# The place of the electrocardiogram in modern cardiology

The electrical activity of the heart generates currents that flow throughout the tissues of the body, creating potential differences between different areas of the body surface. The electrocardiogram is a measurement of these potential differences as a function of time.

## DEVELOPMENT OF THE 12-LEAD ELECTROCARDIOGRAM

The first reported recording of a human electrocardiogram was that of the physiologist Waller (1887), who used a mercury electrometer to record the potential difference between the limbs, and also between the front and back of the chest. The mercury column was seen to pulsate in time with the heartbeat, although the frequency response of this system was poor and so the resulting electrocardiogram did not reflect the underlying electrical activity of the heart accurately.

The development of the string galvanometer, usually credited to the Dutch physician Einthoven, was an important advance in electrocardiography allowing a frequency response comparable to that of modern systems. Using a string galvanometer Einthoven developed the use of bipolar limb leads by recording the potential differences between the left arm and right arm (lead I), left leg and right arm (lead II), and left leg and left arm (lead III; Einthoven, 1901). In the early part of the 20th century this recording system was used to document abnormalities of cardiac rhythm and conduction.

In the United States during the 1920s and 1930s, Wilson linked the electrodes attached to the left and right arms and the left leg via equal resistances to a central terminal with a relatively constant potential (Wilson et al, 1932). The central terminal allowed the measurement of potential variations at a single point using a 'unipolar' exploring electrode.

In the modern 12-lead electrocardiogram the six praecordial leads are derived by measuring the potential difference between the relevant praecordial electrodes and the central terminal. The positions of these leads were defined by the American Heart Association in the 1940s. If the exploring electrode is placed on the limbs a 'unipolar limb lead' is recorded. This arrangement was modified by Goldberger in 1942 who removed the central terminal connection from the limb on which the exploring electrode was placed. The result of this was to increase the potential recorded by the relevant lead by 50%. This gave rise to the 'augmented unipolar limb leads' aVR, aVL and aVF, thus completing the development of the standard 12-lead electrocardiogram.

## RELATIONSHIP BETWEEN THE ELECTROCARDIOGRAM AND CELLULAR ELECTROPHYSIOLOGY

The clinical utility of the electrocardiogram became rapidly apparent, most notably in the diagnosis of cardiac rhythm disorders, but also as a valuable tool for the diagnosis of a wide variety of cardiac diseases. Recent advances in intra- and extracellular recording techniques have provided insight into the electrophysiological events at cellular and tissue level in both health and disease.

Direct measurements from canine and human hearts have established the sequence of cardiac activation and recovery and the corresponding electrocardiographic deflections. In sinus rhythm activation begins in the sinus node in the posterolateral high right atrium. The activation wavefront spreads through the atrial myocardium giving rise to P waves that are positive in the inferior, praecordial and left-sided limb leads.

Conduction through the atrioventricular node is slow and makes the major contribution to the PR interval. Upon exit from the atrioventricular node the activation wavefront is conducted rapidly along the bundle of His, the right and left bundle branches and the subendocardial network of Purkinje fibres. Rapid and virtually simultaneous activation of the ventricles is reflected in the narrow QRS complex.

The cardiac action potential is of long duration with a pronounced plateau. During this phase the majority of the cells in the heart are depolarized to a similar extent. As a result there is little extracellular current flow, giving rise to the isoelectric ST segment. During the terminal portion of the cardiac action potential certain areas of the heart repolarize at different times and potential gradients develop, resulting in the electrocardiographic T wave.

At the cellular level depolarization and repolarization are electrically opposite events. Why then are the QRS complexes (representing activation) and the T waves (representing repolarization) concordant in the normal electrocardiogram? Elegant experiments have shown that there is an inverse relationship between activation time and action potential duration (Cowan et al, 1988). In other words, the later an area of the ventricle is depolarized, the shorter the action potential duration in that area. The vector of repolarization is therefore roughly opposite to the vector of depolarization. Because current flow during repolarization is opposite to that during depolarization, the T wave and QRS complex are concordant. The cellular basis for the electrocardiographic U wave remains the subject of considerable debate (Surawicz, 1998).

