CHAPTER ONE

The ECG and Clinical Decision-Making in the Emergency Department

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KEY POINTS

- The ECG must be interpreted within the context of the clinical presentation, including information such as the patient's age, chief and secondary complaints, physical examination, and other diagnostic test results.
- Clinical judgment has a very important role in the interpretation of the ECG within the individual clinical event.
- The ECG can provide information to confirm a diagnosis, rule out a diagnosis, risk stratify certain conditions, provide an indication for therapy, and predict complications.
- The ECG has numerous limitations in the various clinical scenarios in which it is used. An awareness of these limitations is vital to the correct application of the ECG in clinical care.

Electrocardiography is performed widely throughout emergency medicine, in emergency departments and observation units as well as in the prehospital environment and other out-of-hospital medical settings. In fact, it is appropriate to state that electrocardiographic monitoring is one of the most widely applied diagnostic tools in clinical emergency medicine today. Electrocardiography allows rhythm monitoring using single or multiple leads as well as the 12-lead ECG used to assess patients with a range of primary and secondary cardiopulmonary illnesses. Numerous situations in the emergency department warrant an electrocardiographic evaluation.[1]

The ECG can assist in establishing a diagnosis, ruling out various ailments, guiding diagnostic and management strategies, providing indication for certain therapies, determining inpatient disposition location, and assessing the end-organ impact of a syndrome (Table 1.1). Unfortunately. in the emergency department environment, the ECG does not usually provide a specific diagnosis in isolation. When combined with the clinical presentation, however, ECGs are far more useful. In a study of ECGs obtained in an emergency department, only 8% of the ECGs were diagnostic, but when interpreted within the context of the presentation, they much more frequently were able to help in ruling out various syndromes.[1] The most frequent reasons for obtaining an ECG were chest pain and dyspnea (Figure 1.1). In this same investigation, the ECG influenced the diagnostic approach in one-third of patients; additions included repeat ECGs, serum markers, and rule-out MI protocol. Alterations in therapy were made almost as often with the addition of antiplatelet, anticoagulant, or anti-anginal medication or reperfusion. Disposition was changed in approximately 15% of patient presentations with an inpatient location selected based on the electrocardiographic interpretation. The effects of 12-lead electrocardiographic findings on diagnostic, therapeutic, and dispositional issues in this emergency department population are summarized in Figure 1.2.[1]

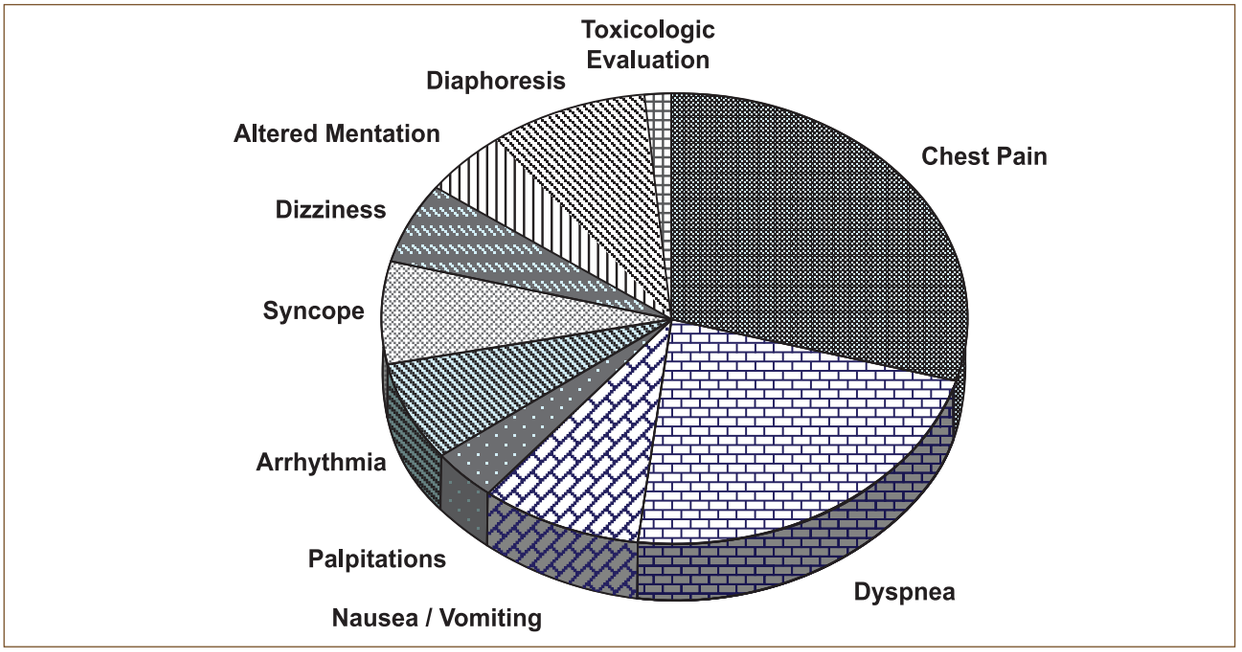

FIGURE 1.1. Clinical reasons for obtaining a 12-lead ECG.[1]

