Dr Brady’s dedication – To my wife, King, for her constant support, patience, and guidance; and for my children, Lauren, Anne, Chip, and Katherine, for their love.

Dr Truwit’s dedication – To my wife Jeanne and my children, Jason, Matthew and Lauren without whom I could not be personally or professionally fulfilled nor accomplish as much.
Critical Decisions in Emergency and Acute Care Electrocardiography

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Electrocardiography is performed widely throughout medicine, ranging from the clinician’s office in a scheduled, routine application to the critical care unit with an unanticipated decompensation during active resuscitation. And, of course, a multitude of other areas rely heavily on the ECG as a valuable tool in the patient evaluation – the prehospital setting in an EMS unit, the emergency department, the surgical suite and post-anesthesia care area, among many others. In fact, it is appropriate to state that some form of electrocardiographic monitoring is one of the most widely applied diagnostic tests in clinical medicine today. Electrocardiography, whether single-lead monitoring for rhythm disorders or 12-lead analysis for ACS or other morphologic abnormality, remains one of the most cost-effective and useful tests in medicine – rapid, non-invasive, inexpensive, portable, easily interpreted – often providing clinical information that will make the difference between life and death.

In acute care medicine, whether it be the acute care ward, emergency department, or critical care unit, the ECG can assist in establishing a diagnosis, ruling-out various ailments, guiding the diagnostic and management strategies in the evaluation, providing indication for certain therapies, determining inpatient disposition location, offering risk assessment, and assessing end-organ impact of a syndrome. In more routine, though no less crucial, settings, the ECG assists in disease surveillance and screening in office-based evaluations as well as risk stratification in pre-operative assessments.

The ECG, similar to other clinical investigations, must be interpreted within the context of the clinical presentation. An understanding of this concept and its application at the bedside is crucial for the appropriate use of the ECG in clinical practice – and is the focus of this textbook, *Critical Decisions in Emergency and Acute Care Electrocardiography*. This textbook focuses on the breadth of acute care medicine – the ward, ED, OR, and critical care unit. Each section is organized around traditional topics such as acute coronary syndrome or dysrhythmia. Within each section, however, are a range of chapters, focusing on a specific use or clinical situation, involving the ECG; each chapter is presented in the form of an inquiry, followed by a series of cases, illustrating the issues, controversies, or questions. For instance, what are the electrocardiographic indications for urgent reperfusion therapy in ACS, can the ECG guide the clinician in the management of the patient with wide complex tachycardia, or what is the value of the 12-lead ECG in the poisoned patient? The chapter itself is the answer to the question with appropriate electrocardiographic examples and adequate supporting evidence.

This work stresses the value of the ECG in the range of clinical situations encountered daily by healthcare providers – it illustrates the appropriate applications of the electrocardiogram in acute care medicine today. We have enjoyed its creation – we hope that you the clinician will find it of value in your care of the patient.

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In clinical medicine, there are a finite number of clinical skills that are considered essential areas of expertise in the management of critically ill patients. Such a short list might include advanced physical assessment, airway management, critical care problem solving, initiation of resuscitation efforts, identification of the need for early surgical intervention, and the immediate diagnostic interpretation of tests. One of the earliest and most common diagnostic studies performed in the critically ill patient is the electrocardiogram. The tremendous value of the electrocardiogram in the acutely and critically ill patient is unequivocally established—in fact, it is considered essential in management. Certainly, the basic electrocardiographic skill set is considered fundamental; the intricacies and nuances of advanced interpretation offers an abundance of clinical data that can alter patient course and outcome—and should also be considered fundamental in acute, emergency, and critical care settings. Drs. Brady and Truwit have assembled such a text which very nicely explores and reviews the impact of the electrocardiogram, from the prehospital arena and emergency department to the inpatient ward and critical care unit.

Patient safety and outcome goals have moved electrocardiographic analysis from the sole responsibility of the cardiologist to the point of care contact for our patients. Expertise in electrocardiographic interpretation is considered the standard of training in emergency medicine and critical care. Appropriately, there has been mounting pressure on acute care and critical care clinicians to rapidly and accurately assess electrocardiograms in a time dependent fashion. Critical time points have been established for electrocardiographic interpretation in acute ST segment elevation myocardial infarctions that directly impact patient treatment strategies, hospital resources and outcomes. Correct interpretation of the electrocardiogram alter treatment decisions for the management of non-ST elevation myocardial infarctions, dysrhythmias, undifferentiated cardiovascular diseases, and poisoning and ingestions. Additionally, electrocardiograms offer insights into other medical conditions that place acutely and critically ill patients in life threatening situations.

Developing expertise in electrocardiographic analysis requires dedicated study, practice and review. The management of critically ill patients at risk for cardiovascular compromise requires not just a basic familiarity in electrocardiography, but an advanced interpretation skill level. Failure to develop expertise in the area of electrocardiography places patients at risk. Acute care electrocardiographic expertise is developed through meaningful self-education, clinical practice, and thoughtful review. Standardizing this process is essential because clinical experience alone is inadequate in addressing the breadth and extent of the required knowledge base.

This textbook on Critical Decisions in Electrocardiography represents an excellent example of standardizing the educational process of electrocardiography. By reviewing case scenarios, learners can explore and actively participate in critical decision making that is required for developing these essential diagnostic skills. The breadth of clinical presentations offers the learner an opportunity to review and reflect on high risk cardiovascular disease states that may not frequently present in their own clinical practice. The text allows for independent study and reflection that can lead to expertise in the field of electrocardiography, providing an integral component in the pursuit of a competency that our patients rely on and deserve.

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William Brady and Jon Truwit have done a masterful job at taking a topic that, while central to the Operating Room, ICU, Emergency Department environments, it is not usually the focus for the personnel regularly working in these areas. As a practicing pulmonary-intensivist, I know that when it comes to ECG abnormalities in the ICU we are exposed to anything and everything, often with very short notice and little time for diagnosis or to think about the most appropriate therapeutic interventions. The common problems such as atrial tachy arrhythmias, ischemic changes, ventricular tachycardia and signs of myocardial infarction occur with such frequency that it is relatively easy to maintain skills necessary to recognize and treat them. However, the uncommon problems are often seen so infrequently that recognition and treatment can be much more of a challenge. Thus Brady and Truwit in *Critical Decisions in Emergency and Acute Care Electrocardiography* have created a text that makes common and obscure ECG findings relevant and accessible.

