Cardiovascular Emergencies

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Amal Mattu, MD, FACEP. Since joining the faculty at the University of Maryland School of Medicine in 1996, Dr. Mattu has had a passion for teaching and writing about emergency cardiology. His commitment to teaching has earned him more than twenty teaching awards, including national awards from the American College of Emergency Physicians (ACEP) and local honors including the Teacher of the Year for the University of Maryland at Baltimore campus and the Maryland State Emergency Physician of the Year Award. He is a regular speaker at national and international conferences on topics pertaining to emergency cardiology. Dr. Mattu has authored or edited 16 textbooks in emergency medicine, including seven focused on emergency cardiology and electrocardiography. He is also the only emergency physician to serve as primary Guest Editor for Cardiology Clinics, which he has done twice. Dr. Mattu is currently a tenured professor, Vice Chair, and director of the Emergency Cardiology Fellowship for the Department of Emergency Medicine at the University of Maryland School of Medicine.

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Scott Silvers, MD, FACEP. Dr. Silvers attended medical school at the University of Rochester School of Medicine in Rochester, New York, and received his training in emergency medicine at the Harvard Affiliated Emergency Residency in Boston, Massachusetts. Currently, he is chair of the Department of Emergency Medicine at Mayo Clinic in Jacksonville, Florida, where he also serves as co-director of the Mayo Clinic Comprehensive Stroke Center and Chest Pain Center. Dr. Silvers is a member of both the ACEP Clinical Policies Committee as well as the American Heart Association’s Emergency Cardiovascular Care Committee where he contributes to the development of national, evidence-based guidelines. He was a co-author of the first Blueprints in Emergency Medicine study guide, and a co-editor of the Textbook of Emergency Cardiovascular Care and CPR. In coordination with the University of Miami’s Center for Research in Medical Education, Dr. Silvers contributed to the development of the national advanced stroke life support curriculum. He is a reviewer for several journals and a member of the editorial board of Emergency Medicine Practice.

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Dedications

I would like to thank my wife, Sejal, for her constant support and encouragement; I thank my children, Nikhil, Eleena, and Kamran, for always reminding me of my proper priorities in life; I thank the residents and students at the University of Maryland School of Medicine for providing me the inspiration for the work I do every day; and finally, thanks to my colleagues and mentors, who continue to exemplify what I hope one day to become.

—Amal Mattu

I am most fortunate and am appreciative of many people—my parents, William and Joann Brady, for providing the opportunities; my wife, King Brady, for her support, patience, and love; my children, Lauren, Anne, Chip, and Katherine, for their love and inspiration; my chair, Robert O’Connor, MD, for his mentorship and leadership; and my colleagues in emergency medical care, both hospital- and prehospital-based, for their partnership in healthcare and dedication to the patient.

—William J. Brady

I would like to dedicate this work to my family, Adrienne, Ben, and Aaron, and to the thousands of emergency physicians whose efforts day and night provide care and comfort for our fellow human beings.

—Michael Jay Bresler

This book is dedicated to my parents for showing me how to live a life of integrity and devotion; to the love of my life, Avery, who is my greatest support and best friend; to my boys, Levi and Austin, who are my best buds and who motivate me to be the best that I can be; and to all of my many mentors in medicine who have challenged me to understand more about why we do what we do.

—Scott Silvers

For all those who let me teach the subtleties of ECG interpretation, medication effects on transmembrane potentials, and the evidence behind ACS risk stratification protocols…at 2 AM and feign to appear interested! I thank you for that gift.

—Sarah A. Stahmer

This text is dedicated to my wife, children, and parents for their support, love, and faith. I thank my colleagues at University of California San Francisco and at the American College of Emergency Physicians for their brilliance, fantastic attitude, and great friendship. I also thank the amazing staff at ACEP who made this all possible and finally, the amazing Amal Mattu, who continues to lead and inspire a generation of physicians.

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Foreword

Emergency physicians serve as front-line clinicians who are expected to evaluate, stabilize, and begin treatment whenever an emergency patient presents to the emergency department (ED). As the specialty of emergency medicine has evolved and matured, so have the expectations for the expertise of the emergency physician. This creates a very exciting but also a very challenging work environment.

Gone are the days when all patients with chest pain get admitted for observation, every patient who is critically ill is whisked to an ICU before the initiation of critical care or a cardiologist is routinely called to the ED for patients with an unusual or unstable rhythm. Physicians working in an ED are now expected to have a high level of sophisticated knowledge in all areas of emergency care, with cardiovascular emergencies being one of the most important.

*Cardiovascular Emergencies* by Mattu, Brady, Bresler, Silvers, Stahmer, and Tabas brings together experts in our specialty to create an authoritative text for emergency providers. It is a book by emergency physicians for emergency physicians. It is also an excellent resource for physicians training in any specialty who will see cardiovascular emergencies. Each of the editors is a renowned educator and they have carefully selected authors for each topic. The text’s value is maximized by extremely clear ECGs and very high quality graphics and illustrations.

