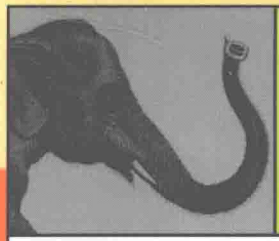

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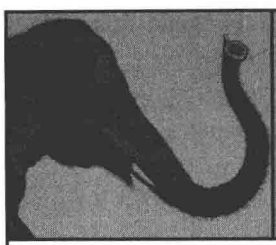

LANGE OUTLINE REVIEW™ USMLE STEP 3

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For My Children, Dan and Kasey

Preface

The typical review book is written in a question-and-answer-type format. It has long existed as the sole product for student examinations, until now. In 1992, I formulated my concept of a rapid-reading review manual, conceived out of the tremendous need for a succinct, yet complete review text. The key component was the extensive coverage of the USMLE "high-impact" disease list, with the inclusion and incorporation of all pertinent test material. In addition, it was necessary to present this material in a concise, easily assimilated format, to allow for a swift and highly effective review.

This text, *Lange Outline Review: USMLE Step 3,* Fifth Edition, exists as a result of the tremendous popularity and widespread use of the Step 2 text, called *The Instant Exam Review for USMLE Step 2.* After the Step 2 text received extraordinary acceptance and acclaim by students and educators across the United States and abroad, I was asked to create a new study book for the Step 3 exam. Thus, *Lange Outline Review: USMLE Step 3!*

In this review manual, my original concept and ideals remain unchanged. Once again, the material in this book encompasses the key test facts, diseases, and disorders listed by the National Board of Medical Examiners for the new Step 3 examination. Our categories in this revised edition have changed to reflect the new examination content, with each chapter encompassing the Board's new list of diseases and disorders.

Finally, I have enlisted as contributors an exceptional group of physicians, widely renowned for their clinical and educational proficiency. These authors have completely revised this book.

Please note that this book was not designed to teach general medicine, nor was it to be a substitute for accepted methods of medical education. Like its predecessor, it was designed as a unique study tool to assist you, the student, in passing the Step 3 examination.

Joel S. Goldberg, DO
Philadelphia, Pennsylvania

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I wish to thank my coauthors for their willingness to participate in this complex endeavor and for investing extensive time and effort in the construction of their chapters, despite their busy professional and personal schedules. They are a group of physicians dedicated to medicine, and their commitment to education is clear.

Finally, I would like to express my gratitude to the staff and faculty of Drexel University College of Medicine for their assistance and unselfish dedication to both the clinical practice of medicine and the education of young physicians in training.

How to Use This Book

This book is an innovative and practical study guide designed to be used in both the initial phase of USMLE Step 3 examination preparation as a comprehensive study outline and in the final few days and hours before the exam as a quick review manual.

USING THE BOOK AS A STUDY OUTLINE

When you begin to study, turn to the Contents to obtain an overview of this text. Review the material supplied by your school and the National Board of Medical Examiners, including the "Step 3 General Instructions, Content Description, and Sample Items." It is important to have a full understanding of the design of the exam and the type of questions that will be asked. Sample test questions and topics may also be found at www.usmle.org.

Once you begin to study, *do not* omit any chapters in this text; instead, start at the beginning and read the book in its entirety. Notice that the outline format is streamlined to allow the rapid assimilation of facts in a minimal amount of reading time. Because extraneous and time-consuming information and phrasing have been omitted, working with *Lange Outline Review: USMLE Step 3* for 1 hour will provide a database equivalent to that procured from several hours' study of any other review text. Because the text is concise, it is vital that you be well rested and in a proper frame of mind for study and concentration. A quiet, comfortable, bright study area without glare is vital (with plenty of snacks nearby, of course!).

USING THE BOOK AS A QUICK REVIEW

In the final several weeks and days prior to your examination, *Lange Outline Review: USMLE Step 3* will serve as a rapid review tool. As in the Step 2 text, this revolutionary new format, which completely covers the "high impact" fact list, will allow the handbook to be read quickly, with successful, easy assimilation of the core facts necessary for exam success.