Intensivists and others who do not regularly work in cardiac units must still maintain skills sufficient to recognize and provide at least the initial management of serious and/or life-threatening diseases manifesting in or resulting from abnormal ECGs. Though complex and challenging, these clinical problems are systematically dealt with by Brady and Truwit in a practical, easily readable format. The format of case presentations followed by a complete and systematic well-organized discussion is designed to give the reader information in a natural flow that facilitates assimilation into practice. The concise but very meaningful discussions of the controversies that loom large in some areas are well-articulated and serve to place much of the information into proper context. The fact that a whole chapter is devoted to the limitations of the ECG in clinical practice is a refreshing testament the pragmatism this volume brings to the field.

The ECG has been around a long time, has many limitations and must be interpreted in the light of the overall clinical presentation including prior probabilities. While the shape of the squiggles on the paper strips have not changed since Einthoven’s work in 1895, the true underlying diseases or processes (diagnoses) these represent have been greatly clarified. In addition the prognostic value of the ECG has greatly improved and we are still learning. Brady and Truwit efficiently takes us right up to the edge of the current state of knowledge.

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Part 1 | **The ECG in Clinical Practice**
Chapter 1  |  What are the clinical applications of the ECG in emergency and critical care?

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Case presentations

Case 1: A 56-year-old man with a history of hypertension and chronic kidney disease is brought to the emergency department (ED) by his wife, with a chief complaint of progressive lethargy and fatigue over several days. On physical exam the patient appears ill. His blood pressure is 90/50 mmHg. The electrocardiogram (ECG) (Figure 1.1a) demonstrates a regular wide complex QRS rhythm with a right bundle branch-like morphology. The QRS complex is not preceded by P waves and occurs at a rate of approximately 48 beats per minute (bpm). The emergency physician initiates appropriate treatment based upon the electrocardiographic findings; laboratory findings confirmed the diagnosis. Four hours later, the ECG (Figure 1.1b) demonstrates sinus tachycardia at a rate of 103 bpm. Note that the P waves have returned and the QRS complex is now narrow.

Case 2: A 24-year-old graduate student is brought to the ED after calling 911 stating that she overdosed on her antidepressant medication. She is alert, crying, and complains of a dry mouth. Her initial heart rate is 96 bpm with a blood pressure of 120/80 mmHg. Her examination is remarkable for dilated pupils, flushed skin, and occasional twitching of muscles in her arms and legs. The baseline ECG demonstrates sinus rhythm with a markedly prolonged QT interval. The patient suddenly becomes lethargic and a 12-lead ECG (Figure 1.2) prompts the emergency physician to consider urgent therapy.

Case 3: A 59-year-old woman with a history of diabetes, hypertension and hyperlipidemia is brought to the ED having experienced increased difficulty breathing and neck discomfort. On physical examination, the patient is tachypneic with a respiratory rate of 30 breaths per minute. Her heart rate is 106 bpm with a blood pressure of 100/60 mmHg. An ECG (Figure 1.3) is obtained. The patient is emergently taken to the cardiac catheterization lab for primary percutaneous coronary intervention (PCI). Repeat ECG after the procedure demonstrates resolution of all electrocardiographic abnormalities.

Case 4: A 74-year-old woman with a history of tobacco use, hypertension, and hyperlipidemia presents with nausea and an episode of syncope. The initial ECG (Figure 1.4) in the ED shows sinus tachycardia and an inferior ST elevation myocardial infarction (STEMI). She presented 4 hours after the onset of symptoms. The cardiac catheterization laboratory is activated to perform primary PCI. Prior to transfer, the patient suddenly becomes diaphoretic and lethargic. Her heart rate decreases to 30 bpm and her blood pressure is 70/50 mmHg. The patient is treated promptly with intravenous atropine, and the sinus tachycardia returns.

Clinical applications of the ECG

Electrocardiography is performed widely throughout emergency and critical care medicine. In fact, some form of electrocardiographic monitoring is one of the most widely applied diagnostic tests in clinical medicine today – including both single- and multiple-lead analysis as well as the 12-lead ECG. The ECG can assist in establishing a diagnosis, ruling-out various ailments, guiding the diagnostic and management strategies in the evaluation, providing indication for certain therapies, determining inpatient disposition location, and assessing end-organ impact of a syndrome. In the ED environment, the ECG less often provides a specific diagnosis.

Metabolic abnormalities: The surface 12-lead ECG is a reflection of the changes in the transmembrane potential of cardiac myocytes that occur with atrial and ventricular depolarization and repolarization. The transmembrane potential is the electrical gradient from the interior to the exterior of cardiac myocytes. This gradient results from differences in the extracellular and intracellular concentrations of specific anions and cations as determined by the Nernst equation. The cations that contribute significantly to the creation or
Figure 1.1 (a) Wide QRS complex bradycardia without P wave activity. (b) Improvement in the ECG from (a) with narrowing of the QRS complex, development of P waves, and increase in the rate. These ECGs are consistent with profound hyperkalemia that has markedly improved with therapy.
Figure 1.2 Polymorphic ventricular tachycardia (PVT). With review of the ECG in sinus rhythm, a prolonged QT interval was noted. With this additional finding, the PVT can be termed torsade de pointes. Note the varying QRS complex morphology (axis, amplitude, and contour) in a pattern suggesting “twisting about a fixed point.”

Figure 1.3 Sinus tachycardia with anterolateral wall STEMI, first-degree AV block, and right bundle branch block. Note the ST segment elevation in leads aV1, V2 and V3, consistent with anterolateral wall STEMI. The ST segment elevation in these leads is concordant with the major, terminal portion of the QRS complex – indicative of STEMI in RBBB.
The maintenance of this gradient include potassium, sodium, calcium, and magnesium. As such, shifts in the extracellular or plasma concentrations of these cations may result in changes in the surface ECG. These changes are readily appreciated when the baseline ECG is completely normal. However, abnormalities from infarction/ischemia can make changes from electrolyte shifts more difficult to appreciate. In critically ill patients, two or more electrolyte abnormalities may co-exist, resulting in several changes in the ECG, some of which may mask each other.

