Cardiovascular I: Atherosclerosis and Hypertension

Overview

Cardiovascular I focuses on the close and dangerous interaction between **atherosclerosis** and **hypertension (HTN)**. Hypertension damages arterial walls and accelerates plaque formation, while atherosclerosis narrows and stiffens arteries, further raising blood pressure. This vicious cycle markedly increases the risk of **heart attack, stroke, and heart failure**, making blood pressure control essential for cardiovascular prevention.

1. Atherosclerosis (Hardening of Arteries)

Definition:

A progressive buildup of plaque (cholesterol, fats, inflammatory cells, debris) within arterial walls, causing arteries to become **narrow and rigid**, reducing blood flow.

Pathogenesis:

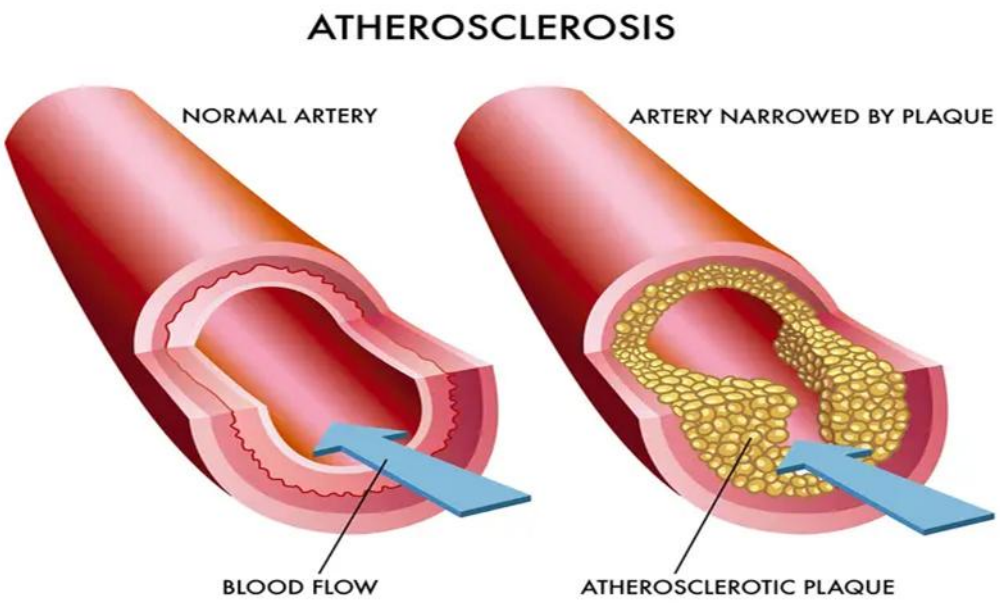
- Endothelial injury → inflammation
- Lipid (LDL) infiltration and foam cell formation
- Plaque growth and arterial narrowing
- Plaque erosion or rupture → thrombosis → acute ischemic events

Key Clinical Concept:

- Narrowing limits blood flow; **clot formation can completely block an artery**, causing tissue ischemia (e.g., myocardial infarction).

Symptoms (often late; usually when >70% blockage):

- **Heart (CAD):** Angina, dyspnea, fatigue, palpitations, nausea
- **Brain (Carotid disease):** TIA or stroke, weakness, speech difficulty, vision loss
- **Limbs (PAD):** Claudication, rest pain, non-healing ulcers, skin changes
- **Kidneys:** Resistant hypertension, edema, urinary changes
- **Digestive system:** Postprandial abdominal pain, weight loss, diarrhea



