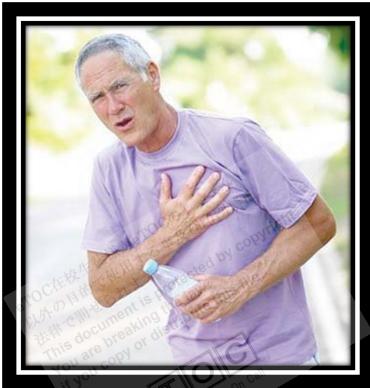




Symptoms of Cardiovascular Disorders

Chest Pain



http://www.belting.org/pics/chest_pain.jpg

Chest pain is a very common complaint. Many patients are well aware that it is a warning of potential life-threatening disorders and seek evaluation for minimal symptoms. Other patients, including many with serious disease, minimize or ignore its warnings. Pain perception (both character and severity) varies greatly between individuals as well as between men and women. However described, chest pain should never be dismissed without an explanation of its cause.

Pathophysiology

The heart, lungs, esophagus, and great vessels provide afferent visceral input through the same thoracic autonomic ganglia. A painful stimulus in these organs is typically perceived as originating in the chest, but because afferent nerve fibers overlap in the dorsal ganglia, thoracic pain may be felt (as referred pain) anywhere between the umbilicus and the ear, including the upper extremities.







Painful stimuli from thoracic organs can cause discomfort described as pressure, tearing, gas with the urge to **eructate**, **indigestion**, **burning**, aching, **stabbing**, and sometimes **sharp needle-like pain**. When the sensation is **visceral** in origin, many patients deny they are having pain and insist it is merely "discomfort."

Etiology

Many disorders cause chest pain or discomfort. These disorders may involve the cardiovascular, GI, pulmonary, neurologic, or musculoskeletal systems.

Some disorders are immediately life threatening:

- Acute coronary syndromes (acute MI/unstable angina)
- Thoracic aortic dissection
- Tension pneumothorax
- Esophageal rupture
- Pulmonary embolism (PE)

Other causes range from serious, potential threats to life to causes that are simply uncomfortable. Often no cause can be confirmed even after full evaluation.

Overall, the most common causes are

Table 1

Some Causes of Chest Pain				
Cause*	Suggestive Findings	Diagnostic Approach†		
Cardiovascular				
¹ Myocardial ischemia (acute MI/unstable angina/angina)	Acute, crushing pain radiating to the jaw or arm	Serial ECGs and cardiac markers; admit or observe		
	Exertional pain relieved by rest (angina pectoris)	Stress imaging test or CT angiography considered in		
	S ₄ gallop	patients with		
	Sometimes systolic murmurs of mitral	negative ECG findings and no		



	regurgitation Often red flag findings‡	cardiac marker elevation Often heart catheterization and coronary angiography if findings are positive
¹ Thoracic aortic dissection	Sudden, tearing pain radiating to the back Some patients have syncope, stroke, or leg ischemia Pulse or BP that may be unequal in extremities Age > 55 Hypertension Red flag findings [‡]	Chest x-ray with findings suggesting diagnosis Enhanced CT scan of aorta for confirmation Transesophageal echocardiography
² Pericarditis	Constant or intermittent sharp pain often aggravated by breathing, swallowing food, or supine position and relieved by sitting leaning forward Pericardial friction rub Jugular venous distention	ECG usually diagnostic Serum cardiac markers (showing elevated troponin with normal CPK level) Transthoracic echocardiography
² Myocarditis	Fever, dyspnea, fatigue, chest pain (if myopericarditis),	ECG Serum cardiac markers





infection

Sometimes findings of heart failure, pericarditis, or both

ESR

C-reactive protein
Usually
echocardiography

GI

1 Esophageal rupture

Sudden, severe pain following vomiting or instrumentation (eg, esophagogastroscopy

or transesophageal echocardiography)

Subcutaneous crepitus

detected during auscultation

Multiple red flag findings‡ Chest x-ray

Esophagography

with water-soluble contrast for confirmation

² Pancreatitis

Pain in the **epigastrium**

or lower chest that is often worse when lying flat and is relieved by leaning forward

