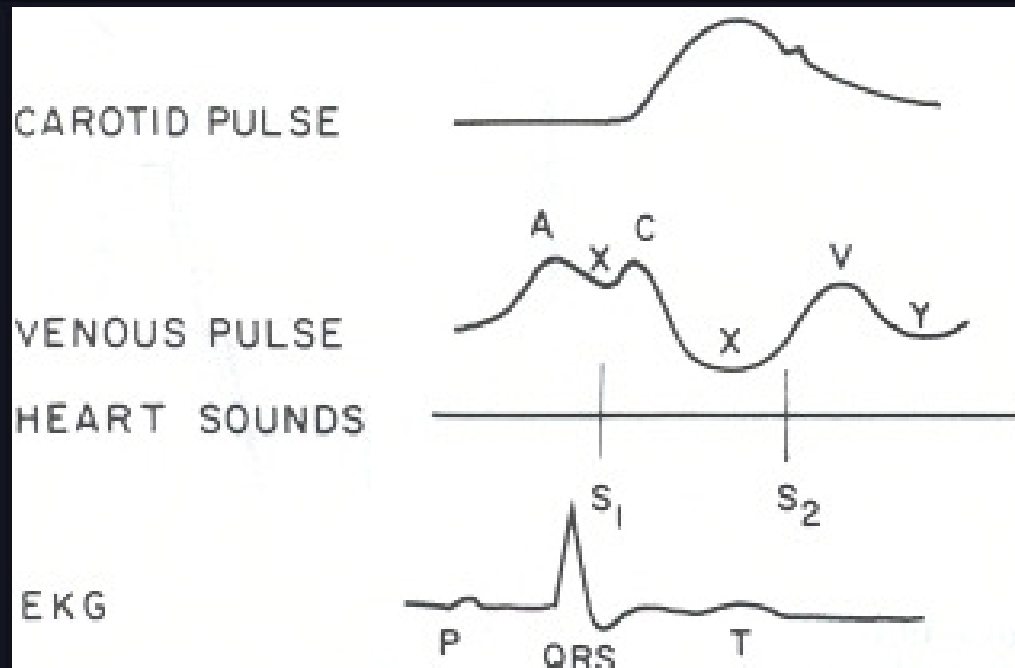


ATRIA = PRIMER PUMPS .

Ventricular filling :

- 80% - direct flow from SVC & IVC
- 20% - atrial contraction.





..... Atrial systole

- **JVP** – ‘a’ wave
- **ECG** – P wave precedes the atrial systole. PR interval – depolarization proceeds to the AVN. The brief pause allows complete ventricular filling
- **Heart sounds** - **S_4** – **pathological**. Vibration of the ventricular wall during atrial contraction. Heard in ‘stiff’ ventricle like in hypertrophy and in elderly. Also heard in massive pulmonary embolism, cor pulmonale, TR

ISOVOLUMETRIC CONTRACTION

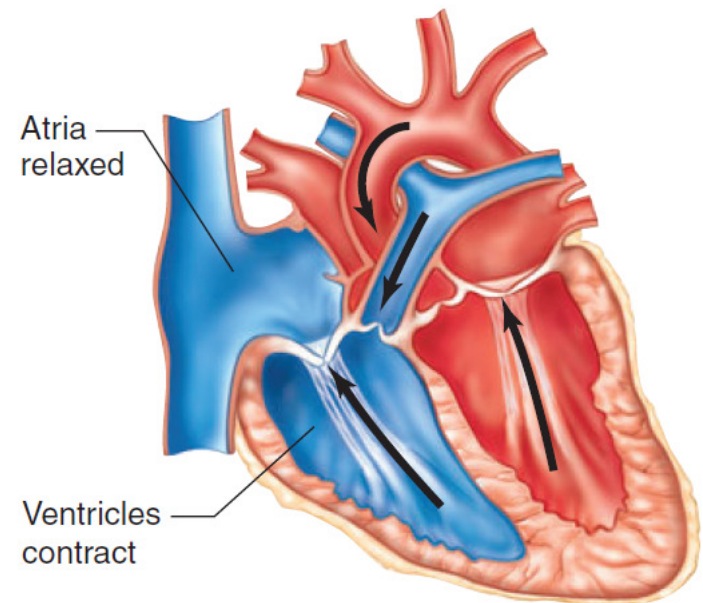
Increase in ventricular pressure $>$ atrial pressure \rightarrow AV valves close

After 0.02s, semilunar valves open

Period between AV valve closure and semilunar valve opening \rightarrow heart prepares for contraction without shortening \rightarrow occurs without emptying

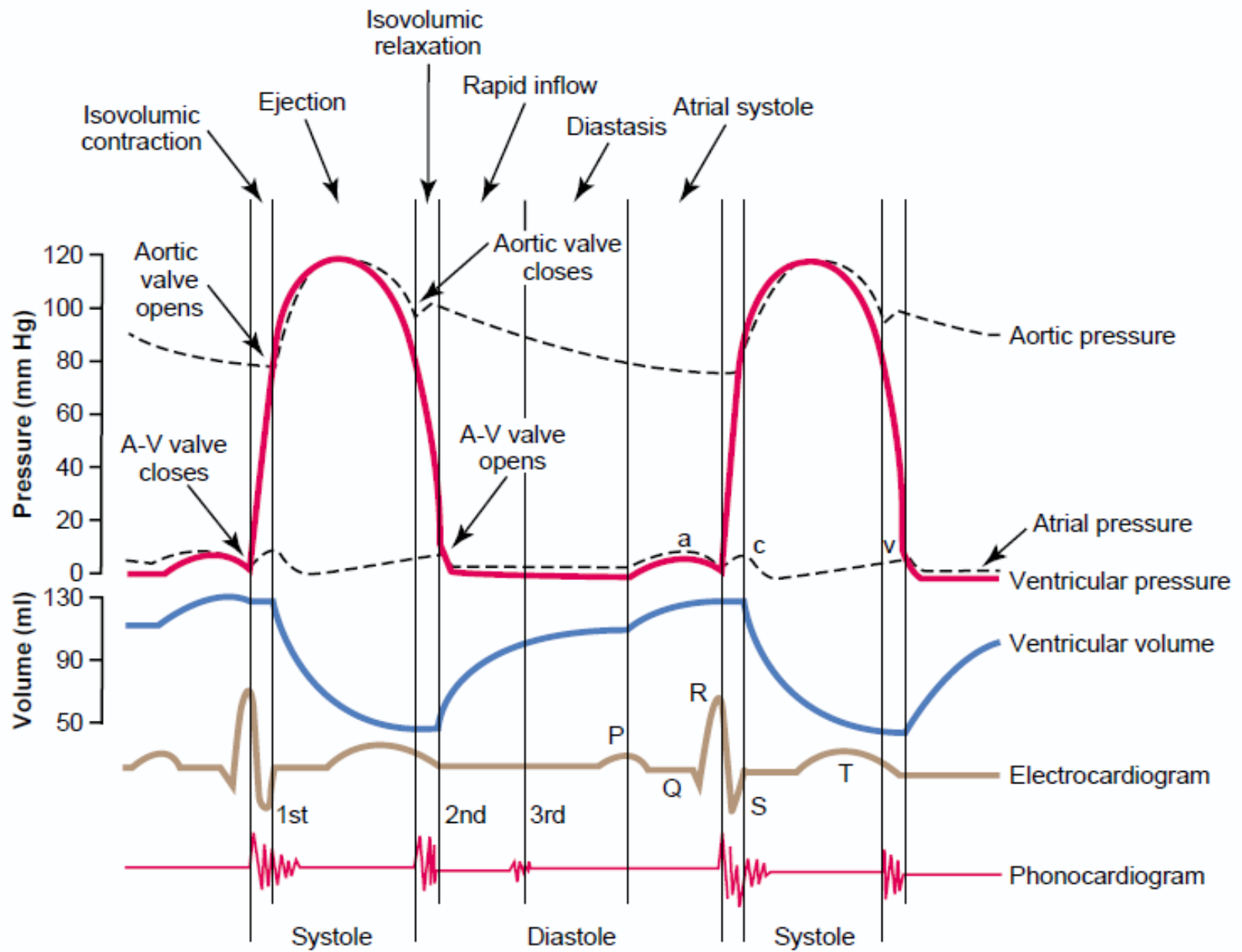
Tension develops without change in muscle length

