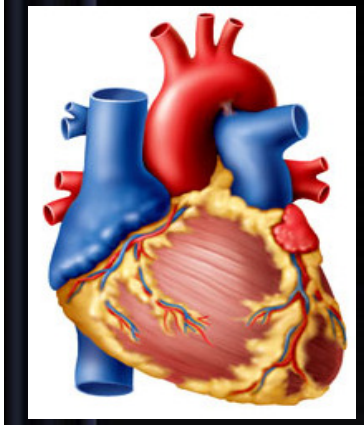
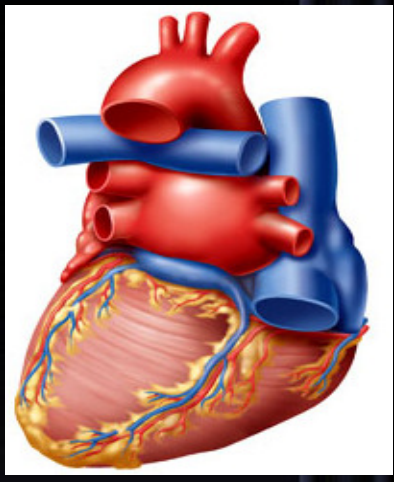
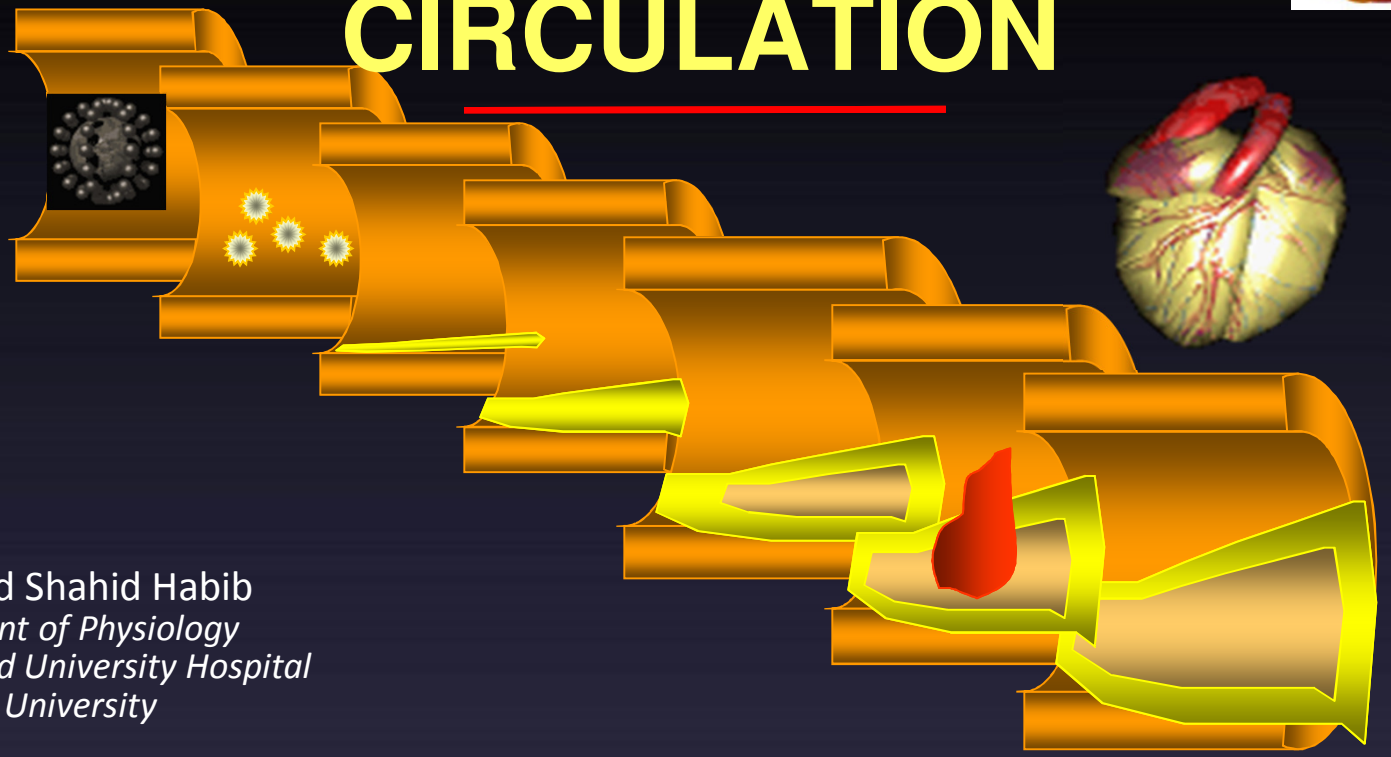




CARDIOVASCULAR SYSTEM

CORONARY CIRCULATION



Prof. Syed Shahid Habib
Department of Physiology
King Khalid University Hospital
King Saud University

OBJECTIVES

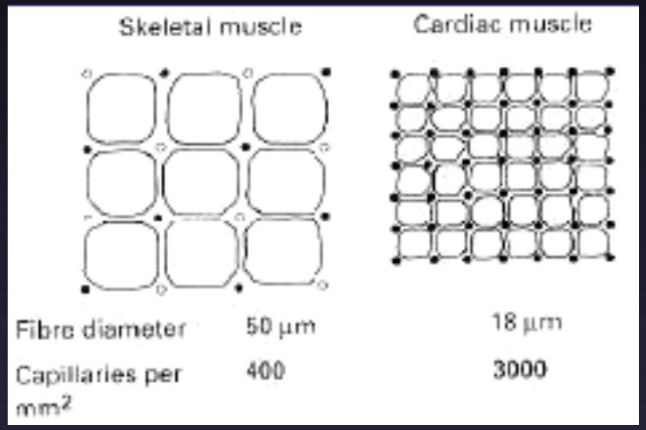
❖ **At the end of the lecture you should be able to …..**

- Facts about Coronary blood flow
- Normal Coronary blood flow
- Coronary blood flow in systole & diastole
- Discuss regulation of coronary blood flow.
- Explain and differentiate between angina and myocardial infarction

IMPORTANT ! WHY?

