Coronary Circulation

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.

Aerobic Requirements of the Heart

Survival requires that the heart and brain receive adequate blood supply at all times.

Coronary arteries supply an enormous number of capillaries.

Systole compresses the coronary blood vessels.

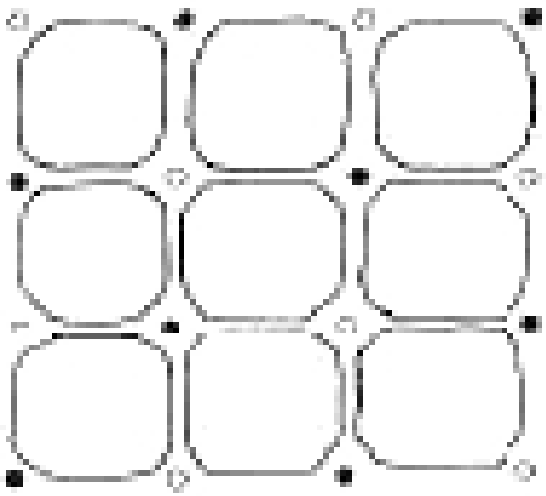
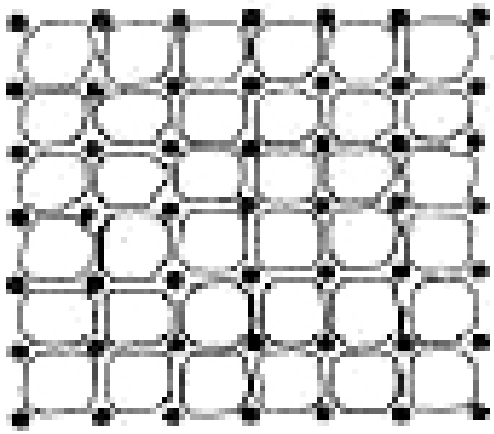
Diastole increases blood flow to the heart muscle.

Myocardium contains large amounts of myoglobin.

Myoglobin stores O_2 during diastole to release during systole.

Heart muscle contains increased number of mitochondria and aerobic respiratory enzymes.

