**V. N. Karazin Kharkiv National University**
Department of Propaedeutics of Internal Medicine and Physical Rehabilitation

Student's scientific community

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Scientific discussion

# Atherosclerosis – clinical features

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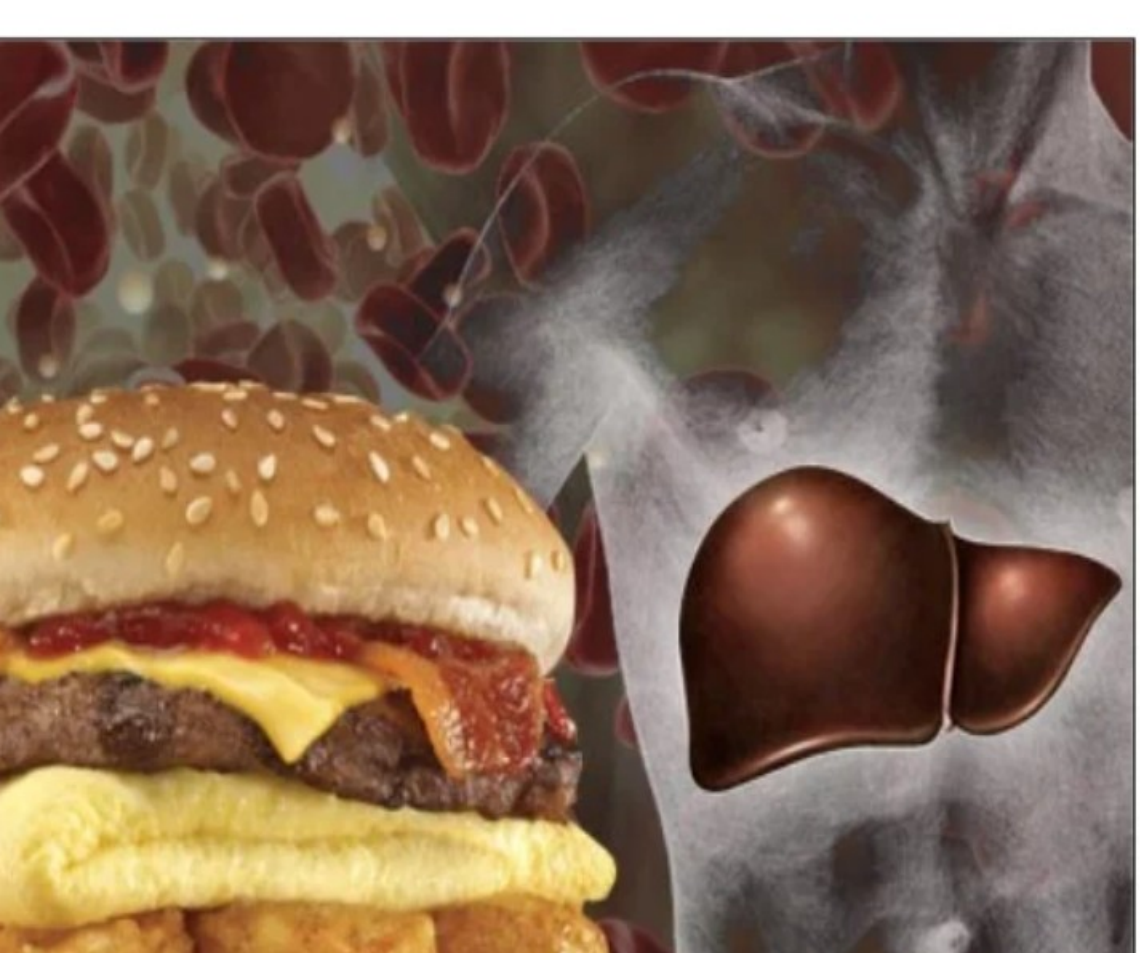
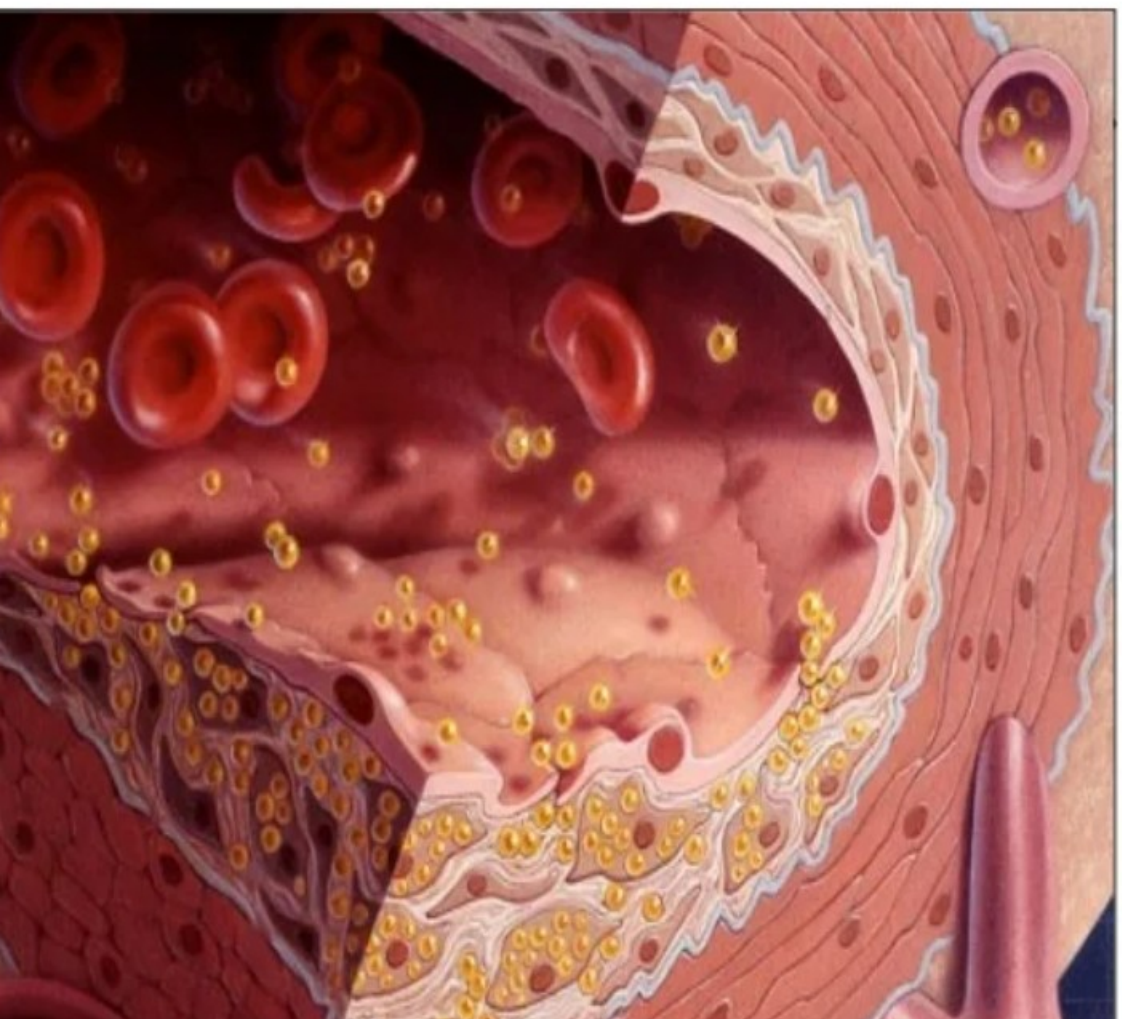
# What Is Cholesterol?



We may associate cholesterol with fatty foods, but most of waxy substance is made by o own bodies. The liver produc 75% of the cholesterol that circulates in our blood. The o 25% comes from food. At no levels, cholesterol actually pl an important role in helping do their jobs. But cholestero levels are precariously high i more than 100 million Indian

Cholesterol is located in every cell of th
ody. It is an oily, waxy ingredient that
s involved
with the making of chemicals,
nembranes, Vitamin D, bile acids or
ther tissue in your body. It also
nsulates the senses in our bodies.
Cholesterol is generally created in the
ver yet we get it from our diet.

# Symptoms of High Cholesterol

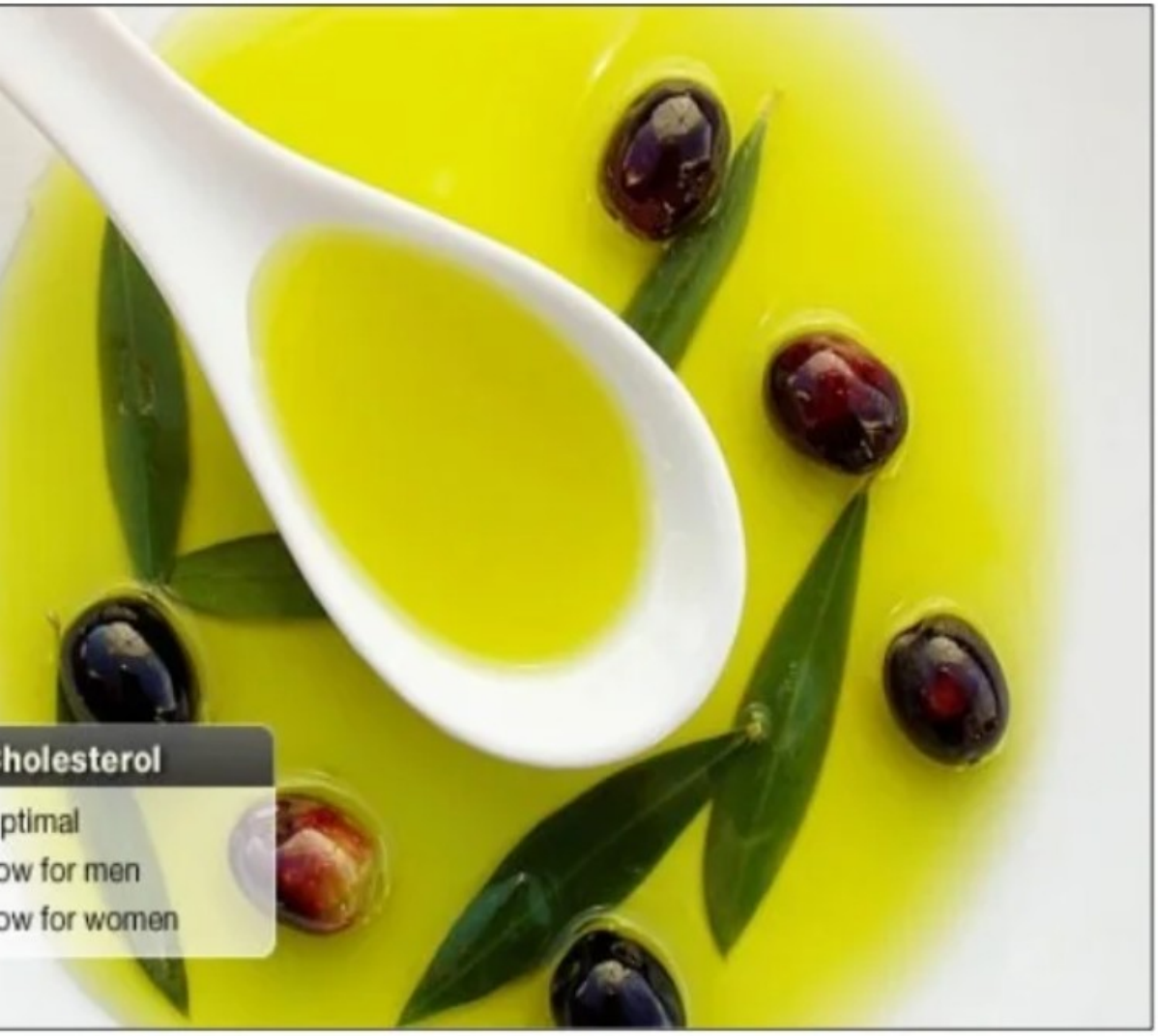


High cholesterol does not ca
any symptoms. But it does ca
damage deep within the bod
Over time, too much cholest
may lead to a buildup of plac
inside the arteries. Known as
atherosclerosis, this conditio
narrows the space available
blood flow and can trigger h
disease. The good news is hi
cholesterol is simple to dete
and there are many ways to
it down.

# "Bad" Cholesterol


Most of the cholesterol in t
blood is carried by proteins
low density lipoproteins or
This is known as the bad
cholesterol because it coml
with other substances to cl
arteries. A diet high in satu
fats and trans fats tends to
the level of LDL cholesterol
most people, an LDL score l
100 is healthy, but people w
heart disease may need to
even lower.

# "Good" Cholesterol




Up to a third of blood cho
is carried by high-density
lipoproteins or HDL. This is
good cholesterol because
remove bad cholesterol,
preventing it from building
inside the arteries. The hig
level of HDL cholesterol, th
better. People with too litt
more likely to develop hea
disease. Eating healthy fat
as olive oil, may help boos
cholesterol.

# Triglycerides


The body converts excess calories, sugar, and alcoho triglycerides, a type of fat t carried in the blood and st fat cells throughout the bo People who are overweigh inactive, smokers, or heavy drinkers tend to have high triglycerides, as do those w a very high-carb diet. A triglycerides score of 150 o higher puts you at risk for metabolic syndrome, whic linked to heart disease and diabetes.

# Total Cholesterol


Total cholesterol measures
combination of LDL, HDL, a
VLDL (very low density
lipoprotein) in your bloods
VLDL is a precursor of LDL,
bad cholesterol. A total
cholesterol score of under
considered healthy in most
People who score in the "h
range have an increased ris
developing heart disease
compared to those who sc
below 200.

