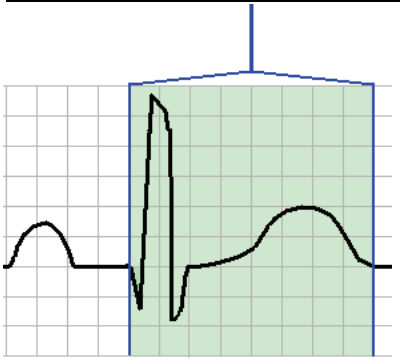
THE Q-T INTERVAL and U WAVES:



VARIES BASED ON PT'S HEART RATE and SEX

The Q-T Interval is measured from the beginning of the QRS complex to the end of the T wave. It represents the total duration of ventricular depolarization and repolarization. U waves, when present, follow T waves, and are most prevalent in the precordial leads V2 and V3.

ACCURATE QT Interval measurement is a critical component of ECG interpretation. Identification of abnormal prolongation of QT Intervals and/or the presence of abnormally large U waves – combined with appropriate patient management – could save the lives of thousands of patients each year. Long QT Syndrome (LQTS) claims the lives of approximately 3000 people per year,[9] many of whom are young, healthy children and adolescents. The mechanism of sudden death in patients with LQTS is development of Torsades de Pointes, a lethal cardiac dysrhythmia. By formal definition, Long QT Syndromes are characterized by: ***prolongation of the corrected QT interval (QTc),*** and ***clinical findings of syncope or aborted sudden death***. There is often a family history of syncope and/or sudden death.[10]

If diagnosed and properly managed, the incidence of mortality from LQTS could be reduced to nearly zero. Current medical literature indicates that the primary diagnostic finding for LQTS is a prolonged QT interval, which in most cases is readily identifiable on the ECG. In some cases, T wave alterans patterns have been reported. Other documented ECG abnormalities associated with LQTS include abnormally large U waves, and or T/U wave fusion.

After extensive review of patient case studies, current medical journal citations, and textbook descriptions of LQTS, we suggest the following guidelines for QT Interval and U wave evaluation:

☞ *Measure QT when patient at resting heart rate.*

Known ECG Indicators of Long QT Syndrome:

- QTc 460ms or longer in females*
- QTc 450ms or longer in males*
- T wave alterans
- U waves >25% of the T wave
- U waves merged with T waves
- U waves >0.1mv (1mm on standard calibrated
- ECG)

ECG Indication of Abnormally SHORT QT Interval:

- QTc less than 390ms

*P. Rautaharju, et al, "Standardization and Interpretation of the ECG, Part IV" JACC2009;53, no. 11:982-991

Etiology of Long QT Syndromes:

- **Congenital** (14 known subtypes) Genetic mutation results in abnormalities of cellular ion channels
- **Acquired**
  - Drug Induced
  - Metabolic/electrolyte induced
  - Very low energy diets / anorexia
  - CNS & Autonomic nervous system disorders
- **Miscellaneous**
  - Coronary Artery Disease
  - Mitral Valve Prolapse

Suspected LQTS Considerations include:

- Avoidance of Medications that are known to prolong the QT Interval. (refer to "Meds Known to Prolong QT Interval" table on next page).
- Immediate expert consultation, such as with cardiologist / electrophysiologist, in order to rule out LQTS
- Continuous ECG monitoring until LQTS ruled out, or until expert consultant deems it safe to discontinue continuous ECG monitoring

Since the ECG indicators of LQTS are usually easily identified on the 12 Lead ECG, it is imperative that all clinicians who interpret ECGs be familiar with these identifying characteristics. When indicators of LQTS are discovered, we recommend that Suspected LQTS Considerations, listed in the table to the above right, be observed.

[9] Fogoros, Richard N, MD, author of "Electrophysiologic Testing."