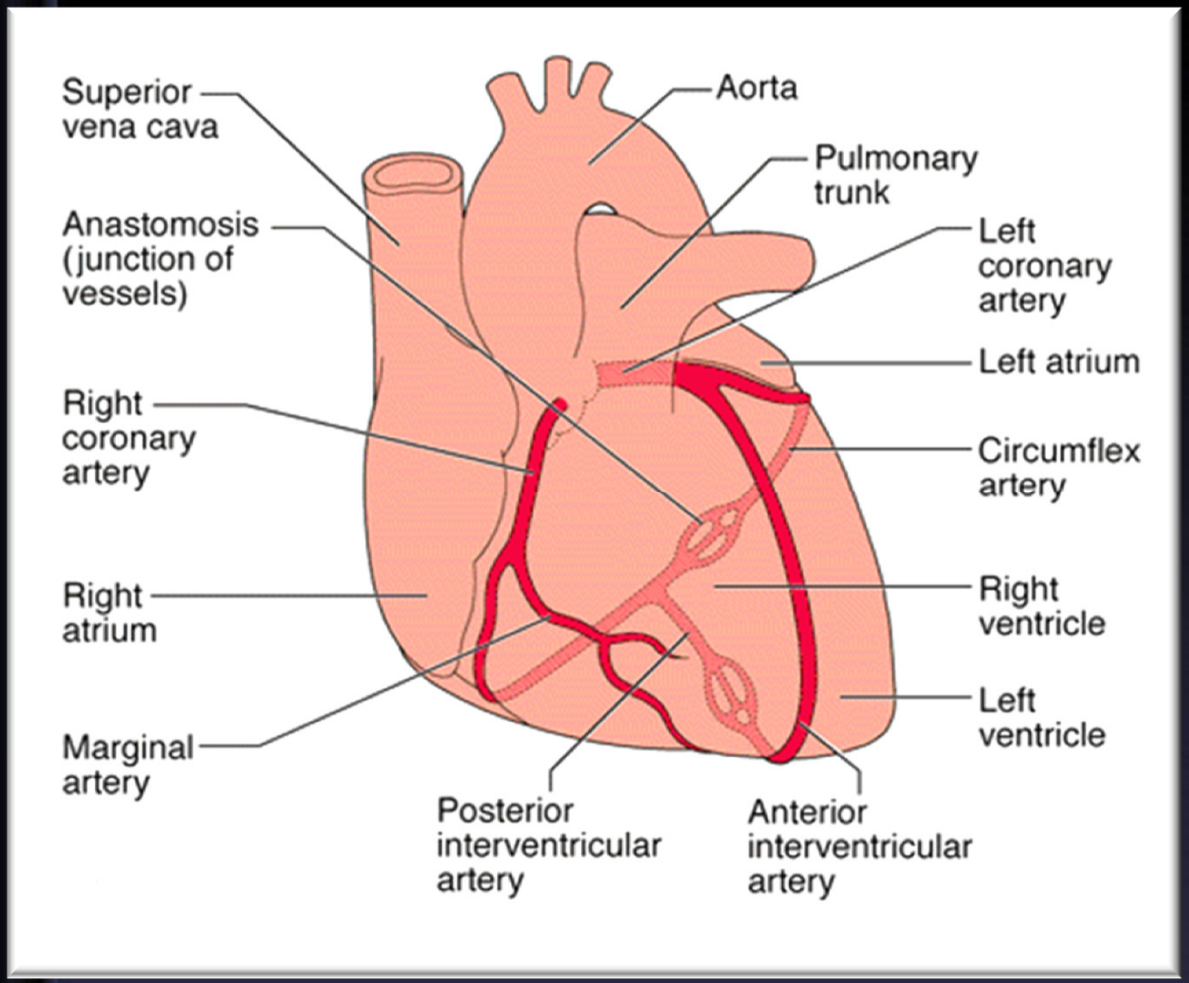
- One third of all deaths in the world result from coronary artery disease.
- Almost all elderly people have at least some impairment of the coronary artery circulation.

Capillary Density in the Heart



Heart uses primarily free fatty acids and to lesser extent glucose and lactate for metabolism.

Coronary arteries



- Left main coronary artery divides into left anterior descending artery (anterior interventricular) and circumflex artery.
- Right coronary artery divides into smaller branches, including the right posterior descending artery (posterior interventricular) and the acute marginal artery.

FACTS ABOUT CORONARY BLOOD FLOW

- Two-thirds of coronary blood flow occurs during diastole .
- Five percent of cardiac output goes to the coronary arteries.
- Seventy percent of oxygen is extracted by the myocardialtissues of the heart, in comparison to the rest of the body at twenty five percent.
- During times of extreme demand, the coronary arteries can dilate up to four times greater than normal
- Coronary blood flow in Humans at rest is about 225-250 ml/minute, about 5% of cardiac output.

Effect of Tachycardia on coronary blood flow:

During increased heart rate, period of diastole is shorter therefore coronary blood flow is reduced to heart during tachycardia.

