Review

Electrocardiographic Interpretation and Abnormalities: A Comprehensive Review of Clinical, Technical, and Pathophysiological Insights

Tyler Hu *

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Electrocardiographic Interpretation and Abnormalities: A Comprehensive Review of Clinical, Technical, and Pathophysiological Insights

Tyler Hu

Independent Researcher; drbball935@gmail.com

Abstract: An electrocardiogram (ECG, or EKG) is a non-invasive recording of the heart's electrical activity, captured via electrodes on the skin [13]. Developed by Willem Einthoven in 1902, who later earned the 1924 Nobel Prize in Medicine, the ECG has become a fundamental tool in cardiac diagnostics. Physiologically, each heartbeat is driven by the coordinated depolarization and repolarization of cardiac muscle cells. The cardiac conduction system (including the sinoatrial node, atrioventricular node, bundle branches, and Purkinje fibers) orchestrates the orderly spread of electrical impulses through the atria and ventricles. The ECG tracings on paper or monitor represent the summation of these electrical currents over time, providing insight into heart rhythm, rate, and the integrity of myocardial tissue. Clinically, ECGs are prevalent in evaluating chest pain, palpitations, syncope, and numerous other cardiovascular presentations, and they are routinely used to screen for heart disease and monitor cardiac therapies [13]. This paper provides an overview of ECG basics, interpretation of normal waveforms and intervals, and an in-depth review of various ECG abnormalities. We will discuss arrhythmias, conduction blocks, ischemic changes, electrolyte disturbances, and structural heart disease indications on ECG, describing their characteristic ECG features, clinical implications, pathophysiology, and common causes.

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ECG Recording and Normal Waveforms

To record a standard 12-lead ECG, electrodes are placed at specific locations on the limbs and chest to measure electrical potential differences along multiple vectors around the heart. The 12 leads consist of six limb leads (I, II, III, aVR, aVL, aVF) and six chest (precordial) leads (V_1–V_6). Each lead "views" the heart's electrical activity from a different angle, allowing localization of electrical events and any regional abnormalities. For example, leads II, III, and aVF reflect the inferior cardiac surface (typically supplied by the right coronary artery), whereas leads V_1–V_4 look at the anterior wall (left anterior descending artery territory), and leads I, aVL, V_5–V_6 view the lateral wall (circumflex artery) [13]. By convention, ECG paper runs at 25 mm/s, so 1 small box = 0.04 s and 1 large box (5 mm) = 0.2 s; vertically, 1 mV = 10 mm. Interpreting an ECG requires systematic assessment of the heart rate, rhythm, axis, waveforms, intervals, and segments for any deviations from normal.

Normal ECG Waves and Intervals

In a normal sinus rhythm, the first deflection is the P wave, corresponding to atrial depolarization. It is typically upright in leads I and II and ≤2.5 mm in amplitude and ~0.08–0.10 s in duration (about 2–3 small boxes) [13]. A normal P wave indicates the electrical impulse originates from the sinoatrial (SA) node. The P wave is followed by the PR interval, measured from the onset of the P wave to the start of the QRS complex. The PR interval reflects the time for the impulse to travel through the atria and atrioventricular (AV) node into the His–Purkinje system. In adults, the PR

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interval is normally 0.12–0.20 s; a prolonged PR (>0.20 s) suggests first-degree AV block [13]. After a brief isoelectric PR segment (when the impulse is traversing the AV node), the QRS complex represents ventricular depolarization. The QRS is normally narrow (≤0.10–0.12 s) because the His–Purkinje network rapidly distributes the impulse through the ventricles. It consists of a Q wave (initial downward deflection, often small or absent in many leads), an R wave (the first upward deflection), and an S wave (the subsequent downward deflection). Following ventricular depolarization, the ST segment is the plateau phase of ventricular repolarization (normally isoelectric), and the T wave represents ventricular repolarization. The T wave is normally concordant with the QRS direction (e.g., upright in leads where QRS is upright) and has a gentle asymmetrical shape. The QT interval, from QRS onset to the end of the T wave, reflects the total duration of ventricular depolarization and repolarization. A normal QTc (rate-corrected QT) is roughly 0.36–0.44 s; prolonged QT can predispose to dangerous arrhythmias (discussed later). A small U wave may occasionally follow the T wave; this is thought to represent final repolarization of the His–Purkinje network or papillary muscles. U waves are usually ≤1 mm if present and have the same polarity as the T wave. Prominent U waves are an abnormal finding often associated with hypokalemia [10]. Overall, a normal ECG shows each P wave followed by a QRS (indicating 1:1 AV conduction), a constant PR interval, narrow QRS complexes, and properly oriented ST segments and T waves.

