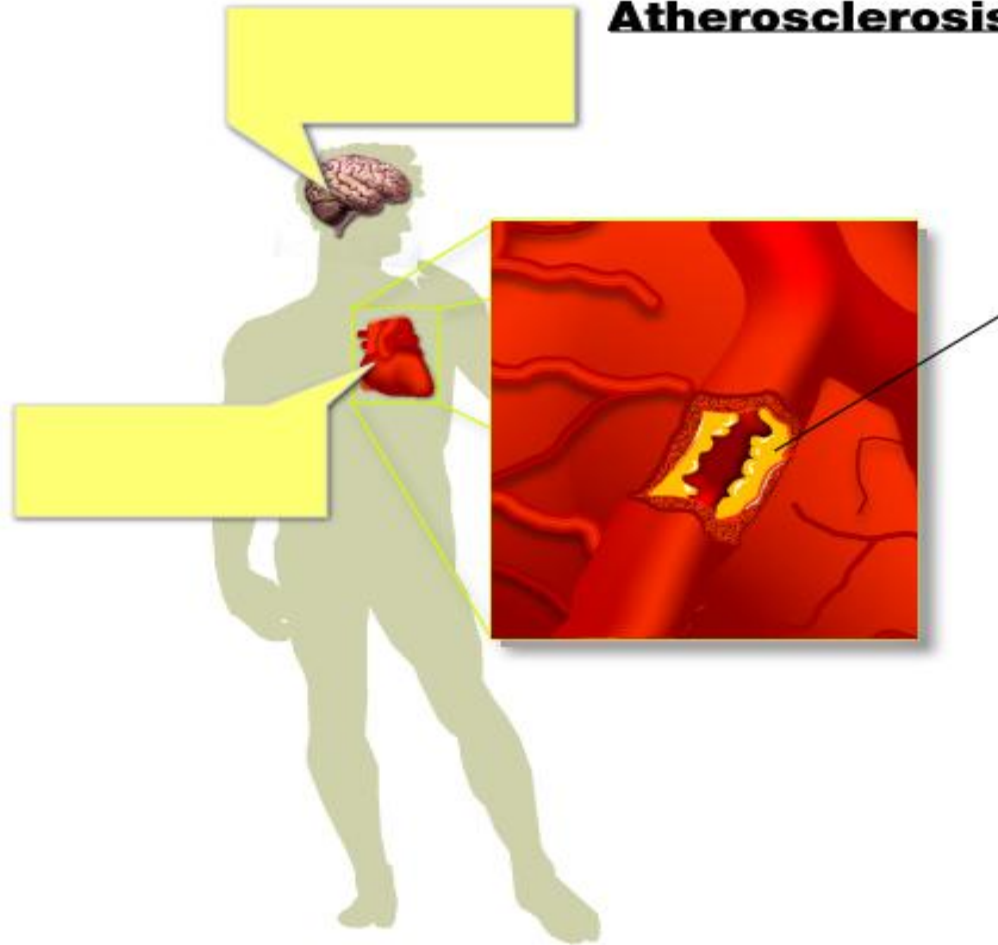


Atherosclerosis



ATHEROSCLEROSIS PATHOLOGY

Ortega RN MSN, CNS

ATHEROSCLEROSIS DISEASE

- Atherosclerosis: Type of blood vessel disorder
 - Begins as soft deposits of fat that harden with age
 - Referred to as “hardening of arteries”
 - Involves progressive narrowing and degeneration of arteries of heart, carotid, abdomen, and extremities.
- Atherosclerosis is the leading cause in majority of cases.



ATHEROSCLEROSIS

- Common Terms:
 - Arteriosclerotic heart disease
 - Cardiovascular heart disease
 - Coronary artery disease (CAD)
 - Peripheral vascular disease (PAD)
 - Carotid Disease



DESCRIPTION

- Typically appears at ages 60s to 80s
- Largely undiagnosed

- Risk factors
 - Cigarette smoking
 - Hyperlipidemia
 - Hypertension
 - Diabetes mellitus



ETIOLOGY

- Characterized by a *focal deposit of cholesterol and lipid*, primarily **within** the **intimal wall** of the artery
- Endothelial *lining altered* as a result of *inflammation and injury*



