Chapter 1 | Evaluation and Management of Patients with Chest Syndromes

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Introduction

Chest pain is a common emergency department (ED) complaint with a well-known differential diagnosis. Yet compared to the abdomen, the chest contains relatively few structures (e.g., the heart, the lungs, the great vessels, the esophagus) to consider as the source of the complaint when evaluating a patient with chest pain. In these few structures, however, there exists the potential for several life-threatening maladies, some of which unfortunately occur rather commonly. In patients with chest pain, initial attention is often devoted to establishing the presence or absence of acute coronary syndrome (ACS), but indeed there are several other syndromes of critical importance and clinical relevance to consider. In this chapter, we consider six pitfalls related to ACS, followed by a variety of pitfalls related to other diseases of the chest: aortic dissection (AD), pulmonary embolism (PE), pericarditis, pneumothorax, esophageal rupture, and finally, herpes zoster.

Pitfall | Over-reliance on the classic presence of chest pain for the diagnosis of acute myocardial infarction (MI)

Although chest pain has long been considered the hallmark clinical feature of acute myocardial infarction (MI), it is important to recognize that the absence of chest pain in no way excludes the diagnosis. In a large observational study, Canto et al. examined the presenting complaints of nearly 435,000 patients with confirmed MI enrolled in the National Registry of Myocardial Infarction 2 (NRMI-2) database and found that one-third of the patients presented to the hospital without chest pain [1]. Other studies have reported similar findings. In one study, over 20% of 2096 patients diagnosed with acute MI presented with symptoms other than chest pain [2]. In another smaller study, nearly half (47%) of 721 patients hospitalized for acute MI presented to the ED without chest pain [3]. Risk factors associated with the absence of chest pain included age, female gender, non-white race, diabetes mellitus, and a prior history of congestive heart failure or stroke (see Table 1.1) [1].

KEY FACT | **Over the age of 85, 60–70% of patients with acute MI present without chest pain.**

Table 1.1 Risk factors for painless acute MI [1].

Risk Factors	% Without Chest Pain
Prior heart failure	51
Prior stroke	47
Age > 75 years	45
Diabetes mellitus	38
Non-white	34
Women	39

In the elderly population, chest pain is reported less frequently according to the NRMI-2 database, patients experiencing an acute MI without chest pain are, on average, 7 years older (74 versus 67 years) [1]. Uretsky et al. reported a mean age of 69.1 years in those patients without chest pain as compared to 58.7 years in those with chest pain [4]. Under the age of 85, chest pain is still present in the majority of patients but other non-pain symptoms (referred to as "anginal equivalents") such as shortness of breath, syncope, weakness, and confusion are common. Over the age of 85, 60–70% of patients with acute MI present without chest pain; shortness of breath is the most frequent anginal equivalent in this population [5].

Women are more likely than men to experience acute MI without chest pain [1–3, 6]. In one study, women over the age of 65 were the most prevalent group to experience acute MI without chest pain [6]. In another study of 515 women surveyed after experiencing an acute MI, only 57% reported chest pain at the time of their MI. The most frequent anginal equivalents reported were shortness of breath (58%), weakness (55%), unusual fatigue (43%), cold sweats (39%), and dizziness (39%) [7].

Patients with diabetes mellitus are at increased risk for acute MI and are more likely to present without chest pain [1, 8]. Medically unrecognized acute MI has been noted in up to 40% of patients with diabetes as compared to 25% of the non-diabetic population [8]. Although the NRMI-2 database noted that diabetics were more likely to experience acute MI without chest pain (32.6% versus 25.4%), two-thirds of those who experienced acute MI without chest pain were still non-diabetics [1].

Patients experiencing an acute MI without chest pain are more likely to suffer delays in their care. Analysis of the NRMI-2 database revealed that these patients were less likely to receive aspirin, heparin, or beta-adrenergic blockers in the initial 24 h and were much less likely to receive fibrinolysis or primary angioplasty (25.3% versus 74.0%) [1]. They were also more likely to die in the hospital compared to patients who presented with chest pain (23.3% versus 9.3%) [1]. Uretsky et al. reported a nearly 50% mortality rate in patients hospitalized with acute MI who presented without chest pain compared to an 18% mortality rate in those presenting with chest pain [4]. The 30- and 365-day mortality rates have also been noted to be higher in this group [2]. Clearly, populations other than diabetics are at risk to present without chest pain while having an acute MI; women and the elderly are among those groups identified to be at particular risk.

Pitfall | **Exclusion of cardiac ischemia based on reproducible chest wall tenderness**

ED visits for chest pain comprise 5–8% of all ED cases [9]. The etiologies of chest pain range from benign to life threatening. The goal of the emergency physicians (EP) is to identify the life-threatening causes, including acute MI. Ruling out acute MI in the clinically stable patient presenting with chest pain and a non-diagnostic ECG represents a particular challenge to the EP.

Certain chest pain characteristics have been shown to decrease the likelihood of acute MI. Lee et al. examined multiple chest pain characteristics to identify patients at low risk for acute MI. The combination of three variables – sharp or stabbing pain, no history of angina or acute MI, and pain that was pleuritic, positional, or reproducible – defined a very low-risk group [10]. Other studies have concluded that positional chest pain suggests a non-ACS etiology [11, 12]. Chest pain localized to a small area of the chest is often thought to suggest a musculoskeletal etiology. In one study, however, 27 of 403 patients (7%) with acute MI localized their pain to an area as small as a coin [13].

Chest wall tenderness, or reproducible chest pain, is a clinical feature that may persuade the EP to make a diagnosis of musculoskeletal pain. On examining the patient, the EP should be careful in determining if the pain induced by chest palpation is the same pain as the presenting pain. If there is no defined injury or event that could have led to a soft tissue injury, the EP should be reluctant to render a diagnosis of musculoskeletal pain.

KEY FACT | **7% of patients with acute MI or unstable angina had their pain partially or fully reproduced on chest wall palpation.**

Several studies have shown that chest wall tenderness can be misleading. In two separate studies, as many as 15% of patients diagnosed with acute MI had some degree of chest wall tenderness on examination [4, 14]. In another study, 17/247 (7%) of patients with acute MI or unstable angina had their pain partially or fully reproduced on chest wall palpation [10]. More recently, Disla et al. noted that 6% of patients with chest wall tenderness on their initial examination were ultimately diagnosed with acute MI [15].

Several other studies have demonstrated that chest wall tenderness "suggests" a non-ACS etiology of chest pain. In one prospective observational study, the presence of chest wall tenderness reduced the probability of acute MI (LR, 0.2; 95% CI, 0.1–1.0) [16]. Panju et al. and Chun and McGee concluded after separate meta-analyses that chest wall tenderness decreased the likelihood of acute MI (LR, 0.2–0.4; LR, 0.3 respectively) [17, 18]. However, considering the pre-test probability of acute MI noted in both meta-analyses (12.5–17.4%), the post-test probability of acute MI was still 4.3–6.3%.

Although certain chest pain characteristics decrease the likelihood of acute MI, none is powerful enough to support discharging at-risk patients without additional testing. In patients with chest pain, chest wall tenderness may suggest that acute MI is less likely but it does not effectively rule out the diagnosis. Given the potential implications of missing the diagnosis of acute MI, using chest wall tenderness as an independent rule out strategy is not recommended in patients at risk for ACS.

Pitfall | **Assumption that acute MI cannot be diagnosed with a 12-lead ECG in the presence of pre-existing left bundle branch block or ventricular paced rhythm**

The 12-lead ECG is an invaluable tool in the diagnosis of acute MI; in fact, it is the defining test of an ST-segment elevation MI (STEMI). There is a tendency to proffer diagnostic surrender when confronted with a patient presenting with signs and symptoms of ACS and an ECG that demonstrates either left bundle branch block (LBBB) or ventricular paced rhythm (VPR); the decision may be made to "wait for the cardiac enzymes" to establish a diagnosis. In fact, whereas these two electrocardiographic entities may confound or obscure the diagnosis of STEMI, there are published criteria that offer fairly specific (if not sensitive) evidence of STEMI in the face of LBBB and VPR.