The best textbooks are broad enough to include all relevant information, but are focused on the core topics readers will need to develop expertise or to serve as a reference. The editors and authors drew on their many years of experience educating students, residents, and fellow physicians to create a comprehensive textbook of cardiovascular emergencies. They begin with a chapter on how to approach chest pain and follow with a chapter on the overt and subtle ECG signs of ischemia and infarction. The evolution of biomarkers as well as the “best” imaging study to evaluate patients for ischemia follows in separate chapters devoted to each topic. The text’s value is maximized by extremely clear ECGs and very high quality graphics and illustrations.

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Chapters are also devoted to the management of arrhythmias seen near daily in the ED, including wide and narrow complex tachycardias along with bradycardias and heart block. Critical care medicine and cardiology share a number of emergencies, and this text provides up-to-date management of acute decompensated heart failure, cardiogenic shock, cardiac arrest, and post-cardiac arrest care, covering each with a detailed but succinct chapter. *Key Point* sections throughout all chapters highlight the most important concepts and clinical insights of the authors.

Other topics also covered in *Cardiovascular Emergencies* are syncope and hypertension, two very common entities, as well as less common but important conditions such as pulmonary hypertension, myocarditis, and pericarditis; complications due to implanted devices including pacemakers, AICDs, and LVADs; and cardiovascular emergencies in pregnant patients. Cardiac pharmacology, as it applies to emergent patients, and the use of the ED for observation are also presented. Because missing a cardiovascular emergency such as a myocardial infarction leads all other causes in dollars lost to malpractice claims paid, the final chapter is devoted to reducing malpractice risks.

*Cardiovascular Emergencies* is a book for anyone who will see a cardiovascular emergency. It is written and edited by expert emergency physicians and is a superb resource. All of us should be indebted to its editors and authors.

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May 2014
Preface

Cardiovascular disease accounts for more deaths in the United States and most other first-world countries than any other cause. This number-one ranking has persisted for many years despite marked advances in preventive medicine, diagnostics, and therapeutics. Not only has this rank remained immobile, but the absolute number of deaths due to cardiac disease continues to rise. Most “experts” predict that this is not going to change in the near future. Therefore, if we in the health care field have any hope of changing these statistics, we must be optimally prepared to diagnose and treat patients when they present with acute cardiovascular conditions or complications.

The specialty of Emergency Medicine bears a great responsibility for the acute care of these patients. We must diagnose and initiate stabilizing treatment for patients with acute coronary syndromes, acute heart failure, pericarditis, myocarditis, arrhythmias, and many other conditions, and we are frequently the key providers who determine the prognosis of patients presenting with cardiac arrest. We are required to carry out these duties while working under significant time constraints; we are forced to make life-and-death decisions, often with minimal objective data; and we are often held to impossible standards of care by society and the legal profession.

The goal of this textbook is to facilitate the efficient and cutting-edge delivery of care to patients who present with acute cardiovascular conditions. To accomplish this goal, we brought together many of the brightest minds in Emergency Medicine from various institutions to collaborate and create best practices for emergency cardiovascular conditions. We believe we have formulated approaches to the workup and management that will optimize patient care.

In the pages that follow, we address many of the most common emergency cardiovascular conditions we face in Emergency Medicine as well as some conditions that are rising in import around the world. Initial chapters focus on the complicated evaluation and differential diagnosis of chest pain and modern approaches to “low-risk” chest pain. Acute coronary syndromes are covered in depth, and subsequent chapters address many of the complications associated with coronary artery disease, including acute heart failure, arrhythmias, and cardiogenic shock. Recent “hot topics” in the Emergency Medicine literature are addressed, including bedside echocardiography, observation units, cardiac arrest, and post-arrest care. Special populations are also discussed: oncologic patients, pregnant patients, transplant patients, patients with HIV, patients with pulmonary hypertension, and patients with implanted devices. A special chapter is devoted to issues related to malpractice.

In overseeing the development of this text, our goal has been to provide an easily understood, highly visual resource that is readable from cover to cover. Although this text might be considered a “bookshelf reference,” that designation is at odds with our goal of cover-to-cover readability. We have tried to format the chapters for quick reference during everyday patient care.

We hope you enjoy reading this book and welcome any and all of your feedback. We would like to thank Linda Kesselring, copyeditor at University of Maryland, and Mary Anne Mitchell, copyeditor at ACEP, whose persistence and insight saw this project through to completion and excellence. We would also like to thank our families for their patience and understanding while we worked on this project, and we thank our colleagues, students, and residents, who have been a constant source of inspiration for our work. We would especially like to thank you, the readers, for your unwavering dedication and commitment to patient care.

Amal Mattu
William J. Brady
Michael J. Bresler
Scott M. Silvers
Sarah A. Stahmer
Jeffrey A. Tabas
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Approach to Acute Chest Pain

Deborah B. Diercks and Nathan Parker

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Pulmonary embolism
Esophageal rupture
Tension pneumothorax
Aortic dissection
Cardiac tamponade
Ancillary tests
Advanced imaging
Clinical decision rules

CHAPTER 1

More than 6 million Americans present to emergency departments every year with the chief complaint of acute chest pain. The differential diagnosis is extremely broad (Table 1-1). Emergency physicians have the difficult task of differentiating life-threatening causes requiring immediate intervention from more benign causes. In this chapter, we focus on the presentation of the most common critical diagnoses, their initial work-up, and the strategies employed to ensure a safe and successful disposition.