INTERPRETATION OF THE ECG WITHIN THE CLINICAL PRESENTATION

As with other diagnostic evaluations, the ECG must be interpreted within the context of the clinical presentation (ie, age, gender, chief complaint, comorbid medical illness, and results of the physical examination). An understanding of this concept and its application at the bedside is crucial for the appropriate use of ECGs in clinical practice. For instance, the meaning of a 12-lead ECG demonstrating normal sinus rhythm with normal ST segments and T waves (a normal ECG) (Figure 1.3) will differ depending on the patient being evaluated. Patient-based issues are the most important and common considerations in the interpretation of an ECG. A normal ECG from a stable 34-year-old man experiencing pleuritic chest pain will be interpreted very differently than a normal reading from a 64-year-old diaphoretic woman with chest pressure, dyspnea, and pulmonary congestion. The young man's presentation induces less concern than the middle-aged woman's; she is in the early stages of acute coronary syndrome (ACS). In these two scenarios, different evaluation and management pathways will be followed even though both patients have a "normal" ECG.

In scenario-based interpretations, the ECG is interpreted within the context of the circumstances leading to the patient's presentation. For example, the presence of a first-degree atrioventricular

TABLE 1.1. Clinical applications of the ECG.

Assessing the end-organ impact of a syndrome
Assessing the impact of therapy
Continuous or intermittent cardiac monitoring
Determining inpatient disposition location
Establishing a diagnosis
Guiding additional diagnostic studies
Guiding management
Predicting risk of cardiovascular complication
Providing an indication for certain therapies
Ruling out a syndrome

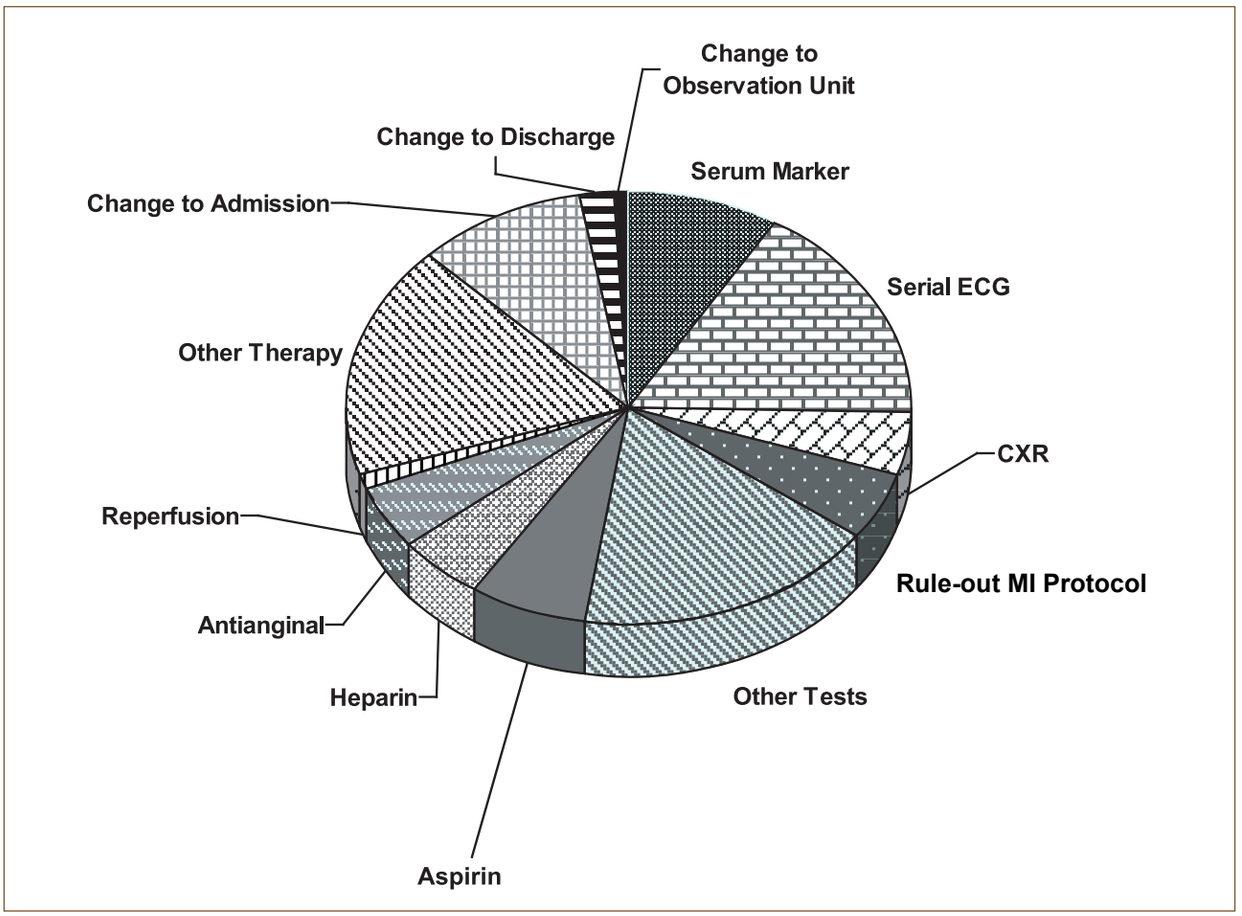

FIGURE 1.2. Impact of the 12-lead ECG on diagnostic, therapeutic, and disposition issues in the emergency department. Note that all changes in evaluation and therapy were additions.[1]