## THE ELECTROCARDIOGRAM IN ACUTE ISCHAEMIA

The diagnosis of acute myocardial ischaemia is made from the history with supportive evidence from the electrocardiogram. Within seconds of experimental coronary occlusion profound changes occur in the transmembrane potential of cells within the ischaemic territory (Janse and Wit, 1989). A reduction in the resting membrane potential occurs accompanied by a reduction in the upstroke velocity and amplitude of the cardiac action potential.

At the same time, changes occur to the duration of the action potential. On the endocardium, progressive shortening of action potential duration occurs while on the epicardium an initial lengthening may be seen. Coronary artery disease typically results in regional ischaemia. As a result of the electrophysiological changes during ischaemia there is current flow between the ischaemic area and surrounding myocardium during the TQ segment (diastole) and the ST segment (systole).

Subepicardial ischaemia, as seen with acute coronary occlusion, results in TQ depression and ST elevation. Subendocardial ischaemia, as seen during angina of effort, gives rise to current flow in the opposite direction, causing TQ elevation and ST depression.

## POSSIBLE FUTURE ROLES FOR ELECTROCARDIOGRAPHY

There is considerable evidence to implicate acute ischaemia in the genesis of life-threatening arrhythmias. An important mechanism linking acute ischaemia with ventricular fibrillation is increased dispersion of repolarization. In the normal heart repolarization is a rapid and orderly process. Certain disease states such as acute ischaemia are characterized by increased dispersion of repolarization. In this state some areas of myocardium may have regained excitability following activation while adjacent areas remain depolarized and inexcitable. This situation creates an environment in which arrhythmias such as ventricular fibrillation may flourish.

The ability to measure dispersion of repolarization in the human heart would be expected to give important insights into the genesis of clinically important arrhythmias. The QT interval of the surface electrocardiogram reflects the duration of ventricular activation and recovery. Interlead variations in the duration of the QT interval ('QT dispersion') have been recognized for many years. It has been suggested that QT dispersion may provide a non-invasive measure of dispersion of repolarization in the human heart and as such may be used to study the mechanisms of human arrhythmogenesis, to provide prognostic information and to assess the effects of therapeutic interventions (Jordaens, 1999). Increased QT dispersion has been observed in certain disease states characterized by a high incidence of sudden arrhythmic death including congenital long QT syndromes and hypertrophic cardiomyopathy.

A number of issues have limited the clinical application of QT dispersion including methodological problems, the lack of an accepted normal range, and a relatively low correlation between QT dispersion and direct measurements of dispersion of repolarization in experimental studies (Surawicz, 1996). In some studies QT dispersion has been measured in disease states where there is little or no experimental evidence to support a role for increased dispersion of repolarization in arrhythmogenesis. Unsurprisingly QT dispersion has not been found to be predictive of arrhythmic events in these settings. Nevertheless a non-invasive measure of dispersion of repolarization in the human heart remains a worthwhile goal and alternative electrocardiographic measures are being explored, including JT dispersion, T peak to T end duration, and T wave area.

### KEY POINTS

- Cellular electrophysiological techniques have established the sequence of cardiac activation and recovery resulting in the deflections of the surface electrocardiogram.
- The electrocardiogram is indispensable for the diagnosis of cardiac arrhythmias and conduction disorders and is a useful adjunct to the diagnosis and management of many other cardiac disorders that affect cardiac electrical activity.
- Research continues to explore new applications of the electrocardiogram including assessment of dispersion of ventricular repolarization, a critical factor in the genesis of certain life-threatening arrhythmias.

## CONCLUSION

The electrocardiogram provides a non-invasive, cheap and widely available means of measuring the heart's electrical activity. It is indispensable for the diagnosis of cardiac arrhythmias and conduction disorders and a useful adjunct to the diagnosis and management of many other cardiac disorders that affect cardiac electrical activity, typified by acute myocardial ischaemia. Our knowledge of the cellular electrophysiological disturbances accompanying disease states is expanding steadily. The electrocardiogram may provide insight into the role of these processes in human arrhythmogenesis. HM

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