LBBB

Delayed depolarization of ventricular myocardium in patients with LBBB results in the following characteristic findings:

1. QRS complex width > 0.12 s;
2. broad QS or rS pattern in the right precordial leads (leads V1, V2, and sometimes V3);

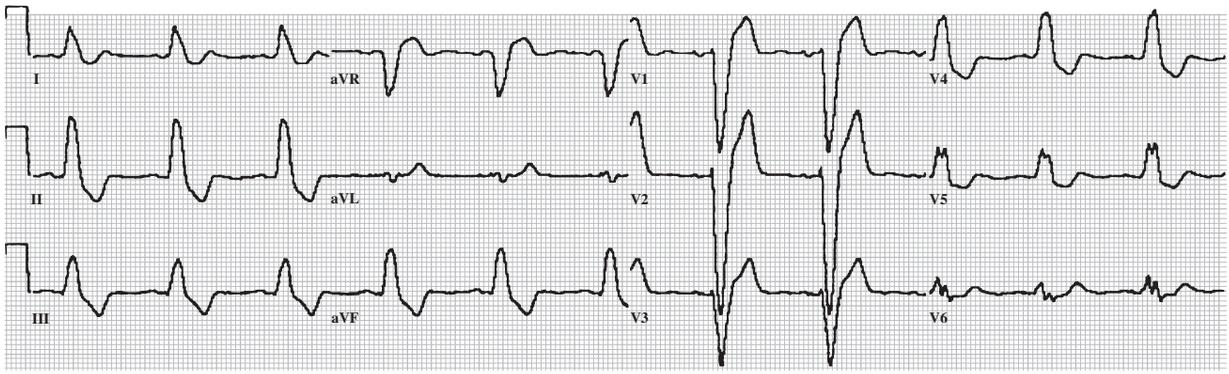

Figure 1.1 LBBB:
This tracing demonstrates an uncomplicated LBBB. Note the widened QRS complex (>0.12 s), the monophasic notched R-wave in the lateral leads (best seen in leads I and V5 here), and the absence of a Q-wave in lateral leads (I, aVL, V5, V6). There is discordance between the major vector of the QRS complex and the major vector of the ST-segment/T-wave complex that follows. Contrast these morphologies to those seen in Figure 1.2.

3. monophasic R-wave in the lateral leads (some, if not all, of leads I, aVL, V5, and V6); the absence of a q-wave in lateral leads.

Characteristically, in LBBB the affected leads also feature discordance of the ST-segment/T-wave complex: when the major QRS vector is directed downward (as in the right precordial leads) the ST-segment will be elevated and the T-wave will be prominently positive. Similarly, if the major QRS vector is directed upward (as in the lateral leads), the ST-segment will be depressed and the T-wave will be inverted (see Figure 1.1). Loss of this characteristic pattern, often referred to as the "rule of appropriate discordance," is an electrocardiographic clue to acute MI in patients with LBBB.

KEY FACT | **… STEMI can be diagnosed on an ECG with LBBB … the ECG is more useful in ruling in the diagnosis than in excluding it.**

Using the GUSTO-1 database, Sgarbossa and colleagues developed electrocardiographic criteria for STEMI in the face of pre-existing LBBB [19]. These criteria, listed in Table 1.2, can be committed to memory, but are perhaps better recalled after examining a tracing that demonstrates the criteria (see Figure 1.2) and comparing it to the appearance of LBBB without ischemia (see Figure 1.1). Meeting the threshold criterion score of ⩾3 points (see Table 1.2) established the diagnosis of acute MI with 90% specificity. Others have reported problems with sensitivity and inter-rater reliability using the Sgarbossa criteria for acute MI in the presence of LBBB [20–22]. Smith and Whitwam argue that the sensitivity of the ECG for acute MI (as defined by CPK-MB elevation) *without* LBBB mirrors that of the ECG *with* LBBB – approximately 45% [23]. The important point to remember here is that the acute MI can be diagnosed on an ECG with LBBB, but that the ECG is more useful in ruling-in the diagnosis than in excluding it – just as is the case in patients with symptoms of ACS and no LBBB (i.e., normal conduction) on their presenting ECG.

Table 1.2 Sgarbossa's criteria for STEMI in the presence of LBBB [19].

ST-segment elevation ⩾1 mm concordant with QRS complex (score 5)
ST-segment depression ⩾1 mm in leads V1, V2, or V3 (score 3)
ST-segment elevation ⩾5 mm discordant to the QRS complex (score 2)

Score ⩾3 means patient is likely experiencing a STEMI; score of <3 means ECG is indeterminate and more information is needed.

VPR

Returning to the GUSTO-1 database, Sgarbossa and colleagues generated electrocardiographic criteria for acute MI in the presence of a VPR [24]. Notably, these criteria were derived from an extremely small subject pool – 17 patients (as opposed to the 131 who had served a parallel role in the data set for LBBB and STEMI discussed above). The criteria that performed best were not surprisingly the same ones that were published for acute MI and LBBB [19]. However, the most useful criterion for acute MI in the presence of VPR was that which performed least well in the LBBB data set – STE ⩾ 5 mm discordant to the QRS complex. Perhaps this is due to the fact that most ECGs with VPR feature very few principally positive QRS complexes; the vector generated by a ventricular pacing spike emanating from the right ventricular apex (where the pacing wire typically sits) results in predominantly negative QRS complexes in most if not all precordial leads and often in the inferior leads as well (see Figure 1.3). Thus, there is more "opportunity" to witness out-of-proportion discordant ST-segment elevation than there is to feature concordant ST-segment elevation or concordant ST-segment depression. However, both may be evident in acute MI in the presence of VPR (see Figure 1.4). And so, as with acute MI and LBBB, the ECG in the presence of VPR is more likely to rule in the diagnosis of acute MI than it is to rule it out.

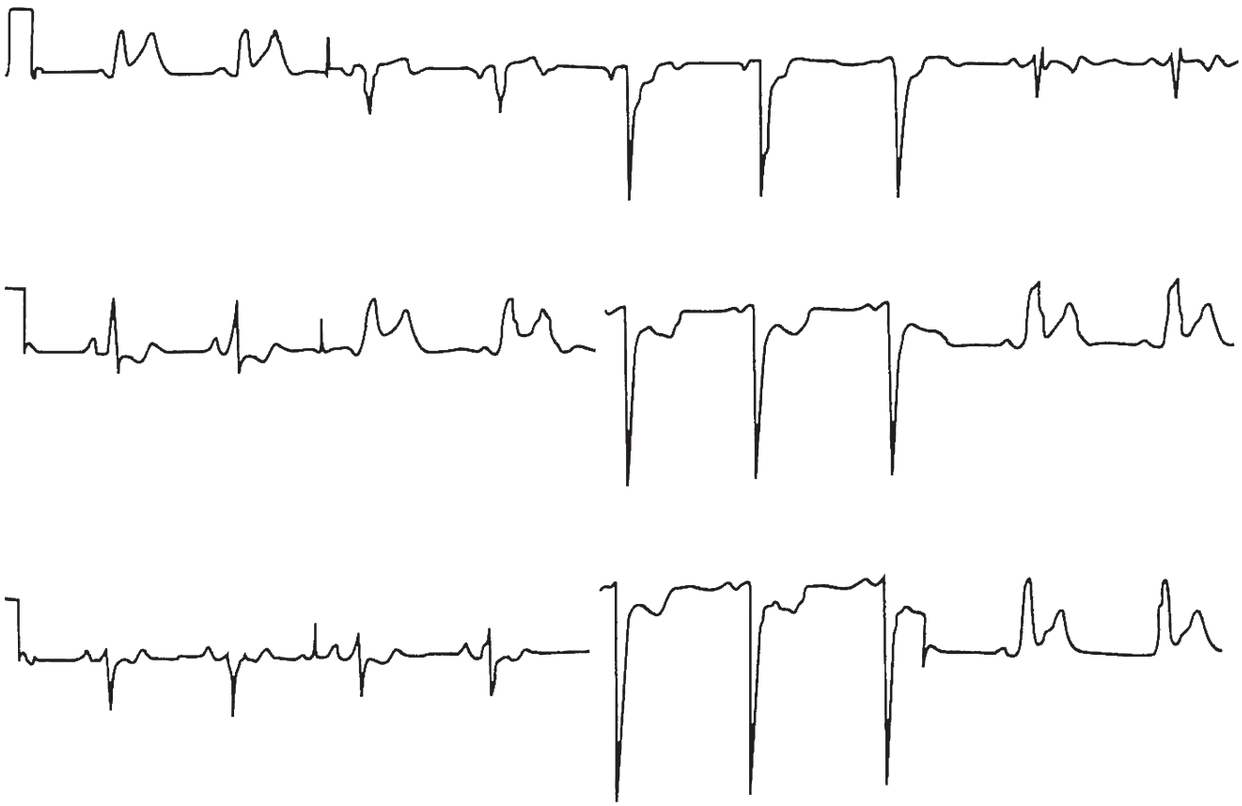

Figure 1.2 Acute MI in the presence of LBBB.
[Reproduced with permission from Elsevier; Brady WJ, Pollack ML. Acute myocardial infarction: confounding patterns. In: Chan TC, Brady WJ, Harrigan RA, et al. (eds). *ECG in Emergency Medicine and Acute Care*. Philadelphia: Elsevier Mosby, 2005, p. 183, Fig. 34-4.]. The ECG demonstrates concordant ST-segment elevation in leads I, aVL, V5, and V6 as well as concordant ST-segment depression in leads V1 to V3, violating the rule of appropriate discordance.