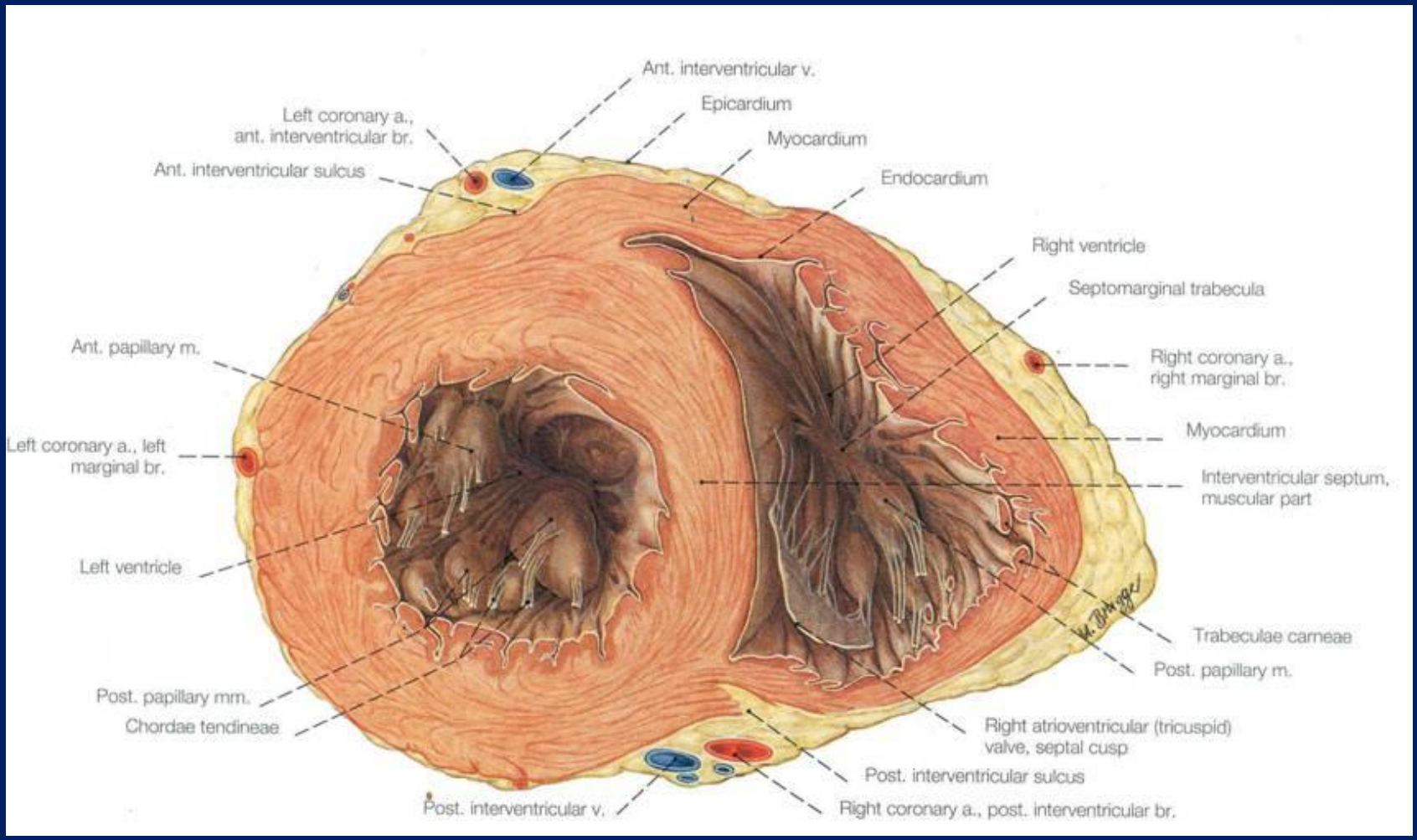
Capillary Density in the Heart

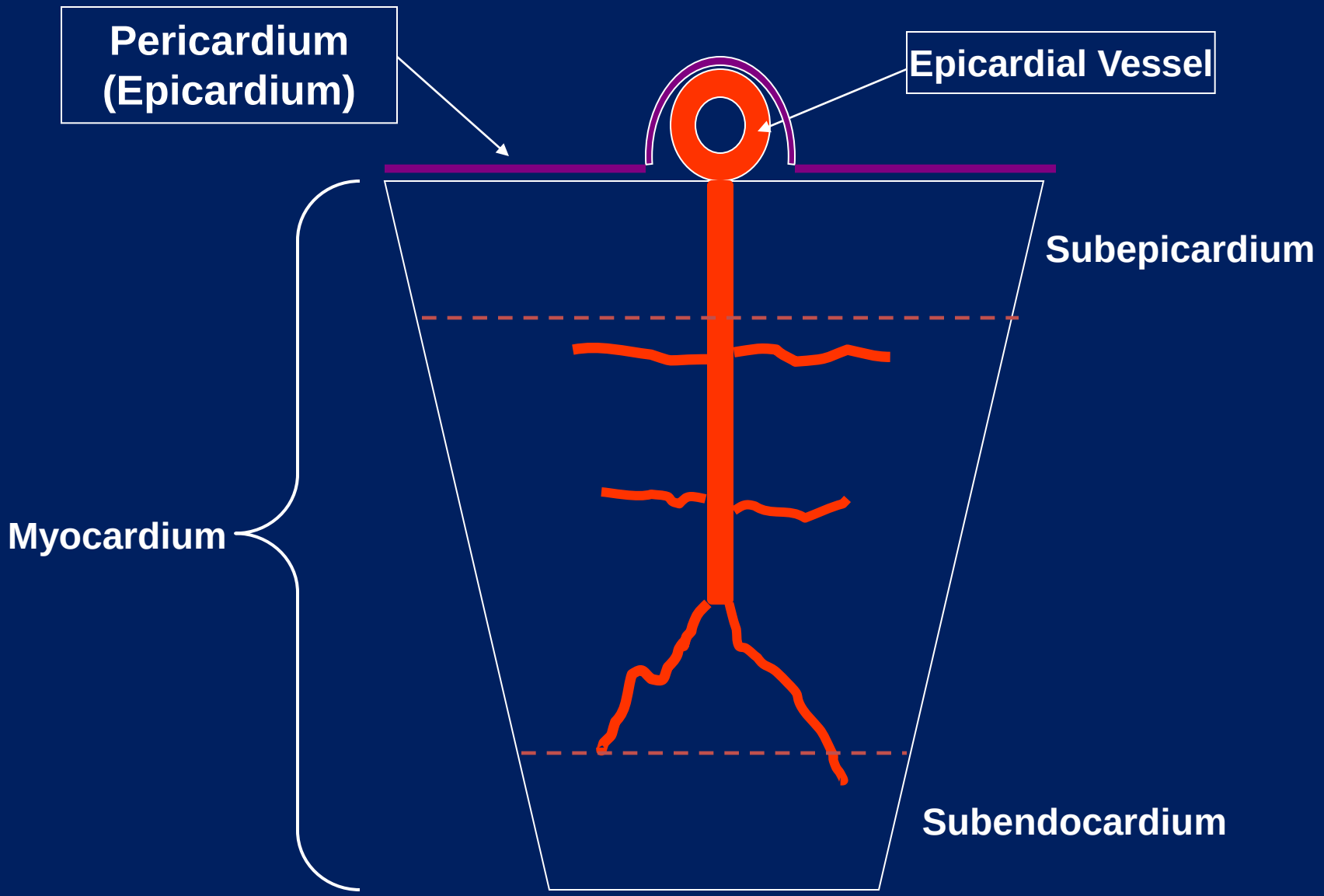
	Skeletal muscle	Cardiac muscle
		
Fibre diameter	50 µm	18 µm
Capillaries per mm^2	400	3000

NEUTRIENT SUPPLY TO HEART

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

Walls of the ventricles:
Left wall is thicker!




Pericardium
(Epicardium)
Epicardial Vessel
Subepicardium
Myocardium
Subendocardium

Coronary Vascular Resistance

Epicardial conductance vessels:

Contribute only to a small % of resistance.

Intramyocardial vessels (arterioles):

Contribute most to total coronary vascular resistance.

Transmural Distribution of Myocardial Blood Flow

Extravascular compressive forces are greater in The subendocardium (inner) and least near the Subepicardial Layer (outer).

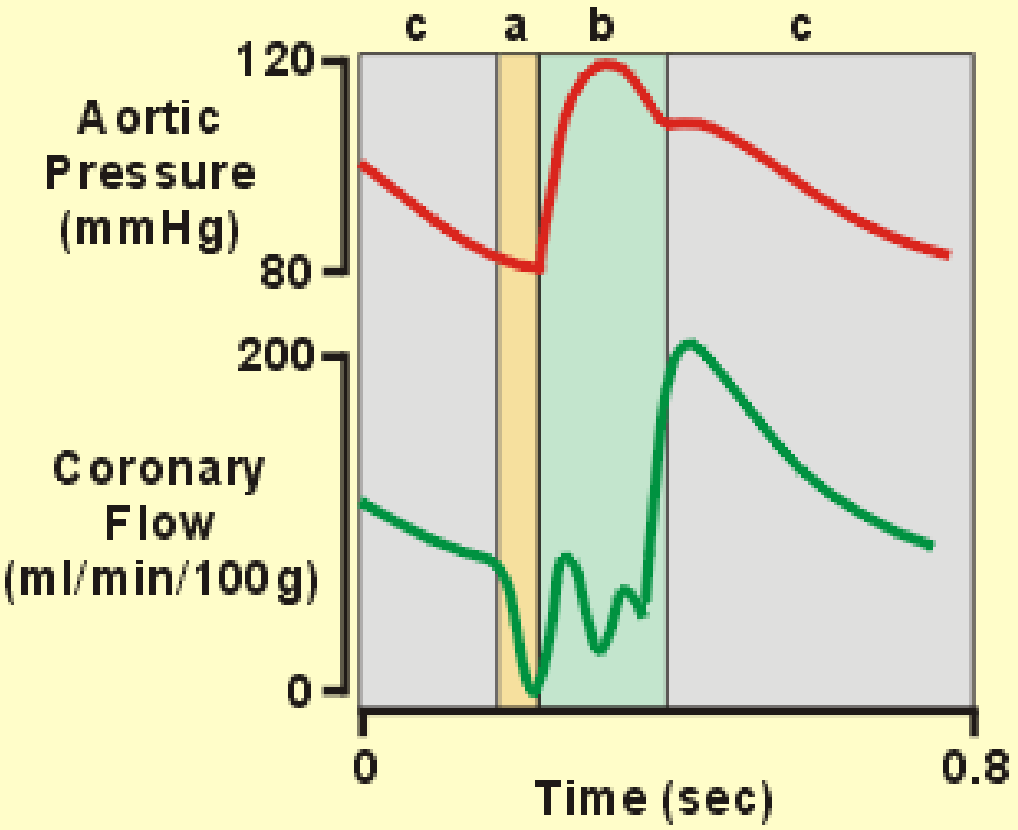
Under normal resting conditions this does not impair subendocardial blood flow as increased flow during diastole compensates.

