Patients presenting with chest pain are a common daily occurrence in emergency medicine. Some authors say that as many as 10% of patients have chest pain as their presenting complaint. Some of these patients will have serious, life threatening causes of their pain, such as an acute myocardial infarction (AMI), unstable angina, pulmonary embolus, aortic dissection, pneumothorax or Boorhave’s. Delay in diagnosis and appropriate treatment can lead to increased morbidity and mortality in these patients. However, many patients with chest pain may have less serious disorders, for example, costochondritis, esophageal pain, gastroesophageal reflux, mitral valve prolapse or referred GI pain. It is obvious that correctly sorting out which patient has life threatening illness from those with minor illness is a high priority for the emergency physician. Unfortunately, this is more easily said than done. Errors in the diagnosis of chest pain account for more than 20% of all medical malpractice judgments rendered against emergency physicians. These errors also account for real morbidity and mortality in our patients. These suits are not nuisance suits but usually involve death or significant disability for the patient and enormous impact on the family.

Why do we fail to recognize serious illness in the patient with chest pain? First of all, multiple organ systems may be the source of the pain because innervation of the thoracic organs is complex and vague. Pain that is felt in the chest may be caused by organs that are within, around or neurologically connected to the thorax. This may include the heart, the lungs, upper GI tract, esophagus, and also muscle, bone, fascia, skin, abdomen and cervical spine. Frequently, chest pain is also a complaint secondary to anxiety. Secondly, the pain sensation from the organs in the thorax is poorly localized. Multiple organs are innervated through the same neuronal network so that the source of the pain is never precisely perceived. Thirdly, thoracic visceral input may converge on somatic afferent fibers in the spinal gray matter. The cerebral cortex interprets the pain as pain in that somatic location. This is referred pain. Thus, the pain sensation may be at a distance from the actual organ causing the original pain. Lastly, because the pain is vague and poorly localized, it requires interpretation by the patient. How the pain is described is dependent on the patient’s social background, prior experience and communication skills. For example, if I took a pin and touched the cornea of several patients (a neurologically richly innervated area) every one would immediately be able to localize the pain in milliseconds, withdraw, and all would describe that pain almost identically. They would tell me that the pain was sharp, like a pin, and all would describe the area of the cornea touched with accuracy. On the other hand, if I took that same pin and stuck it in the myocardium, some would describe a dull ache, some would have arm pain, some would be short of breath, some would have “indigestion” and several would have no pain at all. Instead of a response in milliseconds, there may be no symptom complaint for several minutes. I would expect several patients to think they are having a life threatening illness and others to be completely unconcerned. This is the problem. Identical pathology in different patients produces varying and confounding responses.

Furthermore, patients get very angry at physicians if they miss serious pathology, especially in the chest. An identical pain, interpreted as innocuous when experienced in another part of the body, may seem life threatening when felt in the chest. Patients are trained by the medical profession and the media to take all chest complaints seriously. Look at the magazines in the check out lane at the grocery store the next time you are there. There is bound to be an article addressing the “10 most common symptoms of heart attack” or some related topic. Citizens are bombarded with information that tells them that any pain in the chest may be a heart attack. Remember the TV commercial- “Thank goodness, my doctor said Mylanta” in an elderly male with chest pain and anginal symptoms! Patients and their families are unforgiving when you miss the diagnosis. We know that about 4% of patients who are having heart attacks are sent home from the ED after being examined. 26% of those patients sent home will die from their MI. Is this just bad judgment from emergency physicians? In the majority of those patients sent home from the ED, MI was never seriously entertained as a diagnosis. The patient’s description of the pain never elicited suspicion by the physician that the pain was myocardial. In other words, what the patient complained about never raised the question of myocardial disease in the physician’s mind. Since the diagnosis was never considered, the appropriate diagnostic modalities were not ordered. The physician was completely blindsighted. When the patient
suffers injury, they are not only angry, but they sue. These are often young patients, in their high earning capacity years with dependent children. They rightfully reason that they went to the ED, saw a competent physician in a hospital with a good reputation, waited a long time, may have had multiple tests, were charged “an arm and a leg” for their care and were assured that they were OK. Now several days later they have a major cardiac complication from the “missed” MI. Their anger is justified. What would you do?

