

CARDIOVASCULAR SYSTEM

SUBJECT : physiology

LEC NO. : 14

DONE BY : Abdullah Bani Mustafa

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SCAN ME!

Coronary Circulation

Dr. Waleed R. Ezzat

Lecture Objectives:

1. Explain normal coronary blood flow during systole and diastole to different parts of the myocardium.
2. Discuss the local factors for control of coronary blood flow, local metabolism as primary factor, and the oxygen demand.
3. Describe the effect of autonomic nervous system on coronary arteries, role of Alpha, and Beta-receptors.
4. Define ischemic heart disease, the cause of cardiac pain, and the mechanism of collateral circulation.

Pattern of coronary blood flow

- The normal coronary blood flow in the resting human being is about 4 to 5% of the total cardiac output. *so it's huge amount compared to its size*
- * ■ Under severe conditions the work output of the heart may increase sixfold to ninefold. However, the coronary blood flow increases only threefold to fourfold to supply the extra nutrients needed by the heart. This deficiency of coronary blood supply is overcome by increasing the “**efficiency**” of cardiac utilization of energy. *deficiency doesn't mean decrease the coronary blood flow it means that the increase in its flow when cardiac output increase is less than the increase in cardiac output so it is overcome by increase efficiency*
- The flow in left ventricular capillaries decrease during systole because of the strong compression of the left ventricular muscle around the intramuscular vessels. *explained in the next slide*
- Blood flow in the left coronary artery during cardiac systole is only 10% to 30% of that during diastole, and much of that represents flow to the epicardium. *that means the segments that supplies myocardium & endocardium are completely lost or cut off during systole so that is why during systole the endocardial surface go under local ischemia*
- In heart diseases of all types, therefore, the **subendocardial** layers of the heart suffer more severe impairment and ischemia than do the epicardial layers. *that means if blood flow is decreased to the heart the endocardial surface will suffer much more than the epicardial surface.*
- During diastole blood flows rapidly through relaxed cardiac muscle.
- On the right side of the heart no phasic changes occurs during the cardiac cycle as a result of the lower pressures developed by that chamber.

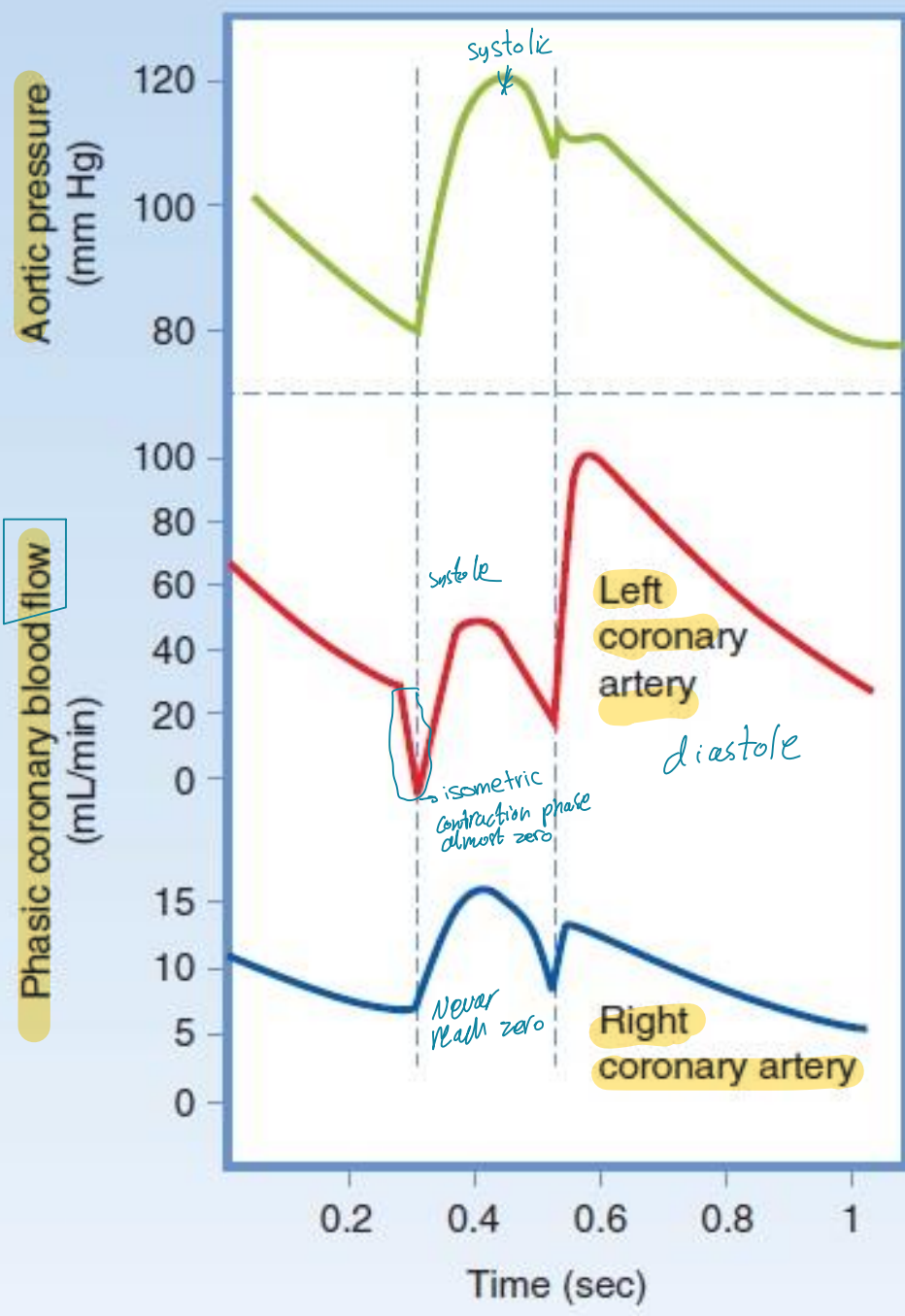
what do we mean by increasing efficiency?