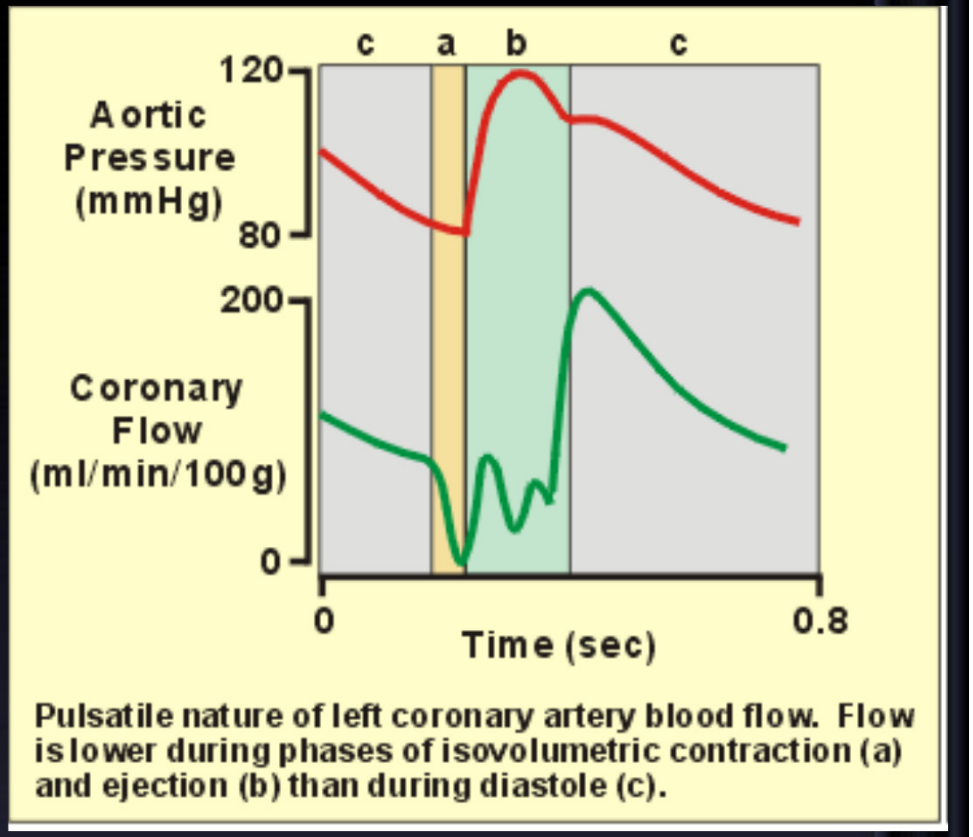
CORONARY BLOOD FLOW

- Why heart is extracting 60-70% of O2?

- **Because heart muscle has more mitochondria, up to 40% of cell is occupied by mitochondria, which generate energy for contraction by aerobic metabolism, therefore, heart needs O_2.**
- **When more oxygen is needed e.g. exercise, O_2 can be increased to heart only by increasing blood flow.**

BLOOD FLOW TO HEART DURING SYSTOLE & DIASTOLE

- During systole when heart muscle contracts it compresses the coronary arteries therefore blood flow is less to the left ventricle during systole and more during diastole.
- Blood flows to the subendocardial portion of Left ventricle ,which occurs only during diastole



Pulsatile nature of left coronary artery blood flow. Flow is lower during phases of isovolumetric contraction (a) and ejection (b) than during diastole (c).

Blood flow through the coronary capillaries of the right ventricle also undergoes phasic changes during the cardiac cycle, but because the force of contraction of the right ventricular muscle is far less than that of the left ventricular muscle, the inverse phasic changes are only partial, in contrast to those in the left ventricular muscle.

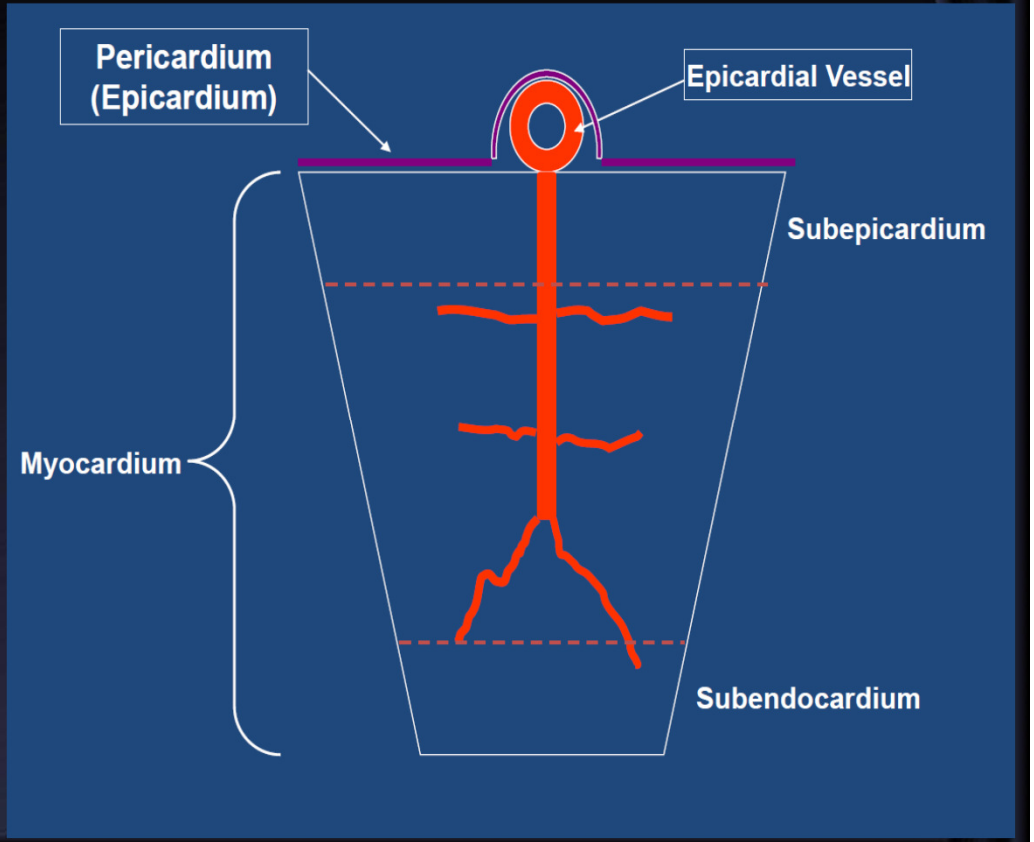
Coronary Vascular Resistance

- **Epicardial conductance vessels:** Contribute only to a small % of resistance.
- **Intramyocardial vessels (arterioles):** Contribute most to total coronary vascular resistance.

As we know during systole blood flow to subendocardial surface of left ventricle is almost not there