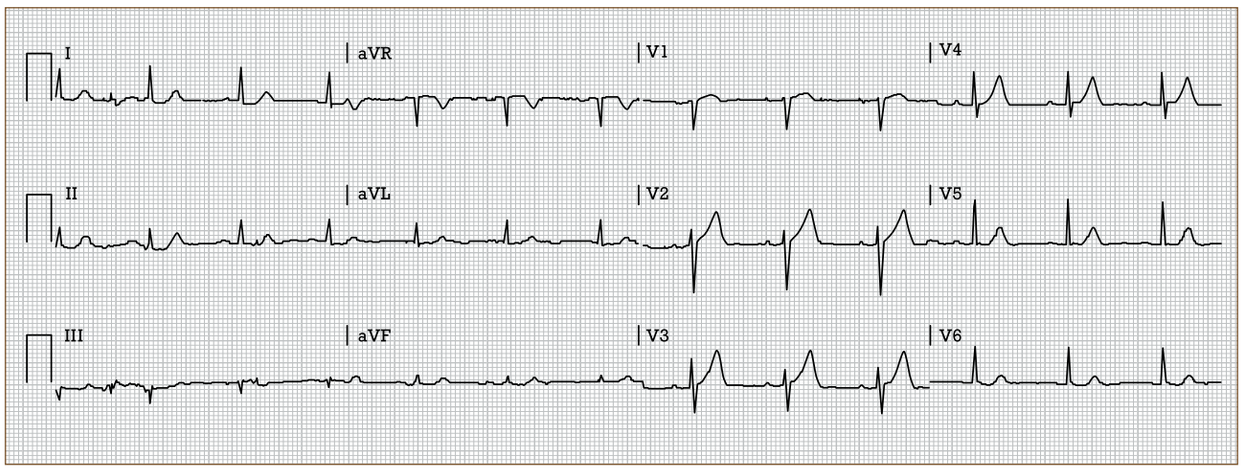

FIGURE 1.3. 12-lead ECG demonstrating normal sinus rhythm with no evidence of ST-segment or T-wave abnormality. A normal 12-lead ECG.

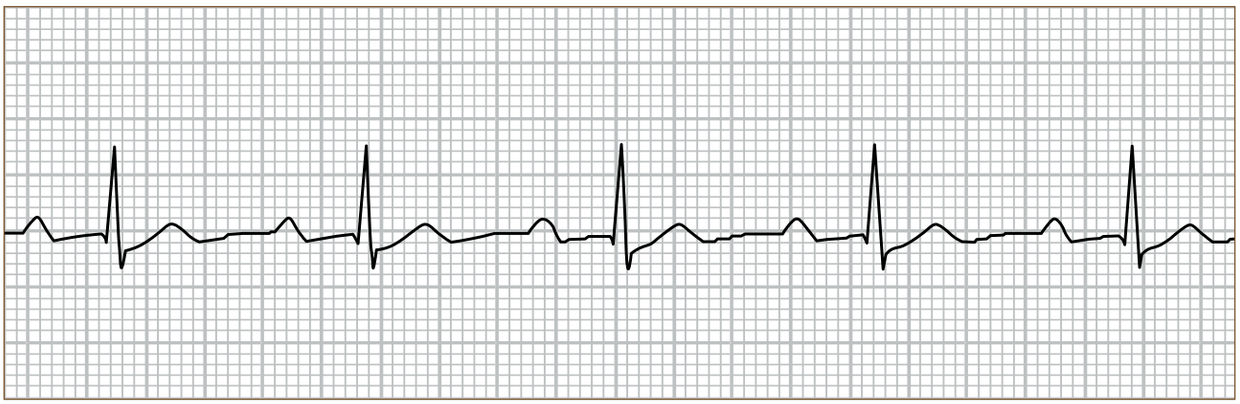

FIGURE 1.4. Normal sinus rhythm with first-degree atrioventricular block.

block (Figure 1.4) induces widely different levels of concern and medical management decisions. It has different meanings in a 27-year-old athletic woman undergoing electrocardiographic evaluation for operative "clearance" after sustaining a trimalleolar fracture of the ankle in a ground-level fall and in a 19-year-old man who ingested a large amount of metoprolol. The same electrocardiographic finding suggests significantly different levels of cardiovascular risk, mandating markedly different management strategies.

The basic, vital message is this: *Interpret the ECG within the context of the clinical presentation evolving before your eyes*. That statement captures the message of this chapter.

CLINICAL SCENARIOS AND THE ECG

The ECG is employed in different situations in the emergency department on a regular basis, including in the evaluation of patients presenting with chest pain, dyspnea, syncope, palpitations, altered mentation, and toxic ingestion and following resuscitation after cardiac arrest. The ECG can be used for many purposes: providing a diagnosis, indicating the extent of an illness, suggesting a therapy, and predicting risk. For instance, in the evaluation of a patient experiencing chest pain and thus suspected of having a coronary event, the ECG is used to help establish that diagnosis or, alternatively, to direct attention to a noncoronary condition. The electrocardiographic findings can also be helpful in selecting appropriate therapy, such as determining the patient's candidacy for fibrinolysis or percutaneous coronary intervention. And the ECG can be used to determine the patient's response to treatments delivered in the emergency department. Lastly, the ECG can help predict the risk of both cardiovascular complications and death.