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Cardiovascular Medicine 1

I. ISCHEMIC HEART DISEASE

A. Acute

1. Unstable Angina

► H&P Keys

Anginal pain with accelerating pattern including new onset or rest symptoms. Midsternal squeezing or heaviness that may radiate to the left shoulder or arm, jaw, neck, etc. Symptoms may be similar to those present previously; however, usual alleviating factors (e.g., nitroglycerin [NTG], rest) may no longer be effective. Diaphoresis, nausea, dyspnea, are common.

With coexisting left ventricular (LV) dysfunction an S_3 or S_4 may be heard. In the presence of global ischemia or LV dysfunction, a dyskinetic cardiac impulse may be palpated, and papillary muscle dysfunction may cause the murmur of mitral regurgitation (MR). Ischemia-induced elevations in cardiac filling pressures often cause pulmonary congestion and rales.

► Diagnosis

Electrocardiogram (ECG): ST depression or elevation or T wave inversion. Exercise tolerance testing (ETT; not performed in patients with unstable angina) or pharmacologic stress testing is combined with an imaging agent, such as thallium, that permits the visualization of myocardial perfusion. Coronary angiography is indicated for patients with high-risk clinical features.

► Disease Severity

High-risk clinical features are advanced age, ST segment depression, heart failure, and elevation of biomarkers of cell damage (troponins). Response to therapy and duration of symptoms dictate evaluation. Patients not medically stabilized or whose symptoms reemerge on therapy and those with prominent ischemic ECG findings usually undergo coronary angiography. Location and severity of stenoses (e.g., left main, three-vessel) dictate management.

► Concept and Application

The vast majority of patients with unstable angina have underlying coronary atherosclerosis (CAD). Unstable symptoms are usually caused by plaque rupture with superimposed thrombosis. Platelet aggregation at site of plaque rupture with release of vasoconstricting mediators plays an important role.

► Treatment Steps

1. Continuous ECG monitoring.
2. Bed rest, mild sedation, and treatment of extracardiac precipitants of increased oxygen demand (e.g., hypoxia, sepsis, anemia, uncontrolled hypo- or hypertension, etc.).
3. Intravenous nitrates, heparin, thienopyridines, and aspirin (acetylsalicylic acid [ASA]) and IIB/IIIA inhibitors are of proven efficacy. Thienopyridines, especially clopidogrel, reduce adverse events including death, myocardial infarction (MI), and stroke. Agents that block the platelet glycoprotein IIB/IIIA receptor substantially reduces mortality and the occurrence of acute MI primarily in patients who undergo coronary intervention. Intravenous NTG often is successful when other routes fail. β-Blockers are useful and also reduce the incidence of acute MI (AMI).

ISCHEMIC HEART DISEASE

Acute Myocardial Infarction
Sudden onset of typical squeezing or crushing substernal chest pain; ECG with ST segment elevation in two or more leads; increased CPK-MB isoenzymes and troponins.

Angina Pectoris
Chest pain lasting 1–15 minutes, precipitated by exertion, relieved by rest; associated ECG changes with ST segment depression. Abnormal exercise stress test or abnormal perfusion on nuclear scan.

Unstable Angina
Angina that increases in frequency or severity, is of new onset or occurs at rest. Coronary angiography defines the extent and severity of disease and need for intervention; exercise testing with perfusion imaging useful for risk stratification and treatment decisions.

4. Calcium channel blockers can provide symptomatic relief but do not decrease event rates.
5. Intra-aortic balloon counterpulsation (IABP) is often used as a bridge to percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass graft (CABG) and is effective in stabilizing medically refractory patients.
6. Lipid-lowering therapy with hydroxymethylglutaryl coenzyme A (HMG CoA) reductase inhibitors should be started for patients with low-density lipoprotein (LDL) > 100 mg/dL.