All the above is to help you see that the history in the patient with chest pain may be very misleading. It is true that many patients with myocardial ischemic syndromes, for example, will have chest pain. Often they describe it in classical terms, a pressure like band around the chest, causing shortness of breath and radiation to the arms, neck or jaw and exacerbated by exertion. It is unfortunately also true that many patients will have no pain at all and suffer a “silent MI”. Likewise, in the elderly, chest pain is increasingly less specific with age. Patients older than 85 will have no pain in over 60% of myocardial infarctions. As age increases, symptoms like “weakness”, acute confusion, syncope and stroke become more common and chest pain and diaphoresis become less common. Good and competent physicians will, and do, send patients home with MI. Experience and evidence dictates that anyone who cares for a sufficient number of patients, will eventually send home a patient with an MI. The resident, who comes to the ED to evaluate a patient and says, “thank goodness, I have never sent home a patient with an MI,” has just declared that he/she is an inexperienced physician!

What can we learn from the history now that we know it may be misleading and unreliable? Among patients who complain of pressure like chest pain, 24% suffered AMI and another 30% met criteria for unstable angina. Of patients who describe “burning” chest pain or “indigestion” in the chest often associated with GI complaints, especially reflux, 23% experienced AMI and 21% had unstable angina. Clearly a description of burning chest pain and pressure-like pain are equally worrisome. The intensity of the pain is not related to the severity of the disease. In other words, you can not speculate that the patient with severe symptoms will have more myocardium at risk that those with milder symptoms. The location of the pain and quality of the pain are likewise not related to the amount of myocardium at risk. However, only 5% who had “sharp” pain proved to have an MI. Reader, beware! In some parts of the country “sharp” means the quality of the pain- a sharp, stabbing or lancinating pain. Very few of these patients may have an MI. In Georgia, “sharp” generally means a “bad” pain- a severe pain. A significant number of these patients have life threatening disease! Since the pain of MI is usually diffuse and poorly localized, a well localized pain that is “sharp” and lasts only seconds is very unlikely to be of life threatening potential. When there is substernal chest pain accompanied by discomfort in the neck, jaw, shoulder or arm, the likelihood of MI increases three or fourfold. However, remember, than any organ in the chest or upper abdomen can refer pain to these locations. The conclusion- although pressure-like discomfort and heaviness is more suggestive of myocardial ischemia, the absence of that description or the presence of a more typical GI or musculoskeletal pain does not exclude the diagnosis of ischemic heart disease. The esophagus may generate pain indistinguishable from that of the myocardium and is the most commonly confused organ system when these patients are admitted.

The presence of associated symptoms may also be helpful, especially the presence of dyspnea. In patients with an MI, 14% have dyspnea as their sole complaint and in 50% it is an associated symptom. Nausea and diaphoresis may also be associated but with a lower predictive value. It is often said that if the patient is sweaty, the doctor should be sweaty. There is some truth in this. “Pleuritic chest pain,” or to use a better term “respirophasic” (pain that increases with respiration), is also nonspecific. Respirophasic pain can be attributed to pain from pleural, pulmonic, pericardial or a musculoskeletal source. Many physicians attempt to use a therapeutic challenge to distinguish myocardial from esophageal pain. These are to be discouraged. Patients with esophageal spasm may experience relief with nitroglycerin and an equal number of patients with myocardial and esophageal disease gain relief with antacids and topical anesthetics.

