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

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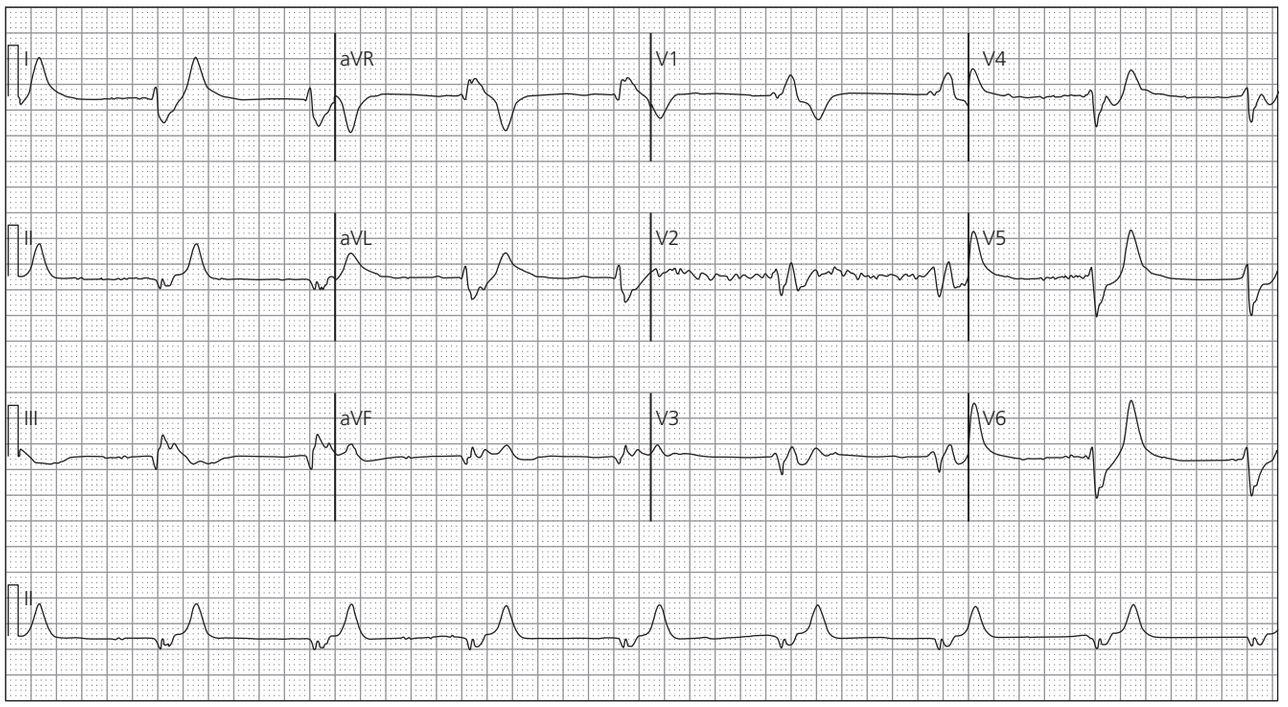
(a)



