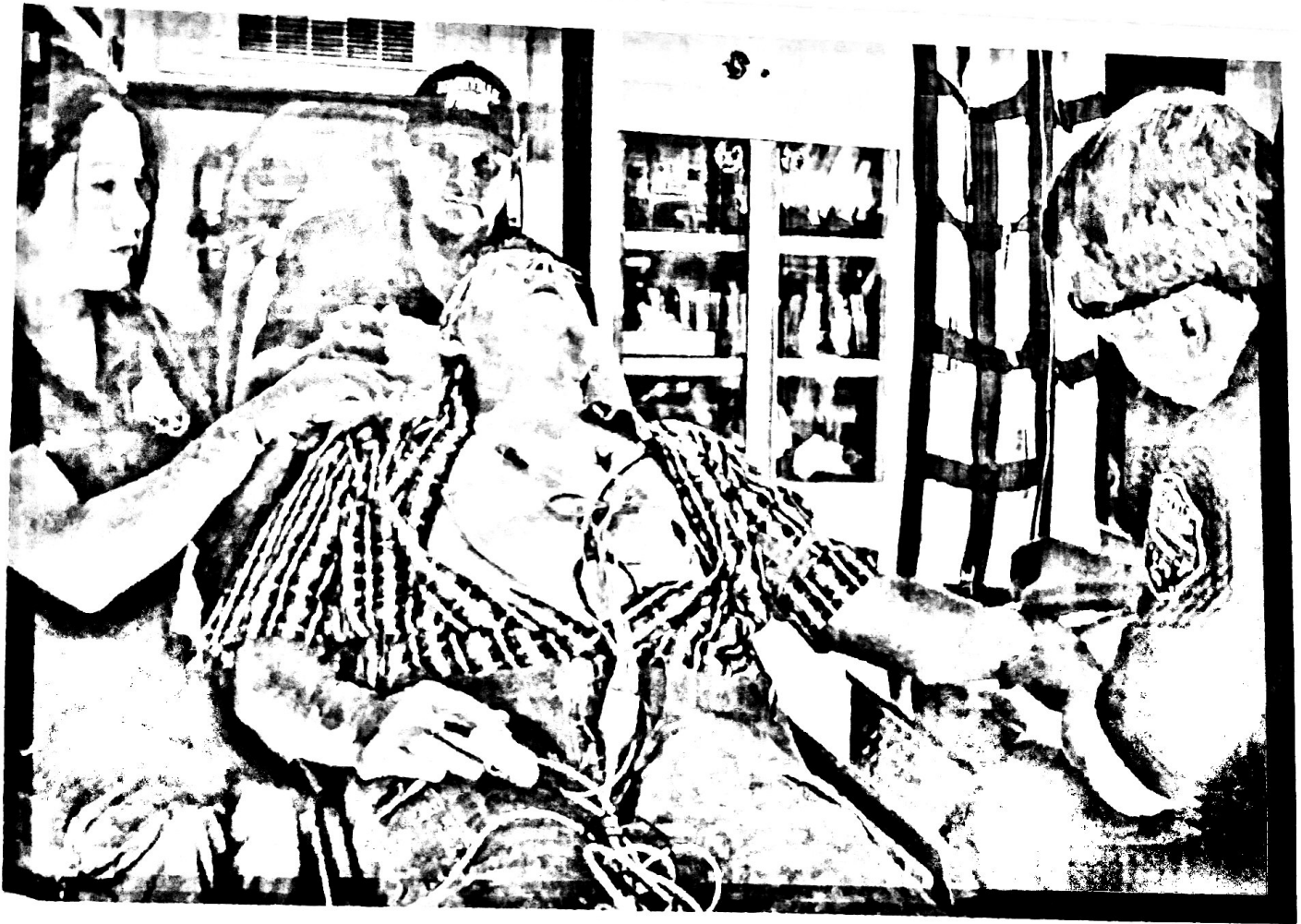


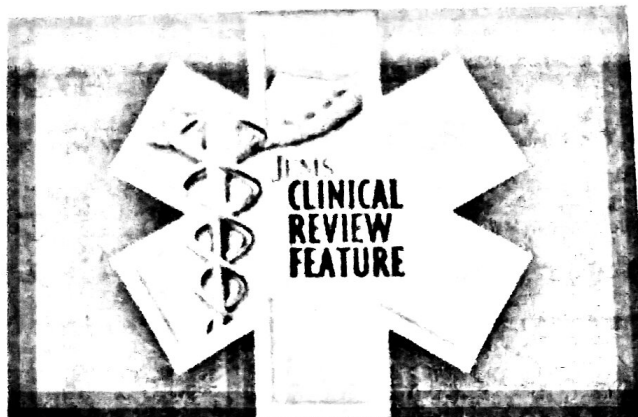
Write a page summary
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The Five Deadly Causes of Chest Pain Other than Myocardial Infarction