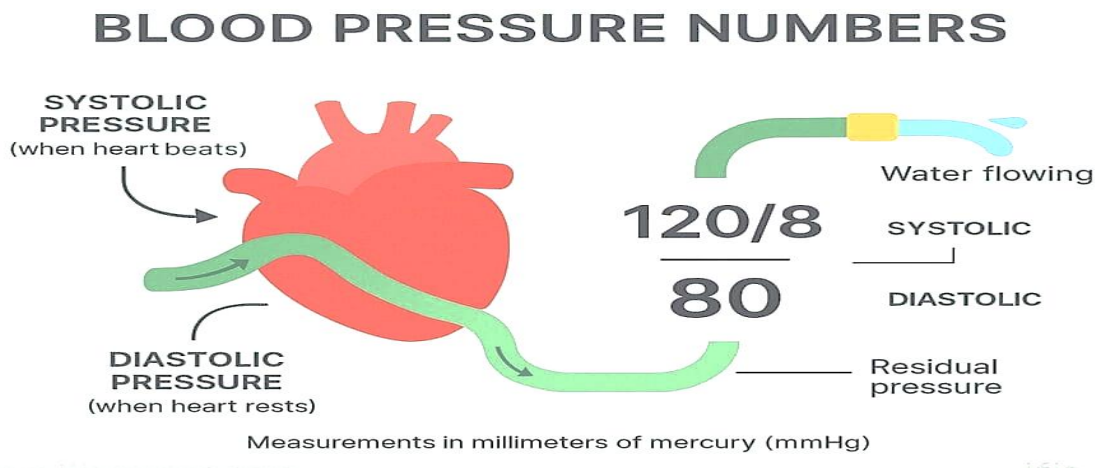
2. Hypertension (High Blood Pressure)

Definition:

Persistent elevation of arterial blood pressure (≥130/80 mmHg).

Classification:

- Normal: <120/80 mmHg
- Elevated: 120–129/<80 mmHg
- Stage 1 HTN: 130–139/80–89 mmHg
- Stage 2 HTN: ≥140/90 mmHg



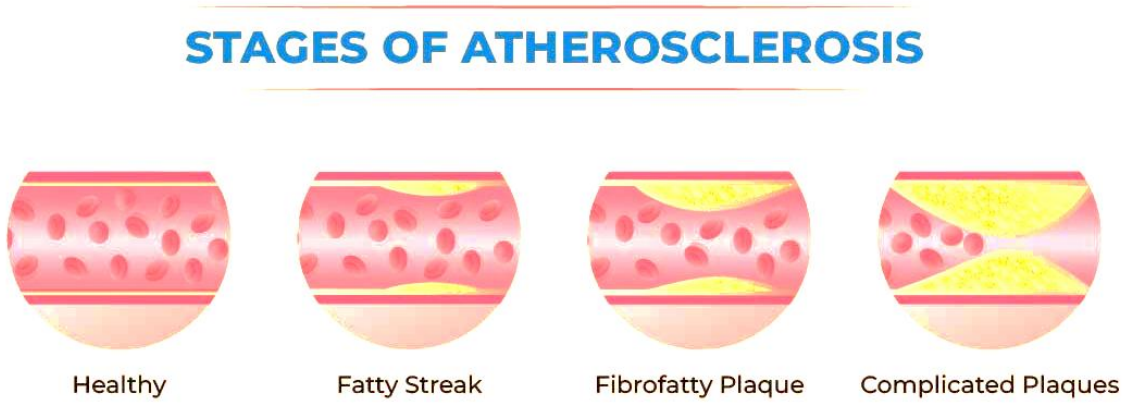
The force against artery walls during a heartbeat is measured by **the systolic blood pressure (top number), whereas the diastolic blood pressure (bottom number)** is measured during a heartbeat. A reading below 120/80 mmHg is considered normal. Both are essential for evaluating cardiovascular health, but in adults over 50, high systolic blood pressure is frequently a more reliable predictor of heart disease risk.

Types of hypertention:

- **Primary (essential):** No identifiable cause
- **Secondary:** Due to conditions like kidney disease
- **Special forms:** Isolated systolic HTN (elderly), white-coat HTN

