**QT interval measurement**

**Andrés R. Pérez Riera MD**

**QT interval measurement:**What’s the QT/QTc intervals and why’s are so important? The QT interval or electric systole on the ECG is measured from the beginning of the QRS complex to the end of the T wave. It represents the time it takes for the ventricles of the heart to depolarize and repolarize. The QT interval is longer when the heart rate (HR) is slower and shorter when the HR is faster. So it’s necessary to calculate the corrected QT interval (QTc) using:

**The Bazett formula:** the Bazett’s formula was proposed in 1920: QT interval divided by the square root of the R-R interval. The R-R interval is measured from one R wave to the next R wave that comes before the QT interval being measured. For example, if the QT interval measures 440ms and the R-R interval measures 860ms, then the QTc is 470ms. The normal QT interval varies depending on age and gender, but it’s usually **360 to 440ms**. QTc values <330 ms are considered short QT interval. Values of QTc >450 ms are considered long QT intervals. Normal values of QTc are between 350 to 440 ms or 446 + - 15%. Anything ≥ 500ms is dangerous for any age or gender. Bazett´s formula has been criticized because it tends to provide an inappropriately short QTc at slow rates and inappropriately long QTc at higher rates. Bazett's correction is used for automated analysis and large clinical trials.The range of normality of QT interval in adults varies between 350 ms and 440 ms. Both short and long QT intervals can be susceptible to life-threatening ventricular arrhythmias.

 Several competing methods have been developed:Friderica: **QTcF=QT⁄3√RR** published an alternative correction using the cuberoot of RR; Framingham: QTc = QT + 0.154 (1-RR) and  Hodges: QTc=QT+105(1+RR-1). None of the formulas has been shown to be clearly superior, so despite its obvious shortcomings.

QT duration is inversely proportional to heart rate.

**Factors that make difficult to measure the QT interval**:

**A wide QRS complex** (RBBB, LBBB, WPW and non-specific intraventricular conduction) on an ECG may give the appearance that the QT interval is prolonged. However, a wide QRS complex represents depolarization, and LQTS is a disorder of repolarization. In these cases, we must use the JT and JTc intervals: interval that extends from the J point to the end of the T wave. The QTc interval constitutes the classical measurement of ventricular repolarization; however, the parameter includes ventricular depolarization. Thus, when there is branch block or WPW ventricular preexcitation, the measurement of ventricular repolarization by QTc may be incorrect. In such cases, the measurement of JTc is more accurate than the QTc interval, because it excludes depolarization. The measurement of JTc may be useful to identify LQTS cases with borderline values, where QTc interval could be normal in rest ECG. We find an example in patients carriers of tetralogy of Fallot who underwent surgery, and as a consequence of RV ventriculotomy, developed CRBBB. In these cases, JTc interval measurement is more sensitive than the QTc interval to detect prolonged repolarization.

**The end of T wave:** Sometimes the end of a T wave isn’t clearly defined, which can make it difficult to get the QT measurement. When measuring QT intervals, it’s important to find a lead on the ECG that has a T wave with a clearly defined end.The end of T wave is defined as T wave return to the baseline, here called T-P segment. For a proper medication of the QT interval, we should be certain of not having included the U wave. To that end, it is advisable to perform the measurement in the aVL lead, because it is usually perpendicular to the U wave axis (SAU).Bimodal or notched T waves may be distinguished from the T-U interval: the second apex of bimodal T wave (T2) is at a distance from the first one (T1) < 150 ms; the T1-U interval is > 150 ms. The second apex of bimodal T wave (T2) is at a distance < 150 ms from the first module (T1): The T1-U interval is always > 150 ms.

**Irregular rhythms( your question)** such as AF or continuous bigeminies’ also make it difficult to obtain a consistent QT interval measurement. For example, AF makes it extremely difficult to measure a QT interval because finding a T wave isn’t always possible with this rhythm. The best thing to do is measure five or six QT intervals and average them together. Each lead of an ECG will most likely give you a slightly different QT measurement.