Sun, Jan 1, 2017 | By Ashley Brown, MD , H. Evan Dingle, MD , Jeremy Brywczynski, MD , Jared McKinney, MD , Corey Slovis, MD



Calls for chest pain are exceedingly common in EMS and result in more than 8 million ED visits yearly in the United States.¹ Of these, almost 1 million may have an acute coronary syndrome (ACS) and about a third of these will have a myocardial infarction (MI).²



Although the vast majority of chest pain patients don't have either ACS or another emergency, EMS providers must be experts in recognizing all of the potential lethal causes of chest pain.

In this article we'll discuss common presentations of ACS and then review the five other acute deadly causes of chest pain: aortic dissection, pericarditis with tamponade, esophageal perforation, pulmonary embolus and tension pneumothorax. Although all may initially appear very similar with chest pain, hypotension, shortness of breath or as a patient in extremis, each has some important red flags to be on the lookout for.

Making matters even more confusing, although the ECG findings of ST elevation or T-wave inversion usually support the diagnosis of ACS, MI-like ECG changes can be seen with each of the other life-threatening causes of chest pain.

Classic & Atypical ACS

Classic angina presents with substernal chest pain that's described as "squeezing" or "pressure-like." It often radiates to the arms or jaw and is made worse by exertion or emotion and made better by rest or nitroglycerin.³

It may be associated with diaphoresis, nausea, weakness or shortness of breath. It usually lasts minutes, not seconds or hours. It's rarely sharp, positional, or pleuritic—that is, it's rarely affected by breathing and doesn't change in intensity with deep or shallow breathing.

Unfortunately, no single symptom can be used to reliably diagnose or exclude a diagnosis of myocardial ischemia or infarction.³ Even more troubling is that studies have shown as many as 1 in 3 patients may have atypical presentation of ACS.¹ That is, they may not have typical chest pain or, in almost 1 in 10 patients, they may have no chest pain at all.⁴

It's imperative that all emergency providers remember that five specific sign-symptom complexes may represent ACS even in patients without chest pain. Acute onset of shortness of breath; diaphoresis; abdominal discomfort and/or nausea; neurological symptoms such as syncope, pre-syncope or dizziness; and global weakness/acute fatigue should prompt prehospital care providers to consider ACS.⁴ Atypical symptoms are more common in the elderly and women, but can occur in any patient.

Assessing Chest Pain

All chest pain patients should have their ABCs (i.e., airway, breathing and cardiovascular stability) assessed rapidly. Asking the patient about their chest pain allows rapid assessment of the airway—the ability to breathe as they talk in full sentences confirms a patent airway and also allows you to evaluate mental status. Listening to each side of the chest allows assessment of air movement, lung sound equality along with the presence of wheezes or wet sounding lungs. Finally, the patient's pulse should be quickly assessed for its strength, rate and regularity.

After the ABCs are assessed they need to be secured. We call this the "opening gambit," as it's the initial move in almost all adult chest pain patients or others who may have an acute cardiac or pulmonary illness: oxygen, oxygen saturation monitoring, IV access, continuous cardiac monitoring and a 12-lead ECG. (See Table 1.) Once done often enough, you'll soon approach these patients thinking, "O₂, O₂ sat, IV, ECG, 12-lead" for critical initial actions before obtaining a full set of vital signs.

**Table 1: Steps to secure ABCs
in typical chest pain patient**

The 'Opening Gambit'

Oxygen	O ₂
Oxygen saturation monitoring	O ₂ sat
IV access line	IV
Cardiac monitoring	ECG
12-lead ECG	12-lead

Once the 12-lead ECG has been performed, it should be carefully analyzed for signs of **ischemia and infarction**. An ST elevation myocardial infarction (STEMI) is identified by ST elevation of 1 mm or more in two anatomically contiguous leads.

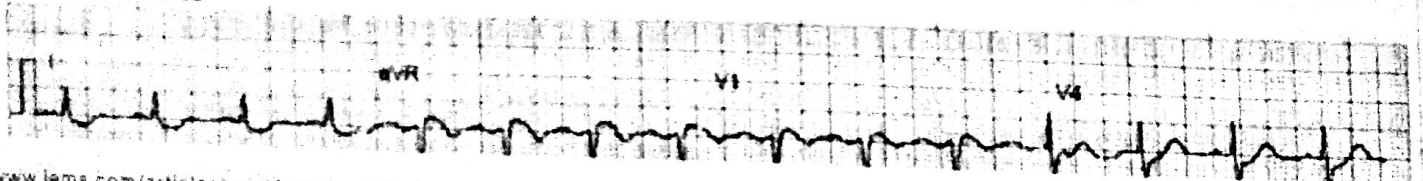
The importance of a prehospital ECG can't be overstated. Prehospital ECGs decrease STEMI mortality by 25–37% and significantly increase the likelihood of reperfusion therapy once that patient reaches the hospital.^{5,6} Because ST changes often evolve over time, a prehospital ECG is essential even if it's non-diagnostic as it will be compared to the in-hospital ECG looking for new changes or resolution of prehospital abnormalities.

