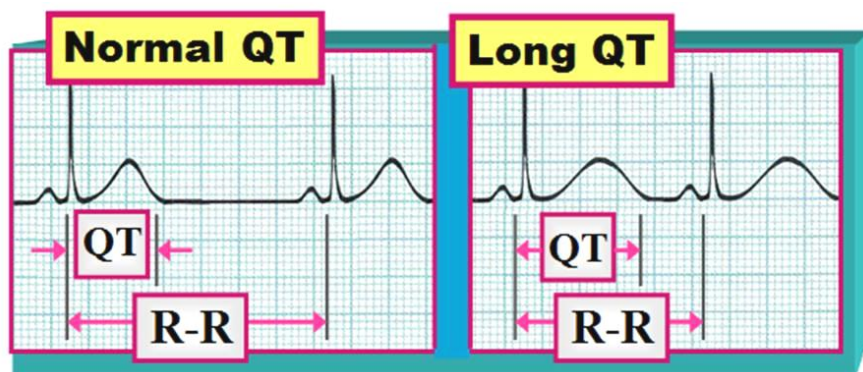




The QT Interval

The **QT interval** is the period that extends from the *beginning* of ventricular depolarization — until the *end* of ventricular repolarization (*Figure below*). For practical purposes, the QT is **prolonged** — IF it clearly measures **more** than **half** the R-R interval. The principal exception to this rule is when the heart rate is rapid (>90 - 100 beats/minute) — in which case it is more difficult to measure the QT and determine its clinical significance.

- **To measure the QT** — Choose a lead where you can clearly see the *end* of the T wave. *Select that lead in which the QT appears to be longest.*



► Is the QT prolonged?

- Answer — The QT interval is clearly *normal* on the left (*since the QT is much less than half the R-R interval*). In contrast — the QT is obviously *long* on the right (*it far exceeds half the R-R interval*).

Clinically — We want to know IF the QT is normal or long. This is usually easy to tell by the above **“eyeball method”** (ie, *Is the QT more than half the R-R interval?*) — provided that the heart rate is *not* excessively fast (*not over 90-100/minute*).

- Practically speaking — one *only* cares IF the QT interval is normal, borderline, or long. (**Hypercalcemia** produces **QT shortening** — but this is *difficult* to recognize clinically and is usually only seen with very high serum calcium values of >12 mg/dL.)
-

CAUSES of QT Prolongation

It is important to be aware of the **Causes** of **QT prolongation**. There are *many*. Fortunately — the most common causes can be divided into 3 basic categories = **“Drugs/Lytes/CNS”** (List #3).

Common Causes of QT Prolongation



♥ **Drugs** — Type 1A (quinidine, procainamide, disopyramide) and Type III (sotalol, dofetilide, amiodarone) antiarrhythmic agents;
— Tricyclic antidepressants/phenothiazines

♥ **“Lytes”** — Hypokalemia (or hypomagnesemia)
— Hypocalcemia

♥ **CNS** — CNS catastrophes (ie, stroke, seizure, coma, intracerebral or brainstem bleeding)

■ **NOTE** - Several *other* conditions (ie, bundle branch block, infarction, and ischemia) may *also* cause QT prolongation. However, the presence of these **other conditions** will usually be *obvious* from inspection of the ECG.

► **Note at the bottom of List #3:** — We pay *less* attention to the QT when the ECG picture is dominated by *other* findings (*such as acute MI/bundle branch block*). However — IF the *only* thing wrong on the tracing is a long QT — Think **“Drugs/Lytes/CNS”** as the cause!

- Some of the most *bizarre* ST-T wave abnormalities (*and some of the longest QT intervals*) may be seen in association with various **CNS catastrophes** (ie, *coma, stroke, seizure, or bleed*). Resultant ST segment elevation that may be seen with CNS catastrophes at times may *mimic* the changes of *acute* MI.
-

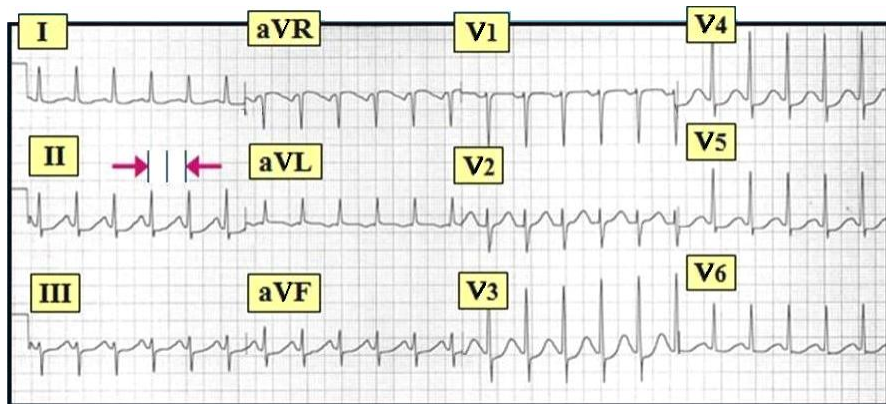
Why Care About the QT? — The answer is to hopefully prevent the rhythm below:



- **Answer:** The rhythm in the above Figure is **Torsades de Pointes** (“twisting” of the points) — so named because of *alternating polarity* of the QRS with respect to the ECG baseline. Torsades is most often associated with a **long QT** — with a *preceding* period of bradycardia (*a long preceding R-R interval sets up ensuing QT prolongation*). **Treatment** — Address and hopefully correct the cause of the long QT; Magnesium (1-2gm IV, which may need to be repeated). **Note** — Torsades $\neq$ VT!

 REVIEW:

Tracing O — Comment on the Rate, Rhythm, and Intervals for the 12-lead ECG shown below:



► **Answer** to **Tracing O** — The Rhythm is rapid and regular. It appears that the R-R interval is approximately 2 large boxes, so that the Rate is ~150/minute. The QRS is narrow and *upright* P waves are seen in lead II with a fixed PR interval. The rhythm is therefore **sinus tachycardia**.

Re Intervals — *both* the PR and QRS intervals are normal. The **QT interval** is difficult to measure because of the *rapid* rate. That said — in at least several leads (*especially* leads V3-V5) — it looks like the QT is clearly *much more* than half the R-R interval. Thus *despite* the rapid rate — We *strongly* suspect **QT prolongation**. List #3 helps us recall the *likely* reasons why (ie, “*Drugs-Lytes-CNS*”). Clinical correlation is needed to determine which of these may be relevant for this patient. **NOTE:** We would not use leads I,aVL,V1 to measure the QT (*because we can’t see the end of the QT in these leads*).

Bottom Line — Think “*Drugs-Lytes-CNS*” whenever you see a long QT in the *absence* of MI/ischemia/BBB.