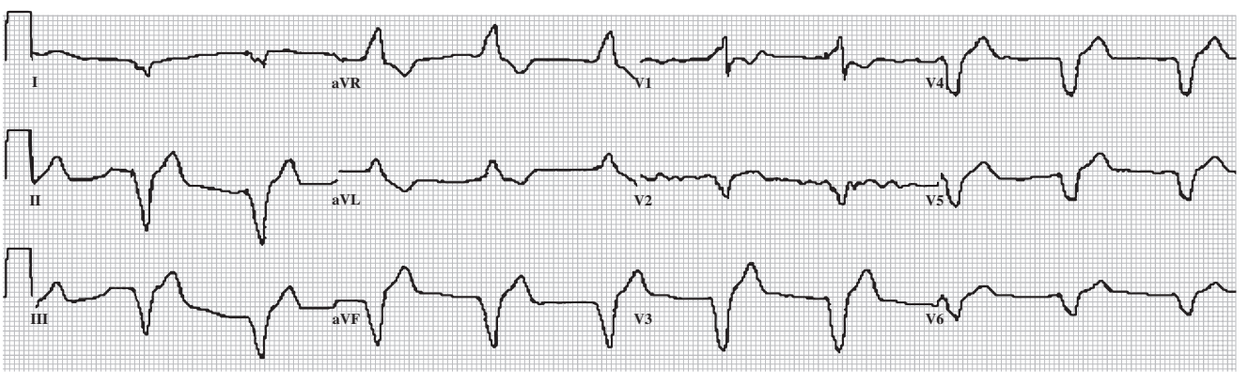

Figure 1.3 VPR:
This tracing shows a functioning ventricular pacemaker set at 60 bpm. Small-amplitude pacemaker spikes can be seen before the widened QRS complexes (these are best seen in leads II and V1 here). Note the predominance of negatively deflected QRS complexes – since 9 of 12 leads have negative QRS complexes, there is less opportunity for concordant ST-segment elevation – the criterion that functioned best in the study defining criteria for detection of acute MI with coexistent LBBB [19]. There is ample opportunity for the detection of discordant ST-segment elevation $\geqslant$ 5 mm, however; this is the criterion that performed best in the study which defined criteria for detection of acute MI with coexistent VPR [24]. There is no evidence of acute MI on this tracing, however.

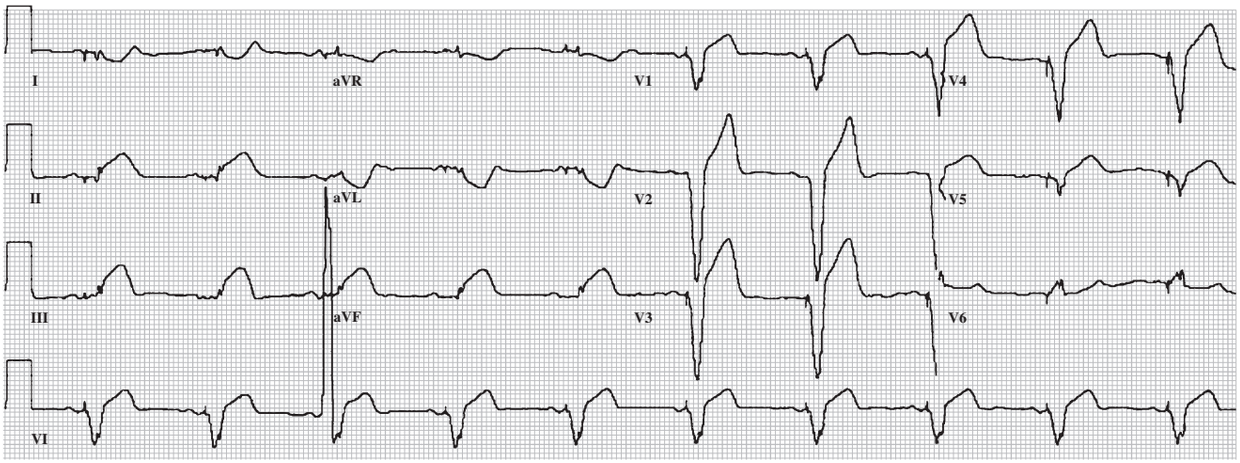

Figure 1.4 Acute MI in the presence of VPR.
[Reproduced with permission from Elsevier; Brady WJ, Pollack ML. Acute myocardial infarction: confounding patterns. In: Chan TC, Brady WJ, Harrigan RA, et al. (eds). *ECG in Emergency Medicine and Acute Care*. Philadelphia, Elsevier Mosby, 2005, p. 187, Fig. 34-10.]. The electrocardiogram demonstrates evidence of concordant ST-segment elevation in leads II, III, and aVF; and reciprocal ST-segment depression in leads I and aVL.

Pitfall | **Use of a "GI cocktail" to distinguish between cardiac versus non-cardiac chest pain**

Distinguishing gastroesophageal pain from ischemic chest pain can be difficult. Both may share similar characteristics such as dyspepsia and response to nitrates; however, one is an emergency and the other is not. A "GI cocktail" is sometimes used in the ED in an attempt to make this differentiation. Compositions vary, but a GI cocktail usually consists of a mixture of a liquid antacid, viscous lidocaine, and a liquid anticholinergic/barbiturate compound [25].

In one small study from the 1970s, Schwartz noted that the administration of a GI cocktail was highly reliable in differentiating ischemic chest pain from gastroesophageal pain. Sixty patients presenting with chest pain, epigastric pain, or both were treated with 20 ml of viscous lidocaine. None of the patients who obtained significant pain relief from the GI cocktail (37/60) were found to have myocardial ischemia. Among those who did not respond to the GI cocktail (23/60), myocardial ischemia or acute MI was diagnosed in more than half (13/23) [26].

More recently, Wrenn et al. performed a retrospective review of ED charts to determine the practice patterns regarding the administration of GI cocktails. During a 3-month period, 97 patients received a GI cocktail for various presenting complaints including abdominal pain (49), chest pain (40), and dyspnea (4). Over two-thirds of the patients (66/97) also received at least one other medication and the median time of administration of the other drug was 9 min before the GI cocktail. The most common medications given included opiates (56), nitroglycerin (22), and aspirin (10). Of the patients admitted for possible myocardial ischemia, 8/11 (73%) were noted to have some degree of relief after administration of a GI cocktail [27].

Beyond the research of Schwartz and Wrenn, the literature on the use of GI cocktails in the evaluation of chest pain is sparse. In one small case series, three patients diagnosed with acute MI had complete relief of their pain after administration of a GI cocktail [28]. One patient, however, did receive nitroglycerin in parallel with the GI cocktail. In another slightly larger case series, 7% of patients with ischemic chest pain got relief of their symptoms after receiving a GI cocktail [29].

Research on the use of the GI cocktail as a diagnostic test in the evaluation of chest pain is clearly limited. In addition, the interpretation of this test remains difficult because the GI cocktail is often administered soon before or after the administration of other potential pain relievers. One thing is clear: there is not enough evidence to suggest that the response of a patient with chest pain to a GI cocktail should in any way direct the disposition decision.

Pitfall | **Assumption that a normal ECG rules out cardiac ischemia**

When working through the differential diagnosis of chest pain, it is often said that the patient cannot be having an MI if ECG is normal. This is not true; in fact, no historical complaint, physical finding, or ECG pattern has a negative predictive value of 100% for MI. The patient may be less likely to be experiencing an MI if the ECG is normal, but more is needed than a normal ECG to discard the diagnosis. Furthermore, when considering the ECG and the literature behind this topic, a "normal" ECG must be strictly defined; that is,

the negative predictive value of a normal ECG differs from that of an ECG with non-specific changes.