[10] Jorge McCormack, MD, FACC, "The Role of Genetic Testing in Paediatric Syndromes of Sudden Death," Cardiology in the Young 2009; 19 (supl. 2) 54-65, Cambridge University Press

A more in-depth discussion of LQTS is beyond the scope of this book. Therefore, we will present standard ECG guidelines and alert criteria for the measurement of QT Intervals and U Waves. In the appendix of this book, we provide three case studies where proper application of LQTS knowledge saved the life of a 15 year old boy, a 22 year old girl and her siblings, and that of a 52 year old man.

QT Interval measurement should be performed in leads where the QT Interval shows the longest; this lead is usually V2 or V3[11]

Normal QT Interval values are based on the patient's heart rate, and to a lesser extent, the patient's gender.

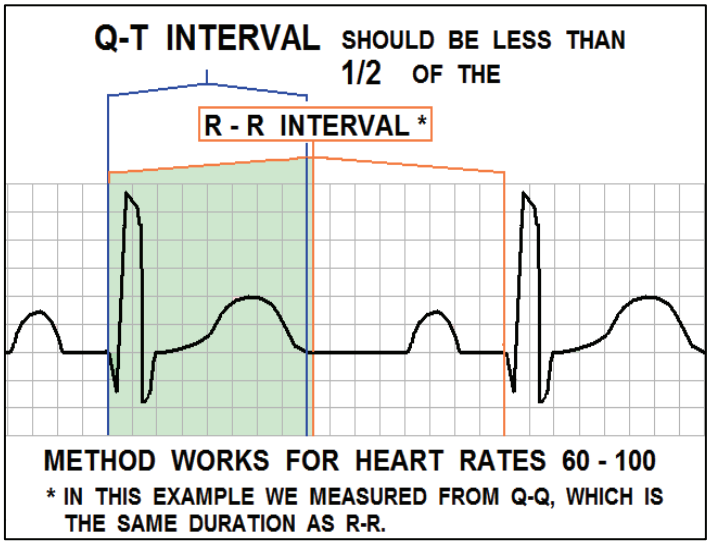
There are several acceptable ways to calculate the corrected Q-T Interval (QTc). One way is to refer to a chart, like the one seen to the right, which provides the patient's QTc, based on the heart rate and gender of the patient. This method is not always practical, unless you don't mind carrying a chart with you at all times. I'm sure by now, there's an "app" for it, however I haven't checked yet.

THE *QTc INTERVAL

*QTc = Q-T interval, *corrected* for heart rate

HEART RATE	MALE	FEMALE
150	0.25	0.28
125	0.26	0.29
100	0.31	0.34
93	0.32	0.35
83	0.34	0.37
71	0.37	0.40
60	0.40	0.44
50	0.44	0.48
43	0.47	0.51

FROM: *Annals of Internal Medicine, 1988 109:905.*



For heart rates between 60-100, use the "Quick Peek" method, shown to the left. This simple method is a favorite of the two electrophysiologists I work with on a regular basis. If it's good enough for them, it's good enough for me!

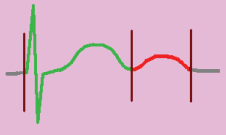
For heart rates less than 60 or greater than 100, or to calculate the precise corrected QT interval, you can use one of the following mathematical formulas, shown in the table below:

QT CORRECTION FORMULAS:

Bazett's	$QTc=QT/\sqrt{RR}$
Fredericia	$QTc=QT/(RR)1/3$
Framingham	$QTc=QT+0.154(1-RR)$
Rautaharju	$QTc=656/(1+HR/100)$

U WAVES:

THE U WAVE



REPORTED CAUSES OF U WAVES:

- *OFTEN SEEN IN BRADYCARDIAS (RATES BELOW 60)*
- *HYPOKALEMIA, HYPOCALCEMIA, HYPOMAGNESEMIA*
- *AFTER-DEPOLARIZATIONS of VENTRICULAR*
- *HYPOTHERMIA*
- *DRUGS THAT PROLONG THE QT INTERVAL*
- *LONG QT SYNDROMES*
- *REPORTED IN APPROX 15% of ISCHEMIC STROKES*

Occasionally, U waves are seen on an ECG. When present, they succeed T waves, which they typically resemble in shape and deflection. They are most prevalent in the precordial leads, most predominantly leads V2 and V3.