Hyperkalemia: The electrocardiographic hallmarks of moderate hyperkalemia are symmetric, tall, peaked T waves and low amplitude P waves. Severe hyperkalemia produces a widened QRS complex without P waves. The electrocardiographic changes associated with hyperkalemia are a reflection of changes in the depolarization/repolarization waveform of the cardiac myocytes. One of the first changes observed on the surface ECG when the extracellular potassium ion concentration increases above 6.0 mM is narrow, peaked, tall T waves. This phenomenon occurs because repolarization of the ventricles occurs more synchronously [1]. As the extracellular potassium ion concentration increases further (6.5–8.0 mM), the resting membrane potential of the cardiac myocytes depolarizes, resulting in a slower conduction velocity. Slow depolarization across the atria is appreciated on the surface ECG as prolonged P wave duration with low amplitude. The HV interval also increases further, contributing to the lengthening of the PR interval. Slow depolarization across the ventricles results in prolongation of the QRS complex. If the extracellular potassium ion concentration increases beyond 8.0 mM, then the P wave may be visible for longer on the surface ECG. In canine models of hyperkalemia, once SA node block occurs, action potential propagation from the atrial pacemaker site to the ventricles may occur via atrianodal pathways [2]. At high extracellular potassium ion concentrations, the QRS complex may resemble a left bundle branch-like or right bundle branch-like waveform. With further increases in extracellular potassium ion concentration, activation of multiple pacemaker foci may result in an irregular rhythm. The QRS complex may become so wide that it takes on the appearance of a sine wave. An explanation of this phenomenon is that cardiac myocytes in one area of the ventricle may repolarize before the action potential wavefront has traversed and depolarized the more distant cardiac myocytes. Once the plasma potassium ion concentration rises to 12–14 mM, ventricular fibrillation and asystole may occur [3]. This phenomenon is exploited during on-pump cardiac surgery by bathing or perfusing the heart with a cardioplegia solution.

Hypokalemia: The electrocardiographic hallmarks of hypokalemia are a prominent U wave and prolongation of the QT(U) interval. With profound hypokalemia or with hypokalemia in the presence of cardiac toxic medications, torsade de pointes may be precipitated. As with hyperkalemia, the electrocardiographic changes associated with hypokalemia can be correlated with changes in the cardiac myocyte action potential waveform. Because the changes observed with hypokalemia affect the QT(U) portion of the ECG, it is changes in the ventricular action potential waveform that provide insight into the surface electrocardiographic
changes. Hypokalemia results in prolongation of phase 3 repolarization. As the extracellular potassium ion concentration drops, the U wave amplitude increases and the T wave amplitude decreases. With a further decrease in potassium ion concentration, the U wave may begin to fuse with the preceding T wave. Hypokalemia can also result in increased arrhythmia. Davidson and Surawicz reported that the incidence of ectopic complexes was three times higher among patients who had a potassium ion concentration of ≤3.2 mEQ/L than control subjects [4]. Paroxysmal atrial tachycardia with block can also be observed in patients with hypokalemia. Severe hypokalemia can also precipitate ventricular tachycardia (VT), ventricular fibrillation (VF), or torsade de pointes [5]. Important non-cardiac manifestations of severe hypokalemia include rhabdomyocytis, metabolic alkalosis, and ascending paralysis.

**Hypokalemia:** During phase 2 depolarization of the cardiac myocyte action potential, calcium slowly enters the cell. The duration of phase 2 correlates directly with the duration of the ST segment [6]. When the extracellular calcium ion concentration is elevated, phase 2 occurs relatively rapidly, producing a short ST segment [7]. Nierenberg and Ransil [8] reported a series in which the Q to apex of T wave interval corrected for rate was 0.27 seconds or less in over 90% of hypokalemia cases. The presence of hypokalemia and hypokalemia produces an interesting ECG. This can be seen with multiple myeloma. The hypokalemia results in a short ST segment and the hypokalemia results in a prominent U wave. Tachyarrhythmias due to hypokalemia are uncommon in the literature. However, bradyarrhythmias with hypocalcemia are well described [9]. The classic case of hypokalemia is that of patients presenting with metastatic non-parathyroid cancer that secretes recombinant parathyroid hormone (rPTH).

**Hypocalcemia:** The surface electrocardiographic changes associated with hypocalcemia are the opposite of those associated with hypocalcemia. Phase 2 depolarization of the cardiac myocyte action potential is lengthened, so the ST segment is prolonged. Suriwicz and Knilans [10] state that hypothermia and hypocalcemia are the only two conditions that increase the length of the ST segment without changing the T wave duration. Suriwicz and Lepeschkin [11] report that isolated hypocalcemia rarely causes the QTc interval to lengthen beyond 140% of normal. If the calculated QTc interval is over 140% of normal, then the measured QT interval may actually be the QU interval due to concomitant hypokalemia. Early after-repolarizations may be observed with hypocalcemia. Importantly, life-threatening arrhythmias can be precipitated with hypocalcemia in the presence of digoxin.

Vigilance for hypocalcemia is critical after thyroidectomy in the case of unintentional parathyroidectomy and, of course, after parathyroidectomy. Clinical scenarios other than primary parathyroidism in which hypocalcemia may occur include acute pancreatitis, rhabdomysisitis, and other specific endocrine disorders involving calcium ion metabolism. Carlstedt and Lind [12] report that as many as 50% of critical care patients may have hypocalcemia. The classic clinical features of hypocalcemia include neuromuscular irritability, tetany, and tonic clonic seizure activity. Bedside tests consistent with hypocalcemia include Chvostek’s and Trousseau’s signs. Therapy for life-threatening arrhythmias and severe symptoms secondary to hypocalcemia includes intravenous calcium solution infusion along with treatment for any other co-existing electrolyte and metabolic conditions.

**Other syndromes:** Magnesium is largely an intracellular cation. Approximately 1% of total body magnesium is in the extracellular space [13]. No specific arrhythmias are associated with hyper- or hypomagnesemia. However, hypomagnesemia may occur in the context of hypocalcemia. Intravenous magnesium sulfate is part of the recommended therapy for torsade de pointes after defibrillation. Additionally, intravenous magnesium sulfate is often given routinely prior to administration of ibutilide for chemical cardioversion of atrial fibrillation.

The presence of isolated hyper- or hyponatremia within the limits compatible with human life is not associated with any specific ECG changes that are well described in the literature. It is noteworthy that hyponatremia in the context of severe hyperkalemia, that would otherwise cause an intraventricular conduction delay, results in a relatively shorter QRS duration than predicted by the degree of hyperkalemia alone. Conversely, the QRS duration is further lengthened in severe hyperkalemia with an intraventricular conduction delay if hyponatremia is present.