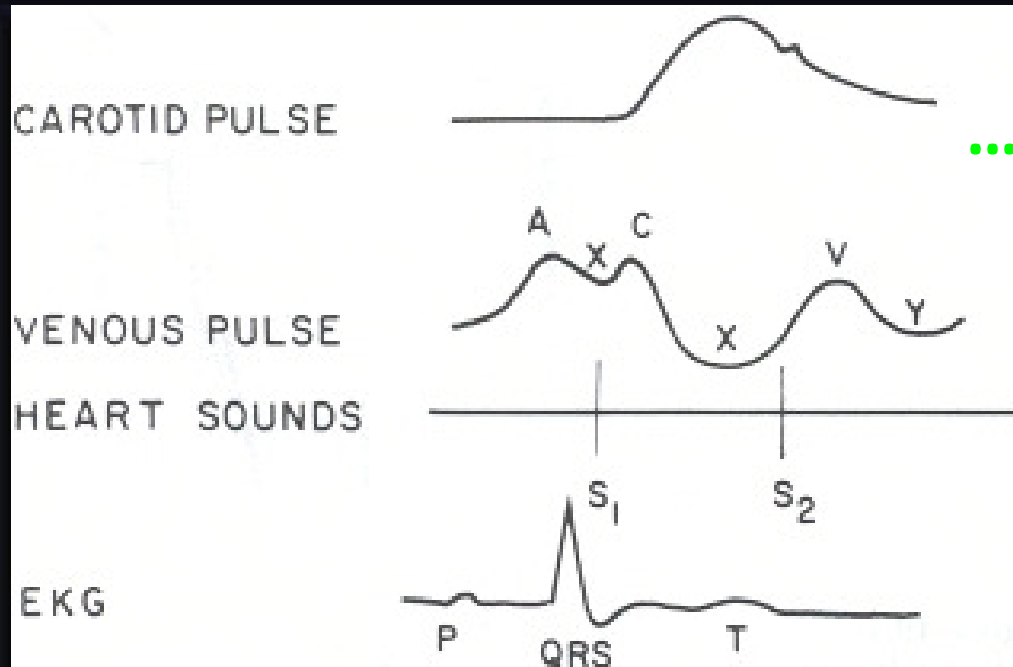
Isovolumetric ventricular contraction



AV valves: Closed

Aortic and pulmonary valves: Closed





... **Isovolumetric contraction**

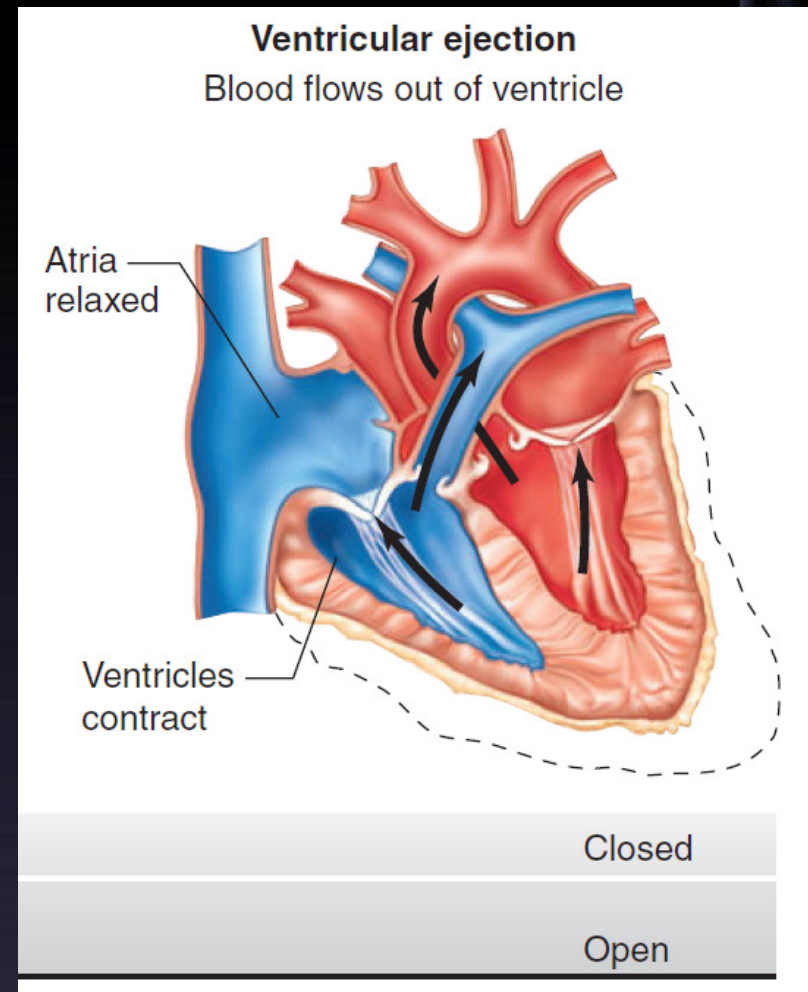
Volume does not change

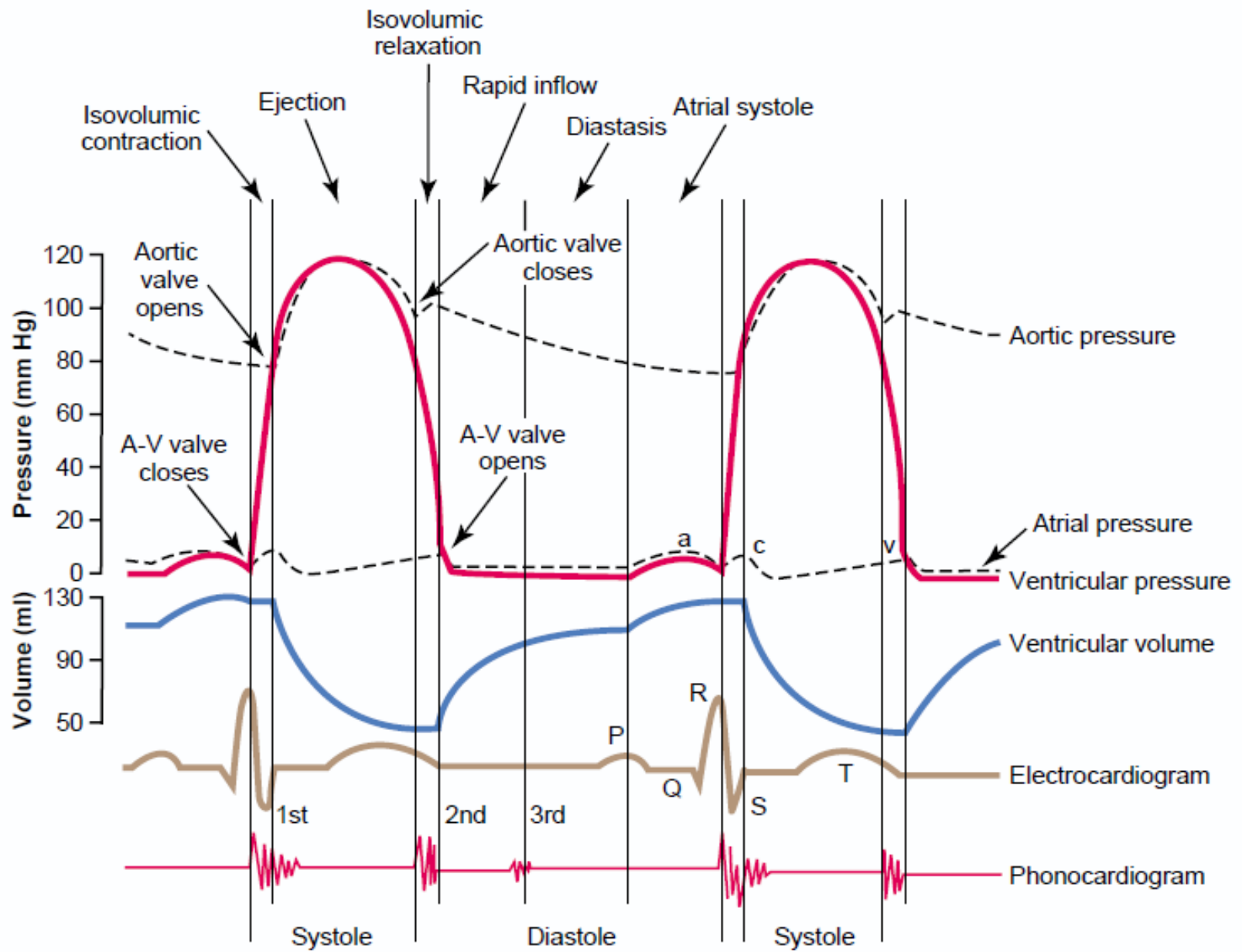
All valves are closed

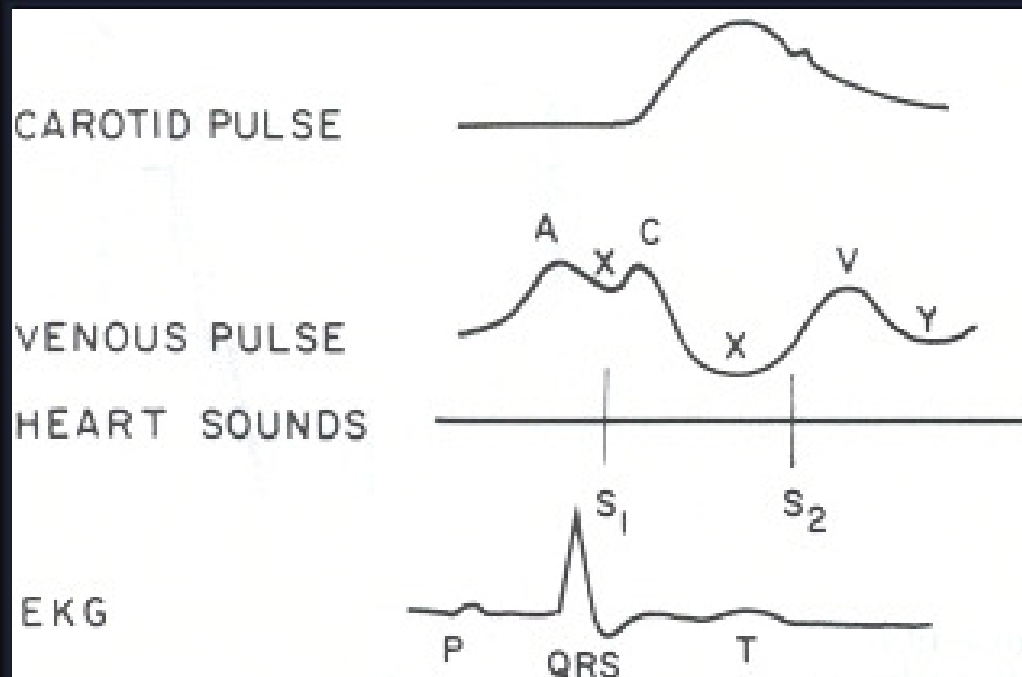
- **JVP** – ‘c’ wave → due to the bulging of the Tricuspid valve into RA secondary to increased pressure in the ventricle.
- **ECG** – End of QRS complex
- **Heart Sounds** – S_1 : closure of the AV valves.

EJECTION

- When LV pres > 80 mm Hg
RV pres > 8 mm Hg,
The semilunar valves open.
- Rapid Ejection – 70% emptying in first 1/3
- Slow Ejection – 30% in last 2/3
- The pressure in the ventricle keeps decreasing until it becomes lower than that of the great vessels







... ejection

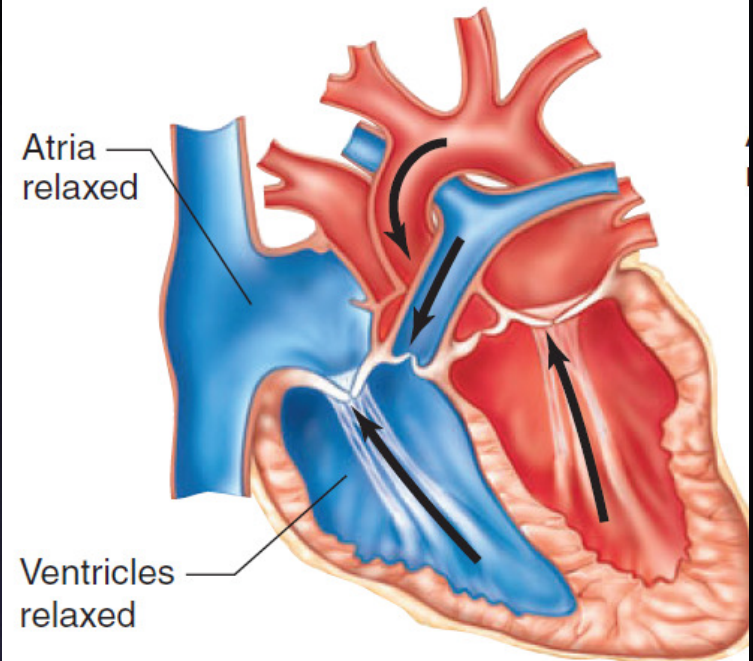
- **JVP** – no waves
- **ECG** – T wave
- **Heart sounds** – none
- **Aortic pressure** - Rapid rise in the pressure = 120 mm Hg. Even at the end of systole pressure in the aorta is maintained at 90 mm Hg because of the elastic recoil