PATHOGENESIS OF ATHEROSCLEROSIS

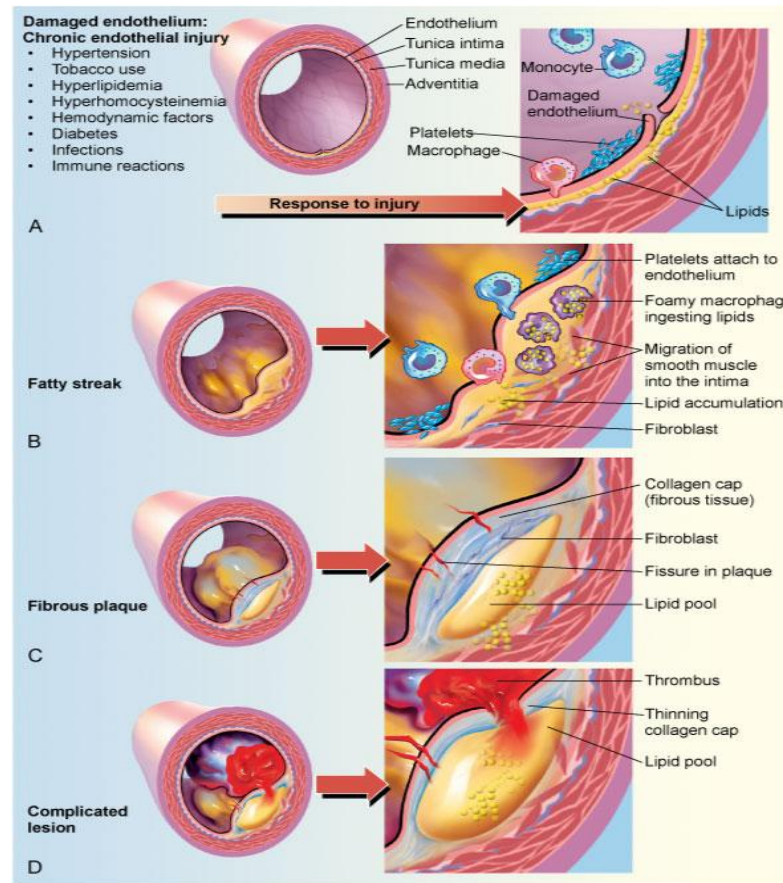


Fig. 34-1. Pathogenesis of atherosclerosis. **A**, Damaged endothelium. **B**, Diagram of fatty streak and lipid core formation. **C**, Diagram of fibrous plaque. Raised plaques are visible: some are yellow, others are white. **D**, Diagram of complicated lesion: thrombus is red, collagen is blue. Plaque is complicated by red thrombus deposition.

PATHOPHYSIOLOGY

- + C-reactive protein (CRP)
 - Non-specific marker of inflammation
 - Increased in many patients with CAD
 - Chronic exposure to CRP associated with unstable plaques and oxidation of LDL cholesterol



PATHOPHYSIOLOGY

- B. Developmental stages: **Fatty streaks**
 - Earliest lesions
 - Characterized by **lipid-filled smooth muscle cells**
 - Potentially **reversible**



PATHOPHYSIOLOGY

- c. Developmental stages: *Fibrous plaque*
 - Beginning of progressive changes in the arterial wall
 - *Lipoproteins* transport cholesterol and other lipids into the *arterial intima*.
 - Fatty streak is *covered by collagen*, forming a *fibrous plaque* that appears *grayish or whitish*.
 - Result = *Narrowing* of vessel lumen