Initial Approach

All patients without an obviously benign cause of chest pain should have their vital signs assessed immediately, be connected to a monitor, and have intravenous access established. Ideally, an electrocardiogram (ECG) should be obtained in the prehospital setting. Recent data have shown that paramedics and nurses, given adequate training, can reliably diagnose ST-elevation myocardial infarction (STEMI) and subsequently alert destination hospitals. Since it has been well established that early reperfusion reduces mortality and morbidity, a system should be in place to facilitate rapid percutaneous intervention or fibrinolysis once STEMI has been confirmed. A focused history and physical examination should be performed promptly by the emergency care provider because successful management of conditions such as tension pneumothorax depends on the provider’s acting within minutes of a patient’s presentation.

KEY POINT

Patients with chest pain should have an ECG obtained on arrival.

Acute Coronary Syndrome

Of the common presenting causes of chest pain, acute coronary syndrome (ACS) presents a particular challenge to emergency physicians. Defined as the syndrome resulting from acute cardiac ischemia, ACS encompasses stable angina, unstable angina, STEMI, and non-STEMI (NSTEMI).

Missed acute myocardial infarctions (AMIs) are frequent causes of litigation against medical providers. Emergency physicians disagree over the acceptable rate of missed acute MI; most accept a rate between 0.01% and 2%. Care providers in emergency departments with low patient volumes and limited resources face particularly difficult challenges in making the diagnosis; miss rates tend to be higher in these facilities.

In the “classic” presentation of ACS, the patient usually describes the pain as pressure, squeezing, or crushing. The pain is located substernally or on the left, and it can radiate to the jaw,
necks, or arms. Associated symptoms usually include diaphoresis, nausea, vomiting, weakness, and syncope. However, none of these signs and symptoms is sensitive or specific enough on its own to rule in or out ACS independent of an ECG, cardiac biomarkers, and other diagnostic tests. Similarly, the presence of traditional risk factors such as hypertension, hyperlipidemia, diabetes mellitus, family history of coronary artery disease (CAD), and history of smoking, although positively correlating with adverse events within 6 months, does not correlate with the incidence of acute MI in the emergency department. However, emergency care providers should be cautious about an initial impression of “noncardiac chest pain” if traditional risk factors are present because 3% of patients with those factors will experience an adverse cardiac event within 30 days.

To further complicate the establishment of a diagnosis, many patients with an eventual diagnosis of acute MI present without chest pain at all. This presentation is more common among women than men (42% and 31%, respectively), but the difference decreases with increasing age. Atypical presentation of acute MI is also associated with diabetes, heart failure, advanced age, and nonwhite races.

KEY POINT

Many patients with ACS present without chest pain.

### Pulmonary Embolism

Pulmonary embolism (PE) accounts for up to 200,000 deaths in the United States annually. Like ACS, PE represents a broad range of disease, from asymptomatic incidental findings to saddle embolus causing shock and sudden death. Among patients presenting in shock, the short-term mortality rate can reach as high as 50%.

Reflecting this broad spectrum of disease, the clinical signs and symptoms are especially difficult to interpret. In a large, prospective study, the following symptoms were present in patients diagnosed with PE: dyspnea (79%), pleuritic pain (49%), cough (43%), wheezing (31%), calf or thigh swelling (39%), and calf or thigh pain (16%). On physical examination, the following signs were present: tachypnea (57%), tachycardia (26%), rales (21%), and signs of deep vein thrombosis (DVT) in the calf or thigh (47%).

Risk factors for acute PE include recent surgery, trauma, immobility, cancer, neurologic disease with lower extremity paresis, oral contraceptive use, hormone therapy, and pregnancy. Given the difficulty in diagnosing PE, multiple clinical decision rules have been devised to aid in the workup. These rules are discussed later in this chapter.

### Esophageal Rupture

Esophageal rupture is a relatively rare cause of acute chest pain among emergency department patients. Although its true incidence is unknown, the diagnosis carries a high mortality rate—approximately 20% despite modern therapies. The mean age of patients with esophageal rupture is the early 60s, and more than two thirds of patients are male. Esophageal perforations are most commonly iatrogenic, usually caused by endoscopic procedures, with a minority of ruptures resulting spontaneously from increased intraabdominal pressures typically associated with vomiting (eg, Boerhaave syndrome). Other causes include caustic ingestions and blunt or penetrating trauma.

The classic features of esophageal rupture include the sudden onset of chest pain precipitated by severe vomiting or retching. The Mackler triad of esophageal rupture—chest pain, vomiting, and subcutaneous emphysema—was first described in 1952; this triad is absent in most patients. Associated symptoms include shortness of breath, dysphonia, dysphagia, abdominal pain, hematemesis, and melena. On physical exam-

