The importance of routine QT interval measurement in rhythm interpretation

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Abstract

When monitoring a patient's cardiac rhythm, the QT interval should be routinely measured. A variety of factors can prolong the QT interval such as drug effects, electrolyte imbalances, acute myocardial infarction, and congenital factors. The biggest risk with prolongation of the QT interval is the development of Torsades de Pointes. One obstacle to the routine measurement of the QT interval is the lack of a standardized and simple approach. The purpose of this article is to detail factors that prolong the QT interval and describe methods used to measure the QT interval.

Torsades de Pointes (TdP) is a form of polymorphic ventricular tachycardia specifically associated with a prolonged QT interval that may be fatal if not immediately treated (American Heart Association, 2005). The classic electrocardiographic changes associated with TdP include a wide complex QRS tachycardia, and a beat-to-beat axis deviation of the QRS complexes around the baseline that is referred to as the 'twisting of the points' (Landen, Schmidt, & Poponick, 2007). The significance of a prolonged QT interval is that as the QT interval gets longer, the risk exists that the next cardiac impulse may occur and strike at the vulnerable relative refractory period, also referred to as the 'R on T' phenomenon (Kass & Moss, 2003). The relative refractory period occurs at the peak of the T wave to the latter portion of the T wave. (See Figure One). If any impulse occurs during the relative refractory period, the cardiac rhythm is in jeopardy of developing a tachyarrhythmia such as polymorphic ventricular tachycardia deteriorating to ventricular fibrillation. (See Figure Two). The QT interval is an indirect measurement of ventricular repolarization and is measured from the beginning of the QRS complex to the end of the T wave on the electrocardiogram (Drew, 2005) (See Figure Three). In this article, the author will identify the practice gap that exists among nurses regarding QT interval measurement, discuss contributing factors that potentially prolong the QT interval, and detail two methods used to measure the QT interval. This article refers to both the adult and pediatric population.

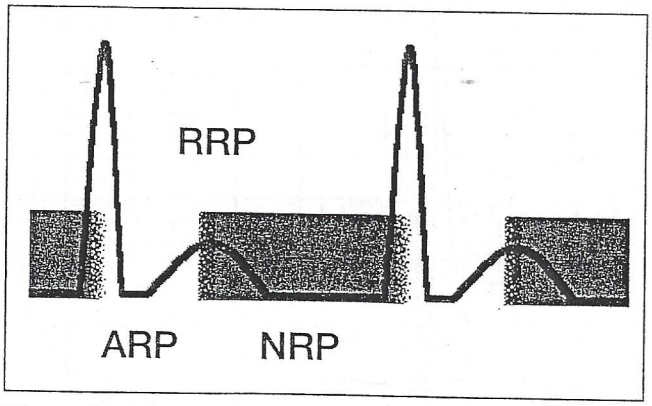

Figure One: ARP= Absolute refractory period. ARP is the time when myocardium is unresponsive to any stimuli. RRP= Relative refractory period. RRP is the time when a stimulus might induce a depolarization. NRP=Non-refractory period. NRP is the time when a stimulus can depolarize myocardial tissue.

Practice gap

There is a practice gap among physicians when measuring QT interval and there is a significant amount of controversy on an established and reliable method of QT interval measurement (Al-Khatib et al., 2005; LaPointe, Al-Khatib, Kramer, & Califf, 2003). Al-Khatib et al. (2005) concluded from a survey distributed to 826 internal medicine and psychiatry physicians attending conferences at six academic institutions in the United States that the majority of physicians could not correctly measure the QT interval and could not correctly identify factors and medications that can prolong the QT interval.