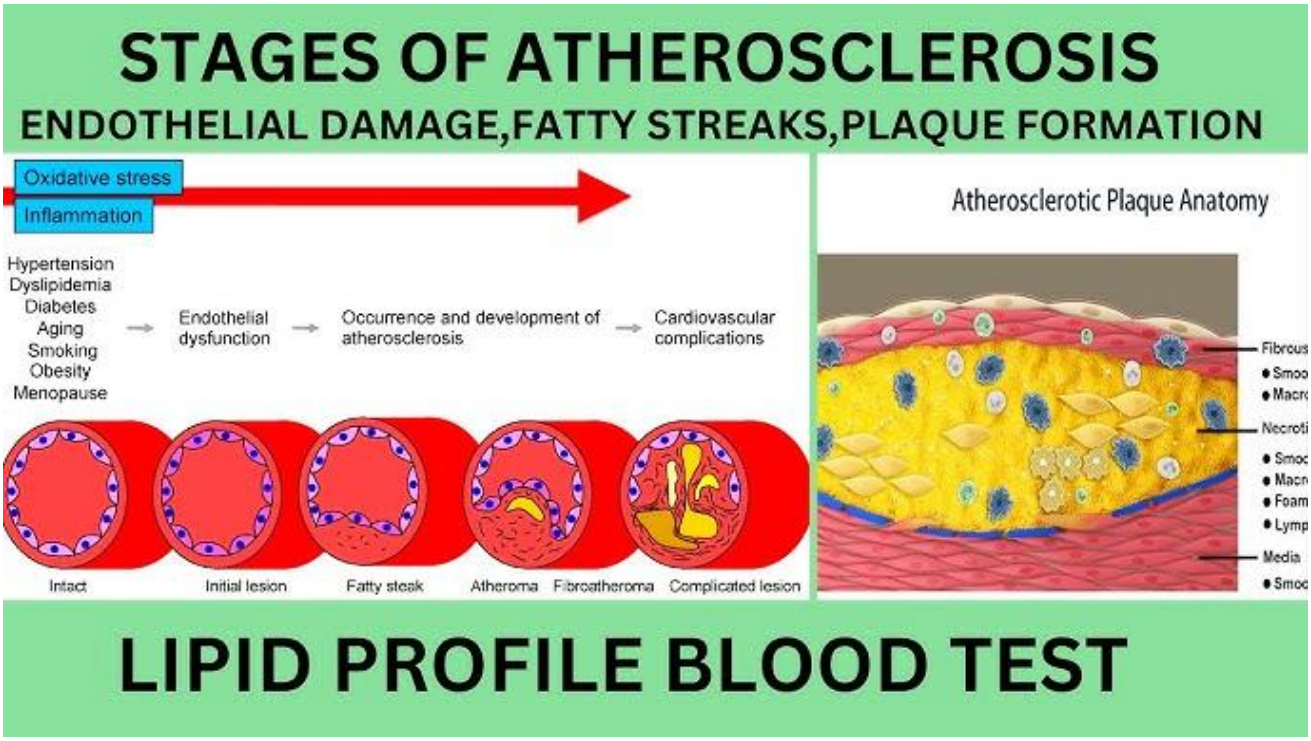
Impact on Arteries:

Chronic high pressure causes **arterial wall thickening, stiffness, and endothelial dysfunction**, promoting vascular disease.



3. How Hypertension Accelerates Atherosclerosis

- **Mechanical Stress:** Elevated pressure injures the endothelium.
- **Endothelial Dysfunction:** Increased permeability to lipids and inflammatory cells.
- **Plaque Progression:** Enhanced inflammation and lipid accumulation.
- **Plaque Instability:** Higher risk of rupture and thrombosis.
- **Magnitude of Effect:** HTN can increase plaque size **up to five-fold**, greatly raising cardiovascular risk.



4. The Vicious Cycle: HTN & Atherosclerosis

- **HTN → Atherosclerosis:** Arterial injury promotes LDL deposition and plaque growth.
- **Atherosclerosis → HTN:** Narrow, stiff arteries increase vascular resistance, raising BP.
- **Shared Risk Factors:** Genetics, dyslipidemia, diabetes, obesity, smoking, sedentary lifestyle.

5. Clinical Significance

- Hypertension is the **most important modifiable risk factor** for cardiovascular disease.
- Effective BP control slows atherosclerosis progression.
- Proper management significantly reduces the risk of **myocardial infarction, ischemic stroke, and heart failure**.

