

QT Interval In The Electrocardiogram (ECG)

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Introduction

The QT interval on an electrocardiogram extends from the beginning of the QRS complex to the end of the T wave and represents the total time required for ventricular depolarization and repolarization. It does not necessarily begin with a visible Q wave, and it should not be interpreted as one fixed normal value for every patient because the interval changes with heart rate, sex, age, QRS duration, medication exposure, electrolyte status, and the method used to define the end of the T wave. Clinical interpretation therefore depends on both measurement quality and context. Automated ECG systems can provide useful QT and corrected QT values, but they may misidentify QRS onset or T-wave termination when the tracing contains artifact, low-amplitude T waves, prominent U waves, atrial fibrillation, premature beats, pacing, or bundle-branch block. When a decision about medication, syncope, congenital long-QT syndrome, or arrhythmia risk depends on the value, clinicians should review the tracing manually and interpret it alongside symptoms, laboratory results, medication history, and the patient's broader cardiovascular condition.

Measurement and Electrophysiological Meaning

Ventricular depolarization begins with the QRS complex, while the ST segment and T wave represent later phases of the ventricular action potential and repolarization. Cardiac myocytes generate these electrical changes through coordinated sodium, calcium, and potassium currents. QT prolongation can occur when outward repolarizing currents are reduced or inward currents persist, which is why many drug-related effects involve the rapid component of the delayed rectifier potassium current associated with the hERG or KCNH2 channel. At the standard ECG paper speed of 25 millimeters per second, one small box equals 40 milliseconds and one large box equals 200 milliseconds, so paper speed must be checked before manual calculation. The interval is measured from the earliest onset of the QRS to the point where the terminal T wave returns to the isoelectric baseline. A distinct U wave is normally excluded. When the T and U waves merge or the T wave is notched or unusually flat, measurement becomes less certain and should be documented as such.

Heart-Rate Correction and Normal Ranges

Raw QT duration generally becomes shorter as heart rate increases and longer as heart rate slows, so comparison across different rates usually requires a corrected QT, or QTc. Bazett's formula, $QTc = QT$ divided by the square root of the RR interval, remains widely used and is commonly printed by ECG machines, but it tends to overcorrect at faster rates and undercorrect at slower rates. Fridericia's cube-root correction and other formulas such as Framingham and Hodges may perform better in selected circumstances. No formula is perfect, and the method should be identified when serial values are compared. Common adult thresholds often regard a QTc above approximately 450 milliseconds in men and 460 milliseconds in women as prolonged, while a QTc of 500 milliseconds or more is associated with substantially greater torsades de pointes risk. These thresholds are clinical guides rather than absolute diagnostic boundaries. A large increase from baseline, symptoms, interacting medications, or severe electrolyte disturbance may be important even when the measured value remains below 500 milliseconds.

Congenital and Acquired QT Prolongation

Congenital long-QT syndrome comprises inherited channel disorders that delay ventricular repolarization and can predispose to syncope, torsades de pointes, ventricular fibrillation, and sudden cardiac death. Diagnosis depends on the ECG together with personal history, family history, symptoms, and sometimes genetic testing; a normal QTc on one tracing does not fully exclude an inherited syndrome because expression can vary. Acquired QT prolongation is more common and may result from medications, bradycardia, electrolyte disturbance, structural heart disease, renal or hepatic dysfunction, or combinations of these factors. Hypokalemia and hypomagnesemia are particularly important because they can intensify the effect of QT-prolonging drugs. Relevant medication classes include selected antiarrhythmics, antipsychotics, antidepressants, antibiotics, antiemetics, opioids, antimalarials, antihistamines, and oncology agents, although risk differs markedly among individual drugs, doses, routes, and patients. Dangerous situations often arise through accumulation: several modest risks can become clinically significant when multiple drugs, impaired clearance, electrolyte loss, and slow heart rate occur together.

Wide QRS, Irregular Rhythms, and Common Measurement Errors

QT interpretation becomes more difficult when the QRS complex is prolonged because the measured interval includes both delayed depolarization and repolarization. Bundle-branch block, ventricular pacing, pre-excitation, and intraventricular conduction delay may therefore make a machine-generated QTc appear more abnormal even when part of the prolongation comes from depolarization rather than from the repolarization phase associated with torsades risk. In these settings, the JT

interval or a validated adjustment may provide additional information, and specialist interpretation can be appropriate. Atrial fibrillation also complicates correction because RR intervals vary from beat to beat, so several beats may need to be averaged according to an established method. Premature beats and immediately post-premature cycles are generally unsuitable for routine measurement. Other common errors include beginning at the Q wave rather than the true QRS onset, including a separate U wave, ignoring paper speed, using a noisy lead, confusing QT with QTc, mixing milliseconds and seconds in a formula, and accepting an automated result without visual review.

Clinical Significance, Monitoring, and Emergency Management

QT prolongation itself may be asymptomatic, but the ventricular arrhythmia torsades de pointes can cause palpitations, lightheadedness, syncope, seizure-like cerebral hypoperfusion, cardiac arrest, or degeneration into ventricular fibrillation. Risk rises when prolonged repolarization occurs with factors such as bradycardia, hypokalemia, hypomagnesemia, structural heart disease, high drug exposure, renal or hepatic impairment, or several QT-prolonging medications. Before starting a higher-risk drug, clinicians may review the baseline ECG, electrolytes, organ function, cardiovascular history, family history, and interacting medications, with repeat ECGs after dose changes or when symptoms develop according to the drug and clinical setting. Sustained polymorphic ventricular tachycardia causing instability requires immediate electrical treatment under resuscitation protocols. Current American Heart Association guidance supports magnesium for recurrent polymorphic ventricular tachycardia associated with long QT and emphasizes correction of contributing electrolyte abnormalities. Patients should not stop an essential prescription independently because an automated ECG reports a prolonged QT; the result requires prompt clinical interpretation.

Drug Regulation, Documentation, and Patient Education

Modern drug development includes formal assessment of a medicine's potential to delay ventricular repolarization and produce proarrhythmic risk. FDA and international E14/S7B guidance combines nonclinical ion-channel evidence, clinical ECG data, concentration-response analysis, pharmacokinetics, and observed adverse events rather than relying on one average QT change. The FDA's final 2025 guidance on QTc information in prescription drug and biologic labeling further emphasizes consistent communication of clinically meaningful evidence. In everyday practice, documentation should identify rhythm, heart rate, raw QT, RR interval, correction formula, QTc, QRS duration, lead or measurement method, and the reason the value is clinically relevant. Medication and electrolyte context should be recorded so a borderline number does not become an unexplained permanent diagnosis. Patients with established long-QT syndrome or important drug-related risk should know which symptoms require urgent attention and should tell clinicians and pharmacists

about the condition before starting new medicines. Education should be specific and balanced, because many medications with potential QT effects remain safe and beneficial when selected and monitored appropriately.

Conclusion

The QT interval is a clinically important measurement because it reflects ventricular depolarization through repolarization and can reveal vulnerability to dangerous ventricular arrhythmias when interpreted correctly. Accurate assessment begins with proper identification of QRS onset and T-wave end, verification of paper speed, and recognition that automated measurements can fail in difficult tracings. Because the interval changes with heart rate, QTc formulas such as Bazett and Fridericia are used, but their limitations must be understood and no single universal threshold should be treated as a diagnosis. Congenital channel disorders, medications, electrolyte abnormalities, bradycardia, organ dysfunction, and wide-QRS rhythms all alter interpretation. A prolonged QT increases susceptibility to torsades de pointes but does not guarantee that the arrhythmia will occur. Safe clinical use therefore combines the ECG with symptoms, baseline change, QRS duration, rhythm, laboratory values, medication exposure, and patient history. Measurement is most valuable when it informs a complete risk assessment rather than when one isolated number is interpreted without context.

References

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