<table>
<thead>
<tr>
<th>TABLE 1-1. Causes of Chest Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
</tr>
<tr>
<td>Acute MI</td>
</tr>
<tr>
<td>Aortic dissection</td>
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<tr>
<td>Cardiac tamponade</td>
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<tr>
<td>Coronary spasm</td>
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<tr>
<td>Pericarditis</td>
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<tr>
<td>Stable angina</td>
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<td>Unstable angina</td>
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<tr>
<td>Pulmonary</td>
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<td>Bronchitis</td>
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<td>Pneumonia</td>
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<tr>
<td>Pneumothorax</td>
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<tr>
<td>Pulmonary embolus</td>
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<tr>
<td>Gastrointestinal</td>
</tr>
<tr>
<td>Cholecystitis</td>
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<tr>
<td>Esophageal reflux</td>
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<tr>
<td>Esophageal rupture</td>
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<td>Esophageal spasm</td>
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<td>Esophageal tear</td>
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<td>Gastritis</td>
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<tr>
<td>Hepatitis</td>
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<tr>
<td>Pancreatitis</td>
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<tr>
<td>Peptic ulcer disease</td>
</tr>
<tr>
<td>Musculoskeletal</td>
</tr>
<tr>
<td>Costochondritis</td>
</tr>
<tr>
<td>Muscle strain</td>
</tr>
<tr>
<td>Rib fracture</td>
</tr>
</tbody>
</table>

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inflation, tachycardia is frequently noted, with fever presenting later. Crepitus in the neck or chest wall is indicative of subcutaneous emphysema. Rapidly progressing pleural effusions can be a late sign.19

**Tension Pneumothorax**

Nontraumatic spontaneous tension pneumothorax is also a relatively uncommon cause of acute chest pain. Approximately 1% to 2% of all spontaneous pneumothoraces present under tension.20 Clinically, a pneumothorax is considered to be under tension when it causes significant respiratory or hemodynamic compromise as a result of positive intrapleural pressure. In an awake patient being ventilated without positive pressure, this process can develop only if the intrapleural pressure is less than the atmospheric pressure during some period of the respiratory cycle. Therefore, the spectrum of tension pneumothorax can range from intrapleural pressure that is positive only at the end expiratory phase to pressure that is positive throughout the entire respiratory cycle.21

Spontaneous pneumothorax can be divided into the following two classifications: primary spontaneous pneumothorax, which occurs in the absence of apparent underlying lung disease, and secondary spontaneous pneumothorax, which develops as a result of underlying lung pathology. Risk factors for primary spontaneous pneumothorax include male sex (6:1 relative risk compared with females), tall stature, smoking, low body mass index, sudden changes in environmental pressure, genetic predisposition, inhalant use, and even exposure to loud music.22,23 Risk factors for secondary spontaneous pneumothorax include chronic obstructive pulmonary disease, interstitial lung disease, infection, neoplasm, and connective tissue disease.22

Symptoms of tension pneumothorax typically include the rapid onset of pleuritic chest pain and shortness of breath. In addition to unilateral reduced breath sounds and hyperresonance, tension pneumothorax also can present with tachycardia, tachypnea, hypotension, and tracheal deviation away from the affected side.24 If a tension pneumothorax is highly suspected based on the history and physical examination alone, steps should be taken immediately to relieve the pressure via needle or tube thoracostomy. A recent review showed tube thoracostomy to be superior to percutaneous aspiration; the reason was initially thought to be that the chest wall thickness exceeded the length of the catheter in percutaneous aspiration, but this does not appear to be the case.25,26 Therefore, in patients with tension pneumothorax, immediate tube thoracostomy is indicated.

**KEY POINT**

Immediate decompression of a suspected tension pneumothorax with tube thoracostomy is indicated before confirmation with chest radiography.

**Aortic Dissection**

Few critical diagnoses are as feared by emergency care providers as acute aortic dissection, which is notoriously difficult to diagnose. Some studies suggest that up to one-third of all aortic dissections are initially misdiagnosed. The difficulty is compounded by its relatively low prevalence (1 case per every 10,000 emergency department visits).27,28 Acute aortic dissection is often mistaken for myocardial infarction. The mortality rate associated with untreated dissection reaches 1% to 2% per hour during the first 48 hours.29,30

**KEY POINT**

Up to one-third of all aortic dissections are misdiagnosed.

Both the DeBakey and Stanford classifications systems have been widely used for describing aortic dissections. DeBakey type I begins in the ascending aorta and extends beyond the arch. Type II involves the ascending aorta only, while type III involves only the descending aorta. Stanford type A is any dissection that involves the ascending aorta, and type B dissections do not. Although both classifications can be used, it is most important to identify if the ascending arch is involved, as dissections involving the ascending arch usually require emergent surgical intervention.31

Recent guidelines published by the American Heart Association, in conjunction with other professional societies, describe important clinical risk factors for assessing the pretest probability of acute aortic dissection in patients with chest pain. These factors include Marfan or Ehlers-Danlos syndrome, a family history of aortic disease, aortic valve disease, recent aortic manipulation, thoracic aortic aneurysm, abrupt onset of pain, pain that is severe, pain that is ripping or tearing, pulse deficit in the upper limbs, focal neurologic deficit, hypotension or shock, and new aortic regurgitation murmur.32 When these features were applied to the International Registry of Acute Aortic Dissection, over 95% of patients with confirmed aortic dissection had at least one of them.28

Research has been done to determine the factors that delay the time from presentation to diagnosis. Female patients, patients transferred from a non-tertiary care facility, and patients who have had previous cardiac surgery all had longer delays in diagnosis. The same is true for patients with mild or no pain, patients with atypical features such as fever, those with heart failure, and those with an initial ECG suggestive of myocardial ischemia.33 A Japanese study suggested that patients who walk into the emergency department are also more likely to have a delay in diagnosis.34 Given the high mortality rate associated with missed diagnosis and the relatively low incidence of this dangerous condition, high suspicion must be maintained to prevent complications.