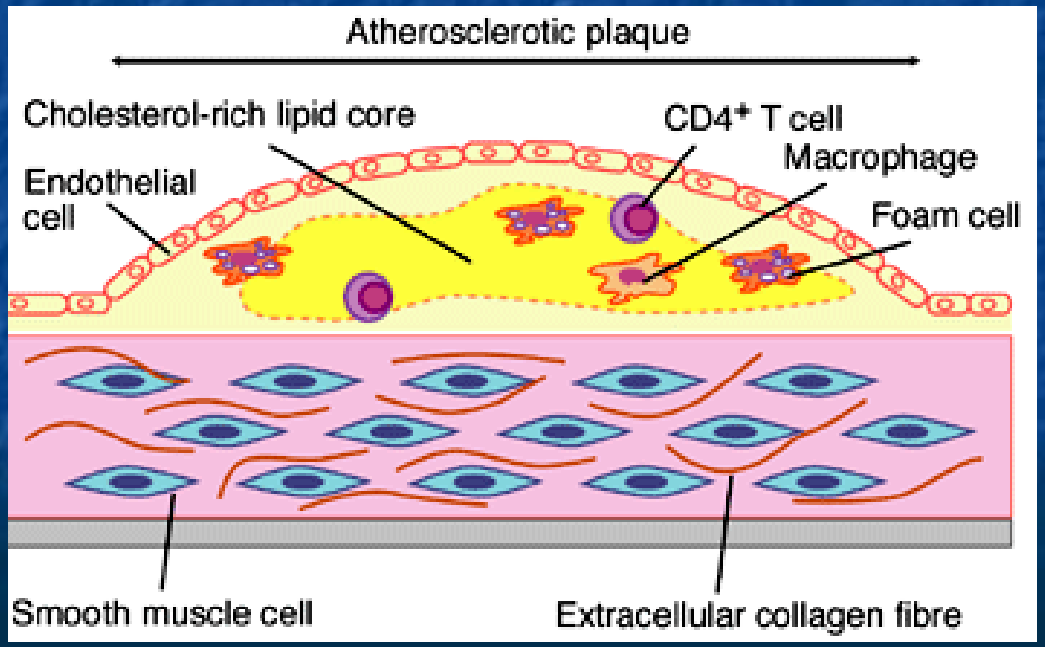
# Cholesterol Ratio


- To calculate your cholesterol ratio, divide your total cholesterol by your HDL cholesterol. For example, a to score of 200 divided by an HD score of 50 equals a cholester ratio of 4 to 1. Doctors recommend maintaining a rat of 4 to 1 or lower. The smalle ratio, the better. While this fi is useful in estimating heart disease risk, it's not as import in guiding treatment. Doctors look at total cholesterol, HDL cholesterol, and LDL choleste to determine treatment.

# Atherosclerosis

**Atherosclerosis** is a process of progressive lipid accumulation with the formation of multiple plaques within the arteries.

- Atherosclerotic plaque contains
  - lipids
  - inflammatory cells
  - smooth muscle cells,
  - connective tissue
  - thrombi,
  - $Ca^{2+}$ deposits.



# Atherosclerosis

- **Arteriosclerosis** - any hardening (and loss of elasticity) of medium or large arteries
- **Arteriolosclerosis** - affectiong of the arterioles (small arteries)
- **Atherosclerosis** is a hardening of an artery specifically due to an **atheromatous plaque** (in Greek, "athero" means "porridge").
  - Atherosclerosis is a form of arteriosclerosis.

Atherosclerosis starts with damage or injury to the inner layer of an artery. The damage may be caused by:

- High blood pressure
- High cholesterol
- An irritant, such as nicotine
- Certain diseases, such as diabetes

us anatomic, physiological & behavioral risk factors for atherosclerosis are own. These can be divided into various categories:, modifiable and non-odifiable.

## Modifiable

**aving diabetes or Impaired glucose tolerance (IGT)**

**yslipoproteinemia (unhealthy patterns of serum proteins rrying fats & cholesterol):**

- **High serum concentration of low-density lipoprotein (LDL, "bad if elevated concentrations and small"), and / or very low density lipoprotein (VLDL) par i.e., "lipoprotein subclass analysis"**
- **Low serum concentration of functioning high density lipoprotein (HDL "prot if large and high enough" particles), i.e., "lipoprotein subclass analysis"**
- **An LDL:HDL ratio greater than 3:1**

**obacco smoking, increases risk by 200% after several pack ars**

**aving high blood pressure, on its own increasing risk by 60%**

**evated serum C-reactive protein concentrations**

# *Physiologic factors that increase ris*

## 2. Non modifiable

- Advanced age
- Male sex
- Having close relatives who have had some complication of atherosclerosis (eg. coronary heart disease or stroke)
- Genetic abnormalities, e.g. familial hypercholesterolemia

## 3. Lesser or uncertain

- **Being obese (in particular ce obesity,**
- **A sedentary lifestyle**
- **Postmenopausal estrogen def**
- **High carbohydrate intake**
- **Elevated serum levels of triglycerides**
- **Elevated serum levels of uric (also responsible for gout)**
- **Elevated serum fibrinogen concentrations**
- **Elevated serum lipoprotein concentrations**
- **Stress or symptoms of clinica depression**
- **Hyperthyroidism**
- **Elevated serum insulin levels**
- **Short sleep duration**
- ***Chlamydia pneumoniae* infec**

Table 1.Conditions That Accelerate the Progression of Atherosclerosis and the Mechanisms Responsible.

| Condition | Mechanism |
|---|---|
| Male gender (and females after menopause) | Estrogen increases cholesterol removal by the liver, and the progression of atherosclerosis is less rapid in premenopausal women that in men |
| Family history of ischemic heart disease, stroke | Probably multiple genetic mechanisms. |
| Primary hyperlipidemia | Inherited disorders causing lipoprotein lipase deficiency (type I), defective LDL receptors (type IIa), abnormal apoprotein E (type III), deficiency of apoprotein C (type V), or unknown cause (types IIb and IV). |
| Secondary hyperlipidemia | Increased circulating triglycerides produced by diuretics, b-adrenergic blocking drugs, excess alcohol intake. |
| Cigarette smoking | Probably carbon monoxide-induced hypoxic injury to endothelial cells. |
| Hypertension | Increased shear stress, with damage to endothelium. |
| Diabetes mellitus (types 1 and 2) | Decreased hepatic removal of LDL from the circulation; increased glycosylation of collagen, which increases LDL binding to blood vessel walls. |
| Obesity, particularly abdominal obesity | Unsettled, but obesity is associated with type 2 diabetes, hypertriglyceridemia, hypercholesterolemia, and hypertension, all of which are risk factors in their own right. |
| Nephrotic syndrome | Increased hepatic production of lipids and lipoprotein (a). |
| Hypothyroidism | Decreased formation of LDL receptors in the liver. |
| Elevated plasma homocysteine | Unsettled. Probably increased homocysteine provides more $H_2O_2$ and other reactive oxygen molecules that foster formation of oxidized LDL. |

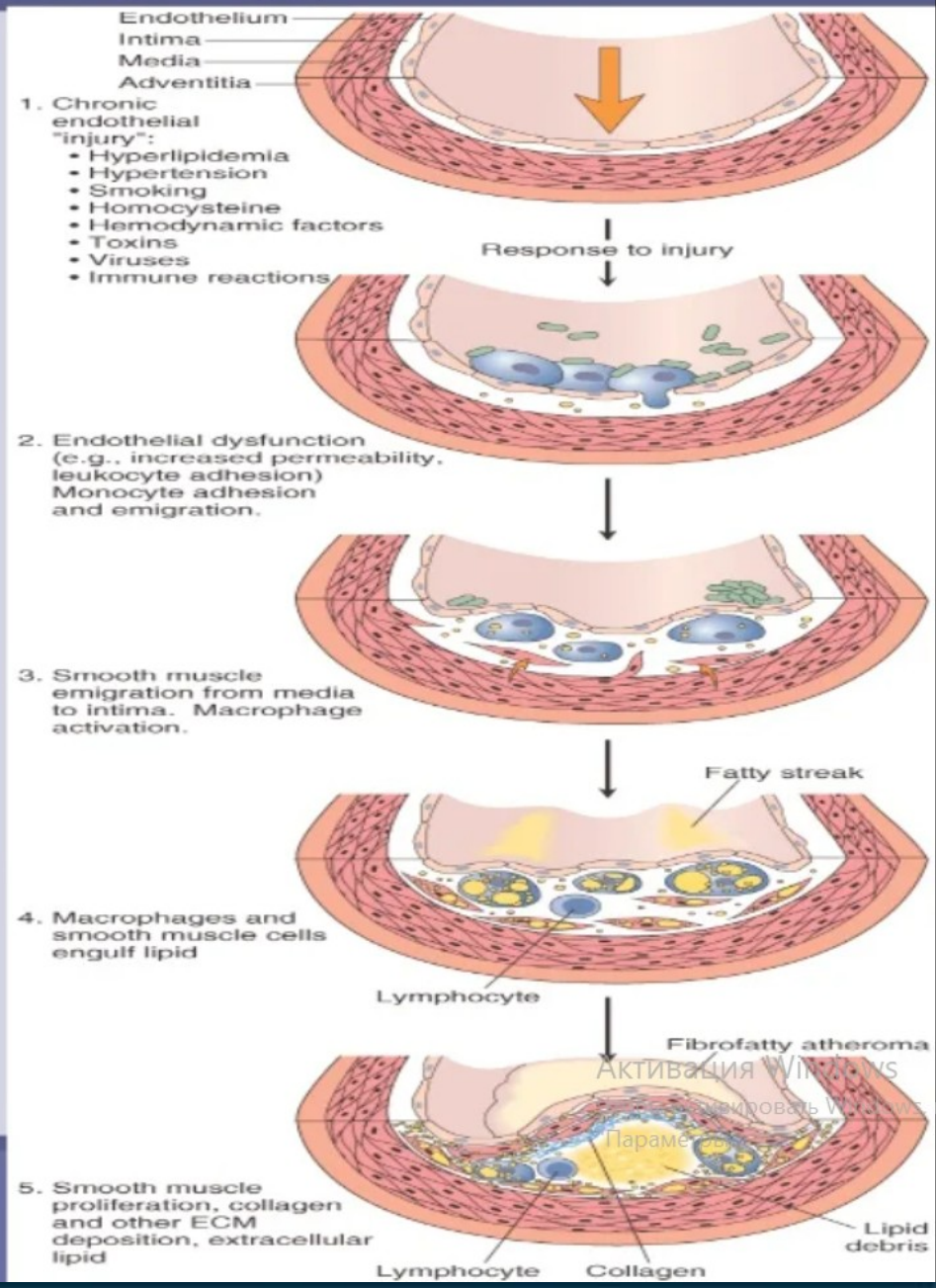
# ***Pathophysiology***

- Atherosclerosis develops as a chronic inflammatory response of the arterial wall to endothelial injury.
- Lesion progression occurs through interactions of modified lipoproteins, monocyte-derived macrophages, T-lymphocytes, and the normal cellular constituent of the arterial wall.
- The contemporary view of atherosclerosis is expressed by the ***response-to-injury hypothesis***.

# *Response-to-injury hypothesis*

- The following are the steps involved in the hypothesis:

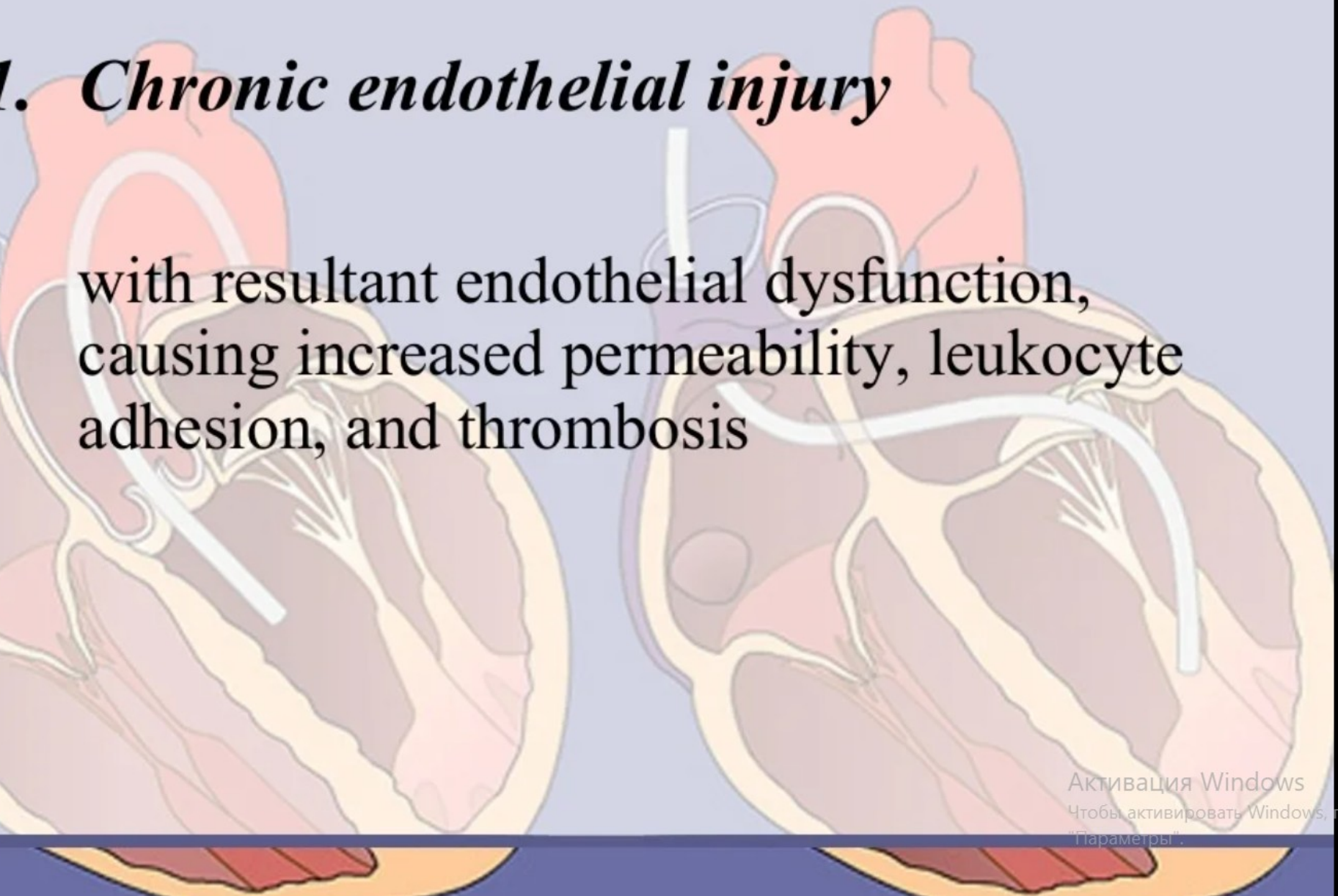
1. ***Chronic endothelial injury***
2. ***Accumulation of lipoproteins***
3. ***Monocyte adhesion to the endothelium***
4. ***SMC proliferations and ECM production***
5. ***factor release***
6. ***platelet adhesion***