ISOVOULUMETRIC RELAXATION

- When ventricle pressure $<$ arterial pressure \rightarrow backflow of blood \rightarrow forces semilunar valves to close.
- For 0.03-0.06 s, ventricle relaxes despite no change in its volume
- AV and Semilunar valves are closed
- Meanwhile, atria fill up and atrial pressure gradually rises
- Pressures in ventricle keep falling till it is $<$ atrial pressure

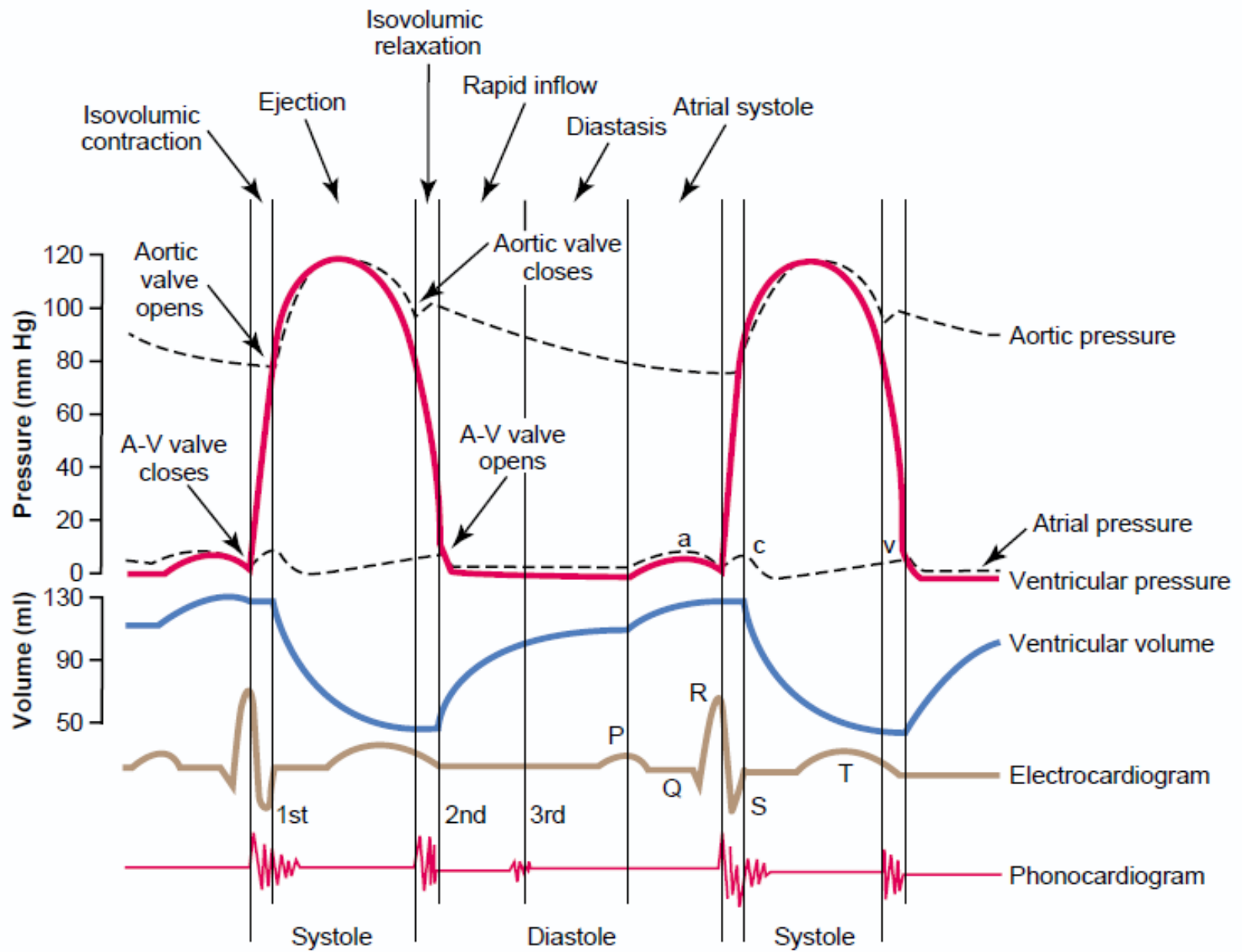
B Diastole

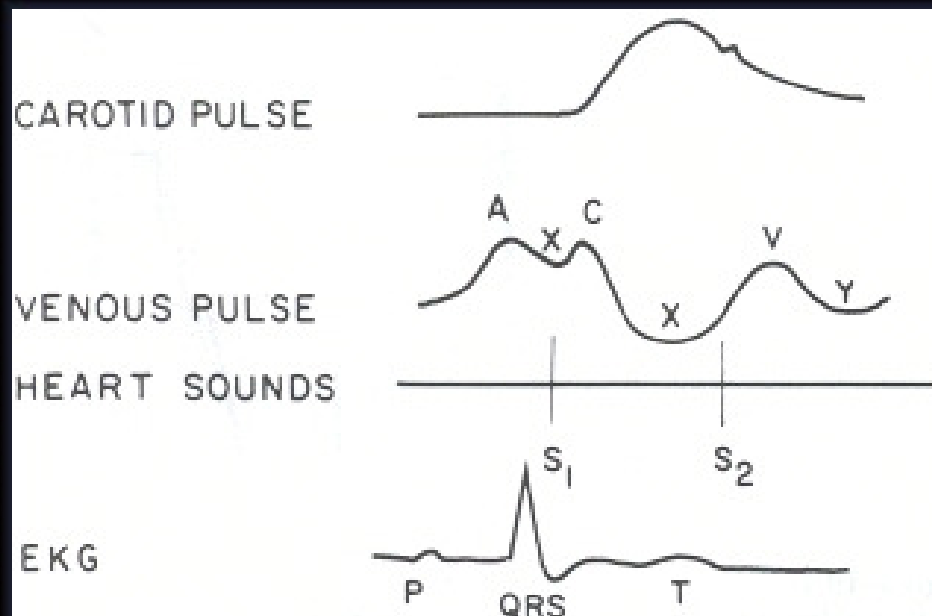
Isovolumetric ventricular relaxation



AV valves: Closed

Aortic and pulmonary valves: Closed





... isovolumetric relaxation

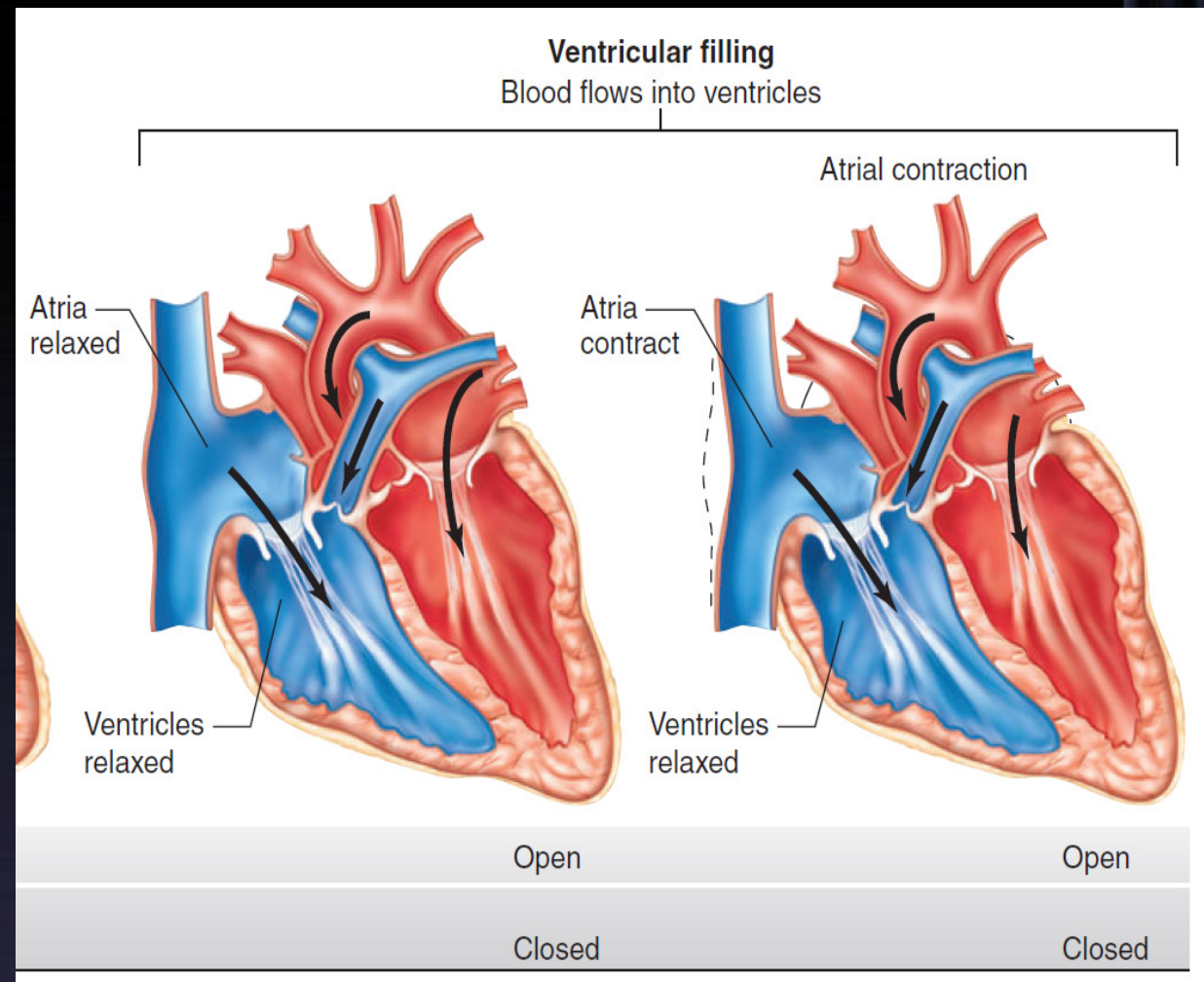
Volume does not change

All valves are closed

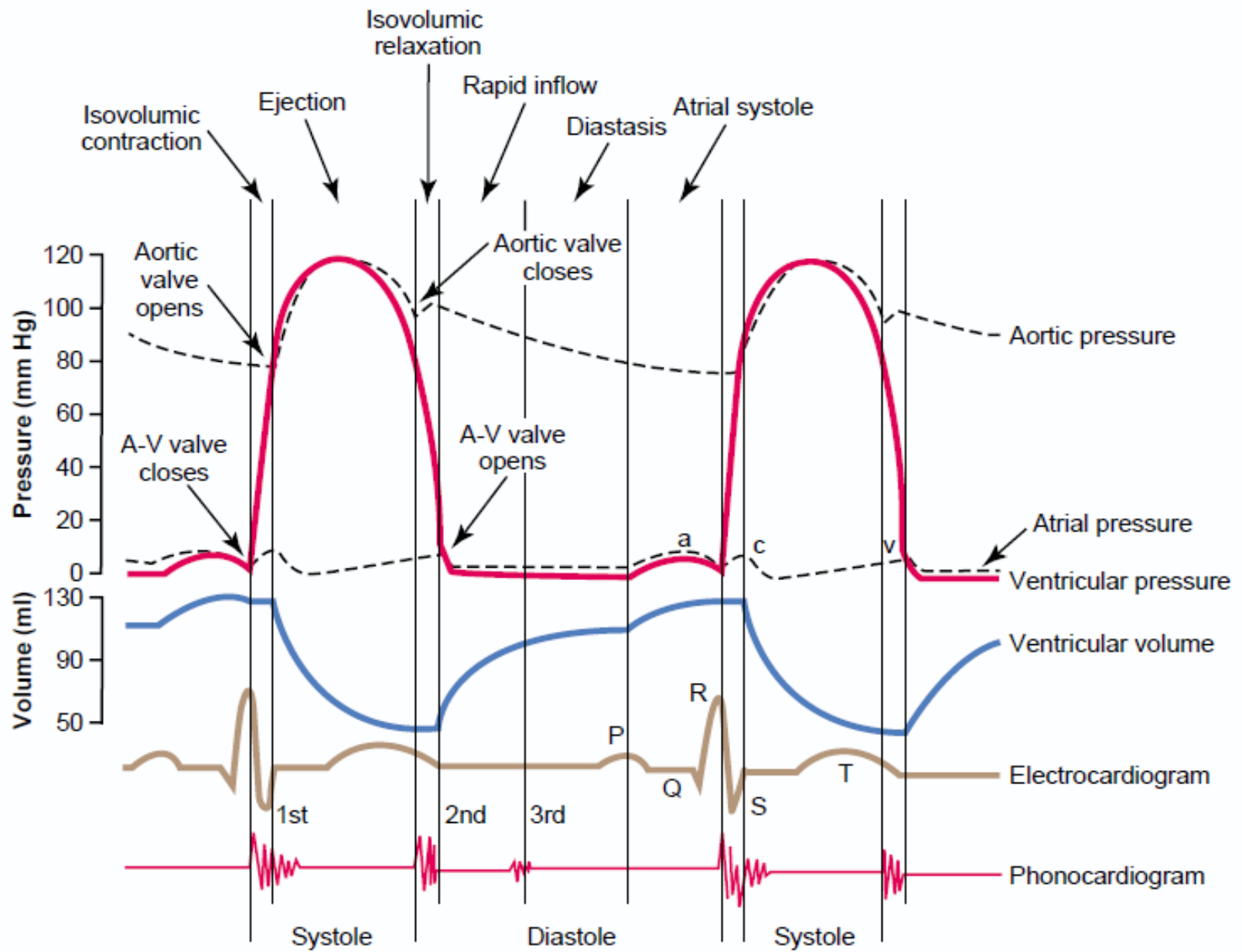
- **JVP:** 'v' wave – due to venous return to the atria from SVC and IVC
- **ECG:** End of T wave
- **Heart sounds:** S_2 : closure of the semilunar valves coincides with this phase.