Blood flow to Heart during Systole & Diastole

- During systole when heart muscle contracts it compresses the coronary arteries therefore blood flow is **reduced** to the left ventricle during systole and **increased** during diastole.

- Blood flow to the **subendocardial** portion of the left ventricle occurs only **during diastole**

Extravascular Compressive Forces



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

Transmural Distribution of Myocardial Blood Flow

The subendocardium is more susceptible to ischemic damage and most common site of myocardial infarction than the midmyocardium or subepicardium.

Coronary Circulation:

Resting coronary blood flow = 225 ml/min.

About 4 to 5 % of total cardiac output.

'Work' of the heart under severe conditions may increase 7 to 9 folds.

Coronary blood flow increases 3 to 4 folds to supply the extra nutrients.

CORONARY BLOOD FLOW

At rest, the heart extracts 60-70% of oxygen from each unit of blood delivered to heart (other tissues extract only 25% of O_2).

Why the heart is extracting 60-70% of O_2?

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 more O_2.

When more oxygen is needed e.g. In exercise, O_2 can be increased to heart only by increasing blood flow.

In aortic stenosis → Ischemia prone to develop.

↓

Increase pressure in ventricle and vessels compressed more.

↓

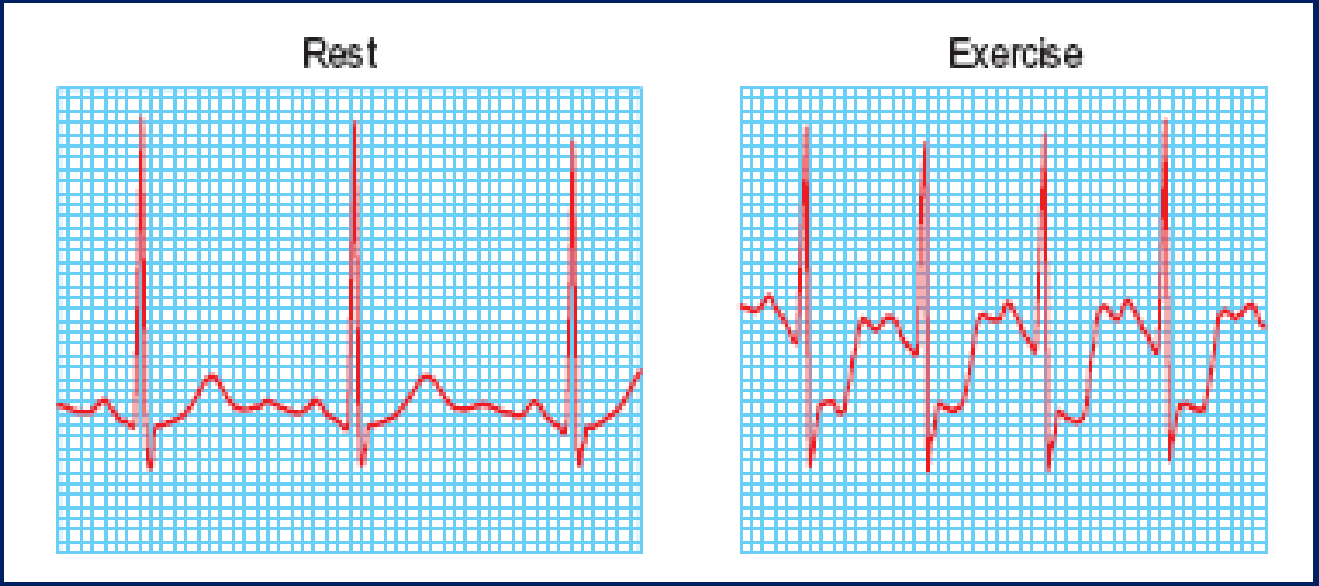
More O_2 needed due to high workload.

↓

Congestive heart failure
Failure of pumping action
Low effective coronary perfusion

Effect of Tachycardia on coronary blood flow:

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



Electrocardiographic changes during exercise test. – significant horizontal ST segment depression during exercise.

Myocardial Oxygen Supply

Determined by:

Coronary Blood Flow & **O_2 Carrying Capacity**

(Flow = Pressure / Resistance)