2. Myocardial Infarction

► H&P Keys

Chest pain, often midsternal squeezing or crushing. The pain may radiate to the neck, jaw, shoulders, arms, etc. Diaphoresis is frequent. Approximately 20% of episodes occur in the absence of pain (silent). Displaced and even dyskinetic cardiac impulse can be palpated. Ischemia-induced papillary muscle dysfunction may cause a MR murmur. Associated right ventricular (RV) infarction, may cause jugular venous distention (JVD). Elevations in cardiac filling pressures often cause pulmonary congestion, allowing auscultation of pulmonary rales.

► Diagnosis

ECG: ST segment elevation MI (STEMI) and T wave inversions with subsequent evolution of Q waves. Non-STEMI: ST depression and T wave inversions are seen. Elevations in cardiac enzymes: creatine kinase (CK), specifically CK-MB isoforms, peaks at 24 hours; troponins I and T, and myoglobin detect cell injury early in the course of MI. L-lactate dehydrogenase (LDH) peaks at 3–5 days after MI. Two-dimensional (2-D) echocardiography and technetium nuclear scans can be valuable in detecting abnormalities in heart function and blood flow.

► Disease Severity

Mortality increases with the number of ECG leads showing ST segment elevation. Cardiac imaging with echocardiography (echo) or radionuclide ventriculography (RVG) can help assess the extent and prognosis of infarction by measuring LV systolic function and ejection fraction. Echo aids in diagnosis of MI complications, e.g., LV thrombus or aneurysm, pericardial effusion, free wall and septal rupture, and MR.

► Concept and Application

MI results from the abrupt cessation of myocardial blood flow. The vast majority of cases of MI are due to CAD. Plaque rupture, platelet aggregation, and release of vasoactive mediators leads to thrombosis, spasm, and coronary occlusion. Elevation of myocardial oxygen demand (e.g., tachycardia) can increase myocardial cell damage. Irreversible cell death occurs, usually within 6 hours, if therapy is not given to restore blood flow or if spontaneous improvement does not occur. Nonatherosclerotic causes of MI, such as embolism, trauma, vasculitis, or hypercoagulable states, are less common.

► Treatment Steps

1. Supplemental oxygen, and continuous monitoring to detect potentially lethal dysrhythmias are important first steps.
2. Analgesia as necessary.
3. Primary angioplasty of occluded artery optimum. Thrombolysis if PTCA unavailable.

► management decisions

ISCHEMIC HEART DISEASE

Acute Myocardial Infarction
Open the occluded artery to restore cardiac blood flow with thrombolytic therapy or angioplasty; monitor and treat serious dysrhythmias, aspirin to decrease clot formation, β-blockers to decrease myocardial oxygen needs, oxygen and pain relief.

Angina Pectoris
Drug therapy with nitrates, β-blockers, aspirin to prevent acute MI; calcium antagonists are second-line therapy. Intervention with bypass surgery or coronary intervention for patients with refractory symptoms or severe coronary disease.

Unstable Angina
Drug therapy for stabilization with intravenous nitroglycerin, aspirin, heparin, clopidogrel, glycoprotein IIB/IIIA inhibitors, and β-blockers. Coronary interventions and coronary bypass surgery for severe coronary disease.

4. Aspirin should be given on day 1 of AMI to all patients without a contraindication.
5. Heparin and ASA are useful in conjunction with thrombolysis.
6. Intravenous NTG is helpful in decreasing oxygen demand and increasing supply.
7. β-Blocker therapy should be administered to all patients without a contraindication within 12 hours of the onset of the MI.
8. Coronary angiography and revascularization wih PTCA or CABG if the patient is in shock, has a large infarct, or is unresponsive to medical therapy. Catheterization may also be required for the diagnosis and treatment of complications.

3. Spasm (Prinzmetal's or Variant Angina)

► H&P Keys

Anginal-type chest pain, typically occurring at rest. High percentage of patients with isolated coronary spasm are cigarette smokers or cocaine abusers. Patients tend to be younger than those with exertional angina. The cardiac exam is usually normal.