Vomiting

Upper abdominal tenderness

Shock

Often history of alcohol abuse or biliary tract disease

³ Peptic ulcer

Recurrent, vague

epigastric or right

upper quadrant

discomfort in a patient

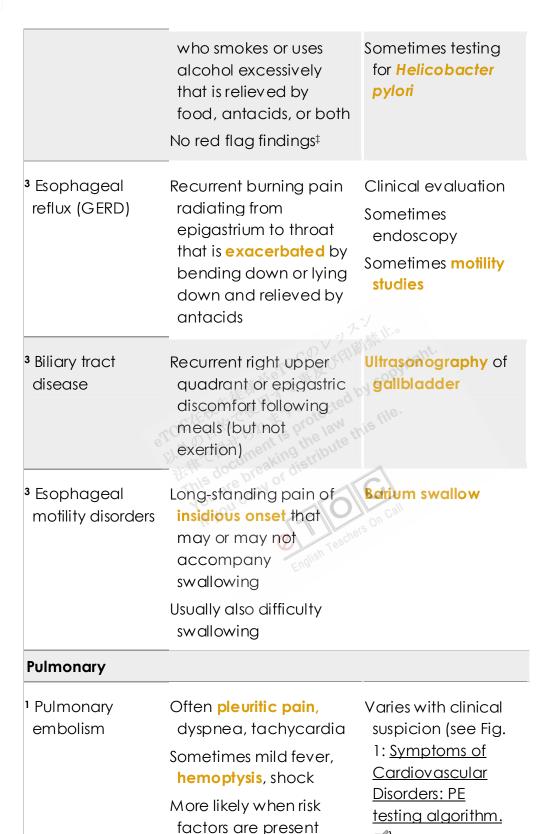
Serum lipase
Sometimes

Sometimes abdominal CT

Clinical evaluation Sometimes

endoscopy





(see Table

S





	2: Symptoms of Cardiovascular Disorders: Clinical Prediction Rule for Diagnosing Pulmonary Embolism)	
¹ Tension pneumothorax	Significant dyspnea, hypotension, neck vein distention, unilateral diminished breath sounds and hyperresonance to percussion Sometimes subcutaneous air	Usually clinical Obvious on chest x- ray
² Pneumonia	Fever, chills, cough, and purulent sputum Often dyspnea,	
² Pneumothorax	Sometimes, unilateral diminished breath sounds, subcutaneous air	Chest x-ray
³ Pleuritis	May have preceding pneumonia, pulmonary embolism, or viral respiratory infection	Usually clinical evaluation
	Pain with breathing, cough	
	Examination	





unremarkable

Other

³ Musculoskeletal chest wall pain (eg, due to

trauma, overuse,

or

costochondritis)

Often suggested by

history

Pain typically persistent (typically days or

longer), worsened with passive and active

motion

Diffuse or focal tenderness

² Various thoracic Variable cancers

Sometimes chronic cough, smoking history, signs of chronic illness (weight loss, fever), cervical lymphadenopathy

Chest x-ray

Chest CT if x-ray findings are suggestive

Clinical evaluation

Bone scan considered for persistent, focal rib pain

Clinical evaluation

³ Herpes zoster

infection

Sharp, band-like pain in the midthorax

unilaterally

Classic linear, vesicular

rash

Pain may precede rash by several days

³ Idiopathic

Various features

No red flag findings[‡]

Diagnosis of exclusion

□ *Seriousness of causes varies as indicated:

□ ¹Immediate life threats.







 ²Potential life threats. ³Uncomfortable but usually not dangerous.
□ †Most patients with chest pain should have pulse oximetry , ECG, and chest x-ray (basic tests). If there is suspicion of coronary ischemia, serum cardiac markers (troponin, CPK) should also be checked.
□ ‡Red flag findings include abnormal vital signs (tachycardia, bradycardia, tachypnea, hypotension), signs of hypoperfusion (eg, confusion, ashen color, diaphoresis), shortness of breath, asymmetric breath sounds or pulses, new heart murmurs, or pulsus paradoxus >10 mm Hg.
\Box S ₄ = 4th heart sound.
 Immediate life threats. ²Potential life threats. ³Uncomfortable but usually not dangerous.

