INTRODUCTION

The Q R S complex is produced by activation of both ventricles and the upper normal value for Q R S duration is traditionally given as less than 120msec (and often as <110msec) measured in the lead with the widest Q R S duration ***(Braunwald's, 2004).***

Prolongation of the Q R S complex has been associated with adverse outcomes in heart failure (H F) patients ***(Hofmann et al., 2005).***

QRS duration greater than 120 ms is associated with increased mortality among these patients ***(Shenkman et al., 2002)***.

Further more, cardiac resynchronization therapy in patients with prolonged Q R S duration has been shown to decrease cardiac morbidity and mortality ***(Bristow et al., 2004).***

The association of Q R S duration with clinical outcomes in the post myocardial infarction (M I) setting is less clear. Prolonged QRS duration even within the normal range, is associated with larger ventricular volumes, reduced systolic function, and an increased risk for development of heart failure (HF), sudden death (SD) and cardiovascular (CV) death after myocardial infarction (MI) but appears to be a marker, rather than an independent predictor for increased risk ***(Yerra et al., 2006).***

AIM OF THE WORK

The aim of this study is to assess the prognostic significance of QRS duration prolongation on initial electrocardiogram after acute MI.

ELECTROCARDIOGRAM AND HAEMODYNAMICS

I- Electrophysiology of Cardiac Conduction

1. Electrical Activity of the Heart:

The heart is a 2 step mechanical pump that is coordinated by prescribedly timed electrical impulses. For the pump to perform optimally, sequential depolarization of atria and then the ventricles allow atrial contraction to provide complete diastolic filling of the ventricles (so, called atrio-ventricular synchrony). Once the ventricle are filled, rapid activation of the ventricular myocardium allows a synchronized contraction to eject blood most effectively to great vessels.

In normal cardiac conduction, electrical excitation of the heart proceeds in a sequential manner from the atria (reflected by the P wave on ECG) to reach the atrio-ventricular node. As the impulse conducts through the atrio-ventricular node, conduction slows allowing time for atrial contraction to occur before ventricular activation (PR segment). Once through the compact atrio-ventricular node, the impulse conducts rapidly through the crux of the heart to the ventricles via the bundle of Hiss (penetrating bundle) to the branching bundle, the bundle branches, the distal purkinje fibers and finally, to the ventricular myocardial cells (Narrow QRS complex). When depolarization is complete, the ventricle repolarizes in preparation for conducting another impulse ***(Ramsaran and Spodick, 1996)***.

2. Normal waves of ECG:

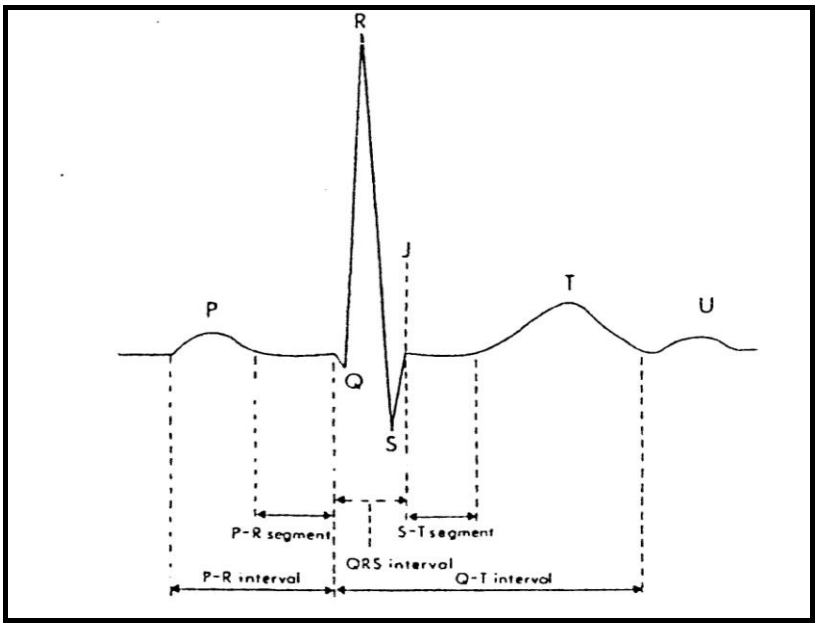

Figure (1): Normal ECG ***(Nishimura et al., 1995).***

P wave:

Represents atrial activation. It is divided into three parts: first part represents activation of the right atrium, middle part represents completion of the activation of right atrium and initiation of left atrial activation and third part represents completion of left atrial activation. P wave duration is normally <0.12 sec. and amplitude <0.20mv ***(Nishimura et al., 1995)***.

