# An Approach to the Initial Care of Patients with Chest Pain in an Emergency Department Located in a Non-Cardiac Center

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## Abstract

Emergency department management of chest pain is a common, with potentially serious problem. With multiple risk factors, target groups, and care pathways, the myriad etiologies of chest pain can be perplexing. This article describes some of the diagnostic and risk stratification scenarios currently used in departments which do not have percutaneous cardiac intervention capabilities.

## Introduction

Patients who appear in the emergency department, urgent care centers, or offices who state that their chief complaint is chest pain deserve an appropriate medical evaluation. In the US, there are approximately six to seven million visits per year to the emergency department or chest pain units due to a chief complaint of chest pain, of which 1.6 million are admitted with a diagnosis of acute coronary syndrome (ACS). Of these, 25 percent (400,000) meet criteria for acute myocardial infarction (AMI) by EKG criteria of ST elevation (STEMI), whereas 75 percent meet criteria for the diagnosis of unstable angina (UA) or non-ST elevation MI (NSTEMI).[1-3] This means that the majority of chest pain complaints are not due to ACS. Non-cardiac chest pain (NCCP) is the second most common reason for presentation to the ED and accounts for approximately two to five percent of all visits.[4-5]

Though it is obvious that only a minority of these patients have a coronary artery problem, the tragedy of a missed diagnosis and the resulting medico-legal problem, requires that we should routinely view chest pain initially as an acute coronary syndrome until proven otherwise. Four other critical causes of acute morbidity or mortality follow closely in the differential; they are: pulmonary embolism, aortic dissection, pneumothorax, and pneumonia.[6] Multiple other etiologies are possible and may need to be considered. It is only after an appropriate medical history, a physical exam, and investigational steps are completed that the physician will be able to exclude some of these entities. Often it takes multiple investigations over time to come to an adequate definition of the etiology of the chest pain. Nonetheless, screening for these critical causes of death are paramount for the emergency physician managing chest pain. Finally, remember, causes of chest pain are not limited to the thorax.

## Causes of Chest Pain – The Short List!

**Cardiovascular:**
ACS/CAD/Angina
Anomalous left coronary
Aortic dissection
Aortic stenosis
Arrhythmias
Cardiac tamponade
Cardiomyopathy
Hypertrophic cardiomyopathy
Kawasaki disease
Mitral valve prolapse
Pericarditis/ myocarditis
Valvular heart disease

**Drug abuse:**
Amphetamine
Cocaine
"Diet pills"
Party Drugs (LSD and Ecstacy)

**Gastric:**
Diverticular disease
Esophageal spasm
Esophagitis
Gastritis
Gastroesophageal reflux disease

**Miscellaneous:**
Anemia (including sickle cell)
Herpes zoster
Thyroid/adrenal problems
Toxins

**Musculoskeletal:**
Arthritis
Costochondritis
Fibromyalgic
Muscular strain
Myofasciitis
Trauma

**Psychological:**
Somataform disorder
Anxiety/panic attacks
Depression

**Respiratory/pulmonary:**
Asthma/reactive airway disease
Cancer
Foreign Body
Hypoxia
Pleurisy
Pneumomediastinum
Pneumonia
Pneumothorax
Pulmonary embolism
Tracheitis

## Chest Pain in Children

Widespread public information messages have made people more aware of heart disease in children, so it is not surprising to find many anxious parents camped out in the ED with a healthy, non-toxic child who has a complaint of chest pain. Some of this anxiety is based on media cases involving sudden cardiac death in young children. It should be remembered that in the majority of cases sudden death in children does not include complaints of chest pain. The etiology of sudden death in children has been postulated to be due to structural defects or to arrhythmias that could have its origin in genetic abnormalities of the ion channels that affect the channel structure and function.[7-8] Of all cases of chest pain reported in children only 5% are attributable to a cardiac etiology.[9]

As you begin the evaluation of a child with chest pain consider that in most cases there will be a child attached to two parents. When you remember that the worst nightmare for any parent is the loss of a child, make certain to pay heed to the fact that you may have three patients and not just one.