When a 12-lead ECG is requested in the emergency department, the patient typically has three simultaneous indications.[1] An adult experiencing chest pain (the chest pain is the first indication) is evaluated according to the "rule-out MI" protocol (the rule-out protocol is the second indication) in consideration of ACS (the ACS evaluation is the third indication). In fact, the most frequent indication for an ECG in the emergency department is chest pain; others are dyspnea and syncope. Symptom-based considerations are the most common reasons for obtaining a 12-lead ECG, but patients can have diagnosis-based (eg, ACS and suspected pulmonary embolism [PE]) and system-related indications (eg, "rule-out myocardial infarction" protocol, admission purposes, and operative clearance) as well.[1] These indications involve consideration of a complaint, but the ECG is performed in a process, following the rule-out MI protocol. Although the value of electrocardiographic rhythm monitoring has not been studied, it is reasonable to assume its usefulness in the emergency department, especially for patients who are ill or who could become quite ill quickly.

Chest Pain

The 12-lead ECG is used widely in the evaluation of patients with chest pain. In fact, the most frequent clinical scenario in which an ECG is obtained is an adult patient with chest pain in whom ACS is being considered or to rule out MI.[1] In this application of the ECG, the clinician is attempting to rule in an ACS event with the demonstration of significant ST-segment and T-wave abnormalities. This same symptom-based approach can involve the 12-lead ECG for the diagnosis of other chest syndromes such as acute myopericarditis.

The ECG has a central role in the diagnostic evaluation of patients with chest discomfort. In fact, it is the major criterion for the diagnosis of STEMI, and it often provides information regarding the anatomic location of the infarct-related artery. In presentations of non-ST-segment elevation acute MI and unstable angina, the ECG provides important diagnostic information, yet less than for STEMI. The information provided by the ECG is less straightforward in that the range of abnormality (from minimal nonspecific ST-segment abnormality to obvious ST-segment depression and T-wave inversion) is quite broad.

Clearly, therapeutic interventions can be suggested or indicated based on the 12-lead ECG from a patient with chest pain suspected of ACS. For instance, the individual with chest discomfort who demonstrates anatomically oriented T-wave inversion or ST-segment depression can be a candidate for anticoagulant, antiplatelet, and antianginal therapies. In fact, the ECG provides clinical information that influences management strategies in one-third of ED patients with chest pain.[1]

The 12-lead ECG provides the major indication for acute reperfusion therapy (fibrinolysis or percutaneous coronary intervention [PCI]) in the STEMI patient. The electrocardiographic indications for acute reperfusion are:

- ST-segment elevation in two or more anatomically contiguous leads or
- left bundle-branch block (LBBB) with Sgarbossa criteria. These both are described in more detail in Chapter 6.

No evidence of benefit from fibrinolytic therapy has been found for patients with ACS presentations who lack either appropriate ST-segment elevation or the LBBB findings. For instance, the Fibrinolytic Therapy Trialists Collaborative Group analyzed randomized fibrinolytic therapy trials of more than 1,000 patients and found benefit of fibrinolytic therapy only in those with ST-segment elevation or LBBB.[2] Patients with an acute MI in anterior, inferior, or lateral anatomic locations benefitted from administration of fibrinolytic therapy if it was administered within 12 hours after onset. Benefit was greatest in patients with LBBB and anterior acute MI and least in those with inferior acute MI. Patients with inferior acute MI and right precordial ST-segment depression (presumably acute posterior wall STEMI) or elevation in the right ventricular leads (right ventricular STEMI) have a worse prognosis and benefit more from fibrinolytic agents than patients with isolated inferior ST-segment elevation.[3-12] Inferior acute MI patients with coexisting right ventricular infarctions, as detected by additional-lead ECGs, are likely to benefit because of the large amount of jeopardized myocardium. Acute, isolated posterior wall MI, diagnosed by posterior leads, could represent yet another electrocardiographic indication for fibrinolysis for the same reason (unproven in large fibrinolytic agent trials).[3-12]

Risk stratification is of great importance to emergency physicians. In broad terms, low-risk patients can be discharged safely for outpatient evaluation while high-risk patients generally require more extensive assessment. A more challenging category of patients is those who are at moderate (or intermediate) risk for ACS. In those chest pain patients, a new electrocardiographic abnormality, a positive cardiac biomarker, or acute heart failure represent high-risk features in the evaluation.[13] The ECG has a central role in the risk assessment strategy.

Clinical decision tools have been suggested as an adjunct in risk stratification of emergency department chest pain patients suspected of ACS. It is extremely important to note that clinical decision tools assist in decision-making, but they do not make decisions for the clinician. And, of course, the

ECG has a pivotal role in each of these tools. Most of these tools, when used alone without other clinical data, cannot clearly demarcate levels of risk, that is, they cannot distinctly separate low-risk from intermediate- and high-risk groups, and thus are of limited value to the clinician. Furthermore, the use of risk scoring systems based on inpatient populations (eg, TIMI) is not appropriate for identifying patients who can be discharged safely from an emergency department.