remember from RS arterial blood O_2 is about 100 while in the venous is about 40 is the utilisation of oxygen is around 60, while in coronary circulation the venous O_2 is about 20 so the utilisation of O_2 is about 80 so that means that the myocardium has a great ability to take the O_2 from the arterial blood so it's much more efficient to utilise O_2 to produce energy

Explanation of point 3:

in the heart we have 2 ventricles L & R which are both supplied by the coronary arteries which originate from the ascending aorta so their pressure is equal to the aortic pressure, the flow in the coronary arteries depend on the difference of pressure gradient usually we say the difference between the arterial & venous side but in the heart we have a trait that is not found in other organs which is that the branching of coronary arteries it passes the wall thickness of ventricles, in LV when it passes through its fibers so when the LV contract it will press on the the blood vessels that passes through it walls, also this contraction produce the pressure in the aorta ≈ 100 & then the aorta through the coronary arteries will supply blood to the ventricles, but LV pressure is stronger so it will be able to compress the coronary arteries to almost close it & cut the circulation this is due to the higher pressure in the LV \rightarrow this is only found in the heart because the pressure in the other organs will never exceed the pressure in the blood vessels

but in the RV the pressure is ≈ 25 and in the aorta is ≈ 100 so it won't be able to compress the R coronary branches that supplies it.



we can conclude from the graph that the L while suffer more in case of ischemia

Regulation of coronary blood flow

the higher the metabolic activity the higher the blood flow in coronary circulation.

auto-regulated means that even if we cut the nerves or in heart transplant patient the blood flow in the coronary arteries depend on the metabolism of myocardium