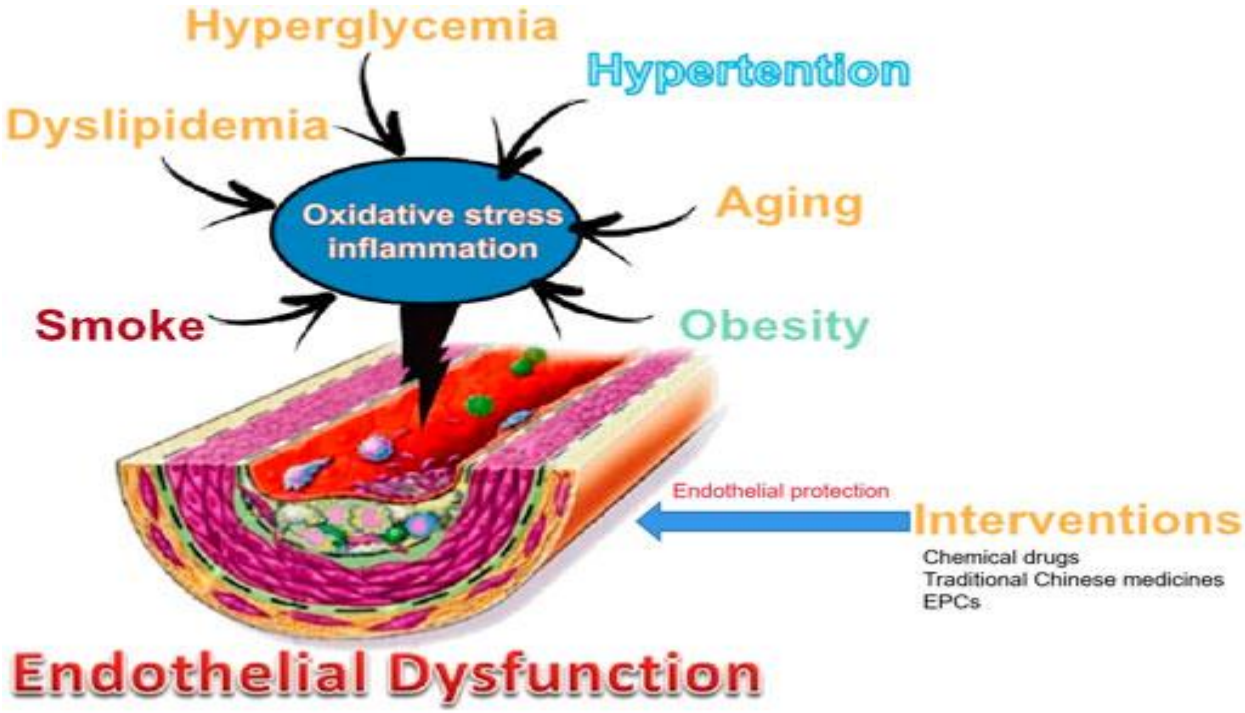
Atherosclerosis and hypertension are interdependent conditions that amplify each other. Early detection and aggressive control of blood pressure are critical to preventing major cardiovascular events.

What are the relation between atherosclerosis and tissues changes?

Atherosclerosis is a chronic, progressive, inflammatory disease characterized by the buildup of fatty plaques (atheromas) in the intima of medium- and large-sized arteries, leading to significant changes in vascular tissue and reduced, or blocked, blood flow. The disease is driven by endothelial dysfunction,

which triggers a cascade of cellular and structural changes, including the migration of smooth muscle cells, inflammation, and the formation of a necrotic core.

- **Vascular Endothelium:** Initial injury to the endothelial layer increases its permeability, allowing low-density lipoprotein (LDL) to enter the subendothelial space.



- **Intimal Layer (Fatty Streaks):** Monocytes migrate into the intima, becoming macrophages, and consume modified (oxidized) LDL to form **foam cells**. These accumulations of lipid-laden cells form "fatty streaks," which are the earliest visible lesions of atherosclerosis.
- **Advanced Plaque (Fibroatheroma):** Smooth muscle cells migrate from the media to the intima, multiplying and producing extracellular matrix to form a **fibrous cap** over the lipid-rich core.
- **Necrotic Core and Calcification:** Foam cells often undergo apoptosis and necrosis, creating a necrotic, lipid-rich core. As the plaque progresses, calcium accumulates within the plaque (calcification), leading to increased stiffening (arteriosclerosis).
- **Tissue Hypoxia:** The growing plaque restricts blood flow, causing oxygen deprivation (hypoxia) in surrounding tissues and organs.

Consequences on Tissue Function

- **Vessel Stiffness:** The accumulation of plaque and calcium leads to hardening (sclerosis) of the artery wall.
- **Reduced Blood Flow:** The narrowed artery (stenosis) restricts blood flow, leading to tissue ischemia.

- **Plaque Rupture:** Advanced, "vulnerable" plaques with thin fibrous caps can rupture, triggering blood clots that can cause acute events like heart attacks or strokes.
- **Organ Damage:** The reduced blood supply can result in damage to the heart (coronary artery disease), brain (stroke), and kidneys (chronic kidney disease).