# *Response-to-injury hypothesis*

## *1. Chronic endothelial injury*

with resultant endothelial dysfunction, causing increased permeability, leukocyte adhesion, and thrombosis



# *Response-to-injury hypothesis*

## *2. Accumulation of lipoproteins*

- (mainly LDL and its oxidized forms) in t
  vessel wall. Low-density lipoprotein mol
  (LDL) becoming oxidized (ldl-ox) by fre
  radicals, particularly oxygen free (ROS).
  oxidized LDL comes in contact with an a
  wall, a series of reactions occur to repair
  damage to the artery wall caused by oxid
  LDL. Cholesterol can move in the bloods
  only by being transported by lipoproteins

# sponse-to-injury hypothesis

## ocyte adhesion to the endothelium

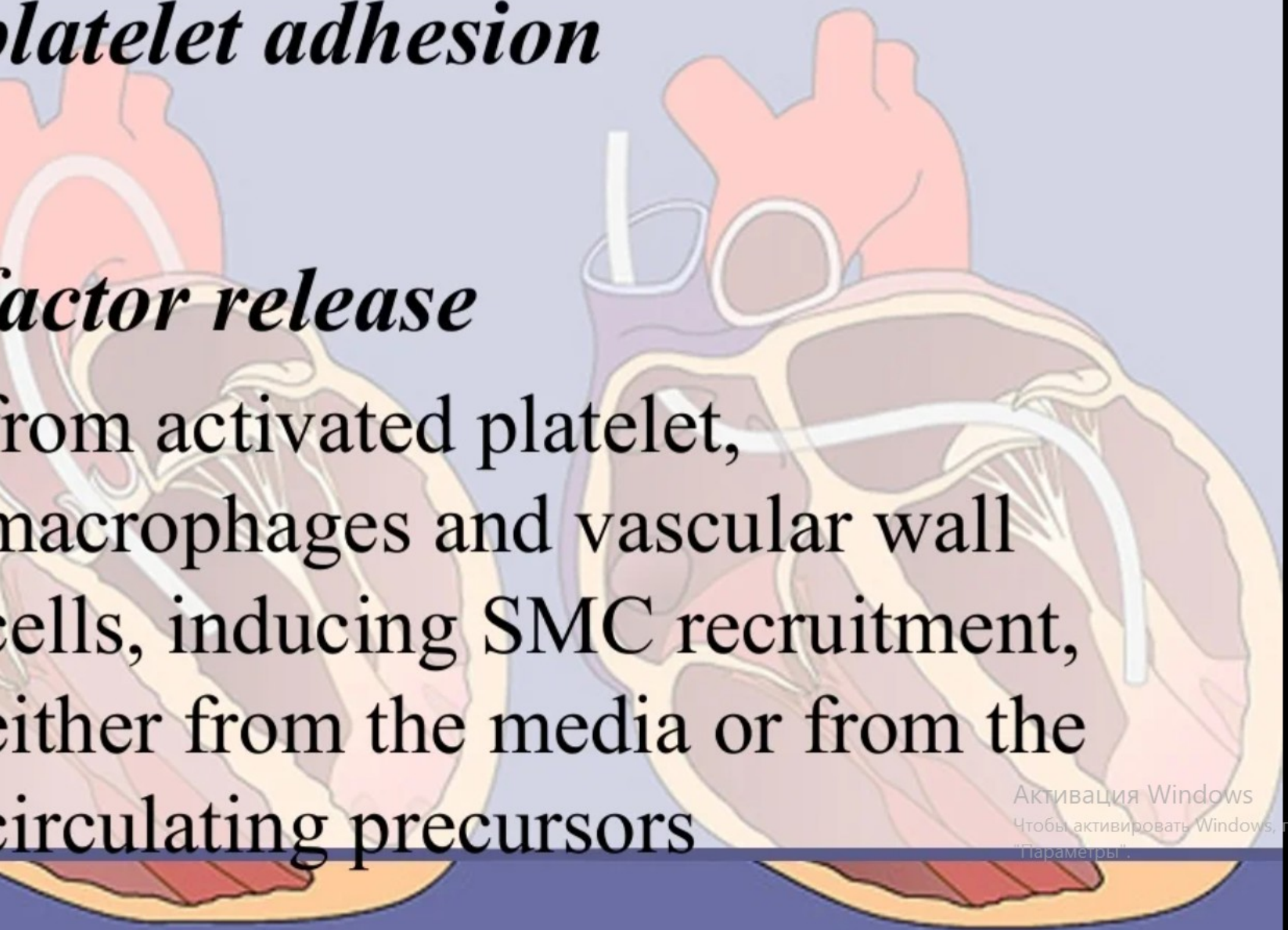
wed by migration into the intima and
formation into macrophages and foam cells. The
's immune system responds to the damage to the
y wall caused by oxidized LDL by sending
alized white blood cells (macrophages and T-
hocytes) to absorb the oxidized-LDL forming
alized foam cells. Unfortunately, these white
d cells are not able to process the oxidized-LDL,
ultimately grow then rupture, depositing a greater
unt of oxidized cholesterol into the artery wall.
triggers more white blood cells, continuing the
e.

# esponse-to-injury hypothesis

## latelet adhesion

## actor release

rom activated platelet,
macrophages and vascular wall
ells, inducing SMC recruitment,
ither from the media or from the
irculating precursors



# *Response-to-injury hypothesis*

## *6. SMC proliferations and ECM production.*

- Eventually, the artery becomes inflamed. The cholesterol plaque causes the smooth muscle cells to enlarge and form a hard cover over the affected area. This hard cover is what causes a narrowing of the artery, reduces the blood flow and increases blood pressure

# *Response-to-injury hypothesis*

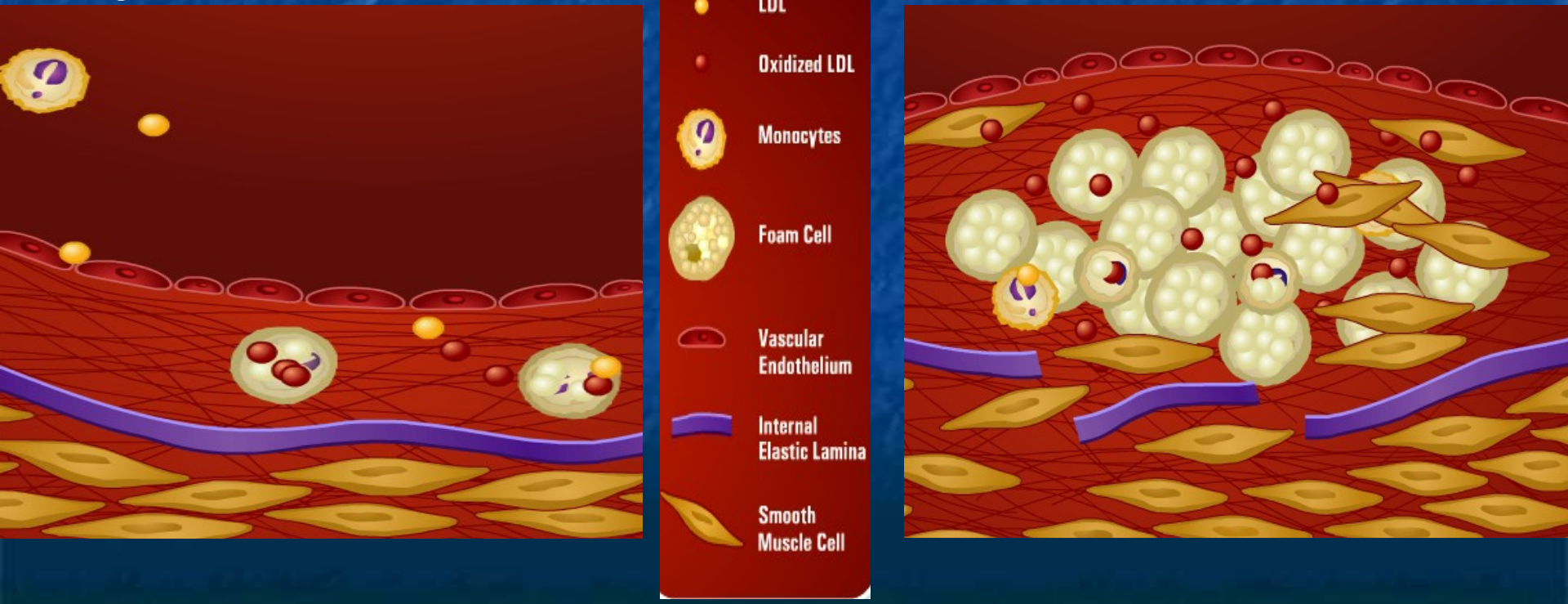
## *7. Lipid accumulation*

- both extracellularly and within cells (macrophages and SMC's). accumulation if lipid-containing macrophages in the intima gives rise to "fatty streaks", with further evolution, a fibrofatty atheroma consisting o proliferated SMC, foam cells, extracellular lipid, and ECM is formed.

# Atherosclerosis pathogenesis

**The lipid hypothesis**

plasma LDL penetration into the arterial wall → lipid accumulation in smooth muscle cells and in macrophages (foam cells)→ smooth muscle cell hyperplasia and migration into the subintimal and intimal region



# Atherosclerosis pathogenesis

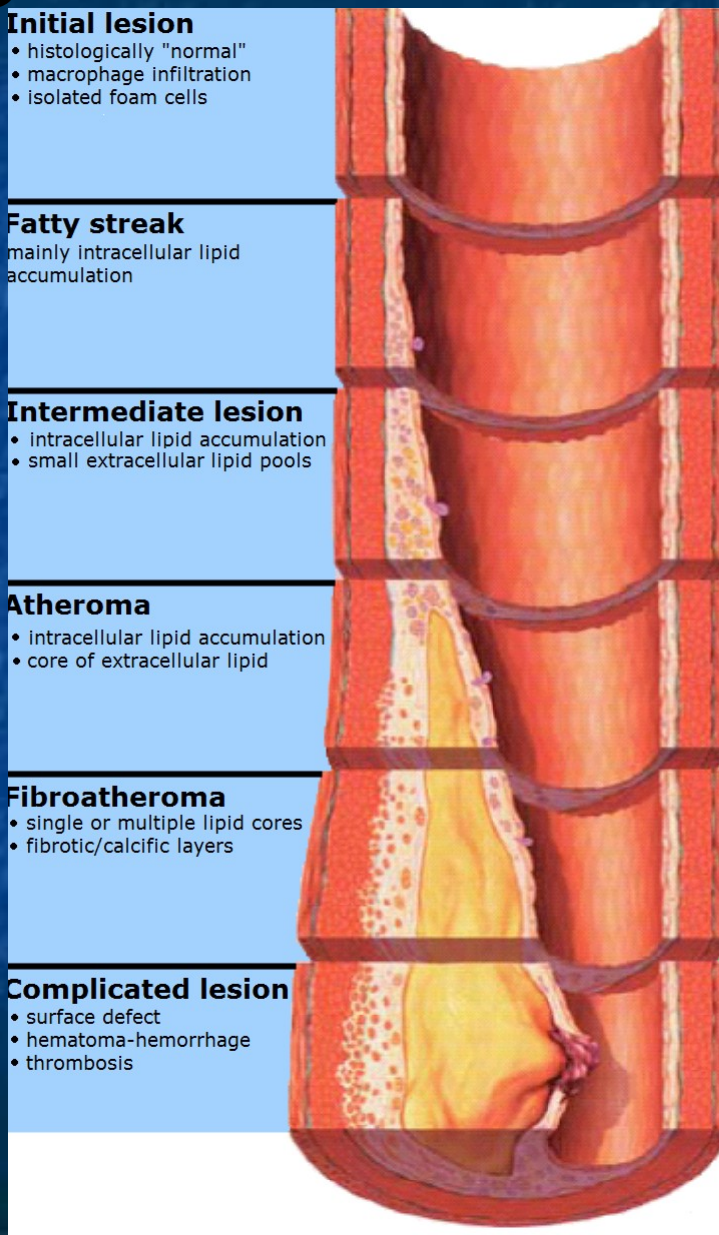
**The chronic endothelial injury hypothesis**

Endothelial injury

- loss of endothelium,
- adhesion of platelets to subendothelium,
- aggregation of platelets,
- chemotaxis of monocytes and T-cell lymphocytes
- release of growth factors
  - induce migration and replication
  - their synthesis of connective tissue and proteoglycans

# Atherosclerosis pathogenesis

- The atherosclerotic plaque may produce a severe stenosis or may progress to total arterial occlusion.
- With time, the plaque becomes calcified.
- Some plaques are stable
- Others may undergo spontaneous fissure or rupture (unstable or vulnerable)
- The ruptured plaque stimulates thrombosis.



# Atherosclerosis: positive risk factors

**Non modifiable**

- Age – middle to late.
- Sex – Males, complications
- Genetic – Familiar Hypercholesterolemia
- Family history.