1. The flow is entirely regulated by local mechanism. which mean it's auto-regulated
2. The greater the myocardial contraction the greater is the coronary blood flow. This relationship is also found in a denervated heart and in a completely isolated heart.
 *more contraction
more O₂ needed
higher metabolic rate → higher blood flow in coronary circulation*
3. The flow is in direct proportion to the cardiac muscle O₂ demand (or consumption). **Adenosine and NO** are the main vasodilators that are believed to be released from the myocardium when O₂ demands increases (when metabolism increases). Blockade of the vasodilator actions of adenosine with **theophylline**, however, does not prevent coronary vasodilation when cardiac work is increased.
 that is why heart transplant is possible
4. The vasodilator effect of sympathetic stimulation on the coronary artery is indirect. The direct effect is vasoconstriction. The positive inotropic and chronotropic effect of sympathetic stimulation increases the myocardial metabolism and O₂ demands. This will trigger the local blood flow regulatory mechanism and dilate coronary vessels. Vagal stimulation ends by indirect vasoconstriction since it decreases the O₂ demands.
5. The coronary artery has β_2 and α_1 adrenergic receptors.

same as any arteries in the body

The main mechanism that controls the blood flow in coronary circulation:

it's believed that nitric oxide (NO) & adenosine are the main regulator for the blood flow in coronary circulation, when O_2 demand increase the myocardium will release NO & adenosine which are consider strong vasodilator which will affect the branches of the coronary arteries that supplies the heart muscle so it will cause vasodilatation in them causing an increase in blood flow but know that they are not the only factors that controls the blood flow in the coronary circulation which is the sympathetic stimulation but remember sympathetic stimulation in all body parts produce Noradrenaline which acts as vasoconstriction in almost all body parts that have α_1 receptors BUT in the heart it will cause vasodilation in indirect way to the coronary arteries through this mechanism:

sympathetic supplies the myocardium so when it's stimulated it will increase the Inotropic which will lead to the production of Adenosine & NO causing vasodilation

Ischemic heart disease (IHD)

- Most of the O_2 in coronary arterial blood is extracted during one passage through the myocardial capillaries. Thus the supply of O_2 to myocardial cells is **flow limited**. Oxygen extraction is nearly maximal even when blood flow is normal.
- IHD is the insufficient coronary blood flow to the heart. The ischemia may be global (affects an entire ventricle) or regional (affects some fraction of the ventricle).
- The most frequent cause of IHD is the **atherosclerosis** which frequently develops in the first few centimeters of the major coronary arteries. *it occur at the beginning of the artery*
atherosclerosis cause narrowing of the coronary arteries so the person become at greater danger to develop ischemic heart disease
- Acute coronary occlusion is a common complication of atherosclerosis.
- Gradual constriction of coronary arteries by atherosclerosis is accompanied by collateral vessels development (long-term regulation of flow), and no acute episodes of cardiac dysfunction may be recognized. *the collateral vessel development is possible because coronary arteries are rich in anastomosis as we taught in anatomy lectures*
myocardial infarction
- Collaterals in **sudden occlusion** supply less than half the flow that is needed to keep the cardiac muscle alive. Then collateral flow begins to increase, doubling by the second or third day and often reaching normal or almost normal coronary flow within about 1 month. *so the issue with atherosclerosis is the symptoms only appear after 60-70% of the artery is closed because the anastomosis can no longer compensate for the narrowing of coronary arteries*

الذبحة الصدرية
stimulated by exercise or emotional stress and disappear at rest

The cardiac pain (angina pectoris)

a temporary / small part occlusion of coronary arteries

→ atherosclerosis occur → No symptoms at rest due to collateral circulation → during exercise or any activity that need the cardiac output to increase → the branches won't give enough supply to the heart → during this short period of time during the need of more CO → O₂ demand is more than the O₂ supplied → causing pain

- It is believed that the pain is due to substances; such as lactic acid, histamine, kinins, or cellular proteolytic enzymes. this pain is caused by the decrease of O₂ lead to the accumulation of metabolite → which stimulate the nerve endings → signal pain to the brain by sympathetic branches
- Pain impulses are conducted through the sympathetic sensory afferent nerve fibers. → the pain is felt in the upper part of the sternum and radiate to right arm left shoulder and may neck & side of the face
- Angina appears when the O₂ demand exceeds the supply by the coronary blood flow.
- Pain is felt beneath the upper sternum and is often referred to the left arm, left shoulder, neck, and even to the side of the face. Pain is triggered by exercise or emotional stress.