**Cardiac Tamponade**

Cardiac tamponade, another relatively rare diagnosis, refers to hemodynamic compromise caused by increased pericardial pressure. The spectrum of this disease ranges from mild and asymptomatic (pericardial pressure <10 mm Hg) to severe, causing shock (pericardial pressure >20 mm Hg).35 Other than trauma and recent cardiac surgery, medical causes of pericardial effusion include acute pericarditis, malignancy, acute MI causing wall rupture, aortic dissection, uremia, heart failure,
bacterial or viral infection, and collagen vascular disease.\textsuperscript{12}

The Beck triad of low arterial blood pressure, distended neck veins, and muffled heart sounds has been used to describe the signs of cardiac tamponade, but these are probably late findings. Increased sympathetic drive usually causes hypertension before the physiologic reserve is exhausted.\textsuperscript{36} A recent review of studies involving patients with pericardial effusion delineated the following signs and symptoms associated with the disease and their related sensitivities: dyspnea (87\%-88\%), pulsus paradoxus greater than 10 mm Hg (82\%), tachycardia (77\%), hypotension (26\%), diminished heart sounds (28\%), and elevated jugular venous pressure (76%).\textsuperscript{37}

**Ancillary Tests**

**Electrocardiography**

The 12-lead ECG is one of the cornerstones in chest pain evaluation. This chapter reviews common ECG changes that, in general, suggest important diagnoses; subtleties of ECG interpretation are discussed in depth elsewhere in this book. ECGs provide a “snapshot” of the heart’s electrophysiology. To fully capture a dynamic process, including an evolving myocardial infarction, serial ECGs repeated 30 to 60 minutes after the initial study are recommended when the initial tracing is nondiagnostic and suspicion remains for ongoing ischemia.\textsuperscript{38} It is important to remember that a normal ECG does not rule out acute ischemia; more than 50\% of patients with missed AMI had a normal initial ECG.\textsuperscript{12}

ST-segment elevation has a variety of causes (Table 1-2). ST-segment elevation should raise suspicion for acute ischemia/infarct when it exists in two or more contiguous leads.\textsuperscript{39} When reciprocal ST-segment depression is present, the diagnosis of STEMI becomes more likely.\textsuperscript{40} When an inferior STEMI is suspected (elevation in lead II, III, or aVF), tracings from leads V\textsubscript{4}R through V\textsubscript{6}R can be obtained to evaluate for right ventricular infarction, as these patients are often preload dependent.\textsuperscript{41}

Although diffuse ST elevation and PR depression (ST depression and PR elevation in aVR) constitute the classic ECG finding in acute pericarditis, once the condition has progressed to a significant pericardial effusion, electrical alternans can be seen.\textsuperscript{12,42} For PE, the ECG is usually of little diagnostic utility. In PE, the classic finding of S\textsubscript{1}Q\textsubscript{3}T\textsubscript{3} is rarely seen. The most common ECG findings are sinus tachycardia or nonspecific ST-segment or T-wave changes.\textsuperscript{44}

A recent study of 159 patients with type A aortic dissection showed that almost half of them had acute changes on the ECG. ST depression (34\%) was the most common, but ST elevation (8\%) was present as well. Finding these ST changes increases the risk of misdiagnosis.\textsuperscript{45} ST elevation in acute dissection is also associated with involvement of the coronary ostia, the right coronary artery being most commonly involved.\textsuperscript{46}

**Chest Radiograph**

The chest radiograph (CXR) is another important diagnostic tool in patients with acute chest pain. Although the CXR rarely provides the diagnosis in isolation, it can rapidly change a treatment algorithm. For example, although a CXR can provide important information in a stable patient, delaying treatment for imaging in a patient with clinically suspected tension pneumothorax is not optimal. Still, tube thoracostomy is not indicated in other diagnoses such as diaphragm rupture, which can mimic the presentation of tension pneumothorax, so the decision to image or not must be individualized for each patient.

The CXR is commonly used to evaluate patients with suspected aortic dissection, but it is neither sensitive nor specific for this disease. In one study, only 73\% of patients with known type A aortic dissection had signs suggesting dissection, most commonly widened mediastinum, while 16\% of normal CXRs were thought to be suspicious for dissection.\textsuperscript{37} Up to 90\% of patients with esophageal rupture have abnormal findings on CXR, most commonly pneumomediastinum, hydropneumothorax, and isolated pleural effusion.\textsuperscript{48} Interestingly, the esophagus ruptures most often on the left, with subsequent development of a pleural effusion on that side.\textsuperscript{49}

**KEY POINT**

A normal chest radiograph does not rule out aortic dissection.

**FIGURE 1-1.**

Right heart strain; note dilated right ventricle. Photo courtesy of K. Kelley.
In one large study of patients known to have PE, cardiomegaly (27%) was the abnormality most frequently seen on CXR; 24% of patients had a normal CXR. The classic Hampton hump is rarely seen. The CXR can provide useful information when the physician is deciding whether to perform ventilation-perfusion scintigraphy or CT angiography, as patients with known chronic lung disease have a higher incidence of nondiagnostic ventilation-perfusion scans.

