QTc Risk Clinical Decision Support: A Primer For Healthcare Providers

MODULE 1: Detecting Proarrhythmic ECG Changes

DETECTING PROARRHYTHMIC ECG CHANGES

Thank you for your interest in this educational program
"QTc Risk Clinical Decision Support: A Primer For Healthcare Providers."
This first educational module "Detecting Proarrhythmic ECG Changes" focuses on the specific changes that occur within cardiac myocytes that lead to changes on the electrocardiogram. In this module you will learn to recognize when your patients are at risk for an arrhythmic event.

Cardiac Electrophysiology

Fortunately we no longer need to rely on placing our patients' hands and feet in electrolyte baths to make a proper heart rhythm diagnosis.

Today, we are able to capture a precise reading of the heart's electrical activity with just a small amount of gel on the skin using a modern electrocardiogram.
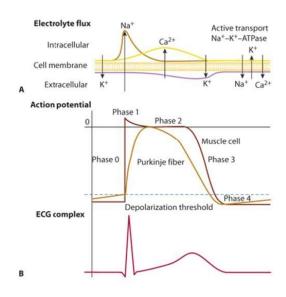
Electrolyte Movement Cardiac Membrane

To begin, let's review some of the electrophysiological principles involved. The above image shows in the top panel the ionic currents that flow across the heart cell membrane during a heart beat. The middle panel shows how those current affect the action potential of a myocyte or a Purkinje (pur-KIN-jee) fiber and the lower panel shows how these processes determine the waveform of an electrocardiogram recording from electrodes on the skin.

Each cardiac cell uses a large amount of energy to actively transport sodium and potassium in different directions across the cell membrane.

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Electrolyte Movement Cardiac Membrane

From: The Fundamental Principles of Medical Toxicology
Goldfrank's Toxicologic Emergencies,

As shown in the top portion of this image, this has the effect of creating both an electrical gradient — a net negative charge across the cell membrane --- because there are more positively charged sodium ions outside, and also a chemical gradient, due to both sodium and potassium.

To contract in a coordinated fashion, channels open and close allowing sodium, potassium and calcium to flow into and then back out of the cell causing the cardiac myocytes to contract and relax in a coordinated fashion.

For example, a signal for a heartbeat begins in pacemaker cells by the opening of voltage-gated sodium channels. Large amounts of sodium ions flood into the cell, both due to the electrical and chemical gradient.
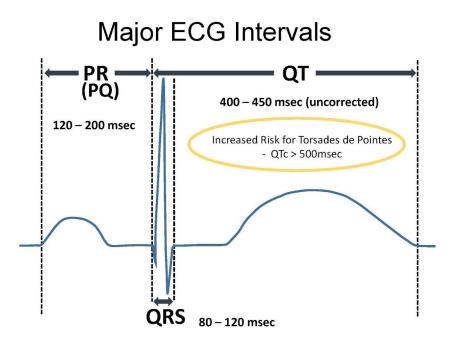
This triggers the sarcoplasmic reticulum to release sequestered calcium, a secondary messenger, through calcium channels.

With each beat, first, we have a sodium channel current occur and then a calcium current.

This calcium released in the cell causes the binding of actin and myosin which actually causes our cells to contract.

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In order to get the cell membrane back to the normal resting potential and prepared for the next heart beat, the cell must move ions to re-establish the normal resting potential. Potassium ions are used for this purpose. The potassium channels in the membrane open to allow potassium to exit the cells. There are several types of potassium channels and you will hear them referred to in different ways. The largest potassium current is often called I_K or the delayed rectifier current. It has two components, one rapid or I_{Kr} and the other slow or I_{Ks}. The I_{Kr} current is also often called the hERG current because it is the product of the hERG gene which amusingly stands for the human Ether-à-go-go gene.