Common Risk Factors and Pathophysiological Links

- **Inflammation:** A chronic inflammatory response is central, involving white blood cells (monocytes and T cells) and inflammatory factors like C-reactive protein (CRP).
- **Metabolic Changes:** In the plaque's hypoxic, inflammatory, and hyperlipidemic environment, metabolic activity shifts to increased anaerobic glycolysis, the pentose-phosphate pathway, and amino acid use.
- **Systemic Conditions:** Hypertension, high cholesterol (hyperlipidemia), diabetes, and obesity are key, often causing long-term, low-grade inflammation that damages vascular tissue

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1. Atherosclerosis is best defined as:
a. Acute vasospasm of arteries
b. Infection of arterial walls
c. **Progressive buildup of lipid-rich plaques within arterial walls**
d. Congenital narrowing of arteries
e. Sudden arterial rupture

2. The primary site of plaque formation in atherosclerosis is the:
a. Media
b. Adventitia
c. **Intima**
d. Perivascular tissue
e. Endocardium

3. The earliest visible lesion of atherosclerosis is known as:
a. Fibrous plaque
b. Necrotic core
c. Calcified plaque
d. **Fatty streak**
e. Thrombus

4. Endothelial injury primarily leads to:
a. Vasodilation
b. Reduced lipid entry
c. **Increased permeability to LDL**

d. Decreased inflammation
e. Vessel regeneration

5. Foam cells are formed from:
a. Platelets ingesting fibrin
b. Smooth muscle cells ingesting HDL
c. **Macrophages ingesting oxidized LDL**
d. Endothelial cells ingesting glucose
e. Neutrophils ingesting bacteria

6. Which blood pressure reading defines hypertension according to current guidelines?
a. ≥120/80 mmHg
b. ≥125/85 mmHg
c. **≥130/80 mmHg**
d. ≥135/85 mmHg
e. ≥140/100 mmHg

7. Stage 2 hypertension is classified as:
a. ≥120/80 mmHg
b. ≥130/85 mmHg
c. ≥135/90 mmHg
d. **≥140/90 mmHg**
e. ≥150/100 mmHg

8. Primary (essential) hypertension is characterized by:
a. Renal artery stenosis
b. Endocrine disorders
c. Cardiac malformations
d. **No identifiable underlying cause**
e. Acute infection

9. Secondary hypertension is commonly caused by:
a. Stress alone
b. Aging
c. Obesity
d. Smoking
e. **Kidney disease**

10. Which hypertension type is common in elderly patients?
a. White-coat hypertension
b. Malignant hypertension
c. Secondary hypertension
d. **Isolated systolic hypertension**
e. Resistant hypertension

11. Hypertension accelerates atherosclerosis mainly by:
a. Reducing LDL levels
b. Enhancing nitric oxide release
c. **Increasing mechanical stress on endothelium**

d. Decreasing inflammation
e. Promoting vasodilation

12. Chronic hypertension causes arterial walls to become:
a. Thin and fragile
b. Dilated
c. Elastic
d. **Thickened and stiff**
e. Permeable to water only

13. Plaque rupture most commonly leads to:
a. Vasodilation
b. Regression of plaque
c. **Thrombosis and acute ischemia**
d. Reduced inflammation
e. Vessel healing

14. Atherosclerosis most commonly affects which type of arteries?
a. Capillaries
b. Veins
c. Lymphatics
d. **Medium and large arteries**
e. Venules

15. Myocardial infarction occurs primarily due to:
a. Chronic anemia
b. Vasospasm alone
c. **Complete arterial occlusion by thrombus**
d. Increased oxygen demand
e. Cardiac hypertrophy

16. Symptoms of atherosclerosis usually appear when arterial narrowing exceeds:
a. 30%
b. 40%
c. 50%
d. 60%
e. **70%**

17. Claudication is most associated with:
a. Coronary artery disease
b. Cerebrovascular disease
c. Renal artery disease
d. **Peripheral arterial disease**
e. Mesenteric ischemia

18. Transient ischemic attack (TIA) results from atherosclerosis of the:
a. Renal arteries
b. Mesenteric arteries
c. **Carotid arteries**

d. Femoral arteries
e. Coronary arteries

19. Resistant hypertension may indicate atherosclerosis affecting the:
a. Pulmonary arteries
b. Cerebral arteries
c. Coronary arteries
d. **Renal arteries**
e. Iliac arteries