**Ultrasonography**

Ultrasonography has become an integral diagnostic tool in the provision of emergency care. In patients who come to the emergency department in shock, a two-dimensional transthoracic echocardiogram (2D-TTE) that shows no signs of right ventricular strain (Figure 1-1) can practically exclude PE as the cause of hypotension. However, a negative ultrasound scan does not rule out PE as a cause of chest pain. Two-dimensional transthoracic echocardiography is the gold standard for diagnosing pericardial effusion in the emergency department, and all patients with suspected pericarditis and any high-risk features should have bedside echocardiography to aid in diagnosis and rule out pericardial tamponade (Figure 1-2).

When ACS is suspected, early 2D-TTE can contribute information to the prognosis by identifying wall motion abnormalities. In a study of patients presenting to an emergency department with acute chest pain and a nondiagnostic ECG, those with a normal 2D-TTE (performed by a cardiologist) had no major cardiac events at 30 days. Structural abnormalities that might change therapeutic management, such as papillary muscle rupture or ventricular septum rupture, can also be identified; images suggesting such abnormalities should be interpreted only by experienced cardiac sonographers.

Limited data exist on the role of ultrasonography in the diagnosis of aortic dissection. In evaluating for an intimal flap, 2D-TTE can be combined with abdominal ultrasonography. The sensitivity of 2D-TTE in detecting acute aortic dissection has been reported to be 67% to 80%, while its specificity is 99% to 100%. When dissection is identified in an unstable patient, emergent cardiothoracic surgery should be expedited.

Bedside ultrasonography performed by emergency physicians has a sensitivity approaching 100% for pneumothorax, compared with a 75% sensitivity for an upright CXR. This degree of sensitivity, as well as ultrasound’s portability, has fostered the use of ultrasonography in military operations and in remote locations where other imaging modalities are unavailable. The detection of occult pneumothorax that was not seen on a CXR might not prove to be clinically significant, but it should prompt the care provider to monitor the patient closely for pneumothorax expansion.

**KEY POINT**

Bedside emergency ultrasonography is a valuable tool in the workup of chest pain.

**Troponins**

Cardiac troponins (T and I) are the preferred markers of myocardial injury in patients presenting with chest pain. They are more sensitive and specific than the biomarkers used in the past. It is important to remember, however, that myocardial necrosis can result from pathologies other than MI or ACS. With the introduction of new highly sensitive troponin (hs-cTn) assays, which are 1,000- to 10,000-fold more sensitive than the original first-generation troponin assays, differentiating the causes of myocardial necrosis (including MI and ACS) will be more important as medical care providers are challenged with interpreting an increasing number of positive tests.

Given our increased ability to measure very low concentrations of cardiac troponins, an upper limit of normal at the population-adjusted 99th percentile has been defined. With the application of this newly defined cutoff, more patients with signs and symptoms suggesting acute MI have an elevated hs-cTn level when they come to the emergency department; a recent study showed an almost 200% increase in the number of hs-cTn–positive patients who were eventually diagnosed with chest pain unrelated to coronary occlusion. Troponin is released during tachyarrhythmia in young healthy people as well as in those undergoing sustained strenuous exercise. However, even small increases in hs-cTn levels in patients presenting with chest pain have been associated with adverse short- and long-term prognoses.

The definition of AMI, which includes the “rise and/or fall of cardiac biomarkers,” may have to be refined with the advent of hs-cTn assays. An acute change in the hs-cTn level of more than 20% has been suggested as representing either new or resolving myocardial injury. However, healthy study subjects have demonstrated a baseline variability of more than 50%. It is important to note that simply changing the diagnostic criterion of AMI to include a hs-cTn change of more than 50% decreases the sensitivity of the test to less than 70%.

It is likely that the introduction of hs-cTn assays will provide several benefits to emergency medicine practice, but its place in our diagnostic algorithm has not been fully defined. It has...
been suggested that, given the fact that hs-cTn levels elevate earlier, the timing of our serial cardiac marker testing could be shortened to less than 3 hours after initial presentation.72 The assay might take a role analogous to the D-dimer test, given its relatively high negative predictive value.73 It will also be important to reevaluate our chest pain clinical decision rules, as most of them were developed before hs-cTn assays became available.

**KEY POINT**
The role of the new highly sensitive troponin assays remains unclear in the evaluation of patients with chest pain.

**D-Dimer**
The D-dimer assay has been used in the evaluation of patients for PE for more than 20 years. Although many assays exist, most used in emergency practice have a high sensitivity (typically in the mid 90% range) but low specificity for venous thromboembolism (VTE).74 In the contemporary diagnostic algorithm, a highly sensitive D-dimer assay that is below the designated cutoff in a non–high-risk population can be used to exclude VTE because of its negative predictive value of more than 99% in this population.75 In fact, the highly sensitive quantitative D-dimer test has one of the highest sensitivities of any test used in the screening of patients for VTE.