KEY FACT | **6.4% of all patients with acute MI had a normal ECG.**

Data from the Acute Cardiac Ischemia-Time Insensitive Predictive Instrument (ACI-TIPI) trial highlights this issue. In that study, 889/10,689 patients were diagnosed with acute MI (by creatine kinase (CK)); 19 of those 889 were mistakenly discharged to home. Seventeen of those 19 (90%) had either a normal (2) or a non-ischemic (15) ECG. Four risks for inappropriate discharge were culled from that data; women <55 years old, non-white race, dyspnea as a chief complaint, and a normal ECG [30]. Combining data from two large studies totaling nearly 12,000 patients, of which nearly 2000 had an acute MI (again defined by CK criteria), Smith and colleagues [31] describe a concerning incidence of acute MI in patients with non-specific, and even normal, ECGs. Four hundred forty-two patients had a *non-specific* ECG yet had an acute MI – meaning 22.5% of all patients with acute MI had a non-specific ECG, and 8.6% of all patients with a non-specific ECG ended up having an acute MI. The normal ECG lessened the likelihood of acute MI, but the numbers here were still impressive: 125 patients had a *normal* ECG yet had an acute MI – translating to 6.4% of all patients with acute MI had a normal ECG, and 3.4% of all patients with a normal ECG had an acute MI. Smith et al. stress several important issues with these studies. They were performed in the pre-troponin era; it is unclear if only initial ECGs were included; and these studies did not differentiate ongoing chest pain from a history of recent chest pain [31].

Singer and associates showed that the negative predictive value of a "normal" ECG for acute MI does not improve as time passes from symptom onset – which seems counterintuitive. Analyzing data from 526 patients, 104 (20%) of whom had acute MI, they restricted their study to the initial ECG, yet did not report if the ischemic symptoms were ongoing. They found that the ECG maintained a 93% negative predictive value for acute MI at 0–12 h after the onset of symptoms [32]. Here, a "normal" ECG included those with non-specific ST-segment/T-wave changes as well as isolated fascicular blocks, illustrating again that this literature is at time confusing, in that the serum biomarker used to define MI varies, as does the definition of a "normal" ECG. What is clear is that the EP must not regard a non-specifically abnormal, or even a normal, ECG as proof-positive that a given patient is not presenting with symptoms of acute MI. Furthermore, a discussion of this issue is notably without reference to the predictive ability of the ECG in excluding unstable angina. The literature discussed above does not include this entity, and due to a lack of a clear gold standard definition of unstable angina, this remains a murky area of concern.

Pitfall | **Discharge of patients after a single set of negative cardiac enzymes**

In recent years, the role of cardiac markers in the diagnosis and treatment of patients with chest pain and suspected ACS has evolved considerably. A recent consensus guideline of the European Society of Cardiology (ESC) and the American College of Cardiology (ACC) redefined acute MI and highlighted the central importance of cardiac markers [33].

KEY FACT | **[Cardiac enzymes] can only detect myocardial cell death but not ischemia.**

Cardiac markers provide a non-invasive means of determining whether myocardial damage has occurred. When ischemia gives way to infarction, the myocardial cell membrane is disrupted and various chemical markers are released into the systemic circulation. The timing of the rise of each cardiac marker is variable. Myoglobin is elevated within 2–4 h after acute MI and rapidly returns to baseline. CK-MB begin rising in the 3–6 h range and falls below the acute MI range at about 2 days. Troponin also begins rising at about 3–6 h post-infarction and gradually returns to baseline over approximately 1 week. In the past, elevation of the CK-MB fraction (CK-MB) was considered the gold standard in diagnosing acute MI. More recently, the cardiac troponins (I or T) have become the preferred cardiac markers for identifying myocardial damage. Regardless of which cardiac enzyme is used, however, it is important to remember that these tests can only detect myocardial cell death but not ischemia.

Cardiac troponins are highly sensitive for the detection of myocardial injury. A single troponin measurement at the time of presentation, however, appears to have limited utility in ruling out acute MI. The sensitivity of a single isolated troponin has been reported to be anywhere from 4% to 100% [34]. Variation in test sensitivity is explained by the timing of the troponin testing. Longer symptom duration yields higher sensitivity. Serial testing, especially when performed at least 6 h after symptom onset, markedly improves the sensitivity of troponins for acute MI. The evidence supporting the use of cardiac troponins in the diagnosis of non-MI ACS is limited. In up to 33% of patients diagnosed with classic unstable angina, cardiac troponins may be slightly elevated [35]. Current thought is now that these "enzyme leaks" are likely caused by micro-infarcts. In patients with ACS, increased troponin levels appear to be an indicator of increased risk for acute MI and death [36].

Once considered the gold standard, CK-MB is outperformed by cardiac troponins in terms of both sensitivity and specificity for acute MI. The sensitivity of a single CK-MB determination in diagnosing acute MI is also dependent on the elapsed time from symptom onset. The overall sensitivity of a single isolated CK-MB has been reported to be anywhere from 14% to 100% [34]. If testing occurs within 3 h

of symptom onset, the sensitivity of CK-MB is only 25–50%. After 3 h, the sensitivity is increased, ranging from 40% to 100%. Because CK-MB rises relatively quickly, serial testing, even over a relatively short time period, has been shown to increase the sensitivity considerably. In one study, a change in a 2-h CK-MB level had a sensitivity of 93.2% for acute MI [37].

Myoglobin is found in both skeletal and cardiac muscle, thereby limiting its specificity. Because myoglobin is rapidly released after myocardial injury, it has been identified as a potential early indicator of acute MI. The sensitivity of a single myoglobin at the time of presentation, however, has been noted to be as low as 21% [34]. Serial testing significantly improves the diagnostic utility of myoglobin. In one study, doubling of the level 1–2 h after the initial measurement was nearly 100% sensitive for the diagnosis of acute MI [38].

More recent studies have looked into the use of serial measurements of multiple markers. McCord et al. noted that when myoglobin and troponin were drawn at presentation and at 90 min, the sensitivity for acute MI was 96.9% and the negative predictive value was 99.6% [39]. Ng et al. reported similar results utilizing a three-marker approach and a 90-min accelerated pathway, reporting nearly 100% sensitivity and 100% negative predicative value for acute MI [40]. It is critical to remember, however, that cardiac enzymes will not be reliably elevated in the setting of cardiac ischemia.

KEY FACT | **Single determinations of cardiac markers at the time of presentation appear to be inadequate to exclude the diagnosis of acute MI and provide no information about the possibility of cardiac ischemia.**

Ultimately, determining the disposition of patients with suspected ACS requires the EP to gather and interpret many pieces of information. The combined data from the history, physical, ECG, and cardiac markers should guide the EP in managing a patient with chest pain or suspected ACS. Single determination of cardiac markers at the time of presentation appears to be inadequate to exclude the diagnosis of acute MI and provides no information about the possibility of cardiac ischemia.

Pitfall | Over-reliance on a "classic" presentation for diagnosis of AD

Acute dissection of the thoracic aorta is, unfortunately, both challenging to diagnose and potentially lethal if the diagnosis is missed. Furthermore, misattributing the chest pain of acute AD to ACS can lead to disastrous results as anticoagulant and fibrinolytic therapy are staples of the treatment of the latter [41, 42]. Classically, the patient with AD has a history of hypertension and experiences the sudden onset of profound ripping or tearing chest pain that radiates to the back (interscapular region – perhaps migrating to the low back) [43]. It is important to note, however, that the absence of this history in no way excludes the diagnosis; symptoms may be atypical – or may even be absent. Indeed, one report [43] looking at pooled data from 16 studies, found a history of any pain to be only 90% sensitive for the diagnosis of acute AD (CI 85–94%) (see Table 1.3), with more precise and classic pain descriptions faring less well. Data reported from the International Registry of Acute Aortic Dissection (IRAD) [44] included 464 patients from 12 referral centers; some type of pain was reported in 94% of Type A dissections and 98% of Type B dissections; it was chest pain in 79% and 63%, respectively. The pain was abrupt in onset in roughly 85% of all dissections, and it was characterized as severe or the "worst ever" in 90% of both groups. Interestingly, it was classified as "sharp" (64%) more often than "ripping or tearing" (51%) [44]. Since the classic description has been well-documented to be less than universal, knowledge of atypical presentations of AD together with an awareness of risk factors enhances diagnostic capability.

Risk factors for AD [43].