The HEART score is an exception because it identifies a discrete population that has a very low rate of adverse events within 4 to 6 weeks.[14-16] The score has five components: history of the chest pain, the ECG, the patient's age, coronary artery disease risk factors, and initial troponin value. Each variable is given three point values (0, 1, and 2). The ECG points are based on the interpretations of normal, nonspecific repolarization disturbance, and significant ST-segment depression, respectively. A score of 3 or lower is associated with a low risk of a major adverse cardiac event (0.9% to 1.7%).[14-16] The American Heart Association Guidelines 2015 recommend combining serial troponin testing with the HEART score or other clinical decision rules. With negative troponin serial test results and a low-risk HEART score (or an equivalent low-risk score from another decision rule), the adverse event rate is less than 1% at 30 days.[17]

The initial ECG correlates well with patient prognosis after acute MI based on the heart rate, QRS duration, infarct location, and amount of ST-segment deviation.[18-20] The initial 12-lead ECG obtained in the emergency department can be a helpful guide for determining cardiovascular risk and therefore in-hospital admission location. Brush and colleagues classified initial ECGs into high- and low-risk groups. Their low-risk group had absolutely normal ECGs, nonspecific ST-T wave changes, or no change from a previous ECG. The high-risk ECGs had a significant abnormality or confounding pattern such as pathologic Q waves, ischemic ST-segment or T-wave changes, left ventricular hypertrophy, LBBB, or ventricular paced rhythm. Patients with initial ECGs classified as low risk had a 14% incidence of acute MI, a 0.6% incidence of life-threatening complications, and a 0% mortality rate. Patients with initial ECGs classified as high risk had a 42% incidence of acute MI, a 14% incidence of life-threatening complications, and a 10% mortality rate.[21] Another approach to risk prediction involves a simple calculation of the number of electrocardiographic leads with ST-segment deviation (elevation or depression), with an increasing number of leads being associated with higher risk. Risk can also be predicted with a summation of the total millivolts of ST-segment deviation; once again, higher totals are associated with greater risk.[21]

The presence of left ventricular hypertrophy on the ECG is associated with an increased long-term risk of sudden death, acute heart failure, angina, and acute MI.[22] The ECG has been shown to predict adverse cardiac events as well as the release of cardiac serum markers in patients with chest pain and new LBBB, ST-segment elevation, or ST-segment depression. Blomkalns and colleagues raised awareness about the potential for adverse events when pathologic Q waves or T-wave inversion is seen on the ECG, so these abnormalities should be considered in treatment decisions regarding patients with risk factors for coronary disease.[19]

To further evaluate risk for ACS, some authors use standardized electrocardiographic classification systems based on the initial ECG. One example was created by the Standardized Reporting Criteria Working Group of the Emergency Medicine Cardiovascular Research and Education Group to determine whether the initial ECG can predict risk of death, acute MI, or need for revascularization at 30 days after presentation.[23] It has demonstrated high reliability in predicting adverse outcomes and presented the hope that it could lead to better risk stratification for low-risk patients with a normal or nondiagnostic ECG.[20,23]

Dyspnea

Dyspnea is the second most frequently encountered indication for ECG performance in the emergency department population. In this complaint-based situation, the clinician considers

not only the anginal-equivalent ACS presentation but also other cardiorespiratory ailments such as PE. Electrocardiographic issues in the setting of ACS are reviewed above in the Chest Pain section.

The diagnosis of PE relies predominantly on the magnitude of clinical suspicion and the interpretation of various diagnostic investigations at that level of diagnostic concern. The diagnosis should be considered in the patient with unexplained dyspnea; certainly, the acute onset of additional symptoms such as pleuritic chest pain and hemoptysis suggest the possibility of PE, yet, as with most classic symptom constellations, these complaints rarely occur simultaneously. In this evaluation, a myriad of tests may be performed, including initial "screening" studies (chest radiography, 12-lead ECG, arterial blood gas measurement) and more advanced diagnostic investigations (chest computed tomography with angiography, ventilation-perfusion imaging). Along with the chest radiograph, the ECG is often obtained as an initial diagnostic test. Despite this widespread application of the ECG, its diagnostic performance in the patient with suspected PE is rather inadequate. In fact, the most common use of the ECG in this presentation is the exclusion of other diagnoses such as ACS. The ECG should not be used as a primary study to rule in PE because its sensitivity is quite low.

In patients with PE, the ECG might be entirely normal or could show any number of rhythm or morphologic abnormalities. The ECG can deviate from the norm with alterations in rhythm; in intraventricular conduction; in the axis of the QRS complex; and in the morphology of the P wave, QRS complex, and ST segment/T wave. The classic electrocardiographic finding of PE was first reported in 1935 by McGinn and White,[24] who described the traditional $S_1Q_3T_3$ pattern in acute cor pulmonale. However, this "classic" electrocardiographic finding is actually not often seen in patients with PE and is occasionally present in those without it, so its diagnostic power has been described as quite poor. The numerous electrocardiographic findings associated with PE include arrhythmias (sinus tachycardia, atrial flutter, atrial fibrillation, atrial tachycardia, and atrial premature contractions), nonspecific ST-segment/T-wave changes, and findings of acute cor pulmonale (including $S_1Q_3T_3$ pattern, T-wave inversions in the right precordial leads, right axis deviation, and right bundle-branch block).[25] The most common are nonspecific ST-segment/T-wave changes with sinus tachycardia. Unfortunately, these findings are extremely nonspecific.[26]

The relatively low sensitivities of these electrocardiographic presentations limit our ability to use the ECG as a sole diagnostic tool. Electrocardiographic changes are seen most frequently in patients with massive or submassive embolization; smaller PEs less often produce significant electrocardiographic abnormality. Various studies have shown that 15% to 30% of ECGs are normal in patients with established PE. Perhaps as a partial explanation of this relatively high rate of the "normal" ECG, it has been noted that the range of electrocardiographic findings in PE is transient, usually appearing during the acute phase of the illness.[27] As an independent marker, the ECG continues to be a limited study due to its poor sensitivity. The transient nature of electrocardiographic abnormalities and the often nonspecific changes reduce the effectiveness of the test as a single agent. The clinician must be aware of these electrocardiographic limitations in the application of the 12-lead ECG in the patient suspected of having PE.