The D-dimer assay has been studied for both prognostic purposes and for evaluation of the burden of disease in PE. High D-dimer levels are associated with increased 15-day and 3-month mortality rates as well as a more central location of clots on CT angiogram.76,77 High levels are also associated with a higher pulmonary artery obstruction index.78

**KEY POINT**
A negative D-dimer does not rule out pulmonary embolus in a population at high risk for the disease.

Recently, the use of the D-dimer assay was studied in the context of aortic dissection. A metaanalysis found a pooled sensitivity of 94%, but specificity remained poor.79 Concern exists regarding the proposed use of the test to rule out aortic dissection in low-risk populations, as isolated intramural hematomas or thromboses have been associated with false-negative tests.80 Given the low incidence of acute aortic dissection in the general population presenting with chest pain, it has been suggested that routine screening with D-dimer assays would increase CT scan utilization by approximately 40% and would not necessarily aid in timely diagnosis.81

**Other Biomarkers**
New research has focused on addressing methods for shortening the time needed to rule out MI in the emergency department. In low-risk populations, adding either N-terminal pro-B type natriuretic peptide (NT-proBNP) or copeptin to the initial troponin assessment at presentation significantly increased the sensitivity and the negative predictive value for MI.82,83 Measuring unbound free fatty acids in addition to conventional or highly sensitive troponin also improves sensitivity and specificity in the detection of ACS.84 Higher levels of NT-proBNP or ST2 (a novel biomarker of cardiac stress) in patients with chest pain have been associated with increased mortality.85,86 Investigating the interesting concept that larger platelets are more active, researchers have found a correlation between mean platelet volume and ACS in patients with acute chest pain.87

**Advanced Imaging**

**Computed Tomography Angiography**
Computed tomography angiography (CTA) has become the gold standard as the initial imaging test in the workup of patients suspected of having aortic dissection or PE. The sensitivity and specificity of CTA in detecting acute aortic dissection have been reported as 100% and 98% to 99%, respectively.88 The data associated with PE have shown larger variations in accuracy. The largest study to date, PIOPED II, reported sensitivity of 83% and specificity of 96%, but most of the scans were performed with four-slice CT.89 It is possible that with the arrival of 64-slice multidetector CT the diagnostic accuracy will increase significantly, thereby allowing a negative CTA alone to be used to effectively rule out PE as the cause of chest pain in all risk groups.88

As diagnostic accuracy increases with improved technology, an increasing number of isolated subsegmental PEs are being diagnosed with CTA.90 No consensus has been reached regarding the proper management of patients with isolated subsegmental PE because 3-month outcomes are generally favorable and the risk of hemorrhage with anticoagulation might outweigh the benefits.91

**Coronary Computed Tomography Angiography**
Coronary CTA (CCTA) has received much attention in recent years, with a flurry of data about its use coming out in a short time. Although the role of CCTA has not been established or extensively validated in the workup of chest pain in the emergency department, its proposed use has been hotly debated. A recent metaanalysis involving only prospective studies estimated the sensitivity and specificity of CCTA for ACS to be 95%

**TABLE 1-3.**

<table>
<thead>
<tr>
<th>PERC Rule101</th>
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<tbody>
<tr>
<td>Age under 50 years</td>
</tr>
<tr>
<td>Heart rate less than 100 beats/min</td>
</tr>
<tr>
<td>Oxygen saturation of more than 94% on room air</td>
</tr>
<tr>
<td>No history of DVT/PE</td>
</tr>
<tr>
<td>No recent trauma/surgery</td>
</tr>
<tr>
<td>No hemoptysis</td>
</tr>
<tr>
<td>No exogenous estrogen</td>
</tr>
<tr>
<td>No clinical signs of DVT</td>
</tr>
</tbody>
</table>

If all eight criteria are met, patient has less than a 2% chance of having PE.
and 87%, respectively.\textsuperscript{92}

Heralded for its high negative predictive value and its potential to rule out ACS with a negative scan, CCTA has been shown to decrease time to diagnosis and disposition.\textsuperscript{93,94} Critics of CCTA point out its low positive predictive value—all patients with significant underlying CAD, stable or not, will likely have a positive scan and thus require additional testing.\textsuperscript{95} The radiation exposure must also be considered, as well as the fact that the study cannot be performed in patients with renal failure, those unable to tolerate beta-blockers, and those with ectopic rhythms.\textsuperscript{96} While not yet standard of care, CCTA will likely find a role in rapidly triaging patients who are at low to intermediate risk for ACS and who have initially normal cardiac markers and an ECG that does not raise concern.\textsuperscript{97,98}

**Triple-Rule-Out Computed Tomography Angiography**

A recently developed protocol combines CCTA with imaging of the pulmonary arteries and thoracic aorta. Triple-rule-out CTA enables patients to be simultaneously evaluated for ACS, PE, and aortic dissection in less than 20 minutes. The protocol has significant limitations: it is technically difficult to perform, it delivers a 50% larger dose of radiation than CCTA, and its image quality varies.\textsuperscript{99} However, because more than 20% of patients evaluated for ACS are simultaneously assessed for PE or aortic dissection, performing only one imaging study would decrease their overall radiation exposure.\textsuperscript{100} As with CCTA, the use of triple-rule-out CTA has not yet been validated in large prospective trials, but it may still find a role in ruling out disease in low- to intermediate-risk patients.\textsuperscript{1}

### Clinical Decision Rules

Considering the difficulty in accurately diagnosing the cause of chest pain while performing an efficient and cost-effective workup, multiple clinical decision rules have been devised to assist emergency physicians. Many of these rules have been well validated in large prospective trials. These rules should never replace the physician’s best judgment, but they can be helpful when the differential diagnosis remains large. In this section, we describe a few of the more recognized clinical decision rules, along with their limitations.