- **Coronary perfusion pressure**
- **Coronary vascular resistance**

- **Oxygen saturation of the blood**
- **Hemoglobin content of the blood**

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

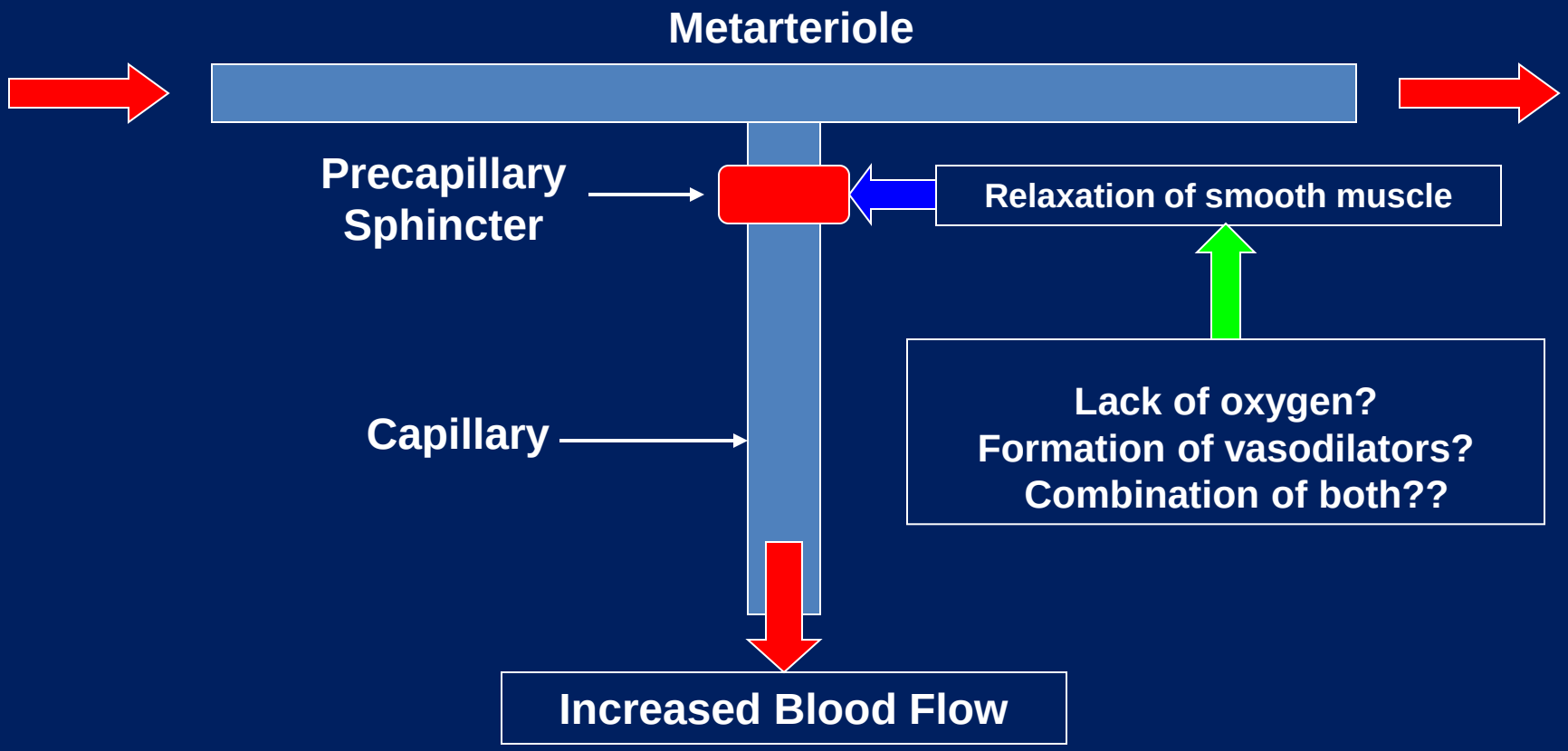
Coronary circulation is very sensitive to myocardial tissue oxygen tension.

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

Metabolic Control of Blood Flow



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.

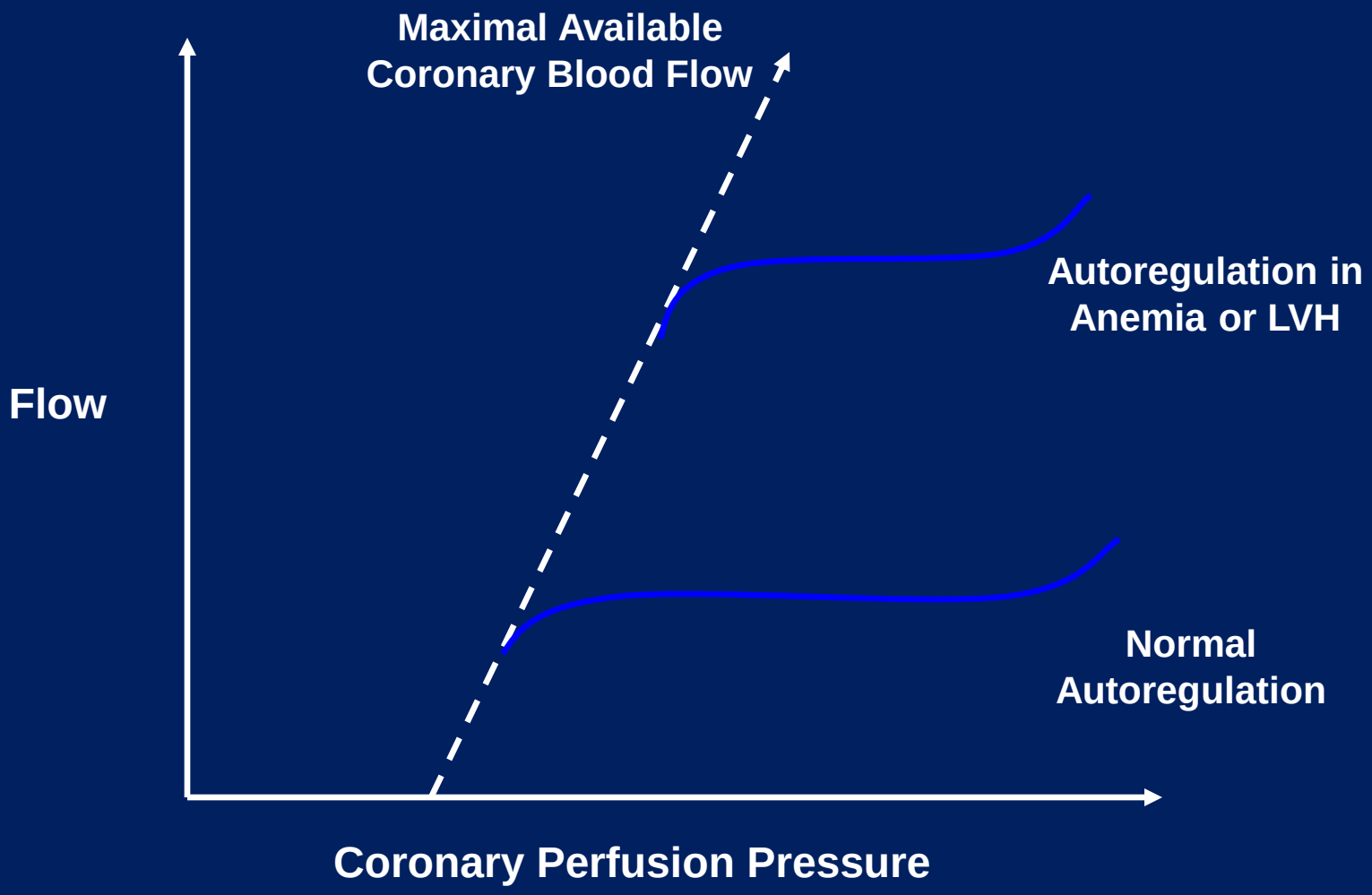
Autoregulation of Coronary Blood Flow

- **Oxygen**
  - Acts as vasoconstrictor
  - As O2 levels drop during ischemia: pre-capillary vasodilation and increased myocardial blood supply
- **Adenosine**
  - Potent vasodilator
  - Prime mediator of coronary vascular tone
  - Binds to receptors on vascular smooth muscle, decreasing calcium entry into cell

Adenosine

- During hypoxemia, aerobic metabolism in mitochondria is inhibited
- Accumulation of ADP and AMP
- Production of adenosine
- Adenosine vasodilates arterioles
- Increased coronary blood flow

Autoregulation



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.