therefore,

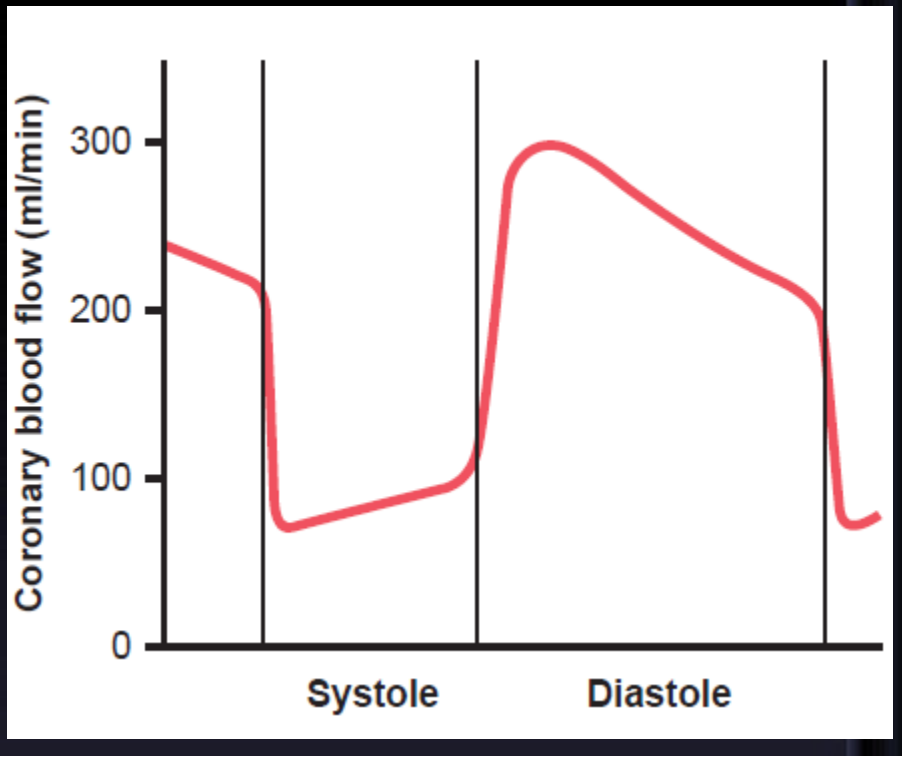
this region is prone to ischemic damage and most common site of Myocardial infarction.



PHASIC CHANGES IN CORONARY BLOOD FLOW

During systole, coronary arteries are compressed & the blood flow to the left ventricle specially subendocardial vessels is reduced.

However, the extra vessels of the subendocardial plexus normally compensate for this reduction.



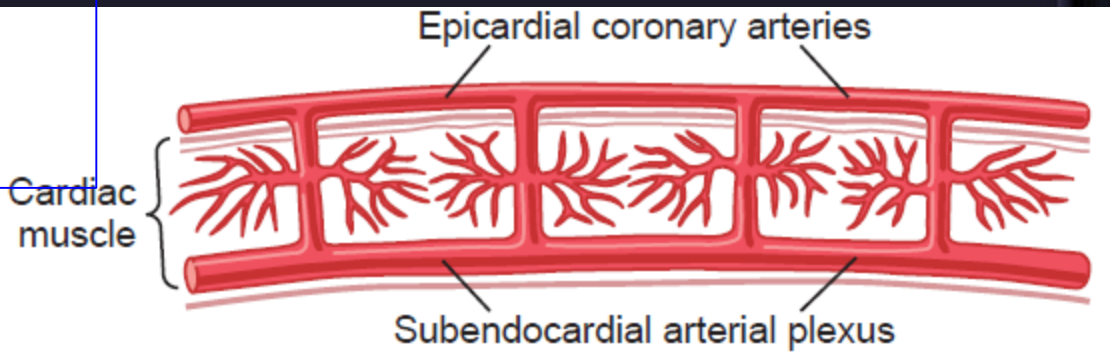


Figure 21-5. Diagram of the epicardial, intramuscular, and subendocardial coronary vasculature.

PRESSURE GRADIENT BETWEEN AORTA & VENTRICLES AFFECTS CBF

CBF to the Right side is not much affected during systole.

Pressure difference between the aorta & Right ventricle is greater during systole than during diastole, therefore more blood flow to Right ventricle occurs during systole

		Pressure in (mmHg)		Pressure difference (mmHg) between aorta& LV-RV	
	Aorta	Left Ventricle	Right Ventricle	Left Ventricle	Right Ventricle
Systole	120	120	25	0	95
Diastole	80	2-0	2-0	80	80

REGULATION OF CORONARY BLOOD FLOW

1- Metabolic control

2- Auto regulation

3- Endothelial control of coronary vascular tone

4- Extravascular compressive forces

5- Neural control

1- METABOLIC CONTROL

Coronary circulation is very sensitive to myocardial tissue oxygen tension.

Increased oxygen demand results in a lower tissue oxygen tension.

This causes:

Vasodilation and increased blood flow by chemical factors like:-

1- ↑ Adenosine 2- Lack of oxygen

3- ↑ Nitric oxide 4- ↑ Prostaglandins

5- ↑ K_+ 6- ↑ H_+

7- ↑ Lactate 8- ↑ Adenine nucleotides

Adenosine, which is formed from ATP during cardiac metabolic activity, causes coronary vasodilatation.

2- AUTO REGULATION

- Ability of a vascular network to maintain constant
- blood flow over a range of arterial pressures.
- Auto regulation is an independent determinant of
- coronary blood flow.
- The set point at which coronary blood flow is
- maintained depends on myocardial oxygen
- consumption (MVO2).

3- ENDOTHELIAL CONTROL OF CORONARY VASCULAR TONE

Damage to endothelial cells will lead to:

1- Decreased Nitric Oxide and Prostacyclin production.

2- Increased Endothelin production.

This will lead to:

1- Vasoconstriction.

2- Vasospasm.

3- Thrombosis.

4- NEURAL CONTROL

- Coronary blood flow is controlled predominantly by local metabolic, auto regulatory, and endothelial factors.
- Neural control of the coronary circulation complements the above local effects.

Sympathetic Control:

Alpha = constrict coronary vessels.

Beta = dilate coronary vessels.

$Beta_1$ in conduit arteries.

$Beta_2$ in resistance arterioles.

Parasympathetic Control:

Acetylcholine

Vasodilation in healthy subjects.

Vasoconstriction in patients with atherosclerosis.

5- EXTRAVASCULAR COMPRESSIVE FORCES

Left Ventricle:

Early Systole > Initial Flow Reversal.

Remainder of Systole > Flow follows aortic pressure curve,

but at a much reduced pressure.

Early Diastole > Abrupt pressure rise (80-90% of LV flow

occurs in early diastole).

