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AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram

Part IV: The ST Segment, T and U Waves, and the QT Interval

A Scientific Statement From the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society

Endorsed by the International Society for Computerized Electrocardiology

Pentti M. Rautaharju, MD, PhD; Borys Surawicz, MD, FAHA, FACC; Leonard S. Gettes, MD, FAHA, FACC

The present article is the fourth in a series of 6 documents focused on providing current guidelines for the standardization and interpretation of the electrocardiogram (ECG). The project was initiated by the Council on Clinical Cardiology of the American Heart Association. The rationale for this project and the process for its implementation were described earlier (1).

Abnormalities in the ST segment, T wave, and duration of the QT interval reflect abnormalities in ventricular repolarization. These abnormalities are common and often difficult to interpret. The U wave most likely represents an electric-mechanical phenomenon that occurs after repolarization is completed. However, it is frequently included in discussions of repolarization and is discussed in this section.

The ST segment corresponds to the plateau phase of the ventricular transmembrane action potential. Under normal conditions, the transmembrane voltage changes slowly during this phase and remains at approximately the same level in all ventricular myocardial cells. As a result, only small voltage gradients are present. This absence of pronounced voltage gradients is similar to that which occurs during electric diastole, that is, from the end of repolarization to the onset of the next depolarization, when ventricular myocardial cells are at their resting transmembrane potential of approximately −85 mV. This corresponds to the TP segment on the ECG. The absence of significant voltage gradients in ventricular myocardial cells during these 2 phases of the cardiac cycle explains why the ST and TP segments are normally nearly flat and at approximately the same level; that is, they are isoelectric.

The T wave corresponds to the phase of rapid ventricular repolarization (phase 3) of the ventricular action potential.

Other members of the Standardization and Interpretation of the Electrocardiogram Writing Group include James J. Bailey, MD; Rory Childers, MD; Barbara J. Deal, MD, FACC; Anton Gorgels, MD; E. William Hancock, MD, FACC; Mark Josephson, MD, FACC, FHRS; Paul Kligfield, MD, FAHA, FACC; Jan A. Kors, PhD; Peter Macfarlane, DSc; Jay W. Mason, MD, FAHA, FACC, FHRS; David M. Mirvis, MD; Peter Okin, MD, FACC; Olle Pahlm, MD, PhD; Gerard van Herpen, MD, PhD; Galen S. Wagner, MD; and Hein Wellens, MD, FAHA, FACC.

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During this phase, the transmembrane action potential repolarizes from its plateau voltage of approximately 10 to −10 mV to its resting level of approximately −85 mV. The interventricular and intraventricular voltage gradients created as the cells undergo rapid sequential repolarization generate the T wave on the body surface ECG. The configuration of the T wave is determined by the spatial-temporal characteristics of ventricular repolarization, particularly the asynchrony of phase 3 of the ventricular action potentials. Our knowledge of these characteristics is still incomplete. In general, repolarization proceeds from epicardium to endocardium, that is, opposite to the direction of ventricular depolarization (2,3), and probably, like during excitation, a significant fraction of simultaneous repolarization wave fronts are mutually canceled. The difference in the spatial sequence of depolarization and repolarization in the left ventricular free wall reflects the observation that there tends to be an inverse relationship between activation time and action potential duration (4). The action potential duration of epicardial cells is shorter than that of the endocardial and midmyocardial cells (5). In addition, it is known that inhomogeneities of repolarization occur over relatively short distances on the surface of the ventricles and most probably also within the ventricular wall (5,6). It is probable that some of these inherent action potential differences are the result of electrotonic interactions during repolarization (7).

Abnormalities in the ST segment and T wave are caused by abnormal voltage gradients during the plateau and rapid repolarization phases of the action potential and by changes in the sequence of repolarization that may occur both with and without abnormal voltage gradients. These abnormalities are often associated with a variety of well-defined anatomic, pathological, physiological, and pharmacological events.

In this section, we address several issues relative to the measurement, description, and interpretation of ST segment, T and U waves, and QT interval. They include the distinction between primary and secondary repolarization abnormalities, appropriate descriptive and interpretive terminology, and measurement of the QT interval and its adjustment for rate, gender, and QRS duration.