ECG Abnormalities: Arrhythmias

An arrhythmia refers to any deviation from normal sinus rhythm—it may be an abnormal heart rate (too fast or slow) or an irregular rhythm arising from abnormal impulse formation or conduction. Arrhythmias are broadly categorized by their site of origin (supraventricular vs. ventricular) and character (tachyarrhythmias vs. bradyarrhythmias). Below we discuss two common clinically significant arrhythmias and their ECG features.

Atrial Fibrillation (AF): One of the most prevalent arrhythmias, atrial fibrillation is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation. On the ECG, AF classically shows an "irregularly irregular" ventricular rhythm with absence of distinct P waves [13]. Instead of discrete P waves, the baseline may exhibit fine fibrillatory waves or appear chaotic as multiple atrial depolarization wavelets continuously bombard the AV node. The QRS complexes in AF are usually narrow (since conduction through the ventricles is normal) but occur at unpredictable intervals due to the irregular AV nodal conduction of atrial impulses. AF may be slow or rapid; "AF with rapid ventricular response (RVR)" indicates the ventricular rate is high (often 120–160 bpm or more) due to many atrial impulses getting through the AV node. Clinically, atrial fibrillation can cause palpitations, dizziness, or even hemodynamic instability if the rate is very fast. The loss of coordinated atrial contraction also predisposes blood stasis in the atria (especially the left atrial appendage), leading to an elevated risk of thromboembolic stroke. Common causes of AF include long-standing hypertension, valvular heart disease (especially mitral valve stenosis), heart failure, hyperthyroidism, and acute triggers like binge alcohol ("holiday heart"). Management centers on rate or rhythm control and anticoagulation to prevent stroke. On an ECG, the hallmark findings are enough to make the diagnosis: irregularly spaced QRS complexes and no identifiable P waves [13].

Ventricular Tachycardia (VT): Ventricular tachycardia is a potentially life-threatening arrhythmia originating in the ventricles. It is defined as a run of three or more consecutive premature ventricular beats at a rate >100 per minute [13]. On ECG, VT typically produces wide QRS complexes (≥0.12 s) because the ventricular activation is occurring via an abnormal pathway (originating within the ventricular myocardium rather than the His–Purkinje system). VT can be monomorphic, with a uniform and stable QRS morphology, or polymorphic, with beat-to-beat variation in QRS shape. A special form of polymorphic VT is Torsades de Pointes ("twisting of the points"), often associated with prolonged QT interval and characterized by QRS complexes of changing amplitude and axis. Clinically, ventricular tachycardia often presents with palpitations, chest pain, dyspnea, lightheadedness, or syncope. Sustained VT (lasting >30 seconds or causing collapse) can degenerate into ventricular fibrillation and cardiac arrest, making prompt recognition critical. The most common

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underlying cause of VT is myocardial infarction (scar-related reentrant circuits), but other causes include cardiomyopathies, heart failure, electrolyte disturbances, and drug toxicity. On the ECG, a monomorphic VT classically shows a regular rhythm with wide QRS complexes at a rapid rate (often 150–200 bpm). There are no preceding P waves (or if present, they bear no fixed relationship to QRS—a phenomenon known as AV dissociation). Sometimes "capture" or "fusion" beats may be seen (occasional normal-narrow QRS amid VT) which support the diagnosis of VT over supraventricular tachycardia with aberrancy. Ventricular tachycardia is a "wide-complex tachycardia" by definition; any wide-QRS tachycardia in an ill patient should be considered VT until proven otherwise. Immediate treatment may involve intravenous antiarrhythmics or electrical cardioversion depending on stability. In summary, VT's ECG hallmarks are wide QRS complexes at a rapid rate, often with a consistent morphology (monomorphic) and AV dissociation. This arrhythmia signifies serious underlying heart disease in many cases and is responsible for a large proportion of sudden cardiac deaths [8].