Remainder of Diastole > Pressure declines slowly as aortic

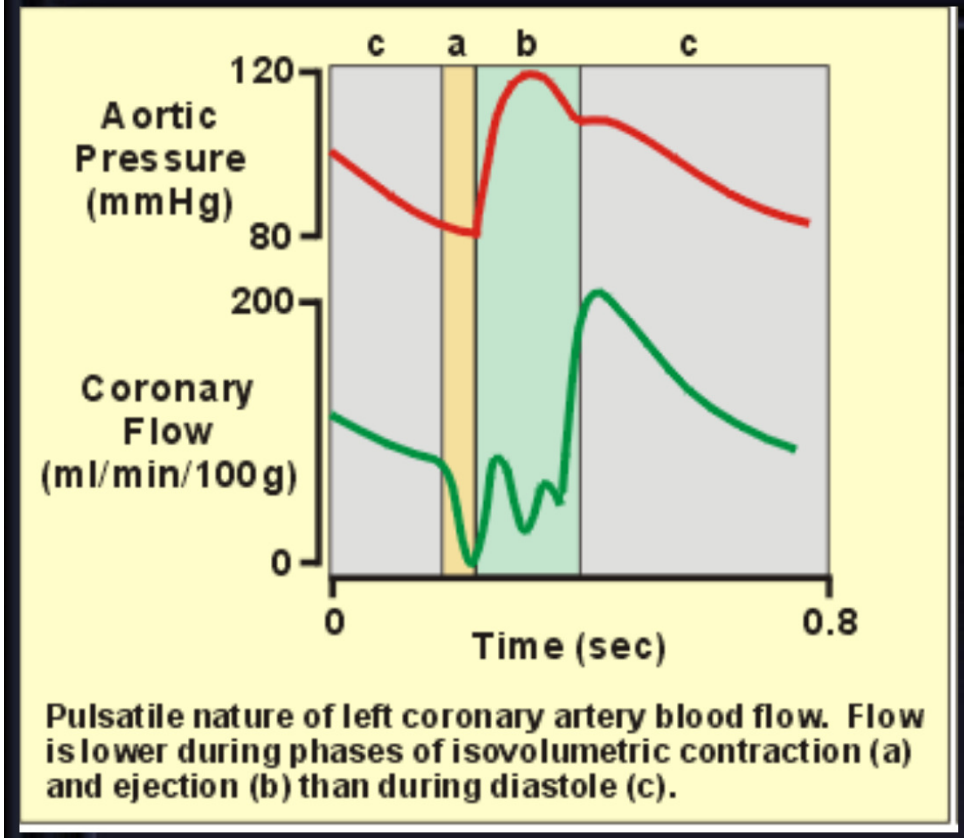
pressure decreases.

Right Ventricle:

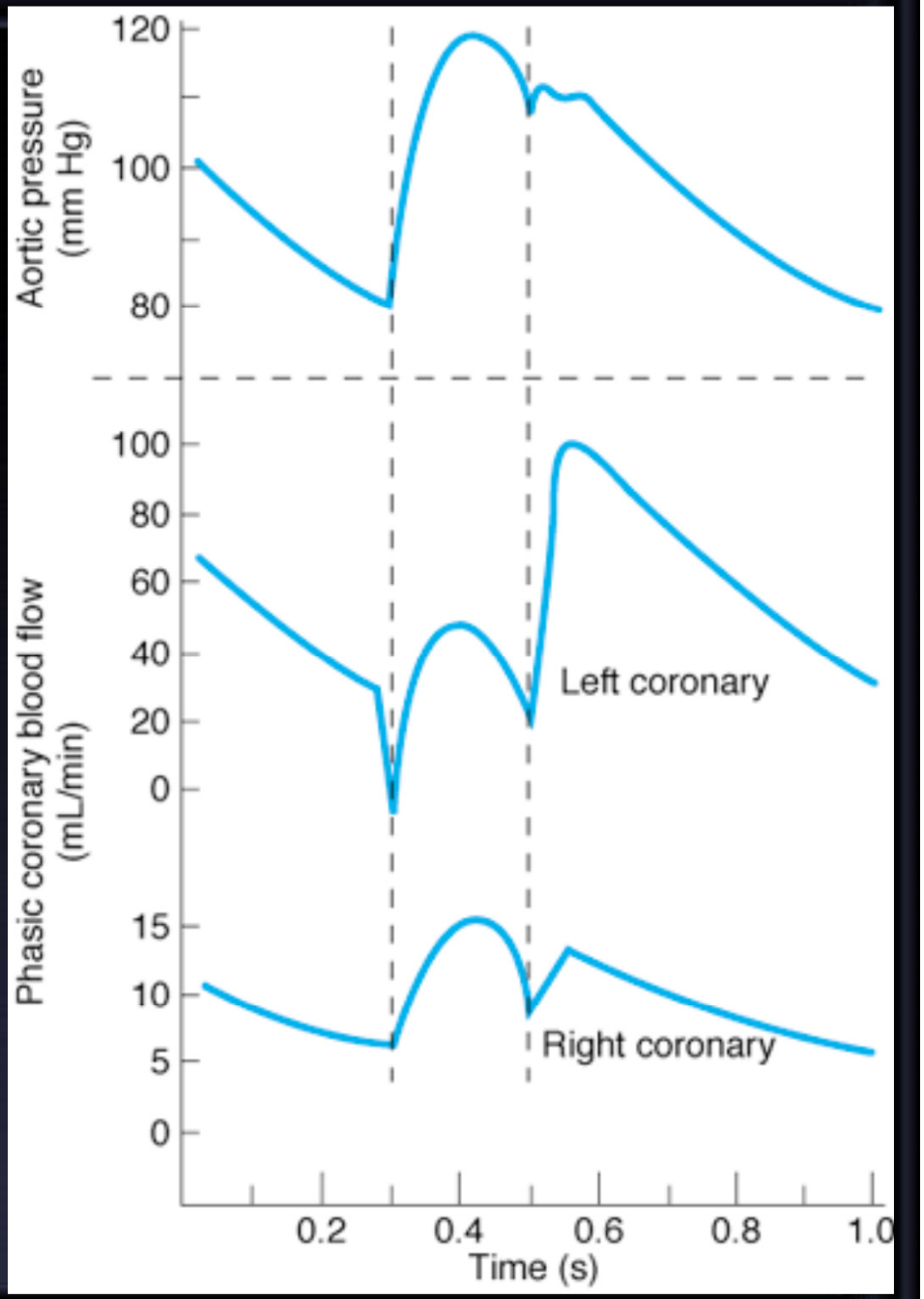
Lower pressure generated by thin right ventricle in Systole.

No reversal of blood flow during early systole.

Systolic blood flow constitutes a much greater proportion of total blood flow.



Pulsatile nature of left coronary artery blood flow. Flow is lower during phases of isovolumetric contraction (a) and ejection (b) than during diastole (c).



FACTORS INCREASING MYOCARDIAL OXYGEN CONSUMPTION

1- Increased Heart Rate.