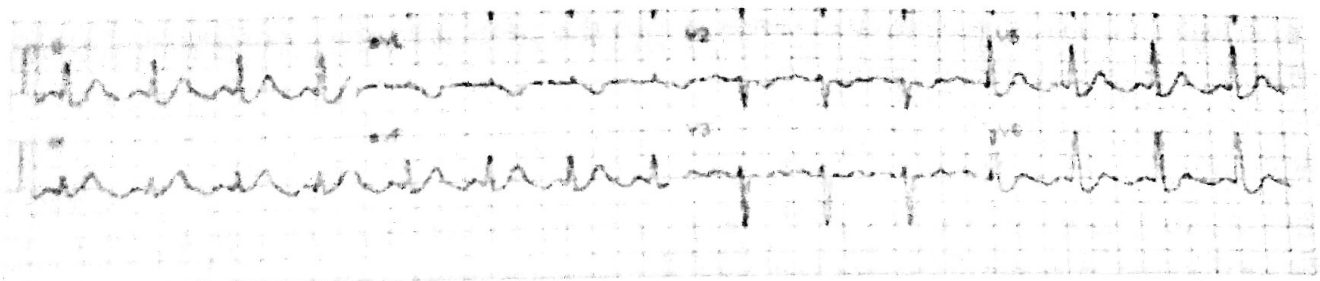
When EMS alerts the ED that they're transporting a STEMI patient, an alert also goes to the cardiac catheterization laboratory team, and door to balloon times can be decreased by 30 minutes or more.⁶

Remember that ECG findings of a STEMI may evolve, or even disappear, quite quickly. (See Figures 1 and 2.) A saying worth remembering in patients at risk for ischemia and infarction, but who have a non-diagnostic ECG, is "one ECG begets another."

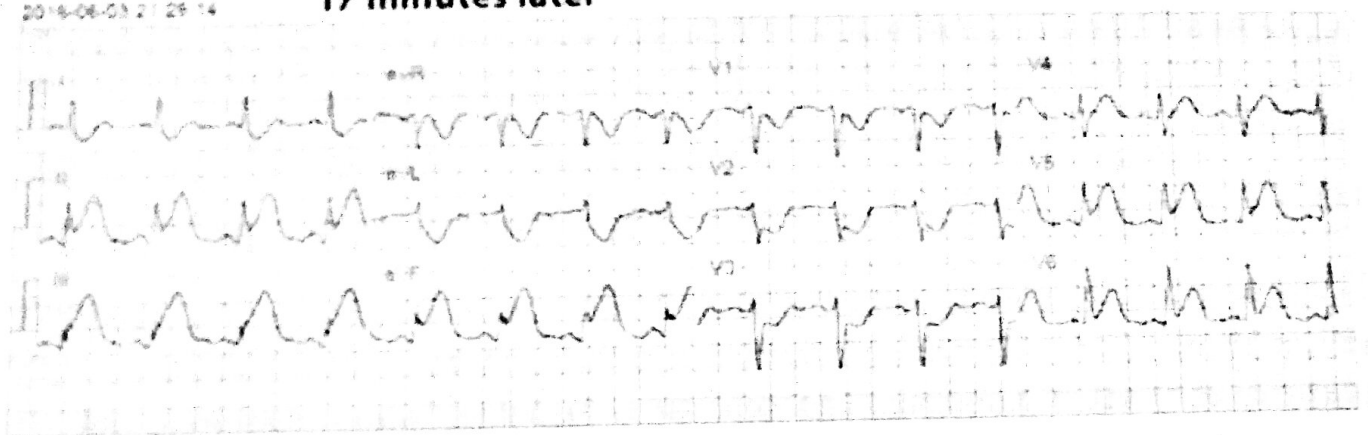
Figures 1 and 2: Evolution of a STEMI

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An abnormal ECG with some lateral ST depression and with no ST elevation rapidly evolves, in just 17 minutes, into an acute inferior lateral STEMI with ST elevation in 2, 3 and AVF and V5-6 along with deep reciprocal changes in V1-V3 suggestive of right ventricular and/or posterior involvement. Images courtesy Vanderbilt University Medical Center

Furthermore, any ECG showing a STEMI indicates an acute MI even if the ST changes resolve. In a study of 83 prehospital STEMI patients, researchers found that 21.6% of the prehospital ECGs showing a STEMI had resolved by the time a second ECG was performed in the ED.