- Chest wall disorders (ie, those involving muscle, rib, or cartilage)
- Pleural disorders
- Gl disorders (eg, esophageal reflux or spasm, ulcer disease, cholelithiasis)
- Idiopathic
- Acute coronary syndromes and stable angina

Evaluation

History: History of present illness should note the location, duration, character, and quality of the pain. The patient should be asked about any precipitating events (eg, straining or overuse of chest muscles), as well as any triggering and relieving factors. Specific factors to note include whether pain is present during exertion or at rest, presence of psychologic stress, whether pain occurs during respiration or coughing, difficulty swallowing, relationship to meals, and positions that relieve or exacerbate pain (eg, lying flat, leaning forward). Previous similar episodes and their circumstances should be noted with attention to the similarity or lack thereof. Important associated symptoms to seek include dyspnea, palpitations, syncope, diaphoresis, nausea or vomiting, cough, fever, and chills.

Review of systems should seek symptoms of possible causes, including leg pain, swelling, or both (deep venous thrombosis [DVT] and therefore possible PE) and chronic weakness, malaise, and weight loss (cancer).

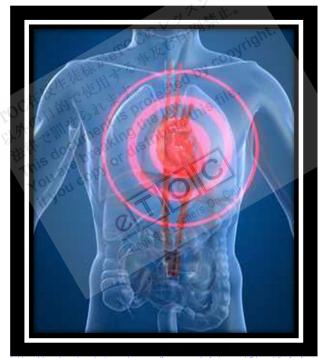




Past medical history should document known causes, particularly cardiovascular and GI disorders, and any cardiac investigations or procedures (eg, stress testing, **catheterization**). Risk factors for coronary artery disease (CAD—eg, hypertension, **hyperlipidemia**, diabetes, **cerebrovascular disease**, tobacco use) or PE (eg, lower extremity injury, recent surgery, immobilization, known cancer, pregnancy) should also be noted.

Drug history should note use of drugs that can trigger **coronary artery spasm** (eg, cocaine, triptans, **phosphodiesterase inhibitors**) or GI disease (particularly alcohol, NSAIDs).

Family history should note history of MI (particularly at an early age) and hyperlipidemia.



http://mykentuckyheart.com/images/pictures/ChestPain.ipa

Physical examination: Vital signs and weight are measured, and **body mass index (BMI)** is calculated. Pulses are palpated in both arms and both legs, BP is measured in both arms, and pulsus paradoxus is measured.

General appearance is noted (eg, pallor, diaphoresis, cyanosis, anxiety).

Neck is inspected for venous distention and **hepatojugular reflux**, and the venous wave forms are noted. The neck is palpated for carotid pulses,







lymphadenopathy, or thyroid abnormality. The carotid arteries are auscultated for bruit.

Lungs are **percussed** and **auscultated** for presence and symmetry of breath sounds, signs of congestion (dry or wet rales, rhonchi), consolidation (**pectorilloquy**), **pleural friction rubs**, and **effusion** (decreased breath sounds, dullness to percussion).

The cardiac examination notes the intensity and timing of the 1st heart sound (S_1) and 2nd heart sound (S_2) , the respiratory movement of the pulmonic component of S_2 , clicks and snap of the mitral apparatus, pericardial friction rubs, murmurs, and gallops. When murmurs are detected, the timing, duration, pitch, shape, and intensity and the response to changes of position, handgrip, and the Valsalva maneuver should be noted. When gallops are detected, differentiation should be made between the 4th heart sound (S_4) , which is often present with diastolic dysfunction or myocardial ischemia, and the 3rd heart sound (S_3) , which is present with systolic dysfunction.

The chest is inspected for skin lesions of trauma or herpes zoster infection and palpated for **crepitance** (suggesting subcutaneous air) and tenderness. The abdomen is palpated for tenderness, **organomegaly**, and masses or tenderness, particularly in the epigastric and right upper quadrant regions.

The legs are examined for arterial pulses, adequacy of perfusion, edema, varicose veins, and signs of DVT (eg, swelling, erythema, tenderness).

Red flags: Certain findings raise suspicion of a more serious etiology of chest pain:

- Abnormal vital signs (tachycardia, bradycardia, tachypnea, hypotension)
- Signs of hypoperfusion (eg, confusion, ashen color, diaphoresis)
- Shortness of breath
- Asymmetric breath sounds or pulses
- New heart murmurs
- Pulsus paradoxus > 10 mm Hg

Interpretation of findings: Symptoms and signs of thoracic disorders vary greatly, and those of serious and non-serious conditions often overlap. Although red flag findings indicate a high likelihood of serious disease, and many disorders have





"classic" manifestations, many patients who have serious illness do not present with these classic symptoms and signs. For example, patients with myocardial ischemia may complain only of indigestion or have a very tender chest wall on palpation. A high index of suspicion is important when evaluating patients with chest pain. Nonetheless, some distinctions and generalizations are possible.