Physicians often use the presence of “risk factors” to aid in their disposition of the patient with chest pain. They reason that if there are multiple risk factors present, coronary artery disease will be more likely as a diagnosis. This is dangerous for several reasons. First, if the patient has a history of coronary artery disease or MI, their risk is 100%. They have the disease- you don’t have to be a rocket scientist and go into more detail. Secondly, many physicians use risk factors inappropriately. Risk factors compare different cohorts of patients relative to the cohort risk of coronary artery disease. Risk factors are termed...
“pseudodiagnostic criteria” in emergency medicine. Risk factors are derived from population statistics and epidemiologic data and have little bearing on whether or not the patient in front of you is having an MI. If the patient in front of you has a pulmonary embolus or thoracic aortic dissection, documentation of risk factors are unnecessary and may potentially delay diagnosis or life saving treatment. Risk factors can clearly not exclude any of the life-threatening causes of chest pain. Nevertheless, in most cases, documentation of a family history of CAD (close male relative with CAD before the age of 65), hypertension, diabetes, smoking and elevated cholesterol is reasonable for completeness if you are not using them for decision making in individual cases. It is true that uncontrolled hypertension can lead to end organ disease and uncontrolled hypertension increases myocardial oxygen consumption and may be implicated in myocardial ischemic syndromes. Also, patients with diabetes have a high propensity for coronary artery disease. Patients who are diabetic for greater than 5 years should be assumed to have some degree of coronary artery abnormality and patients who have M.I.’s and diabetes have a poorer outcome than the nondiabetic population. Cigarette smoking causes micro-ischemia of the myocardium that may persist for several hours. This is like a stress test of the myocardium every time you smoke. However, unfortunately, the presence or absence of one or several of these risk factors can not predict whether or not the patient in front of you right now is having a myocardial ischemic syndrome, dyspepsia, dissection of the aorta or an anxiety attack.

The physical exam in chest pain syndromes is of limited usefulness. Only a small minority of patients will have a physical finding that either eliminates or enhances the diagnosis. Tachycardia, hypotension and diaphoresis are a sign of “badness” and should alert the physician to be diaphoretic and tachycardia as long as the patient is the same. In fact, no patient should ever be considered for discharge from the ED with abnormal vital signs that are unexplained. However, none of these are either sensitive or specific as diagnostic criteria. The appearance of the patient may be the single most important finding. An apprehensive, anxious, diaphoretic patient with a sense of impending doom often has life-threatening illness. A new murmur may suggest papillary muscle dysfunction, but since emergency physicians do not provide continuity care, they are usually unaware if the murmur is new or not. An S3 or S4 may be observed but is likewise nonspecific and of questionable usefulness. In pulmonary embolism, a normal exam is the rule. Physical examination of the lower extremities for DVT is unreliable and the pulse oximetry and ABG may be normal or only slightly suggestive of the actual diagnosis. There are no confirmatory findings in esophageal reflux disease that reliably differentiate it from ischemic syndromes. Musculoskeletal chest wall pain can be reproduced by palpating the chest wall. Unfortunately, a large proportion of the population has chest wall tenderness on palpation. As many as 50% of normal individuals will complain of chest tenderness on palpation and 15-50% of patients with an acute MI will have chest wall tenderness that partially or completely reproduces the pain they are experiencing from their myocardial ischemic syndrome. Using the presence of chest wall tenderness to diminish the diagnostic likelihood of myocardial ischemia can be disastrous!