**Torsade de pointes:** Torsade de pointes is a syndrome of ventricular tachycardia in which the electrical axis “twists” around. The QRS complex exhibits a crescendo-decrescendo variation in amplitude. The R-R interval is frequently in the range of 200–250 bpm. One of the characteristic features of torsade is a long period of ventricular repolarization so that the QT interval is typically at least 500 ms long. This prolonged QT interval is most readily observed in the QT interval immediately prior to the onset of torsade. Most cases of torsade are preceded by long-short R-R cycles [14]. For example, after a premature ventricular complex a compensatory pause will occur, and then a sinus beat with a long QT interval will occur. If another PVC occurs, torsade may be initiated. If a premature stimulus occurs near the zenith of the T wave, it may be more likely to induce a ventricular arrhythmia [15]. However, a short couple variant with a particularly high mortality has been described [16, 17]. It is important to distinguish polymorphic VT with a normal QT interval from torsade as the treatment and prognosis may be different.

The QT interval is measured from the onset of the Q wave to the end of the T wave [18]. The QT interval can vary with
heart rate. Bradycardia is often associated with a prolonged QT interval while tachycardia is associated with a shortened QT interval. The QT interval can be corrected (QTc) for heart rate using Bazett’s formula (the QTc equals the longest QT interval divided by the square root of the preceding R-R interval [19]). If atrial fibrillation is present, the QTc should be measured for 10 consecutive beats and averaged. Correct assessment of the QTc is critical during initiation of sotalol in patients with atrial fibrillation.

Torsade may devolve into ventricular fibrillation, return to the baseline rhythm, or end with asystole. Therefore, the first line of therapy is usually defibrillation followed by intravenous magnesium sulfate. Once the patient has hemodynamics that allow perfusion of vital organs, the goal is identifying the underlying cause. Common causes include extreme bradycardia, congenital causes of long QT syndrome, anti-arrhythmic drugs, and one or more combinations of drugs that prolong the QT interval. Both drug overdose and reduced drug clearance can prolong the QTc sufficiently to cause torsade. In a retrospective study of 249 cases of torsade not attributed to cardiac drugs, Zeltser and colleagues [20] noted that 71% of the cases involved female patients. Other risk factors in this series included hypokalemia, the use of multiple drugs that prolong the QT interval, increased drug dosage, a history of prior torsades, and a family history of long QT syndrome. Among cardiac medications, the class IA and class III anti-arrhythmics are associated with the development of torsade. The class IA drugs are most well known for blocking sodium channels. However, at low serum drug concentrations, potassium ion current blockage occurs. The association of quinidine is well described in the literature. Disopyramide has also been implicated. N-acetylcyanamidine, a metabolite of procainamide, can cause torsade via QT prolongation by blocking the I_{Kr} channel. Class III anti-arrhythmics are potent I_{Kr} channel-blockers. High serum concentrations of these drugs, either due to overdose or decreased clearance, can result in torsade. As these drugs exhibit reverse use dependence, I_{Kr} is more effectively blocked at slow heart rates. Thus, bradycardia increases the risk of torsade with class III agents [21–23]. Interestingly, the class III agent amiodarone is rarely associated with torsade [24]. Drouin and colleagues [25] demonstrated that amiodarone decreases heterogeneous repolarization, thus reducing the susceptibility of re-entry. Torsade is also associated with overdose of tricyclic anti-depressants [26] and with use of the neuroleptics, including phenothiazines and haloperidol [27,28]. Among antimicrobials, the macrolides erythromycin and clarithromycin have been reported to prolong the QT interval and cause torsade [29,30]. Both of these medications inhibit the CYP3A4 system. Therefore, QT prolongation may occur in a patient taking either of these antibiotics with another drug that is metabolized by the CYP3A4 system. Such drugs will cause QT prolongation with increasing serum concentrations. The incidence of torsade among patients taking azithromycin is substantially less than that of patients taking erythromycin [31]. An interesting historical footnote is cisapride, a promotility drug withdrawn from the U.S. market because it has a high incidence of QT prolongation and arrhythmia. Cisapride blocks the I_{Kr} channel. Finally, in the case of bradycardia with long QT, temporary pacing may be necessary to prevent recurrence until the etiology of the slow heart rate can be diagnosed and treated.

**Acute anterior myocardial infarction and right bundle branch block:** The formal criteria for right bundle branch block (RBBB) are as follows: (1) the QRS duration must be ≥120 ms; (2) an rSR’ pattern must be present in lead v1 or v2; (3) the S wave in V6 and I must be longer than 40 ms or at least longer than the R wave duration; and (4) the time to the peak of the R wave must be ≥50 ms in v1, but within normal limits in v5–6 [32]. With RBBB, the secondary (R’) deflection is typically of greater amplitude than the first (R) deflection. Furthermore, there may be associated T wave inversion. Sometimes downsloping ST segments are observed. The presence of RBBB does not prevent the diagnosis of anterior MI. ST segment elevation and Q waves may be observed in V1–V4 precordial leads despite the presence of high amplitude R waves. The Q waves of an anterior infarct can obscure the initial R of a RBBB pattern, so that there is a qR in V4, rather than an RSR’.

The incidence of RBBB in the population has been reported to be approximately 1.8/1000 people [33]. In patients with isolated RBBB and a structurally normal heart, the conduction delay does not portend a worse prognosis. However, there are many pathologic conditions that may cause RBBB including Ebstein’s anomaly, cor pulmonale, myocarditis, hypertensive heart disease, Lenegre’s disease, and Lev’s disease. RBBB may also occur in patients with repaired tetralogy of Fallot who are left with significant pulmonary valve insufficiency resulting in right ventricular volume overload [34]. The development of RBBB in the context of acute MI occurs more frequently than left bundle branch block (LBBB). This may be due to the fact that the right bundle is a smaller, more discrete structure relative to the left bundle.

Prior to the thrombolytic era, the development of RBBB in the context of acute MI was associated with increased mortality [35]. Among patients who received thrombolysis, Go and colleagues [36] reported that the presence of RBBB was associated with a 69% increase in the risk of in-hospital death compared with acute MI patients who did not have RBBB and ST segment elevation. Moreno and colleagues [37] reported data from 681 patients with acute MI (74% had RBBB). Those with new irreversible RBBB had a 1-year mortality of 73%. The mortality of acute MI patients has decreased with advances in medical and mechanical therapy. More recently, Wong and colleagues [38] reported a 30-day mortality of 27.2% in patients with RBBB and a QRS duration <160 ms. If the QRS duration was ≥160 ms, then the
30-day mortality was 37.2%. If new RBBB developed within 60 minutes of treatment, then the mortality was 24.5% if the QRS duration was less than 160 ms and 46.2% if the QRS duration was ≥ 160 ms. The development of a new bundle branch block in the context of an acute MI is generally a consequence of a large area of necrosis. If a new bundle branch block is a surrogate for a large MI, this explains, in part, the worse prognosis with the development of a new RBBB in the context of an acute MI. By the same logic, a longer QRS duration implies more myocardium is involved in the infarction. Nonetheless, the mortality of acute MI patients continues to decrease with early ECG recognition and treatment of STEMI.