Their patients were, however, just as likely to have an acute coronary occlusion as those who still have ST elevation acute MIs on their ECGs.⁷ This reinforces the need to do ECGs in the field and to repeat them if possible while en route to the hospital.

1. Aortic Dissection

Aortic dissection occurs when there's a tear in the aortic intima, the aorta's innermost layer, which leads to the creation of a false lumen.

The force exerted by the patient's blood pressure and heart rate (i.e., shear forces) further extends the injury along the length of the aorta, which can lead to

occlusion of branch vessels or extension into the pericardium or coronary arteries leading to **tamponade** or acute MI.

Although much less common than ACS, an acute aortic dissection carries a high rate of morbidity and mortality.^{8,9} This is complicated by the fact that ACS and dissection can present very similarly yet are treated totally differently.

Aortic dissection is more common in men and most patients have a history of hypertension. Other risk factors include **Marfan syndrome**, aortic valve disorders/surgery, cocaine ingestion, cardiac surgery, atherosclerosis, and a prior known aortic aneurysm.^{8,9}

The presenting signs and symptoms of aortic dissection are diverse. The classic presentation of aortic dissection involves the acute onset of chest pain, which is maximal at onset and described as "tearing" or "ripping," with radiation to the back.

Over 70% of patients will describe chest pain,⁹ but as compared with acute MI, the pain is less likely to radiate to the arm or jaw. The pain is more often described as sharp,⁸ and involvement of the descending aorta can cause pain in the back and abdomen.

Any chest pain patient who says their pain has moved or migrated should raise your suspicion for an aortic dissection. Patients suspected of having an aortic dissection should have the opening gambit performed rapidly.

Patients may also present without pain, which often leads to delayed or missed diagnosis.

Aortic dissection should also be considered in the differential diagnosis of patients who present with syncope, signs/symptoms of stroke, or spinal cord syndromes.

Physical exam findings in patients with acute aortic dissection are varied. As mentioned above, most patients will be hypertensive at presentation. Hypotension should raise the concern for cardiac tamponade and you should consider aortic dissection for any MI patient with hypotension.

Patients can also present with a discrepancy in blood pressure between the upper

extremities as a result of dissection down the subclavian artery, or with a pulse deficit between the upper and lower extremities. Patients may also have a murmur consistent with aortic regurgitation, although this isn't likely to be heard in the noisy prehospital setting.

Patients with acute aortic dissection will often have ECG abnormalities, with only about 30% of patients having a normal ECG at presentation.^{8,9,10} These ECG changes may mimic ACS/MI, which can lead to delayed or missed diagnosis.

Common ECG findings include T-wave inversion, fragmentation of the QRS complex, ST depression, and ST elevation.^{10,11} ECG changes are more common in patients presenting in shock.¹⁰ ST elevation can be seen with dissection into the coronary artery, ostial or coronary occlusion from the false lumen, or hypotension.

The right coronary is most often involved and will lead to classic inferior MI changes on ECG with ST elevation in leads II, III, and aVF.

Incorrect diagnosis of ACS/MI based on ECG changes can lead to the administration of antiplatelet therapy, anticoagulation, and thrombolytic agents. This increases the risk of propagation of the dissection, cardiac tamponade, aortic rupture, and sudden death, so antiplatelet therapy such as aspirin should be withheld until discussion with medical control.

Caution should also be given to the administration of nitroglycerin if dissection is suspected as it could lead to reflex tachycardia and increased shear forces with potential for propagation of the dissection.

The diagnosis of aortic dissection is difficult to make and should be highly suspected in patients presenting with the acute onset of chest pain, which is maximal at onset, with radiation or migration to the back, abdomen or neck, and in chest pain patients who have chest pain with neurologic symptoms. Common incorrect initial diagnoses include ACS, pulmonary embolism, and musculoskeletal strain.¹¹

2. Pericarditis & Pericardial Tamponade

Pericarditis, an inflammation of the pericardial sac that lines, insulates and contains the heart, is a common and often forgotten cause of chest pain in adult

The disease most frequently affects young and middle-aged individuals, often recurs, has a much higher incidence in men, and has an overall mortality rate of 1.1%.¹²