**Potentially Modifiable**

- Hyperlipidemia – HDL/LDL ratio.
- Hypertension.
- Smoking.
- Diabetes
- Life style, diet, exercise

# Atherosclerosis risk factors

- Negative risk factors
  - high levels of circulating high density lipoproteins
  - moderate alcohol consumption
  - cardiovascular fitness

# Symptoms

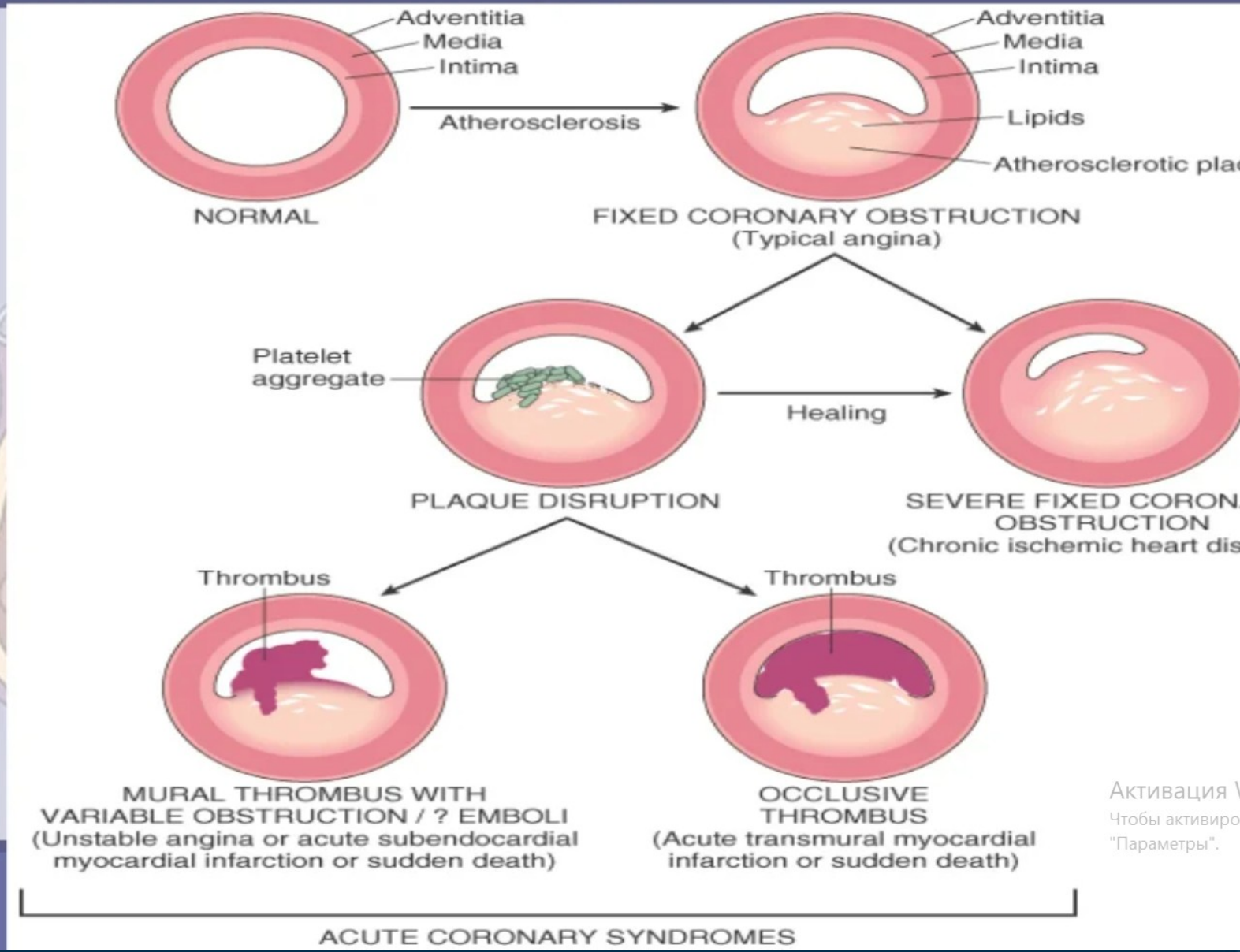
- Atherosclerosis develops gradually, typically begins in early adolescence, and is usually found in most major arteries. There are usually no atherosclerosis symptoms until an artery is so narrowed or clogged that it can't supply adequate blood to your organs and tissues. Sometimes a blood clot completely obstructs blood flow, or even breaks apart and causes blood clots that can trigger a heart attack or stroke.

# Symptoms

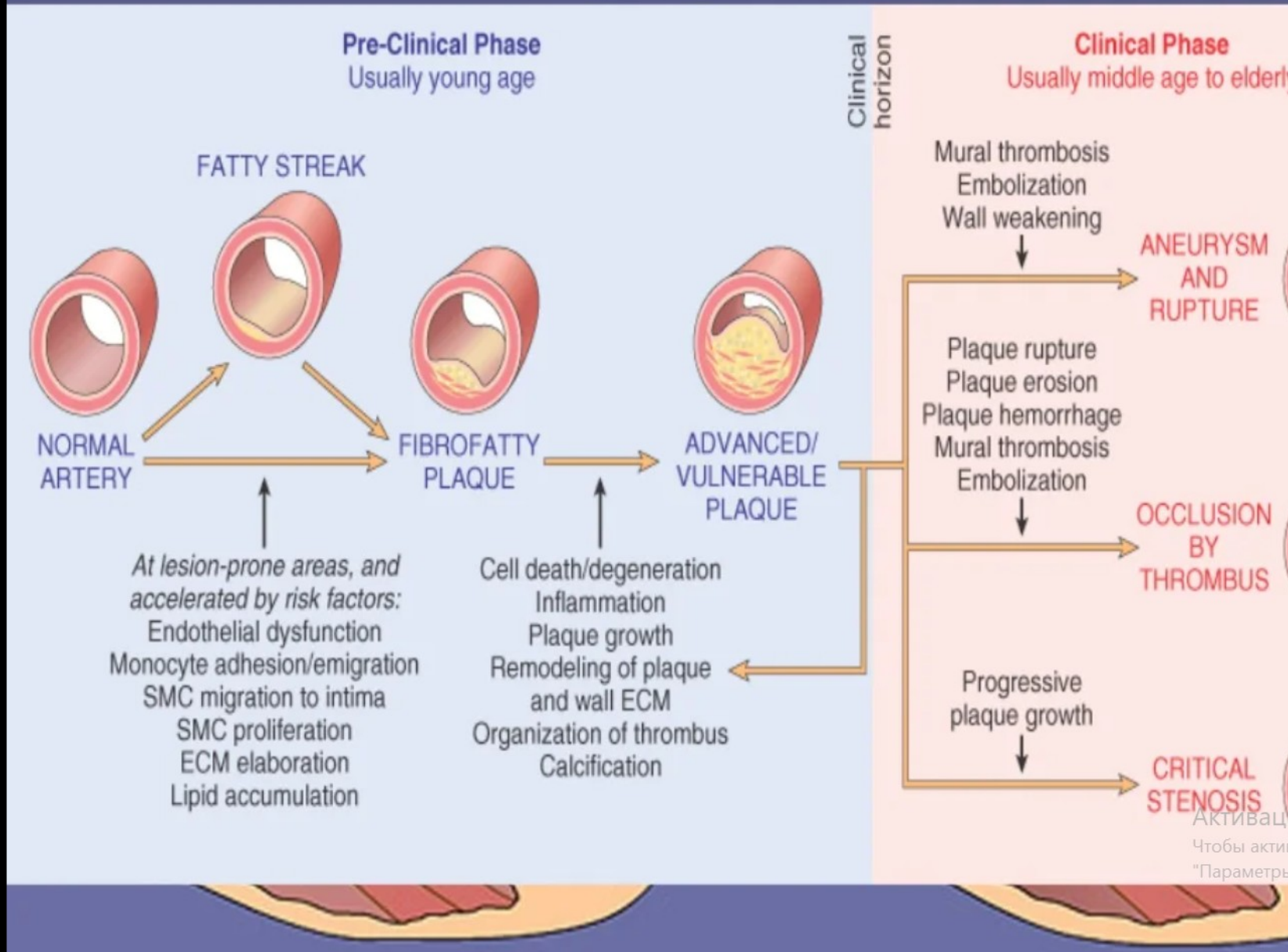
Atherosclerosis symptoms depend on which arteries are affected. For example:

- **Atherosclerosis in heart arteries,** have symptoms sim to those of a heart attack, such as chest pain (angina).
- **Atherosclerosis in the arteries leading to brain,** have symptoms such as sudden numbness or weakness in you arms or legs, difficulty speaking or slurred speech, or drooping muscles in your face.
- **Atherosclerosis in the arteries in arms and legs,** prod decreased blood flow is called peripheral artery occlusiv disease (PAOD).have symptoms such as leg pain when walking
- Sometimes atherosclerosis causes erectile dysfunction i men.

# Symptoms



# Symptoms


Pre-Clinical Phase
Usually young age
Clinical horizon
Clinical Phase
Usually middle age to elderly
FATTY STREAK
NORMAL ARTERY
FIBROFATTY PLAQUE
ADVANCED/ VULNERABLE PLAQUE
At lesion-prone areas, and accelerated by risk factors:
Endothelial dysfunction
Monocyte adhesion/emigration
SMC migration to intima
SMC proliferation
ECM elaboration
Lipid accumulation
Cell death/degeneration
Inflammation
Plaque growth
Remodeling of plaque and wall ECM
Organization of thrombus
Calcification
Mural thrombosis
Embolization
Wall weakening
ANEURYSM AND RUPTURE
Plaque rupture
Plaque erosion
Plaque hemorrhage
Mural thrombosis
Embolization
OCCLUSION BY THROMBUS
Progressive plaque growth
CRITICAL STENOSIS

# Complications

The complications of atherosclerosis depend on the location of the blocked arteries. For example:

- **Coronary artery disease.** When atherosclerosis narrows the arteries close to your heart, you may develop coronary artery disease, which can cause chest pain (angina) or a heart attack.
- **Carotid artery disease.** When atherosclerosis narrows the arteries close to your brain, you may develop carotid artery disease, which can cause a transient ischemic attack (TIA) or stroke.

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- **Carotid artery disease.** When atheroscler
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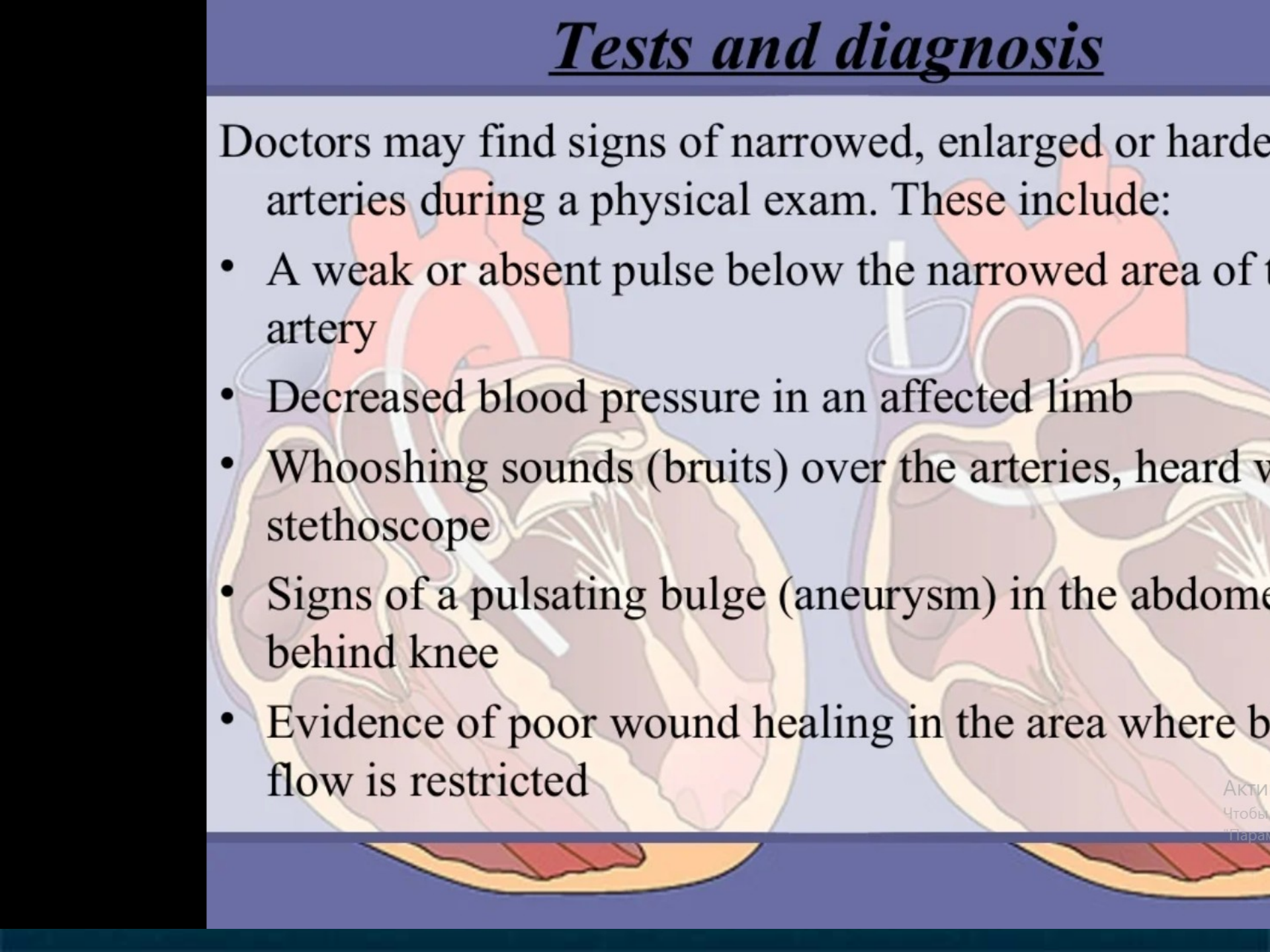
develop carotid artery disease, which can c
transient ischemic attack (TIA) or stroke.