Complete heart block: Complete heart block, also called third-degree heart block, describes a condition in which atrial depolarization is not conducted to the ventricles. Without action potential propagation from the atria through the atrioventricular (AV) node via the conduction system to the ventricles, the ventricular rate falls to that of automatic pacemakers located in the ventricular tissue. The ventricular rate will be in the range of 20 to 40 bpm or sometimes slower. The ventricular escape rate depends on the location of the ectopic escape pacemaker site. The atrial rate will generally be that of the sinus node (i.e., faster than the ventricular rate). As such, the atria and the ventricles are asynchronous. This phenomenon is referred to as AV dissociation. In patients with complete heart block, AV dissociation can be observed on the surface ECG as independent P waves that are not associated to QRS complexes in the usual 1 : 1 relationship. Furthermore, the atrial rate is faster than the ventricular rate. Therefore, the P waves are described as “marching though” the ECG with no fixed relationship to the QRS complexes [39]. Not all patients with AV dissociation have complete heart block. For example, sometimes in ventricular tachycardia P waves can be observed that are slower than the ventricular rate.

The etiology of complete heart block can be considered as primary or secondary. Primary complete heart block is due to pathology intrinsic to the conduction system. Secondary heart block is due to primary pathology outside the conduction system that affects the conduction system. Examples of secondary cases include permanent electronic pacemaker malfunction, neurologic causes, metabolic etiology, and medication toxicity due to overdose or reduced clearance. Iatrogenic complete heart block is a rare complication of aortic valve replacement surgery or atrioventricular nodal re-entry tachycardia catheter ablation. For this reason, temporary epicardial pacing leads are placed in patients undergoing cardiac surgery and transvenous pacing leads are paced in patients undergoing certain catheter ablation procedures in the electrophysiology lab.

Myocardial infarction is an important etiology of complete heart block. The infarct-related artery responsible for inferior infarction is often the right coronary artery (RCA). The AV nodal branch artery is often derived from the RCA, therefore inferior infarctions may cause heart block at the level of the AV node. As such, the escape rhythm will often originate directly below the AV node, from the His bundle. This mechanism generally provides a narrow QRS complex rhythm with a rate of at least 40 bpm. Another etiology of complete heart block with inferior MI is vasovagal reaction. Both are effectively treated with atropine [40]. Heart block secondary to inferior MI is usually transient and generally does not require permanent pacemaker placement unless the heart block persists. In contrast, if complete heart block occurs in the context of an anterior MI, the infarct zone is usually very large. The mechanism of complete heart block with an anterior MI is infarction of the infra-nodal conduction system, therefore the escape rhythm is wide, complex, and slow, typically less than 40 bpm. In the past, when a patient presented sufficiently early in the course of an evolving MI, progressive degrees of AV block were observed prior to complete heart block. As such, the American Heart Association/American College of Cardiology have given temporary pacemaker placement a class I indication for anterior MI patients with progressive AV block. Today, with early reperfusion via primary PCI or fibrinolytic agents, this complication is rarely seen. However, given the risk to the patient, it is critical to recognize progressive heart block in the context of an anterior MI early in the course of treatment so that a temporary pacemaker can be placed before a patient’s life depends on emergency pacing [41].

**Case conclusions**

**Case 1** represents a common presentation of severe hyperkalemia. The history suggests a patient with chronic kidney disease. Patients with chronic kidney disease are unable to efficiently excrete excess potassium ion in the urine. It is critical to make the diagnosis of hyperkalemia from an ECG, particularly in the setting of a sinusoidal QRS complex, in that treatment needs to be initiated immediately.

**Case 2** presents a common scenario of tricyclic anti-depressant overdose. The patient presented with anti-cholinergic symptoms. Intravenous sodium bicarbonate is the antidote for this type of ingestion with a widened QRS complex – this patient received multiple doses of sodium bicarbonate coupled with endotracheal intubation and other critical management supportive care. Occasionally, the medication or medications upon which the patient overdosed are known. When the drugs are not known, the health care provider must rely on the history and examination for clues. The patient must be stabilized and monitored closely while the laboratory is performing a toxicology or overdose panel. The baseline ECG may provide clues regarding the drugs on which the patient overdosed, and for what acute or sub-acute adverse effects the patient may be at risk.
Case 3 is a classic presentation of an acute MI. This patient was experiencing a large anterior STEMI with RBBB; the patient underwent PCI with stenting of the left anterior descending artery with good outcome. It is critical to recognize the presence of an anterior MI in the presence of RBBB. The presence of RBBB should not distract the health care provider from the diagnosis of MI – as is the case in patients with LBBB presentations. Such infarctions are typically larger and patients have the potential to receive significant benefit from early recognition and treatment.

Case 4 represents a common situation in which complete heart block may occur. Regardless of the clinical context it is important to recognize complete heart block and immediately initiate treatment to stabilize the patient. In this case, the underlying etiology was the inferior MI. If the underlying etiology was known, once the patient has been stabilized, the patient should be admitted and considered for a permanent pacemaker.

References


Chapter 2 | What are the indications for the ECG in the pediatric emergency department?

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Case presentations

Case 1: A 14-year-old female presents to the emergency department (ED) with a chief complaint of chest pain for 1 hour. The pain is characterized as crushing in nature without radiation. There are no other associated symptoms. This is the third such episode since joining the soccer team. She does not take any medications. There is no family history of sudden, unexplained death or congenital heart disease. On examination, her heart rate is 90 beats per minute (bpm), blood pressure is 110/68 mmHg, and oxygen saturation is 95% on room air. Her physical examination is unremarkable and you are unable to reproduce her pain. Her electrocardiogram (ECG) is shown in Figure 2.1.

Case 2: A 10-year-old male presents to the ED after a syncopal episode while running at school. The patient has no memory of the event. Witnesses deny any seizure-type activity. He has no significant past medical history and no habitual medication use. His family history is significant for a distant relative who died suddenly of unknown causes. On examination his heart rate is 85 bpm, blood pressure is 95/53 mmHg, and oxygen saturation is 100% on room air. His physical exam is unremarkable. An ECG is performed, shown in Figure 2.2.

Case 3: A 4-year-old male presents to the ED with a complaint of episodic chest pain that started while watching television. According to the patient’s mother, the pain starts and stops abruptly and lasts between 1 and 5 min. The child has not had syncope. His mother reports six of these episodes this morning and denies any such episodes prior to today. He has no significant past medical history, takes no habitual medications, and has a non-contributory family history. On examination, his heart rate suddenly increases to > 250 bpm, his ECG is shown in Figure 2.3. You have the child perform a valsalva maneuver and his heart rate decreases to 120 bpm and the repeat ECG is demonstrated in Figure 2.4.