P-R interval:

It estimates the conduction through atrio-ventricular node, Hiss bundle, bundle branches and main divisions of the left bundle branch as well as through those parts of the

atria located between sinus and atrio-ventricular node. Its duration varies from 0.12 to 0.2 or 0.22 sec ***(Scanu et al., 1995).***

QRS complex:

Its significance is that it represents the time required for a stimulus to spread through the ventricle. Its timing is from the beginning of the Q wave to the end of S-wave ***(Line et al., 1995)***.

* *Q wave:*

It is a negative wave at the onset of the QRS complex represents the onset of ventricular depolarization, it reflect activation of left side of septum to the right side producing q wave in V_6 and small r wave in V_1 ***(Line et al., 1995 and Mabo et al., 1992)***.

Table (1): Normal Q wave in different ECG leads

Limb leads		**Pericardial leads**	
Lead	**Upper limit**	**Lead**	**Upper limit**
I	<0.03 sec	V_1	Any
II	<0.03 sec.	V_2	Any
III	-	V_3	Any
avR	-	V_4	<0.02 sec
avL	<0.03 sec.	V_5	<0.03 sec
avF	<0.03 sec	V_6	<0.03 sec

(Mabo et al., 1992)

* *R wave:*

Represents the progression of activation wave from thinner right ventricle to the thicker left ventricle. It is the first positive deflection which increases in amplitude and duration from V1-V6. Its duration is 0.04-0.06 sec ***(Daubert et al., 1998).***

* *S wave:*

Represents the end of ventricular depolarization. It represents the second negative deflection. Its amplitude is large in V1 & V2 and is getting smaller in V3-V6. Its duration <0.03 sec. Its significance is that it represents the time from activation of endocardial insertion of purkinje network to the time of arrival of electrical impulse at the precordial surface beneath the recording electrodes on body surface ***(Panidis et al., 1986).***

* *Duration of QRS:*

It is normally ranged from 0.07 to 0.12 sec. It is slightly longer in males than in females and it is measured from the beginning of the first appearing Q or R wave to the end of the last appearing of the last R, S, Ŕ or Ś wave ***(Mabo et al., 1992)***.

***** *Amplitude of QRS:*

It varies with age, increasing until about age 30 and then gradually decrease the amplitude is generally large in

males than in females. It is measured between the peaks of the tallest positive and negative wave forms, occasionally up to 30mm have seen in normal individuals ***(Mabo et al., 1992)***.

S-T segment:

Represents the period of time when ventricular myocardium remains in an activated state. It is iso-electric line starts from the end of QRS to the beginning of T wave ***(Appleton et al., 1991)***.

T wave:

This represents ventricular repolarization. The amplitude of T-wave varies in each lead from 0.1mv up to 0.7mv. The right and left precordial T-waves are upright in 57% of newborns respectively. In adult, all the unipoler leads inscribe upright T-wave except avR and occasionally V1. A negative T-waves persists into early adulthood termed the Juvenile T-wave ***(Ishikawa et al., 1992)***.

U wave:

Represents a surface reflection of a negative after potential. The two prevailing concepts of the mechanism of the U wave include repolarization of the purkinje fibers and a mechanical event, presumably, ventricular relaxation. The U-waves is upright and its amplitude is 5 to 50% that of the T-wave. The tallest U-wave is recorded in leads V2 and V3 where its amplitude may reach 0.2mv ***(Ishikawa et al., 1992)***.

Q-T interval:

Represents the duration of activation and recovery of ventricular myocardium. Its timing is from the beginning of Q wave to the end of T-wave. Its duration is dependant on heart rate, increased heart rate shorten the QT interval.

* *QTC (Corrected QT interval)*:

Importance come from the fact that the normally of the QT interval can be determined only by the correction for the heart rate. It is calculated by Bazett's Formula:

QTc = QT/RR or QTc = QT / cycle length.

Its duration <0.44 sec. ***(Hodges et al., 1983)***.