Distinction Between Primary and Secondary Repolarization Abnormalities

Abnormalities in the ST segment and T wave, which are the result of changes in the shape and/or duration of the repolarization phases of the transmembrane action potential and occur in the absence of changes in depolarization, are referred to as primary repolarization abnormalities. They may be localized or diffuse and may be caused by a variety of events, including ischemia, myocarditis, drugs, toxins, and electrolyte abnormalities, particularly abnormalities of serum calcium and potassium. An abrupt change in heart rate, hyperventilation, changes in body position, catecholamines, sympathetic stimulation or ablation of the stellate ganglion, and temperature changes also can cause primary repolarization abnormalities (8,9).

Abnormalities in the ST segment and T wave that occur as the direct result of changes in the sequence and/or duration of ventricular depolarization, manifested electrocardiographically as changes in QRS shape and/or duration, are referred to as secondary repolarization abnormalities. These changes do not require changes in the shape or duration of phase 2 and phase 3 of ventricular action potential of individual cells. Rather, they may be due to voltage gradients that are normally largely canceled but become manifest when the changes in the sequence of depolarization alter the repolarization sequence. The ST- and T-wave changes that occur in association with bundle-branch blocks, ventricular preexcitation, and ectopic and paced ventricular complexes are examples of secondary repolarization abnormalities.

The classic ventricular gradient concept introduced by Wilson et al (10) in 1931 is of some theoretical interest concerning primary versus secondary repolarization abnormalities. Ventricular gradient in a single ECG lead is the net time integral of the ECG voltage from the beginning of the P wave to the end of the U wave. Its spatial counterpart is the ventricular gradient vector determined from the orthogonal XYZ leads. The practical utility of the ventricular gradient in differentiating primary from secondary repolarization abnormalities has not been demonstrated (11). When the direction of the QRS axis is normal, an abnormal direction of the T-wave axis is generally an indication of primary repolarization abnormalities.

Recognition of secondary repolarization abnormalities is usually not difficult. In left bundle-branch block, the ST- segment and T-wave vectors are generally directed opposite to the mean QRS vector. In right bundle-branch block, they are directed opposite to the slow terminal component of the QRS complex. In ventricular preexcitation, ST-T changes are directed opposite to the delta wave of the QRS complex. The magnitude of the ST-T change is dependent on the magnitude of the QRS-waveform changes when the excitation pathways change.

The secondary ST- and T-wave changes associated with transiently altered ventricular conduction such as those that occur with ectopic ventricular complexes or transient bundle-branch blocks usually revert promptly to the pattern that existed before the ventricular conduction changes developed. However, some secondary repolarization changes take longer (hours or days) to develop and to dissipate. The repolarization changes associated with prolonged ventricular pacing are examples of this phenomenon (12).

Primary and secondary repolarization abnormalities may occur concurrently. For example, ventricular hypertrophy is associated with changes in the shape and duration of the ventricular action potential of isolated ventricular cells, particularly on the endocardial surface (13). These changes may contribute to ST- and T-wave changes and are independent of the changes that are secondary to QRS-amplitude changes and prolongation of the QRS complex. A combination of primary and secondary repolarization abnormalities should also be considered when T-wave polarity does not change as anticipated by the changes in the QRS complex.

Recommendation

The distinction between primary and secondary repolarization abnormalities is clinically relevant because primary abnormalities indicate changes in the repolarization characteristics of ventricular myocytes whereas secondary changes do not. The designation of the ST- and T-wave abnormalities as primary or

secondary is appropriate, and it is recommended that automated interpretative algorithms be programmed to identify them.

ST-Segment Abnormalities

ST- and T-wave amplitudes are referenced against the TP or PR segments of the ECG. When low-frequency filtering is done in ECG acquisition to remove baseline drift, the actual DC voltage levels of various ECG waves or segments cannot be determined. Thus, elevation of the ST segment may reflect PR/TP depression, true ST elevation, or both; conversely, ST depression may reflect PR/TP elevation, true ST depression, or both (14–16). When considering deviations of the ST segment, one should bear in mind total QRS amplitude because this variable also affects the amplitude of ST-segment abnormalities. Displacement of the ST segment is usually measured at its junction with the end of the QRS complex, the "J point," and, in some settings such as exercise testing, 40 and up to 80 ms after the J point. The ST segment can be described as elevated, depressed, upsloping, horizontal, or downsloping. In addition, the magnitude of abnormal deviations and the leads showing them should be identified. A depressed ST segment may be further characterized as horizontal, downsloping, or upsloping (rapidly or slowly).