# Tests and diagnosis

Doctors may find signs of narrowed, enlarged or harde
arteries during a physical exam. These include:

- A weak or absent pulse below the narrowed area of t
artery
- Decreased blood pressure in an affected limb
- Whooshing sounds (bruits) over the arteries, heard w
stethoscope
- Signs of a pulsating bulge (aneurysm) in the abdome
behind knee
- Evidence of poor wound healing in the area where b
flow is restricted



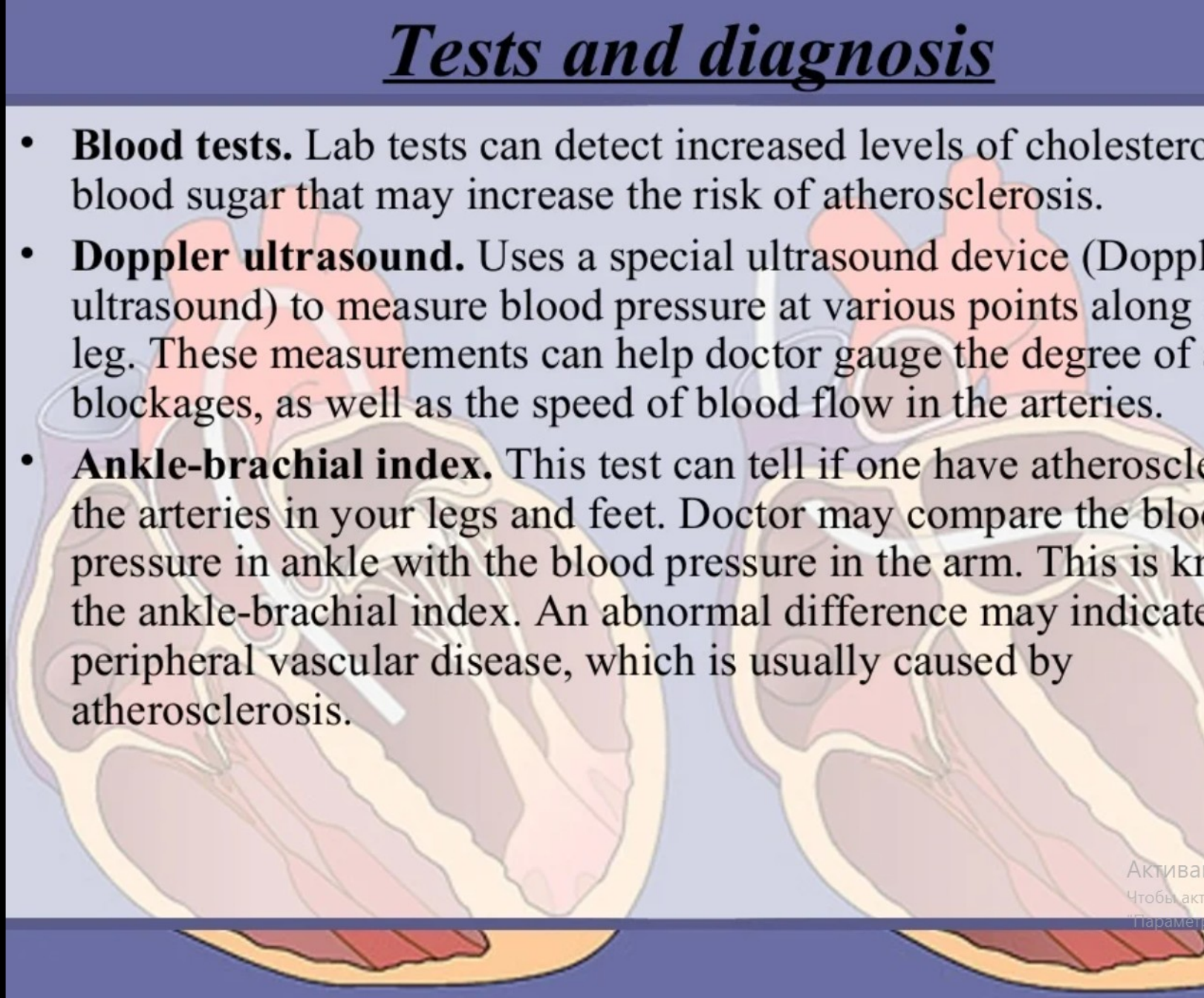
# Tests and diagnosis

Depending on the results of the physical ex
may suggest one or more diagnostic tests

- **Blood tests.**
- **Doppler ultrasound**
- **Ankle-brachial index.**
- **Other imaging tests.**
- **Angiogram.**
- **Electrocardiogram (ECG).**

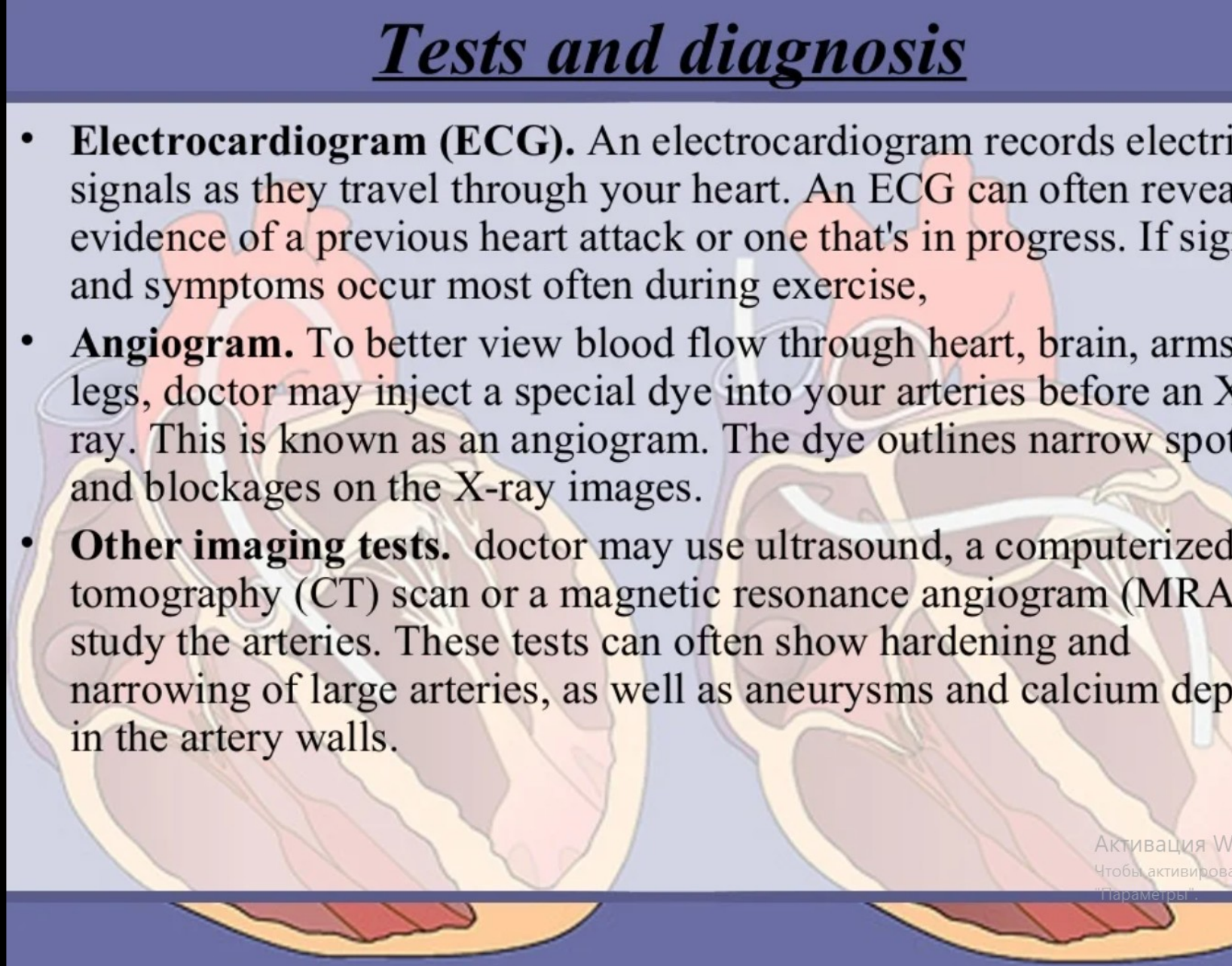
# Tests and diagnosis

- **Blood tests.** Lab tests can detect increased levels of cholesterol blood sugar that may increase the risk of atherosclerosis.
- **Doppler ultrasound.** Uses a special ultrasound device (Doppler ultrasound) to measure blood pressure at various points along leg. These measurements can help doctor gauge the degree of blockages, as well as the speed of blood flow in the arteries.
- **Ankle-brachial index.** This test can tell if one have atherosclerosis the arteries in your legs and feet. Doctor may compare the blood pressure in ankle with the blood pressure in the arm. This is known the ankle-brachial index. An abnormal difference may indicate peripheral vascular disease, which is usually caused by atherosclerosis.



# Tests and diagnosis

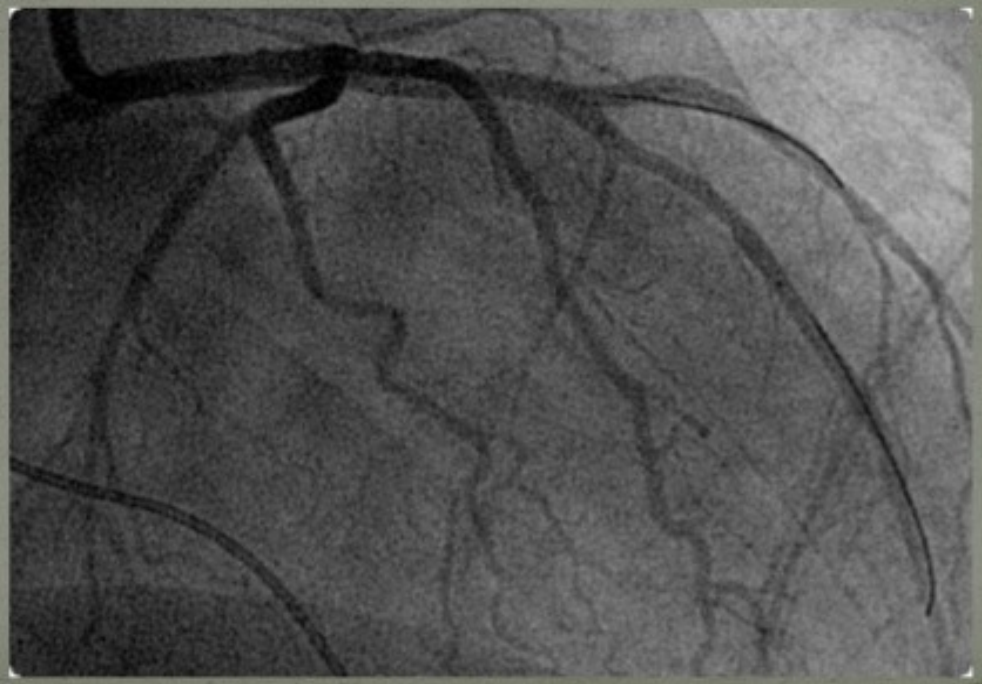
- **Electrocardiogram (ECG).** An electrocardiogram records electri signals as they travel through your heart. An ECG can often revea evidence of a previous heart attack or one that's in progress. If sig and symptoms occur most often during exercise,
- **Angiogram.** To better view blood flow through heart, brain, arms legs, doctor may inject a special dye into your arteries before an X ray. This is known as an angiogram. The dye outlines narrow spot and blockages on the X-ray images.
- **Other imaging tests.** doctor may use ultrasound, a computerized tomography (CT) scan or a magnetic resonance angiogram (MRA study the arteries. These tests can often show hardening and narrowing of large arteries, as well as aneurysms and calcium dep in the artery walls.



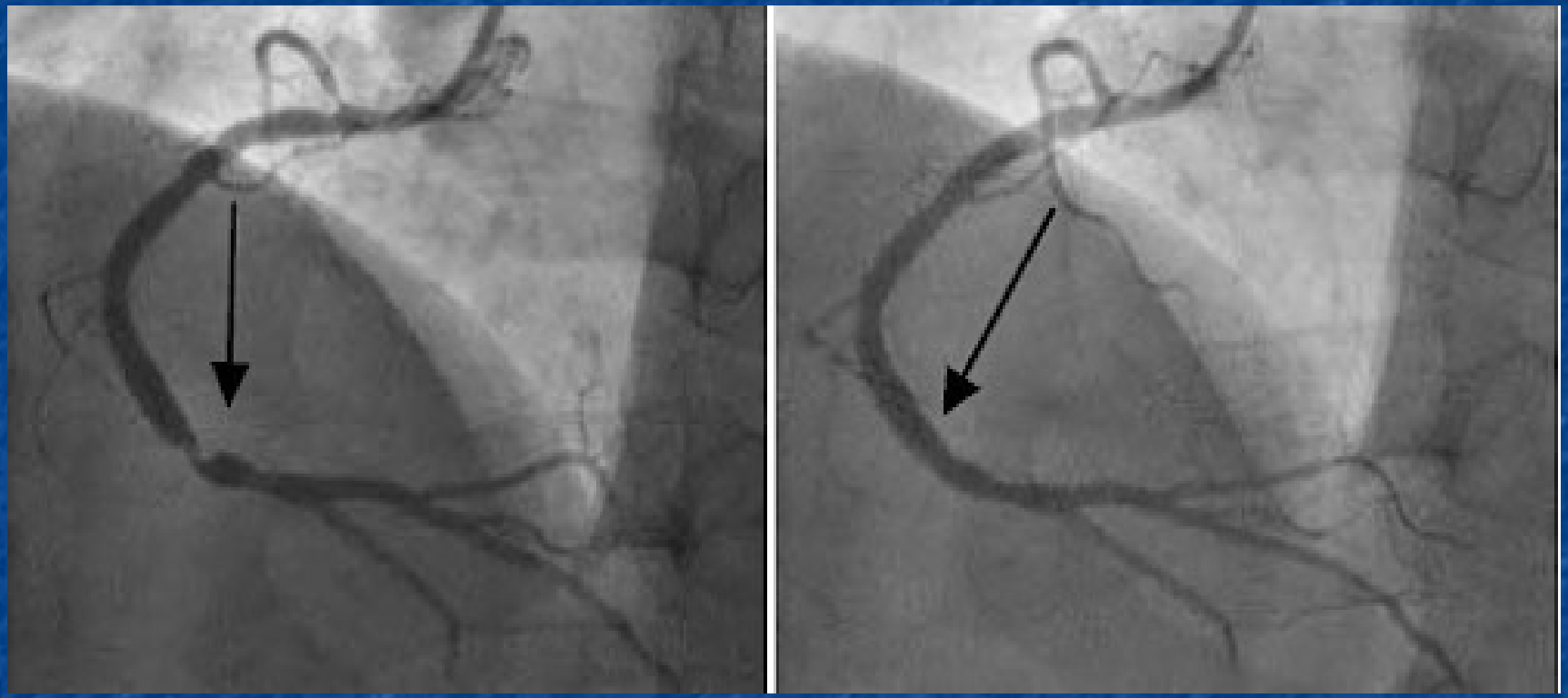
# A coronary angiography to see how well blood flows through the arteries