Neural Control

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

The heart influences its blood supply by The squeezing effect of the contracting Myocardium on the blood vessels coursing through the heart.

Extravascular Compressive Forces

Left Ventricle:

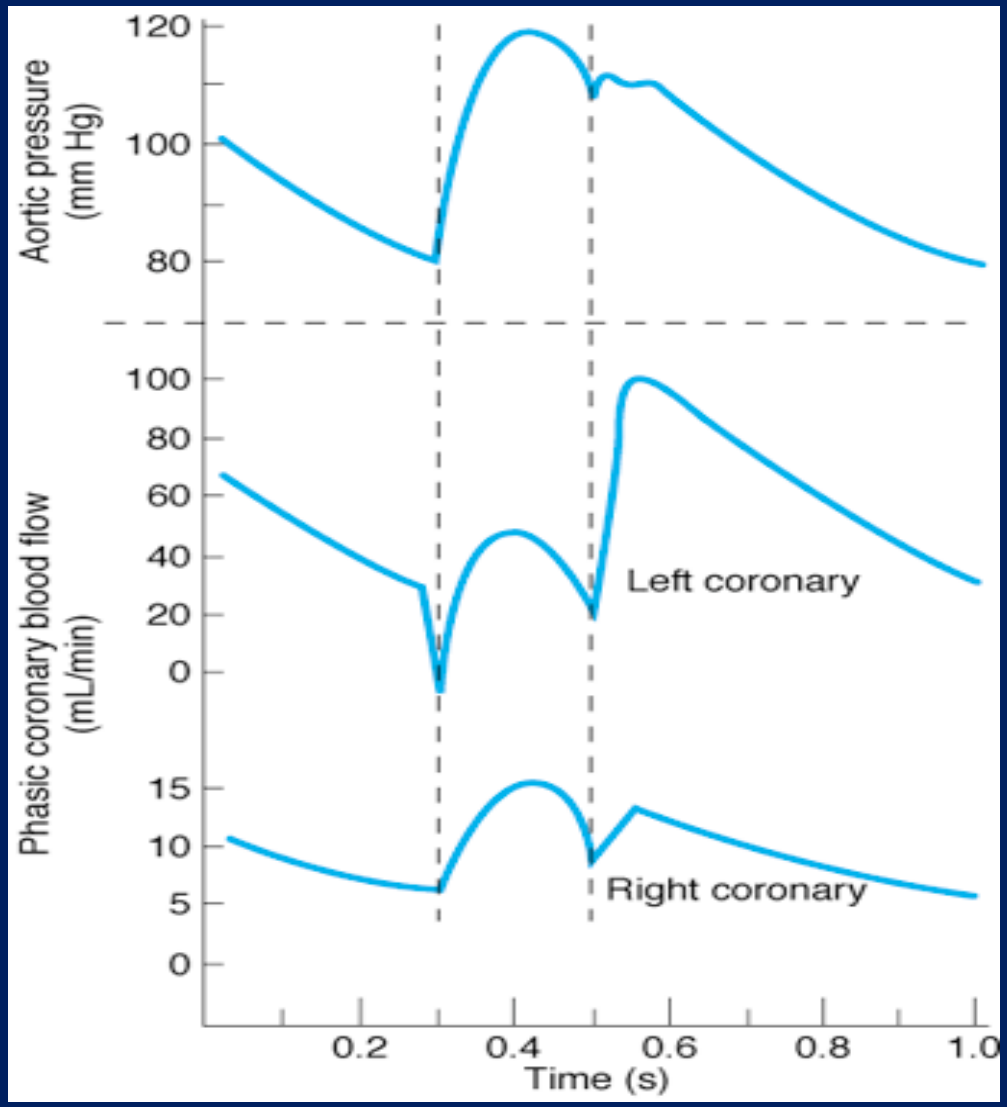
Early Systole > Initial Flow Reversal.

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.