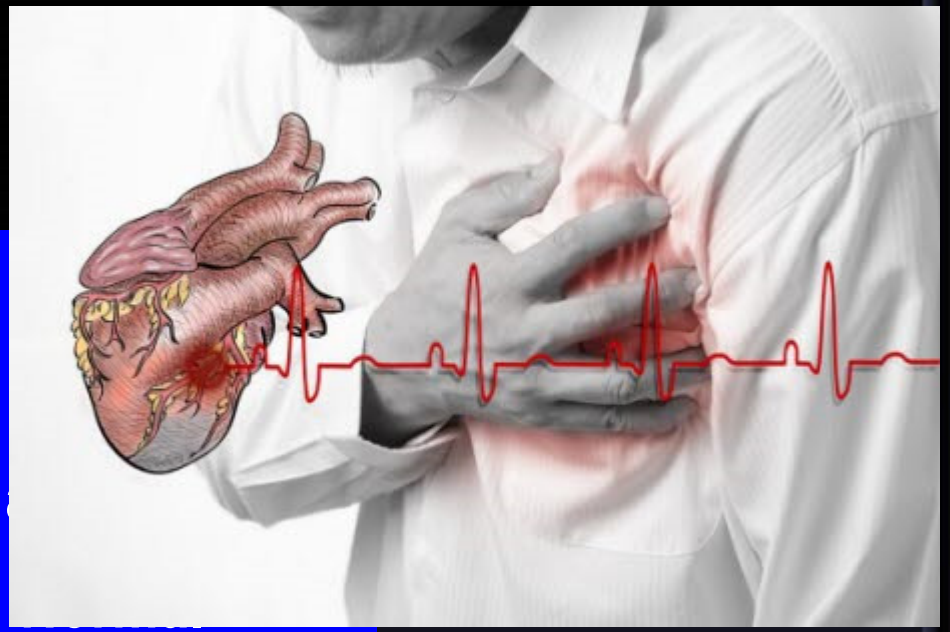
2- Increased Inotropy (Contractility).

3- Increased Afterload.

4- Increased Preload.

Changes in preload affect myocardial oxygen consumption less than do changes in the other factors. % of O2?

ANGINA:



Investigation:

ECG may be normal between

Exercise ECG – 75% positive,

results does not exclude the condition.

Treatment for attack:

Stop exercise

Glyceryl trinitrate 0.5mg under the tongue.

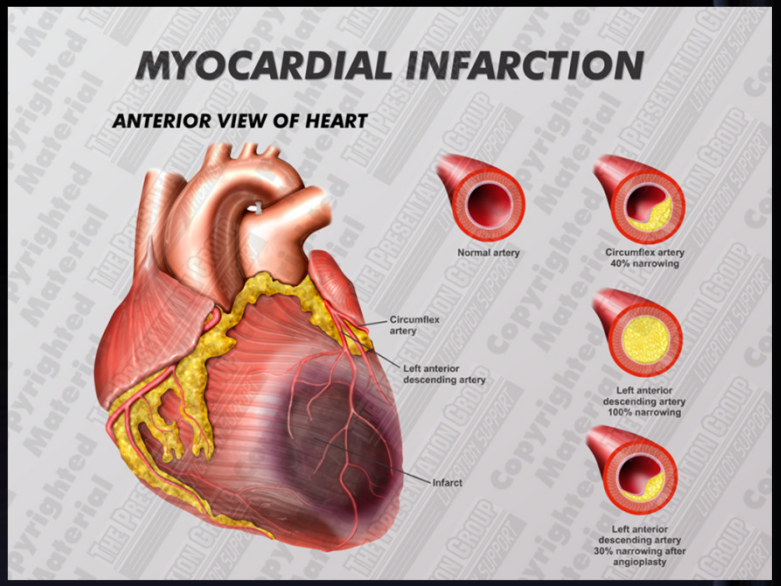
(side effects → headache)

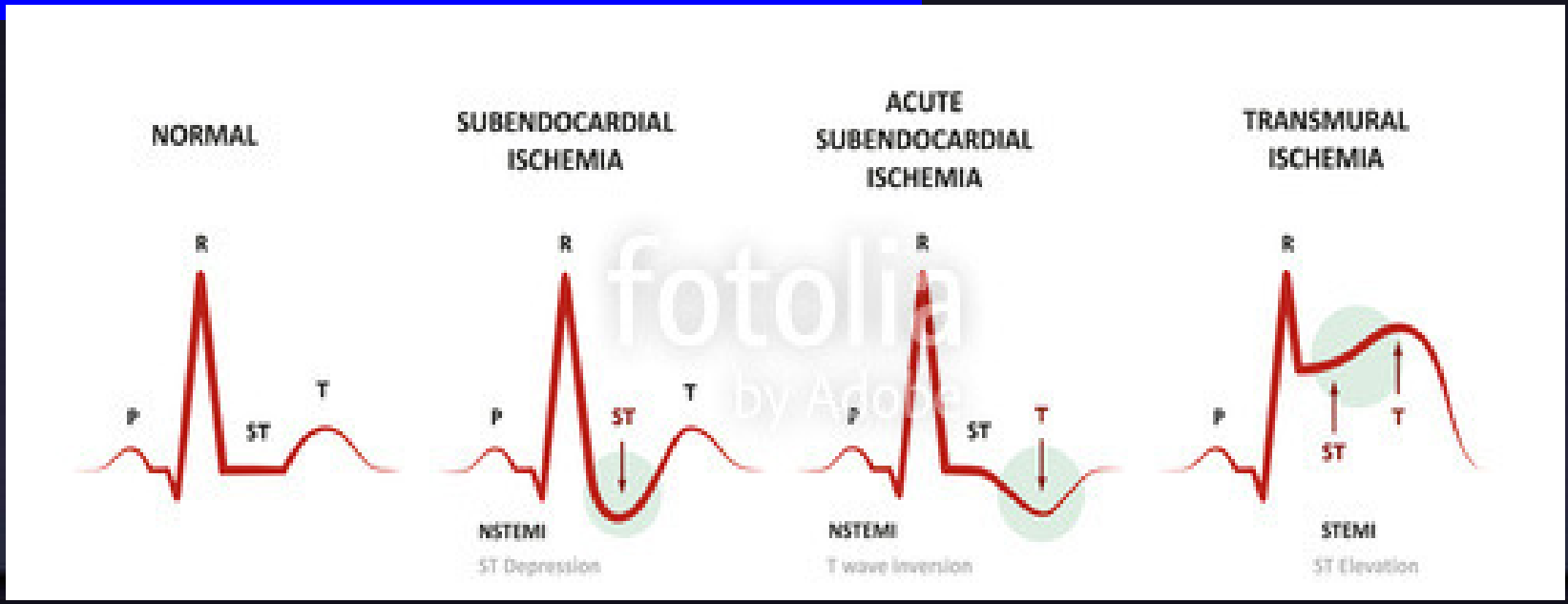
MYOCARDIAL INFRACTION:

Most common cause of death. Clinical features:

Chest pain – even at rest & last for hours. Severe pain – sudden onset, but can develop gradually.