Start your evaluation by inspecting the vital signs (they are called vital for a reason and the emergency physician will be held responsible for any abnormal vital sign that remains unexplained) making sure to document the four cardinal ones, plus the $O_2$ saturation, as you observe the child. An active, playful child may be the best sign of reassurance that you, the doctor, can obtain, but it is rarely sufficient for the parent. Therefore, you must obtain the complete history of pain documenting the quality, intensity, location, temporal relationship to events, provocative and palliative factors, and associated symptoms. Trauma, including child abuse, must be sought and excluded. Look to the history and review of systems for weight loss, fatigue, headache, syncope, altered mental status, palpitations, pain on exertion, cough, wheezing, vomiting, and recent fevers/URIs that may be associated with myocarditis, athletic heart syndrome, or drug abuse.

Continue your evaluation by performing an appropriate physical exam, concentrating on the cardiovascular, respiratory, gastrointestinal, and musculoskeletal systems. Remember to document your findings ("if you didn't write it, you didn't do it"). The following tests could be performed in order to reassure yourself and the parents and to clarify doubts about the diagnosis: the chest x-ray (PA and lateral) and 12-lead EKG are usually useful, and additional hematologic and chemistry profiles may be appropriate for specific cases.

All chest pain associated with dyspnea, syncope, and palpitations, especially if it occurs during strenuous physical activity, will need to be more thoroughly evaluated and often results in a pediatric cardiologic consultation. But, in the vast majority of cases, at the end of your evaluation you will not find a definite cause for chest pain in children seen in the ED. Be honest with the parents, but always tell them if you do not find any of the serious causes of pain that are associated with acute morbidity or mortality. Consult as needed; follow up is almost always appropriate.

## Chest Pain in Adults

Heart disease remains the leading cause of death in the United States with an estimated direct and indirect cost of approximately $142.5 billion for 2006,[4,10-11] but certainly not all people who arrive in the ED or clinic with the complaint of chest pain are experiencing heart disease. Certainly, as patients become older, more obese, and less active, the risk of developing coronary artery disease and dying from it increases. It is also important to remember that it is not only ACS (whether STEMI/NSTEMI) that kills patients, but other etiologies such as pulmonary embolisms, dissecting aortas, pneumothorax, and pneumonias that may also kill patients.

Pulmonary embolism is a legendary challenge for emergency physicians. The classic description of sudden onset of pleuritic chest pain associated with shortness of breath, abnormal vital signs, and hypoxia is not the most common presentation. Well's criteria is the most widely used method for evaluating patients by stratifying them into risk categories. Low-risk patients currently are being excluded by use of the d-dimer. All others require investigations by ultrasonography and/or CT angiography; various strategies exist for thrombolysis. Patients with aortic dissection are described as presenting with severe tearing or sharp chest pain that is sudden in onset. However, this classical description is unfortunately not always seen. Many patients present with neurological symptoms or syncope as the initial complaint. Hypertension in the elderly remains the main risk group, but those with historical findings such as connective tissue disorders and sudden death in family members are also frequently found in the high-risk group.

Patients with pneumonia should present with fever, cough, and infiltrates on the x-ray; however, it is altogether too common that they present with merely lack of energy, perception of being "sick," and myalgia, especially in the elderly. In addition, there are numerous other causes for chest pain (see Table 1) including patients with palpable discomfort on chest wall examination. Remember that up to 15% of acute MI patients may present with this finding and it certainly does not prevent them from having a concomitant coronary event.[12]

## Scoring Risk in ACS

We are all aware of the "door to diagnosis/drug/transfer guidelines" set forth for patients who state that they have chest pain. At triage, most all hospitals have implemented protocols to stratify patients into high-, moderate-, and low-risk groups, but remember that protocols are not perfect. Our population has dramatically increased its incidence of the obese and morbidly obese (2/3 and 1/3 of the population respectively) and the number of patients with metabolic syndrome has burgeoned. The percentage of children afflicted has increased and will continue to do so. The rate of smoking by females has reached or exceeded that of males and in conjunction with birth control pills and hormone replacement therapy has led to more thromboembolic problems.[13]

There are tools that help in stratifying these patient risks. One of the earliest tools was Rouan's (see below). The Goldman prediction rules have been used by some. Another system has become part of ACLS training over the last decade.