Specific to nursing, there is a paucity of information related to practice patterns and guidelines for QT interval measurement. A review of the best practice guidelines project developed by

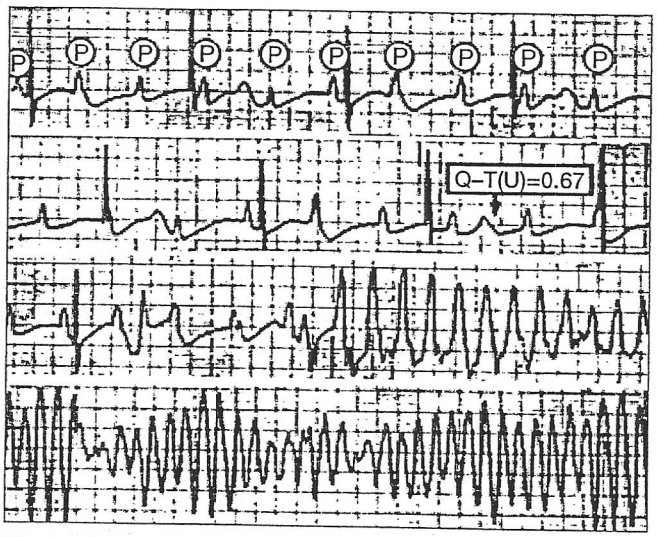

Figure Two: 49-year-old female presenting with syncope. Ventricular rate 36/min with a complete AV Block, progressing to torsades de pointes.

the College of Nurses of Ontario and the Registered Nurses of Ontario resulted in no published guidelines on caring for the patient on a cardiac monitor or on QT interval measurement (CNO, 2006). A review of the Canadian Nurses Association and the Canadian Association of Critical Care Nurses also resulted in no published practice guidelines for QT interval measurement.

In 2005, the American Heart Association (AHA) developed guidelines for electrocardiogram and cardiac monitoring based on clinical experience and related research in the field of electrocardiography. The rationale for basing these guidelines on clinical experience rather than evidence-based research was because randomized clinical trials in this area were almost nonexistent (Drew, 2005). The AHA practice standards statement is a consensus document prepared from councils on cardiovascular nursing, clinical cardiology, and cardiovascular disease in the young, and is endorsed by the International Society of Computerized Electrocardiology and the American Association of Critical Care Nurses. According to Drew, the AHA statement represents the first attempt to encompass all areas of hospital cardiac monitoring including QT interval measurement in children and adults. The AHA recommends the QT interval should be measured in patients who have received any antiarrhythmic drug known to cause TdP, overdosed from a potentially proarrhythmic agent, developed new-onset bradyarrhythmias, severe hypokalemia or hypomagnesemia, require antipsychotics, and experienced an acute neurological event (Drew, 2005). In these patients, QT interval monitoring is a high priority if the patient has risk factors for TdP.

With no established Canadian evidence-based guidelines on QT interval measurement, standards on QT interval measurement have been implemented on a hospital-by-hospital basis. To explore this, the author developed a survey to determine the frequency of QT interval measurement and identify reasons why QT interval measurement was not being done. Between September and November 2007, 180 nurses working with patients requiring cardiac monitoring or on telemetry completed a five-question survey. These surveys were conducted with nurses working at three Toronto-area hospitals and four hospitals in the Vancouver area, as well as five hospitals in the British Columbia interior. When asked how often the QT interval is assessed, 30% of the nurses responded they always measure this interval, 45% responded they sometimes measure this interval, and 25% responded they never measure this interval. For nurses who responded they sometimes or never measure the QT interval (70%), the majority of the participants reported the main reason for not measuring this interval routinely was because of a lack of knowing how to measure or not understanding the significance of why to measure it.

There are a wide variety of factors that can cause a prolonged QT interval. Therefore, all rhythm strips and electrocardiograms should include routine measurement of the QT interval. Because there are many non-cardiac medications and other factors that may contribute to the development of a prolonged QT interval and potential TdP, it is far safer to adopt a practice of measuring the QT interval every time a patient's rhythm is analyzed. According to Landen et al. (2007), a complicating factor in predicting TdP is that there is no direct correlation between the length of the QT interval and the incidence of the arrhythmia so the unpredictability in any given patient warrants vigilance of QT interval measurement. From a report published by Kowalski, Kenigsberg, Sinno, Krishnan and Khanal (2006), antiarrhythmic medications (AAM) are associated with an increase in cardiac mortality due to their proarrhythmic effects and, from a meta-analysis of 16,365 patients, AAM were also associated with an increase in non-cardiac mortality.

