Some Electrophysiological Concepts of the U Wave of ECG

Wg Cdr N Mohan Murali*, VSM (Retd)

Introduction

THE normal ECG complexes have been correlated with the sequence of electrical events in the heart. P wave is the result of atrial muscle depolarization, the QRS complex is produced by the depolarization of the ventricular myocardium and the T wave results from the return of ventricular muscle to the resting state due to repolarization. At times the T wave is followed by a small wave called the U wave, low in amplitude and not always recorded. It is best observed in the precordial leads. The electrophysiology of the U wave is not yet properly understood.

Clinical Significance

Clinical significance of changes in the U wave of the ECG are now attracting the attention of cardiologists in diagnosing heart diseases. The changes that have been observed are the inversion of U wave and the appearance of pronounced U wave in exercise ECG. In certain cases of hypertension, coronary artery sclerosis and other organic heart diseases, inversion of the U wave has been found to be one of the earliest electrocardiographic changes preceeding the classic ECG findings. It has also been noticed that exercise precipitates the appearance of a prominent U wave which is probably due to inadequate supply of oxygen in the presence of increased demand during exercise. The recognition of the U wave will, therefore, enhance the value of the ECG in clinical diagnosis.

Some Postulated Theories

Some theories have been postulated to explain the genesis of the U wave. According to Hoff and Nahum[1], it forms part of the ventricular complex and is coincident in time with the super normal phase. While some others suggested that it originates in the intraventricular septum and is a result of retardation of repolarization in that structure due to its compression caused by the contraction of the ventricles. The U wave has also been linked with certain mechanical factors like the outward thrust of the apex. Lepeschkin has proposed that it mainfests from after potentials in the ventricular action potential, i.e. failure of restoration of the normal membrane resting potential at completion of repolarization. Available data do not allow complete acceptance of any theory of genesis of the U wave.

The New Concept

In this paper an attempt has been made to understand the causation of the U wave. The U wave appears after the T wave and has same polarity as the T wave in a normal ECG. The T wave represents the repolarization of the ventricles. It would therefore be quite pertinent to correlate the U wave with some repolarization event taking place after the repolarization of the ventricles. In order to understand the genesis of the wave, it is necessary to examine some of the electrical features of the ventricles.

The stimulus from the S. A. node after reaching the A.V. node spreads through the bundle of His and then through the Purkinje fibres which directly activate the inner most layers of the myocardium. The excitation then spreads through the ventricular muscle from apex to the base and generally from endocardium to epicardium at about 0.3 meter per sec. In general, excitation of the epicardial surface occurs later than its endocardial counterpart. For purposes of didactic discussions the muscular mass comprising the free walls of the ventricles may be considered to consist of an inner two-thirds (sub-endocardial) and an outer one-third (sub-epicardial)[5]. Propagation through the former is rapid, figures

* Dixit Bungalow, Belgaum Road, Dharwar-8, Karnataka.

of 1 to 2 meters per sec. being commonly found. The conduction rate decreases as excitation moves away from the endocardium with the result that the impulse travels through the outer or sub-epicardial third of the wall at speeds of only 300 to 400 millimeter per sec. This data strongly suggests that branches of Purkinje network penetrate deeply into the myocardial wall and is in agreement with the anatomic studies. The last regions to be activated are the posterobasal portion of the left ventricle, the pulmonary conus and the upper portion or interventricular septum because there is little Purkinje tissue in these regions.

The sequence of depolarization in ventricles is such that the contraction of the myocardium is from endocardium to epicardium and from apex towards base which is necessary so that blood is forced out efficiently through the outlet valves of the ventricle. In most electrocardiographic leads, the T wave of the ECG has the same polarity as the QRS complex indicating that the sequence of repolarization in ventricles does not follow the same pathway as depolarization and indeed that the pathways tend to be opposite. The author[2,3] had developed a concept to explain this paradoxical orientation of ventricular repolarization in the human electrocardiogram. This concept explains that the inner layers have a much smaller radius and circumference especially near the apex than those of the outer layers of the left ventricle and that the internal cross section increases from apex towards base. Due to these differences the inner layers would shorten more than the outer layers and the degree of contraction would be more at the apex, decreasing towards the basal regions, in ejecting a particular volume. In other words, the tension is decremental from endocardium to epicardium and from apex towards base. This decremental contraction would act as a retarding agent or the factor which causes the repolarization to proceed in a direction opposite to that of depolarization which remains unchanged. Thus the process of relaxation in the ventricles would be from outside to inside of the ventricular walls and from base towards apex which is in a direction opposite to that of contraction. Such a sequence of contraction and relaxation of the ventricles conforms to the requirements of the ventricle as a pump where in-flow during filling is opposite in direction to outflow during ejection as the inlet and outlet valves are located on the same side.

It has been mentioned above that the Purkinje network penetrates deeply into the myocardial wall (sub-endocardial). After the ventricular walls have repolarized in the manner as explained, the repolarization has to commence in the Purkinje network. Since the repolarization takes place from epicardium to endocardium and from base to apex, the Purkinje network embedded in the sub-endocardial layers would also have to repolarize in a similar fashion but later than the ventricular walls. Further, the action potentials of the Purkinje fibres are considerably longer than that of the ventricular muscle. This may be to prevent re-entry of activity back into the parts of this conduction system from late firing ventricular muscle fibres. This would also explain the repolarization of the Purkinje system taking place later than that of the ventricular muscle. The U wave appearing after the T wave and having the same polarity as the T wave in a normal ECG may therefore be correlated with the repolarization of the Purkinje network penetrated into the myocardial wall. Since the action potentials in the Purkinje system produce small differences of potentials, they are not normally recordable through external electrodes. Palmer[4] noticed U wave inversion without other electrocardiographic changes in some patients and observed that this abnormality was probably related to organic changes in the myocardium. This further lends support to the above concept.

Conclusion

U wave appears after the T wave having the same polarity as the T wave in a normal ECG. As the T wave represents the repolarization of the ventricles, it would be quite pertinent to correlate the U wave with some repolarization event taking place after the repolarization of the ventricles. An attempt has been made to correlate this wave with the repolarization of the Purkinje network penetrated into the myocardial wall.

References

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