Acute pericarditis is a diagnostic challenge in that it can present very similar to other disorders and have life-threatening complications. Chest pain is the most common presentation, which is classically described as "sharp" and "pleuritic," but may also be dull or aching. The patient may be febrile. The pain may radiate to the trapezius ridge, neck or shoulder. The pain is made better by leaning forward and worse by reclining backward.¹³

The etiology of pericarditis is idiopathic in up to 90% of cases and it's assumed by the medical community that these are viral infections.¹⁴

The remaining cases are associated with post-cardiac injury syndromes, rheumatologic manifestations of heart disease (especially systemic lupus erythematosus), and complications of cancer.¹⁴

The EMS diagnosis of pericarditis is tentatively made by seeing typical ECG findings or diffuse ST elevation in multiple leads without reciprocal depressions often in association with P-R depression. It's important to remember that the diffuse PR depression and ST elevation in the majority of ECG leads that is classic for acute pericarditis is only seen in cases of infectious pericarditis.¹⁴ (See Figure 3.)

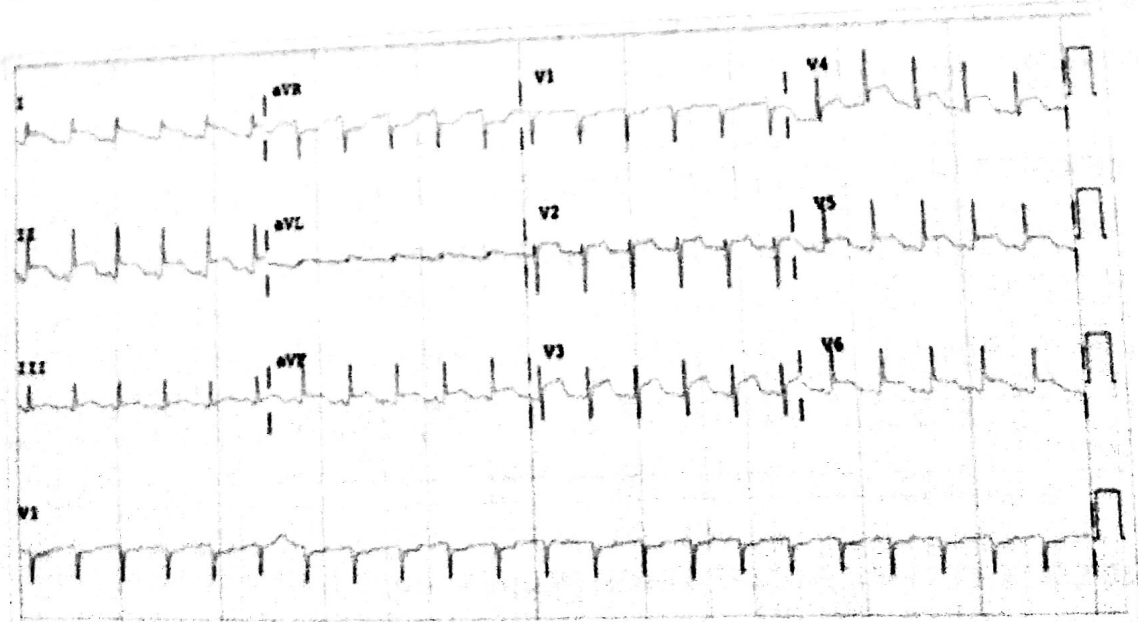


Figure 3: ECG demonstrating diffuse ST elevation of pericarditis

General treatment of pericarditis is rest, nonsteroidal anti-inflammatory drugs and colchicine. Patients will typically recover in 1-2 weeks, with some experiencing several recurrences over a lifetime. The dreaded complication of pericarditis is the development of a large pericardial effusion with resultant pericardial tamponade.

Up to 3% of patients with pericarditis will develop cardiac tamponade, which clinically is diagnosed by distended neck veins, muffled heart tones, and hypotension, known as Beck's triad.¹⁴

The most common type of pericarditis known to progress to a pericardial effusion causing tamponade physiology is pyogenic (staphylococcal or streptococcal), tuberculous or malignant.¹⁵ (See Figure 4.)

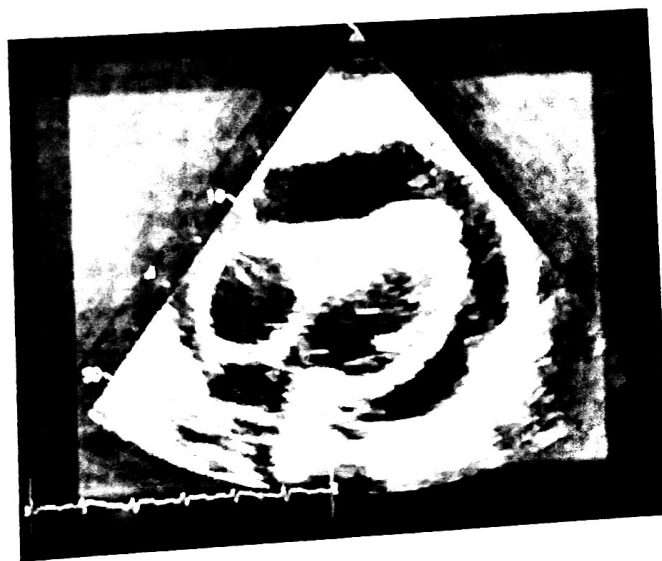


Figure 4: Pericardial fluid (black) compressing the heart in cardiac tamponade.