- **Aortic pressure curve:** **INCISURA** - when the aortic valve closes. Caused by a short period of backflow before the valve closes followed by sudden cessation of the backflow when the valve closes.

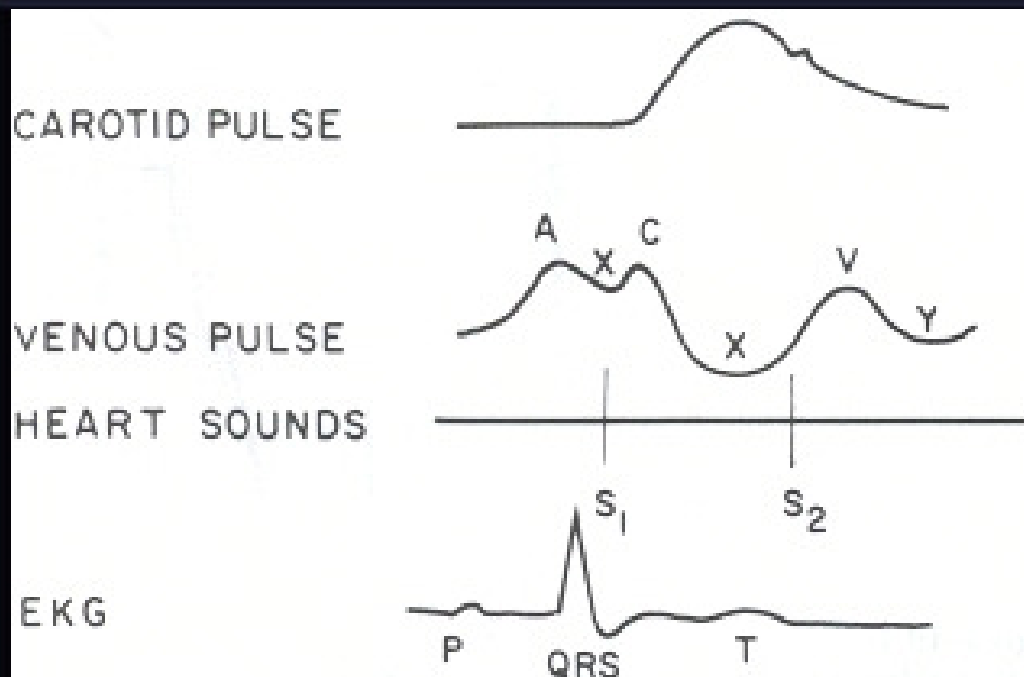
VENTRICULAR FILLING

- Begins with the opening of AV valves
- **Rapid filling** – first 1/3 of diastole
- **Reduced filling (Diastasis)** – middle 1/3 of diastole
- **Atrial contraction** – last 1/3 of diastole



As the atrial pressures fall, the AV valves close and left ventricular volume is now maximum → EDV (120 ml in LV)





... ventricular filling

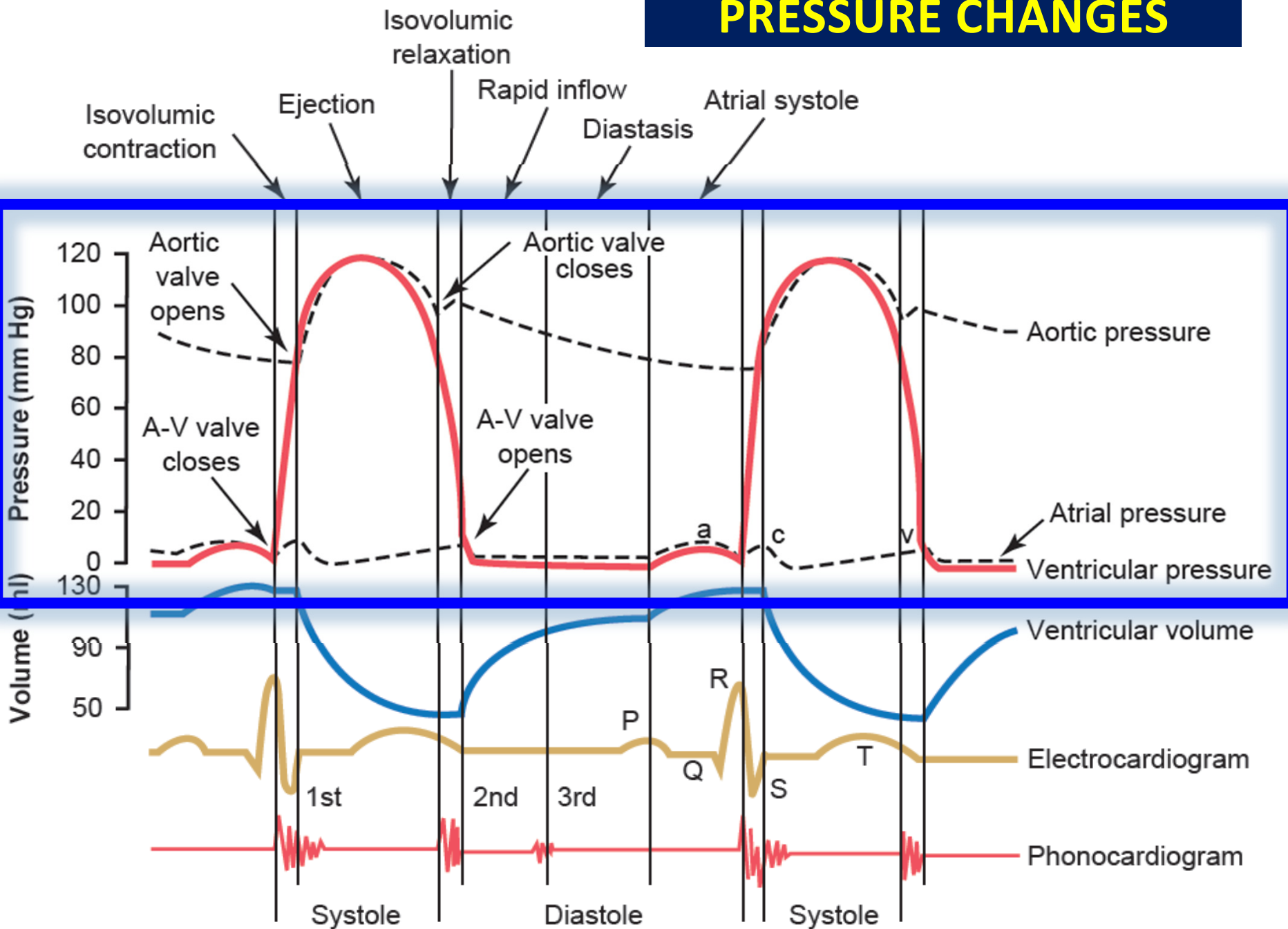
- Rapid filling

- Reduced filling

- Atrial contraction

- **JVP** – ‘y’ descent in first 2/3 & ‘a’ wave in last 1/3
- **ECG** – P wave before atrial systole
- **Heart sounds - S₃ - Pathological in adults.** Seen in dilated congestive heart failure, MI, MR, severe hypertension. Normal in children.