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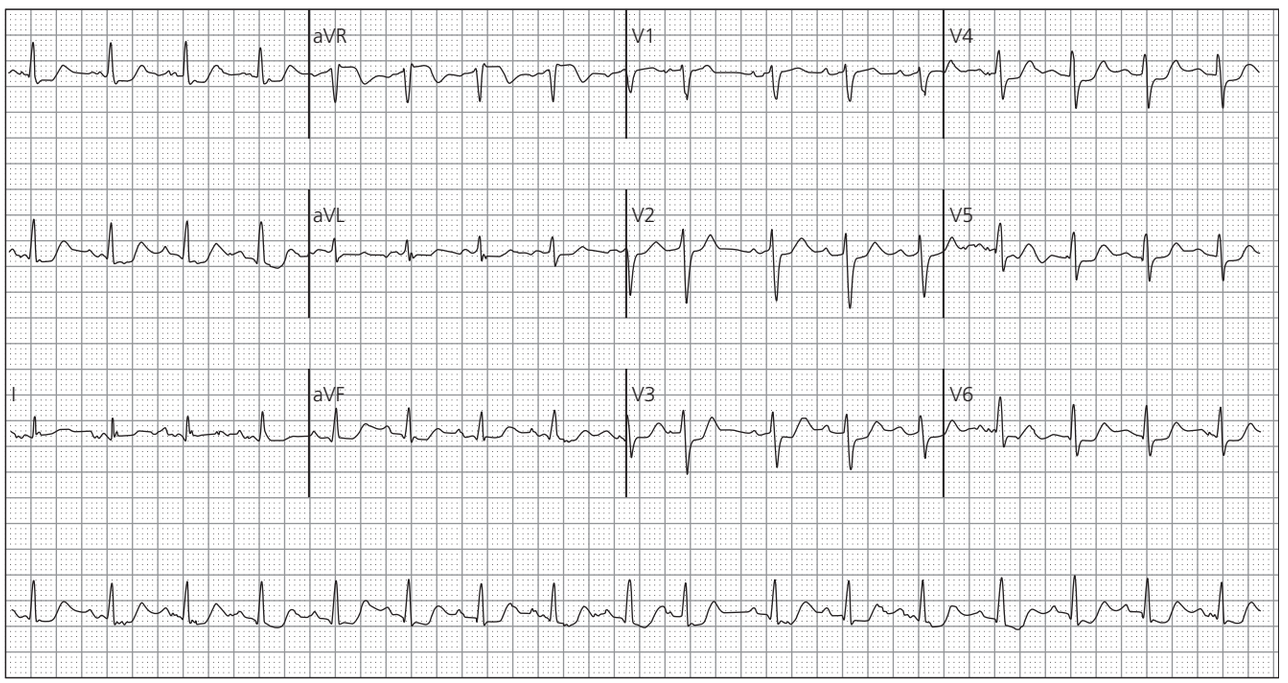

Figure 1.1 (a) Wide QRS complex bradycardia without P wave activity. (b) Improvement in the ECG from (a) with narrowing of the QRS complex, development of P waves, and increase in the rate. These ECGs are consistent with profound hyperkalemia that has markedly improved with therapy.