The etiology of U waves is most likely from several possible causes. Traditional thought attributes the U wave to "hypokalemia," however with respect to electrolytes, we know that hypocalcaemia, hypomagnesaemia, are also known to cause U waves. Therefore, when U waves are noted, the patient's electrolyte levels should be evaluated.

[11] P. Rautaharju, MD, FACC et al, "Standardization and Interpretation of the ECG, Part IV", JACC 2009; Vol 53 No. 11:982-991

Recall that serum magnesium does not accurately reflect intracellular magnesium. If your patient is exhibiting symptoms of low magnesium; for example is presenting with runs of Torsades, sub-normal ***intracellular magnesium levels*** may be present, despite the reporting of normal ***serum magnesium levels*** on the electrolyte panel[12].

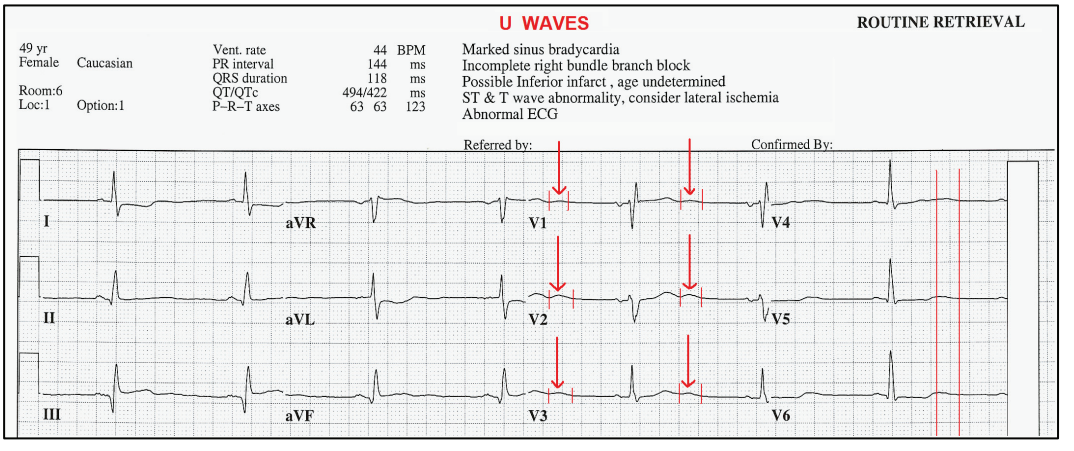
According to a study performed by Warren Jackman, et. Al., at the University of Oklahoma, U waves are indicative of "early afterdepolarizations," a condition linked to increased incidence of Torsades.

According to Marriott and Cooper[13], when U waves are seen with *deflections opposite that of T waves* (i.e. *positive* T waves with *negative* U waves), it is a sign of possible myocardial ischemia.

My response to seeing U waves on a patient's ECG is to rule out known causes of U waves:

- obtain a thorough patient history, to rule out incidence of syncope and family history of sudden death/ near sudden death.
- evaluate the patient's electrolyte levels, and
- evaluate patient's medications list for meds that prolong the QT Interval.
- rule out hypothermia
- rule out CVA
- monitor the patient's ECG for runs of Torsades.
- Expert consult for LQTS evaluation

The ECG featured to the left provides an example of U waves.