- Hypertension
- Bicuspid aortic valve
- Previous cardiac surgery, particularly aortic valve replacement
- Coarctation of the aorta
- Marfan syndrome
- Ehlers–Danlos syndrome
- Turner syndrome
- Giant cell arteritis
- Third-trimester pregnancy
- Cocaine abuse
- Trauma

Table 1.3 Sensitivity of clinical history of pain in acute thoracic AD [43].

Pain Description	Sensitivity (%)	Confidence Intervals (%)
Any pain	90	85–94
Chest pain	67	56–77
Anterior chest pain	57	48–66
Posterior chest pain	32	24–40
Back pain	32	19–47
Abdominal pain	23	16–31
Sudden-onset pain	84	80–89
Severe pain	90	88–92
Ripping/tearing pain	39	14–69

So how do patients with acute AD present, if not with chest pain, or indeed any pain? *Syncope* was reported in 13% of Type A AD in IRAD; 2% of those patients did not have any pain or neurological findings (only 4% of Type B dissections presented with syncope) [44]. Others have reported syncope (at times painless) in acute AD as well [42, 45–47]. Another common diagnosis associated with acute AD is *acute stroke*, this being mediated by flap occlusion of a carotid artery in Type A dissection. IRAD data found 17/289 (6%) to present with acute stroke symptoms [44]; the more broadly defined finding of a new focal neurologic deficit was reported in 17% of pooled studies [43]. The neurologic deficit may be peripheral rather than central, due to the site of occlusion; motor and sensory findings in a lower extremity have been reported with acute AD in the absence of pain [48]. AD may also present as an acutely painful ischemic leg or as acute chest pain radiating to the back with simultaneous incontinence and bilateral lower extremity paralysis. Other atypical presentations of acute AD include abdominal or flank pain, hoarseness (recurrent laryngeal nerve compression), swelling and bruising of the neck, cough (mainstem bronchus compression), dysphagia (esophageal compression), Horner's syndrome (sympathetic chain compression), pulsatile sternoclavicular joint, superior vena cava syndrome, and testicular/groin pain [43–46, 49–51].

Pitfall | **Use of the chest X-ray to exclude the diagnosis of AD**

AD is the most common fatal condition involving the aorta [45]. Left untreated, about 75% of patients with AD involving the ascending aorta will die within 2 weeks. If diagnosed early and treated successfully, the 5-year survival rate approaches 75% [49]. Because early diagnosis is so important, the EP must maintain a high level of suspicion for AD. In the setting of chronic hypertension, AD should be considered in any patient with sudden and severe chest or back pain.

When AD is being considered, a chest X-ray should be obtained and examined for abnormalities of the aortic silhouette. This is best accomplished with a standing posteroanterior (PA) view. Portable anteroposterior (AP) views may falsely enlarge the cardiomediastinal silhouette and lateral chest X-rays rarely show evidence of AD [52]. Many radiographic findings have been noted in AD but unfortunately the majority of these findings are subjective and not well defined. Although the chest X-ray may suggest the diagnosis, it is rarely definitive.

Radiographic findings in AD may include widening of the mediastinum, abnormalities of the aortic knob and aortic contour, increased aortic diameter, left-sided pleural effusion, tracheal deviation, and esophageal deviation [49, 53]. The double density sign is observed when the false lumen is less radiopaque than the true lumen [49]. The calcium sign, consisting of the displacement of the aorta's intimal calcification from the aortic knob by 1 cm or more, is highly suggestive of AD but is only present in a minority of cases [43, 49].

Widened mediastinum, defined as a measurement $\geqslant 8$ cm at the level of the aortic knob, is considered by many to be the most sensitive radiographic finding. According to one study, widening of the mediastinum and widening of the aortic knob were the only two radiographic features of significance in predicting dissection [54]. A tortuous aorta, common in hypertensive patients, may widen the mediastinum and be hard to distinguish from AD. Other causes of mediastinal widening include adenopathy, lymphoma, and an enlarged thyroid.

KEY FACT | **A widened mediastinum was noted in only 62% of all patients and an abnormal aortic contour was noted in only 50% of all patients.**

The IRAD, consisting of 12 international referral centers, published data on 464 patients diagnosed with AD. A widened mediastinum was noted in only 62% of all patients and an abnormal aortic contour was noted in only 50% of all patients (see Table 1.4). However, 21.3% of the patients were noted to have an absence of both a widened mediastinum and an abnormal aortic contour and 12.4% did not have any abnormalities noted on their chest X-rays [44].

In a meta-analysis of 13 studies, which included 1337 radiographs of patients diagnosed with AD, the sensitivity of plain chest X-rays was noted to be 90%. Absence of a widened mediastinum and abnormal aortic contour, in particular, decreased the probability of disease (negative LR, 0.3; 95% CI, 0.2–0.4). However, no specific radiographic abnormality was dependably present and therefore the absence of any one particular finding could not be used to rule out AD [43].

When asked to evaluate the presenting chest X-ray of patients with and without AD in a blinded manner, physicians from various specialties read 84% of the normal films as "not suspicious" for AD and only 73% of the AD films as "suspicious" for AD [55]. The most frequent finding identified on the AD chest X-rays was a widened mediastinum (38%). In another study, EP read 32% of AD chest X-rays as

Table 1.4 Chest X-ray findings in AD (Types A and B) [44].

Radiographic Finding	% Present
No abnormalities	12
Absence of widened mediastinum or abnormal aortic contour	21
Widened mediastinum	62
Abnormal aortic contour	50
Abnormal cardiac contour	26
Pleural effusion	19
Displacement/calcification of aorta	14

"normal" and noted a widened mediastinum only 10% of the time [45].

Although an apparently normal chest X-ray may decrease the likelihood of AD, it cannot be used exclusively to rule out the diagnosis of AD. If the clinical history and/or physical examination raise the suspicion for AD, further imaging should always be pursued.

Pitfall | **Over-reliance on the presence of classic pleuritic chest pain and dyspnea in the evaluation of PE**

PE remains a common cause of morbidity and mortality. Because so many cases of PE go undiagnosed, the actual incidence of PE remains unknown. Most cases of fatal PE are not actually diagnosed until autopsy. Despite advances in diagnostic methods and treatment over the last several decades, mortality rates have changed very little [56].

When promptly diagnosed and treated, PE rarely causes death. In fact, less than 10% of deaths caused by PE occur in those patients in which treatment is initiated. The majority of deaths (90%) occur in patients who are never treated because the diagnosis is never made [57].

The clinical presentation of PE is often subtle and many patients may actually be asymptomatic. The true rate of asymptomatic PE in the general population is unknown. In one study, of 387 patients diagnosed with PE, 34% of the patients were asymptomatic [56]. Atypical presentations may also occur. Patients may present with non-pleuritic chest pain, abdominal pain, back pain, fever, cough, wheezing, palpitations, and syncope [56, 58].

The classic triad of pleuritic chest pain, dyspnea, and hemoptysis is not only non-specific but it is also not sensitive. At most it is present 20% of the time [58]. The combination of chest pain, dyspnea, and tachypnea has been noted be as high as 97% sensitive for PE in various studies. However, the patients enrolled in these studies had symptoms suggestive of PE. Thus, patients with atypical features and asymptomatic patients were excluded [56].

KEY FACT | **The presence of pleurisy is neither sensitive nor specific for PE. In one study … pleuritic chest pain occurred in only 44% of patients with PE versus 30% of patients without PE.**

Pleuritic chest pain has long been considered one of the classic symptoms of PE. However, its presence is neither sensitive nor specific for PE. In one study, for example, pleuritic chest pain occurred in only 44% of patients with PE versus 30% of patients without PE. In fact, the pain was described instead as substernal chest pressure, typical of cardiac ischemia, in 16% [59]. Chest pain is more common if pulmonary infarction has occurred because of pleural irritation. Pulmonary infarction is more likely to occur in older patients with underlying cardiopulmonary disease [58]. In one study, nearly three quarters of patients with proven PE had pulmonary infarction [56].

Patients with PE are more likely to report dyspnea. In one study, sudden onset of dyspnea was by far the most frequent symptom in patients with PE, occurring in nearly 80% of the patients diagnosed with PE [59]. In another study, as many as 92% of patients diagnosed with PE reported dyspnea [60]. However, the severity of dyspnea is not always related to the degree of obstruction within the pulmonary vasculature. It has been suggested that many patients can be asymptomatic with as much as a 50% obstruction [61].