## Diagnosis

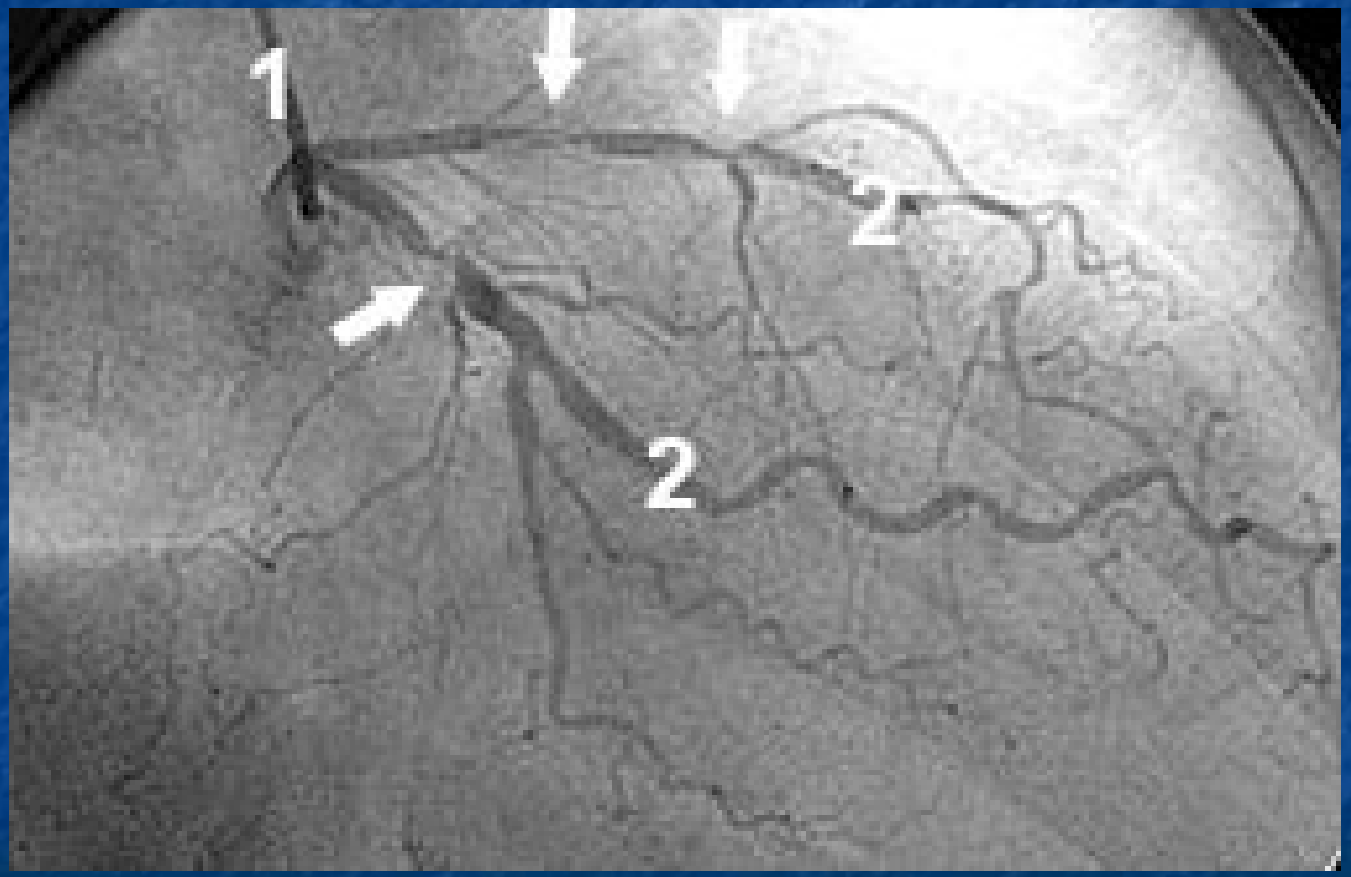
- **Coronary angiogram. Its gold standard of** diagnostics of Coronary Heart Disease.