20. Postprandial abdominal pain is a sign of atherosclerosis in the:
a. Coronary circulation
b. Cerebral circulation
c. Peripheral circulation
d. Renal circulation
e. **Mesenteric arteries**

21. The fibrous cap of an atherosclerotic plaque is mainly produced by:
a. Endothelial cells
b. Platelets
c. Macrophages
d. **Smooth muscle cells**
e. Neutrophils

22. Calcification within plaques leads to:
a. Plaque regression
b. Increased elasticity
c. **Increased arterial stiffness**
d. Reduced BP
e. Vessel dilation

23. Tissue hypoxia in atherosclerosis results primarily from:
a. Increased oxygen demand
b. Hemoglobin deficiency
c. **Reduced blood flow due to stenosis**
d. Hyperventilation
e. Venous obstruction

24. Which metabolic shift occurs in hypoxic atherosclerotic plaques?
a. Increased oxidative phosphorylation
b. Fatty acid oxidation
c. **Increased anaerobic glycolysis**
d. Ketone utilization
e. Reduced glucose use

25. The necrotic core of a plaque forms mainly due to:
a. Excess collagen synthesis
b. Endothelial proliferation
c. **Death of foam cells**

d. Platelet aggregation
e. Vasodilation

26. White-coat hypertension refers to:
a. Chronic uncontrolled BP
b. Nocturnal hypertension
c. **Elevated BP only in clinical settings**
d. BP elevation during sleep
e. Drug-induced hypertension

27. Which factor is central to both hypertension and atherosclerosis?
a. Infection
b. Dehydration
c. **Chronic inflammation**
d. Hypoglycemia
e. Hypercalcemia

28. C-reactive protein (CRP) is associated with:
a. Lipid digestion
b. Blood clot dissolution
c. **Inflammation in atherosclerosis**
d. Oxygen transport
e. Renal filtration

29. A major modifiable risk factor for cardiovascular disease is:
a. Age
b. Sex
c. Genetics
d. **Hypertension**
e. Ethnicity

30. The vicious cycle between hypertension and atherosclerosis means:
a. Each condition resolves the other
b. One prevents the other
c. **Each condition worsens the other**
d. They occur independently
e. Only one causes symptoms

31. Increased vascular resistance in atherosclerosis is mainly due to:
a. Vasodilation
b. Decreased blood viscosity
c. **Narrowed and stiff arteries**
d. Increased cardiac output
e. Reduced heart rate

32. Which organ damage is commonly linked to long-standing hypertension?
a. Liver cirrhosis
b. Lung fibrosis
c. **Heart failure**

d. Peptic ulcer disease
e. Pancreatitis

33. Stroke due to atherosclerosis is usually:
a. Hemorrhagic only
b. Infectious
c. **Ischemic**
d. Traumatic
e. Congenital

34. Which lifestyle factor contributes to both conditions?
a. Adequate exercise
b. Balanced diet
c. Adequate sleep
d. **Smoking**
e. Hydration

35. The main consequence of arterial sclerosis is:
a. Increased elasticity
b. Vessel dilation
c. **Loss of compliance**
d. Reduced pressure
e. Improved perfusion

36. Which cells migrate from media to intima during plaque formation?
a. Endothelial cells
b. Platelets
c. Fibroblasts
d. **Smooth muscle cells**
e. Neutrophils