(Other arrhythmias: There are many other arrhythmias detectable on ECG. For instance, atrial flutter presents with rapid "sawtooth" atrial waves (usually ~300/min) and a regular ventricular response at a fraction of the atrial rate (like 150 bpm if 2:1 conduction). Supraventricular tachycardias (AV nodal reentrant tachycardia, AV reentrant tachycardia in WPW syndrome, etc.) typically have narrow QRS and high rates with abrupt onset/termination. Ventricular fibrillation is a chaotic, irregular waveform with no discernible QRS complexes, reflecting cardiac arrest. Bradyarrhythmias such as sinus bradycardia or junctional rhythms show slow rates; sinus pauses or arrest can be seen as gaps in ECG activity, and so on. While these are beyond the scope of this paper, a skilled interpreter can diagnose each by characteristic ECG patterns.)*

Conduction Blocks

Conduction blocks occur when electrical impulses are delayed or interrupted as they propagate through the heart's conduction system. The two major categories are atrioventricular (AV) blocks (impaired conduction between atria and ventricles) and bundle branch blocks (impaired conduction in the left or right bundle branch within the ventricles).

Atrioventricular (AV) Blocks: AV blocks are classified into first, second, or third degree based on severity. First-degree AV block is simply prolongation of AV conduction—defined by a PR interval >0.20 s while every P still conducts to QRS [13]. It shows up on ECG as a consistently prolonged PR interval, but each P wave is followed by a QRS. Patients are usually asymptomatic in first-degree block; it can be seen in athletes or due to enhanced vagal tone, AV nodal blocking medications (beta blockers, calcium channel blockers, digoxin), or ischemia. Second-degree AV block means intermittent failure of AV conduction. In Mobitz Type I (Wenckebach) second-degree block, the ECG shows gradual prolongation of the PR interval on consecutive beats until a P wave is eventually not conducted (dropped QRS), after which the cycle repeats [13]. This usually indicates a block at the AV node and is often benign; it can occur in inferior myocardial infarction or with high vagal tone, and patients may be asymptomatic. Mobitz Type II second-degree block is characterized by sudden, unpredictable dropped QRS complexes without the progressive PR prolongation. For example, the ECG may show a pattern like 2:1 or 3:1 conduction (every second or third P wave fails to conduct). Mobitz II typically implies a block in the His–Purkinje system below the AV node. It is more serious, often progressing to complete heart block, and usually warrants pacing. Third-degree AV block (Complete Heart Block) is complete failure of all atrial impulses to reach the ventricles. On ECG, P waves and QRS complexes are present but totally independent of each other (AV dissociation). The atria fire at their sinus rate, and an escape pacemaker in the AV junction or ventricles takes over to activate the ventricles at a slower rate. The QRS in complete heart block may be narrow (junctional escape) or wide (ventricular escape) but will be bradycardic. Complete AV block causes fatigue, dizziness, syncope (Stokes-Adams attacks), and is an indication for an urgent pacemaker. Causes include acute myocardial infarction (classically, an inferior MI can cause transient AV block by affecting the AV nodal artery [6], whereas an anterior MI can cause sudden block in the

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bundle branches), degeneration of the conduction system in the elderly (Lenègre's or Lev's disease), or drug toxicity. In summary, AV blocks produce characteristic relationships between P and QRS: first-degree has a long PR; second-degree has intermittent dropped beats (with or without Wenckebach progressive prolongation); third-degree has P–QRS dissociation. ECG recognition of high-grade AV block is critical, as it may necessitate acute intervention (like atropine, transcutaneous pacing) especially if the patient is unstable.