Contributing factors for prolonged QT interval

There are many medications that are known to potentially prolong the QT interval including antiarrhythmics, antibiotics, non-sedating antihistamines, antipsychotics, antidepressants and others (Al-Khatib, LaPointe, Kramer, & Califf, 2003; Kass & Moss, 2003; The University of Arizona Center for Education and Research on Therapeutics, 2005). Refer to Table One for examples of drugs that can prolong the QT interval. The antiarrhythmic medications that are given to patients to prevent potentially dangerous arrhythmias have proarrhythmic properties. Proarrhythmic properties mean that these medications may cause arrhythmias due to their ability to alter the ion channels, which then extends the ventricular repolarization phase resulting in a prolonged QT interval, possibly leading to TdP (Al-Khatib et al., 2003).

Electrolyte imbalances play a major role in the development of TdP including hypomagnesemia, hypokalemia and hypocalcemia (Al-Khatib et al., 2003; Kass & Moss, 2003; Ruskin, 2005). The QT interval is dependent on a normal flow of ions in and out of cardiac cells. A rapid inflow of sodium and calcium, which are positive ions, results in depolarization. Repolarization results when the inflow of sodium and calcium is exceeded

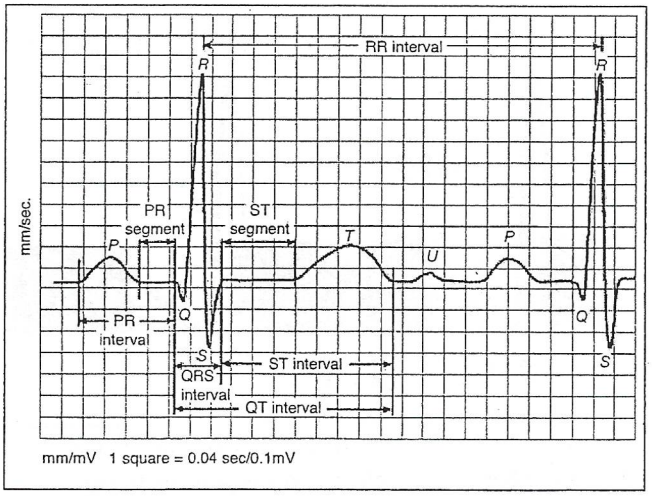

Figure Three: The QT interval is measured from the start of the QRS complex to the end of the T wave. Figure retrieved March 6, 2008, from http://www.merck.com/mmpe/sec07/ch070/ch070e.html. *Used with permission.*

by the outflow of potassium. Magnesium is a crucial cofactor in the sodium-potassium adenosine triphosphase (Na/K-ATPase) system. Any disturbance in these electrolytes extends ventricular repolarization resulting in a potentially prolonged QT interval (Al-Khatib et al., 2003; Urden, Stacy, & Lough, 2002).

An acute myocardial infarction is another factor that affects the QT interval (Kenigsberg, Khanal, Kowalski, & Krishnan, 2007). According to results of a prospective study conducted by Kenigsberg et al. with 74 patients, the QT corrected (QTc) interval lengthened in early transmural ischemia in 100% of the cases. Comparatively, T-wave changes, ST segment elevation, or ST depression occurred in 7%, 15%, and 7% respectively in early transmural ischemia, concluding that QTc is the first electrocardiographic abnormality in early transmural ischemia (Kenigsberg et al., 2007). The reason the QTc prolongs in early transmural ischemia relates to prolonged local recovery time in the area of ischemia (AHA, 2007).

Since repolarization normally proceeds in an epicardial-to-endocardial direction, delayed recovery in the subendocardial region due to ischemia does not reverse the direction of repolarization, but merely lengthens it. This generally results in a prolonged QT interval or increased amplitude of the T wave or both (AHA, 2007, n.p.).

A prolonged QT interval has also been observed in patients who present with hypothermia or have hypothermia induced as medical management. The physiology of how the QT interval prolongs in hypothermia has not been well-described other than causing a delayed repolarization, which prolongs the QT interval (Al-Khatib et al., 2003).