The single most important diagnostic test for the patient with chest pain is the EKG. Its predictive value is greater than that of the history and the physical exam. The American College of Emergency Physicians has developed a clinical policy that recommends an EKG be obtained on almost all individuals presenting to the emergency department over the age of 35 with any symptom complex that may be suggestive (even remotely) of a myocardial ischemic syndrome. Thus any patient with chest pain, dyspnea, stroke, syncope, confusion etc. will normally get an EKG. If there is recurrence of chest pain while the patient is in the ED or if there is a decline in the patient’s condition the EKG should be repeated. What actually constitutes an EKG that is diagnostic of an AMI is more controversial than you might imagine. Numerous EKG findings have been used as criteria for the diagnosis of AMI. Large thrombolytic trials have used the GISSI criteria which have a specificity of 94%. The criteria are 1) ST segment elevation or depression of 1 mm or greater in any limb lead or 2) ST segment elevation of 2 mm or greater in any precordial lead. Unfortunately, the sensitivity of these criteria even in patients with a strong clinical history is only about 40% in those proven to have an AMI. New ST segment elevation of 2 mm or greater in two contiguous leads is the standard criteria for initiation of thrombolytic therapy. Some will also add the development of a new bundle branch block as thrombolytic criteria along with the ST segment elevation. Of note is that some patients will have ST segment depression with their AMI. ST segment depression is not included as thrombolytic criteria. These patients have an increased morbidity and mortality with thrombolytics for an unexplained reason and thrombolytics should not be given. Also noteworthy is that not all patients having new ST segment
elevation are having an AMI. Clearly pericarditis and thoracic aortic dissection can cause elevated ST segments as can a ventricular aneurysm. Anticoagulation in these patients is disastrous. A good rule of thumb is that if you think you have thrombolytic criteria- think twice, act once. Consider everything before acting- but then act. Don’t be paralyzed by fear or uncertainty- but do take the above into consideration.

Unfortunately, a significant number of patients having an MI will have either ST segment depression or a normal EKG. Although, it is true that most patients who have an MI will show some EKG abnormality sometime during the course of their illness, unfortunately, those abnormalities may not be present when they are in the ED. Across the board, up to 20% of patients who have an MI may have a perfectly normal EKG. Normal EKGs are repeated in up to 50% of patients who present very early, within the first minutes to hours, to the ED with their ischemia/infarction. The EKG, although it is the best criteria we have, is far from perfect for several reasons. First, unstable ischemia syndromes have rapidly changing demand and supply characteristics. A single EKG rarely tells the whole story. A patient with unstable angina may have a normal EKG one minute and a very different tracing the next minute. Secondly, the standard EKG is limited because of its lack of perfect detection in areas of the myocardium it samples. Small areas of ischemia or infarction may not be detected. Conventional leads do not sample the right ventricle or the posterior or lateral walls of the ventricles. Additionally, AMI’s in the area of the circumflex artery are likely to have a nondiagnostic EKG. A third reason why the EKG is limited is that some baseline abnormalities of the EKG make interpretation difficult to impossible. Baseline findings like early repolarization, left ventricular hypertrophy, bundle branch block, arrhythmias and prior Q waves may mask zones of infarction. Lastly, there may be disagreement in interpretation between readers. One man’s ST segment elevation consistent with ischemia/infarction may be another man’s nonspecific ST-T segment abnormality. However, in a study of patients sent home from the ED with M.I.’s who had EKG’s in the ED, 23% were found to have EKG diagnostic criteria consistent with AMI on review. Clearly, emergency physicians must make sure that their EKG reading skills are honed to perfection before making important decisions about patient care based on their EKG interpretative skills.

The chest x-ray is commonly ordered in patients with chest pain but is only of limited value. In myocardial ischemic syndromes the chest x-ray is normally noncontributory in making the diagnosis. In patients with pulmonary embolus the chest x-ray is usually normal or demonstrates minimal nondiagnostic findings. A V/Q scan or pulmonary arteriogram will be necessary to make the diagnosis. Even in patients with chest wall trauma, rib fractures are commonly missed the first few days after injury and may be demonstrated only as they show calcification secondary to healing. Pneumonias can be demonstrated on CXR but are usually anticipated by the history and the finding of rales or rhonchi on the physical exam and is rarely a surprise for the mature physician.

Aortic dissection may be suspected by a wide mediastinum but this finding alone is nondiagnostic and must be followed by an trans-esophageal echo or an aortogram. The take home lesson is that the CXR, while considered standard practice for the patient with chest pain, is rarely diagnostic or of help in defining unexpected conditions.