Bundle Branch Blocks (BBB): A bundle branch block occurs when there is a block in the right or left bundle branch, delaying depolarization of the respective ventricle. The QRS complex becomes prolonged (>0.12 s), and specific morphologic changes are seen on the ECG. In Right Bundle Branch Block (RBBB), activation of the right ventricle is delayed, causing a characteristic RSR′ pattern ("M-shaped" QRS) in lead V_1 (and V_2) and a prominent terminal S wave in leads I and V_6 [3]. The QRS duration is ≥120 ms, and secondary ST-T changes (often T wave inversion) appear in the right precordial leads due to the altered depolarization sequence [4]. In classic RBBB, leads V_1–V_3 show an rSR′ (small initial r, S dip, then a tall R′) and leads I, aVL, V_5–V_6 show a wide S wave [3]. Clinically, RBBB can be seen in healthy individuals (especially incomplete RBBB) or caused by structural changes (e.g., atrial septal defect, pulmonary hypertension, chronic lung disease) but by itself it is not as strongly associated with adverse outcomes as LBBB. Left Bundle Branch Block (LBBB) causes the left ventricle to be depolarized late via cell-to-cell spread from the right ventricle. ECG criteria for LBBB include QRS ≥120 ms, broad/notched or "M"-shaped R waves in leads I, aVL, V_5–V_6, and absent normal initial Q waves in those leads [14]. Additionally, LBBB produces discordant ST-T changes—meaning the ST segment and T wave are directed opposite to the major QRS deflection (often ST depression and T inversion in left-sided leads). For example, in LBBB, leads V_5–V_6 show tall broad R waves with associated ST-T that is subtly inverted, whereas V_1 may show a deep broad S wave with ST elevation and upright T (the "appropriate" discordance). These repolarization changes can mask or mimic ischemia on the ECG, complicating interpretation of LBBB patients with chest pain. New-onset LBBB in the context of acute chest pain is concerning and considered an acute myocardial infarction "equivalent" in older guidelines [14]—it may warrant urgent therapy if accompanied by symptoms. Causes of LBBB include long-standing hypertension leading to left ventricular hypertrophy, coronary artery disease (especially when it causes intraventricular septum scarring), cardiomyopathies, or degenerative conduction system disease. Both RBBB and LBBB can be chronic or transient. Importantly, chronic LBBB is associated with mechanical dyssynchrony of the left ventricle; in patients with heart failure and LBBB, this forms the basis for cardiac resynchronization therapy (biventricular pacing). In summary, bundle branch blocks show widened QRS complexes with distinctive patterns: RBBB produces an rSR′ in V_1 and wide S in V_6 [3], whereas LBBB produces broad notched R in lateral leads with absent Q waves [14]. Recognition of BBB on ECG provides clues to underlying pathology (e.g., LBBB often indicates structural heart disease and can affect the interpretation of other ECG changes.

Ischemic ECG Changes (Myocardial Ischemia and Infarction)

Myocardial ischemia (insufficient blood flow to the heart muscle) and infarction (irreversible muscle death, typically from prolonged ischemia) produce well-known patterns on the ECG. Ischemic changes primarily involve alterations in the ST segment, T wave, and Q waves. Clinicians divide the ECG manifestations of acute coronary syndromes into ST-elevation MI (STEMI) and non-ST-elevation MI (NSTEMI)/unstable angina, as this guides urgent management. Here we outline the key ECG signs:

- **ST Segment Elevation:** Elevation of the ST segment is a hallmark of acute transmural injury (as in an ongoing myocardial infarction involving the full thickness of the wall). By convention, significant ST elevation is defined as ≥1 mm (0.1 mV) in two or more contiguous limb leads, or ≥2 mm in contiguous precordial leads (with some gender differences in cutoff). A classic STEMI shows ST elevations in specific lead territories, often with reciprocal ST depression in opposite leads. For example, an anterior STEMI (LAD occlusion) causes ST elevations in V_1–V_4

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with reciprocal depressions in inferior leads. Pathophysiologically, ST elevation indicates current flow due to injury currents in the border zone of ischemic myocardium. Transient ST elevation can also occur in Prinzmetal's (variant) angina during coronary vasospasm, which resolves when the spasm abates [6]. In the early minutes of an acute MI, hyperacute T waves (tall, peaked T waves) may precede ST elevation. Within hours, ST segments elevate and T waves often invert later. For example, in the case of resolved vasospastic angina or an aborted MI, deep T wave inversions may follow transient ST elevation. Clinically, ST-elevation MI is an emergency; prompt reperfusion therapy (angioplasty or thrombolysis) is indicated to salvage myocardium. Of note, new LBBB with symptoms was traditionally treated as STEMI equivalent. In patients with prior MI, re-elevation of ST segments in those same leads can indicate acute reinfarction [6].