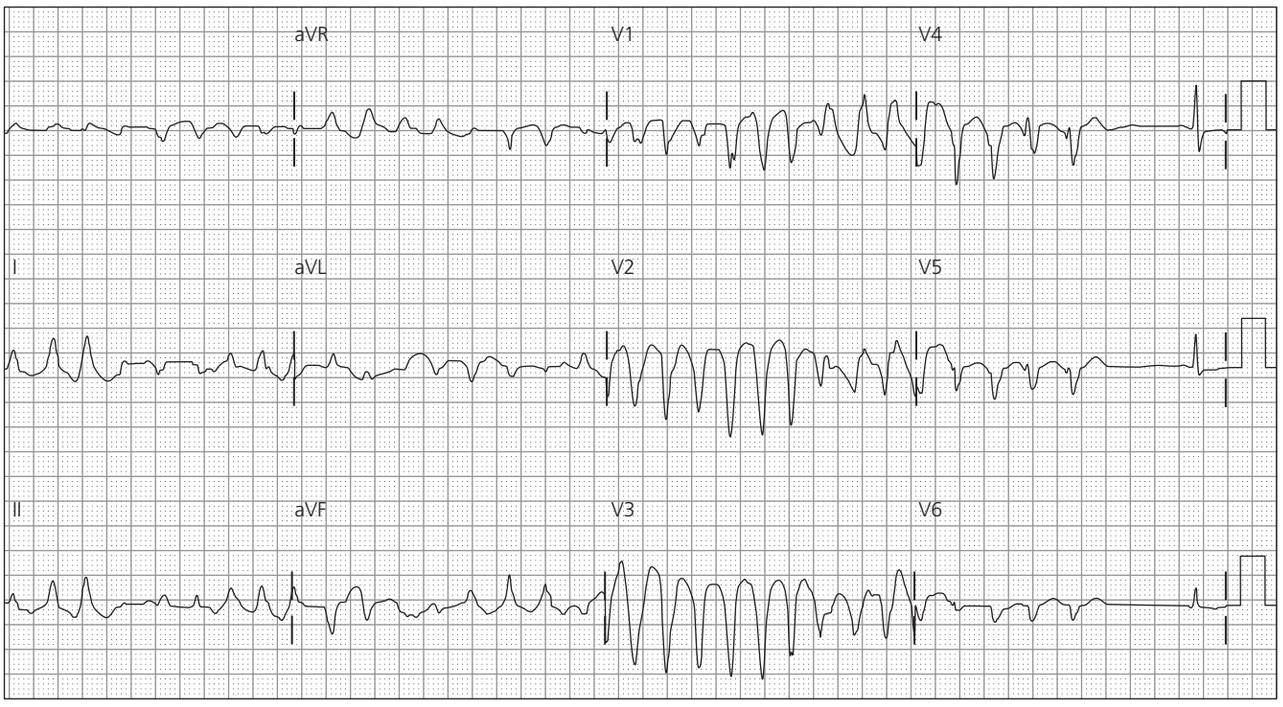

Figure 1.2 Polymorphic ventricular tachycardia (PVT). With review of the ECG in sinus rhythm, a prolonged QT interval was noted. With this additional finding, the PVT can be termed torsade de pointes. Note the varying QRS complex morphology (axis, amplitude, and contour) in a pattern suggesting "twisting about a fixed point."

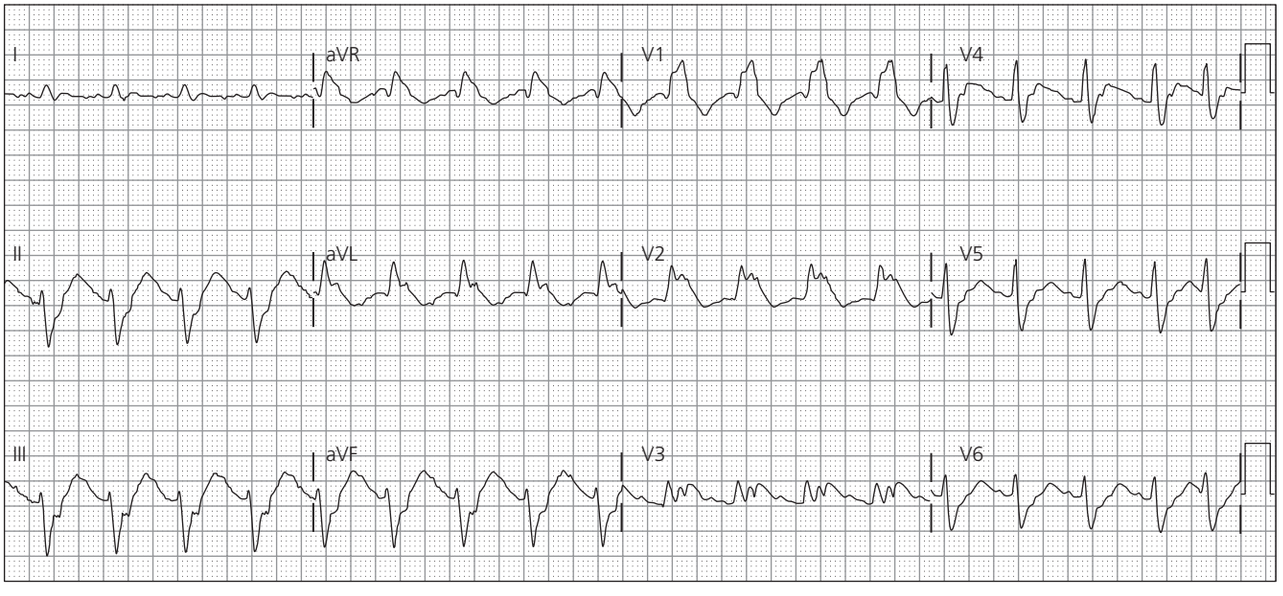

Figure 1.3 Sinus tachycardia with anterolateral wall STEMI, first-degree AV block, and right bundle branch block. Note the ST segment elevation in leads aVl, V2 and V3, consistent with anterolateral wall STEMI. The ST segment elevation in these leads is concordant with the major, terminal portion of the QRS complex – indicative of STEMI in RBBB.