Duration of pain can provide clues to the severity of the disorder. Long-standing pain (ie, for weeks or months) is not a manifestation of a disorder that is immediately life threatening. Such pain is often musculoskeletal in origin, although GI origin or a cancer should be considered, particularly in patients who are elderly. Similarly, brief (< 5 sec), sharp, intermittent pains rarely result from serious disorders. Serious disorders typically manifest pain lasting minutes to hours, although episodes may be recurrent (eg, unstable angina may cause several bouts of pain over 1 or more days).

Patient age is helpful in evaluating chest pain. Chest pain in children and young adults (< 30 yr) is less likely to result from myocardial ischemia, although MI can occur in people in their 20s. Musculoskeletal and pulmonary disorders are more common causes in these age groups.

Exacerbation and relief of symptoms also are helpful in evaluating chest pain. Although angina can be felt anywhere between the ear and the umbilicus (and often not in the chest), it is typically consistently related to physical or emotional stress, ie, patients do not experience angina from climbing one flight of stairs one day and tolerate 3 flights the next day. **Nocturnal angina** is characteristic of acute coronary syndromes, heart failure, or coronary artery spasm.

Pain from many disorders, both serious and minor, can be exacerbated by respiration, movement, or palpation of the chest. These findings are not specific for origin in the chest wall; about 15% of patients with acute MI have chest tenderness on palpation.

Nitroglycerin may relieve pain of both myocardial ischemia and non-cardiac smooth muscle spasm (eg, **esophageal** or **biliary disorders**); its efficacy or lack thereof should not be used for diagnosis.

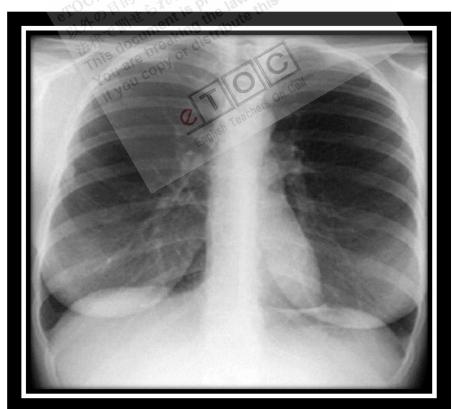
Associated findings may also suggest a cause. Fever is nonspecific but, if accompanied by cough, suggests a pulmonary cause. Patients with **Raynaud syndrome** or **migraine headaches** sometimes have coronary spasm.





The presence or absence of risk factors for CAD (eg, hypertension, hypercholesterolemia, smoking, obesity, diabetes, positive family history—alters the probability of underlying CAD but does not help diagnose the cause of a given episode of acute chest pain. Patients with those factors may well have another cause of chest pain, and patients without them may have an acute coronary syndrome. However, known CAD in a patient with chest pain raises the likelihood of that diagnosis as the cause (particularly if the patient describes the symptoms as "like my angina" or "like my last heart attack"). A history of peripheral vascular disease also raises the likelihood that angina is the cause of chest pain.

Testing: For adults with acute chest pain, immediate life threats must be **ruled out**. Most patients should initially have pulse oximetry, ECG, and chest x-ray. If symptoms suggest an acute coronary syndrome or if no other cause is clear (particularly in at-risk patients), **troponin levels** are measured. If a PE is considered possible, D-dimer testing is done. Expeditious evaluation is essential because if MI or other acute coronary syndrome is present, the patient should be considered for urgent heart catheterization (when available).



http://www.meddean.luc.edu/lumen/meded/medicine/pulmonar/cxr/atlas/images/71bl.jpg



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Some abnormal findings on these tests confirm a diagnosis (eg, acute MI, pneumothorax, pneumonia). Other abnormalities suggest a diagnosis or at least the need to pursue further investigation (eg, abnormal aortic contour suggests need for testing for thoracic aortic dissection). Thus, if these initial test results are normal, thoracic aortic dissection, tension pneumothorax, and esophageal rupture are highly unlikely. However, in acute coronary syndromes, ECG may not change for several hours or sometimes not at all, and in PE, oxygenation may be normal. Thus, other studies may need to be obtained based on findings from the history and physical examination.