Syncope

The patient with syncope presents a significant challenge to emergency physicians; this scenario is yet another common indication for electrocardiography in the emergency department. Most of these patients ultimately will have a favorable outcome; a significant minority, however, will be diagnosed with a life- or limb-threatening event or will die. Several clinical variables have demonstrated utility in the evaluation of patients with syncope; the ECG, of course, is one of them.

Certain obvious electrocardiographic presentations in the syncope patient will not only provide a reason for the loss of consciousness but also guide early therapy and disposition. Bradycardia, atrioventricular block, intraventricular conduction

abnormality, and tachyarrhythmia in the appropriate clinical setting provide an answer for the syncopal event. Morphologic findings suggesting the range of cardiovascular malady are also encountered; they are far ranging, including the various ST-segment and T-wave abnormalities of ACS, ventricular hypertrophy suggestive of hypertrophic cardiomyopathy, ventricular preexcitation as seen in the Wolff-Parkinson-White syndrome, prolonged QT interval common in the diverse long QT interval presentations, and Brugada syndrome with the associated tendency for sudden death. Of course, this list is by no means inclusive.

Investigators have studied ECGs from patients with syncope with the aim of identifying individuals at risk for adverse outcome. For instance, Martin and colleagues endeavored to develop and validate a risk classification system for patients presenting to the emergency department with syncope. In a two-step analysis, they reviewed the presentations of 612 patients and found that an abnormal ECG was associated with arrhythmia or death with an odds ratio (OR) of 3.2. Other factors suggestive of poor outcome included histories of acute heart failure (AHF) (OR 3.2) and ventricular arrhythmia (OR 4.8).[28] Additional work by Sarasin and colleagues[29] considered the subset of patients with unexplained syncope after an initial emergency department evaluation. In 344 patients, those investigators found that an abnormal ECG was a predictor of arrhythmia, with an OR of 8.1. Other factors of significance associated with arrhythmic syncope included older age (OR 5.4) and a history of AHF (OR 5.3). In patients with one risk factor, arrhythmia was encountered rarely (0–2%). In patients with identified risk factors, arrhythmia occurred at the following frequencies: one risk factor, 0 to 2%; two, 35% to 41%; and three, 27% to 60%. The San Francisco Syncope Rule incorporates the ECG into the evaluation of patients with syncope. Quinn and associates considered 684 presentations of syncope and reviewed clinical variables with the intent of identifying patients at risk of poor short-term outcome.[30] An abnormal ECG was associated with an increased risk of short-term adverse event, and dyspnea, low hematocrit, and hypotension were predictors of poor outcome.

The most appropriate electrocardiographic approach to these patients is an initial review aimed at the detection of malignant arrhythmia. This first evaluation most often involves the rhythm strip. The detection at this stage of the evaluation is diagnostic of the cause and will mandate therapy. If the electrocardiographic rhythm strip does not yield an answer, then a 12-lead ECG can be performed. It can provide a more detailed review of a challenging rhythm presentation as well as the various morphologic findings noted above. Of course, a "negative" ECG itself does not rule out cardiac pathology.

Toxic Ingestion

The clinician approaches the poisoned patient with numerous important diagnostic tools, including the history of the ingestion, the physical examination demonstrating various toxidrome findings, and selected investigative tools. One of these tools is the ECG. In the poisoned patient, the ECG takes the form of the electrocardiographic rhythm strip and the 12-lead ECG. The ECG is used to establish the diagnosis, assess for end-organ toxicity, and guide therapeutic interventions. Not unlike patients with syncope, individuals presenting with significant cardiotoxicity manifested by arrhythmia will be assessed with an electrocardiographic monitor. Further diagnostic and management decisions will be suggested based on bedside interpretation of the rhythm strip.

For "stable" patients (ie, patients with a perfusing, stable cardiac rhythm), a 12-lead ECG is obtained. In addition to rhythm interpretation, this ECG is reviewed for abnormalities of the various structures, intervals, complexes, and axes. Beyond rhythm considerations, the primary electrocardiographic determinants of impending or established cardiotoxicity include the PR interval, the QRS complex, the T wave, the ST segment, and the QT interval.