Major ECG Intervals

PR (PQ) ←————→ QT ←————————————————→

400 – 450 msec (uncorrected)

120 – 200 msec

Increased Risk for Torsades de Pointes
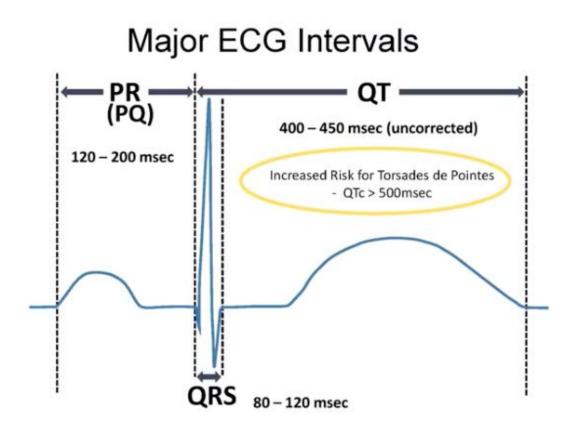- QTc > 500msec

QRS 80 – 120 msec

In the lower panel of the above image, the electrocardiogram shows the QRS caused by the depolarization of the ventricle followed by the T-wave which represents the movement of ions through potassium channels.

In this module we are particularly interested in these potassium channels.

There are a number of medications and other xenobiotics that can block or otherwise interfere with these potassium channels.

Major ECG Intervals

The above image shows the P wave caused by contraction of the atria and the PR interval is the time it takes to be conducted through the bundle of His and reach the ventricles.

The ventricles depolarize and contract to generate the QRS complex and then, as the heart re-polarizes to reach its resting membrane potential, we see what we call the T wave.

From the beginning of the QRS to the end of the T-wave, we measure the QT interval which represents the time taken for depolarization and complete repolarization for a heartbeat. In a normal human being, this takes somewhere in the neighborhood of 400 to 450 ms. If the QT is greater than 500 ms, the patient is at risk for the life-threatening cardiac dysrhythmic condition called torsades de pointes.

We will discuss the QTc versus the QT later in the module

QT INTERVAL

It is important to recognize that nature has addressed the fact that there is less time to re-polarize when your heart is beating at very fast rates. Therefore the QT interval is shorter at fast heart rates. This makes it difficult to compare two QT intervals if they were recorded at different heart rates. What we actually do is correct the QT based on the heart rate in order to normalize it to a common rate of 60.

Traditionally, Bazett's formula has been used to correct the QT because it is the easiest to calculate mathematically. However, please be aware that Bazett's is not a perfect correction and other formulas have been found preferable, especially at higher heart rates where it gives larger values than appropriate.

In some cases, you may see the Fridericia or Framingham formula used. Today, we can easily utilize an app on our phone or a Web calculator to make these more complex calculations for us.

For links to more information and suggested applications, please refer to the tools section of the web page that corresponds with this Module, found at:

www.qtccds.crediblemeds.org

NORMAL RANGE FOR A QT INTERVAL

One thing to keep in mind is that the normal range for QTc Interval is different between men and women and varies with age.

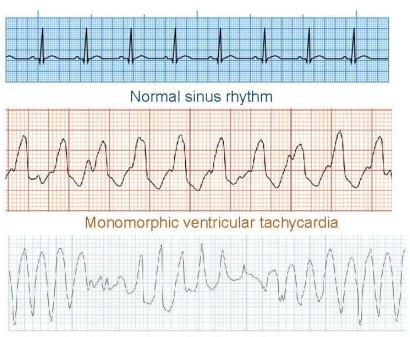Women have a significantly longer QTc than men at most times throughout their lives.

Extensive research has shown the importance of hormones on the QTc in various physiologic conditions such as puberty, menses, pregnancy and menopause.

There is considerable evidence that the longer QTc in women contributes to a greater incidence of torsades de pointes in women, at least during their early adult life.

For normal adult women the upper limit of normal for the QTc is ≤ 460 msec
For adult men, the upper limit of normal for the QTc is ≤ 450 msec

TORSADES DE POINTES

Normal sinus rhythm

Monomorphic ventricular tachycardia

Torsades de pointes

Represented in the above image is a tracing of normal sinus rhythm on the upper recording. The second image is an arrhythmia known as monomorphic ventricular tachycardia. Typically this arrhythmia occurs in a clinical setting that has disrupted sodium channels, such as cardiomyopathy, an acute myocardial infarction or perhaps an overdose with a drug that blocks sodium channels.