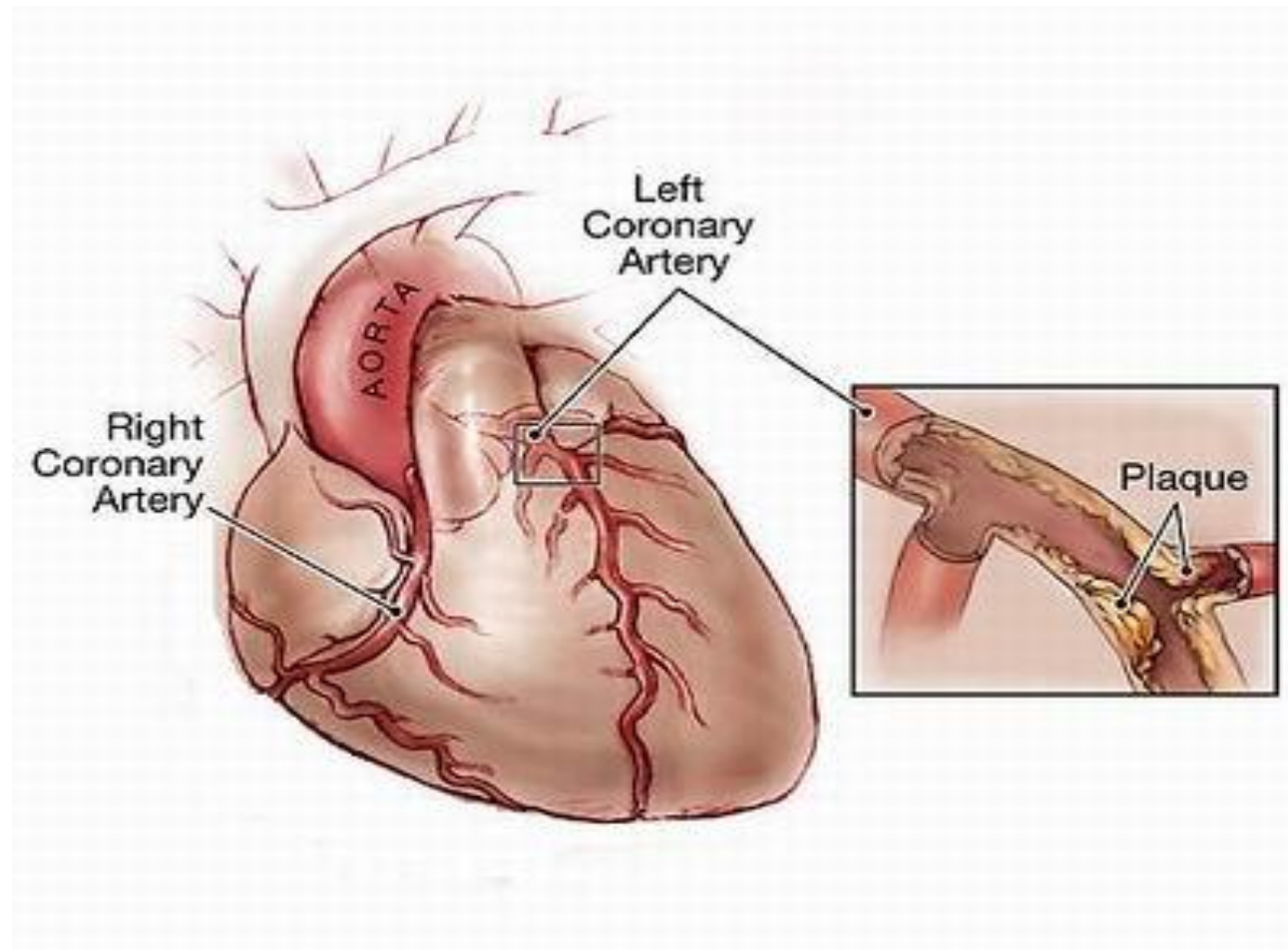
PATHOPHYSIOLOGY

- D. Developmental stages: *Complicated lesion*
 - Continued *inflammation* can result in plaque *instability, ulceration, and rupture*.
 - *Platelets accumulate* and *thrombus forms*.
 - Increased *narrowing* or *total occlusion* of lumen