Elevation of the ST segment in leads V_1, V_2, and V_3 should be referenced against the elevation that occurs normally in these leads and is greater in young and middle-aged males than in females (17–21) and greater in blacks than in whites (21). ST elevation is usually most pronounced in chest lead V_2. The upper normal limit for J-point elevation in V_2 varies to a certain degree in various reference sources, probably largely as a result of differing selection criteria for the normal group. One reference source (20) reports the upper 98th percentile normal limit as approximately 0.3 mV in white men less than 40 years of age (up to 0.33 mV in the 24- to 29-year age group) and approximately 0.25 mV in white men 40 years old and older. The corresponding limits for white women remained relatively unchanged with age, staying at approximately 0.15 mV.

Another reference source (21) lists normal limits for J-point and ST_{60} (ST at 60 ms past the J point) amplitudes for white and black men and women 40 years old and older in 2 age groups. The upper normal limit (98th percentile) for J-point amplitude in V_2 was approximately 0.15 mV in white men and 0.20 mV in black men. The corresponding limits were approximately 0.10 mV for white women and 0.15 mV for black women. The upper normal limits for ST_{60} in V_2 were approximately 0.3 mV in white men and approximately 0.35 mV in black men. The corresponding limits were approximately 0.2 mV in white women and approximately 0.25 mV in black women.

Evaluation of ST elevation is of particular concern in connection with myocardial ischemia in acute myocardial infarction, as discussed in detail in part 6 of this series of recommendations (Acute Ischemia/Infarction). The threshold value for abnormal J-point elevation in V_2 and V_3 recommended in that part is 0.2 mV for men 40 years of age and older and 0.25 mV for men less than 40 years of age. The recommended threshold value for adult women in V_2 and V_3 is 0.15 mV. The threshold recommended for abnormal J-point elevation for men and women in all other standard leads is 0.1 mV. These threshold values appear to be an appropriate compromise for practical clinical use in the evaluation of ST elevation.

In the evaluation of ST elevation, it is important to consider ST-segment waveform in addition to the normal limits for ST amplitudes. The ST segment in normal J-point elevation in V_2 and particularly V_1 is generally sloping down steeply. Normal ST elevation at 60 ms past the J point is combined with an upsloping ST segment rather than with the more horizontal ST segment present in myocardial ischemia.

The reference values established in adequately large population-based normal groups stratified by age, gender, and race should be incorporated into computer-ECG ST-segment classification algorithms to avoid the inappropriate diagnosis of injury currents associated with myocardial ischemia, myocardial infarction, or pericarditis.

ST-segment elevation can most often be attributed to 3 specific causes: 1) a normal variant, frequently referred to as *early repolarization*, commonly characterized by J-point elevation and rapidly upsloping or normal ST segment; 2) injury currents associated with acute ischemia or ventricular dyskinesis; and 3) injury currents usually associated with pericarditis. Criteria exist to differentiate these causes (22–24) and should be incorporated into the descriptive and diagnostic algorithms of the various computer-ECG algorithms. However, it is important to recognize that in practice it is often difficult to differentiate between them. In addition, a variety of other conditions may be associated with ST-segment elevation.

ST depression may be caused by various physiological, pathological, and pharmacological interventions that change the plateau phase of the ventricular action potential. Examples include the effects of ischemia, hypokalemia, and a variety of cardiac and noncardiac drugs. These are primary ST-segment changes. Depression of the ST-segment also may occur concurrently with T-wave changes. Examples include the ST-segment depression associated with hypertrophy and, as secondary repolarization abnormalities, in ventricular conduction disturbances.

The ST-segment changes on the standard ECG that are associated with acute ischemia or infarction are due to the flow of current across the boundary between the ischemic and nonischemic zones referred to as *injury current*. ST-segment elevation generally occurs with reciprocal ST depression in ECG leads in which the axis is opposite in direction from those with ST elevation. These ST-segment abnormalities are discussed in part VI (Acute Ischemia/Infarction), which gives −0.05 as the recommended threshold value for abnormal J-point depression in leads V_2 and V_3 in men and women and −0.1 mV in all other leads (66).

Consideration of ST-segment changes as a response to exercise stress testing is outside the scope of the present working group.

Recommendation

Although it may be difficult to differentiate various causes of ST-segment abnormalities, the ECG interpretative report should include a qualitative description of the ST segment with due consideration of the age and gender of the patient, including a

note if ST depression is 0.1 mV or more pronounced. One or more of the possible causes, depending on the presence of other ECG abnormalities and the knowledge of any pertinent clinical information, also may be included. For evaluation of ST elevation, reference values established in adequately large population-based normal groups stratified by age, gender, and race should be incorporated into computer-ECG ST-segment classification algorithms to avoid the inappropriate diagnosis of injury currents associated with myocardial ischemia/infarction or pericarditis.