According to Al-Khatib et al. (2003), other conditions that cause a delay in repolarization, thus potentially causing a prolonged QT interval, are congestive heart failure, myocardial contusion, myocarditis, mitral valve prolapse, chronic alcohol use, organophosphorus insecticide poisoning, subarachnoid hemorrhage, bradycardia, hepatic dysfunction, starvation diet, and radical neck surgery. In cases where the QT interval is prolonged or is getting longer, the nurse needs to inform the physician and monitor the patient closely. Even though treatment is not specifically geared to correcting the QT interval, alleviation of the causative factor will cause the QT interval to return to normal in most situations (Al-Khatib et al., 2003).

Another factor that may contribute to the development of prolonged QT interval and TdP is congenital long QT syndrome (LQTS). LQTS is a rare, usually inherited disorder affecting the heart's electrical system and is associated with ventricular tachyarrhythmias, syncope and sudden death (Kass & Moss, 2003; Makielski, 2006; Zhang, Timothy, & Vincent, 2000). The more common form of LQTS is Romano-Ward syndrome and is estimated to occur in about one in 5,000 people (Kass & Moss, 2003; Mayo Clinic, 2006). Scientists are continuing to investigate the link between sudden infant death syndrome (SIDS) and long QT syndrome and suspect that a small percentage of babies with SIDS had LQTS (Priori et al., 2003). About one-third of people with LQTS have no symptoms. A warning signal for a health care provider to become suspicious of LQTS is in a young person presenting with syncope, palpitations or ventricular tachyarrhythmias, especially if there is a family history or any of the following: LQTS, sudden and unexplained fainting, death in newborn infants, children or young adults (Daley, Tranebjaerg, Samson, & Green, 2004; Kass & Moss; Makielski; Priori et al.). LQTS is often detected through routine ECG or cardiac monitoring. However, if the QT interval is within normal limits and the physician still suspects LQTS, an exercise stress test or a pharmacological stress test may be done. Treatment of LQTS depends on the patient, the family history, and the type of LQTS. Many times, treatment with high-dose beta blockers will alleviate symptoms of syncope in about 70% of patients with LQTS (Al-Khatib et al, 2003; Locati, et al., 1998; Priori et al.). Permanent pacing in combination with beta blockers may also be effective in managing symptoms. In cases where the patient is at high risk (cardiac arrest, premature death of family member, syncope) or is resistant to first-line therapy, a cardioverter defibrillator can be implanted (Priori et al.).

QT interval measurement

The first step in assessing whether the QT interval is within normal limits or prolonged is to identify the QT interval. The QT interval is measured from the start of the QRS complex to

Table One: Examples of drugs affecting QT interval

Class 1: Drugs that are accepted by authorities to have a risk of prolonging QT interval and TdP	Class 2: Drugs that in some reports may be associated with TdP	Class 3: Drugs to be avoided for use in patients with diagnosed or suspected congenital long QT syndrome. (With these patients, drugs on Class 1, 2 and 4 should also be avoided)	Class 4: Drugs that are weakly associated with TdP and/or QT prolongation but are unlikely to be a risk for TdP when used in usual recommended dosages and in patients without other risk factors*.
amiodarone, biaxin chloroquine, chlorpromazine cisaprid, clarithromycin, disopyramide, domperidone, haldol, mellaril, methadone, procainamide, sotalol	chloral hydrate, indapamide, lithium, serevent, ventolin, zithromax	cocaine as local anaesthetic, dexatrine, dobutamine, dopamine, ephedrine, epinephrine, levophed, appetite suppressant phentermine, dexatrim®, acutrim®, sibutramine, ritilin, ventolin	amitriptyline, ampicillin, bactrim, ciproflaxin, celexa, doxapin, ketoconazole, paxil, prozac, sulfa, tamoxifen, zoloft

the end of the T wave. (See Figure Three). Choose a lead on the ECG that offers good visualization of the start of the QRS complex and the end of the T wave. The next step is to visualize this QRS complex and the QRS complex immediately to the right. For heart rates ranging from 60 to 100 beats per minute, a QT interval is normal if it is less than half the distance between these two QRS complexes (Normal QT=<? R-R) (Luo, Michler, Johnston, & Macfarlane, 2004; Urden et al., 2002). (See Figure Four). This formula can be used as a general reference to determine if the QT interval is within the normal limits.