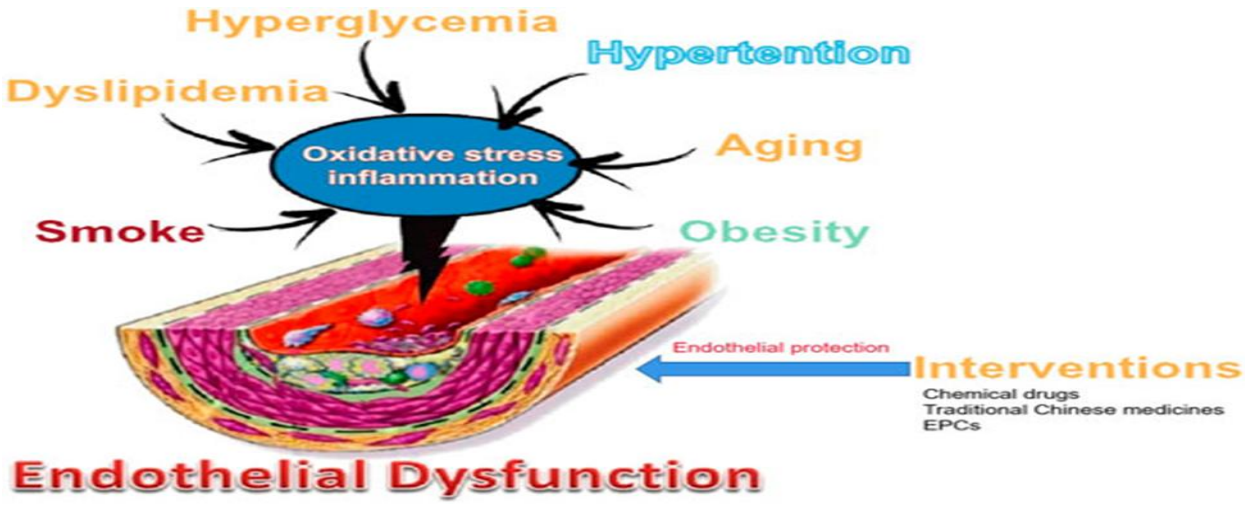
37. Hyperlipidemia contributes to atherosclerosis by:
a. Reducing LDL oxidation
b. Lowering inflammation
c. **Increasing lipid deposition in arteries**
d. Improving endothelial function
e. Enhancing fibrinolysis

38. Non-healing ulcers in legs suggest:
a. Venous insufficiency only
b. Neuropathy
c. Infection
d. **Peripheral arterial disease**
e. Lymphatic obstruction

39. Effective blood pressure control results in:
a. Increased plaque rupture
b. Accelerated plaque growth
c. **Slowed progression of atherosclerosis**

d. Immediate plaque removal
e. Complete cure

40. The most critical strategy to prevent major cardiovascular events is:
a. Treating symptoms only
b. Late surgical intervention
c. Ignoring early disease
d. **Early detection and aggressive BP control**
e. Calcium supplementation



1. The central mechanism linking multiple cardiovascular risk factors in the image is:
a. Thrombosis
b. Vasodilation
c. Lipid metabolism
d. **Oxidative stress and inflammation**
e. Calcification

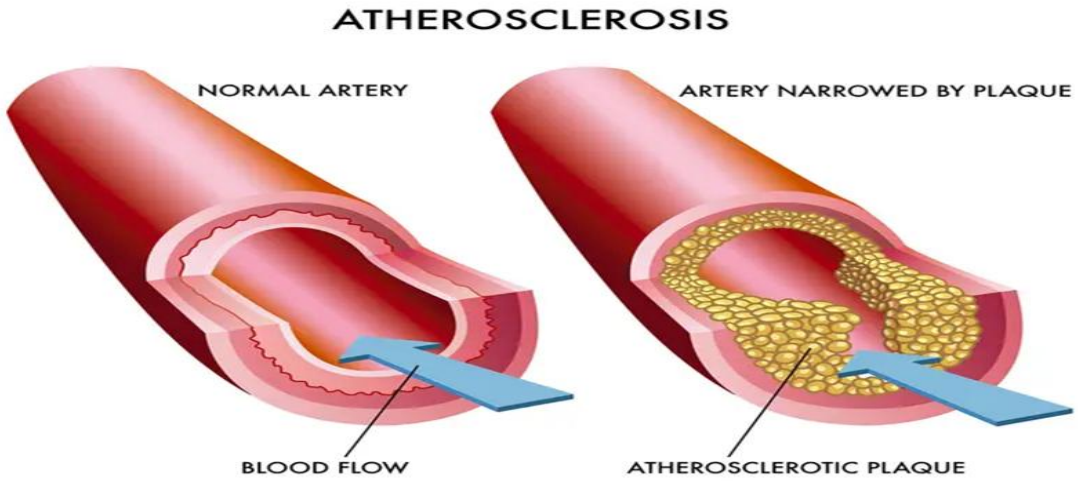
2. Which of the following is shown as a contributor to endothelial dysfunction in the image?
a. Hypoglycemia
b. Hypotension
c. **Dyslipidemia**
d. Anemia
e. Dehydration