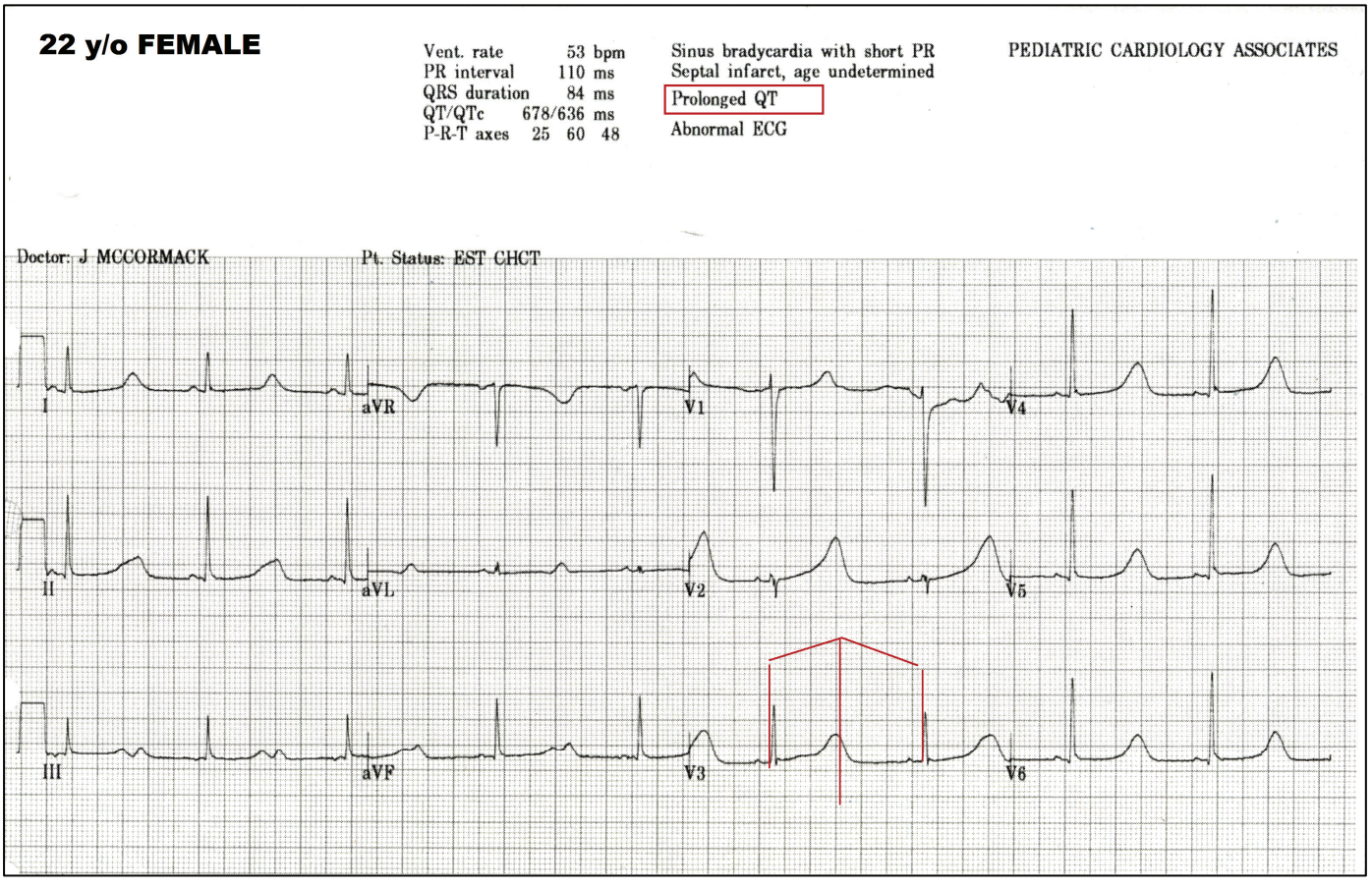
Prolongation of the Q-T interval and/or the presence of abnormal U waves has been linked to an increase incidence of Torsades de Pointes, a potentially fatal dysrhythmia. Patients with a prolonged QT interval should be monitored closely for the development of Torsades. Electrolytes should be checked, with specific emphasis on the patient's Magnesium, Potassium, Calcium and Sodium levels. **Patients with NORMAL serum Potassium levels may have intracellular hypomagnesaemia – therefore we DO NOT withhold administration of Magnesium Sulfate, when Torsades is present.** Abnormal electrolyte levels should be corrected. Patients in Torsades may have no cardiac output and may present in cardiac arrest. **Patients in Torsades who present in cardiac arrest should be managed using the American Heart Association ACLS algorithm for Ventricular Fibrillation. Patients in Torsades whom are hemodynamically unstable should receive an unsynchronized DC countershock, (defibrillation) as per ACLS protocol. Patients in Torsades, who are stable, per AHA ACLS guidelines, should receive Magnesium Sulfate 1 – 2 gm. over 5 to 60 minutes.**