T-Wave Abnormalities

Just as ST-segment segment abnormalities can occur with or without T-wave abnormalities, T-wave abnormalities can occur in the presence or absence of ST-segment segment abnormalities. The T-wave amplitude in limb leads is influenced by the frontal-plane T axis, which in turn is influenced by the QRS axis.

In children older than 1 month, the T wave is often inverted in leads V_1, V_2, and V_3. In adolescents 12 years old and older and in young adults less than 20 years of age, the T wave may be slightly inverted in aVF and inverted in lead V_2. In adults 20 years old and older, the normal T wave is inverted in aVR; upright or inverted in leads aVL, III, and V_1; and upright in leads I and II and in chest leads V_3 through V_6.

In evaluations of T-wave abnormalities, T-wave negativity in lateral chest leads V_5 and V_6 is clinically particularly important. In these leads, the T wave is slightly negative (less than 0.1 mV) in 2% of white men and women 60 years of age and older and in 2% of black men and women 40 years of age and older; it is negative by 0.1 mV or more in 5% of black men and women 60 years of age and older (21).

In normal adults, the T-wave amplitude is most positive in lead V_2 or V_3. The reported normal standards for the T wave in various reports from community-based populations vary to some extent by age, gender, and race (20,21). T-wave amplitudes for V_2 from 1.0 to 1.4 mV have been listed as upper normal thresholds in men (up to 1.6 mV in the 18- to 29-year age group) and from 0.7 up to 1.0 mV in women.

A number of terms such as *peaked*, *symmetrical*, *biphasic*, *flat*, and *inverted* are being used as appropriate qualitative T-wave descriptors. As more quantitative descriptors, it is proposed that the T wave in leads I, II, aVL, and V_2 to V_6 be reported as *inverted* when the T-wave amplitude is from -0.1 to -0.5 mV, as *deep negative* when the amplitude is from -0.5 to -1.0 mV, and as *giant negative* when the amplitude is less than -1.0 mV (25). In addition, the T wave may be called *low* when its amplitude is less than 10% of the R-wave amplitude in the same lead and as *flat* when the peak T-wave amplitude is between 0.1 and -0.1 mV in leads I, II, aVL (with an R wave taller than 0.3 mV), and V_4 to V_6.

Interpreting isolated T-wave abnormalities is difficult and often the source of ambiguous and inaccurate statements. The inappropriate diagnoses of myocardial ischemia and infarction are common errors. As indicated above, ST- and T-wave abnormalities that are secondary to abnormalities in ventricular conduction should be labeled as such. ST- and T-wave changes associated with hypertrophy, hypokalemia, and drugs can be attributed to one of these factors. Giant T-wave inversion is usually limited to one of several entities, including hypertrophic cardiomyopathies, non–ST-segment elevation myocardial infarctions, and neurological events, particularly intracranial hemorrhage. The interpretation of such T-wave changes should be descriptive, and a statement listing the most common causes is appropriate.

It is virtually impossible to develop a cause-specific classification for minor T-wave abnormalities. For these, classification as *slight* or *indeterminate* T-wave abnormality is appropriate. The overreader can then apply analysis of other features, and the clinical condition if available, to provide a more likely list of diagnostic possibilities. In these situations, comparison with prior ECGs (if available) is often helpful.

Notching of the T wave may be difficult to discriminate from a U wave that is superimposed on the downslope of an upright T wave. It is important to recognize that the T wave is rarely notched in all 12 leads and that the interval between the 2 summits of a notched T wave is usually less than the interval between the peak of a monophasic T wave and the U wave, which usually exceeds 150 ms at heart rates of 50 to 100 bpm (26).

Recommendation

The ECG report should include a description of T-wave abnormalities, identification of associated ST-segment changes if present, and a statement as to whether the changes are indeterminate or more likely to be associated with a specific cause.

T-Wave Alternans

T-wave alternans signifies T-wave amplitude variations that alternate every second beat. These amplitude variations are quantified with various modifications of moving-average analysis or as the variance of specific frequency components in spectral analysis. T-wave alternans is typically observed as microvolt-level variation (microvolt T-wave alternans) and, at times, as more pronounced variations in alternating complexes or as slower components outside the range of the proper T-wave alternans, generally most prominent in phase with respiration.