Table (2): QT interval values corrected for heart rate

Measured RR interval (sec)	Heart rate (per min)	QT interval upper no mal limits (sec)
1.50	40	0.50
1.20	50	0.45
1.00	60	0.42
0.86	70	0.40
0.80	75	0.38
0.75	80	0.37
0.67	90	0.35
0.60	100	0.34
0.50	120	0.31
0.40	150	0.25

(Richard et al., 2003)

II- Electrocardiogram and left ventricular dysfunction

The value of electrocardiogram (ECG) in identifying heart failure due to left ventricular (LV) systolic dysfunction is evaluated by many researches. More cost effective approach to the diagnosis of suspected chronic heart failure is to use ECG as initial investigation, if the tracing is normal other diagnosis should be considered. If the ECG is abnormal, ECHO is indicated ***(Itliano et al., 2002)***.

Among patients with normal ECG, Left Ventricular Ejection Fraction (LVEF) >50% was found in 92% and abnormal ECG had high sensitivity 90% and modest specificity 34% in identifying patients with abnormal left ventricular functions. The presence of any of the following ECG abnormalities significantly increases the likelihood of a patient heaving an abnormal LVEF: T wave abnormalities, bundle branch block, left ventricular hypertrophy (LVH) and myocardial infarction ***(Baldasseroni et al., 2002)***.

Abnormal ECG include T-wave and ST segment changes (any ST segment deviation from iso-electric line 80m sec. after J point or any biphasic or inverted T waves in 10 standard leads after exclusion of avR and lead III), Q-wave >20m sec. in duration, bundle or fascicular block, LV hypertrophy, ventricular pacing rhythm or any other deviation from normal classified patient as having abnormal ECG. The prevalence of normal resting LVEF value was 96% in patients with normal resting ECG versus 75% for

patients with an abnormal resting ECG ***(Bode-Schnurbus et al., 2003)***.

Table (3): Variables significantly associated with left ventricular systolic dysfunction

	No.	LVEF ≥0.50	LVEF <0. 50
Rest ECG			
Normal	752	96%	4%
Abnormal	1.515	75%	25%

(Bode-Schnurbus et al., 2003)

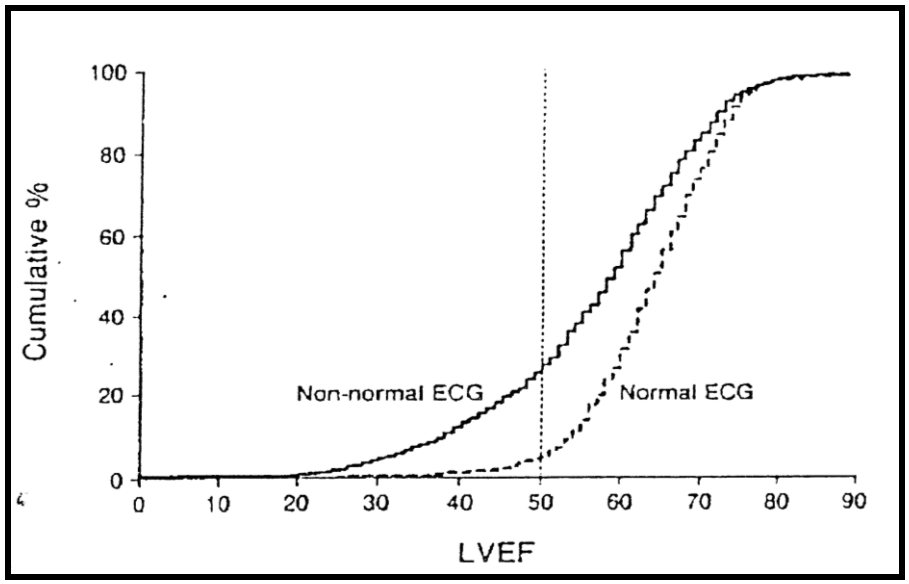

Figure (2): The cumulative frequency of left ventricular ejection fraction (LVEF) values for patients with either normal or abnormal rest electrocardiogram (ECG) ***(Bode-Schnurbus et al., 2003)***.

Participants with normal ECG, 98% had no evidence of left ventricular systolic dysfunction (high negative

predictive value) and of all participants with ECHO proved left ventricular systolic dysfunction, ECG abnormalities were observed in 54%. This value increases in those above the age of 70 years to 69% ***(Sandhu and Bahler, 2004)***.