CORONARY BLOOD FLOW DURING SYSTOLE AND DIASTOLE



Extravascular Compressive Forces

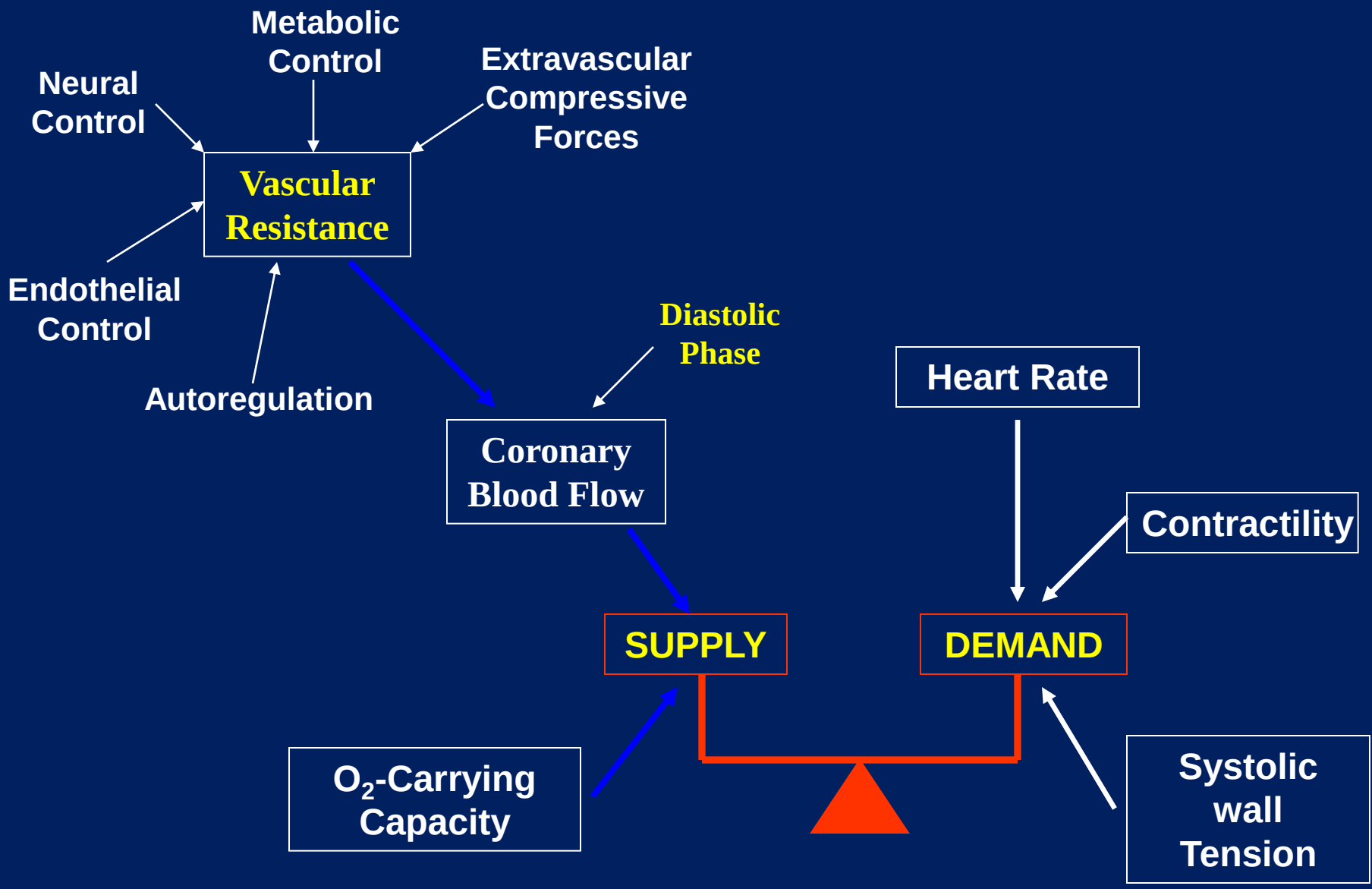
Right Ventricle:

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.

Determinants of Myocardial Oxygen Supply and Demand


Metabolic Control
Neural Control
Extravascular Compressive Forces
Vascular Resistance
Endothelial Control
Autoregulation
Diastolic Phase
Heart Rate
Coronary Blood Flow
Contractility
SUPPLY
DEMAND
O2-Carrying Capacity
Systolic wall Tension

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.

- ISCHEMIC HEART DISEASE (IHD) (ANGINA PECTORIS)

- MYOCARDIAL INFARCTION

ANGINA PECTORIS:

THERE IS REDUCED CORONARY ARTERY BLOOD FLOW DUE TO ATHEROSCLEROSIS (CHOLESTROL DEPOSITION)

SUBENDOCARDIALLY -- Plaque)

CAUSES OF IHD:

- CIGARETTE SMOKING
- HYPERTENSION
- DIABETES MELLITUS
- INCREASED LIPIDS (CHOLESTROL)
- OTHER FACTORS: LACK OF EXERCISE, ANXIETY etc.

Myocardial infraction:

Most common cause of death.

ATLEAST 75 % OF LUMEN OF CORONARY ARTERY IS BLOCKED BY THROMBUS.

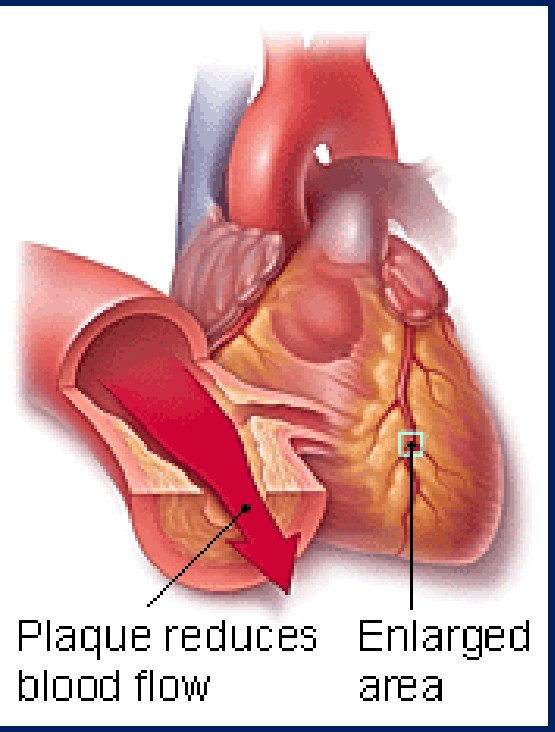
Clinical features:
Chest pain – even at rest & last for hours.

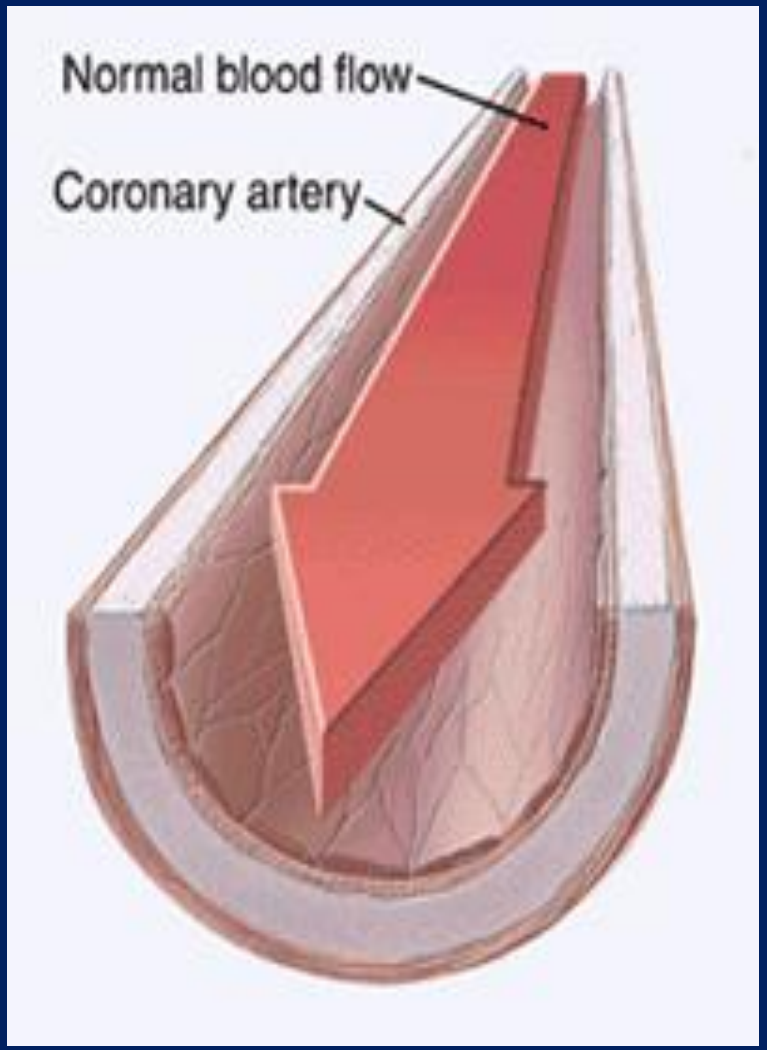
Severe pain – sudden onset, but can develop gradually.