The signs and symptoms of PE are relatively non-specific and therefore the clinical recognition of PE is difficult. Although pleuritic chest pain and dyspnea make the diagnosis of PE more likely, the absence of these symptoms should not rule out the diagnosis.

Pitfall | **The use of ECG findings to rule in or rule out PE**

Patients with PE typically present with some combination of dyspnea, chest pain, tachypnea, and tachycardia – yet as with most illnesses, there is no combination of findings on the history and physical examination that either clinches or excludes the diagnosis. Thus, the EP looks to other easily obtainable tests (e.g., ECG, chest X-ray, D-dimer assay) when confronted with a patient with these signs and symptoms. The $S_1Q_3T_3$ pattern on ECG has long been linked with the diagnosis of PE, yet the literature suggests it is neither sensitive nor specific for PE.

Roughly 70 years ago, the $S_1Q_3T_3$ pattern was first reported in a series of seven patients with acute right heart strain secondary to PE, and was defined as such: an S-wave in lead I and a Q-wave in lead III with an amplitude of at least 0.15 mV (1.5 mm), and an associated inverted T-wave in lead III [62]. Others have avoided a strict criterion amplitude for these findings, or have used a variation of this finding, when looking at the ECG in PE [59, 63, 64]. Combining their data with those of three other studies, Ferrari and colleagues found the incidence of $S_1Q_3T_3$ to range from 12% to 50% in patients with *confirmed* PE [65]. Others stress the importance of looking at the incidence of a finding (such as $S_1Q_3T_3$) in patients with *suspected* PE (which generalizes more readily to our situation in the ED, where we are seeking a diagnosis) – where it has been found with equivalent frequency (approximately 12%) in patients with and without PE [66]. In either patient population, the $S_1Q_3T_3$ pattern is clearly not sensitive or specific for PE.

KEY FACT | **Sinus tachycardia was found in only 8–69% of patients (with PE).**

Another ECG finding that is classically linked with PE – sinus tachycardia – should be recognized as less than universal; sinus tachycardia was found in only 8–69% of patients over six studies [67]. Other electrocardiographic findings occur at relatively low rates as well, including right atrial strain (2–31%) and right bundle branch block (6–67%) [67]. There are scattered reports of other entities, including atrial fibrillation and flutter, new changes in frontal plane QRS axis (especially rightward shift), clockwise shift in the precordial transition zone (i.e., toward the left precordial leads), low QRS voltage, ST-segment depression, and $S_1S_2S_3$ [63, 67, 68].

KEY FACT | **Precordial T-wave inversion was the most common finding, occurring in 68% of patients with confirmed PE.**

So what ECG finding, if any, should be linked with PE? Ferrari [65] found precordial T-wave inversion was the most common finding in their series of 80 patients, occurring in 68% of patients with confirmed PE. The frequency of this finding exceeded those of sinus tachycardia (26%) and $S_1Q_3T_3$ (50%) in their series.

Two points should be emphasized with regard to this topic. First, the literature on the incidence of any ECG finding in PE comes principally from populations where the people are known to have the disease – thus they may have more obvious disease (i.e., large PEs) since they entered the subject pool when someone made the diagnosis. Second, ECG changes that resemble cardiac ischemia, especially T-wave inversions, can occur in patients with PE. Physicians should never rule in or rule out PE simply based on ECG finding.

Pitfall | **Failure to differentiate pericarditis from other chest syndromes**

On the surface, pericarditis seems as though it would be easy to recognize. Classically, pericarditis features the rather sudden onset of progressive, central, pleuritic chest pain that is worse with lying supine and improved with sitting up and leaning forward. On physical examination, a mono-, di-, or tri-phasic pericardial friction rub will be heard best when the patient is sitting up and leaning forward. The ECG shows diffuse ST-segment elevation, usually with PR-segment depression, while lead aVR (due to its opposite vector polarity) often demonstrates PR-segment elevation with ST-segment depression [69]. In actuality, however, acute pericarditis may be the great masquerader, in that historical features may vary, the elusive rub may be difficult to capture with the stethoscope, and the ECG bears some similarity to other syndromes, most notably ACS and benign early repolarization (BER).

Confusingly, pericarditis shares historical characteristics with other diseases such as pleurisy, PE, pneumothorax, pneumonia, acute MI, AD, and chest wall pain. All may feature pleuritic chest pain; the location of the pleural irritation may localize the pain away from the heart, moving other diagnoses up on the differential hierarchy. Proximal AD may be complicated by the development of a pericardial effusion, as might pericarditis – thus sudden onset of chest pain plus pericardial effusion on bedside ultrasound does not necessarily equal either disease. Like acute MI, the chest pain in pericarditis may radiate to the neck or shoulder area; however, radiation to the trapezius ridge(s) suggests pericarditis, because both phrenic nerves course through the anterior pericardium and innervate each trapezius ridge [69, 70]. Thus, the history is important but often insufficient in distinguishing the cause of the pain.

How often a pericardial rub is detectable in acute pericarditis is really not known; rubs are notoriously transient and unpredictable, although if present, they are virtually pathognomonic for pericarditis [69, 71, 72]. Rubs vary in description (e.g., rasping, creaking, scraping, grating, scratching, squeaking) and seem to overlie normal heart sounds. Typically best heard along the mid-to-lower left sternal border, rubs are best accentuated by positioning that brings the heart closer to the anterior chest wall – sitting up and leaning forward, or examination of the patient on all fours [70–72]. Experts differ on which phase of respiration optimizes auscultation of the rub – end-expiration [70, 72] or inspiration (if there is increased pericardial fluid) [71]. Pleural rubs are best distinguished from pericardial ones by location and phasic variation – the former varies with breathing, the latter with the heart cycle [70, 72]. If three phases occur with a pericardial rub, they are attributed to atrial systole, ventricular systole, and early diastolic filling [70, 71]. Signs of pericardial tamponade – hypotension, tachycardia, elevated jugular venous distension, muffled heart sounds, pulsus paradoxus – should be sought; however, these will be absent in pericarditis without sufficient effusion to cause or approach tamponade physiology. Tamponade may be expected in approximately 15% of cases of idiopathic origin, and as many as 60% of cases due to neoplastic, purulent, or tuberculous causes [70].

It is important to realize that laboratory diagnosis offers another juncture for confusion in this disease. Elevation of the peripheral leukocyte count, sedimentation rate, and C-reactive protein are neither sensitive nor specific. Disturbingly, serum troponin levels are elevated in 35–50% of patients – due to either epicardial inflammation or, more rarely, myocardial involvement in the form of myocarditis. Serum troponin elevation varies directly with the magnitude of ST-segment elevation on the ECG [70, 73]. Thus, three hallmarks of acute MI – chest pain radiating to the neck and shoulder, elevation of serum troponin, and ST-segment elevation on the ECG – may be seen with pericarditis. With that being said, the subtleties of the ST-segment elevation are usually helpful in distinguishing the two diseases.

ST-segment elevation, at times subtle and at times pronounced, can be seen in both acute MI and acute pericarditis.

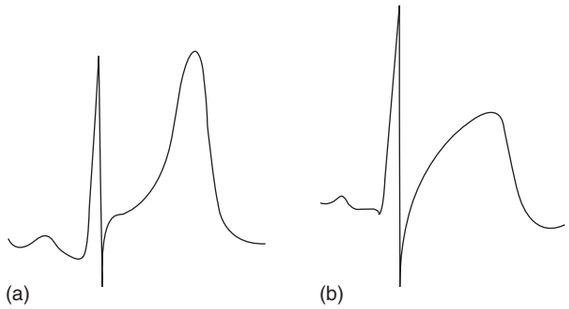

Figure 1.5 ST-segments in acute pericarditis and acute MI. [Reproduced with permission from Elsevier; Chan, TC. Myopericarditis. In: Chan TC, Brady WJ, Harrigan RA, et al. (eds). *ECG in Emergency Medicine and Acute Care*. Philadelphia: Elsevier Mosby, 2005, p. 203, Fig. 37-7.]. (a) demonstrates concave ST-segment elevation typical of acute pericarditis. Acute MI may also demonstrate this same type of ST-segment morphology. However, the presence of concurrent PR-segment depression confirms the diagnosis of acute pericarditis; (b) demonstrates convex ST-segment elevation highly specific for acute MI.