Most of these strategies are based on age, history of previous coronary artery disease, type of pain, EKG findings, cardiac markers, and physical examination findings. Utilizing them will help you determine which patients deserve more complete work ups, monitoring, follow ups, and referrals. It is important to notice that one to four percent of patients with normal EKG findings could have an acute myocardial infarction[14-15] and that cardiac enzymes take from two to six hours to become positive in the majority of cases.[16]

Another tool rating risk assessment came from Braunwald et al in 2000. Its usefulness in the early evaluation of ED patients is somewhat diminished by its complexity and lack of scoring specificity, which seems to target its usefulness to post hoc assessments by consultants rather than the ED physician attempting to stratify the patient.

**Rouan's Decision Rule for Myocardial Infarction**

| Clinical Characteristics (Each count as one point) | |
|---|---|
| Age greater than 60 years<br>Diaphoresis<br>History of MI or angina<br>Male sex<br>Pain described as pressure<br>Pain radiating to jaw, neck, shoulder, or arms | |
| Score | Risk of MI (%) |
| 0 | Up to 0.6 |
| 1 | Up to 3.4 |
| 2 | Up to 4.8 |
| 3 | Up to 12.0 |
| 4 | Up to 26.0 |

Rouan GW, Lee TH, Cook EF, Brand DA, Weisberg MC, Goldman L. Clinical characteristics and outcome of acute myocardial infarction in patients with initially normal or nonspecific electrocardiograms (a report from the Multicenter Chest Pain Study). Am J Cardiology 1989; 64:1087-92.

**Risk Assessment Myocardial Infarction/Unstable Angina/NSTEMI**

| Short-term high risk for death / myocardial infarction in unstable angina/NSTEMI | Short-term intermediate risk for death / myocardial infarction in unstable angina /NSTEMI | Short-term low risk for death / myocardial infarction in unstable angina/NSTEMI |
|---|---|---|
| At least one of: | No high risk features, but must have one of: | No high or intermediate risk features, but may have any of: |
| **History** | | |
| Accelerating tempo of ischemic symptoms in preceding 48 hrs | Prior myocardial infarction, peripheral vascular disease, or cerebrovascular disease<br>Coronary artery bypass grafting<br>Prior aspirin use | None indicated |
| **Character of pain** | | |
| Prolonged ongoing (>20 min) rest pain | Prolonged (>20 min) rest angina now resolved, with moderate or high likelihood of coronary artery disease<br>Rest angina (<20 min) or relieved with rest or sublingual nitroglycerin | New-onset Canadian Cardiovascular Society class III or IV angina in past 2 weeks without prolonged (>20 min) rest pain, but with moderate or high likelihood of coronary artery disease |
| **Clinical findings** | | |
| Pulmonary edema, most likely caused by ischemia<br>New or worsening mitral valve regurgitation murmur<br>S3 or new/worsening rales<br>Hypotension, bradycardia, tachycardia<br>Age over 75 years | Age over 70 years | None indicated |
| **EKG** | | |
| Rest angina with transient ST changes greater than 0.05mV<br>Bundle branch block<br>New sustained ventricular tachycardia | T-wave inversions greater than 0.2mV<br>Pathologic Q waves | Normal or unchanged EKG during chest discomfort |
| **Cardiac markers** | | |
| Markedly elevated (eg. Troponin T or Troponin I >0.1 ng/mL) | Slightly elevated (eg. Troponin T or Troponin I <0.1 ng/mL) | Normal |

From Braunwald E, Antman EM, Beasley JW, et al. ACC/AHA guidelines for the management of patients with unstable angina and non-ST segment elevation myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina). J Am Coll Cardiology 2000;36:1062.

**TIMI Risk Score for Patients with Unstable Angina and Non-ST Elevation MI: Predictor Variables**

| Predictor Variable | Point Value of Variable | Definition |
|---|---|---|
| Age > 65 | 1 | |
| > 3 risk factors for CAD | 1 | Risk factor:<br>• Family history of CAD<br>• Hypertension<br>• Hypercholesterolemia<br>• Diabetes<br>• Current smoker |
| Aspirin use in last 7 days | 1 | |
| Recent, severe symptoms of angina | 1 | > Anginal events in last 24 hrs |
| Elevated cardiac markers | 1 | CK-MB, or Troponin levels |
| ST deviations > 0.5mm | 1 | ST depression > 0.5mm is significant<br>ST elevation > 0.5mm for < 20 minutes is treated as ST-segment depression and is high risk<br>ST elevation > 1mm for more than 20 minutes places these patients in the STEMI treatment category |
| Prior coronary artery stenosis > 50% | 1 | Risk predictor remains valid even if this information is unknown |
| Calculated TIMI Risk Score | Risk of > 1 Primary End Point in < 14 Days | Risk Status |
| 0 or 1<br>2<br>3<br>4<br>5<br>6 or 7 | 5%<br>8%<br>13%<br>20%<br>26%<br>41% | Low<br><br>Intermediate<br><br>High |

Taken from the American Heart Association Guidelines CPR/ECC 2005: Handbook of Emergency Cardiovascular Care for Healthcare Providers. Page 38.