PRESSURE CHANGES



PRESSURE CHANGES DURING CARDIAC CYCLE

Rt- Atrium = 2-8mm Hg.

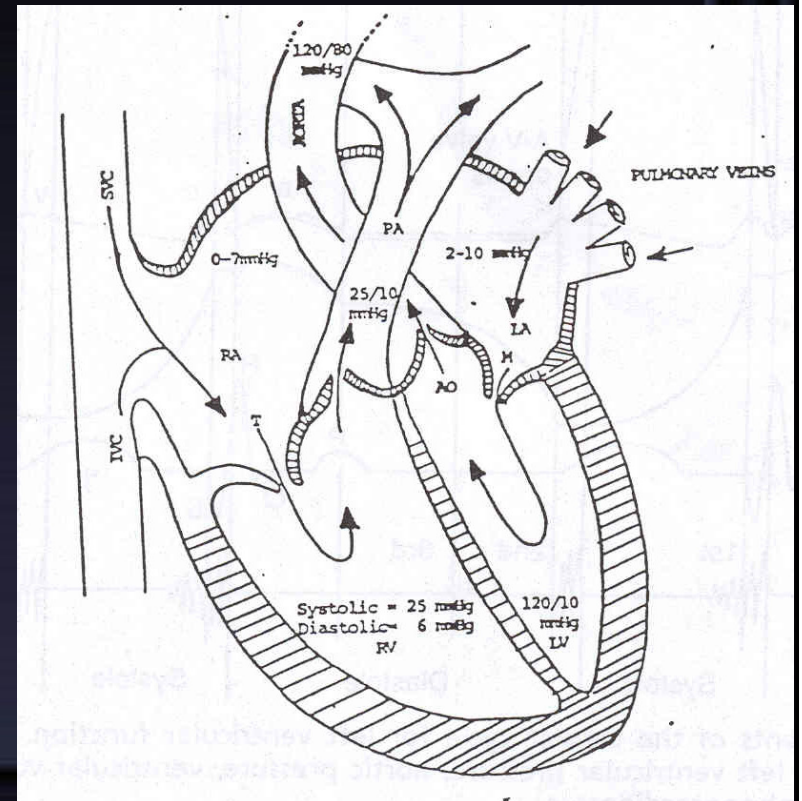
Lt- Atrium = 2-10 mm Hg.

Rt. Ventricle = 25/6 (2-8) mm Hg.

Lt. Ventricle = 120/8 (2-10) mm Hg.

Aorta = 120 / 80 mm Hg.

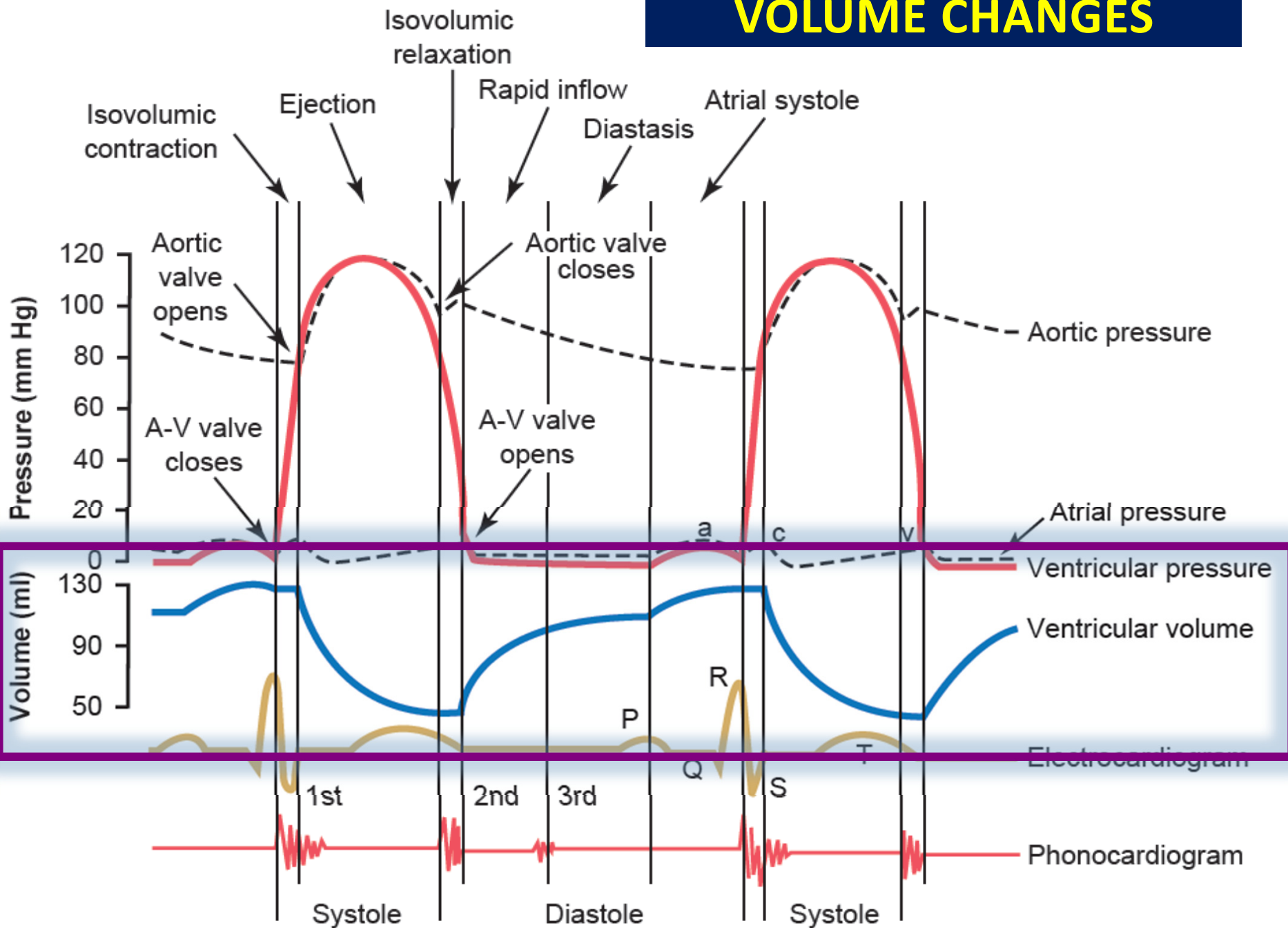
Pul. Artery = 25 / 10 (8-15) mm Hg



VARIOUS PRESSURE VALUES

CHAMBERS	NORMAL RANGE (mm of Hg)
Right Atrium	2 – 8
Right Ventricle (systolic)	15 – 25
(diastolic)	2 -8
Pulmonary Artery (systolic)	15 – 25
(diastolic)	8 - 15
Left Atrium	2-10
Left Ventricle (systolic)	100 – 120
(diastolic)	2 – 10

VOLUME CHANGES



- **End Diastolic Volume:** Volume of blood in each ventricle at the end of diastole.

It is about 110 – 120 ml.

- **End Systolic Volume:** Volume of blood in each ventricle at the end of Systole. It is about 40 to 50 ml

- **Stroke Volume:** It is a volume of blood pumped out by each ventricle per beat. It is about 70 ml.

$$\text{Stroke volume (SV)} = \text{EDV} - \text{ESV}$$

EJECTION FRACTION (EF) is the percentage of ventricular end diastolic volume (EDV) which is ejected with each stroke.

$$EF = \frac{SV (EDV - ESV)}{EDV} \times 100$$

$$\frac{75}{120} \times 100 = 62.5\%$$

Normal ejection fraction is about 60 – 65 %.
Ejection fraction is good index of ventricular function.

FRANK – STARLING'S LAW

Within physiologic limits, the heart pumps all the blood that returns to it by the way of the veins.

OR

The greater the stretch of the cardiac muscle the greater would be the force of contraction.

OR

“The energy of contraction is proportional to the initial length of the cardiac muscle fibers” and for the muscle is proportional to the End Diastolic Volume.

Preload & Afterload

Preload: It is the amount of blood that returns to the heart from veins.

Is end-diastolic volume, which is related to right atrial pressure. When venous return increases, end-diastolic volume increases and stretches or lengthens the ventricular muscle fibers

Afterload: It is the resistance against which the ventricles contract.

For the left ventricle is aortic pressure. Increases in aortic pressure cause an increase in afterload on the left ventricle and for the right ventricle is pulmonary artery pressure. Increases in pulmonary artery pressure cause an increase in afterload on the right ventricle.



Practical Box 13.2

Measurement of jugular venous pressure

The patient is positioned at about 45° to the horizontal (between 30° and 60°), wherever the top of the venous pulsation can be seen in a good light.

The jugular venous pressure is measured as the vertical distance between the manubriosternal angle and the top of the venous column.

The normal jugular venous pressure is usually less than 3 cmH₂O, which is equivalent to a right atrial pressure of 8 cmH₂O when measured with reference to a point midway between the anterior and posterior surfaces of the chest.

The venous pulsations are not usually palpable (except for the forceful venous distension associated with tricuspid regurgitation).

Compression of the right upper abdomen causes a temporary increase in venous pressure and makes the JVP more visible (hepatojugular reflux).