**Observation:** When measuring the QT interval, the ECG is best recorded at a paper speed of 50 mm/s and at an amplitude of 0.5 mV/cm using a multichannel recorder capable of simultaneously recording all 12 leads. A tangent line to the steepest part of the descending portion of the T wave is then drawn. The intercept between the tangent line and the isoelectric line is defined as the end of the T wave The QT interval is measured from the beginning of the QRS complex to the end of the T wave on a standard ECG. There are no available data on which lead or leads to use for QT interval measurement. Traditionally, lead II has been used for QT interval measurement because in this lead, the vectors of repolarization usually result in a long single wave rather than discrete T and U waves. Generally, QT prolongation is considered when the QTc interval is greater than 440 ms (men) and 460 ms (women), although arrhythmias are most often associated with values of ≥500 ms. The severity of pro-arrhythmia at a given QT interval varies from drug to drug and from patient to patient. Unfortunately, the extent of QT prolongation and risk of TdP with a given drug may not be linearly related to the dose or plasma concentration of the drug because patient and metabolic factors are also important (for example, sex, electrolyte concentrations, etc). Furthermore, there is not a simple relation between the degree of drug induced QT prolongation and the likelihood of the development of TdP, which can occasionally occur without any substantial prolongation of the QT interval.

LQTS is a result of the heart’s electrical system recharging abnormally. There are two types:

1.      [**Acquired LQTS:** it is caused by an underlying medical condition, such as drugs that prolong the QT interval, electrolyte imbalances (such as caused by anorexia), and bradycardia. There are many drugs that can prolong the QT interval, such as some antibiotics, antidysrhythmics, antihistamines, antifungals, and antipsychotics. Other categories of drugs that cause QT prolongation are some heart medications, cholesterol-lowering drugs, and diabetes medications. This doesn’t mean that all drugs in these categories can cause it, but many of them can. A person is more likely to develop QT prolongation from these drugs if they have the following risk factors: renal dysfunction, hypokalemia, hypomagnesaemia, bradycardia, advanced age, female gender, underlying heart disease, and polypharmacy. Be aware of these risk factors in your patients and be aware of the drugs they’re on. Whenever you start a patient on a new drug that may prolong the QT interval, always document the baseline QT interval before administering the drug. Continue monitoring and documenting the QT interval at least once every 8 hours.

2.      **Congenital LQTS:** Congenital LQTS is inherited from one or both parents. In addition, there are 12 different subtypes of LQTS, labeled LQT1 to LQT12. Talk to the healthcare provider about correcting any electrolyte imbalances, such as low potassium, magnesium, calcium, and sodium, before starting the drug. Congenital LQTS is a genetic mutation of the ion channels in the cardiac cells. It affects the flow of potassium and sodium in and out of the cells. Genetic testing issued to determine whether a person has the congenital form. Three of the congenital types are more common than the others: LQT1, LQT2, and LQT3. People with congenital LQTS are prone to go into TdP when there’s an external stimulus. The triggers for TdP are somewhat specific to the type of LQTS a person has. β-blockers are the main medication used for the management of congenital LQTS. If medication isn’t successful, then the patient may need dual chamber pacing at a rate to shorten the QT interval. For patients at increased risk or those who continue to experience severe life threatening cardiac events, an implantable cardioverter defibrillator (ICD) is strongly recommended. If you take care of a patient newly diagnosed with congenital LQTS, patient education needs to be a priority. Teach the patient used to determine whether a person has the congenital form. Three of the congenital types are more common than the others: LQT1, LQT2, and LQT3.  Triggers

Exercise, especially swimming in LQT1 variant;

Emotion or stress and noises: LQT2;

Events during sleep or at rest: LQT3.

How should we proceed to read the ECG in suspicion of LQTS?

Do not perform the measurement of intervals and waves by the computerized method.

Conduct an independent review of ECG.

The measurement of the QT interval should be made by an experienced cardiologist.

The general cardiologist, before the suspicion of LQTS, should refer the patient to a colleague familiar with this for cardiological evaluation.