Because a single normal set of cardiac markers does not rule out a cardiac cause, patients whose symptoms suggest an acute coronary syndrome should have serial measurement of the cardiac marker troponin and ECGs at least 6 h apart. Some clinicians follow these tests (acutely or within several days) with a stress ECG or a stress imaging test. Drug treatment is begun while awaiting results from the 2nd troponin level unless there is a clear contraindication. A diagnostic trial of sublingual nitroglycerin or an oral liquid antacid does not adequately differentiate myocardial ischemia from gastroesophageal reflux disease or gastritis. Either drug may relieve symptoms of either disorder. Troponin will be elevated in all acute coronary syndromes causing cardiac injury and often in other disorders that damage the myocardium (eg, myocarditis, pericarditis, agric dissection involving coronary artery flow, PE, heart failure, severe sepsis). CPK may be elevated from damage to any muscle tissue, but CPK-MB elevation is specific to damage to the myocardium. However, troponin is now the standard marker of cardiac muscle injury. ST-segment abnormality on the ECG may be nonspecific or due to antecedent disorders, so comparison with previous ECGs is important.

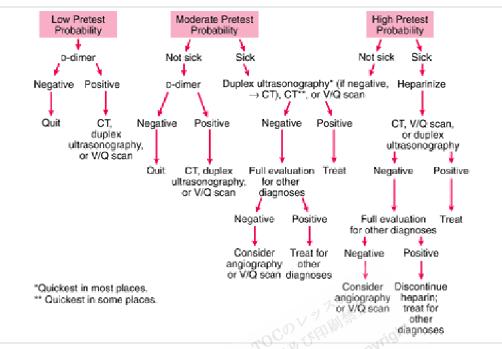
The likelihood of PE is affected by a number of factors, which can be used in an algorithm to derive an approach to testing.

In patients with chronic chest pain, immediate threats to life are unlikely. Most clinicians initially obtain a chest x-ray and do other tests based on symptoms and signs.

Fig. 1

PE testing algorithm.





Use pretest probability to determine testing.

Table 2

Clinical Prediction Rule for Diagnosing Pulmonary Embolism

I. Establish clinical probability—add points to determine total score and thus probability.

Clinical Risk	Points
Clinical signs and symptoms of DVT (objective leg swelling, pain with palpation)	3
PE as likely as or more likely than alternative diagnosis	3
Heart rate > 100 beats/min	1.5
Immobilization ≥ 3 days	1.5
Surgery in previous 4 wk	1.5
Previous DVT or PE	1.5
Hemoptysis	1
Malignancy (including in patients stopping cancer	1



treatment within 6 mo)		
Total Score	Probability	
> 6	High	
2–6	Moderate	
< 2	Low	
II. Use pretest probability to determine testing.		
 DVT = deep venous thrombosis; PE = pulmonary embolism; V/Q = ventilation/perfusion. 		

Treatment

Specific identified disorders are treated. If etiology is not clearly benign, patients are usually admitted to the hospital or an observation unit for cardiac monitoring and more extensive evaluation. Symptoms are treated with acetaminophen or opioids as needed, pending a diagnosis. Pain relief following opioid treatment should not diminish the urgency of ruling out serious and life-threatening disease.

Geriatrics Essentials

The probability of serious and life-threatening disease increases with age. Many elderly patients recover more slowly than younger patients but survive for significant time if properly diagnosed and treated. Drug doses are usually lower, and rapidity of dose escalation is slower. Chronic disorders (eg, decreased renal function) are often present and may complicate diagnosis and treatment.

Key Points

- Immediate life threats must be ruled out first.
- Some serious disorders, particularly coronary ischemia and PE, often do not have a classic presentation.
- Most patients should have pulse oximetry, ECG, cardiac markers, and chest x-ray.
- Evaluation must be prompt so that patients with ST-elevation MI can be in the heart catheterization laboratory (or have thrombolysis) within the 90-min standard.



 If PE is highly likely, antithrombin drugs should be given while the diagnosis is pursued; another embolus in a patient who is not receiving anticoagulants may be fatal.

Reference: http://www.merckmanuals.com