Because heart rate affects the QT interval, practitioners may also use a formula to determine the QTcorrected (QTc). Therefore, for heart rates slower than 60 or faster than 100 beats per minute, the QTc should be measured.

Currently, there is a lack of standardization and lack of data regarding the best way to adjust for heart rate (Al-Khatib et al., 2003; Al-Khatib et al., 2005; Drew, 2005; Luo et al., 2004). Drew (2005) notes that a normal QTc is <0.46 seconds in women and <0.45 seconds in men, and a QTc> 0.50 seconds in either sex has been linked to a higher risk for TdP. There are different methods that can be used to correct the QT interval to the heart rate. The most common formula used is the Bazett's formula where QTc= QT interval divided by the square root of the R-R interval measured in seconds (Al-Khatib et al., 2003; Drew, 2005; Luo et al., 2004). This formula, however, has been criticized because some evidence exists that the formula overcorrects the QT interval at fast heart rates and undercorrects it at low heart rates (Drew, 2005).

Other obstacles that make accurate measurement of the QTc difficult are the presence of atrial fibrillation and a wide QRS complex. In the presence of atrial fibrillation, there is no consensus on how to measure QT interval (Al-Khatib et al., 2003). Some clinicians advise averaging the QT interval over 10 beats, whereas others prefer to measure the QT intervals that follow the shortest and longest R-R intervals and divide each QT interval by the square root of the R-R interval preceding it (Al-Khatib et al., 2003). In the presence of a wide QRS complex, there are no consensus guidelines on how to measure the QT or QTc interval.

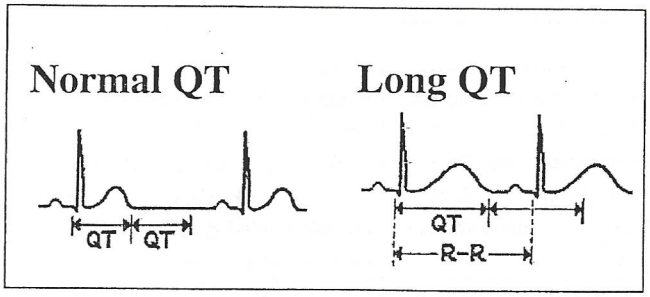

Figure Four: Normal QT. The QT interval on the left is within the normal limit because it is measured to be within half the R-R interval as indicated by the arrows. Long QT: the QT interval on the right is prolonged; note the QT interval extends beyond the half-way mark of the R-R interval.

Conclusion

As a nurse caring for a patient who is on a cardiac monitor, there are three messages to take from this article. First, because there are many factors that play a role in the development of a prolonged QT interval, which could potentially cause TdP, measurement of the QT interval with all rhythm strips and electrocardiograms should become routine practice. Second, when measuring the QT interval, there is a lack of consensus about how the QT interval measurement should be done, what QT interval threshold should be considered dangerously long, and what is the best correction formula. The author advises and prefers using the method that measures the QT interval as less than half of the R-R interval if the heart rate is between 60 and 100 beats per minute. In settings of atrial fibrillation, tachycardia or bradycardia, the QTc needs to be measured. Drew (2005) suggests that if a health care professional is uncertain on how to measure QTc, to record a 12-lead electrocardiogram (ECG). Standard ECG algorithms provide both uncorrected and corrected QT intervals. If the computer measurement of the uncorrected QT interval is confirmed by manual measurement, then health care professionals can trust the corrected value of the algorithm. Third, despite the method used to measure QT interval, all subsequent rhythm and ECG records should be compared against the baseline QT interval. If the nurse observes a lengthening in the QT interval, the physician should be informed. As nurses working with patients who are on a cardiac monitor or have electrocardiograms performed, we play an important role in monitoring the patients and being proactive in our assessment skills. Measurement of the QT interval should be as critical as measurement of the PR and QRS intervals.

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