ABNORMALITIES OF “a” WAVE

- **Elevated a wave**

Tricuspid stenosis

Decreased ventricular compliance (ventricular failure, pulmonic valve stenosis, or pulmonary hypertension)

- **Cannon a wave**

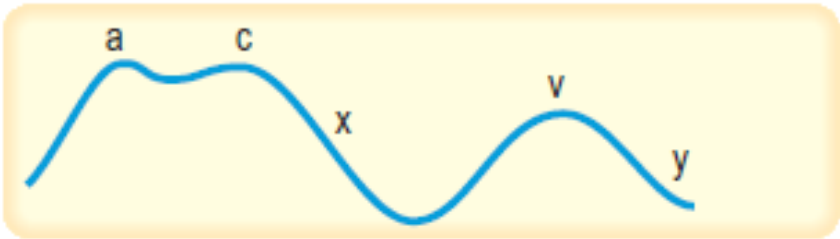
Atrial-ventricular asynchrony (atria contract against a closed tricuspid valve)

complete heart block, following premature ventricular contraction, during ventricular tachycardia, with ventricular pacemaker

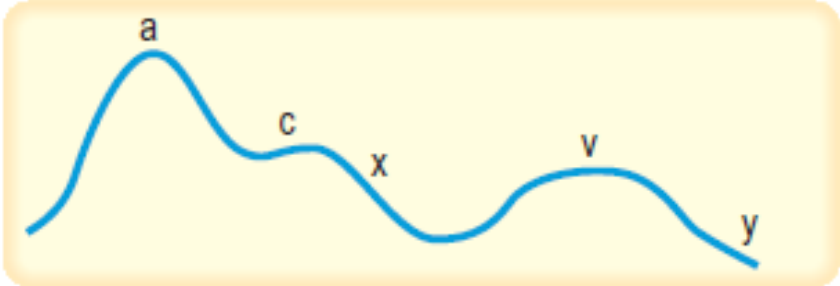
- **Absent a wave**

Atrial fibrillation or atrial standstill
Atrial flutter

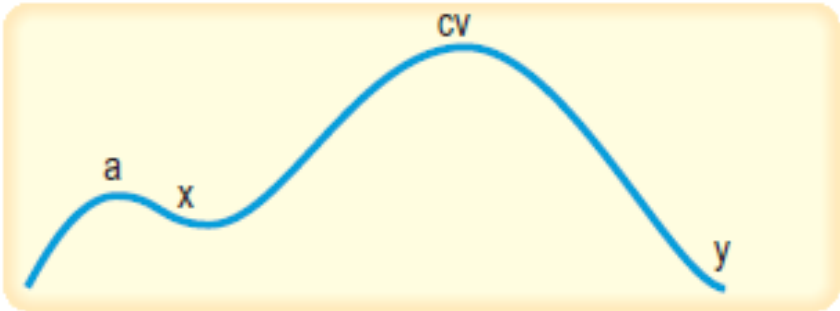
Normal



Pulmonary hypertension
Tricuspid stenosis



Tricuspid regurgitation



Constrictive pericarditis

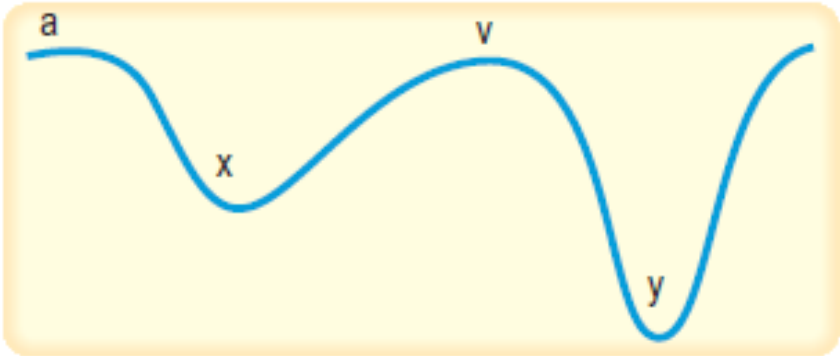


Fig. 13.12 Jugular venous waveforms.

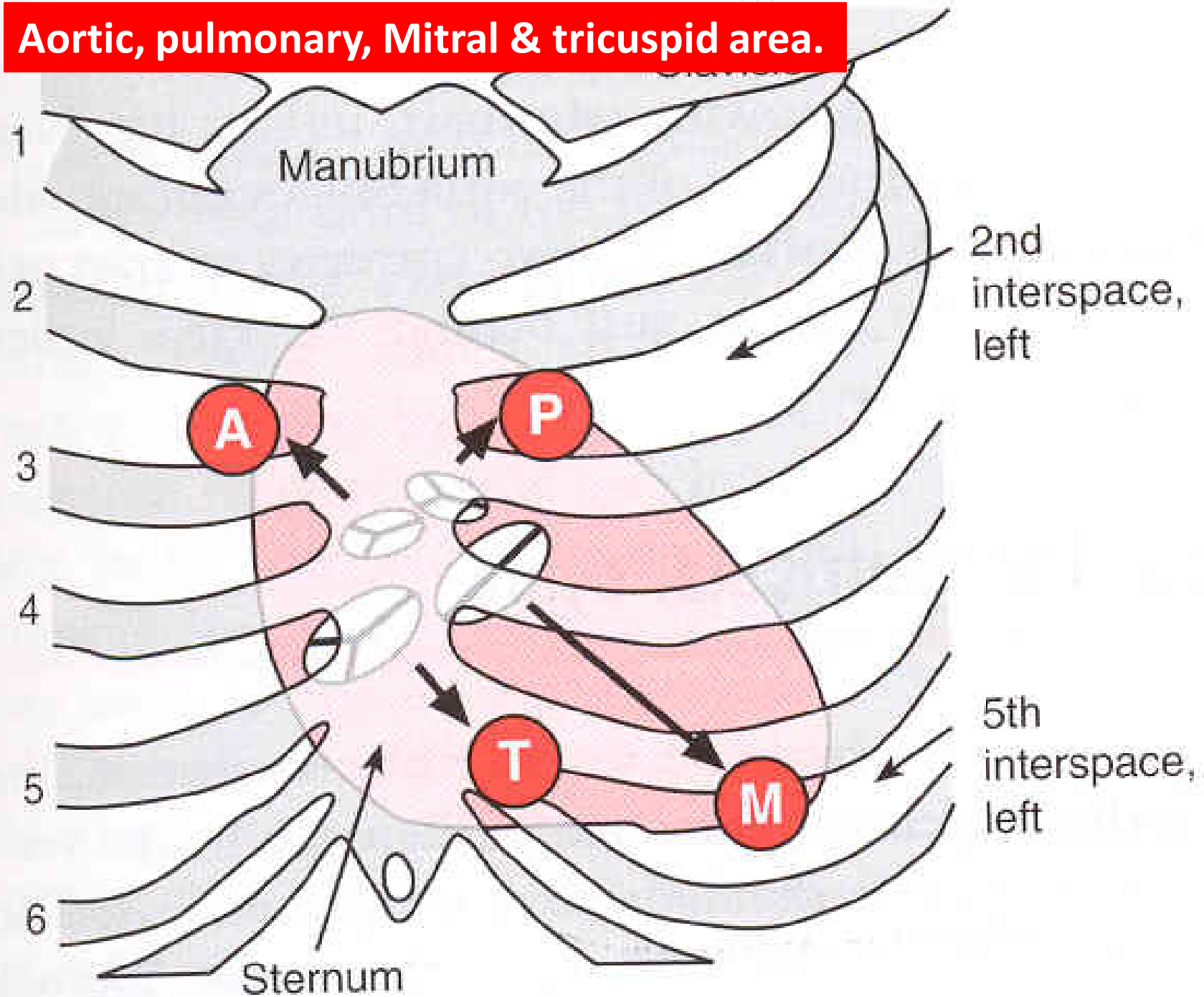


HEART SOUNDS

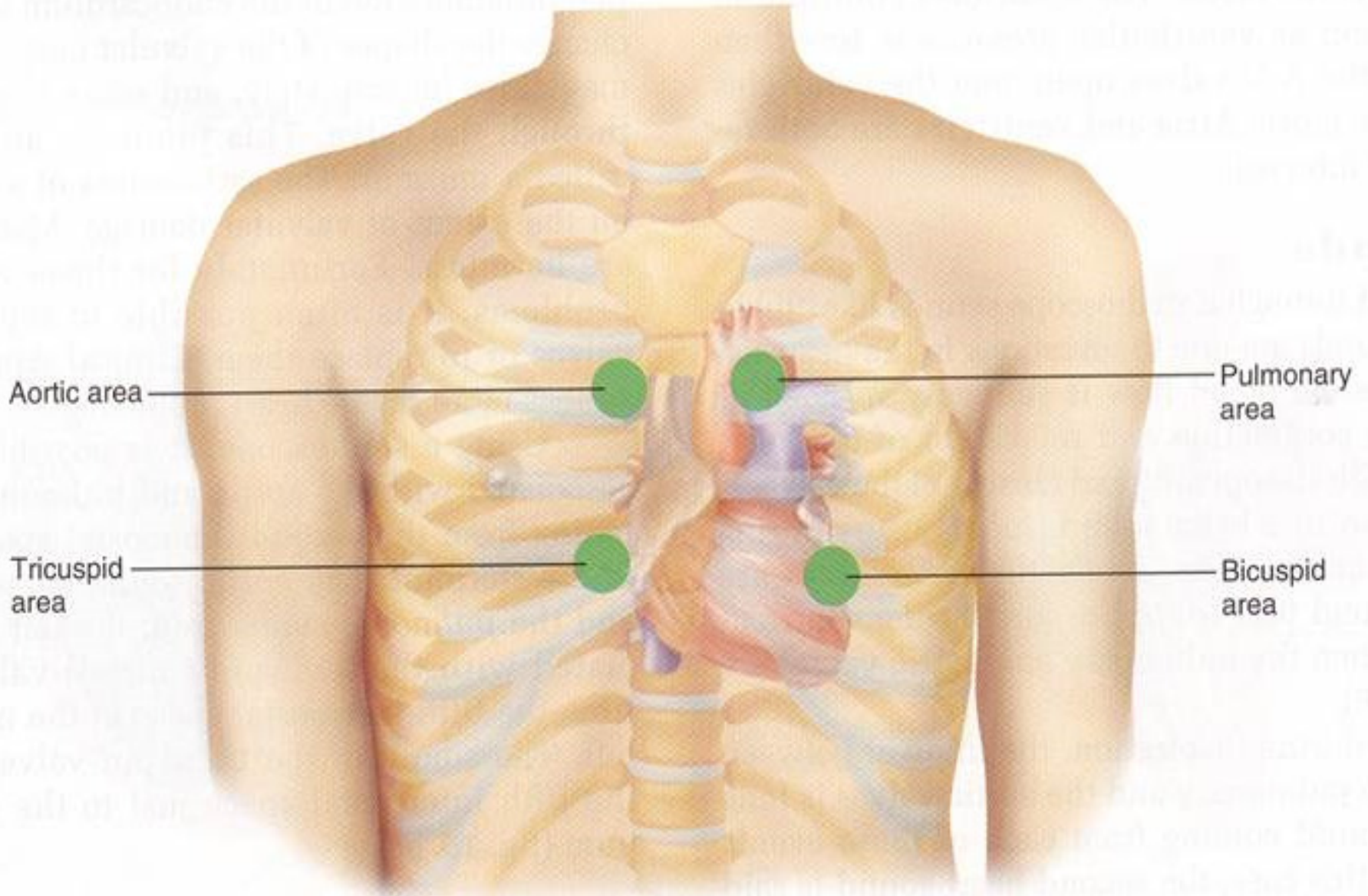


Heart sounds

Aortic, pulmonary, Mitral & tricuspid area.



AREAS OF AUSCULTATION



HEART SOUNDS

- There are four heart sounds S1, S2, S3 & S4.
- Two heart sound are audible with stethoscope S1 & S2 (Lub - Dub).
- S3 & S4 are not audible with stethoscope Under normal conditions because they are low frequency sounds.
- Ventricular Systole is between First and second Heart sound.
- Ventricular diastole is between Second and First heart sounds.