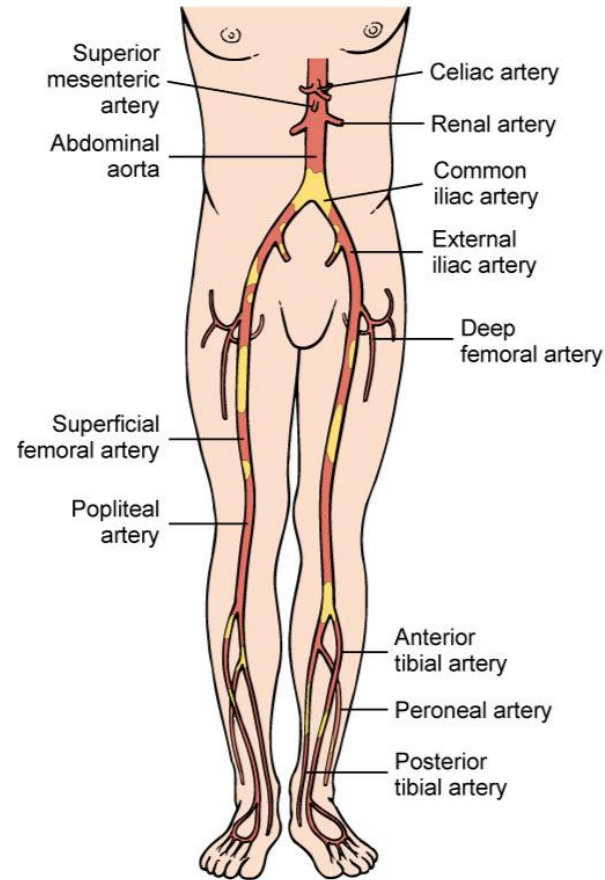


COMMON SITES IN THE CORONARIES: CAD

- *Affinity for coronaries*



COMMON SITES OF ATHEROSCLEROTIC LESIONS

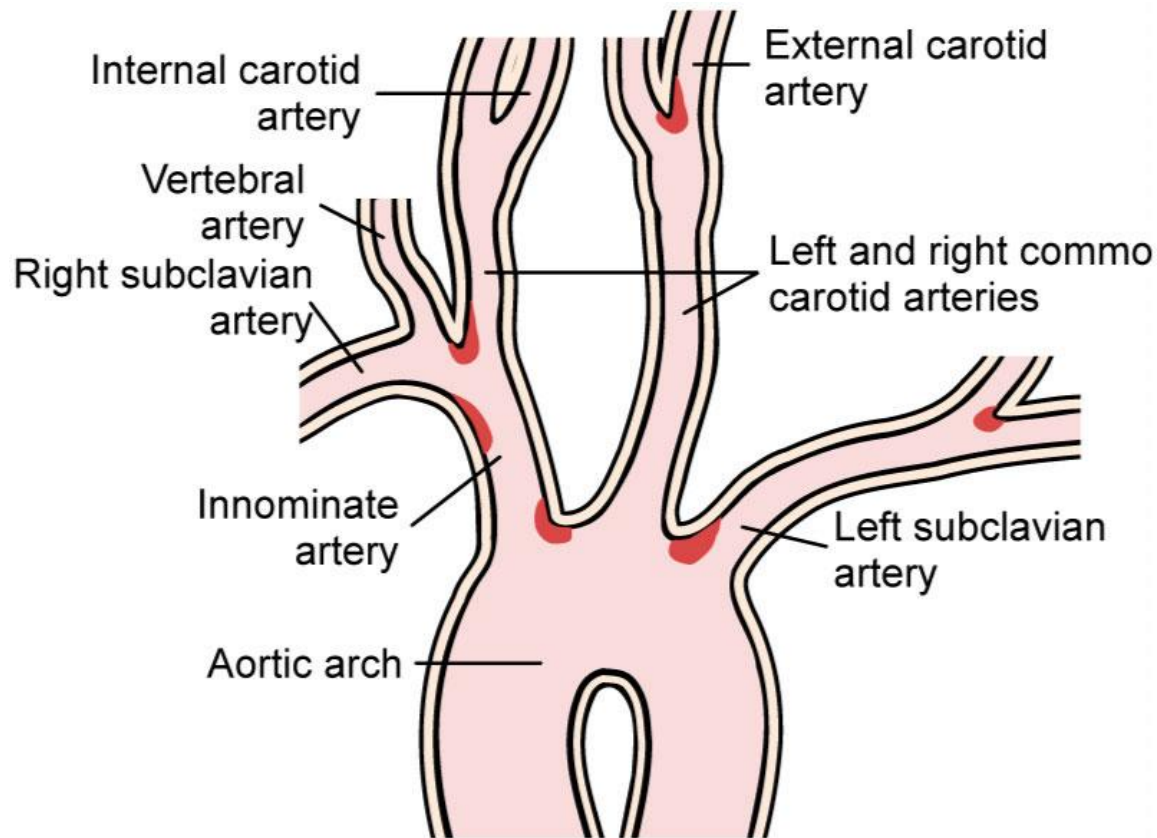


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Fig. 38-1. Common anatomic locations of atherosclerotic lesions (shown in yellow) of the abdominal aorta and lower extremities.