The lower tracing is a very different arrhythmia and the topic of this section, torsades de pointes which translates from the French as "twisting of points". This arrhythmia is seen when the QTc is excessively prolonged, either as an inherited disorder, the congenital long QT syndrome, or the acquired long QT syndrome caused by electrolyte disturbances or drugs that prolong the QT, most often because they block potassium channels.

TORSADES DE POINTES

The image below shows an actual patient rhythm strip showing torsades de pointes. The critical thing about TdP is that it can degenerate into other more lethal dysrhythmias

and often times by the time a team arrives to take care of a patient or EMS arrives the ECG is no longer TdP but the patients is in full ventricular fibrillation or asystole.

Torsades de Pointes (TdP)

- TdP is a life-threatening polymorphic ventricular tachycardia characterized by a gradual change in the amplitude and twisting of the QRS complexes around the isoelectric baseline in the setting of marked QT prolongation.

- TdP episodes can be transient causing loss of consciousness or they can persist to degenerate into ventricular fibrillation and sudden death.

The good news is that the treatment being delivered by the code team, defibrillation, is appropriate in either case. However, without the early tracing that shows QT prolongation and the classic twisting pattern, the underlying cause can be missed and errors made in future management.
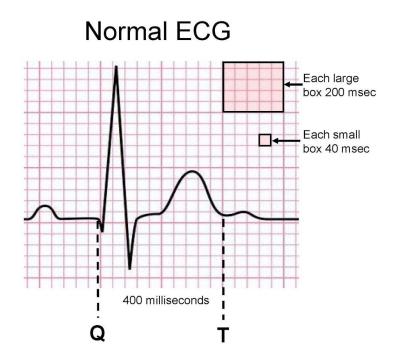
In fact, studies undertaken in Belgium and Germany have demonstrated that about 7% of their in-ICU cardiac arrests actually initiated as TdP which is more than are typically recognized.

NORMAL ECG

To interpret a normal electrocardiogram, remember that, at the standard paper speed of 10mm/sec, each of these large boxes represent about 200 ms of time. In the tracing shown below the QT starts with the first dotted line which marks the beginning of the QRS and goes to the end of the T wave spanning two large boxes or 400 ms. Since each large box is 200 ms, each small box is 40 ms. When one considers that a 60 ms

change is a clinically relevant increase, we are only talking about a small box and a half. Be mindful of how you read these and that you carefully mark the beginning of the Q and the end of the T.

Normal ECG

Each large box 200 msec

Each small box 40 msec

400 milliseconds

Q   T

Computerized systems accurately measure QT in most cases within 10 or 20ms. However, in some cases, particularly when there is a u- wave or a T-wave that's notched or slurred, your computerized system may not measure it correctly. In these instances, it is important that you are able to measure a QT accurately.

More information on best practices for measuring QT intervals can be found in the Tools section of this module's corresponding web page:
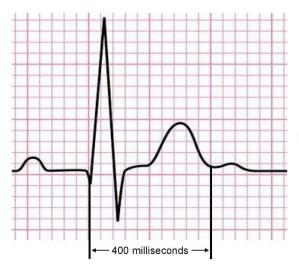
MODULE ONE TOOLS

Calculating QTc using the Bazett's Formula

The image below displays what the actual QTc calculation looks like in a particular case with a heart rate of 70 bpm

Calculating QTc using the Bazett's Formula

$$RR = \frac{60}{HR} = \frac{60}{70} = 0.857\,\text{sec}$$

$$QTc = \frac{QT}{\sqrt{RR}} = \frac{0.40}{\sqrt{0.857}} = 0.432 = 432\,\text{msec}$$

← 400 milliseconds →

Heart rate = 70 beats per min

The use of a web or app calculator for this is recommended.

To complete this module, please continue to the knowledge test section of this module's webpage for a short quiz that will test your knowledge of the presented information before advancing to Module Two, found at:

MODULE ONE KNOWLEDGE TEST

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