The TIMI scoring scheme shown above is currently in widespread use. It must be remembered that, for each patient, the disease is either present or absent and numbers are only a guide based on group studies. It is certain new guidelines will appear ad infinitum and that the trial attorneys will continue to be aware of their existence.

## Examination of the Adult Chest Pain Patient

Begin your evaluation of the patient with chest pain by visual observation of the patient and inspection of the vital signs. An emergency department patient should by this time be in a room attached to a cardiac monitor. Do not forget to get $O_2$ saturation! The ABCs should be attended to, of course; if the patient is stable, then get a 12-lead EKG, put the patient on oxygen if indicated, give an aspirin dose of 325mg (if not allergic), and start the most important part of your assessment, the history.

The history should focus on the onset and evolution of the patient's chest pain. Document its location, quality, severity (using a 1 to 10 scale), radiation, timing, duration, and aggravating and alleviating factors. Pay close attention to any kind of pain that radiates to the jaw, neck, arms, back or abdomen. Associated symptoms such as diaphoresis, weakness, vomiting, nausea, palpitations, and syncope should draw special attention. By this time, and within the ten-minute framework, a 12-lead EKG should be in your hands and, if no contraindications have been discovered (such as hypotension), sublingual nitroglycerine (0.4mg) should be given to the patient with written orders to repeat every two to five minutes times two if chest pain continues and if the systolic blood pressure (SBP) is above 100mmHg. Alternatively a nitroglycerin IV infusion of 10 to 20 micrograms per minute may be used. Titrate to effect, by increasing 5 to 10 micrograms every five to ten minutes until the desired effect is obtained. Nitroglycerine must be discontinued if the blood pressure drops below 90. If pain persists, the use of morphine is indicated; you can use 2 to 4 mg IV (over 1-5 minutes) every 5-30 minutes until the pain improves or the patient develops problems such as hypotension or decreased respiratory rate.[17] Large bore IVs are suggested, as thrombolytics may be required or bolus infusions for hypotension may be necessary. Normal saline is commonly used on a "keep open" basis; if boluses are necessary, 250 to 500mL amounts are commonly given in sequence to bring the pressure back up.

When inspecting the EKG, look for ST elevation, ST depression, Q waves, T wave changes, and bundle branch blocks. Any patient with chest pain and an EKG showing a left bundle branch block (LBBB) needs scrutiny for an acute myocardial infarction. It is very helpful to locate a previous EKG to inspect

for changes, especially in the bundle branch block patients. Solid evidence as to the utility of strategies used to determine the presence of ACS on top of a LBBB remains elusive. Remember that T-wave changes are early signs of ischemia produced by transient hyperkalemia in the ischemic cardiac muscle. Treatment and disposition of the patient is guided by EKG changes and the patient's history, especially their risk factors. Patients (other than those on cocaine) should be given B-blockers (i.e., within the first 24 hours) if no contraindications such as hypotension, bradycardia, heart block (other than first degree), or severe airway disease/pulmonary edema exists.[17] For example, the patient may be given metoprolol 5mg IV in a two to five minute infusion every five minutes times three while in the ED.

The at risk patient population is then divided on the basis of the EKG evaluation into ST elevation MI's (STEMI), non-ST elevation MI's and unstable anginas (NSTEMI/UA), or non-diagnostic EKG patients still with clinical suspicion of ACS. The level of suspicion of cardiac damage and therapy and management is different in each of the three groups. The third group is divided into high risk, intermediate risk, and low risk groups. Patients can move between groups if conditions change, i.e., there is a change in their cardiac markers, their EKG, or their symptomatology.