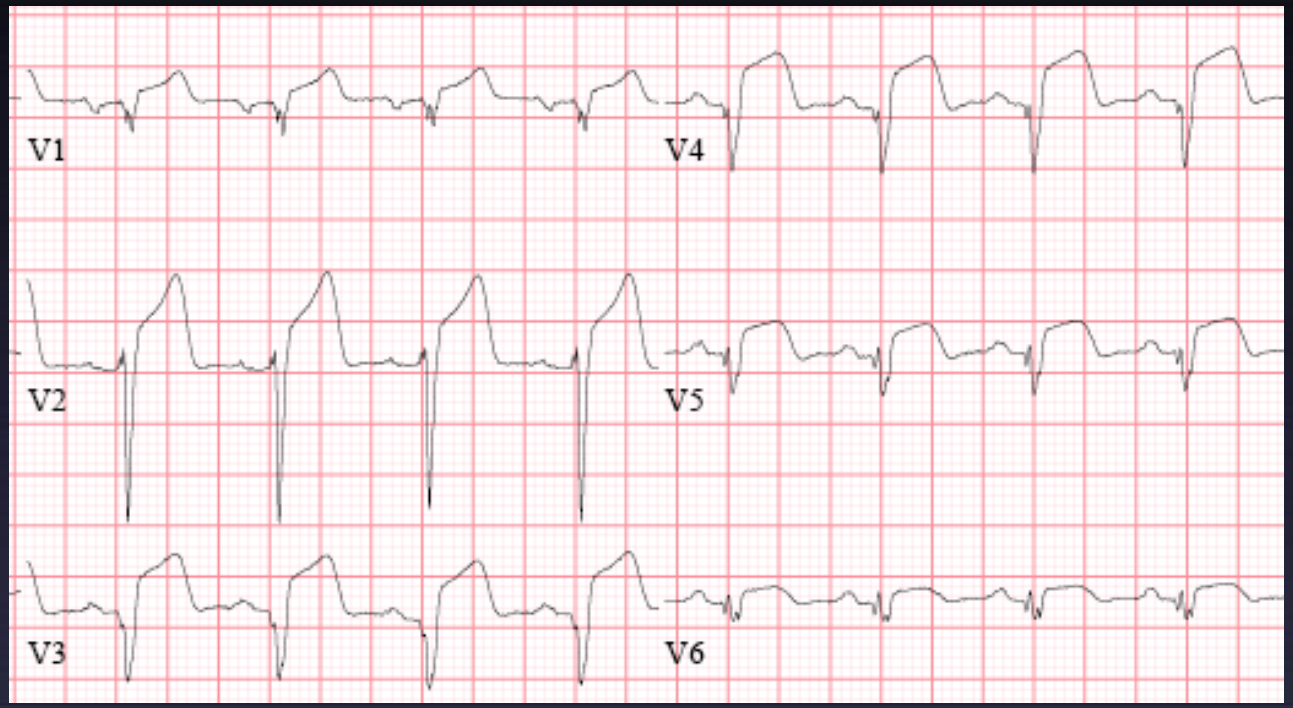
Associated with: sweating, vomiting. 20% no pain. Hypotension.





INVESTIGATIONS:-

- Cardiac enzyme – CK (creatine kinase), AST (aspartate aminotrasferase), LDH (lactic dehydrogenase)
- ECG: Q wave, ST elevation, T inversion. Q wave – full thickness infraction.