Morphologically, the ST-segments of acute pericarditis are classically concave upward, whereas the ST-segments in acute MI can be concave upward, straight, or convex upward (see Figure 1.5) [74]. One morphologic feature of the ST-segment that distinguishes acute MI from pericarditis is reciprocal ST-segment depression; with the former, this dramatically increases the specificity of the ECG. Reciprocal ST-segment depression may logically appear on the ECG in the area representing the opposing electrical view from that of the infarcted territory; for example, in inferior (leads II, III, and aVF) STEMI, lead aVL (which is directed 150° opposite to lead III in the frontal plane) may demonstrate ST-segment depression (which also may be seen, to a lesser extent, in lead I) [75]. Save for lead aVR and at times lead V1, the presence of ST-segment depression on the ECG in acute pericarditis is extremely rare [71, 75, 76]. This emphasizes another key distinction on the ECG between acute MI and pericarditis – the former features regional abnormalities that reflect infarct territory of the affected coronary artery, whereas most cases of acute pericarditis demonstrate diffuse ST-segment elevation. Similarly, regional development of Q-waves in the company of ST-segment elevation favors acute MI [72, 75].

PR-segment depression is another distinguishing feature of the ECG in acute pericarditis. As with ST-segment changes, diffuse changes suggest pericarditis; focal, regional changes do not. PR-segment depression is itself of undetermined specificity, and can be seen in atrial infarction. Leads II, V5, and V6 often feature the most obvious PR-segment depression; lead aVR may again behave oppositely, revealing PR-segment elevation in acute pericarditis. PR-segment depression may coincide with or even precede ST-segment elevation in pericarditis [69, 71, 72, 76]. PR-segment depression is most specific for acute pericarditis when it occurs in multiple leads; however, the finding is transient and is therefore not universally present in all patients with pericarditis.

KEY FACT | **T-waves do not invert in pericarditis until the resolution of the ST-segment elevation phase, whereas in acute MI, they may invert while the ST-segments remain elevated.**

Like the PR- and ST-segments, the T-wave behaves differently in acute MI and pericarditis. While both diseases can feature T-wave inversions following ST-segment elevation, there is an important distinction: T-waves do not invert in pericarditis until the resolution of ST-segment elevation phase, whereas in acute MI, they many invert while the ST-segments remain elevated [70, 77]. This characteristic serves to emphasize the value of serial ECG sampling; regional ST-segment evolution, and the timing of dynamic T-wave changes will aid in securing a diagnosis.

Stepping away from the electrocardiographic similarities and differences of acute MI and pericarditis, some attention must be given to differentiating acute pericarditis from BER on the ECG – should a patient with baseline BER on the ECG present with chest pain consistent with pericarditis. Both pericarditis and BER feature diffuse ST-segment elevation with concave upward morphology. Marked PR depression may occur in pericarditis, whereas mild PR depression, as a function of the natural process of atrial depolarization [75], may be seen on any ECG, including those with BER. One useful distinguishing factor is that the ST-segment elevation of BER is stable over time (i.e., should be present on old tracings), whereas the ST-segment elevation of acute pericarditis, though not given to minute-to-minute change (unlike ACS), should be absent on old ECGs, should they be available for comparison [78]. Another useful distinguishing characteristic focuses on the relative amplitudes of the J point and the T-wave. If the height of the J point in lead V6 measures more than 25% of the amplitude of the corresponding T-wave peak, the ECG diagnosis is likely pericarditis, rather than BER. The end of the PR-segment should be used as a baseline when making this comparison [79, 80]. Restated, pericarditis yields more ST-segment elevation per T-wave amplitude (in lead V6) than does BER (see Figure 1.6).

Pericarditis can be difficult to diagnose – with similarities to other diseases in history, laboratory test results, and electrocardiographic appearance. The evidence must be weighed in total in difficult cases. Furthermore, a case can be made for urgent echocardiography as a diagnostic adjunct. Since any form of pericardial inflammation can lead to pericardial effusion, echocardiography is recommended when making the diagnosis of pericarditis [69]. Thus, the degree of effusion can be assessed, which has important implications for treatment and disposition. Moreover, regional wall motion abnormalities may be seen in acute MI, whereas they would not be expected (unless pre-existing) in pericarditis or BER. Echocardiography is also the best test to detect ventricular aneurysm, which also may cause ST-segment elevation on the ECG [81].

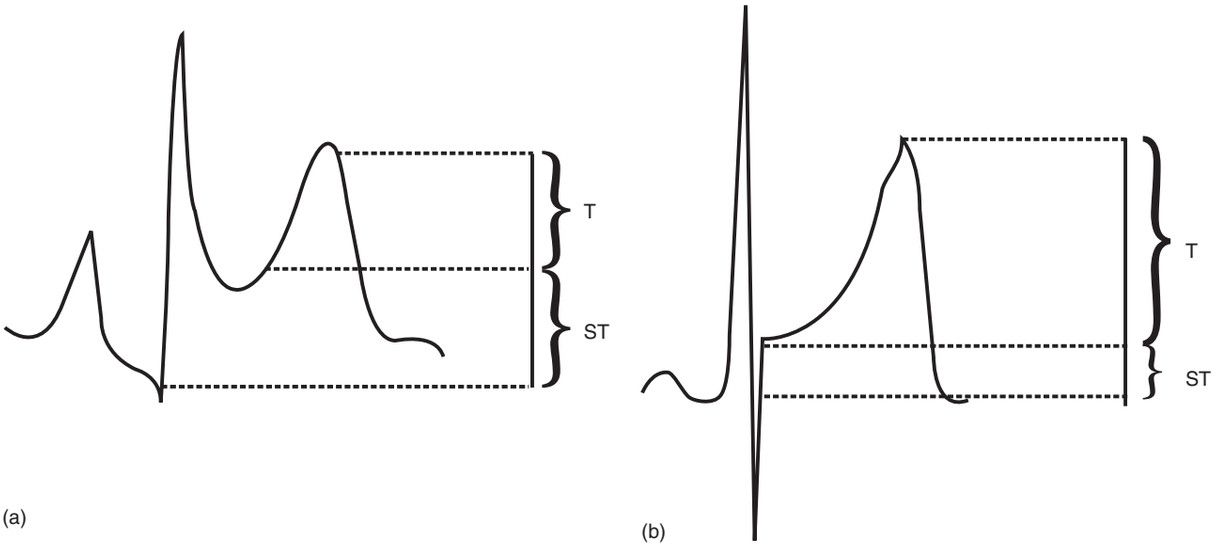

Figure 1.6 Pericarditis versus BER.
Acute pericarditis and BER can be differentiated on the ECG by determining the ratio of the ST segment and T wave amplitudes. The terminal portion of the PR segment should be used as the baseline for the purpose of performing this calculation. Typically lead V6 is evaluated. A ratio (ST segment/T wave) of $\geqslant 0.25$ favors a diagnosis of pericarditis (a), whereas BER (b) is more likely if the ratio is <0.25. [79]

Pitfall | **Assumption that the standard chest X-ray completely rules out pneumothorax**

Pneumothorax is a common condition affecting all age groups. It may occur spontaneously or as the result of trauma. Primary spontaneous pneumothoraces most often occur in tall, young males without any underlying parenchymal lung disease. A history of smoking is very common (90%). Secondary spontaneous pneumothoraces occur in older patients with known lung disease, primarily chronic obstructive pulmonary disease (COPD). Depending on the severity of the underlying disease and the size of the pneumothorax, secondary spontaneous pneumothoraces can be life threatening. In fact, COPD patients have a 3.5 fold increase in mortality when spontaneous pneumothoraces occur. Tension pneumothorax in the absence of trauma is relatively rare, and is associated with spontaneous pneumothorax in only 1–3% of cases [82]. Traumatic pneumothoraces occur as the result of blunt or penetrating trauma or as complication of a medical procedure. Tension pneumothoraces are much more likely to develop in the setting of trauma.

The classic symptoms of pneumothorax include pleuritic chest pain and shortness of breath. However, nearly one-third of patients (30%) may be asymptomatic or present only with minor complaints [82]. On physical examination, there may be decreased chest wall movement, hyperresonance to percussion, and decreased or absent breath sounds on the affected side.

The chest X-ray is the primary diagnostic modality used to screen for pneumothorax. The overall sensitivity of chest X-rays in detecting pneumothorax has been reported to be as high as 80%. Diagnosis is typically made by identifying a visceral pleural line on an upright, inspiratory chest X-ray. This line is seen initially at the apex of the lung and along the lateral pleural margin. The absence of lung markings peripheral to the pleural line may also be noted. With small pneumothoraces, an overlying rib may obscure the pleural line. Skin folds, the inner borders of the scapula, large bullae, and indwelling lines may all be mistaken for a pneumothorax. In most cases, an upright, inspiratory chest X-ray is the only study required to make the diagnosis.