Line of treatment

* β blockers reduce this drug activity how?
because NO cause vasodilation which lead to \downarrow BP \rightarrow baroreceptors will sense it cause reflex sympathetic stimulation so β blockers will reduce the reflex effect
 \rightarrow this is used as immediate medication when angina occur but to prevent it from happening we give β blockers & Ca²⁺ channels antagonist

- Many drugs are available for use in patients with coronary artery disease to relieve angina pectoris, the chest pain associated with myocardial ischemia. These compounds include **organic nitrates/nitrites**, **calcium channel antagonists**, and **β -adrenoceptor antagonists**.

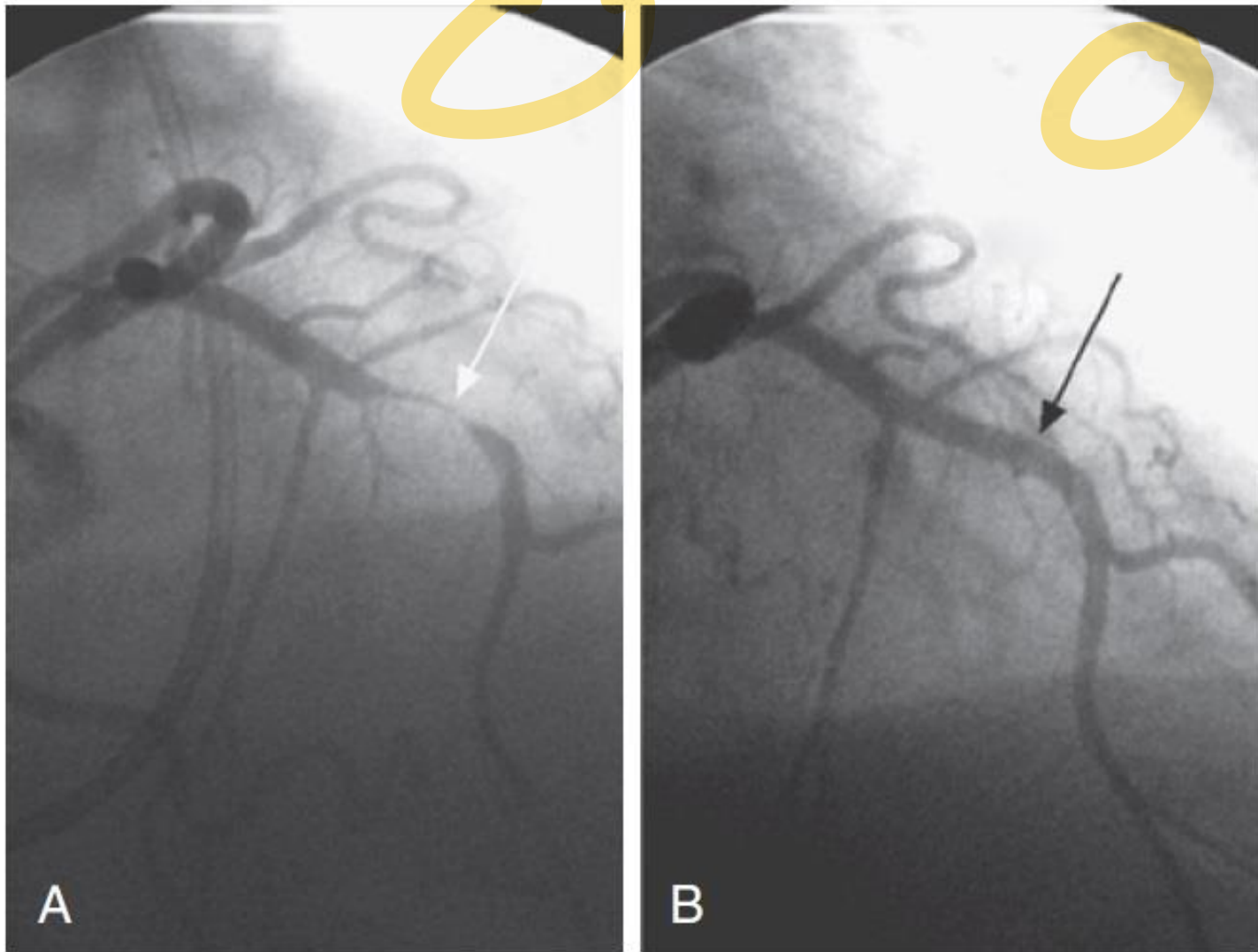
- On crisis a vasodilator of the venous system is used to reduce the venous return and preload on the heart such as nitrate drugs.