- **ST Segment Depression and T Wave Inversion:** Myocardial ischemia that is subendocardial (not full-thickness) typically produces ST depression rather than elevation. ST depression may be horizontal or downsloping and is often accompanied by T wave inversions in the affected leads. Horizontal ST depression ≥1 mm strongly suggests ischemia (such as in unstable angina or NSTEMI), whereas downsloping ST depression is less specific and can also occur with ventricular hypertrophy or digitalis effect [6]. For instance, during an exercise stress test, horizontal or downsloping ST depressions are positive indicators of inducible ischemia. Diffuse ST depression with ST elevation only in aVR can signify left main or multi-vessel ischemia. T wave inversions are another sign of ischemia or infarction. Deep, symmetric T wave inversions in anterior leads (V_2–V_4) may indicate a reperfused anterior STEMI or critical proximal LAD artery stenosis (e.g., Wellens' syndrome). More mild T inversions can be nonspecific but are often seen in NSTEMI or unstable angina (e.g., inverted T waves in V5–V6 with chest pain suggest lateral ischemia). It is important to remember that some T wave inversions can be a normal variant (e.g., isolated inversion in lead III or V_1 is normal. Persistent T wave inversion after an MI typically reflects scar. In summary, NSTEMI/ischemia ECG changes usually include ST depressions and T wave inversions in the leads corresponding to the ischemic region, without the ST elevations that define STEMI. These changes, while subtler, carry prognostic significance—for example, ≥2 mm of widespread ST depression portends a higher risk and often signifies more extensive coronary disease [6].
- **Pathological Q Waves:** Q waves are the initial negative deflections of the QRS and can be normal in certain leads (small "septal Qs" in I and aVL, etc.). However, pathological Q waves are defined by greater depth and duration (e.g., >0.04 s in duration and depth >25–33% of the ensuing R wave) and indicate an area of myocardium that is electrically inert (dead) from a prior infarction [7]. Essentially, a pathologic Q wave signifies that the infarcted tissue no longer conducts electrical current, so the ECG lead "sees through" to the unopposed signals from the opposite wall. Pathological Q waves usually take several hours to develop after an MI and often persist indefinitely. For example, after a transmural anterior MI, leads V_1–V4 may develop deep Q waves. The presence of Q waves on an ECG is evidence of an old myocardial infarction in that territory [7]. One must be careful to distinguish these from normal tiny Q waves due to septal activation. Diagnostic criteria typically require Q waves in at least two contiguous leads for an MI diagnosis. Notably, successful early reperfusion of an MI can sometimes prevent Q wave formation or even result in the disappearance of Q waves over time [7]. Clinically, Q waves tell us about infarct age and location (e.g., Q waves in II, III, aVF indicate an old inferior MI). However, their absence does not exclude MI, especially in NSTEMIs or small infarcts that heal without Q waves. Thus, Q waves are a useful electrocardiographic marker of necrosis and help in retrospective diagnosis of MI.

In patients with chest pain, recognizing these ischemic changes on ECG is critical. A STEMI (with ST elevations) demands immediate reperfusion therapy. NSTEMI/unstable angina (ST depressions/T inversions without ST elevations) still carries significant risk and warrants aggressive medical management and possible early invasive evaluation. It is important to integrate the ECG findings

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with clinical context; about 20% of acute MIs may initially show nonspecific or minimal changes on ECG [6], so serial tracings and adjunct testing (cardiac enzymes, imaging) are used when suspicion is high. Nonetheless, when present, ST-T deviations and Q waves on ECG provide invaluable information about myocardial ischemia, infarction, and their evolution over time.

Electrolyte Imbalances and ECG Changes

Disturbances in serum electrolytes—particularly potassium and calcium—have characteristic effects on the ECG due to their influence on cardiac action potentials. Prompt recognition of these changes can be lifesaving, as severe electrolyte disorders can precipitate dangerous arrhythmias. Below we review classic ECG findings in key electrolyte abnormalities:

- **Hyperkalemia:** Elevated serum potassium levels cause a progressive slowing of impulse conduction and alterations in repolarization. The earliest ECG change with hyperkalemia is peaking of the T waves—classically tall, narrow, tented T waves best seen in precordial leads [11]. As K^+ rises, the QT interval shortens (rapid repolarization), the PR interval prolongs, and the P waves diminish in amplitude. Moderate hyperkalemia (e.g., >6.5 mEq/L) often leads to P wave flattening and eventual disappearance, as atrial activity is suppressed. The QRS complex then begins to widen due to delayed ventricular depolarization. With severe hyperkalemia (>7.0–8.0 mEq/L), the QRS can become markedly broad and merge with the T wave, producing a sine-wave pattern. This is a pre-arrest state—ventricular fibrillation or asystole can occur if no intervention is taken. Hyperkalemia can thus present on ECG along a spectrum: peaked T waves → P wave loss → wide QRS → sine wave → ventricular standstill. Clinically, hyperkalemia is most often due to renal failure, potassium-sparing drugs, or cell breakdown (tumor lysis, rhabdomyolysis). It can cause symptoms of weakness or, most critically, arrhythmias. The ECG changes of hyperkalemia correlate roughly with toxicity and guide therapy urgency. The presence of ECG changes (especially QRS widening) is an indication for immediate treatment (such as IV calcium to stabilize myocardium). In summary, tall peaked T waves are an early clue to hyperkalemia on ECG, and progression to a wide QRS and sine wave forebodes cardiac arrest if untreated [11].
- **Hypokalemia:** Low serum potassium has essentially opposite electrophysiological effects, tending to lengthen repolarization. On ECG, flattening of the T waves is an early finding [10]. As K^+ drops further (<3 mEq/L), ST-segment depressions, T wave inversions, and prominent U waves appear. The U wave is a positive deflection after the T wave; in hypokalemia it becomes larger and more evident, often exceeding the T wave in amplitude (especially in leads V_2 and V_3). Essentially, the T wave may merge into a U wave, creating a long QU interval (sometimes misread as a prolonged QT). True QT prolongation can be considered present (due to the QU prolongation), and this predisposes to a specific polymorphic ventricular tachycardia known as Torsades de Pointes. In severe hypokalemia, patients are at risk for ventricular arrhythmias (VT/VF) and even AV block [10]. Hypokalemia also increases susceptibility to digoxin toxicity and can cause muscle cramps or weakness. Common causes include diuretic therapy, gastrointestinal losses, or endocrine disorders (hyperaldosteronism). The ECG hallmark of hypokalemia is thus a combination of ST depression, low-amplitude or inverted T waves, and prominent U waves. Recognition is important because replenishing potassium (and sometimes magnesium) will usually correct the ECG changes and reduce arrhythmia risk [10].
- **Hypercalcemia:** High calcium levels shorten the plateau phase of the cardiac action potential. The classic ECG finding is a shortened QT interval due to abbreviation of the ST segment [9] [12]. In mild to moderate hypercalcemia, the T waves may appear right after the QRS because of the short ST segment. Hypercalcemia can also cause slight prolongation of the PR interval and QRS duration [12], although these are less prominent than the QT shortening. In severe hypercalcemia (e.g., Ca^{2+} > 14–15 mg/dL), Osborn waves (J waves)—an extra deflection at the end of the QRS—have been reported, somewhat similar to hypothermia changes [9]. Hypercalcemia's cardiac effects include reduced excitability and predisposition to

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bradyarrhythmias or AV block [12], but interestingly, hypercalcemia is less often a direct cause of lethal arrhythmias compared to potassium imbalances. Clinically, hypercalcemia is most commonly due to hyperparathyroidism or malignancy and causes fatigue, confusion, polyuria, etc. The ECG clue of QT shortening can support the diagnosis. For instance, a patient with unexplained weakness and very short QT on ECG should prompt a check of calcium level. Treatment of severe hypercalcemia (IV fluids, bisphosphonates, etc.) will normalize the QT interval. In summary, think "short QT" when observing hypercalcemia [12]—the opposite of what is visualized with hypocalcemia.

- **Hypocalcemia:** Low calcium prolongs the plateau of the action potential, thus prolonging the ST segment and QT interval on ECG [9]. Unlike hypokalemia, which alters the T wave, hypocalcemia typically leaves T wave morphology unchanged; it simply stretches out the ST segment (and thus QT). A QTc significantly >0.46 s may be observed. This predisposes to torsades de pointes (a form of polymorphic VT) particularly when hypocalcemia is severe or combined with other QT-prolonging factors. Patients with hypocalcemia may experience neuromuscular irritability (tetany, tingling, spasms) before arrhythmias occur. Common causes are postsurgical hypoparathyroidism, vitamin D deficiency, or renal failure. On the ECG, one should be cautious that a "long QT" in a patient might be partly due to hypocalcemia—for example, patients with acute pancreatitis often have hypocalcemia that can prolong QT. Treatment of the underlying cause and calcium supplementation will shorten the QT again. Thus, "long QT" (specifically due to long ST) is the signature of hypocalcemia on ECG. Continuous ECG monitoring is advised in severe cases to watch for torsades. Additionally, hypomagnesemia often accompanies and exacerbates the effects of hypocalcemia and should be corrected as well [9].

In summary, electrolyte imbalances produce distinctive ECG patterns: Hyperkalemia—peaked T to sine wave; Hypokalemia—flattened T, U waves; Hypercalcemia—short QT; Hypocalcemia—long QT. Recognizing these can guide urgent electrolyte correction and prevent arrhythmias [10][11][12]. It is also important to consider clinical context, as patients with renal failure (risk for high K^+) or those on diuretics (risk for low K^+) should have careful ECG assessment for such changes.

Indicators of Structural Heart Disease on ECG

Beyond rhythm and ischemia, ECGs can also reflect chronic structural changes in the heart. Increased chamber size or wall thickness (hypertrophy) alters the voltages and depolarization patterns seen on the ECG. While imaging (echocardiography, MRI) is more definitive for diagnosing structural heart disease, the ECG provides clues such as hypertrophy or enlargement of specific chambers.

- **Left Ventricular Hypertrophy (LVH):** When the left ventricle enlarges and thickens (commonly due to hypertension or aortic stenosis), the ECG QRS voltages often increase in leads oriented to the left ventricle. Classic criteria for LVH are based on high QRS amplitudes. For example, the Sokolow–Lyon criteria: S wave in V_1 + R wave in V_5 (or V_6) > 35 mm is suggestive of LVH [1]. The Cornell voltage criteria uses R in aVL + S in V3 > 28 mm (men) or > 20 mm (women). In general, LVH produces tall R waves in left lateral leads (I, aVL, V_5–V_6) and deep S waves in the right precordial leads (V_1–V_3). The QRS interval may be slightly prolonged and the axis often shifts leftward. Additionally, LVH is associated with secondary ST-T changes known as the "strain pattern": slight ST depression and T wave inversion in the left chest leads (V_5–V_6, I, aVL). This reflects delayed repolarization in a hypertrophied ventricle. An example ECG in LVH might show R wave of 30 mm in V_5 with ST depression and inverted T in that lead. Patients with LVH on ECG have an increased risk of adverse outcomes, and regression of LVH with therapy correlates with improved prognosis. The presence of LVH suggests conditions like chronic pressure overload (e.g., long-standing hypertension, aortic stenosis) or volume overload (aortic or mitral regurgitation). Thus, the ECG findings of LVH—high voltage QRS and ST-T strain—are important to recognize. However, ECG has limited

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sensitivity for LVH; many patients with anatomical LVH won't meet voltage criteria on ECG, especially if obesity or COPD attenuates the voltages. But if criteria are met, it strengthens clinical suspicion. In summary, voltage criteria (such as $R_5 + S_1 > 35$ mm) combined with "strain" ST-T changes in lateral leads strongly indicate LVH on the ECG [1].

- **Right Ventricular Hypertrophy (RVH):** In right ventricular hypertrophy, seen in conditions like pulmonary hypertension, COPD, or congenital heart disease, the ECG shows a dominance of right-sided forces. There is often right axis deviation (> +110°) and an abnormally tall R wave in lead V_1 ($R_1 > 7$ mm or R/S ratio > 1 in V_1) [4]. Conversely, the left precordial leads (V_5–V_6) may have a small R and deep S (S wave in $V_6 > 7$ mm). Essentially, the usual voltage pattern is flipped: V_1, normally with a small R, now has a large R; V_6, normally with a large R, now shows a large S. The QRS may still be <0.12 s (unless coexisting with RBBB). RVH often also produces a "strain" pattern in V_1–V_3 (downsloping ST depression and T wave inversion) and sometimes in the inferior leads. Peaked P waves (>2.5 mm) in lead II (P pulmonale) may be present if right atrial enlargement coexists. As an example, an ECG in cor pulmonale (chronic lung disease causing RVH) might show R waves as tall as S waves in V_1, right axis deviation, and T inversions in V_1–V3. The presence of RVH on ECG suggests right-sided pressure overload, such as from pulmonary artery hypertension (e.g., due to chronic pulmonary emboli or emphysema) or volume overload (e.g., atrial septal defect). Like LVH, ECG has limited sensitivity for RVH, but when extreme (e.g., "dominant R in V_1") it's quite specific. In summary, RAD with R wave dominance in V_1 and strain in V_1–V_3 are the key features of RVH on ECG [4].
- **Atrial Enlargement:** ECG can suggest enlargement of the atria by characteristic P wave changes. Right atrial enlargement (RAE) is typically indicated by tall, peaked P waves (P pulmonale). Specifically, a P wave amplitude >2.5 mm in the inferior leads (II, III, aVF) is a sign of RAE [13]. This reflects the increased contribution of the right atrium to the P wave (often due to pulmonary hypertension, tricuspid stenosis, or chronic lung disease causing cor pulmonale). Left atrial enlargement (LAE) manifests as broad, notched P waves (P mitrale) in lead II and a biphasic P wave in V_1 with a deep terminal negative component >1 mm² area. Essentially, the P wave duration becomes ≥0.12 s and may look "m-shaped" in lead II due to sequential activation of an enlarged left atrium. LAE is commonly caused by mitral valve disease (stenosis or regurgitation) or longstanding hypertension. While P wave changes are minor compared to QRS or ST changes, they provide useful clues: for example, a patient with COPD might show both RAE (peaked P in II) and RVH on ECG. These findings direct attention to possible right-heart strain. In severe atrial enlargement, the P wave axis may shift and the morphology differences become pronounced. Overall, P wave height >2.5 mm (inferior leads) suggests RAE, and P wave width >0.12 s with notching (lead II) or biphasic V_1 with deep terminal portion suggests LAE [13].
- **Other Structural Indicators:** There are a few other notable ECG signs of structural changes. Left atrial enlargement was mentioned with P mitrale. Right atrial enlargement with P pulmonale. Biventricular hypertrophy can be hard to diagnose by ECG because criteria for LVH and RVH can mask each other, but sometimes combined patterns are seen (e.g., criteria for LVH in limb leads and an R/S >1 in V_1). Extreme increases in QRS voltage (>50 mm total) could indicate hypertrophic cardiomyopathy. Deep, narrow Q waves in multiple leads (especially V_4–V_6) in a young patient may also suggest hypertrophic cardiomyopathy (septal hypertrophy) rather than infarction [2]. Low QRS voltage (diminished height in all leads) can signal diseases like infiltrative cardiomyopathy (amyloid) or pericardial effusion. Diffuse low voltage combined with electrical alternans (beat-to-beat QRS variation) is classic for cardiac tamponade. These are beyond the main scope, but it is worth noting that the ECG offers many subtle hints about structural pathology.

In clinical practice, when ECG evidence of hypertrophy or chamber enlargement is found, it prompts further investigation (e.g., echocardiography) to confirm and assess severity. For instance,

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ECG criteria of LVH in a hypertensive patient correlate with echocardiographic LV mass [1] and would influence management. Likewise, new onset of LBBB or other intraventricular conduction delays might suggest an underlying cardiomyopathy and indicate the need for imaging. Thus, understanding these ECG signs helps integrate the electrical findings with the anatomical state of the heart.

Conclusion

The ECG remains an indispensable clinical tool, condensing complex cardiac electrophysiology into a simple tracing that clinicians can interpret for clues about a patient's cardiac rhythm, conduction, ischemia, and structural status. This paper has reviewed how a normal ECG is generated and how to recognize its main waveforms and intervals. We then explored a range of ECG abnormalities—from arrhythmias like atrial fibrillation and ventricular tachycardia to conduction blocks, ischemic changes of myocardial infarction, electrolyte-induced patterns, and signs of chamber hypertrophy. Each of these findings offers insight into underlying pathophysiology: for example, an irregularly irregular rhythm with no P waves signifies chaotic atrial activity in atrial fibrillation [13]; ST-segment elevation in specific leads localizes an acute infarct [6]; peaked T waves warn of dangerous hyperkalemia [11]; deep S waves in V_1 with strain pattern point to left ventricular hypertrophy [5]; and so on. Importantly, ECG interpretation requires correlation with the clinical context—an ECG abnormality is most meaningful when considered alongside the patient's symptoms, history, and other tests. Modern guidelines and large studies continue to refine the prognostic and diagnostic significance of various ECG changes. Nevertheless, the fundamental patterns described—P-QRS-T morphology, timing intervals, and deviations in these—remain constant and are grounded in cardiac anatomy and physiology. Mastery of ECG interpretation is a core skill for healthcare providers. It allows early diagnosis of life-threatening conditions (like STEMI or VT), guides therapy (as in AV block or electrolyte disorders), and often provides the first hint of occult pathology (like a silent prior MI evidenced by Q waves [7] or asymptomatic hypertrophy due to hypertension). In an era of advanced imaging and analytics, the ECG's elegance lies in its immediacy, accessibility, and breadth of information from a simple set of skin electrodes. Ongoing research and experience continue to validate the ECG's role—as both a clinical workhorse and a window into the electrical and structural state of the heart [13]. By combining diligent pattern recognition with knowledge of cardiac pathophysiology, clinicians can unlock a wealth of diagnostic and prognostic data from every ECG encountered.

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