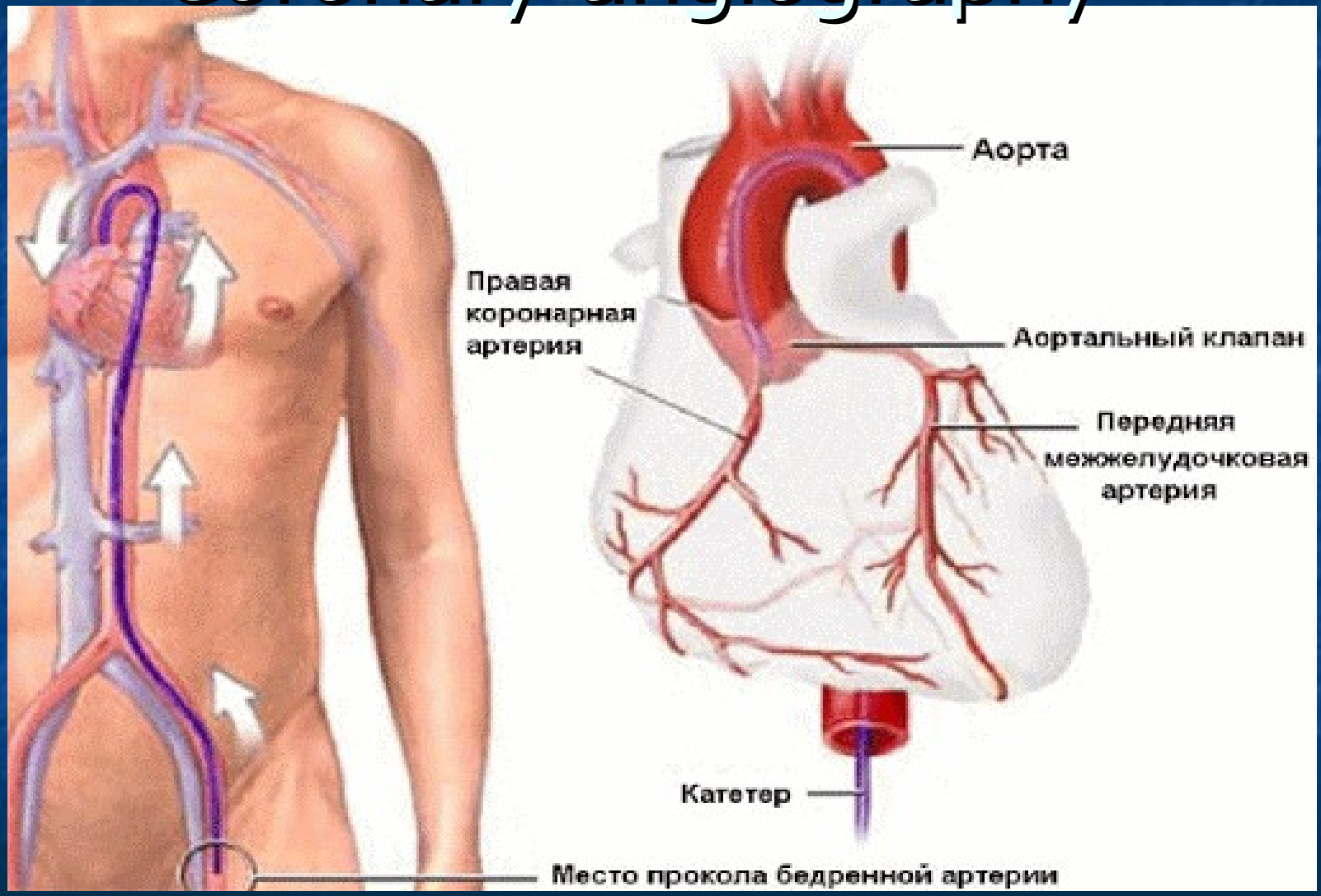
# coronary angiography



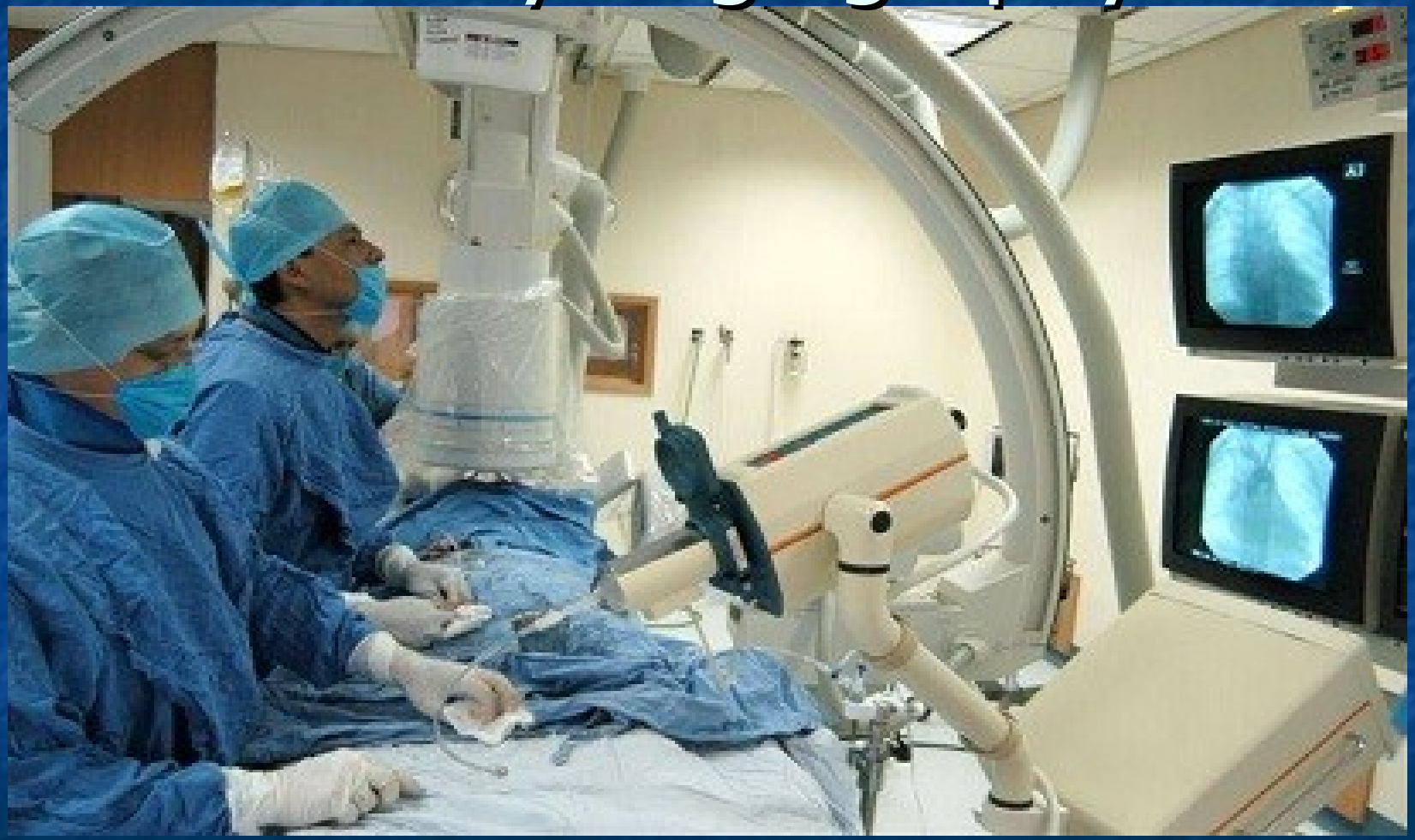
# Coronary angiography



# Coronary angiography

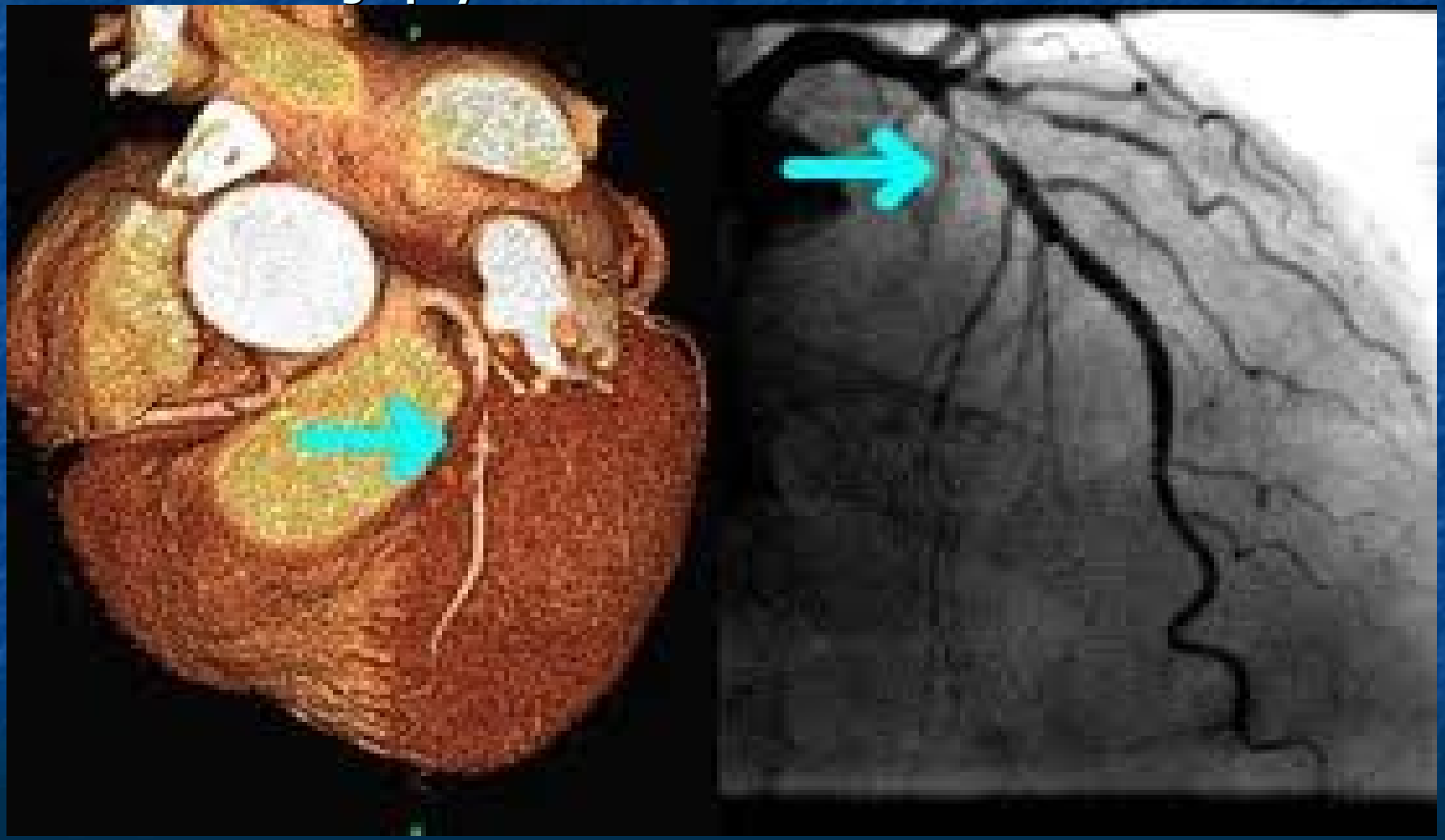


# Coronary angiography



Multispiral computed tomography

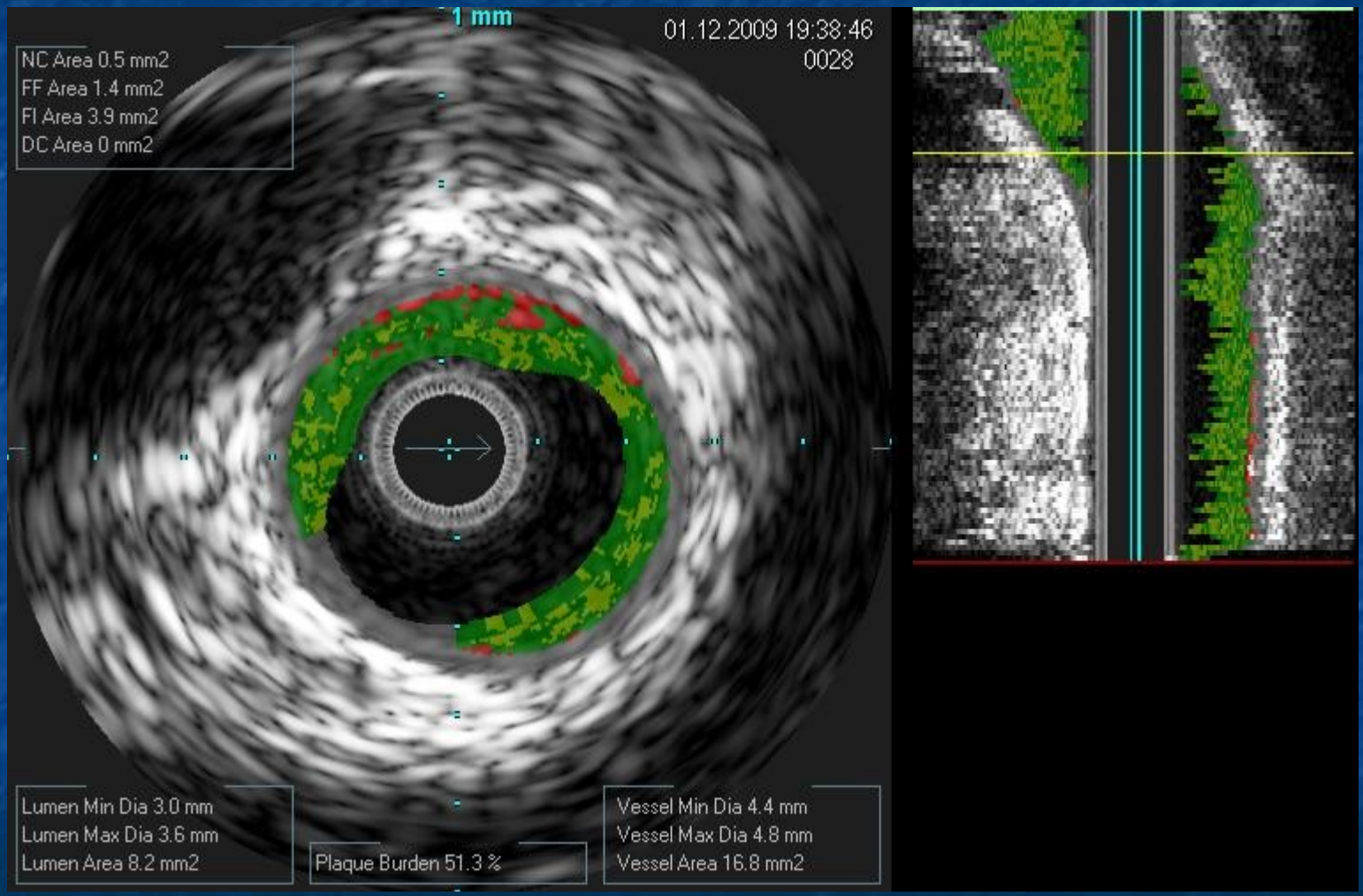
Coronary angiography



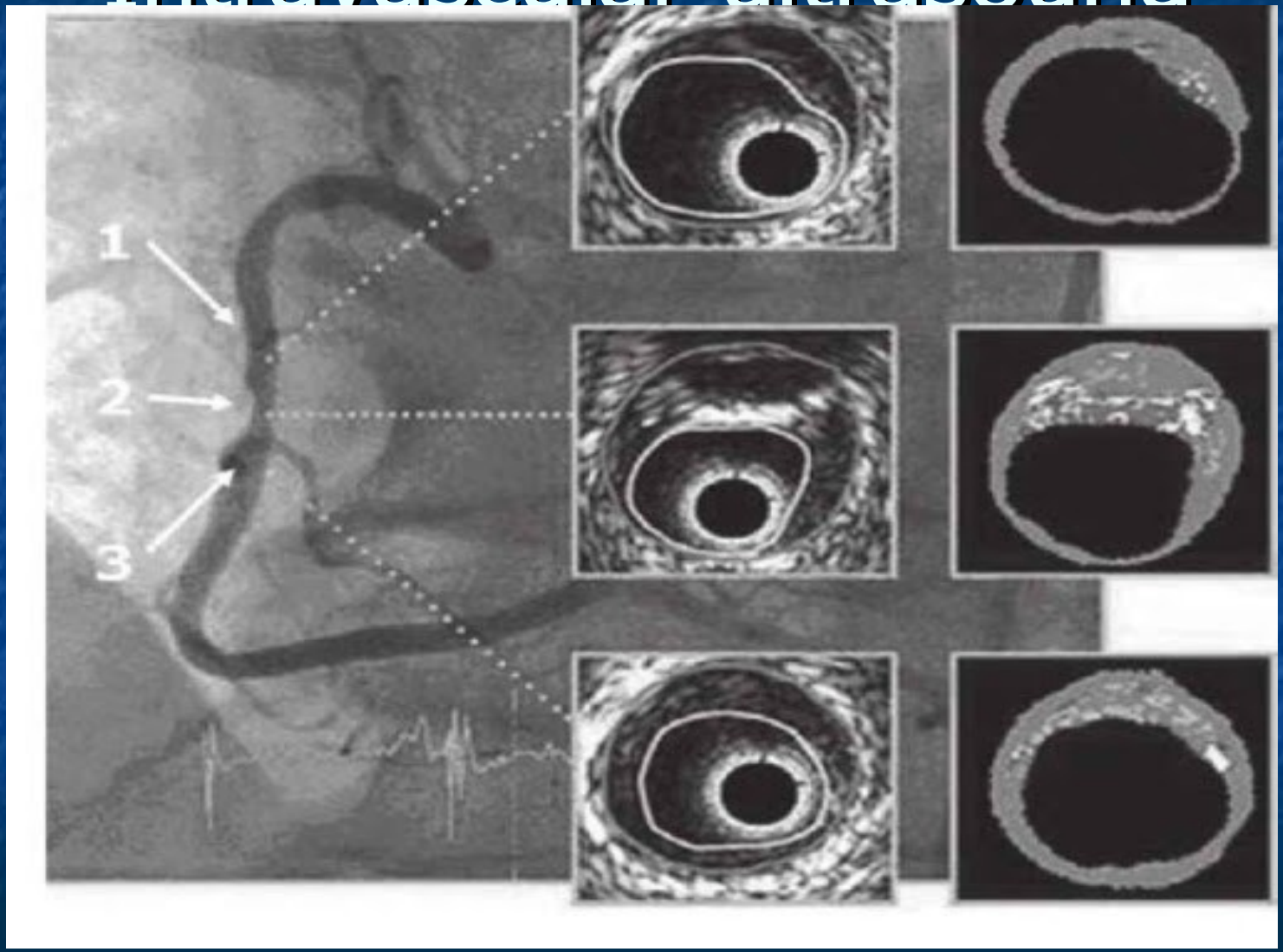
# Intravascular ultrasound

- **IVUS** is performed during cardiac catheterisation using miniature ultrasound probes mounted on the tip of a coronary catheter. The IVUS probe emits high ultrasound frequencies, typically centred at 20–50 MHz. The ultrasound signal reflected from arterial wall structures is used to generate a grey scale image.

# Intravascular ultrasound



# Intravascular ultrasound



- Ultrasound is strongly reflected at the interface of different tissue structures.
- Using IVUS, some morphologic features of atherosclerotic coronary plaques can be readily recognised. The reliability of ultrasound imaging in predicting the composition of atherosclerotic plaque components has been demonstrated in comparative studies of histology. Lipid laden lesions appear as hypoechoic, “soft” areas and fibrous or calcified tissues are recognised as bright echoes. In lipid laden lesions with prominent overlying fibrous “caps”, a more reflective structure separating the soft echoes from the lumen is identified on the corresponding images. The integration of information from adjacent images in a coronary segment allows three dimensional reconstruction and the calculation of atheroma volume

# Treatments and drugs

Lifestyle changes, such as eating a healthy diet and exercisi
are often the first line of defense in treating atherosclerosi
But sometimes, medication or surgical procedures may be
recommended as well.

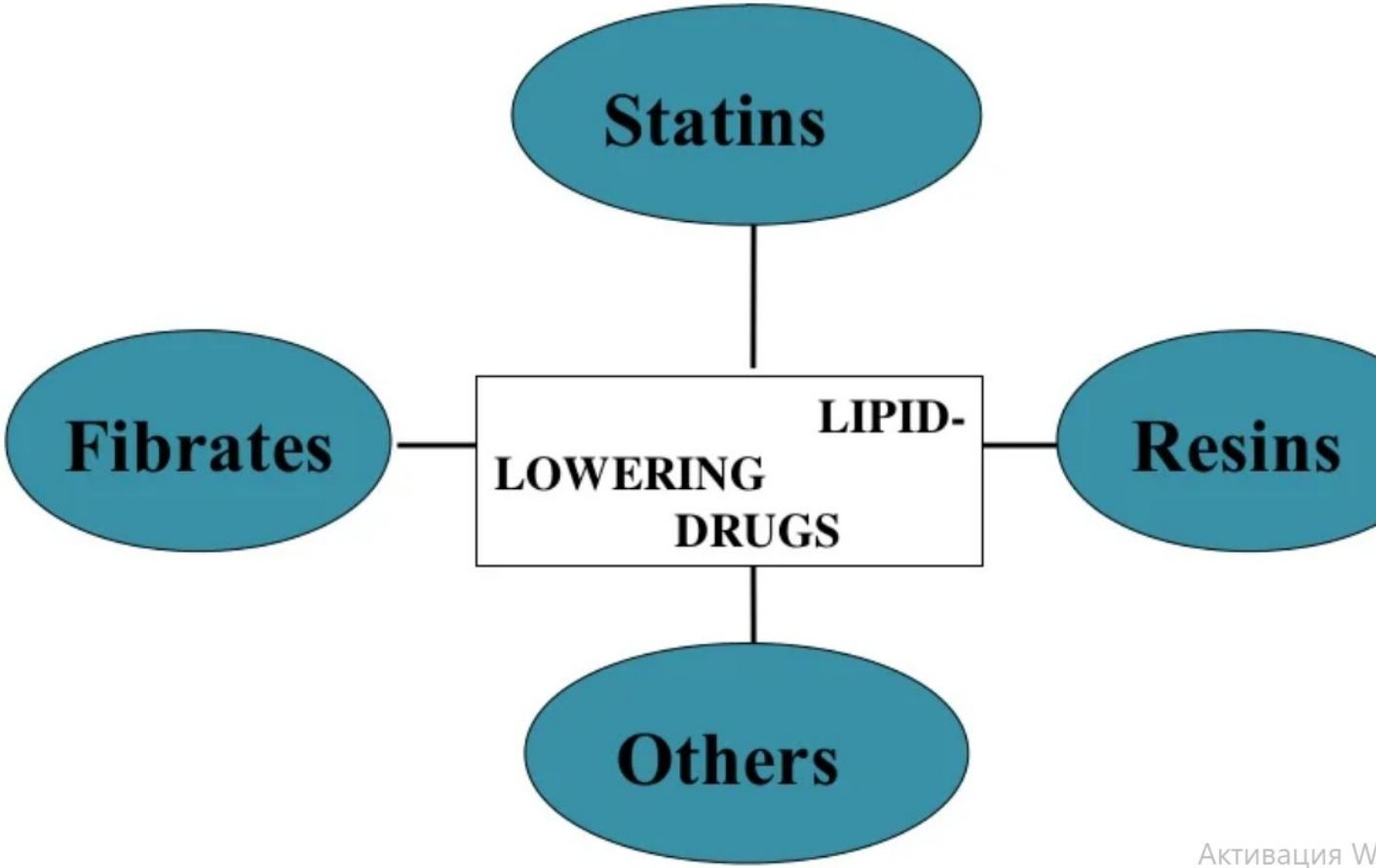
Various drugs can slow — or sometimes even reverse — th
effects of atherosclerosis. Here are some common choice

- **Cholesterol medications.** Aggressively lowering low-
density lipoprotein (LDL) cholesterol, the "bad" cholester
can slow, stop or even reverse the buildup of fatty deposi
in arteries. Boosting your high-density lipoprotein (HDL)
cholesterol, the "good" cholesterol, may help, too.
cholesterol medications includes drugs known as statins a
fibrates.
- .

# Lipid-lowering drugs

- Several drugs are used to decrease plasm LDL-CHO
- Drug therapy to lower plasma lipids is onl one approach to treatment

- And is used in addition to ***dietar management and correction of othe modifiable cardiovascular risk factors***



# Statins:

## ***MOA***

- ✓ HMG-CoA (3-hydroxy-3-methylglutaryl-coenzyme A) reductase inhibitors.

The reductase catalyses the conversion of HMG-CoA to mevalonic acid

- ✓ ***Simvastatin + pravastatin + atorvastatin***
  - ❖ decrease hepatic CHO synthesis
- up regulates LDL receptor synthesis, increasing and LDL clearance from plasma into liver ce
  - **Atorvastatin and rosuvastatin** are long-lasting inhibitors.