If pneumothorax is strongly suspected and a pleural line is not visualized, an expiratory chest X-ray can be obtained. In full expiration the lung density is increased while the volume of air in the pleural space remains constant, in theory making it easier to detect a pneumothorax. A recent randomized controlled trial, however, revealed no difference in the ability of radiologists to detect pneumothoraces on inspiratory and expiratory films [83]. A lateral decubitus film can also be obtained. Although a lateral decubitus chest X-ray may be diagnostic, when clinically feasible, an upright chest X-ray is the procedure of choice for suspected pneumothoraces [84, 85].

Although the standard chest X-ray is usually sufficient to diagnose a pneumothorax, the literature demonstrates that missed pneumothoraces are still relatively common [85]. In 200 intensive care unit patients, 47 patients (23.5%) had missed pneumothoraces on routine chest X-rays [86]. In one study of 90 trauma patients, the initial supine chest X-ray failed to detect pneumothorax in 35 patients (39%) [87]. In another study of 103 severely injured patients with blunt

trauma, 27 (26%) had pneumothoraces missed on their initial chest X-ray only to be picked up on thoracic CT [88]. In yet another study, one-third of all traumatic pneumothoraces were missed on the initial chest X-ray and diagnosed on abdominal CT [89]. If the initial chest X-ray is inconclusive and there is a significant suspicion of pneumothorax, CT imaging should be pursued in any high-risk patient group (COPD, trauma, mechanically ventilated). As the diagnostic sensitivity of a test (chest CT) increases, the issue of clinical relevance emerges. Clearly some trivial pneumothoraces found only on chest CT need no treatment.

Pitfall | **Excluding the diagnosis of Boerhaave's syndrome due to an absence of antecedent retching or vomiting**

First described by the Dutch physician Herman Boerhaave in 1724, Boerhaave's syndrome refers to rupture of the esophagus – and is associated with high morbidity and mortality. At times referred to as spontaneous rupture of the esophagus, Boerhaave's syndrome is probably best thought of as rupture due to the development of a tear after a rise in the intraluminal pressure of this structure. The classic triad for this syndrome includes forceful emesis, chest pain, and subcutaneous emphysema. Patients usually appear very ill, prefer to sit up and lean forward, and may have lateralizing pulmonary findings on examination (rales, wheezing, decreased breath sounds) in addition to the subcutaneous emphysema, if it is present. Chest X-ray abnormalities include atelectasis, infiltrates, and pleural effusion, usually on the left because 90% of cases are due to a tear in the left posterolateral wall of the lower third of the esophagus, which communicates with the left pleural cavity in 80% of cases. Pneumomediastinum and hydropneumothorax may be apparent on the chest X-ray as well. Definitive diagnosis is usually made by computed tomographic scan of the thorax or by esophagram, although false negative studies may occur with either [90–93].

KEY FACT | **Antecedent retching or vomiting was absent in 21% of cases of Boerhaave's syndrome … the diagnosis should not be excluded in the absence of this historical feature.**

In one literature review [90] antecedent retching or vomiting was absent in 21% of cases of Boerhaave's syndrome. Thus, it should be emphasized that the diagnosis should not be excluded in the absence of this historical feature. Indeed, Boerhaave's syndrome has been reported after a variety of events, some less dramatic than others. Belching [94], simply swallowing a sandwich [95], violent cough [96], defecation, childbirth, weight lifting, asthma attacks, seizures, and blunt abdominal trauma [97, 98] have all been reported as precipitant events for Boerhaave's syndrome. It has been reported to complicate the vomiting associated with acute MI [99]. It should especially be considered in patients with chest pain after a recent esophageal endoscopic procedure. Notably, it is also seen in children [97, 98], and may present with a right-sided esophageal tear – leading to findings on physical examination and chest X-ray on the right side rather than the classic occurrence on the left [91, 98]. Thus, as with most diseases, atypical isolated features of the history and physical examination, and even negative initial diagnostic tests, should not dissuade the EP from pursuing the diagnosis of Boerhaave's syndrome if the patient appears ill and the diagnosis remains possible yet illusive.

Pitfall | **Failure to evaluate a patient with chest tenderness for herpes zoster**

We have all seen patients in a less-than optimal setting (e.g., in a chair; multiple layers of clothes on; no curtain for privacy) where we take the chest pain history and find that the pain is reproducible with palpation on physical examination. When entertaining the diagnosis of chest wall pain or costochondritis, consider herpes zoster (shingles) as well.

Herpes zoster is generally a clinical diagnosis. It occurs in patients due to reactivation of latent varicella zoster virus, dormant in the dorsal root ganglia. It is seen in both children and adults, although incidence varies directly with age [100, 101]. Annualized incidence is 1.5–3.0 case per 1000 persons; in patients >75 years of age, this rate increases to 10 cases per 1000 persons [100]. The increased incidence with age, as well as an association with states of impaired cell-mediated immunity (e.g., immunosuppressive therapy, cancer, human immunodeficiency virus) is evident, but an outbreak of herpes zoster is not specific for a state of impaired immunity [100, 102]. Indeed, herpes zoster develops in approximately 20,000 apparently healthy children each year in the USA; chicken pox at an age of less than 1 year is a risk factor [102].

Herpes zoster typically presents with abnormal skin sensations (itching, tingling, and/or pain – which may be severe) in a dermatomal distribution that precede the appearance of skin lesions – typically by 1–5 days [100], although visible lesions may not develop for a week to 10 days [103, 104]. *Zoster sine herpete* is an uncommon variant in which the lesions never appear [104]. The history together with visible evidence of the lesions (classically an erythematous maculopapular rash which progresses to the vesicular stage, followed by pustulation, ulceration, and finally crusting before disappearance) in a dermatomal distribution is key to the diagnosis [100]. Pain on light touch (allodynia) or overly sensitive skin (hyperesthesia) in a dermatomal distribution is also consistent with the diagnosis; these findings may precede the outbreak of the skin lesions [104]. Generally

speaking, the rash is unilateral, does not cross the midline, and is confined to one dermatome in immunocompetent persons. Overlap with adjacent dermatomes is relatively common (20%), and the appearance of a few lesions outside the affected dermatome is also not unusual [100]. Resolution occurs over 2–4 weeks, although it may be followed by the persistence of pain – so-called post-herpetic neuralgia.

Thus, in patients with a presumptive diagnosis of chest wall pain, carefully inspect the skin for signs of herpes zoster. Furthermore, if no lesions are visible, but the history (pain, oftentimes severe, in a band-like, dermatomal distribution, and perhaps accompanied by itching or paresthesias) and physical examination (hyperesthesia or allodynia in the same dermatomal distribution) are consistent with the prodromal stage of herpes zoster, instruct the patient to watch carefully for the appearance of any lesions. Prompt treatment (generally within 3 days of appearance of the rash) with antiviral therapy is indicated [100].

Pearls for Improving Patient Outcomes

- Do not exclude the diagnosis of acute cardiac ischemia or MI based on the absence of pain, especially when evaluating diabetic patients, the elderly, and women.
- Never use reproducible chest wall tenderness to exclude the diagnosis of acute MI.
- When the ECG shows LBBB or VPR, examine it closely for signs of inappropriately large, discordant ST-segment elevation; concordant ST-segment elevation; or concordant ST-segment depression (in the right precordial leads) – these may indicate an acute MI.
- Never use the response to antacids as a diagnostic test for distinguishing cardiac versus gastric pain.
- Neither a single normal ECG nor a single negative set of cardiac enzymes should be used to rule out acute cardiac ischemia.
- The chest X-ray can be used to suggest the diagnosis of AD, but it cannot definitively exclude the diagnosis.
- Consider AD and PE in the differential diagnosis of patients presenting with syncope.
- Pleuritic chest pain should prompt diagnostic consideration of PE as well as acute pericarditis.
- Precordial T-wave inversions in patients with chest pain should prompt consideration of not only acute cardiac ischemia but also of acute PE.
- Boerhaave's syndrome should be considered in the differential diagnosis for all patients with chest pain, even in the absence of a history of retching or vomiting.
- Always visualize the skin whenever a patient has reproducible chest well tenderness, and look for evidence of herpes zoster.

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