T-wave alternans indicates latent instability of repolarization predictive of malignant arrhythmias. It is generally not present at the resting state even in high-risk patients, and a stress test (exercise or pharmacological stress or pacing), requiring special equipment and analysis software, is needed to provoke it. These procedures are outside the scope of the present document. It is sufficient to state that although the role of T-wave alternans regarding its clinical utility has not been fully defined, it holds substantial potential in identifying patients at high risk of serious arrhythmic events.

The U Wave

The U wave is a mechanoelectric phenomenon (26) that results in a low-amplitude, low-frequency deflection that occurs after the T wave. It is frequently absent in the limb leads and is most evident in leads V_2 and V_3, where its

amplitude has been suggested to be approximately 0.33 mV or 11% of the T wave (27). Its presence is heart-rate dependent; it is rarely present at rates greater than 95 bpm. Bradycardia enhances the U-wave amplitude and is present in 90% of cases at heart rates less than 65 complexes per minute (28).

It has long been held that an increase in U-wave amplitude, usually in association with depression of the ST segment and a decrease in T-wave amplitude, may be caused by a variety of cardioactive drugs with quinidine-like effects and by hypokalemia and that with more advanced hypokalemia, that is, K less than 2.7 mmol/L, the U-wave amplitude may exceed the T-wave amplitude in the same lead. However, more recent information suggests that this may be due to fusion of the U wave with the T wave rather than to an increase in U-wave amplitude per se.

Fusion of the U wave with the T wave also occurs in association with an increase in sympathetic tone (29) and in the presence of a markedly prolonged QT interval such as that which occurs in congenital and acquired long-QT syndromes (LQTS).

An inverted U wave in leads V_2 through V_5 is abnormal (30). It may appear transiently during acute ischemia or in the presence of hypertension (31,32). An abnormal U wave is often quite subtle and is rarely an isolated ECG abnormality. Thus, its presence is often not recognized or is overlooked by ECG readers and automated systems. For these reasons, no specific descriptive or diagnostic statements are recommended for inclusion in the automated list of terms. It remains the responsibility of the overreader to recognize abnormal U waves and to determine their clinical relevance.

Recommendation

Statements concerning the U wave should be included in the ECG interpretation when the U wave is inverted, when it is merged with the T wave, or when its amplitude is greater than that of the T wave.

The QT Interval

Measurement of the QT interval and its adjustment for rate, gender, and QRS prolongation represent 2 of the major challenges in electrocardiography. They are matters of great importance to physicians, drug manufacturers, and regulatory agencies because of the relationship between prolongation of the QT interval and potentially lethal ventricular arrhythmias. The document released in October 2005 by the Food and Drug Administration (FDA) provides guidance for the design, conduct, analysis, and interpretation of clinical studies for evaluation of QT-interval prolongation (33).

QT and ST-T patterns vary a great deal in various genotypes of the LQTS. Zhang et al (34) described 10 different ST-T patterns in first 3 genotypes of the syndrome (4 in LQT1, 4 in LQT2, and 2 in LQT3), and these patterns were present in the majority of genotyped LQTS patients.

The QT interval is defined as the interval from the onset of the QRS complex, that is, the earliest indication of ventricular depolarization, to the end of the T wave, that is, the latest indication of ventricular repolarization. The problems associated with this measurement include the following: 1) recognizing the onset of the QRS complex and the end of the T wave, 2) determining the appropriate lead(s) in which to measure the QT interval, and 3) adjusting the QT interval for increases in QRS duration, gender, and rate.

When the majority of ECGs were recorded on single-channel analog machines, various leads were recorded sequentially, and the QT interval was measured manually in the individual leads. Determination of the end of the T wave was often difficult and sometimes impossible, and the onset of the QRS complex and the end of the T wave varied in different leads, appearing shorter when the axis of an individual lead was more perpendicular to the spatial vector of the onset of the QRS complex or the end of the T wave. The onset of the QRS complex tends to occur up to 20 ms earlier in V_2 and V_3 than in the limb leads (35). Some regard differences of up to 50 ms in QT intervals measured in the various leads in normal subjects as being normal (36); others have suggested that differences of up to 65 ms were still within the limit of normal (37). This value is reported to be less in women than in men (38).