Numerous authorities have explored the ECG in patients with suspected or known toxic diagnoses. It has been thoroughly explored in presentations after tricyclic antidepressant (TCA) ingestion and

digoxin exposure, but little clinical information is available about the general use of the ECG in poisoned patients. Homer and associates undertook such a study,[31] with the goal of reviewing the range of electrocardiographic abnormalities encountered in poisoned patients. All patients evaluated for poisoning by the toxicology service at a tertiary referral center who underwent electrocardiographic analysis within 6 hours after ingestion were entered in the study. Each ECG was reviewed for rhythm and morphological diagnoses as well as interval/complex duration. Two hundred seventy-seven patients underwent electrocardiographic evaluation; 32% of them had a normal ECG. Of the patients (68%) with abnormal ECGs, 62% had a rhythm abnormality and 38% had morphologic abnormality. Rhythm disturbances included sinus tachycardia (51%), sinus bradycardia (7%), atrioventricular block (7%), nonsinus atrial tachycardias (3%), and nodal bradycardia (3%). Morphologic abnormalities included abnormal QRS configuration (35%), QRS complex widening (33%), QT-interval prolongation (33%), PR-interval prolongation (12%), ST-segment abnormality (9% elevated, 25% depressed), and T-wave inversion (20%). Interestingly, the degree of abnormality was directly related to the number of toxins ingested, but the cardiovascular agents (beta-adrenergic blockers and calcium channel antagonists) were no more likely to produce electrocardiographic abnormality than were noncardiovascular substances (sedative-hypnotic medications and stimulants). Importantly, this analysis did not include patients who underwent electrocardiographic rhythm analysis via the monitor. It likely would have missed the more malignant rhythm presentations, such as ventricular tachycardia or complete atrioventricular block. This study demonstrated that the ECG is, in fact, frequently abnormal in the poisoned patient. It did not explore the impact of an abnormal ECG on medical decision-making and patient management.

The ECGs of patients exposed to sedative-hypnotic or psychotropic medications have been explored thoroughly; importantly, the impact of the electrocardiographic findings has also been reviewed in various studies. For instance, patients with TCA poisoning have a range of electrocardiographic abnormalities, including arrhythmia, QRS-complex widening, QRS-complex configuration (prominent, terminal R wave in lead aVR, and S wave in lead I), and QT-interval prolongation. Specific electrocardiographic findings have different clinical implications; for example, sinus tachycardia, although present in many TCA-poisoned patients, is a nonspecific finding. Widening of the QRS complex, a more specific finding suggestive of TCA cardiotoxicity, is more useful. QRS complexes more than 100 milliseconds in duration are predictive of convulsion.[32] Conversely, a normal QRS complex duration is not "protective" in that convulsion and malignant arrhythmia can be seen in this group as well.[32,33] In general, with increasing QRS complex duration, the clinician is more likely to encounter significant end-organ toxicity. Rightward deviation of the terminal 40 milliseconds of the QRS complex frontal plane axis is also associated with both neurotoxicity and cardiotoxicity. A rightward axis of the terminal QRS complex is easily detected on the 12-lead ECG via observation of a prominent R wave in lead aVR and a deep S wave in lead I. This finding is reasonably predictive of either seizure or ventricular arrhythmia, with a sensitivity of 81%. As with increasingly wider QRS complexes, progressively larger R waves are associated with greater toxicity.[34] QTc interval prolongation is also seen in these patients but is not necessarily indicative of TCA cardiotoxicity or predictive of an impending adverse event.

Numerous studies[35,36] have noted that these and other electrocardiographic abnormalities occur commonly in the TCA-poisoned patient. The authors also point out that, even when these electrocardiographic findings are applied in collective fashion, they demonstrate less-than-reliable sensitivity and specificity for both the diagnosis as well as the occurrence of convulsion or malignant arrhythmia, meaning that the clinician should not employ these criteria alone as the reason to either rule in or rule out TCA poisoning. Baily and colleagues performed a meta-analysis of electrocardiographic prognostic indicators[37] and reported the frequent occurrence of these electrocardiographic abnormalities in the

ill TCA-poisoned patient. Unfortunately, these abnormalities were not entirely predictive of the development of significant end-organ toxicity, with sensitivities and specificities ranging from 69% to 81% and 46% to 69%, respectively.

When these electrocardiographic findings are interpreted within the context of the clinical presentation, they are markedly more powerful. In the TCA-poisoned patient who is fully alert and oriented, sinus tachycardia is an abnormal but nonspecific finding that is not necessarily indicative of impending cardiotoxicity. On the other hand, the same finding in a lethargic TCA-overdosed patient, is a stronger predictor of significant toxicity. If sinus tachycardia is complicated by a widened QRS complex and prominent R wave in lead aVR, the patient's mental status (normal or altered) does not significantly influence interpretation of the ECG: in both scenarios, the patient is at extreme risk of an adverse event.

The 12-lead ECG can be applied in serial fashion as a screening tool in the patient who is asymptomatic at presentation. This use of the ECG in the diagnosis of an asymptomatic patient (ie, fully alert with normal mentation and the absence of tachycardia) can aid the clinician in ruling out significant TCA poisoning. This type of patient can be monitored over a 6-hour period. If serial electrocardiography does not demonstrate tachycardia, QRS-complex widening, terminal QRS-complex rightward axis shift, or QT-interval prolongation, then the patient is unlikely to have significant TCA poisoning.[38]