BEWARE of giving MEDS that INCREASE the QT INTERVAL to patients with abnormally prolonged QT intervals and/or abnormal U waves – this practice can lead to TORSADES and CARDIAC ARREST. There are hundreds of medications known to prolong the QT interval, many of which are frequently used, such as Amiodarone, Avalox, Biaxin, Cardene, Corvert, Erythromycin, Geodon, Haloperidol, Levoflaxin, Lithium, Procainamide, Z-pak, Zithromax; many more are listed on page XX in the appendix of this book. Please visit: www.longQT.org and www.azcert.org for additional information, precautions and advice.

[12] Rinehart, R et al, Arch Intern Med 1988;148(11):2415-2420
[13] Cooper, J and Marriott, H, "ECG Essentials and Beyond," American College of Cardiovascular Nurses

LONG QT SYNDROME CASE STUDY 1:

The ECG below is that of a 22 year old female who was previously diagnosed with "epileptic seizure disorders." She was prescribed several antiseizure medications, none of which were effective in controlling her periodic seizure activity. After visiting several physicians, only one obtained a 12 Lead ECG, which is shown below. Using the "Quick Peek" method (above left), one can rapidly see that the patient's QT duration exceeds that of the R-R Interval!



This patient was referred to a local electrophysiologist specializing in the pediatric patient population, and is well known for his expertise in the area of Long QT Syndromes. During exercise stress testing, the patient developed Torsades. At this point, an AICD was implanted.