When the QT interval is measured in individual leads, the lead showing the longest QT should be used (39). This is usually V_2 or V_3. However, if this measurement differs by more than 40 ms from that in other leads, the measurement may be in error, and measurements from adjacent leads should be considered. If the T wave and U wave are superimposed or cannot be separated, it is recommended that the QT be measured in the leads not showing U waves, often aVR and aVL (39), or that the downslope of the T wave be extended by drawing a tangent to the steepest proportion of the downslope until it crosses the TP segment. It should be recognized that defining the end of the T wave in these ways might underestimate the QT interval.

As detailed in the section on ECG technology (1), most currently used automated digital machines record all leads simultaneously. This technique permits their temporal alignment and superimposition, which facilitates a more accurate assessment of the beginning of the QRS complex, the end of the T wave, and the separation of the U wave from the T wave. As a result, the automatically measured QT interval is often longer than the QT interval as measured in any individual lead, and the values currently regarded as normal, which were established with single-channel sequential recordings, may no longer be valid. Most automated systems do not routinely display the superimposed tracings or the points used to derive the QT interval.

In view of the clinical importance of the QT-interval prolongation, it is essential to visually validate QT-interval prolongation reported by a computer algorithm.

In addition to administration of QT-prolonging cardioactive drugs, a number of conditions can induce QT prolongation. It is often possible to identify a specific cause of QT prolongation when appropriate clinical information is available; for instance, both hypokalemia and hypocalcemia can prolong phase 2 and phase 3 of the action potential and prolong the QT interval. It is not feasible here to compile a comprehensive list of all possible causes of QT prolongation. It is sufficient to emphasize that its presence in an ECG report should call for a careful clinical evaluation of possible causes.

Recommendation

It is recommended that selective subsets of temporally aligned superimposed ECG leads be made available as an optional display to facilitate QT measurement and to validate the onset and end points of the QT interval. In view of the clinical importance of QT-interval prolongation, it is essential to visually validate QT-interval prolongation reported by a computer algorithm.

QT Correction for Rate

Many formulas have been proposed to adjust the QT interval for rate (40,41). The most widely used is the formula derived by Bazett (42) in 1920 from a graphic plot of measured QT intervals in 39 young subjects. This adjustment procedure divides the measured QT by the square root of the RR interval to derive the rate-adjusted value. The formula introduced by Fridericia (43), also in 1920, uses the cube root of RR. Bazett's formula leaves a strong positive residual correlation ($r=0.32$) and Fridericia's formula leaves a negative correlation ($r=-0.26$ to -0.32) with heart rate (44,45), and the adjusted QT values may be substantially in error, particularly when the heart rate is high. More recently introduced formulas for QT adjustment as a linear or power function of RR or heart rate for adults (44–48) and for children (49) effectively remove the rate dependence of the adjusted QT, and they are clearly preferable to both Bazett's and Fridericia's formulas. Some investigators have introduced separate normal limits or rate correction factors for each heart rate subinterval using the so-called "bin method" (46,50).

Recommendation

It is recommended that linear regression functions rather than the Bazett's formula be used for QT-rate correction and that the method used for rate correction be identified in ECG analysis reports. In addition, rate correction of the QT interval should not be attempted when RR interval variability is large, as often occurs with atrial fibrillation, or when identification of the end of the T wave is unreliable.

QT Correction for Gender and the Limits for Prolonged and Short QT Interval

Although Bazett's and Fridericia's formulas make no adjustment for gender, many studies have demonstrated that the QT interval is longer in young and middle-aged females than in males. The gender difference is potentially important because women are generally considered to be more prone to malignant arrhythmias in LQTS than men. The gender difference appears during adolescence (51), when the rate-adjusted QT shortens in boys, possibly as a testosterone effect, but undergoes little change in girls.

The reported gender difference in various studies varies from 6 to 10 ms in older age groups and from 12 to 15 ms in younger adults. Overall, the gender difference in rate-adjusted QT interval becomes small after 40 years of age and practically disappears in older men and women. Separate gender- and age-specific QT-adjustment formulas have been proposed to accommodate these differences (21,44,46,47). Normal limits proposed in different studies vary to a certain extent, depending on the characteristics of the study population and particularly on the type of QT-adjustment function used. It is important to recognize that normal limits established using the upper and lower limits of actual percentile distributions of the rate-adjusted QT are preferable to those as mean values $\pm 2\times$SD because these distributions are strongly skewed (44).

Normal standards for thresholds for abnormal QT from large subgroups of community-based populations are available (21,44–48). These limits are relatively uniform in reports that have appropriately used linear regression functions with QT-rate adjustment as a linear or power function of RR or heart rate (21,44,46–48). The following normal limits are suggested as a practical compromise for the evaluation of QT-interval prolongation and shortening in adult men and women: prolonged QT: women, 460 ms or longer; men, longer than 450 ms; and short QT: women and men, 390 ms or shorter.

