



AMBOSS

FOR INSTITUTIONS



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resource
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students

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Discover the comprehensive resource students love for all of medical school



Digital Library: Read & reference key curricular content

- Comprehensive collection of pre-clinical and clinical learning material designed to support both exam prep and applied clinical skills
- 17,000+ searchable medical terms and over 200,000 cross-links between topics
- Up-to-date, scientifically sourced, and portable

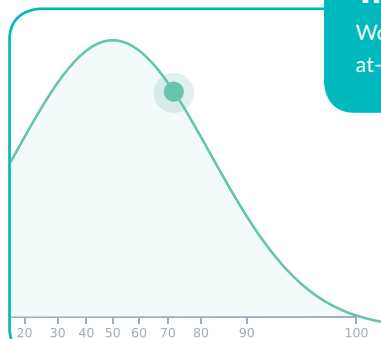


Advanced Qbank: Effective NBME® Step & Shelf prep

- 4,700+ exam-style practice questions cover relevant topics strategically
- Unique clinical vignettes with sophisticated explanations challenge and teach students
- Detailed performance analytics guide students towards personalized study goals
- Teaches critical thinking skills for patient care in practice

Track performance & monitor trends

Work closely with our data to better support your at-risk students and inform your curricular decisions.



University features

Educators can curate sets of questions from the AMBOSS Qbank and assign them with corresponding Learning Cards to students.

Knowledge just a click away

Open up the AMBOSS Knowledge Library side by side with questions and review past mistakes, hone in on high-yield content points, or explore topics in depth.

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Reinforce skills & understanding
Every case-based multiple choice question breaks down suggested stem highlights, clinical reasoning advice, and unique explanations for each answer choice.

Videos, illustrations, overlays
The Library contains thousands of high-quality images, flowcharts, and interactive media (like quizzes and overlays) which boost the learning experience and help train the clinical eye.

Question Vignette:
A 61-year-old woman is brought to the emergency department because of crushing substernal chest pain at rest for the past 2 hours. She is diaphoretic. Her temperature is 37.5°C (99.5°F), pulse is 110/min, respirations are 21/min, and blood pressure is 115/65 mm Hg. An ECG shows ST elevation in I, aVL, and V2-V4. Coronary angiography shows an 80% stenosis in the left anterior descending artery. Which of the following is the most likely initial step in the pathogenesis of this patient's coronary condition?

Answer Choices:
A. Intimal smooth muscle cell migration (1%)
B. Intimal monocyte infiltration (1%)
C. Platelet activation (2%)
D. Low density lipoprotein oxidation (12%)
E. Endothelial cell dysfunction (69%)
F. Intimal foam cell accumulation (10%)
G. Fibrous plaque formation (4%)

Explanation:
The development of atherosclerosis begins with an external factor (e.g., hypertension, smoking, advanced glycation end-products) damaging the endothelium, which increases adhesiveness and leads to local inflammation and invasion of smooth muscle cells into the tunica intima. Inflammatory cytokines attract macrophages, which then form foam cells by taking up cholesterol from oxidized low-density lipids. These foam cells accumulate to form fatty streaks (early atherosclerotic lesions). Finally, collagen production by lipid-laden macrophages and SMCs form these fatty streaks into a fibrous plaque.

Knowledge Article: Pathogenesis of atherosclerosis
1. Chronic stress on the endothelium
2. Endothelial dysfunction, which leads to:
• Invasion of inflammatory cells (mainly monocytes and lymphocytes) through the disrupted endothelial barrier
• Adhesion of platelets to the damaged vessel wall → platelets release inflammatory mediators (e.g., cytokines) and platelet-derived growth factor (PDGF)
• PDGF stimulates migration and proliferation of smooth muscle cells (SCM) in the tunica intima and mediates differentiation of fibroblasts into myofibroblasts
3. Inflammation of the vessel wall
4. Macrophages and SMCs ingest cholesterol from oxidized LDL and transform into foam cells
5. Foam cells accumulate to form fatty streaks (early atherosclerotic lesions).
6. Lipid-laden macrophages and SMCs produce extracellular matrix (e.g., collagen) → development of a fibrous plaque (atheroma)
7. Inflammatory cells in the atheroma (e.g., macrophages) secrete matrix metalloproteinases → weakening of the fibrous cap of the plaque due to the breakdown of extracellular matrix → minor stress ruptures the fibrous cap
8. Plaque rupture → exposure of thrombogenic material (e.g., collagen) → thrombus formation with vascular occlusion or spreading of thrombogenic material

Common sites (in order of frequency):
• Abdominal aorta
• Coronary arteries
• Popliteal arteries
• Carotid arteries

Atherosclerotic diseases:
• arterial aneurysm or dissection
• Demand-supply mismatch; coronary heart disease; peripheral artery disease; subcortical vascular dementia
• Thrombosis; acute coronary syndrome; stroke
• Renovascular hypertension: atherosclerosis of the renal artery → activation of the renin-angiotensin-aldosterone system

Media:
Includes a chest X-ray, a flowchart of the pathogenesis, histological images of atherosclerotic plaques, and a video player.

Partner with AMBOSS to improve your students' success

- Founded and managed by doctors
- 250+ AMBOSS employees support 500,000 users worldwide
- Institutional partnerships with dozens of universities in the USA and faculties in over 20 countries
- Customize your license for an integrated curriculum and work with our analytics to improve your students' scores

Contact us for trial access & next steps:
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