Other Scenarios

Presentations involving metabolic abnormality, altered mentation, cardiorespiratory arrest, or blunt chest trauma are evaluated with numerous diagnostic studies. In the renal failure patient, hyperkalemia can be diagnosed early, even before serum laboratory test results become available, with the ECG. Obviously, abnormalities of the T wave and/or QRS complex suggest the diagnosis and allow potentially life-saving therapy to be delivered expeditiously. The ECG provides the reason for altered mental status in 7% of patients presenting to the emergency department with abnormal mentation.[39] Patients experiencing cardiorespiratory arrest are managed with the ECG while in active arrest; after resuscitation, the ECG continues to guide therapy; and after stabilization, a 12-lead ECG might yield clues as to the cause of the hemodynamic collapse such as STEMI or PE. Unexplained hypotension in the blunt trauma patient can result from myocardial contusion; the ECG can confirm the diagnosis during the early phase of the trauma evaluation.[40]

LIMITATIONS OF THE ECG

The ECG has numerous limitations when used in the patient suspected of ACS or another acute event. For instance, the adult with chest pain who is ultimately diagnosed with STEMI can demonstrate a normal or minimally abnormal ECG on presentation; the ECG then evolves over minutes to hours into STEMI. In non-ACS scenarios, the initial ECG after TCA ingestion might not reveal pathologic abnormality in a patient with impending toxicity, and the ECG from a patient found to have PE might show nonspecific findings such as sinus tachycardia. These clinical situations demonstrate the importance of having a sound understanding of the ECG's limitations, which will guide emergency physicians in appropriate applications of the 12-lead ECG.

For potential ACS patients, the ECG has additional shortcomings in the following scenarios: "normal" and "nondiagnostic" electrocardiographic presentations; evolving, confounding, and mimicking syndromes; and the "electrocardiographically silent" areas of the heart (eg, isolated acute posterior wall STEMI). The ECG that is diagnostic for acute MI at emergency department presentation is seen in only 50% of patients ultimately diagnosed with acute MI. The remaining patients have ECGs that are entirely normal, nonspecifically abnormal, or clearly abnormal yet without pathologic ST-segment elevation indicative of STEMI. Lee and colleagues[41] reported that a significant portion of emergency department patients suspected of ACS had a normal or minimally abnormal ECG yet were ultimately diagnosed with ACS (4%–20% had unstable angina and 1%-4% had acute MI). Pope and colleagues,[22] in a

description of emergency department patients with the missed ACS diagnosis (2.1% unstable angina and 2.3% acute MI), noted a number of factors that could have contributed to the initial incorrect assessment; one was a normal ECG on presentation. Overreliance on a normal or nonspecifically abnormal ECG from a patient with potential ACS who is currently pain free should be avoided. Furthermore, the elapsed time from chest pain onset in patients with these nondiagnostic electrocardiographic patterns does not assist in ruling out the possibility of acute MI with a single ECG.[42]

It is important to understand that the single, initial emergency department ECG is merely a "snapshot" of the status of coronary perfusion and its effect on the myocardium, that is, an electrocardiographic abnormality that suggests ACS. Patients with nondiagnostic ECGs have probably presented during an early phase of their syndrome. ACS is a dynamic, evolving process—it follows that the ECG will change and evolve over time as the syndrome progresses. The history and other clinical data must be relied on heavily in patients with either normal or minimally abnormal ECGs and a convincing description of ischemic chest discomfort; in these patients with reasonable clinical suspicion for ACS, serial ECGs might reduce the initial relatively poor sensitivity for acute MI. Management and disposition decisions must be based on the total clinical picture, not on a nondiagnostic ECG.

A broad range of electrocardiographic abnormalities is encountered in adult emergency department patients with chest pain, some of whom are presenting with ACS while others are experiencing a noncoronary ailment. Certain electrocardiographic syndromes commonly mimic ischemia such as benign early repolarization, acute pericarditis, left ventricular hypertrophy, and bundle-branch block. For instance, ST-segment elevation in an adult with chest pain does not equate with STEMI. In fact, a minority of chest pain patients with ST-segment elevation are diagnosed with STEMI; most of them are ultimately diagnosed with non-STEMI syndromes. This observation has been noted in prehospital, emergency department, and coronary care unit populations.[43-46] A sound, thorough understanding of the various electrocardiographic syndromes encountered in the emergency department is crucial in the initial evaluation and subsequent management of these patients.[47,48] The electrocardiographic abnormalities associated with acute MI can be masked by the altered patterns of ventricular conduction encountered in patients with confounding patterns, including LBBB, ventricular paced rhythm, and left ventricular hypertrophy. These electrocardiographic syndromes produce ST-segment and T-wave changes that are the new "normal" findings in these patients. These electrocardiographic findings can obscure or mimic the typical electrocardiographic findings of ACS, including STEMI. Emergency physicians must approach these patients with the realization that the ECG is of limited diagnostic power. Further diagnostic and management decisions must be made with this caveat in mind.

The limitations of the ECG in ACS patients are well known and reasonably well elucidated in the literature. Other clinical scenarios, as described above in the Clinical Scenarios section, demonstrate similar limitations, ranging from major to minor. For instance, the 12-lead ECG in a patient suspected of having PE offers very little diagnostic information; conversely, the ECG in a patient with hyperkalemia provides evidence of the degree of cardiotoxicity in most instances, whether it demonstrates prominent T waves or QRS-complex abnormalities.

The important issue to consider is the presence of these limitations and their magnitude. With this knowledge in mind, the clinician is able to approach the patient and use the ECG in appropriate fashion.

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