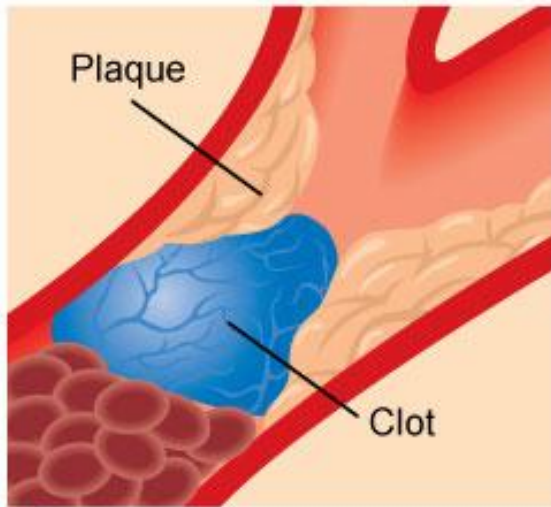
COMMON LESION: CAROTID VESSELS



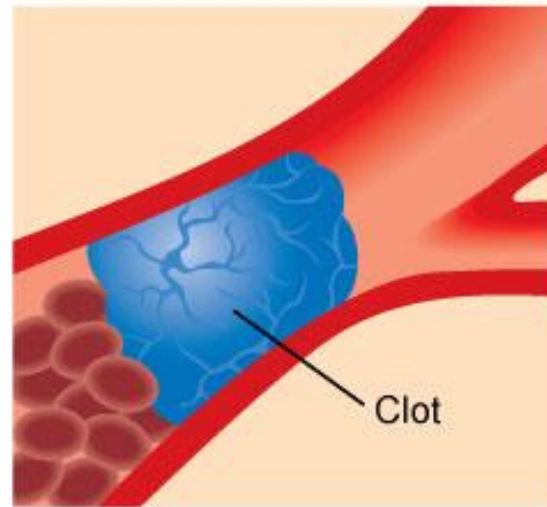
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Fig. 58-2. Common sites for the development of atherosclerosis in extracranial and intracranial arteries. The main locations are just above the common carotid bifurcation (most common site) and the start of the branches from the aorta, innominate, and subclavian arteries.

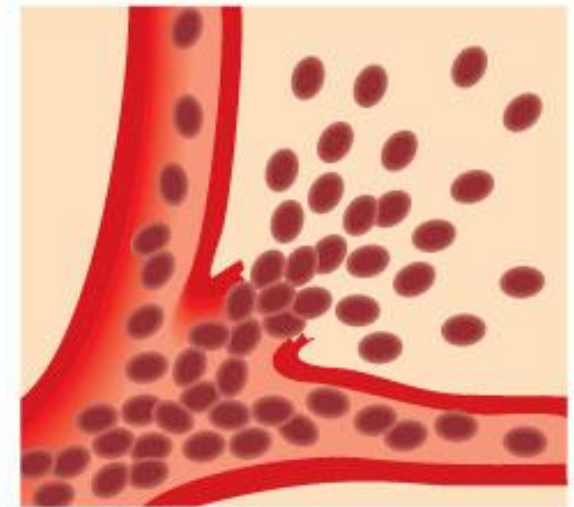
MAJOR TYPES OF STROKE



Thrombotic stroke. The process of clot formation (thrombosis) results in a narrowing of the lumen, which blocks the passage of the blood through the artery.



Embolic stroke. An embolus is a blood clot or other debris circulating in the blood. When it reaches an artery in the brain that is too narrow to pass through, it lodges there and blocks the flow of blood.



Hemorrhagic stroke. A burst blood vessel may allow blood to seep into and damage brain tissues until clotting shuts off the leak.

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Fig. 58-3. Major types of stroke.



ISCHEMIC STROKE

- Ischemic strokes result from
 - Inadequate blood flow to the brain from partial or complete occlusion of an artery
- 80% of all strokes are ischemic strokes.