- Organic nitrates/nitrites are metabolized to **NO**. NO dilates the great veins and reduce venous return, thereby reducing cardiac work and myocardial O_2 requirements. In addition, NO dilates the coronary arteries to increase collateral flow. *low flow \rightarrow increase diameter increase tension \rightarrow more O_2 needed vice versa*

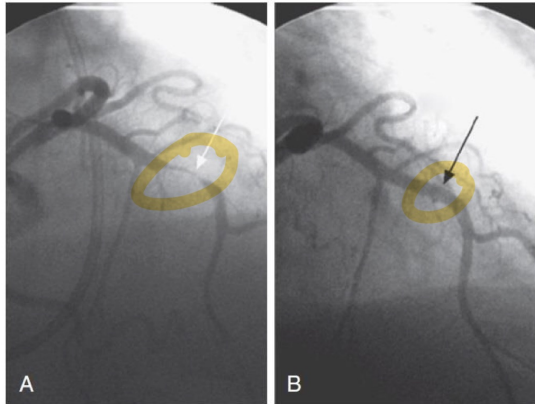
- For prophylaxis β blockers are used to block the sympathetic effect on the heart. Also the β -adrenoceptor antagonists reduce the heart rate to indirectly increase coronary flow and oppose the reflex tachycardia that has been observed with organic nitrates/nitrites. *sympathetic stimulation increase myocardial contractility \rightarrow increase O_2 demand \rightarrow so β blockers reduce it*

- Calcium channel antagonists also cause vasodilation; none selectively dilates the coronary vessels.

- Frequently, the narrow segment can be dilated by insertion of a balloon-tipped catheter into the diseased vessel via a peripheral artery and then inflation of the balloon (**angioplasty**). This procedure can produce lasting dilation of a narrowed coronary artery, particularly when a drug-eluting stent (the drugs help prevent restenosis) is inserted during angioplasty. *the closed branches will won't be effected by the adenosine or nitric oxide \rightarrow because when originally the blood supply were reduced the No x A were at their maximum level, so when we give them we won't increase them in the affected area; it will work in collateral*



A, Angiogram (with intracoronary radiopaque dye) of marked narrowing of the left anterior descending branch of the left coronary artery (*white arrow*).
B, The same segment of the coronary artery (*black arrow*) after angioplasty and insertion of a drug-eluting stent.



A, Angiogram (with intracoronary radiopaque dye) of marked narrowing of the left anterior descending branch of the left coronary artery (white arrow).
B, The same segment of the coronary artery (black arrow) after angioplasty and insertion of a drug-eluting stent.

the white arrow is pointing at the blocked area of the coronary artery caused by atherosclerosis this cause the angina in patients during exercise, so we insert stent which inserted through inserting catheter which at its end there is a balloon which can be inflated, the catheter reach the partially closed area then they inflate the balloon causing dilation of coronary artery & to prevent it from closing again, the same catheter contain a wire called "guide" which through it they insert a stent that pass through it until it reached the closed area then they inflate the balloon again (note that the balloon is inside the stent then the stent open and stay open but in might cause clot so it's coated by anticoagulant material such as heparin

major causes of atherosclerosis:

- 1) smoking
- 2) diabetes
- 3) hyperlipidemia → LDL

<https://youtu.be/j9VY6FSIr64?si=JdAt3CMIL42tDTXq>



video for how angioplasty is done to make it more clear watch it before you read this slide

Test:

Pain due to poor coronary blood flow (angina) may be relieved by any of the following EXCEPT?

- A. Cutting the sympathetic nerve trunks supplying the heart.
- B. Correcting anemia if present.
- C. Providing the patient with a cold environment.
- D. Angioplasty and stent placement.
- E. Drugs causing peripheral vasodilation.