# Promising pharmacodynamic actions of statins:

- *Improved endothelial function*
- *Reduced vascular inflammation and platelet aggregability*
- *Antithrombotic action*
- *Stabilisation of atherosclerotic plaques*
- *Increased neovascularisation of ischaemic tissue*
- *Enhanced fibrinolysis*
- *Immune suppression*
- *Osteoclast apoptosis and increased synthetic activity in osteoblasts*

# Pharmacokinetics of Statints

- *well absorbed when given orally*
- *extracted by the liver (target tissue), undergo extensive presystemic biotransformation*

*Simvastatin is an inactive pro-drug*

# Clinical uses:

- ***Secondary prevention of myocardial infarction stroke in patients who have symptom atherosclerotic disease*** *(angina, transient isch attacks)* ***following acute myocardial infarctio stroke***
- ***Primary prevention of arterial disease in pat who are at high risk because of elevated s CHO concentration, especially it there are risk factors for atherosclerosis***
- ***Atorvastatin lowers serum CHO in patients homozygous familiar hypercholesterolemia***

# Adverse effects:

- **mild gastrointestinal disturbances**

- Raised concentrations of liver enzymes in plasma
- **Severe myositis (rhabdomyolysis)**
- **Angio-oedema (rare)**

# 2- Fibrates:

- ***stimulate the beta-oxidative degradatio fatty acids***
- - liberate free fatty acids for storage in fat metabolism in striated muscle
- increase the activity of lipoprotein lipase,
- hence increasing hydrolysis of triglyceri chylomicrons and VLDL particles

- reduce hepatic VLDL production and inc hepatic LDL uptake

- **Other effects:**
- improve glucose tolerance
- inhibit vascular smooth muscle inflammation

- ✓ ***Fenofibrate***
- ✓ ***Clofibrate***
- ✓ ***Gemfibrozil***
- ✓ ***Ciprofibrate***

# Adverse effects:

In patients with renal impairment

- ✓ myositis (rhabdomyolysis)
- ✓ myoglobulinuria, acute renal failure

- Fibrates should be avoided in such patient
- Mild GIT symptoms

# Clinical uses:

- Mixed dyslipidaemia (i.e. raised ser triglyceride as well as cholesterol)
- In patients with low high-density lipopro and high risk of atheromatous disease (o type 2 diabetic patients
- Combined with other lipid-lowering drug patients with severe treatment-resist dyslipidaemia

# Bile acid binding resins: (Colestyramin colestipol)

- sequester bile acids in the GIT → prevent the reabsorption and enterohepatic recirculation
- *The result is:*

decreased absorption of exogenous CHO and increas metabolism of endogenous CHO into bile acid acids

increased expression of LDL receptors on liver cells

↓

increased removal of LDL from the blood

↓

reduced concentration of LDL CHO in plasma

(while an unwanted increase in TG)

# Clinical uses:

- ✓ **an addition to a statin if response has been inadequate**
- ✓ **for Hypercholesterolemia when a statin is contraindicated**
- ❖ ***Uses unrelated to atherosclerosis, including*:**
  - ➢ **pruritus** in patients with partial biliary obstruction
  - ➢ **bile acid diarrhea** (diabetic neuropathy)

# Adverse effects:

- GIT symptoms - nauzea, abdominal bloatin constipation or diarrhea
- Resins are unappetising. This can be mini by suspending them in fruit juice
- Interfere with the absorption of fat-s vitamins and drugs (chlorothiazide, dig warfarin)

- These drugs should be given at last 1 hour before or 4- after a resin

# Others

- *Nicotinic acid* inhibits hepatic T production and VLDL secretion
- Modest reduction in LDL and increase HDL

- **Adverse effects:**
- flushing, palpitations , GIT disturbances

- **Fish oil** (rich in highly unsaturated fatty acids
the omega-3 marine TG
reduce plasma TG but increase CHO (CHO i
more strongly associated wih coronary arter
disease)
the effects on cardiac morbidity or mortality i
unproven
although there is epidemiological evidence tha
eating fish regularly does reduce ischemic hear
disease)

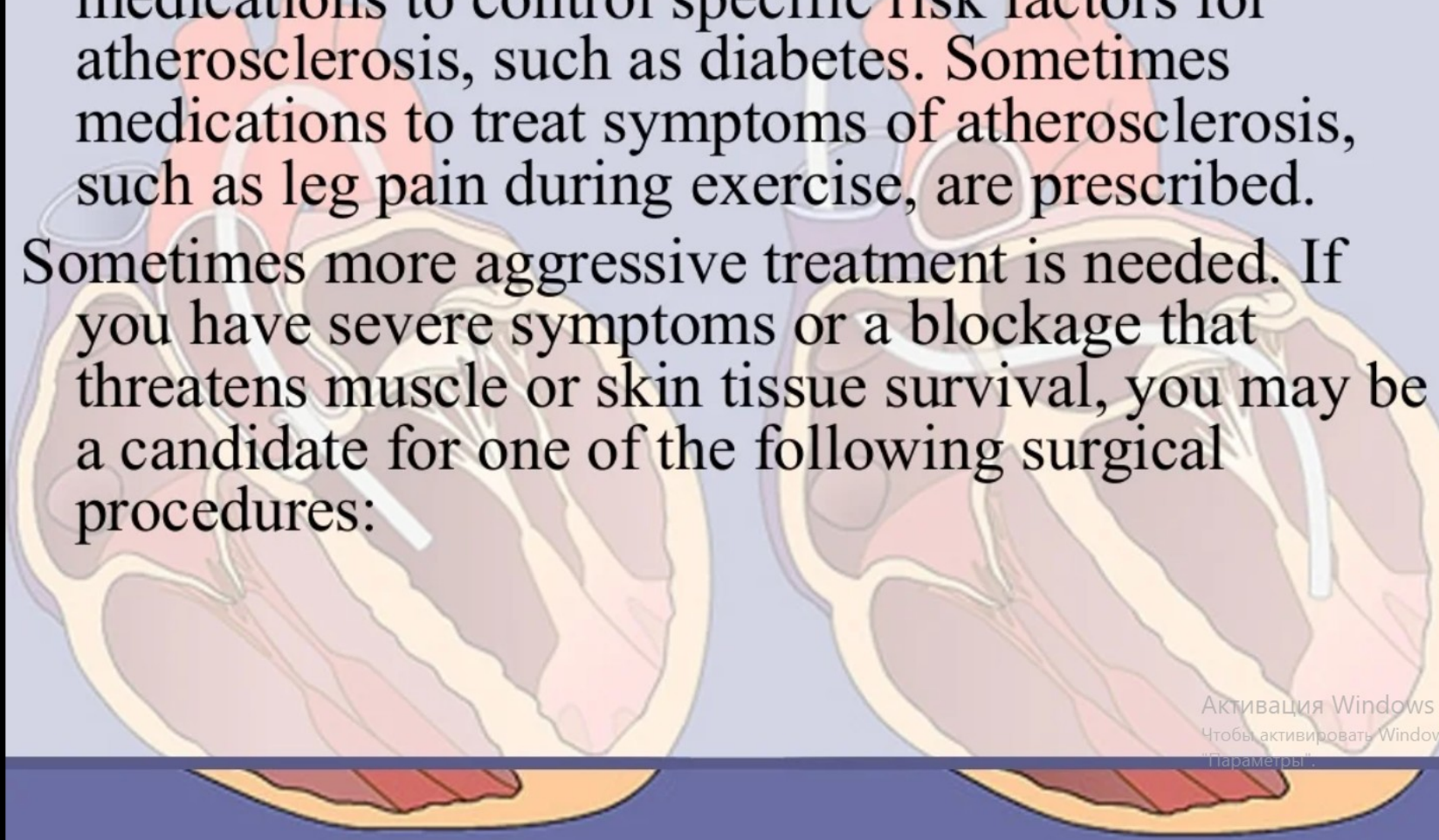
# Treatments and drugs

- **Anti-platelet medications.** Doctors may prescribe anti-platelet medications, such as aspirin, to reduce the likelihood that platelets will clump in narrowed arteries, form a blood clot and cause further blockage.
- **Anticoagulants.** An anticoagulant, such as heparin or warfarin (Coumadin), can help thin blood to prevent clots from forming.
- **Blood pressure medications.** Medications to control blood pressure — such as beta blockers, angiotensin-converting enzyme (ACE) inhibitors and calcium channel blockers — can help slow the progression of atherosclerosis

# Treatments and drugs

**Other medications.** doctor may suggest certain medications to control specific risk factors for atherosclerosis, such as diabetes. Sometimes medications to treat symptoms of atherosclerosis, such as leg pain during exercise, are prescribed.

Sometimes more aggressive treatment is needed. If you have severe symptoms or a blockage that threatens muscle or skin tissue survival, you may be a candidate for one of the following surgical procedures:



# Treatments and drugs

- **Angioplasty.** In this procedure, your doctor inserts a long, thin tube (catheter) into the blocked or narrowed part of your artery. A wire with a deflated balloon is passed through the catheter to the narrowed area. The balloon is then inflated, compressing the deposits agair your artery walls. A mesh tube (stent) is usually left in the artery to help keep the artery open. Angioplasty ma also be done with laser technology.
- **Endarterectomy.** In some cases, fatty deposits must be surgically removed from the walls of a narrowed artery When the procedure is done on arteries in the neck (the carotid arteries), it's known as carotid endarterectomy.

# Treatments and drugs

- **Thrombolytic therapy.** If you have an artery that's blocked by a blood clot, your doctor may insert a clot-dissolving drug into your artery at the point of the clot to break it up.
- **Bypass surgery.** Your doctor may create a graft bypass using a vessel from another part of your body or a tube made of synthetic fabric. This allows blood to flow around the blocked or narrowed artery.

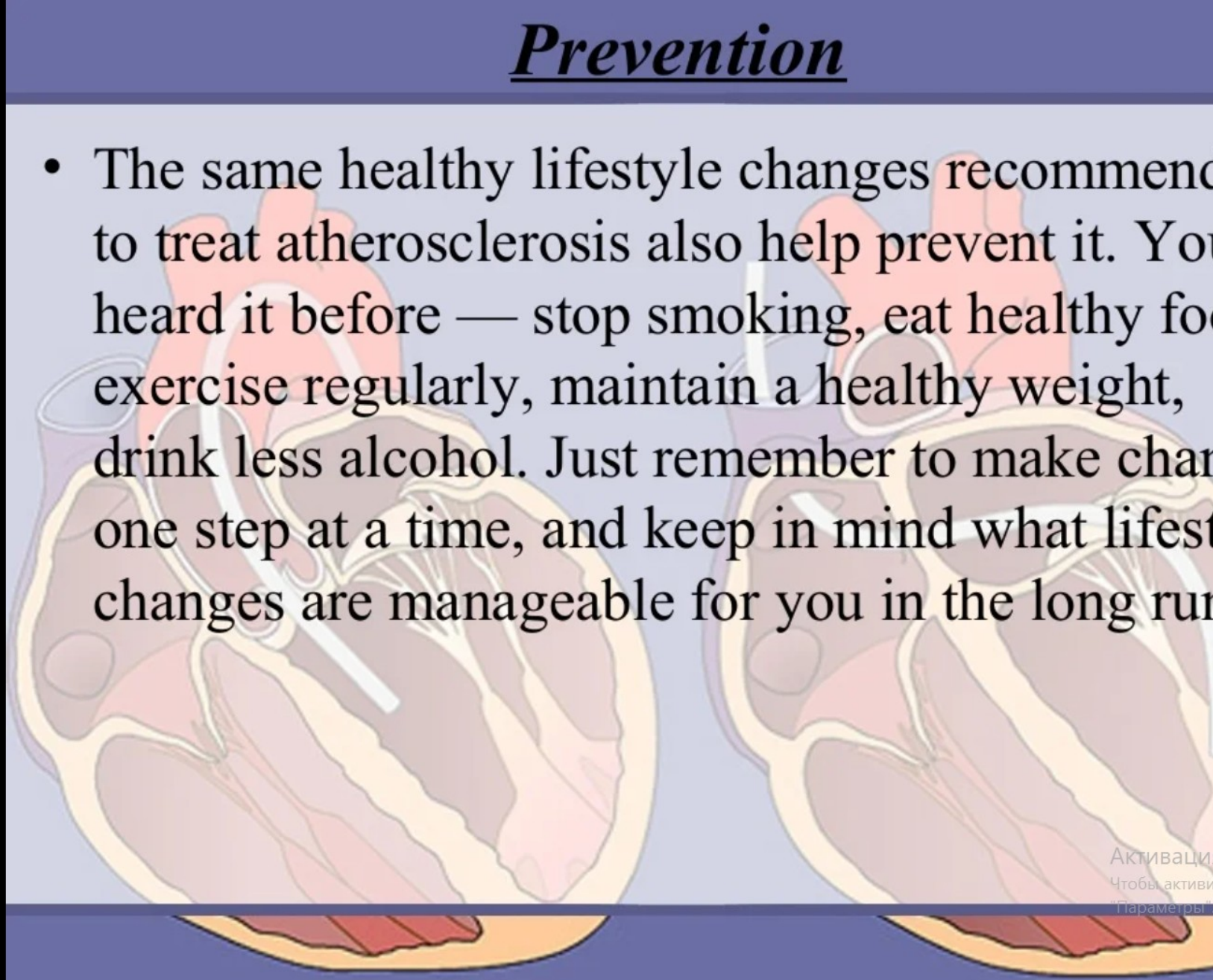
# Lifestyle and home remedies

Lifestyle changes can help prevent or slow the progression of atherosclerosis.

- **Stop smoking.**
- **Exercise most days of the week.**
- **Eat healthy foods**
- **Manage stress**
- **manage the condition of high cholesterol, high blood pressure, diabetes or other chronic disease**

# Prevention

- The same healthy lifestyle changes recommend to treat atherosclerosis also help prevent it. You heard it before — stop smoking, eat healthy foo exercise regularly, maintain a healthy weight, drink less alcohol. Just remember to make chan one step at a time, and keep in mind what lifest changes are manageable for you in the long run



# Conclusion

- Although atherosclerosis is considered a hea
  disease it can happen in any part of the bodi
- Atherosclerosis can be prevented by life sty
  factor and home remidies by eating healthy
  diet.
- Atherosclerosis is a preventable and treatab
  condition.