Clinical indication for the ECG in pediatric emergency and critical care medicine

Clinical indications for the electrocardiogram (ECG) are well documented for adult patients [1,2]; however, studies evaluating its efficacy in the pediatric population are lacking. A systematic approach to ECG interpretation in the pediatric population is required to minimize the occurrence of misdiagnosis. Familiarity with the indication for and interpretation of the pediatric ECG is critical to the clinician managing children with acute illness and injury, as misdiagnosis may lead to mismanagement [3–5]. Based on the literature, ECGs should be considered in the pediatric emergency department in the following scenarios: chest pain with a supportive history, palpitations with a supportive history, most instances of syncope, most cases of apparent life-threatening event (ALTE) or near-miss sudden infant death syndrome (SIDS), ingestions, suspected metabolic derangements, and patients with an episode of commotio cordis; of course, other presentations are appropriate and ECGs may also be performed in these other settings.

Chest pain: Unlike the adult population, fewer than 5% of cases of chest pain in children and adolescents are of cardiac etiology [7–11]. Chest pain in this population, however, has been shown to account for nearly 20% of all new pediatric cardiology consults in the ED [11]. It commonly accounts for more than 50% of all ECGs obtained in pediatric EDs [5], yet accounts for less than 1% of all visits to the pediatric ED [7, 9, 10], and often does not demonstrate any changes even when cardiac pathology is present [10,12,13]. Costochondritis, asthma, gastroesophageal reflux, trauma, and anxiety constitute the majority of presentations for chest pain in this population [7–11,14].

When evaluating chest pain in a pediatric ED, the clinician should consider myocardial ischemia or infarction; refer to
Figure 2.1 This ECG demonstrates upright P waves in leads I, aVF, and V3 to V6, consistent with a sinus rhythm. Her rate of 85 bpm is appropriate for her age. The predominant QRS voltages are upright in leads I and aVF, consistent with a leftward QRS axis that is normal for her age. There is no evidence for ventricular hypertrophy by voltage criteria, no ST segment or T wave abnormalities and no evidence of pre-excitation or “delta waves.” The QT interval is less than one-half of the R to R interval, best seen in leads II and aVF, which is typically consistent with a normal corrected QT interval. This is a normal ECG for this girl who was found to have exercise-induced asthma.

Figure 2.2 As in Figure 2.1, there is normal sinus rhythm and an appropriate heart rate and leftward QRS axis. There are inverted T waves in the early precordial leads (V1 to V3), which is normal into late adolescence. There is no ST segment elevation or depression or pre-excitation. The QT interval is at least one-half the R to R interval, which is typically consistent with long QT syndrome, this patient’s diagnosis. The QTc is calculated by dividing the QT interval in leads II or V5 by the square root of the preceding R to R interval; normal values are less than 460 ms.
Table 2.1 for a listing of possible causes of myocardial ischemia. The differential diagnosis for myocardial ischemia is more diverse in the pediatric patient, as obstruction of a coronary artery lumen by an atherosclerotic plaque is extremely rare. Severe left ventricular hypertrophy (i.e., aortic stenosis, coarctation of the aorta, or hypertrophic cardiomyopathy) can exhibit large QRS voltages and T wave inversions in the lateral precordial leads with or without ST segment changes. “Kinking” of transplanted coronary arteries can produce ST segment changes consistent with ischemia.

Figure 2.3 The most striking aspect of this ECG is the heart rate of nearly 300 bpm, which is never physiologic. This is a narrow complex tachycardia without clear evidence of P waves.

Figure 2.4 This follow-up ECG of the same patient as in Figure 2.3 demonstrates a normal sinus rhythm with an appropriate heart rate and QRS axis. The most notable aspect of this ECG is the pre-excitation or delta waves seen in leads II, III, aVF, and V2 through V4. This patient was diagnosed as having Wolff-Parkinson-White syndrome.
with psychogenic manifestations in children origin and several authors have found chest pain associated thrombosis of coronary arteries. Lastly, ST segment changes years after the diagnosis of Kawasaki’s disease from the of the affected coronary artery. This pattern can also occur years after the diagnosis of Kawasaki’s disease from the thrombosis of coronary arteries. Lastly, ST segment changes may be seen in the setting of cocaine overdose.

As noted, most chest pain in children is not cardiac in origin and several authors have found chest pain associated with psychogenic manifestations in children > 12 years and respiratory diseases in those < 12 years [7,8,10]. Wiens and colleagues were able to reproduce chest pain with an exercise stress test and alleviate symptoms with albuterol in 64 of 88 children with a presenting complaint of chest pain [14]. A history of chest pain with palpitations, syncope, progressive exercise intolerance, prior Kawasaki’s disease, or a positive family history of sudden death or defibrillator implantation should raise suspicion for cardiac pathology. However, information regarding chest pain duration, location, and frequency has limited diagnostic utility in the pediatric population [7,8,10]. A focused physical examination should confirm their original suspicions. Evidence of wheezing, decreased breath sounds, stridor, or chest wall tenderness during the examination suggest non-cardiac chest pain. [8,9].

In 168 consecutive patients evaluated in an ED for chest pain, Massin and colleagues reported a friction rub, murmur, pallor, abnormal heart rhythm, or signs of decreased cardiac output in all seven patients ultimately diagnosed with a cardiac etiology [10]. Although 83 ECGs were ordered during this retrospective study, the ECG was abnormal in only seven patients.

Chest pain is a common problem in the pediatric population, which is only rarely associated with myocardial ischemia. Considering the variability in ECG interpretation, its use should be limited to those patients with both a positive history and physical exam. Any patient producing a high index of suspicion, as well as ECG evidence for myocardial ischemia, should undergo prompt evaluation by a pediatric cardiologist.