#### Pulmonary Embolism Rule-out Criteria

The pulmonary embolism rule-out criteria (PERC) rule (Table 1-3) was devised to avoid additional testing in patients with a low pretest probability for PE. If a patient meets all eight criteria, his or her probability of having PE is less than 2%, which is an appropriate cutoff to discontinue further testing.\textsuperscript{101} The rule’s sensitivity for detecting PE is 97%; however, caution is advised, because the rule applies only to patients for whom the clinical suspicion for PE is low (<15%).\textsuperscript{102} In patients for whom the probability is high, the PERC rule does not safely exclude the condition.\textsuperscript{103}

#### TABLE 1-4.

**PE Risk Stratification**\textsuperscript{104,105}

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical signs and symptoms of DVT</td>
<td>+3</td>
</tr>
<tr>
<td>PE is most likely diagnosis</td>
<td>+3</td>
</tr>
<tr>
<td>Heart rate above 100 beats/min</td>
<td>+1.5</td>
</tr>
<tr>
<td>Immobilization in past 3 days or surgery in the past 4 weeks</td>
<td>+1.5</td>
</tr>
<tr>
<td>Previous DVT/PE</td>
<td>+1.5</td>
</tr>
<tr>
<td>Hemothysis</td>
<td>+1</td>
</tr>
<tr>
<td>Malignancy with treatment in past 6 months or palliative care</td>
<td>+1</td>
</tr>
<tr>
<td>Low risk (15%) &lt;2 points</td>
<td></td>
</tr>
<tr>
<td>Intermediate risk (29%) 2-6 points</td>
<td></td>
</tr>
<tr>
<td>High risk (59%) &gt;6 points</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lower extremity tenderness and unilateral edema</td>
<td>+4</td>
</tr>
<tr>
<td>Unilateral leg pain</td>
<td>+3</td>
</tr>
<tr>
<td>Heart rate between 75 and 94 beats/min</td>
<td>+3</td>
</tr>
<tr>
<td>Heart rate above 95 beats/min</td>
<td>+5</td>
</tr>
<tr>
<td>Surgery or fracture within 1 month</td>
<td>+2</td>
</tr>
<tr>
<td>Previous DVT/PE</td>
<td>+3</td>
</tr>
<tr>
<td>Hemothysis</td>
<td>+2</td>
</tr>
<tr>
<td>Active malignancy</td>
<td>+2</td>
</tr>
<tr>
<td>Patient older than 65 years</td>
<td>+1</td>
</tr>
<tr>
<td>Low risk (8%) &lt;4 points</td>
<td></td>
</tr>
<tr>
<td>Intermediate risk (28%) 4-10 points</td>
<td></td>
</tr>
<tr>
<td>High risk (74%) &gt;10 points</td>
<td></td>
</tr>
</tbody>
</table>
KEY POINT
In a low-risk population, a negative PERC score can preclude the need for further workup for pulmonary embolus.

Wells and Revised Geneva Scores
The Wells and revised Geneva scores (Table 1-4) were developed by assessing the clinical probability of PE and have been validated in large clinical trials. Both scores successfully stratify patients into low-, intermediate-, and high-risk groups for PE, but the Wells score is superior for the identification of patients in the high-risk group. Critics of the Wells score point to the subjective nature of the score. It asks care providers to determine if PE is the most likely diagnosis. In contrast, the revised Geneva score is based solely on objective data. In patients with an “unlikely” probability in the Wells or revised Geneva score (≤4 or <4, respectively), a negative highly sensitive D-dimer assay may be used to exclude PE without additional testing.

Thrombolysis In Myocardial Infarction Risk Score
The Thrombolysis In Myocardial Infarction (TIMI) risk score (Table 1-5) has been well validated as a method to stratify patients diagnosed with unstable angina/NSTEMI according to their risk for adverse events. The score is used to guide therapeutic and prognostic decision making. The TIMI risk score also correlates with outcomes when applied to emergency department patients; however, its sensitivity alone has been shown repeatedly to be inadequate at ruling out ACS in the unique population of emergency department patients. Although attempts have been made to create a “modified TIMI score” with improved performance in risk stratification, the new decision rule was still unable to screen patients safely for adverse events in the emergency department.

HEART Score and North American Chest Pain Rule
The HEART score and the North American Chest Pain Rule (NACPR) were devised to identify patients who experienced chest pain but are suitable for early discharge without stress testing or cardiac imaging. Both tools have yet to be validated in large multicenter prospective trials, but they have been shown initially to have an acceptable miss rate (<1%) for adverse events when combined with serial troponin measurements. It is unlikely that these rules will change clinical practice, as they generally describe the current standard of care for ACS evaluation in the emergency department.

Conclusion
Chest pain is a common emergency department presentation and has multiple causes. Emergency providers should take a careful and systematic approach to evaluating patients with chest pain. As technology has improved and tests have become more sensitive, providers must navigate the increasingly complex workup and allocate resources appropriately. By using the strategies outlined in this chapter, emergency physicians should be able to diagnose the life-threatening causes of chest pain safely and successfully.

References
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