Patients with tamponade will present with shortness of breath, chest pain and heaviness, and even signs and symptoms of cardiogenic shock.¹⁵ In the prehospital environment, the patient will be preload-dependent and the treatment will center around fluid resuscitation with normal saline or lactated Ringer's.

Intubation and mechanical ventilation should be approached with much caution and avoided if at all possible; the change from negative intrathoracic pressure to positive pressure ventilation decreases preload further and can precipitate an abrupt cardiovascular collapse and cardiac arrest. The definite treatment is pericardiocentesis or pericardial window, optimally in the operative suite.

3. Esophageal Perforation & Rupture

Although it's a relatively rare cause of chest pain, esophageal perforation can become deadly quickly; therefore, its timely diagnosis is essential.

The esophagus contains two major tissue layers, a mucosal layer and a muscular layer. Unlike most of the gastrointestinal tract, the esophagus lacks a strong outer serosal layer. This increases susceptibility for full-thickness perforation.¹⁶ Morbidity and mortality increase with delay in diagnosis as esophageal contents such as food, stomach acid and bacteria leak into the normally sterile mediastinum.



ECG changes that are consistent with myocardial infarction can be seen with other life-threatening causes of chest pain. It's important to continuously monitor the patient's cardiac status as it may evolve or change.

There are specific elements in the patient's history that make a perforated esophagus more likely. Recent instrumentation of the esophagus should prompt consideration of this diagnosis as medical procedures are responsible for 60% of perforations.¹⁷

Some procedures associated with rupture include upper GI endoscopy, esophageal biopsy procedures, nasogastric tube placement, and balloon dilation of strictures. The increasing number of endoscopic procedures is the primary reason esophageal perforations have increased in frequency throughout the past

reason esophageal perforations have increased in frequency throughout the past two decades and will likely continue to rise.

Perforations may also occur spontaneously following sudden increases in intraesophageal pressure. Boerhaave syndrome describes a full-thickness tear of the esophagus, typically following retching or forceful vomiting, and may be seen in alcoholics.

This is contrasted with the more common and more benign Mallory-Weiss tear, which only involves the mucosal layer and presents with hematemesis and typically less pain.^{17,18}

Perforations are also seen following penetrating trauma to the neck and, less commonly, with blunt trauma. Perforations have even been reported following administration of the Heimlich maneuver and following difficult endotracheal intubations.¹⁷

The diagnosis of an esophageal perforation is suggested by chest pain with **subcutaneous emphysema** around the neck. The subcutaneous air may be heard during heart auscultation as crunching sounds, classically referred to as Hamman's crunch.

Although classically associated with esophageal perforation, Mackler's triad (chest pain, vomiting and subcutaneous emphysema) is found in less than one-third of patients. Patients often have vomiting, dyspnea, difficulty speaking and, depending on the location of the perforation along the esophagus, may complain of neck pain from cervical perforations or epigastric pain from intra-abdominal perforations.

Pain may radiate into the back. Septic shock with severe hypotension and tachycardia may predominate in delayed cases.^{17,19} Nonspecific T-wave change may be seen on ECG.²⁰

Prehospital care of suspected esophageal perforations involves maintaining a low threshold of suspicion, obtaining key elements of patient history that suggest perforation, and supporting the ABCs.

IV access should be obtained and crystalloid resuscitation administered,

particularly in cases with tachycardia and hypotension. Early recognition is vital for patient outcome. Following diagnosis by imaging or direct visualization in the hospital setting, most will require surgical repair, except for contained perforations, which may be managed by broad-spectrum antibiotics and parenteral nutrition.¹⁹

EMS providers should think of esophageal perforation whenever they encounter a chest pain patient who has recently had endoscopy or had protracted vomiting.