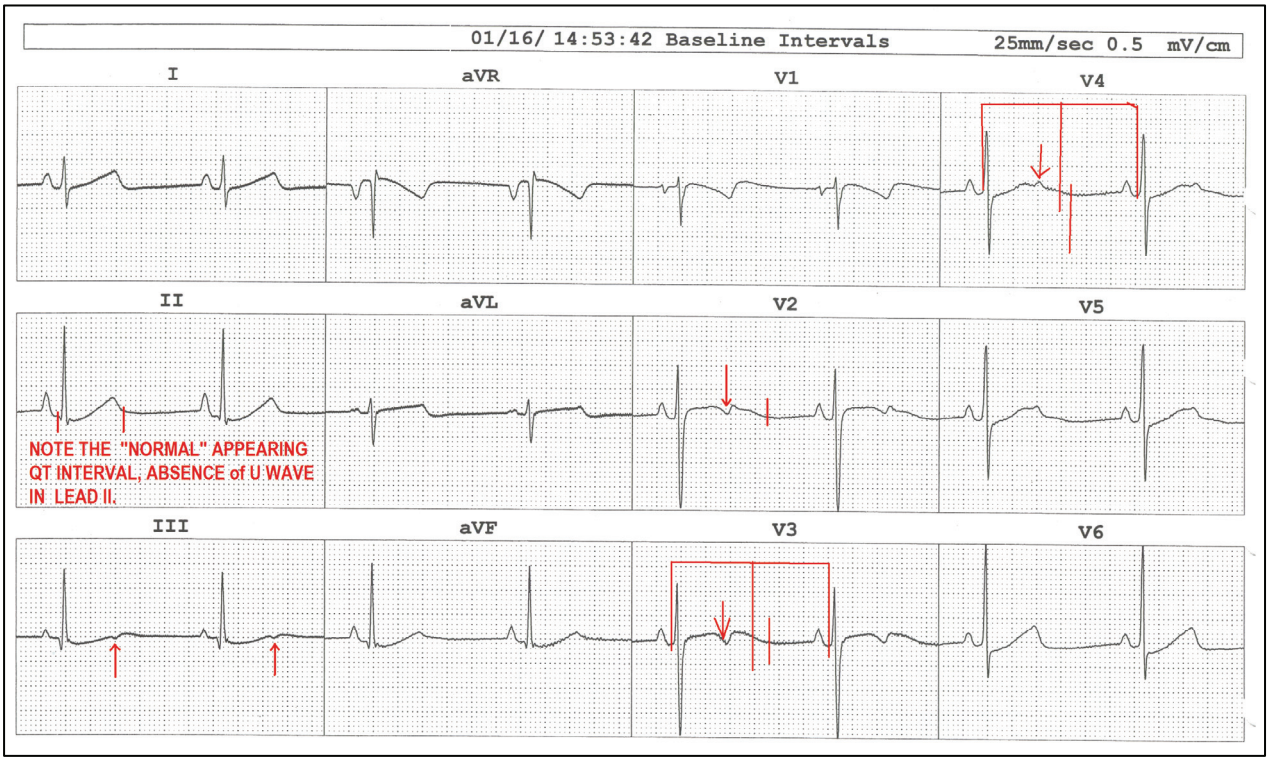
Subsequent genetic testing confirmed an abnormality of gene KCNQ1, confirming the physician's suspected diagnosis of LQTS Type I. It is significant to note that other members of her family, including a small child, suffered syncopal episodes, tested positive for Type 1 LQTS, and received AICDs. All family members are currently alive and well.

Cardiac events (Torsades, sudden death) in LQTS Type 1 are typically triggered by Exercise and exertional stress. Several incidents have been described as occurring during athletic events and while swimming.[111]

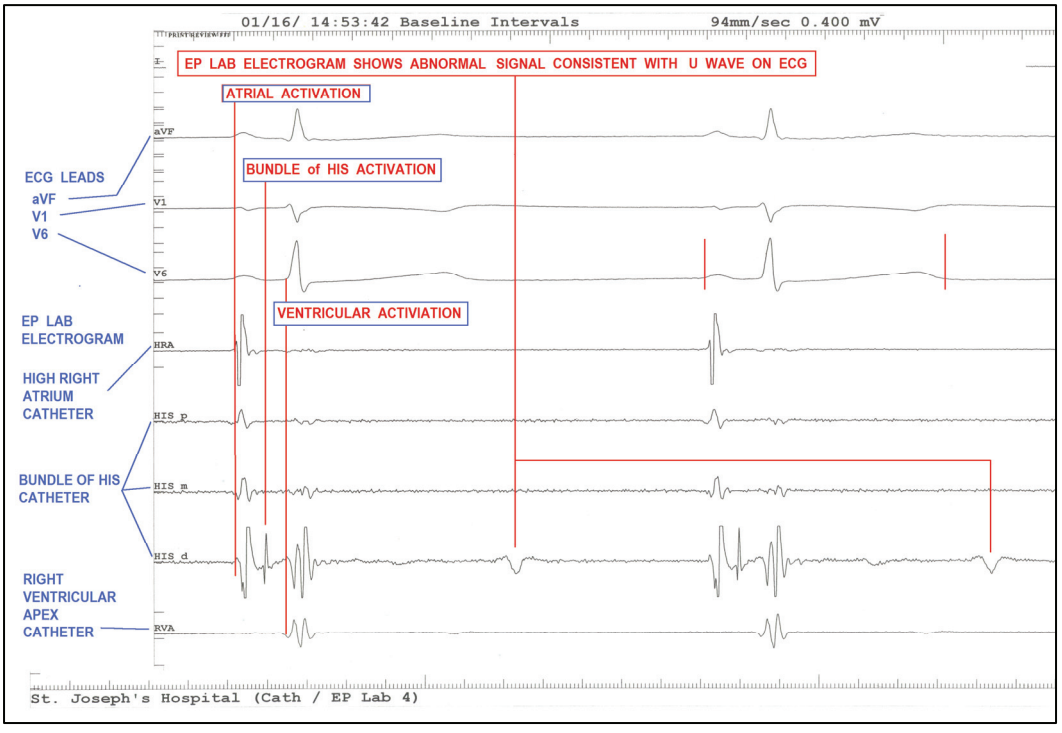
Case contributed by Jorge McCormack, MD, FACC

