Figure captions

Fig. 1. Demographics and outcomes of patients who present to emergency departments in the U.S. with chest pain.

Fig. 2. Time line for chest pain evaluation centers. From Perfecting MI Ruleout. Best Practices for emergency evaluation of chest pain. Cardiology Preeminence Roundtable, Washington, DC, 1994, used with permission.

Fig. 3. Plot of the appearance of cardiac markers in blood vs time after onset of symptoms. Peak A, early release of myoglobin or CK-MB isoforms after AMI; peak B, cardiac troponin after AMI; peak C, CK-MB after AMI; peak D, cardiac troponin after unstable angina. Data are plotted on a relative scale, where 1.0 is set at the AMI cutoff concentration.

Fig. 4. Pathophysiology of acute coronary syndromes. A. Cross-section of coronary artery showing the presence of a lipid-filled plaque with a thin fibrous cap. B. Rupture occurring at the shoulder region of the plaque, which is an area of vulnerability due to high circulatory shear stress. C. Exposure of plaque core elements propagates thrombus formation. D. Totally occlusive thrombus causing AMI. Reprint from Clinical Laboratory News, Jun 1998, page 12-14, with permission from the American Association for Clinical Chemistry.

Fig. 5. Summary of pathophysiologic events in acute coronary syndromes. Reprint from Clinical Laboratory News, Jun 1996, poster insert, with permission from the American Association for Clinical Chemistry.

Fig. 6. Cutoff concentration for use of a non-specific marker such as CK-MB have traditionally been set to differentiate between patients with unstable angina and AMI. Use of a biochemical marker that is highly specific for cardiac injury enables the selection of two cutoff concentrations: differentiation between unstable angina vs. AMI, and stable angina vs. unstable angina. Used with permission from Wu AHB, Clin Chim Acta 1998;272:11-21.

Fig. 7. A. List of biochemical events occurring after total occlusion of a coronary artery. B. Possible routes for release of markers from tissue to blood. C. Release pattern of marker vs. time after onset of infarction. Used with permission from Hearse DJ. Cellular damage

during myocardial ischaemia: metabolic changes leading to enzyme leakage. In: Hearse DJ, de Leiris, eds., Enzymes in cardiology. Diagnosis and research. Chichester: Wiley, 1979:4-14.

Fig. 8. Relative response for troponin assays to prepared samples of cTnI.

Fig. 9. A. National Heart Attack Alert Program, 60 minutes to Treatment Working Group “Door to Drug” program. B. NACB "Arm to Report" recommendation for a 1-hour turnaround time for collection, transportation, analysis, and delivery of results for acute cardiac marker testing.