► Diagnosis

ECG typically shows ST elevation during symptomatic periods.

► Concept and Application

Most patients have CAD, and spasm occurs in close proximity to a diseased segment, although approximately one-third have angiographically normal coronaries. Diseased coronary vasculature loses the ability to manifest endothelial-dependent vasodilation and may react paradoxically to what are normally vasorelaxant stimuli.

► Treatment Steps

1. Avoid smoking and cocaine.
2. Nitrates and calcium channel blockers are usually effective; the effect of β-blockers is unpredictable; in some they can precipitate spasm.
3. α-Adrenergic β-blockers (e.g., prazosin) can be helpful.

B. Chronic

1. Stable Angina Pectoris

► H&P Keys

Episodic chest discomfort, often described as heaviness or squeezing lasting 1–15 minutes. Pain may radiate to the jaw, neck, shoulder, or the left arm. Symptoms typically are precipitated by exertion, cold weather, or emotional upset, and relieved by rest. Family history of premature CAD, diabetes, hyperlipidemia, hypertension, cigarette smoking. Exam may be normal, but if the patient is examined during an ischemic episode, an S_3 or S_4 may be heard.

► Diagnosis

ECG may be normal if patient is asymptomatic, but evidence of a prior MI or ischemic ST and T wave changes may be noted. ETT, pharmacologic stress testing, and exercise echo are useful.

► Disease Severity

Global ECG changes suggest multivessel CAD. Quantitation of ischemic burden can be accomplished with perfusion imaging. Coronary arteriography documents presence, extent, and severity of CAD and suitability for revascularization.

► Concept and Application

Angina results from myocardial oxygen supply–demand imbalance. Obstructive coronary lesions limit the blood flood to myocardial segments. Dilatation of myocardial arteriolar resistance vessels mitigates ischemia, but this mechanism eventually is inadequate as stenosis severity increases. Angina can be precipitated in the absence of CAD in patients with augmented myocardial oxygen demand, e.g., thyrotoxicosis, hypertrophy, or aortic stenosis.

► Treatment Steps

1. Reduction of ischemic precipitants and treatment of coexisting illnesses (e.g., hyperthyroidism) that increase oxygen demand.
2. Sublingual NTG is valuable for prompt relief.
3. Aspirin reduces the risk of future adverse events by 33%.
4. Lipid-lowering therapy with HMG CoA reductase inhibitors also substantially reduce the risk of future fatal and nonfatal MI.
5. β-Blockers stimultaneously improve angina and ischemia while also preventing MI and death.
6. Calcium channel blocking drugs also relieve ischemic symptoms.
7. PTCA and CABG relieve symptoms and improve outcomes in appropriate patients.

2. Silent Ischemia

► H&P Keys

Patients are asymptomatic, and the physical exam is usually normal.

► Diagnosis

Holter monitoring and ETT can uncover ST segment changes indicative of ischemia.

► Disease Severity

Degree of ST segment depression and number of leads involved can suggest disease extent; nocturnal ST segment depression often means multivessel CAD.

► Concept and Application

Most patients with symptomatic angina have episodes of silent ischemia. Silent ischemia is more common in diabetics, who often have an abnormality in pain perception.

► Treatment Steps

1. Medical therapy, as outlined for angina pectoris, is often used; however, specific therapy depends on extent of disease, patient's age, occupation, etc.
2. Most patients develop symptomatic angina before additional adverse events occur.

II. HEART FAILURE (HF)

A. Left-Sided

1. Low Output

► H&P Keys

History may elicit cause, e.g., CAD, prior MI, hypertension, or valvular disease. Symptoms: fatigue, weakness, reduced exercise tolerance, exertional or rest dyspnea, paroxysmal nocturnal dyspnea (PND), orthopnea, nocturia. Physical findings: displaced cardiac impulse; S_3 murmurs, especially MR; rales; rarely Cheyne–Stokes respiration.