Up to this point, we have seen that the history and physical exam may be helpful at times and misleading other times. We have seen that the chest x-ray is unlikely to be helpful and the EKG, while very helpful if it shows the characteristic changes of infarction or ischemia, may also be normal in the presence of an infarction or unstable angina. The “gold standard” for the diagnosis of infarction is the characteristic rise and fall of the cardiac enzymes. The best marker we have is the creatinine kinase (CK) and its MB fraction (CK-MB). This enzyme is released from necrotic heart muscle and enters the circulation through the lymph system. The CK and CK-MB may be detectable in the serum as early as 4 hours post infarction and will peak 12 to 20 hours post infarction. The CK-MB is considered positive for myocardial muscle necrosis if the fraction is greater than 2-3 % of the total CK. The CK, while it is the best marker we have, is of little use in the emergency department for several reasons. Generally, we see patients early before the CK rise and since we are a time limited specialty and generally do not follow patients or admit them, we can not benefit diagnostically from the longitudinal rise in the CK-MB. It is not uncommon in the ED in the patient with an MI to have a normal CK and CK-MB only to show the rise after admission. Another limitation is that since CK is released only from necrotic cells, it is not helpful in the diagnosis of unstable angina when muscle necrosis has not yet occurred. In other words, it will not help us predict which patients with coronary artery disease, but without infarction, will have complications hours or days from their visit.
to the ED. Also improving our tests to increase the sensitivity of detection of CK and CK-MB in serum are limited in that, no matter the sensitivity of the test, there is time required for the muscle cell to die and for that biochemical to enter the general circulation before we can detect it. Tests that detect the enzyme earlier will be helpful, but the problem of detection very early in MI and detection of the patient with unstable angina remain. Other biochemical markers for myocardial muscle necrosis include LDH and isoenzymes which rise 8-12 hours after MI and peak 72-144 hours after MI share the same limitations as the CK. Troponin T and I are proteins specific for myocardial cells and are reported to be helpful in the early detection of the MI patient, however, clinical experience is still too limited to comment on its ultimate place in our diagnostic armamentarium.

What should now be apparent is that the diagnosis of the cause of chest pain in the emergency department is fraught with difficulty and uncertainty.

The history can be misleading, the physical exam nonspecific and all ancillary testing normal and the patient can still harbor an acute life threatening illness from which he/she will suffer significant morbidity or mortality is the succeeding hours or days. The prudent physician knows when to trust data and when to suspect data. It will be the wise physician who is cautious in the approach to the patient with chest pain. A physician who says that he/she has never sent home a patient with unstable angina or an MI or pulmonary embolus is most likely a physician who is relatively inexperienced and has not had a great deal of patient contact. The very nature of the patient who presents to the emergency department with chest pain makes eventual error almost inevitable.
Annotated Bibliography of Recent References on
Evaluation and Treatment of the Patient with Chest Pain
(Not in any order)

1. GISSI, Lancet 1986;1:397-402. (STK reduced mortality in acute MI)
   (Timolol improves long term survival and decreases reinfarction rates)
3. BHAT JAMA 1981;246:2073-2080
   (Propranolol shows a 26% reduction in mortality after MI)
   (Acebutolol reduces mortality by 58% 1 year after MI)
5. Science 1983;220:1181-1183
   (rt-PA can selectively lyse coronary thrombi improving myocardial perfusion)
   (Metoprolol reduced mortality by 13% as well as enzyme and ECG evolution)
7. VA Cooperative Trial JAMA 1973;225:724-9
   (Anticoagulants reduced systemic thrombi after MI but not mortality)
8. Circulation 1986;74;III-1-10
   (Embolization prophylaxis recommendation for patients for large anterior M.I.'s)
   (Use of intracoronary STK in Acute MI)
   (Compared STK and TPA- TPA had much higher incidence of reperfusion but there was no
   difference in hospital mortality)
   (Patients given TPA have a 75% patent coronary artery- no change in LV function at 7 days
   between those with and without TPA)
   (TPA reduces mortality and reinfarction compared to placebo)
   (STK verses ASA, neither or both- STK and ASA used alone or both reduced mortality at 5
   weeks compared to placebo)
   (TPA reduces mortality early and reduces 1 month mortality over placebo)