### Table 2.1a Possible causes of myocardial ischemia in the pediatric patient

<table>
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<tr>
<th>Possible causes of myocardial ischemia in the pediatric patient</th>
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<td>Congenital heart diseases</td>
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<td>Anomalous left coronary artery arising from the pulmonary artery (ALCAPA)</td>
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<td>Coarctation of the aorta</td>
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<td>Aortic stenosis</td>
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<td>Hypertrophic cardiomyopathy</td>
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<td>Congenital heart disease – postoperative</td>
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<td>Transposition of the great arteries status postarterial switch operation</td>
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<tr>
<td>Single ventricle physiology (i.e., hypoplastic left heart syndrome)</td>
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<tr>
<td>Aortic stenosis status post-balloon valvuloplasty</td>
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<tr>
<td>Acquired heart disease</td>
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<td>History of Kawasaki’s disease with or without echocardiographic evidence of coronary artery involvement</td>
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<tr>
<td>Other</td>
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<td>Cocaine abuse</td>
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<td>Pulmonary hypertension</td>
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### Table 2.1b Possible causes of myocardial ischemia in the pediatric patient by age

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<tr>
<th>Possible causes of myocardial ischemia in the pediatric patient by age</th>
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<td>0–6 months</td>
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<td>Anomalous left coronary artery arising from the pulmonary artery (ALCAPA)</td>
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<td>Coarctation of the aorta prior to intervention</td>
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<td>Aortic stenosis</td>
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<td>Cyanotic congenital heart disease – preoperative</td>
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<td>Cyanotic congenital heart disease – postoperative</td>
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<td>Transposition of the great arteries status postarterial switch operation</td>
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<td>Aortic stenosis status post-balloon valvuloplasty</td>
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<tr>
<td>Greater than 5 years old</td>
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<td>History of Kawasaki’s disease with or without echocardiographic evidence of coronary artery involvement</td>
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<td>Cocaine abuse</td>
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Syncope and presyncope: Syncope is a transient, self-limited loss of consciousness caused by global cerebral hypoperfusion; presyncope is the feeling that syncope is imminent. As with chest pain, syncope is common in the pediatric population, occurring in up to 15% of adolescents [12,15,16], and is also rarely cardiac in origin. However, with subsequent annual mortality rates of 24% in those with cardiac syncope [15], a thorough evaluation of this presenting symptom is justified.

Any syncopal episode in the setting of known cardiac disease should be considered an aborted sudden death until proven otherwise. Ventricular tachycardia, ventricular fibrillation, or torsade de pointes are the three dysrhythmias most commonly cited as the cause of cardiac syncope, but are only rarely documented in patients who survive to receive emergency medical care. Severe pulmonary hypertension, long QT syndrome, hypertrophic cardiomyopathy, and Wolff-Parkinson-White syndrome are the most likely substrates leading to these ventricular dysrhythmias. In order to properly recognize these rare disease states, the clinician must familiarize him/herself with the most common findings on history, physical exam, and ECG.

A history of dehydration, dizziness, fatigue, vertigo, medication use, or intentional termination of activities prior to the loss of consciousness all suggest a vasovagal etiology. Conversely, syncope during exercise or exertion, or when immediately preceded by palpitations or chest pain is consistent with dysrhythmia. It is generally agreed that all patients with an episode of exercise-induced syncope should be evaluated by a pediatric cardiologist [13,17]. To assess the risk for long QT syndrome and hypertrophic cardiomyopathy (HCM), the clinician should explore any family history of syncope, sudden unexplained deaths particularly with exercise, and defibrillator and, pacemaker placements [18].
A cardiac examination will often be normal in patients with cardiogenic syncope. Two notable exceptions can occur in HCM with left ventricular outflow tract obstruction and severe pulmonary hypertension. In general, however, patients with long QT syndrome, pulmonary hypertension, Wolff-Parkinson-White syndrome, or HCM without left ventricular outflow tract obstruction will have unremarkable physical examination. This fact underscores the importance of a thorough medical and family history, as well as appropriate ordering and interpretation of the ECG. Most pediatric patients referred for a syncopal episode should undergo an ECG. A normal ECG, however, does not rule out a cardiac cause as studies have demonstrated significant variability in ECG interpretation and the common finding of normal QTc intervals on resting ECGs of individuals with long QT syndrome [13].

ECG interpretation in this setting must focus on identifying findings suggestive of HCM, long QT syndrome, Wolff-Parkinson-White syndrome, or pulmonary hypertension with right ventricular hypertrophy. Patients with HCM may demonstrate large R wave voltages in leads I, II, III, aVF, and V3 through V6, consistent with left ventricular hypertrophy; ischemia or left ventricular “strain” may also be seen as ST segment changes or T wave inversion in the lateral precordial leads. Though often normal at rest, long QT syndrome is diagnosed as having a corrected QT interval of greater than 460 ms.

Syncope in patients found to have Wolff–Parkinson–White syndrome is thought to occur due to rapid conduction of atrial fibrillation across the accessory pathway with resultant ventricular fibrillation. The incidence of spontaneous atrial fibrillation is thought to be higher in these patients as compared with the general population, but the incidence of sudden cardiac death is estimated to occur in only 1 of 10,000 patients [19,20]. A short PR interval, along with evidence of pre-excitation, is required to make the diagnosis of this conduction anomaly [21] and should prompt consultation with a pediatric cardiologist.

Severe pulmonary hypertension can result in syncope through two possible mechanisms. In one scenario, right ventricular strain against an acutely elevated pulmonary artery pressure could result in myocardial ischemia and ventricular dysrhythmias. Alternatively, acute right ventricular failure in the setting of elevated pulmonary artery pressures would impede left ventricular preload and systemic cardiac output, ultimately producing cerebral hypoperfusion. Regardless of the mechanism, severe right ventricular hypertrophy can produce large R waves in the early precordial leads (V1 to V3) in addition to a rightward deviation of the QRS axis. T wave inversion in these leads may also be seen, but this finding is normal into adolescence. While these findings could be considered strong evidence in support of the respective diagnoses, their absence does not exclude them. For example, the pre-excitation or “delta wave” seen in Wolff-Parkinson-White syndrome can be intermittently expressed [20]. The corrected QT interval is commonly normal in patients with documented abnormalities in the identified electrolyte channels, the pathologic hallmark of long QT syndrome. Thus, as a screening test for cardiac causes of syncope, the surface ECG is a relatively specific test with poor sensitivity. The clinician should recognize this limitation, and therefore focus attention on the history and physical examination to screen for those patients at most risk for having a cardiac etiology for their syncope.

**Palpitations and supraventricular dysrhythmia:**

Palpitations are the perception of an abnormal heart rate or rhythm. Assessment by both 24-h Holter monitors and event monitors frequently demonstrates this sensation to be associated with premature atrial and ventricular contractions, as well as sinus tachycardia. Sustained periods of abnormal cardiac conduction are relatively uncommon. Primary ventricular dysrhythmias (i.e., ventricular tachycardia) are rare in pediatric patients without congenital or acquired heart disease, and are typically associated with syncope or chest pain. Simple palpitations with a true cardiac etiology are most commonly associated with ventricular electrical conduction via the His-Purkinje fibers and effective ventricular contractions. This scenario occurs in the setting of either normal sinus tachycardia or supraventricular tachycardia. Supraventricular tachycardia is the most common cause of palpitations from a primary cardiac dysrythmia in pediatric patients [21]; fortunately, less than 1 in 10,000 [20] of such patients are estimated to be at risk for degenerating into a lethal ventricular dysrhythmia.