P wave abnormalities:

Atrial conduction disturbances are frequently observed in dilated cardiomyopathic patients and may severely affect atrio-ventricular synchrony in the left heart by delaying the timing of the atrial activation and contraction. In a group of patients with left ventricular ejection fraction (LVEF) = 18 ± 6% referred for electro-physiologic and heamodynamic evaluation before pacing therapy as primary treatment for drug refractory congestive heart failure (CHF), the mean inter-atrial conduction time measured during spontaneous sinus rhythm was 102 ± 25m sec. (rang 75-130m sec) ***(Gerber et al., 2001)***.

Atrio-ventricular conduction defects:

Serial electrocardiographic changes in necropsy-proven idiopathic dilated cardiomyopathy were evaluated in 34 patients by Wilensky et al., seventy one percent of patients remained in stable sinus rhythm until the time of death, the other 29% developed permanent atrial tachyarrhythmia. In patients with stable sinus rhythm, the mean PR interval was nearly normal (on average 180 ± 30m sec) at entry into the study but prolonged (on average, 210 ± 30m sec) after a mean follow up period of 2.9 years ***(Nelson et al., 2000)***.

In other studies, including patients considered for pacemaker implantation to treat end stage CHF, the prevalence of PR interval >200m sec. varied from 30% to 53%. Individual values ranged from 100 to 400m sec. with an average of 209 ± 40m sec ***(Leclercq and Kass, 2002).***

The heamodynamic disorders resulting from ultra long PR intervals are well known in patients without structural heart disease. The early occurrence of left atrial (LA) contraction during the rapid or intermediate phase of left ventricular (LV) filling results in a complete or partial loss of LA contribution to LV filling ***(Douglas et al., 1989)***.

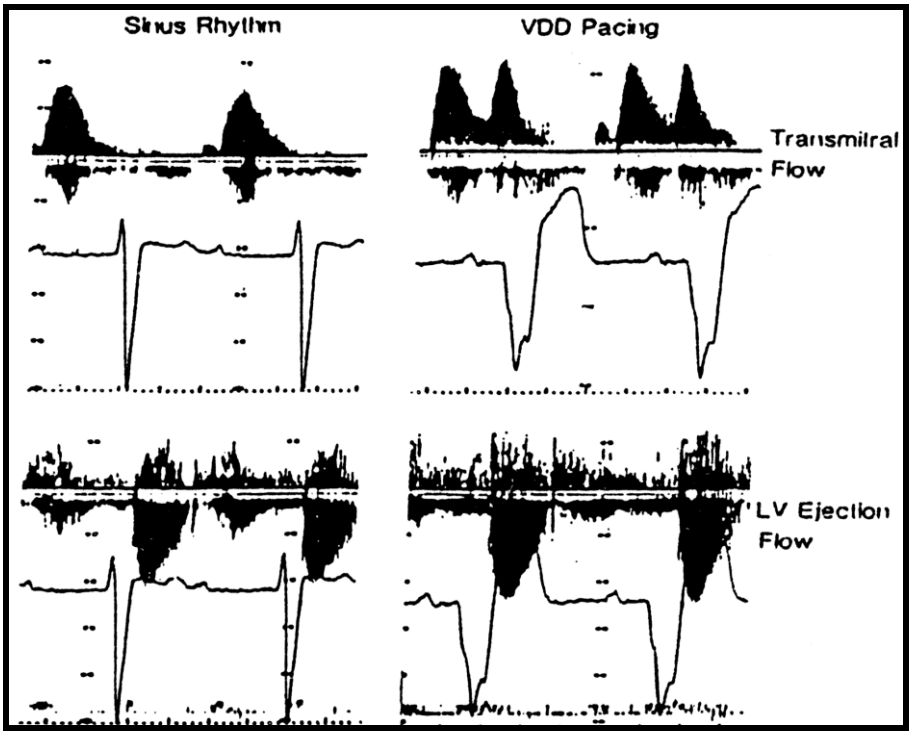

Figure (3): Deleterious consequences of ultra long PR interval alone in a patient without structural heart disease (left). VDD pacing resulted in normalization of transmitral and transaortic flow (right) ***(Charanjit et al., 1994)***.