The management of patients with ST elevations will vary by institution and often depends on access and availability of percutaneous coronary intervention (PCI). There is persuasive evidence that PCI is more effective than fibromyalgia if it can be accomplished within the 90 minutes time frame.[18] In many parts of rural America, patients with ST elevations will benefit from thrombolytic therapy if PCI is not available within 90 minutes (after making certain to document the presence or absence of contraindications). Remember every minute counts as "time is muscle." The national goal for administration of fibrinolysis is 30 minutes and door to PCI is 90 minutes.[19] Make sure to document any factors that prevent you from achieving these goals. If you are not fortunate enough to be at a cardiac center, then arrange for appropriate consultation or transfer expeditiously. Anti-coagulant, anti-platelet, and fibrinolytic therapy should be considered and discussed with the receiving physician. If appropriate, knowledge of the receiving physician's preferences and standing protocols are quite helpful, although by now the standard of practice allows emergency physicians to deliver such care using widely distributed protocols without direct supervision or specific recommendations by cardiologists.

Most clinicians order the following laboratory and ancillary tests in order to more fully evaluate patients who often have multiple medical problems, are on a myriad of medications, and are unknown to the emergency physician. Such test include: complete blood count, chemistry profile, liver panel, troponin, myoglobin, CK-MB markers, TSH and coagulation studies, though there is abundant evidence that results from these tests rarely change the immediate therapy or management.

Serial EKGs and cardiac markers are standard over a period of time (8-12 hours) although once positive the patient can be stratified and referred. Some institutions use continuous EKG monitoring, though the value is uncertain. Otherwise, the EKGs should be repeated at any time if symptoms warrant, or at least every two to four hours.[2] Of the cardiac markers, myoglobin rise is the earliest to be detected and is very sensitive though not very specific; frequently increases are due to non-specific factors such as renal impairment and muscle contusions. Myoglobin levels may be useful for ruling out acute myocardial infarction if the levels remain normal during the first several hours (4 to 8 hours). Troponins and CK-MB may take more than four hours to rise and may stay positive for quite some time; in the case of troponin up to two weeks. The risk of death due to a MI correlates with the quantitative value of the troponin.[16] After excluding an acute coronary syndrome (i.e., STEMI, NSTEMI/UA) the workup for non-acute coronary syndrome and non-cardiac chest pain can be started. These workups can be accomplished by referral for provocative testing and may take place on an outpatient basis.

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## Sidebar

1. While not all cases of chest pain turn out to be heart attacks, when you are wearing a white coat, treat all chest pain as such during the early management phase.
2. In chest pain, time is muscle, but also, time is money. You are being timed! If you fail to diagnose and treat according to CMS and other standards in the future, funding may be withheld to the institution.
3. As soon as the chief complaint of chest pain is documented, GET INVOLVED! You, as the captain of the ship, will be accountable from the moment that the patient gets into your building. Make sure the staff understands this.
4. Teach and train all health-related personnel in your area to recognize the signs and symptoms of a heart attack. The nurse, respiratory technician, janitor, and receptionist are people who may interface with patients at any time, and when they tell you something, heed their advice. Remember, you may be the new doctor, but the people in your work area will have had many years of experience.
5. When you see a patient who declares chest pain, look, listen, and feel. If you think that the patient is not stable, give treatment immediately; put them on a stretcher, and let them "lie with MONA" (M=morphine, O=oxygen, N=nitroglycerin, A=aspirin).
6. Patients with high-risk scores who have histories of diabetes, hypertension, lipid disorders, and thrombotic events must be evaluated very thoroughly. This means appropriate observation, follow up, including stress tests, 2D-Echos, nuclear medicine scans, consultations, and so forth.
7. Once you start the work up for chest pain, finish it. One test or set of markers is never enough. Medico-legally you will find few experts to testify in your favor if you "rule out" an acute coronary syndrome with a single cardiac marker. If the patient tries to leave, make them sign out AMA and clearly document the steps taken.
8. Know your limits. If you are in a facility (non PCI, non CABG) that cannot manage the problem the patient has, TRANSFER the patient. Do not ignore EMTALA. Many experts urge PCI if access can be accomplished rapidly; most everyone recommends lytics, anti-platelet drugs, and anti-coagulants early. Know your local referral options.
9. Get prepared. Make sure you know what is available in your hospital and where the equipment and medications in your department are. Check the intubation equipment, i.e., light bulbs, and make sure you have the proper blade tube sizes and an assortment of back-up airway devices.
10. Wall-mount the ACS protocols so that you can find them rapidly, check them frequently, and keep your information current. While ACLS may have detractors, it is a good place to start.