LONG QT SYNDROME CASE STUDY 2:

ECG of a 15 year old male who suffered sudden cardiac arrest. Immediate bystander CPR and application of AED resulted in successful resuscitation. Prior to the event, patient had no known history of any medical problems. There was a familial history of sudden death.

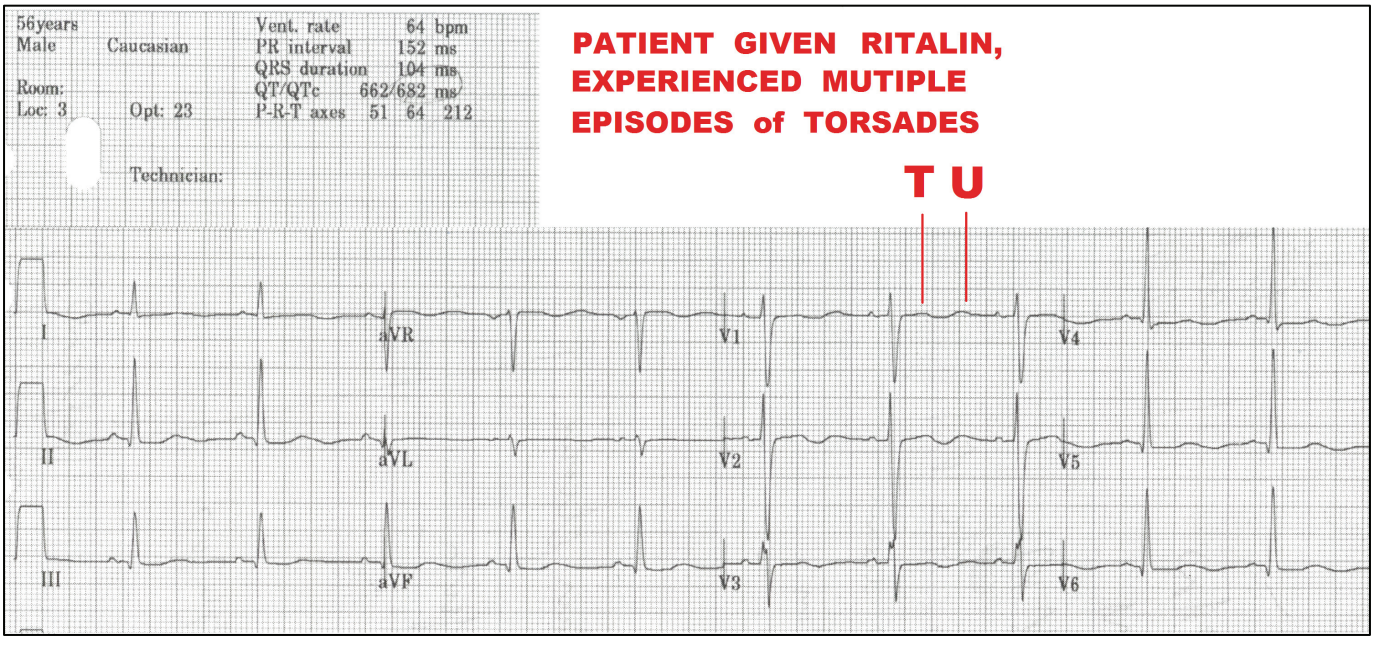


An EP study (shown below) revealed the presence of abnormal electrical signals originating in the ventricles consistent with timing of the U wave (or second half of the "notched" T wave), combined with positive genetic testing for LQTS Type 2 resulted in the patient receiving an AICD.

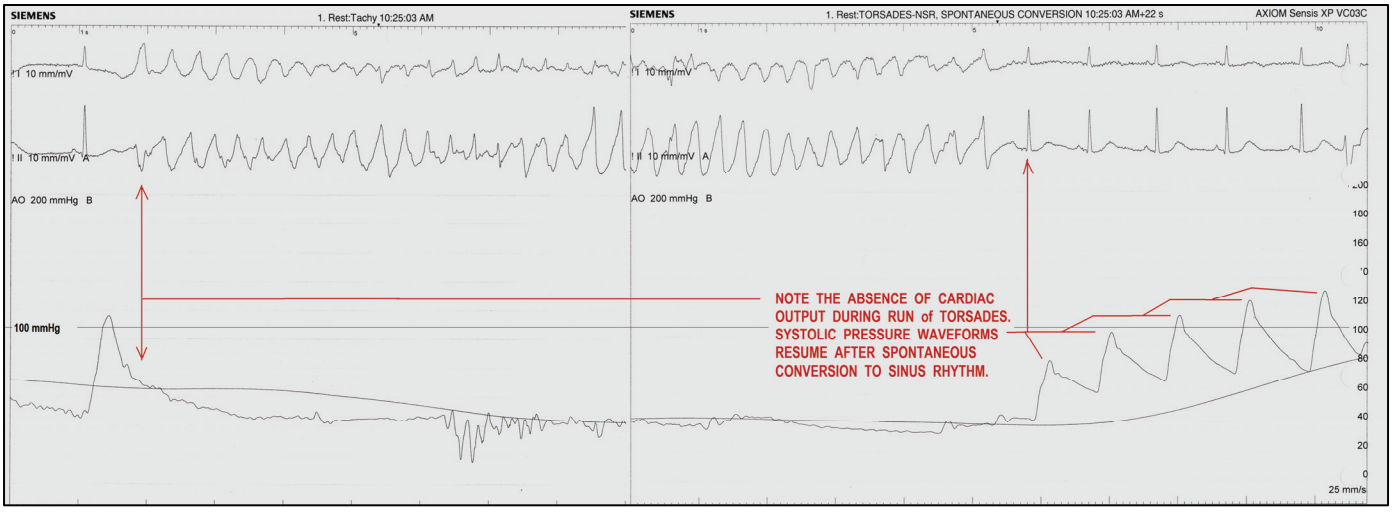


LONG QT SYNDROME CASE STUDY 3:

The ECG below is that of a 56 year old man with no previous history of cardiac disorders who suffered from episodes of syncope after being put on Ritalin. Note the prevalence of U waves in leads aVR, V1, V2 and V3. U waves of opposite polarity (positive T, negative U) are noted in leads V4, V5 and V6.



Part of his diagnostic workup included cardiac catheterization to rule out CAD. (U waves of opposite polarity from T waves are considered a sign of ischemia). During cardiac catheterization, he was found to have no obstructive CAD, however he suffered another episode of syncope – caused by run of Torsades, recorded on the Cath Lab's hemodynamic monitoring system.



This patient was found to have the Acquired form of Long QT Syndrome, induced by Ritalin, which is one of many medications known to increase QT intervals.