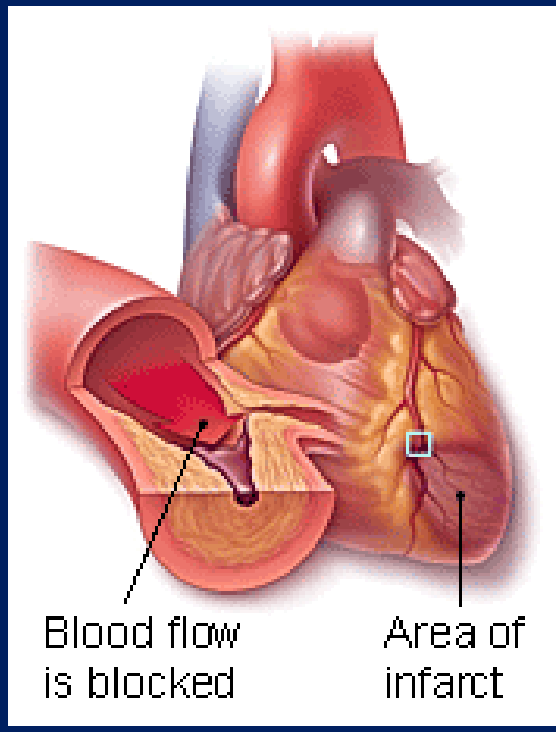
Associated with: sweating, vomiting.

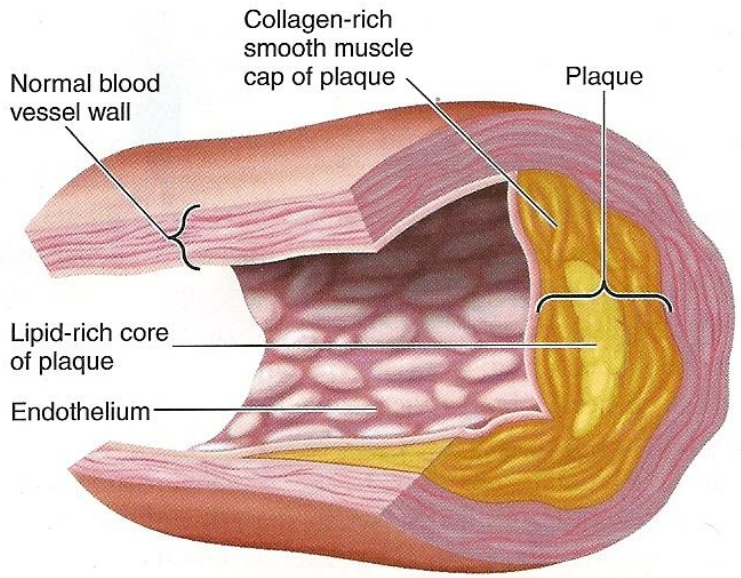
20% no pain.

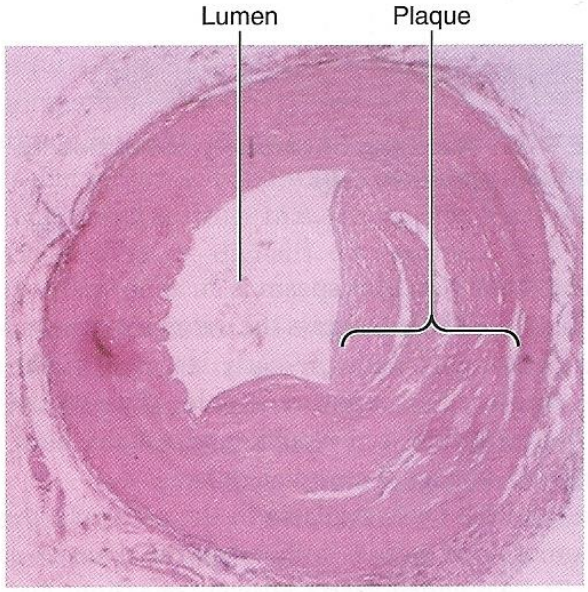
Hypotension.