FIRST HEART SOUND (S1)

- It is produced due to the closure of Atrioventricular valves (Mitral & Tricuspid)
- It occurs at the beginning of the systole and sounds like LUB
- Frequency: 50-60 Hz
- Time: 0.15 sec
- It is heavier when compared to the 2nd heart sound.

SECOND HEART SOUND (S2)

- It is produced due to the closure of Semilunar valves (Aortic & Pulmonary)
- It occurs at the end of the systole and sounds like DUB
- Frequency: 80-90 Hz
- Time: 0.12 sec
- It is short and sharp compared to the 1st heart sound

THIRD HEART SOUND (S3)

- **It occurs at the beginning of middle third of Diastole**
- **Cause of 3rd sound – Rush of blood from Atria to Ventricle during rapid filling phase of Cardiac Cycle. It causes vibration in the blood**
- **Frequency: 20-30 Hz**
- **Time: 0.1 sec**
- **S3 may be heard in children and young slim adults but usually pathological in old age.**

FOURTH HEART SOUND (S4) OR ATRIAL SOUND

- It occurs at the last one third of Diastole (just before S1)
- Cause of Fourth heart sound – Due to Atrial systole which causes rapid flow of blood from Atria to Ventricle and vibration in the blood.
- Frequency: < 20 Htz

Note:

- Third and Fourth heart sound are low pitched sounds therefore not audible normally with stethoscope
- S4 may be heard in elderly but is usually pathologic in the young.

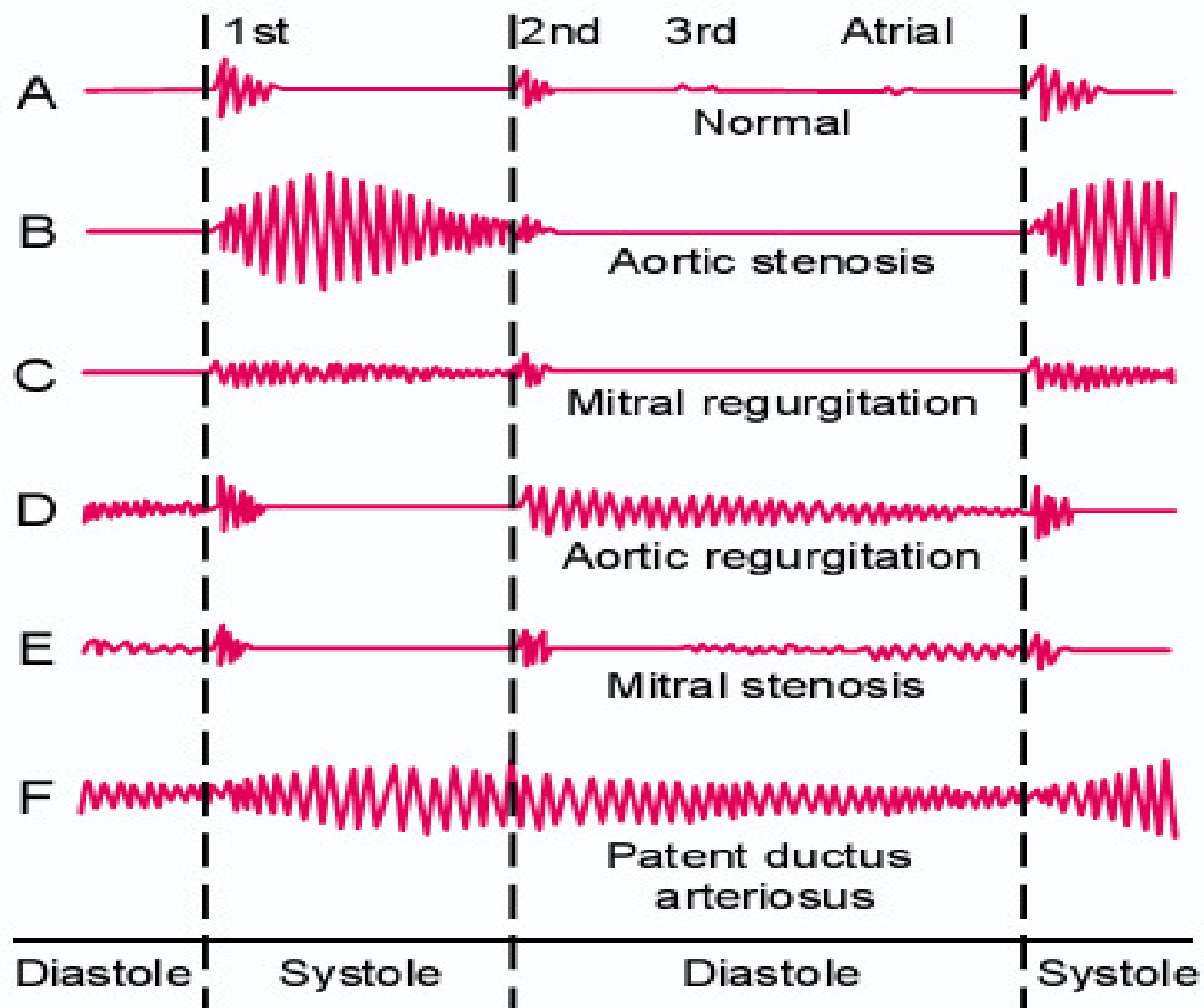


Figure 23-3

Phonocardiograms from normal and abnormal hearts.

HEART MURMURS

- Murmurs are abnormal sounds produced due to abnormal flow of blood.
- OR
- Murmurs are pathologic and added heart sounds that are produced as a result of turbulent blood flow

TABLE 30-2 Heart murmurs.

Valve	Abnormality	Timing of Murmur
Aortic or pulmonary	Stenosis	Systolic
	Insufficiency	Diastolic
Mitral or tricuspid	Stenosis	Diastolic
	Insufficiency	Systolic

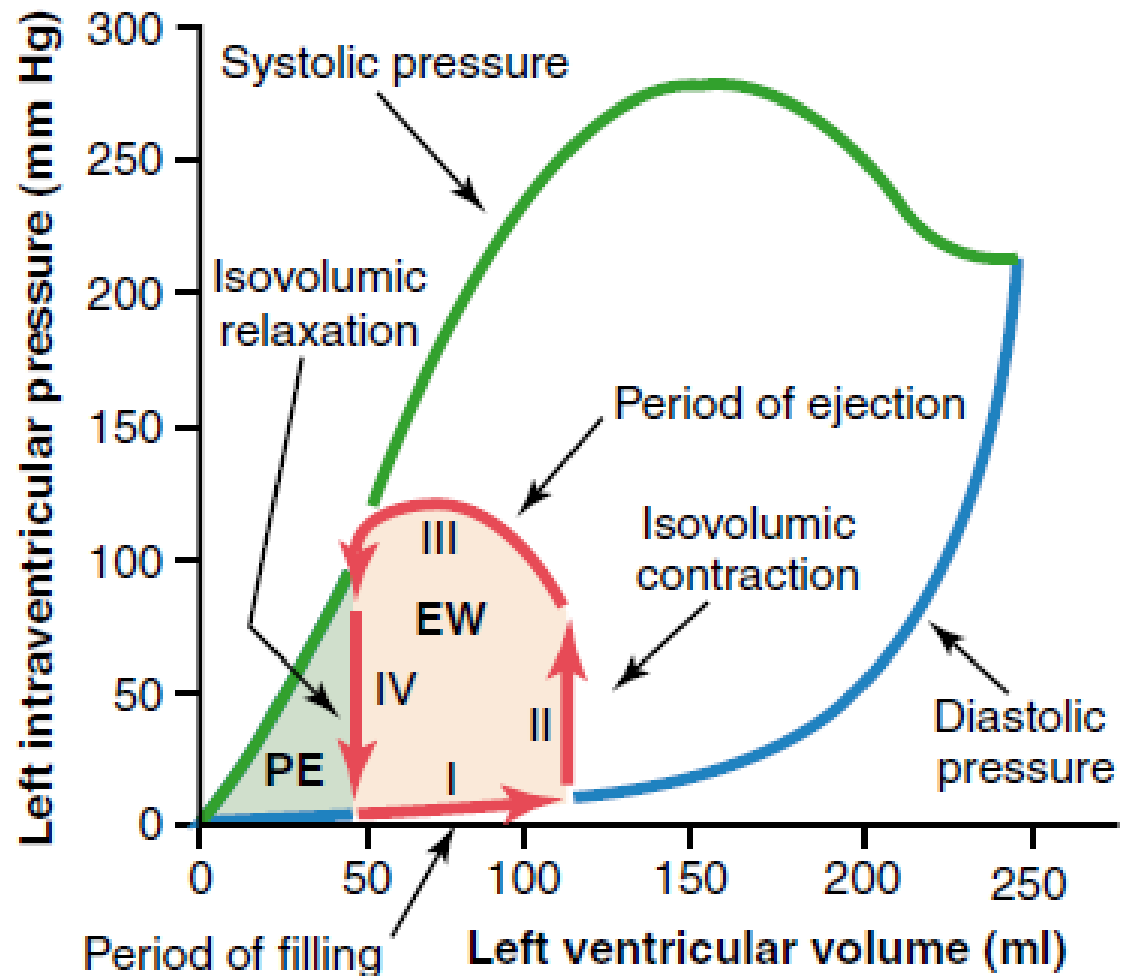


Figure 9-8 Relationship between left ventricular volume and intraventricular pressure during diastole and systole. Also shown by the heavy red lines is the "volume-pressure diagram," demon-