Simultaneous LV filling time is shortened due to premature atriogenic (diastolic) closure of the mitral valve .Finally, this usually incomplete atriogenic closure and the frequent late reopening of the mitral valve in diastole may results in various degrees of end-diastolic (presystolic) mitral regurgitation i.e. the mitral valve retains an open midstream configuration during late diastole (after atrial contraction), which promotes regurgitation during the onset of ventricular systole ***(Gerald et al., 1993; Appleton et al., 1987; Hattle et al., 1989)***.

In DCM patients, several studies have clearly shown that moderately prolonged PR intervals (or even nearly normal PR intervals) could produce exactly the same haemodynamic consequence with the same degree of AV dyssynchrony as ultra long PR intervals in patients with an apparently normal heart. This apparent paradox can probably be explained by the high prevalence of severe intra-ventricular conduction block that results in delay and asynchronous LV contraction and relaxation in DCM patients ***(Lucas et al., 1983; Douglas et al., 2005)***.

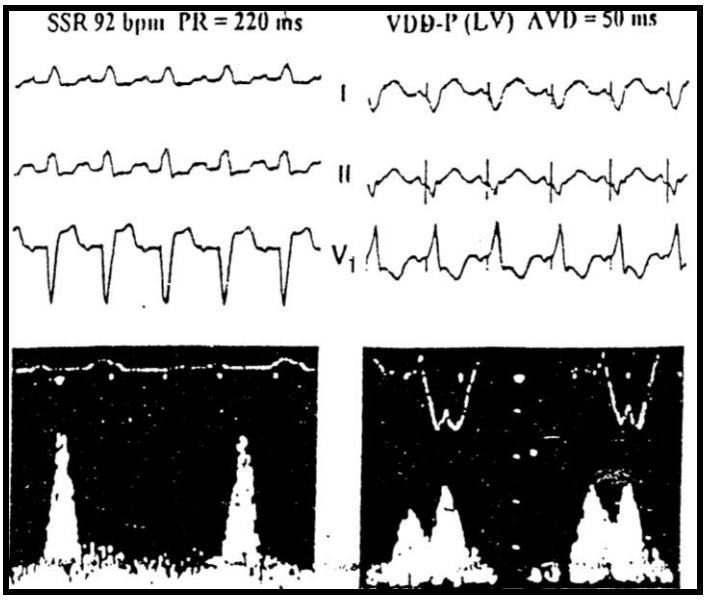

Figure (4): Example of "concealed long PR interval" in a patient with DCM. Despite nearly normal PR interval, transmitral Doppler flow shows a single summation pulse with very short diastolic filling time (left). VDD pacing at short AV delay improved mitral flow pattern with prolongation of filling time (right) ***(Charanjit et al., 1994)***.

Intra-ventricular conduction defects (IVCD):

Intra-ventricular conduction block is the general name given to a varied group of electrocardiographic entities. All share a common finding of some degree of delay in ventricular activation; recognition of these blocks hinges upon analysis of the QRS complex, as well as the ST-T changes associated with them. Bundle branch block (right or left), and fascicular block (left anterior or left posterior) are all examples of intra-ventricular conduction block. Causation of intra-ventricular conduction block may be cardiac or non-cardiac ***(Lapovitz and Pearson, 1987)***.

Intra-ventricular conduction delay with concomitant left ventricular (LV) dyssynchrony is common in patients with heart disease and/or ventricular pacing, and independently predicts sudden death in patients with heart failure (HF) ***(Tatsuji et al., 1992; D'Cruze; et al., 1991; Jing et al., 1997)***.

Moderate or severe mitral regurgitation was seen in 8% and 20% of patients with HF with QRS < 120 ms and ≥ 120 ms, respectively. Increasing QRS duration was also associated with more severe tricuspid regurgitation ***(Godly et al., 1981)***.

In the study conducted by ***Wilensky et al.*** 82% of patients had significant intra-ventricular conduction disturbances in the last ECG recorded within 60 days before death. Of the patients with the conduction abnormalities at the first examination, 68% had progressive disturbances in the time period studied. The mean QRS duration increased from 100 ± 20m sec. at the first examination to 130 ± 30m sec. at the end of follow up period ***(Nelson et al., 2000)***.

Complete bundle branch block was seen in 38% of patients, principally left bundle branch block (29%). Right bundle branch (9%) but associated with left axis deviation indicating probable associated left anterior fascicular block ***(Nelson et al., 2000)***.