Subendocardial Infarction

- Persistent ST depression

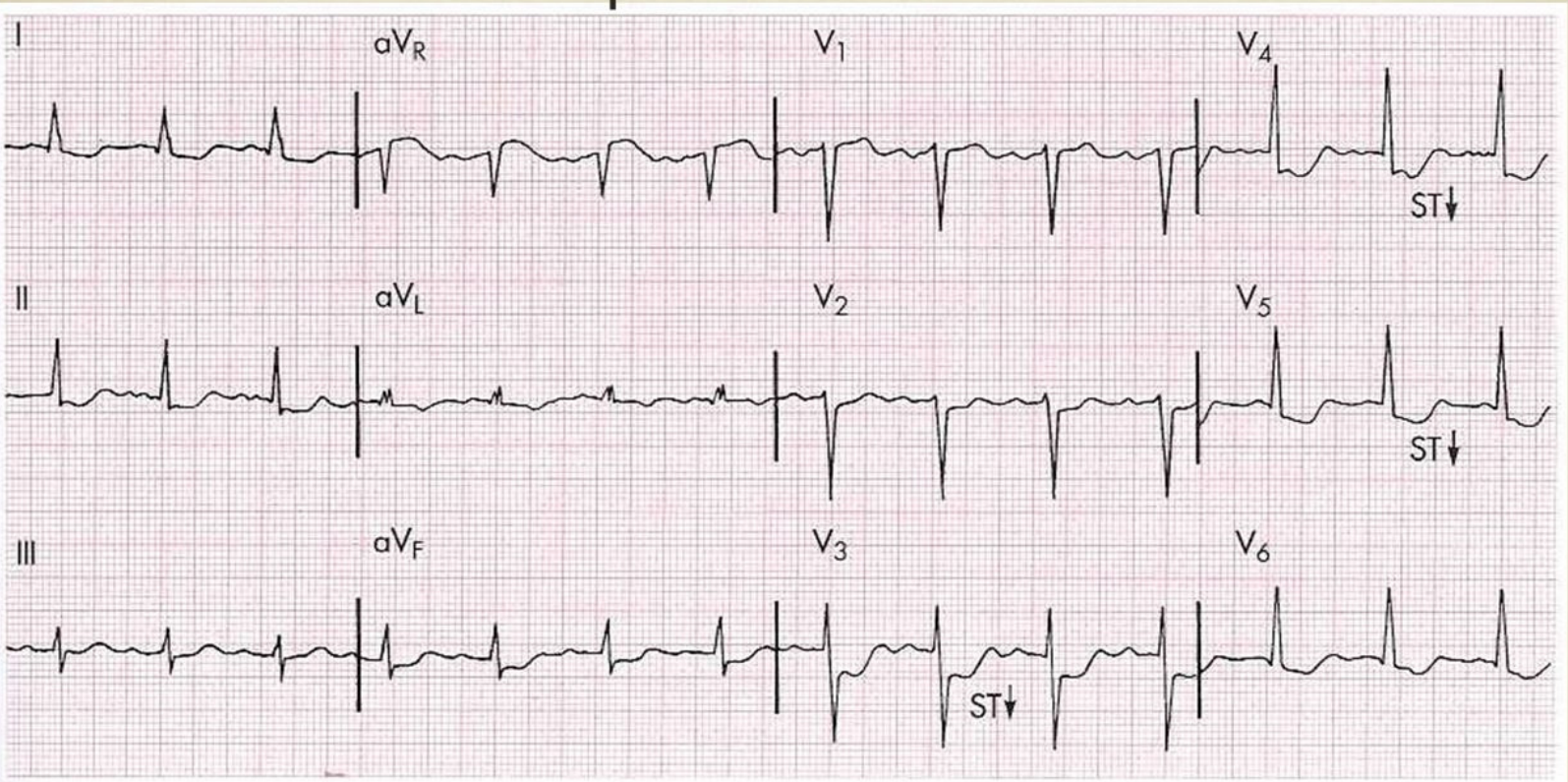


FIGURE 9-6 Non–Q wave infarction in a patient who complained of severe chest pain. Subsequently, the patient's cardiac enzyme levels were elevated. Notice the marked, diffuse ST depressions in leads I, II, III, aV_L, aV_F, and V_2 to V_6, in conjunction with the ST elevation in lead aV_R. These findings are consistent with severe subendocardial ischemia. Other abnormalities include a prolonged PR interval (0.28 sec) and left atrial abnormality.

When the normal portions of the ventricular muscle contract, the ischemic portion of the muscle, whether it is dead or simply nonfunctional, instead of contracting is forced outward by the pressure that develops inside the ventricle.

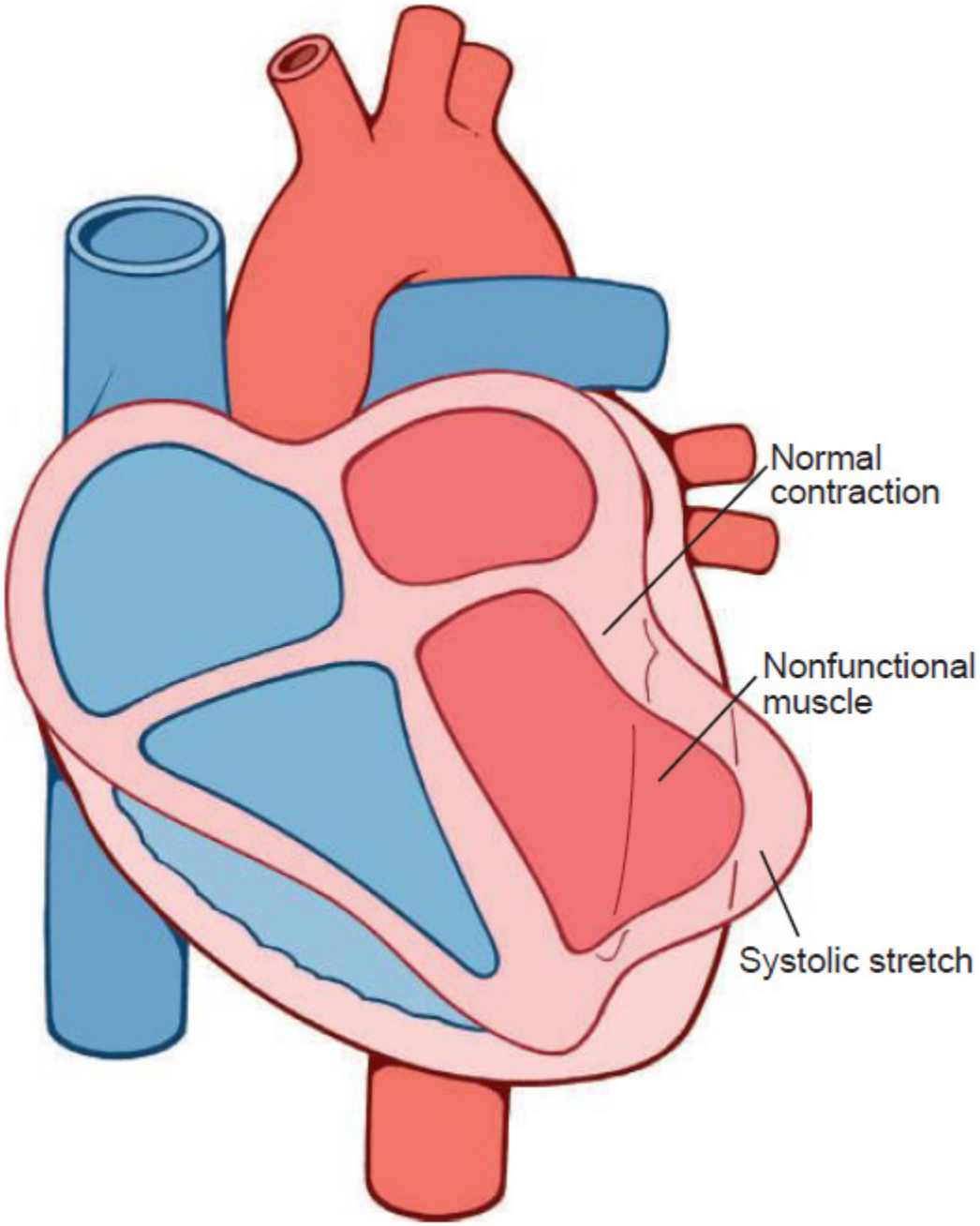


Figure 21-7. Systolic stretch in an area of ischemic cardiac muscle.