4. Pulmonary Embolism (PE)

Pulmonary embolism (PE) is another life-threatening cause of acute chest pain, and is diagnosed in one out of every 400–1500 adult ED patients.²¹ It's caused by an obstruction of the main pulmonary arteries or one of their branches.²²

PE generally occurs when a thrombus or blood clot forms in another part of the venous system, dislodges, and embolizes through the right atrium and ventricle and lodges in the pulmonary arteries. These thrombi can develop in any part of the venous system but most originate in the lower extremity as a deep venous thrombosis (DVT).²²

In addition to chest pain, patients may also complain of dyspnea; however, presentations range from asymptomatic to full cardiac arrest.²² Due to the subtle and varied presentation, a PE is difficult to diagnose and is often missed, leading to significant morbidity and mortality, with a three-month mortality rate of about 15%.²³

Risk factors for PE include conditions that predispose to clot formation. Virchow's triad describes three broad categories that predispose to thrombus formation: vascular injury, hypercoagulable states (either genetic or acquired), and venous stasis.²²

Active cancer is a risk factor as it induces a hypercoagulable state.^{21,22} Other risk factors include recent surgery or immobilization, recent travel > 4–6 hours duration, prior PE or DVT, pelvic or lower extremity trauma within the last three months, malignancy, smoking and exogenous estrogen use.²²

Pregnant and immediately postpartum women are at increased risk of venous thromboembolism, though this risk seems to be greater in the postpartum period.^{21,22} Age is a significant risk factor, with risk increasing between ages 50–90.²¹

Clinical presentation of PE can be quite varied. The classic presentation of pleuritic chest pain, shortness of breath, and hemoptysis is seen in less than 20% of patients.²²

Many times, a patient's presentation may mimic other pathology such as ACS or pneumonia.^{22,23} The typical complaint of sharp, pleuritic chest pain is due to pulmonary infarction leading to inflammation.²²

Patients may also complain of new-onset dyspnea, sometimes at rest but more typically with exertion.^{21,24} Patients may present with syncope or lightheadedness. Depending on the size of the clot burden, pulmonary embolism may also lead to significant outflow obstruction, causing both right and left ventricular failure and cardiovascular collapse.²²

A patient's clinical presentation will depend on the size of the embolism as well as their underlying medical problems and physiologic reserve. For example, those with a small embolism who are relatively healthy may have stable vital signs and be relatively asymptomatic; alternatively, a patient who has significant pre-existing cardiac or pulmonary disease may become quite symptomatic and unstable with even a small embolism.

In general, PE should be considered in any patient presenting with chest pain, dyspnea, syncope, hemoptysis, or cardiovascular collapse, particularly in patients who also have signs or symptoms of DVT.

Physical exam findings in PE patients are nonspecific. Vital sign abnormalities of tachycardia (> 100 bpm) and pulse oximetry readings < 95% on room air have been shown to increase the probability of PE.²¹

There's conflicting data on the association between tachypnea and the probability of PE. About 10% of patients with PE have a temperature > 100.4 degrees F (38

degrees C), but high fever (> 102.5 degrees F or 39.2 degrees C) was found in $< 2\%$ of PE patients.²¹

Unilateral leg swelling, which is suggestive of DVT, also raises the probability of PE,²¹ as about one-third of patients with DVT also have a PE. In general, though, there are no physical exam findings that can reliably rule in or exclude PE.

Although not diagnostic, an ECG can be a helpful adjunct in the assessment for PE.²⁴ Between 20-25% of patients with PE may have a completely normal ECG,^{24,25} and up to 70% of PE patients may have ECG changes also seen in ACS.²³

The most common abnormal finding is sinus tachycardia,²⁴ which occurs due to increased demand for cardiac output secondary to decreased left-sided stroke volume. The classic pattern of an S1Q3T3, a sign of right heart strain, may also be observed.²⁴

Massive PE is associated with T-wave inversions in the precordial leads.²⁴ Signs of right ventricular strain have been correlated with increased degree of pulmonary artery obstruction and increased risk of death and clinical deterioration, even in patients with normal blood pressures at presentation.^{24,25}

ECG features such as low voltages, right bundle branch block and ST elevation in lead V1 have been associated with cardiogenic shock. Other features such as Q waves in leads III and aVF, ST segment changes in the left precordial leads were found more commonly in patients who died.²⁴

Once in the ED, these patients can be further risk stratified with a combination of clinical decision rules, laboratory testing and imaging.²²

However, in the prehospital setting it will likely be impossible to accurately diagnose a PE, particularly as the signs and symptoms of PE can overlap with a variety of other complaints, such as MI, sepsis, congestive heart failure and chronic obstructive pulmonary disease.

positive pressure ventilation, should be used for those who are hypotensive.

5. Pneumothorax & Tension Pneumothorax

Pneumothorax occurs when air accumulates in the pleural space outside of the lung and may be due to spontaneous or traumatic causes (penetrating and blunt). In blunt trauma, most are caused by fractured ribs that puncture the visceral pleura.