There are 11 different types of supraventricular tachycardia. These conduction disturbances manifest at a rate faster than that of the sinoatrial node, and are intrinsically unresponsive to the needs of the body. Patients subjected to the persistent state of any one of these dysrhythmias can develop a dilated cardiomyopathy over time; nearly 50% of infants will exhibit signs of congestive heart failure in as little as 48 h of a continuous non-sinus rhythm [22]. While important to make a timely diagnosis, the incidence of sudden cardiac death associated with true supraventricular tachycardia is exceedingly rare and has only been associated with one type: Wolff-Parkinson-White syndrome (WPW).

The electrocardiographic findings necessary to make the diagnosis of Wolff-Parkinson-White syndrome include a PR interval that is shortened due to aberrant prograde conduction over an accessory pathway [21]. This finding is not present during the typical tachycardia cycle, as conduction is prograde down the atrioventricular (AV) node, into the His-Purkinje fibers, and retrograde up the accessory pathway back into the atria – perpetuating the cycle until some part of the circuit is made refractory to further conduction. Most commonly, the AV node is the focus of interventions through either vagal maneuvers or adenosine, which both slow conduction through this structure and terminate the circuit. Unlike in the other 10 types of supraventricular
tachycardia, the accessory pathway in Wolff-Parkinson-White syndrome has the potential to allow rapid prograde conduction into the ventricles. In the setting of atrial fibrillation with rates up to 300 bpm, this will quickly degenerate into an unstable ventricular rhythm.

Paul and colleagues [19] published a landmark study about patients with Wolff-Parkinson-White syndrome, linking a predisposition to atrial fibrillation with syncope. Of 74 adolescents with this diagnosis who underwent formal electrophysiology testing, 14 presented with a history of syncope. This subset demonstrated a significantly increased ability to be induced into atrial fibrillation as compared with the other 60 patients who did not present with a history of syncope. Prograde conduction down the accessory pathway in patients with Wolff-Parkinson-White syndrome places them at increased risk for sudden cardiac death.

Psychiatric history should be thoroughly addressed, as studies have demonstrated non-cardiac palpitations to be present in nearly half of all patients suffering from anxiety disorders [23]. As physical examination is unlikely to guide the diagnosis, the clinician must rely on ECG interpretation to detect underlying processes. After determining whether the absolute heart rate is appropriate for the particular patient, the clinician must focus attention on the cardiac rhythm. Sinus rhythm is a heart rate that is being controlled by the rightward, superior, and posteriorly located sinoatrial node. This anatomic position can be verified by the P wave axis. When originating from the sinoatrial node, there should exist upright P waves in leads I, II, III, aVF, and the precordial leads V3 through V6. Any variation from this pattern implies that another region of the atria is controlling the heart rate. Once the rhythm is determined, attention should be given to the presence of pre-excitation. This will produce a gradual upstroke that terminates into the rapid upstroke of the QRS complex, signifying the less efficient conduction through the accessory pathway as compared with the His-Purkinje fibers. In patients with Wolff-Parkinson-White syndrome this pattern should be seen in more than one lead, but typically will not be seen in all leads.

Apparent life-threatening event (ALTE) and near-miss sudden infant death syndrome (SIDS): Formerly known as “crib death,” SIDS is a devastating loss of life that classically occurs in the first 5–12 weeks [24] in an estimated 7 out of 10,000 live births [25]. Several authors have suggested a correlation between SIDS and the long QT syndrome by citing that 14% of all patients with long QT syndrome will die with their sentinel event, of which 30% occur in the first year of life [25].

Schwartz and colleagues [25] prospectively followed more than 33,000 infants who underwent a resting ECG on day three or four of life. A QT interval was measured in lead II and corrected for heart rate (QTc). The mean QTc was 400 ms and the 97.5th percentile was 440 ms. At 1-year follow-up, the overall risk of non-traumatic death was 1.53% in those infants with a QTc greater than 440 ms, compared with 0.037% in those with a QTc < 440 ms. None of the infants with the longer QTc had a positive family history for long QT syndrome. An ECG should be considered as a portion of the initial evaluation of any patient with a suspected ALTE or near-miss SIDS [26].

Brugada syndrome is another electrocardiographic diagnosis that has been suggested to have a link to sudden cardiac death in children. The findings of a right bundle branch block (RBBB) pattern and ST segment elevation in precordial leads V1 to V3 are necessary for this diagnosis, which occurs most commonly in Asian and South American males, with an overall estimated prevalence of 1 in 5000 [27]. Priori and colleagues [28] reported a family in which four children died between 2 and 36 months of age. Though less likely in the United States, this diagnosis should also be considered in the setting of ALTE.

Commotio cordis: Commotio cordis is the onset of cardiovascular collapse immediately following a blunt, non-penetrating blow to the chest, in the absence of underlying cardiovascular abnormalities. In patients who have survived long enough to receive prompt medical attention, the three most commonly documented rhythms are ventricular tachycardia, ventricular fibrillation and asystole. Animal studies have suggested the mechanism is similar to the R-on-T phenomenon that destabilizes patients with long QT syndrome into unstable ventricular dysrhythmias [28]. In the largest retrospective study on the subject, 84% of the 128 patients identified over a 16-year period died as a consequence of their event; more than 75% of these victims received appropriate resuscitative measures in under 3 min [29]. Despite these results, death is not universal if the problem is identified quickly [30]. Therefore, any pediatric patient presenting to the ED following an episode of significant chest trauma should have an ECG to confirm a normal sinus rhythm.

Case conclusions

Case 1, a normal ECG in a child with non-cardiac chest pain, was discharged from the ED without consequence. Case 2 illustrates a potential long QT syndrome presentation; this child was admitted for further monitoring. The child in Case 3 presented with symptomatic paroxysmal supraventricular tachycardia. With resolution of the tachycardia, a 12-lead ECG in sinus rhythm demonstrated Wolff-Parkinson-White syndrome. Due to this finding, he was admitted for further therapy and monitoring. No recurrence was found and he was ultimately discharged without therapy.

References

1 Antman EM, Anbe DT, Armstrong PW, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial


