Clinical Scenario:
Jessica is a 23-year-old university student who presents to hospital with anorexia and symptoms of a lower respiratory tract infection. She is otherwise healthy and is on no medications. The remainder of her history is unremarkable except for a family history of sudden, unexplained death in a maternal aunt. Baseline blood work shows mild hypokalemia and a left lower lobe infiltrate on her chest x-ray. She is admitted to hospital for correction of her electrolytes and is given clarithromycin for her pneumonia. The following day, she suffers a cardiac arrest. Polymorphic wide QRS complex tachycardia is seen on the monitor. A post-resuscitation ECG demonstrates striking QT findings (Figure 1).

Question: How does the clinician approach the measurement and interpretation of the QT interval on the ECG?

The QT interval is the electrocardiographic representation of ventricular depolarization and subsequent repolarization. This myocardial electrical activity is mediated by ion channels within the cardiacmyocyte cellmembrane. Reduced or absent function of key ion channels results in excess intracellular sodium or potassium, which can lengthen the repolarization period and prolong the QT interval. This leads to ventricular arrhythmia caused by an entity known as early afterdepolarizations, which can have significant prognostic implications.
Long QT syndrome (LQTS) can be overt or subclinical and is usually the result of a genetic predisposition. Abnormally long QT intervals have been associated with a potentially fatal form of polymorphic ventricular tachycardia, known as Torsades de pointes (TDP) (Figure 2). The risk of developing TDP is variable, but generally requires a precipitating factor such as ischemia, bradycardia, electrolyte imbalances or a QT-prolonging medication. In patients with congenital LQTS, specific triggers such as swimming or auditory stimuli may trigger events.

The prevalence of QT prolonging agents make the QT interval an unavoidable and important issue. Many common drugs, including certain classes of antibiotics, antipsychotics, antiemetics and antiarrhythmics can prolong the QT interval and precipitate TDP. Unfortunately, several studies have shown that QT prolongation is poorly measured and interpreted by healthcare team members. Fortunately, it is a skill that can be easily taught, without any special background training.

The goal of this article is to educate medical professionals on how to accurately measure the QT interval in order to facilitate the recognition of QT prolongation and avoid potential adverse clinical outcomes.

Figure 1. Resting ECG on Jessica, a 23-year-old woman with a family history of sudden death.
Figure 2. Torsades de pointes: note the two premature ventricular contractions induce a pause, followed by a QRS with a dramatically prolonged QT interval. This is followed by polymorphic ventricular tachycardia, characterized by wide complexes which appear to be twisting around the isoelectric line. The rhythm most often terminates spontaneously.

Figure 3. The QT interval is defined from the beginning of the QRS complex to the end of the T wave. The maximum slope intercept method defines the end of the T wave as the intercept between the isoelectric line with the tangent drawn through the maximum down slope of the T wave (left). When notched T waves are present (right), the QT interval is measured from the beginning of the QRS complex extending to the intersection point between the isoelectric line and the tangent drawn from the maximum down slope of the second notch, T2.

How to measure the QT interval

On a 12-lead ECG, the QT interval is measured from the beginning of the QRS complex to the end of the T wave. Both manual and automatic measurements of this interval are often complicated by a variety of factors including a noisy baseline, variations in T wave morphology, U waves and merging of the T waves with U and/or P waves. Manual measurements of the QT interval should be taken from leads II or V5 and averaged over three to five successive beats, with the maximum measured interval taken as the final result. Measurements made from these leads have the greatest positive and negative predictive value in detecting abnormal QT intervals.(3) The QT interval is influenced by a variety of factors including gender, heart rate, underlying rhythm and conduction defects. There is a range of opinions as to the normal values of the corrected QT interval and at least as many suggested approaches on how to correct for the
above variables. The most reported criteria in the medical literature uses Bazett’s formula, which defines the normal value as < 440 ms for men (borderline 440 ms to 460 ms) and < 460 ms for women (borderline 460 ms to 480 ms).(4) Several basic algorithms for measuring the QT interval have been derived. Most vary according to how the T wave offset is determined. The various methods for determining the end of the T wave can be grouped into either the slope methods or threshold methods. The slope-based methods have the greatest reliability and thus are preferred.\(^{(3)}\) The widely used maximum slope intercept method defines the end of the T wave as the intersection point between the tangent drawn at the maximum down slope of the T wave and the isoelectric line (Figure 3). Figure 4 illustrates how the maximum slope intercept method can be applied to determine Jessica’s QT interval.

**U waves**

U waves can sometimes mimic the appearance of notched T waves making it difficult to correctly identify the end of the T wave. If notched T waves are present, the tangent is applied at the maximum down slope of the second notch (Figure 3). U waves < 0.1 mV in amplitude or independent from the T wave should be excluded from the QT measurement. When larger U waves merge with the T wave, they should be included in the QT measurement.\(^{(5)}\)

<table>
<thead>
<tr>
<th>Correction</th>
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<th>Comment</th>
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<tbody>
<tr>
<td>Bazett</td>
<td>QTc = QT/√RR</td>
<td>Widely used for its simplicity; vercorrects at heart rates &gt; 100 bpm and undercorrects at heart rates &lt; 60 bpm</td>
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<tr>
<td>Fredericia</td>
<td>QTc = QT/(RR(^{1/3}))</td>
<td>Maintains accurate correction at higher heart rates</td>
</tr>
<tr>
<td>Framinghamham</td>
<td>QTc = QT + 0.154(1-RR)</td>
<td>Relatively consistent correction from bradycardic to tachycardic heart rates</td>
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**Figure 4.** Application of the maximum slope technique to Jessica’s ECG in lead II (left) and V5 (right) yields a QT interval of 490 msec in II and 480 msec in V5. Using the maximum measured interval and an RR interval of 0.88 seconds, the QTc is 522 msec (QTc = QT/√RR), dramatically prolonged compared to the accepted upper limit of normal.
Correcting for heart rate

Because QT interval duration is dependent on heart rate, a rate correction should be applied to QT interval measurements. This allows QT measurements to be compared over time and to normal cut offs independent of heart rate. Many corrections exist, each with its own benefits and shortcomings. Unfortunately, no consensus exists on which correction is most effective (Table 1). The most commonly used Bazett’s formula (QTc=QT/√RR) provides an adequate correction for heart rates ranging from 60 bpm to 100 bpm. At heart rates outside of that range, the Fredericia (QTc=QT/[RR]1/3) or Framingham (QTc=QT+0.154[1-RR]) corrections should instead be applied.

Conclusion:

1. Individuals with long QT syndrome are at risk for a potentially fatal arrhythmia known as Torsades de pointes

2. Torsades de pointes can be precipitated by a variety of factors, including QT-prolonging medications

3. Algorithms on how to measure the QT interval are easy to learn and apply

4. A systematic approach will improve recognition and reduce adverse outcomes

For more information on QT-prolonging medications and drug interactions: www.qtdrugs.org

References:


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