FDA guidelines for industry recommend that 3 severity levels for rate-corrected QT be reported when considering possible QT-prolonging effects of drugs: longer than 350 ms, longer than 480 ms, and longer than 500 ms (32).

Although the upper normal limits for QT adjusted for rate as a linear function of RR in small groups of children stratified by age, gender, and heart rate have been published (52), the limits for prolonged and short QT established in reasonably large groups of children have been reported only for Bazett's formula (53). In that report, the 98th percentile limit for rate-adjusted QT was approximately 450 ms in children younger than 12 years of age. The gender difference of 8 ms appeared in the 12- to 16-year age group. It should be noted that QT adjusted by Bazett's formula may produce false QT prolongations (45).

Recommendation

It is recommended that, in addition to rate, an adjustment for gender and age be incorporated into QT adjustment. As practical clinical limits for considering the QT interval as abnormal, it is recommended that the adjusted QT of 460 ms or longer in women and 450 ms or longer in men be considered a prolonged QT interval and that QT 390 ms and shorter be considered a short QT interval.

Correction for QRS Duration

The QT interval prolongs in ventricular conduction defects, and an adjustment for QRS duration becomes necessary (54–56). This can be accomplished best by incorporating QRS duration and RR interval as covariates into the QT-adjustment formula or by using the JT interval (QT duration–QRS duration) (55). If the JT interval is chosen, normal standards established specifically for the JT interval should be used (55).

Recommendation

QT- and JT-adjustment formulas have recently been introduced for use in the setting of prolonged ventricular conduction. With confirmation, they may be incorporated into automated algorithms to provide appropriate correction factors.

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Evaluation of QT Intervals in Sequential Tracings

Evaluation of QT intervals from sequential ECG recordings is essential for identification of QT prolongation induced by pharmacological agents in drug studies. It is also helpful as an adjunct to ST analysis in determining the presence of active ischemia in patients suspected of having myocardial infarction.

When sequential tracings from patients recorded at different times of day and at different locations are compared, it is important to recognize that the time of day can influence the QT interval (57,58), that differences may exist between the different recording systems and between the programs used for QT measurement, and that different formulas for QT-rate adjustment may have been used. Moreover, there is a significant interreader variability in the measurement of QT interval (59). Rigorous standardization of the recording and evaluation procedures should be followed when serial comparison of QT intervals is undertaken.

The FDA guide for industry suggests that in the evaluation of possible QT prolongation induced by drugs, 2 levels of change in serial ECGs in the rate-corrected QT from the baseline be reported: an increase of greater than 30 ms and an increase of greater than 60 ms (33).

Recommendation

It is recommended that for serial comparisons ECG recorders meet specific performance standards and, if possible, be of the same type; that uniform, carefully standardized ECG acquisition and QT-measurement procedures be used; and that, whenever possible, a single reader be responsible for overreading sequential tracings of an individual patient or research subject.

QT Dispersion

Increased heterogeneity of myocardial repolarization may predispose patients to the development of malignant ventricular arrhythmias (60). As indicated above, significant differences exist in the duration of the QT interval when measured in the individual leads. Visualization of these differences is facilitated by the display of a suitable subset of temporally aligned simultaneous leads with a slight separation on the amplitude scale.

The difference between the longest and shortest QT intervals is referred to as QT dispersion. This concept was introduced in 1990 for risk identification in patients with LQTS (61). Since its introduction, QT dispersion has been one of the most popular topics in ECG research. In November 2006, a PubMed search cited 670 publications with QT dispersion in the title, and a Google search under "QT dispersion measurement" revealed 171 000 communications.

The QT-dispersion concept has led to the expectation that QT dispersion is a measure of regional or localized heterogeneity of myocardial repolarization. Numerous studies have suggested an increased risk of morbidity and mortality for an increase in QT dispersion. However, substantial methodological problems with the QT-dispersion measurement have been identified that have raised fundamental questions about the validity of the concept (62–65). In principle, the expectation that QT dispersion is a measure of the regional or localized heterogeneity of myocardial repolarization implies that the leads with the shortest and longest QT contain signal information at the terminal part of the T wave that is not present in the first 3 orthogonal components of the 12-lead ECG (or the composite global T wave) (65).