17. AIMS (APSAC Intervention Mortality Study) Lancet 1988;ii:545-549
   (Overall reduction in mortality 47% but reinfarction was common)

   (Compared TPA and STK- reperfusion better with TPA but reocclusion higher so that the net result was the same with both drugs.)

19. TIMI 2 (Thrombolysis in MI) JAMA 1988;260-2849-56
   (Immediate coronary arteriography or angioplasty provides no advantage)

   (Angioplasty can be safely deferred until patients have recurrent ischemia. Patients given IV metoprolol verses oral drug- patients given Beta blockers had lower incidence of ischemia and reinfarction- patients treated within first 2 hours and low risk patient benefited the most)

   (STK verses TPA using LV function as end point- EF the same, reinfarction the same, patency the same but mortality lower with TPA at 30 days and 9 months)

   (All patients got standard therapy for MI plus STK with or without heparin or TPA with or without heparin- No advantage for subQ heparin)

23. SCATI (Subcutaneous calcium-heparin in acute MI) Lancet 1989;182-186
   (Mortality was lower in heparin and STK group compared to controls. Also less thrombus in heparin group by echo)

24. HART (Heparin Aspirin Reperfusion Group) J Am Coll Cardiol 1990;15 (suppl A):64A
   (Patients either received heparin + TPA or ASA+TPA,-heparin and TPA lead to better patency at day 1)

25. 2nd ACCP Conference on Antithrombotic Therapy Chest 1989;95:73S-97S
   (No recommendation on which agent to use, recommends use of various adjunct agents, e.g ASA, Beta Blockers, NTG etc.)

26. ISIS-1 Lancet 1986;2:57
   (Atenolol decreased mortality at 7 days by 15%)

   (NTG reduces death rate in acute MI)

   (NTG reduces infarct size by 27%, infarct expansion reduced by 87%, thrombus formation by 77%, cardiogenic shock by 67% and inhospital mortality by 50%)

   (Magnesium reduces overall mortality by greater than 50% and reduces arrhythmias by more than 50%)

   (Calcium Channel Antagonists and Non Q Wave MI)

   (Effects of Diltiazem on non Q wave MI- No difference on mortality but a decrease incidence of reinfarction- those with diltiazem had a high incidence of adverse effects)

32. HINT Research Group Br Heart J 1986;56:400-413
   (Trial of Ca Channel antagonists- verapamil and nifedipine- with unstable angina more likely to have an MI or recurrent ischemia with Ca Channel antagonists than with metoprolol)
Danish Verapamil Trial; Norwegian Nifedipine Multicenter Trial; TRENT Study- (all say the same- verapamil or nifedipine either have no effect or an increased mortality on acute Q wave MI)

(Ca Channel Blockers do not reduce risk of infarction of death in acute MI)

Arch Intern Med 1989;149:1669-1677 (Ca Channel antagonists are contraindicated in patients who have acute MI or in patients with LV dysfunction)

Multicenter Diltiazem Postinfarction Research Group  N Engl J Med 1988; 319:385-392  (No decreased mortality or cardiac events in patients on long term diltiazem after MI- increased mortality in patients early on and in patients with pulmonary congestion)

JAMA 1988; 260:1910-1916  (Lidocaine can reduce the odds of ventricular fibrillation by 1/3. There is no beneficial effect on mortality- but mortality is increased by 1/3 in the lidocaine group)