3. Smoking contributes to endothelial dysfunction primarily by increasing:
a. Oxygen delivery
b. Nitric oxide production
c. Vessel elasticity
d. **Oxidative stress and inflammation**
e. Blood flow velocity

4. The structural change highlighted at the blood vessel level in the image is:
a. Venous valve failure

b. Capillary leakage
c. Myocardial hypertrophy
d. **Endothelial dysfunction**
e. Aneurysm formation

5. According to the image, which intervention strategy aims to counteract endothelial dysfunction?
a. Surgical bypass
b. Radiation therapy
c. Gene deletion
d. Lifestyle neglect
e. **Endothelial protection therapies**



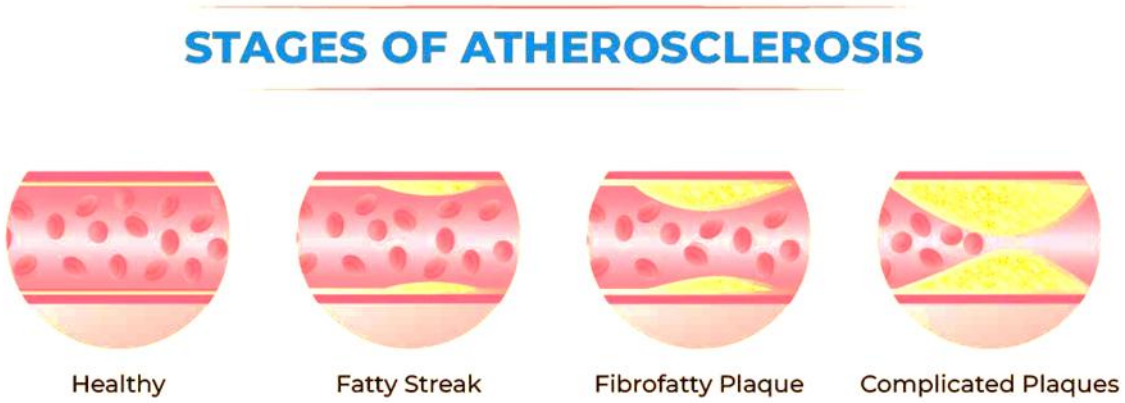
1. What is atherosclerosis primarily characterized by?
a. Sudden rupture of veins
b. Infection of the arterial wall
c. **Buildup of fatty plaques inside the arteries**
d. Enlargement of red blood cells
e. Increased elasticity of arteries

2. Compared to a normal artery, an artery affected by atherosclerosis shows:
a. Wider lumen and faster flow
b. Thinner arterial walls
c. Complete absence of blood flow
d. **Narrowed lumen due to plaque accumulation**
e. Increased oxygen content

3. What is the main effect of plaque formation on blood flow?
a. It increases blood pressure instantly
b. It improves oxygen delivery
c. **It reduces and restricts blood flow**
d. It converts arteries into veins
e. It prevents clot formation

4. Where does atherosclerotic plaque develop?
a. Outside the blood vessel
b. Only in capillaries
c. In the heart valves
d. **Along the inner lining of the artery**
e. Inside white blood cells

5. Which structure allows smooth blood movement in a normal artery?
a. Thick plaque layer
b. Irregular lumen
c. Collapsed vessel walls
d. Blocked passage
e. **Open and unobstructed lumen**



1. What is the first stage in the development of atherosclerosis?
a. Fibrofatty plaque
b. Complicated plaque
c. Thrombus formation
d. **Fatty streak**
e. Calcified artery

2. Which stage represents a normal artery with no lipid accumulation?
a. Fibrous plaque
b. Fatty streak
c. Complicated lesion
d. Advanced blockage
e. **Healthy**

3. Fibrofatty plaque is best described as:
a. Temporary widening of the artery
b. Dissolution of cholesterol deposits
c. **A thicker plaque that begins to narrow the arterial lumen**
d. Complete arterial rupture
e. Increased blood oxygen levels

4. What is a major risk associated with complicated plaques?
a. Improved blood circulation
b. Decreased blood viscosity
c. Expansion of the artery
d. **Severe blockage or possible clot formation**
e. Reduced heart workload

5. As atherosclerosis progresses from fatty streak to complicated plaque, the artery typically becomes:
a. More flexible
b. Wider
c. Free of lipids
d. Filled with oxygen
e. **Increasingly narrowed**