Until adequately validated data in specific clinical conditions are presented showing that QT dispersion on the body surface ECG is the counterpart of localized dispersion of myocardial repolarization and conveys adequately strong nondipolar signal information that cannot be extracted from the X,Y,Z components, it seems unwise to include it as a part of the routine ECG report.

Recommendation

It is recommended that QT dispersion not be included in routine ECG reports. However, because of the fundamental importance of the heterogeneity of myocardial repolarization in the genesis of malignant ventricular arrhythmias, continued research into the identification of markers of increased dispersion of myocardial repolarization on the body surface ECG is encouraged.

Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
James J. Bailey	National Institutes of Health	None	None	None	None	None	None
Rory Childers	University of Chicago	None	None	None	None	None	None
Barbara J. Deal	Northwestern University	None	None	None	None	None	None
Leonard S. Gettes	University of North Carolina	None	None	None	None	None	None
Anton Gorgels	University Hospital Maastricht	None	None	None	None	None	None
E. William Hancock	Stanford University Medical Center (retired Professor Emeritus)	None	None	None	None	Philips Medical Systems†; Covance Diagnostics†	None
Mark Josephson	Harvard Medical Faculty Physicians for Beth Israel Deaconess Medical Center	None	None	None	None	Medtronic*	None
Paul Kligfield	Weill Medical College of Cornell University	None	None	None	None	Philips Medical*; Mortara Instrument*; GE Healthcare*; MDS Pharma Services†; Cardiac Science*	None
Jan A. Kors	Erasmus Medical Center	None	None	None	None	None	Welch Allyn*
Peter Macfarlane	University of Glasgow	Cardiac Science Corporation†; Medtronic Physio Control†; Spacelabs Health Care†; Draeger Medical†; Heartlab†; McKesson†	None	None	None	Cardiac Science Corp†; Medtronic Physio Control†; Spacelabs Health Care†; Draeger Medical†; Heartlab†; McKesson†	None
Jay W. Mason	Independent consultant	None	None	None	None	None	None
David M. Mirvis	University of Tennessee	None	None	None	None	None	None
Peter Okin	Weill Medical College of Cornell University	Merck & Co, Inc†	None	None	None	None	None
Olle Pahlm	BFC Klin	None	None	None	None	None	None
Pentti M. Rautaharju	Wake Forest University Medical School (retired)	None	None	None	None	Philips Medical Systems†	None
Borys Surawicz	CARE Group	None	None	None	None	None	None
Gerard van Herpen	Erasmus Medical Center	None	None	None	None	None	Welch Allyn*
Galen S. Wagner	Duke University Medical Center	Medtronic† Physiocontrol† Welch Allyn†	None	None	None	None	None
Hein Wellens	University of Maastricht	None	None	None	None	Medtronic*	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

Reviewer Disclosures

Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/Advisory Board	Other
Jeffrey L. Anderson	Intermountain Medical Center	None	None	None	None	None	None	None
Leonard S. Dreifus	Hahnemann University Hospital	None	None	None	None	None	None	None
Mark Eisenberg	McGill University	None	None	None	None	None	None	None
Nora Goldschlager	University of California, San Francisco	None	None	None	None	None	None	None
Cindy Grines	William Beaumont Hospital	None	None	None	None	None	None	None
Mark Hlatky	Stanford University	None	None	None	None	None	None	None
Peter Kowey	Lankenau Medical Office	None	None	None	None	Cardionet†	Transoma*; Cardionet†; NewCardio*	None
Rachel Lampert	Yale University	Medtronic, Inc†; Guidant/Boston Scientific†; St Jude†	None	None	None	None	Medtronic*	None
Robert Lichtenberg	Heart Care Centers of Illinois	None	None	None	None	None	None	None
Jonathan Lindner	Oregon Health and Sciences University	Genentech*	None	None	None	None	Genentech*; VisualSonics*	None
Frank Marcus	University of Arizona	None	None	None	None	None	None	None
Robert J. Myerburg	University of Miami	None	None	None	None	None	None	None
Gerald M. Pohost	University of Southern California, Keck School of Medicine	None	None	None	None	None	None	None
Richard Schofield	University of Florida Health Sciences Center	None	None	None	None	None	None	None
Samuel Shubrooks	Beth Israel Deaconess Medical Center	None	None	None	None	None	None	None
John Strobel	IMA, Inc	None	None	None	None	None	None	None
Stuart A. Winston	Michigan Heart, PC	Medtronic*; Boston Scientific*	None	Boston Scientific*	None	None	None	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

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†Significant.

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