Am J Cardiol 63:772 1989  Comparison of clinical presentation of AMI in patients older than 65. Multicenter Chest Pain Study (Older you are, the less typical your presentation is for MI)

Am J Cardiol 1987;60:219-214  (Study of characteristics of patients with MI who are sent home. Miss patients who are having pain at rest. 50% of patients with I sent home could have been diagnosed if physician had better EKG reading skills)

N Engl J Med  Vol 327 No 3 Pg 175  Use of ASA in Ischemic Heart disease
(a review of the literature)

N Engl J Med 1988 :319:1105-11  (Montreal Heart institute study of heparin in unstable angina) Aspirin, heparin or both to treat acute unstable angina  (both aspirin and heparin are associated with a reduced incidence of AMI in unstable angina.  Trend favors heparin)


LIMIT-2   Lancet 1994;343:816-19
(Magnesium given before thrombolytics decreases mortality and decreases LV dysfunction)

Clinical characteristics and outcome of AMI in pts with initially normal or nonspecific EKG.  Am J Cardiol  1989;64:1087-92.  (Pts had lower CPK and mortality was lower)

Clinical characteristics and natural history of Pts with AMI sent home from the ED.  Am J Cardiol 1987;60:219-224.  (Pts with missed AMI younger, had less typical symptoms, less likely to have prior ASCVD and had normal EKG.  Mortality higher than controls.  50% could have been diagnosed if physician had better EKG reading skills)

(25% of pts with missed MI could have been diagnosed if EP had better EKG reading skills.)

(good review article on current state of knowledge on unstable angina)

Reperfusion in AMI, Mayo clin Proc  65:549-564, 1990
(good review article on current state of knowledge on reperfusion injury after thrombolytics)

(This is an annotated bibliography of articles on Mag in AMI)
49. Acute MI: Initial manifestations, management and prognosis

50. The Golden Hours of the MI: Nonthrombolytic therapy
   (This is a review article covering lidocaine, nitrates, Ca Channel blockers, B Blockers, Aspirin)

51. ACC/AHA Guidelines for the Early Management of patients with AMI
   Circ 1990;82(2):664-707. (Exhaustive review of the literature on all aspects of the care of patients
   with AMI- a must read for all emergency physicians)

52. Silent myocardial ischemia
   (Review of current understanding of silent ischemia)

53. Hostility Predicts Restenosis after PTCA
   Patients who had higher hostility index had > # restenosis of coronary artery after PTCA than those with lesser
   hostility index.

54. Effect on Outcome of the Presence or Absence of Chest Pain at the initiation of TPA in AMI
   Am J Cardiol 73:729-736, 1994
   (Pts with EKG evidence of AMI but without chest pain still benefit from early initiation of TPA)

55. Influence of pseudodiagnostic information on the evaluation of ischemic heart disease
   Green and Yates
   (Physicians in ED rely heavily on epidemiologic factors to make disposition on patients with chest pain although epidemiologic criteria on
   populations bear no relationship to individual patients risk factors)

56. An evaluation of technologies for identifying acute cardiac ischemia in the emergency department:
   Executive summary of a National Heart Attack Program Working Group Report, Selker HP, Zalenski RJ, Antman EM, et al
   Annals of Emergency Medicine, Vol 29, Number 1, Jan 1997, pages 1-88
   (Literature review with recommendations on the current state of understanding of diagnostic modalities for acute coronary event. Review includes, standard EKG, Prehospital EKG, continuous EKG, nonstandard EKG and body mapping, EKG stress test, ACI predictive instrument, ACI-TIPI, Goldman chest pain protocol, computer based decision aids, Creatinine kinase, other biochemical tests, echocardiogram, thallium scanning, sestamibi and technecium 99m scanning- conclusions are that standard EKG is useful as is the ACI predictive instrument. Less important is the prehospital EKG, ACI-TIPI, Goldman Chest Pain Protocol)