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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

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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.

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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