**"Volume-Pressure Diagram" During the Cardiac Cycle;
Cardiac Work Output.**

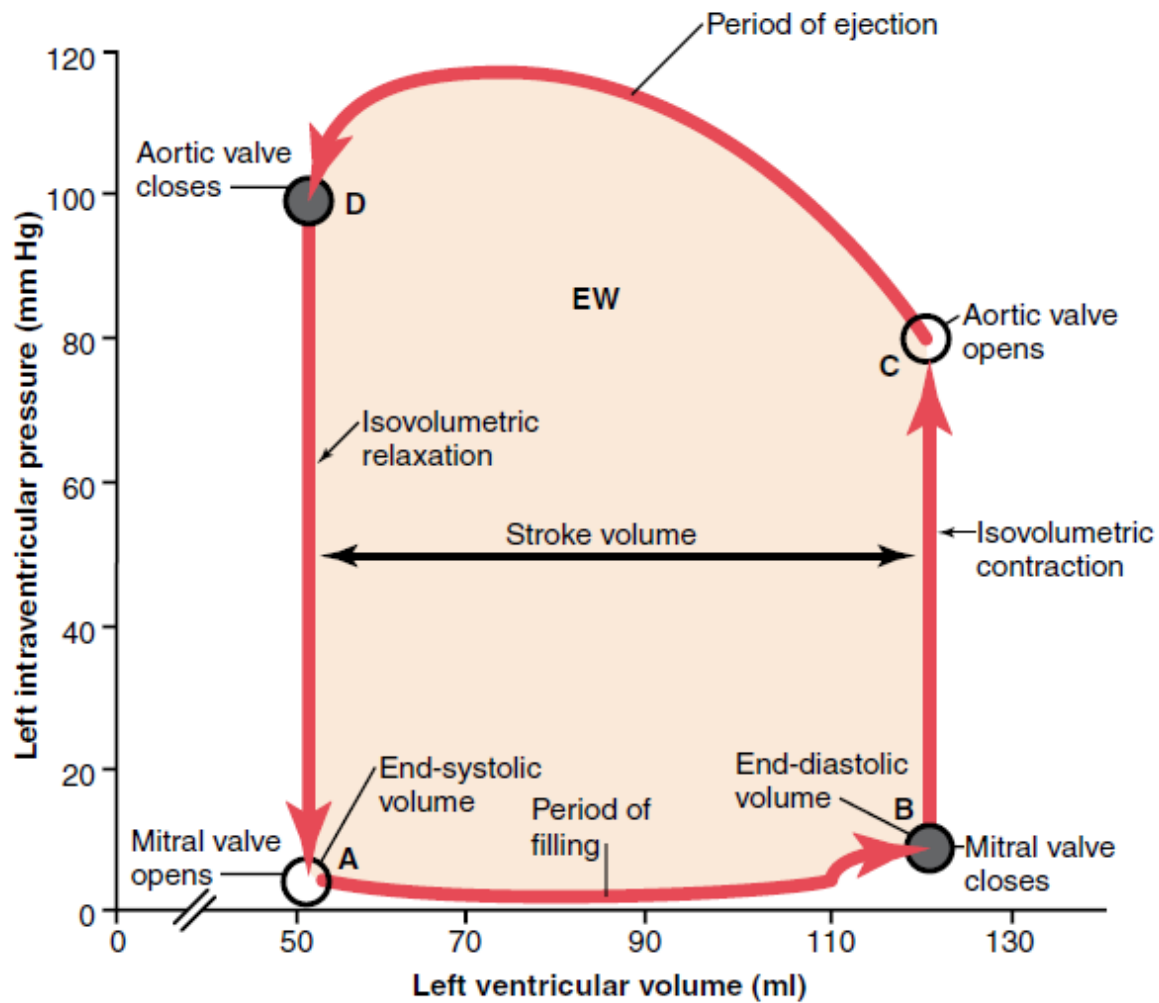
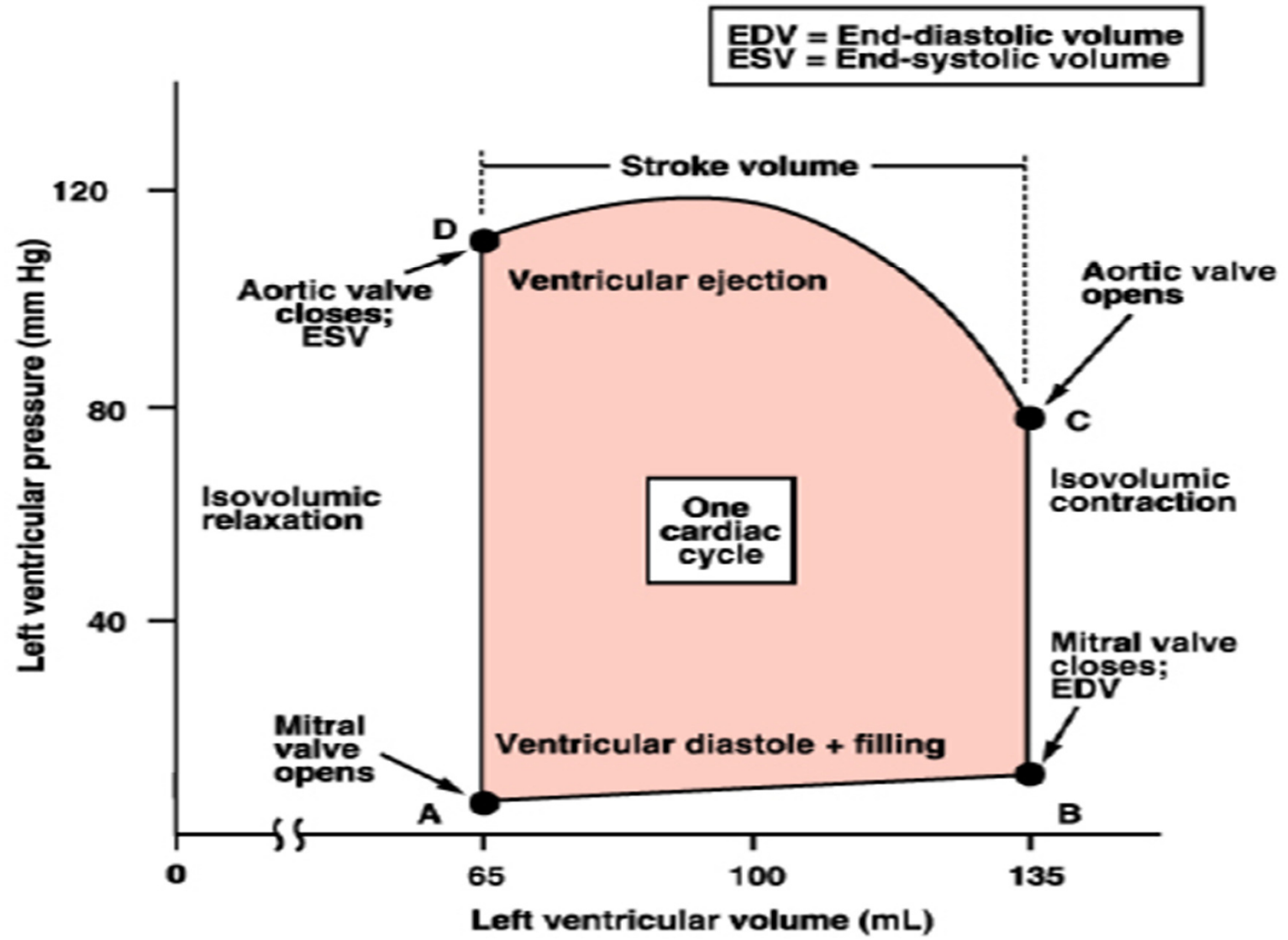


Figure 9-9 The “volume-pressure diagram” demonstrating changes in intraventricular volume and pressure during a single cardiac cycle (*red line*). The tan shaded area represents the net external work (*EW*) output by the left ventricle during the cardiac cycle.



- A → B: Passive filling and atrial contraction
- B → C: Isovolumic contraction
- C → D: Ejection of blood into aorta
- D → A: Isovolumic relaxation

a. **Increased preload:** refers to an increase in end-diastolic volume and is the result of increased venous return. It causes an **increase in stroke volume** based on the Frank–Starling relationship....reflected in **increased width** of the pressure–volume loop.

b. **Increased afterload** refers to an increase in aortic pressure. The ventricle must eject blood against a higher pressure, resulting in a decrease in stroke volume....is reflected in **decreased width** of the pressure–volume loop. The decrease in stroke volume results in an increase in end-systolic volume.

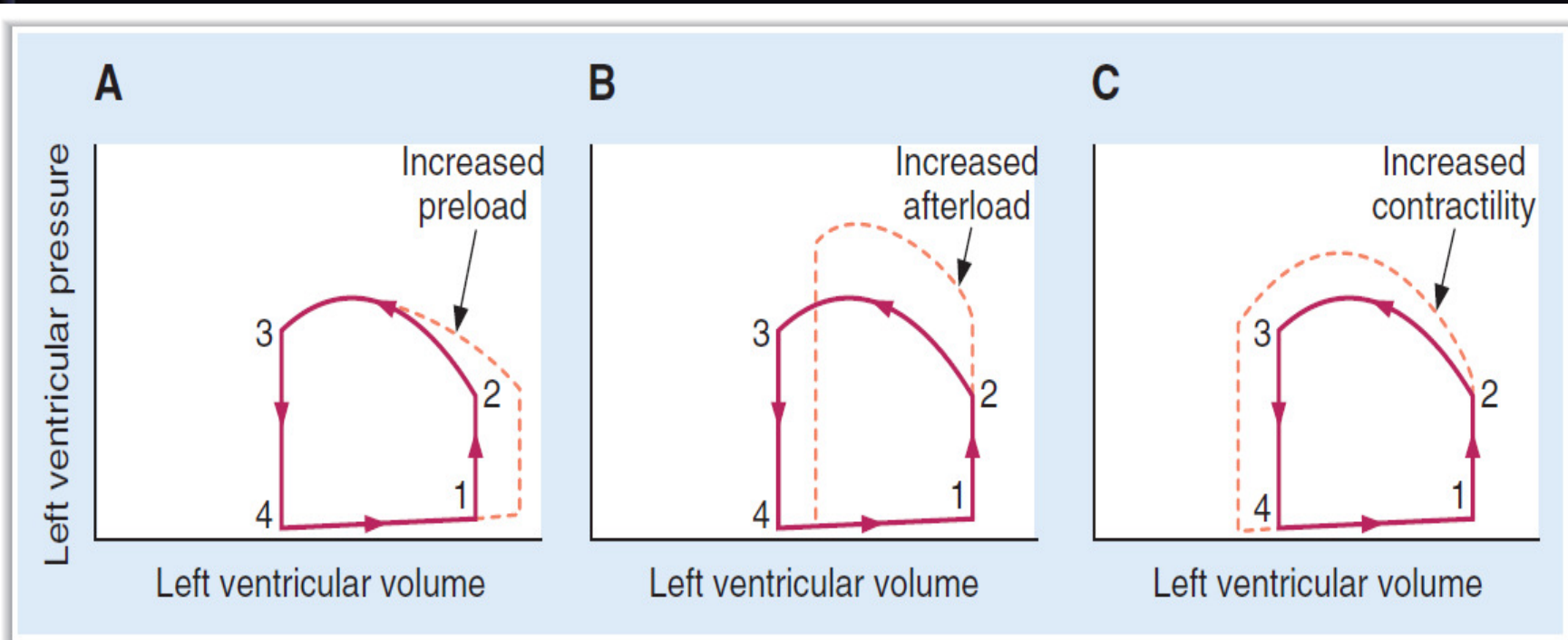


FIGURE 3-10 Effects of changes in (A) preload, (B) afterload, and (C) contractility on the ventricular pressure–volume loop.