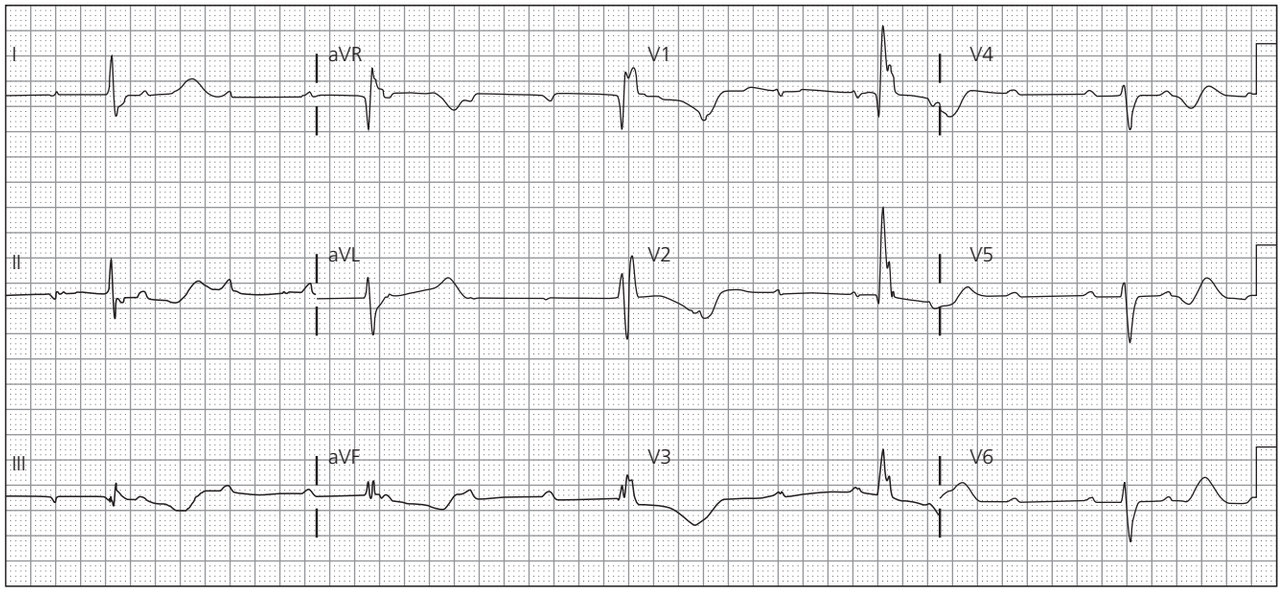

Figure 1.4 Complete heart block with no association of the atria with the ventricles. The P waves are occurring independently of the QRS complexes. Also note that atrial rate is greater than the ventricular rate.

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]. Paroxsymal 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.

Hypercalcemia: 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 hypercalcemia cases.

The presence of hypercalcemia and hypokalemia produces an interesting ECG. This can be seen with multiple myeloma. The hypercalcemia results in a short ST segment and the hypokalemia results in a prominent U wave. Tachyarrhythmias due to hypercalcemia are uncommon in the literature. However, bradyarrhythmias with hypercalcemia are well described [9]. The classic case of hypercalcemia 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 hypercalcemia. 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, rhabdomyositis, 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 hypokalemia. 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 hypernatremia 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-acteylprocainamide, a metabolite or procainamide, can cause torsade via QT prolongation by blocking the I_{kr} channel. Class III anti-arrthymics 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 neuroleptins, 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 V_1–V_3 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 V_1 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 is not known, once the patient has been stabilized, the patient should be admitted and considered for a permanent pacemaker.

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