Spontaneous pneumothoraxes occur in the absence of trauma and are often due to ruptured apical blebs. They occur more frequently in smokers and tall, thin males between the ages of 10 and 30.^{26,27}

Certain underlying conditions place patients at higher risk for spontaneous pneumothorax development including cystic fibrosis, asthma and connective tissue disorders, particularly Marfan syndrome and Ehlers-Danlos syndrome.²⁶

Severity of symptoms is determined by the size of the pneumothorax, a patient's underlying lung reserve, and the presence of tension physiology.²⁶ Patients with a pneumothorax typically complain of sudden onset pleuritic chest pain and shortness of breath, which may occur even at rest.²⁷

On exam, decreased or absent breath sounds may be appreciated on the affected side. Tachycardia is the most common finding on physical exam.²⁷

Patients may even have palpable subcutaneous emphysema if air dissects into the soft tissues of the thorax and neck. Some pneumothoraxes may develop life-threatening tension physiology.

A tension pneumothorax occurs when air enters the pleural space during inspiration through a "one-way valve" but can't escape during expiration. As air pressure exceeds venous pressure in the thorax, return of blood to the heart is impeded and cardiac output drops.²⁶ This will lead to vital sign abnormalities of tachycardia, hypotension and hypoxia.

Elevated intrathoracic pressure is sometimes reflected on physical exam as distended neck veins; however, this is a late finding. Similarly, tracheal deviation away from the pneumothorax also occurs late as high pressure from trapped air pushes the mediastinal structures toward the opposite side.²⁸

Patients may appear agitated and restless with decreasing mental status, tachycardia and hypoxia. If no intervention is taken, these patients develop hypotension and ultimately cardiac arrest.

A third and more obvious type of pneumothorax, an open pneumothorax, sometimes referred to as a "sucking chest wound," occurs in penetrating trauma when the chest wall wound is approximately two-thirds the diameter of the trachea or greater. This causes air to preferentially enter the pleural space through the chest wall during inspiration.²⁹ The flow of air through the defect is heard during respirations, hence the term "sucking chest wound." Open pneumothorax is most common in combat situations or from gunshot wounds.



Lung sounds, or the lack thereof, may clue you in to causes of chest pain that aren't due to angina or myocardial infarction.

Prehospital care of pneumothorax should include administration of 100% oxygen, IV access, and placement on pulse oximetry and cardiac monitoring.

Patients with suspected pneumothorax, particularly trauma patients receiving positive pressure ventilation, should be vigilantly monitored for development of a

positive pressure ventilation, should be vigilantly monitored for development of a tension pneumothorax.²⁸

An increase in resistance to ventilation is an early sign of developing tension pneumothorax. Evidence of tension pneumothorax requires prompt decompression. Absence of breath sounds alone isn't an indication for decompression.

Significant respiratory or hemodynamic compromise should be a prerequisite for decompression.³⁰ Historically, emergent decompression has been accomplished by placement of a large bore catheter through the second intercostal space at the midclavicular line. However, studies have demonstrated that this site is often associated with failure to enter the pleural space, particularly in obese patients.^{31,32}

One study found that using 8-cm angiocatheters at the midclavicular line, rather than 5-cm catheters as recommended by Advanced Trauma Life Support guidelines, improved effectiveness of decompression in the prehospital setting.³³ However, the midclavicular approach may be associated with serious injury, particularly to the subclavian and great vessels and even to the heart.²⁸

An alternative needle insertion site is the 4th or 5th intercostal space at the anterior axillary line. Regardless of which site is chosen, needle insertion should occur just above the rib (at the bottom of the intercostal space), avoiding the intercostal arteries which run along the inferior edge of each rib.

Finally, sucking chest wounds should be covered with an occlusive dressing on three sides, allowing air to escape during expiration through the unsecured side, but preventing air entrance during inspiration. Erroneously securing all edges of the occlusive dressing can result in tension pneumothorax when concomitant lung injury allows air to enter the pleural space without a means of escape.²⁹

Although seen most commonly in trauma patients, pneumothorax can also occur spontaneously and should be considered in the differential for patients with sudden onset chest pain or shortness of breath, particularly when breath sounds are decreased on one side. Patients should be monitored closely for development of tension pneumothorax, and prehospital providers should be prepared to

perform urgent decompression.

Conclusion

Although ischemic changes are common in patients with ACS, similar changes may be seen in other deadly causes of chest pain. We've reviewed the five deadly causes of chest pain other than MI and examined the similarities and differences of each. Understanding them is important, but the key to all of these potentially life threatening diseases is early expert care and a high index of suspicion.