 People with congenital LQTS are prone to go into TdP when there’s an external stimulus. The triggers for TdP are somewhat specific to the type of LQTS a person has. If you take care of a patient newly diagnosed with congenital LQTS, patient education needs to be a priority. Teach the patient if he or she has any illnesses that lower the potassium levels, such as vomiting or diarrhea, because this could trigger an episode of TdP. Ask the patient whether he or she has any family history of SCD. With any patient in your care, always consider LQTS if the patient has fainted and it can’t be explained by any other medical condition. Getting to the point of TdP What can happen if the QT interval is too long? If the QT interval is ≥ 500 ms, then a patient’s heart rhythm is more likely to progress into TdP, an irregular chaotic heartbeat that’s a type of polymorphic VT. To determine whether a polymorphic VT is TdP, look at the beats around it. First the QT interval will be prolonged, usually with a pause in the heartbeat followed by a beat with a bizarre T wave. This is where TdP usually starts. When this happens, the cardiac output drops and the patient doesn’t get enough oxygen to the brain and can faint. If the heart doesn’t return to a normal sinus rhythm, it will eventually go into VF. VF requires immediate defibrillation because it can lead to SCD if left untreated.

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1. Genetic entity
2. Long QTc in ECG.
3. Hallmark of arrhythmia: TdP
4. The most common symptom is: unexplained syncope.
5. Syncope during exercise in pediatric patients should be considered malignant until the contrary is proved.
6. SCD in children or young adults.
7. Family history of dizziness or deafness.

**Causes of arrhythmic syncope**

1) Very fast VT or TdP with hypotension;

2) AF or atrial flutter with high rate of ventricular response in WPW;

3) AV block;

4) Sinus arrest.

**Symptoms**

Syncope.

Dizziness.

Palpitations or chest pain.

SCD.

ECG:  long QT interval and Torsade de Pointes (TdP).

**Diagnosis criteria**

**HISTORY:** dizziness, syncope, aborted SCD;

**FAMILY BACKGROUND:** early sudden death in first degree family members. Unexplained early sudden death.

**ECG:** long QTc interval, TdP, T wave with notches, low HR for the age;

There are some signs of impending TdP that you need to be aware of:

QTc > 500 ms  after starting a QT-prolonging. Patients with QTc intervals >600 ms are considered to be in high risk of arrhythmic SCD by TdP. In these cases, if the pharmacological treatment with β- blockers is insufficient to abolish TdP or in patients carriers of severe bronchial asthma or type 1 diabetes mellitus, where they are contraindicated

Frequent premature ventricular contractions (PVCs)

Couplets (2 PVCs back to back) seen while monitoring the heart rhythm

Height of the T wave alternates from beat to beat (this may or may not occur). The alternation of T wave polarity is a characteristic of patients carriers of LQTS. Isolated T wave alternans is not related to tachycardia or extra-systole, and it usually indicates advanced heart disease or severe electrolytic disorder.  Are causes of T wave alternans Tachycardia, sudden changes in cycle length or HR cycle, severe hyperpotassemia of uremia, experimentally, in hypocalcemia in dogs, severe myocardial impairment: cardiomyopathy, acute myocardial ischemia, particularly in variant angina, post-resuscitation, acute pulmonary embolism, after administration of amiodarone or quinidine (rare), congenital long QT syndromes of the Romano-Ward or Jervell-Lange Nielsen types, Brugada syndrome, and non-sustained runs of TdP after a pause.

When a patient is in TdP, always look at him or her and listen. Remember to ask the right questions. Ask the patient whether he or she has any chest pain or shortness of breath. Look to see whether there’s a change in level of consciousness or BP. Print a rhythm strip and place it in the chart. Review the patient’s drug list and stop any medications that prolong the QT interval. Obtain lab work to check electrolyte levels: potassium, sodium, magnesium, and calcium. There are various ways to manage TdP. Give I.V. magnesium per the healthcare provider’s orders. The healthcare provider may start the patient on a beta-blocker to shorten the QT interval. Digoxin can also shorten the QT interval. If the patient is still in TdP after this, overdrive pacing will be needed. A transvenous pacemaker is inserted and set at a rate between 100 and 110 bpm. This high HR prevents pauses and shortens the QT interval. A patient in sustained TdP may progress to VF and needs to be defibrillated immediately.

Listen to what they tell you. Your vital sign readings and cardiac monitors only give you part of the information you need to take care of your patient. Your patient gives you the rest.

Andrés.