Plaque reduces blood flow
Enlarged area


Normal blood flow
Coronary artery


Blood flow is blocked
Area of infarct

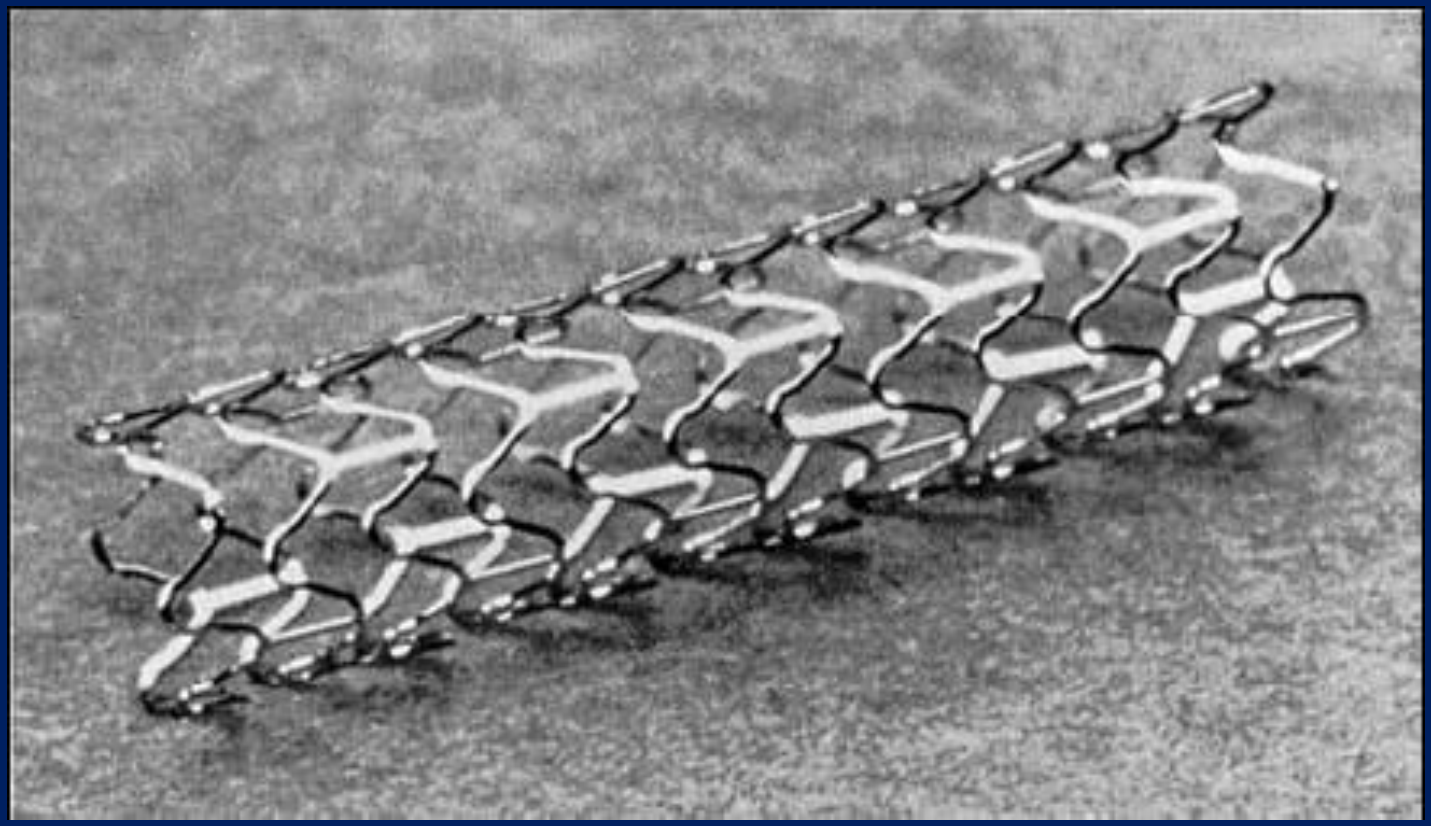




● **FIGURE 9-28** **Atherosclerotic plaque in a coronary vessel.**

TREATMENT:

- CORONARY DILATORS E.g. NITRATES
- BETA-BLOCKERS
- ANGIOPLASTY (DILATE AREA OF CONSTRICTION)
- STENT
- BYPASS SURGERY



An intracoronary stent.

CORONARY ARTERY BYPASS SURGERY

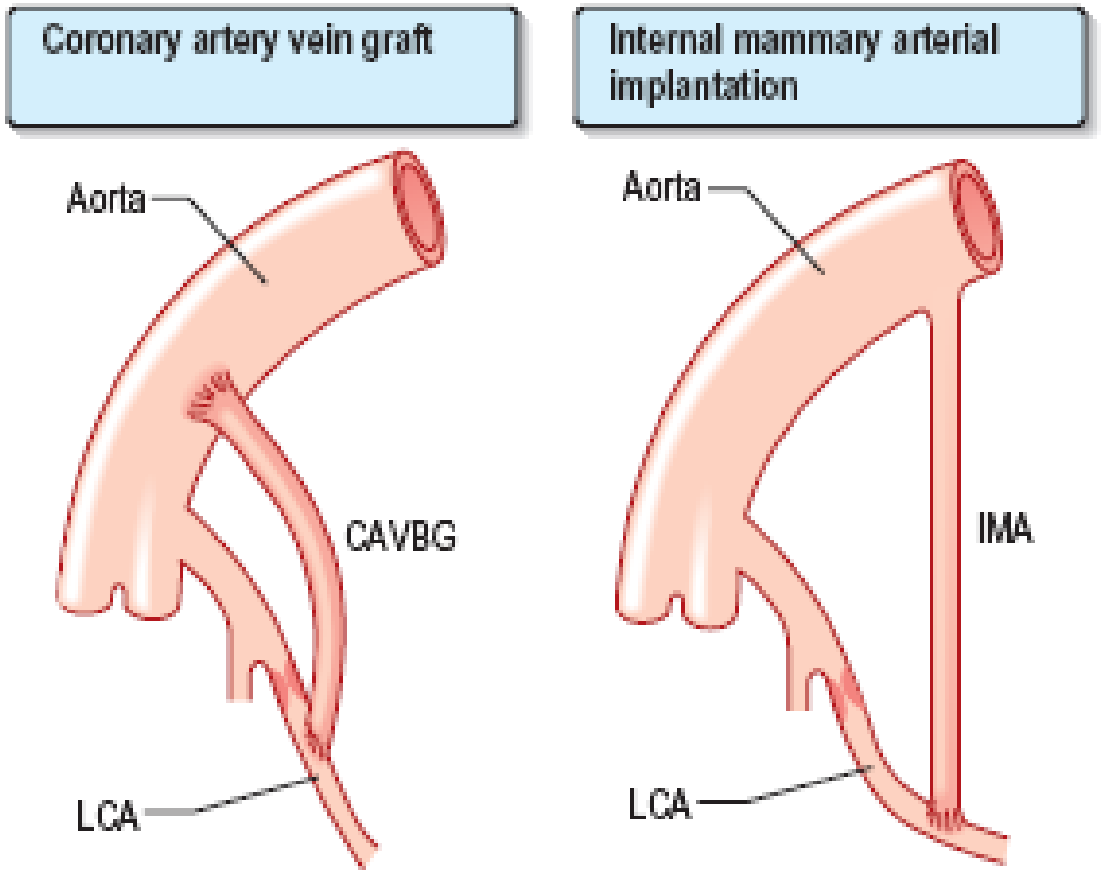


Fig. 13.71 **Relief of coronary obstruction by surgical techniques:** coronary artery vein bypass grafting (CAVBG) or internal mammary arterial implantation (IMA). In both of these examples, the graft bypasses a coronary obstruction in the left coronary artery (LCA).