The Prognostic Significance of QRS Complex Duration In Patients With ST Segment Elevation Myocardial Infarction Receiving Thrombolytic Therapy

Tarek Heussien El-Badawy[1]; Mohammed Mostafa Abd El-Salam Megahed[2]; and Mahmoud Abd ELhamied Etman[2]

[1]Cardiology Dep . [2]Critical Care Medicine Dep. Faculty of medicine University of Alexandria

Abstract: BACK GROUND: Complete right and left bundle branch block and advanced atrioventricular block present on admission electrocardiograms of patients with acute myocardial infarction, are associated with poor short and long-term outcome. Little is known about the impact of intermediate QRS prolongation (0.09–0.11 s) on the prognosis of acute myocardial infarction and its relation to the systolic function of the heart, **AIM OF THE WORK:** The aim of this study was to evaluate the importance of the QRS complex duration and its relations to mechanical performance of the heart as well as its in hospital prognostic significance in patients presented to Critical Care Department in Alexandria Main University Hospital with ST segment elevation myocardial infarction who received thrombolytic therapy. **PATIENTS**: This prospective study conducted on 30 adult patients of both sexes with ST segment elevation myocardial infarction who were admitted to Critical Care Department in Alexandria main university hospital and receiving thrombolytic therapy. **METHODS**: Beside regular treatment and monitoring Standard resting 12-lead electrocardiograms was obtained at the time of admission, after receiving thrombolytic therapy, daily and before discharge at 25 mm /s and 50mm /s and 1 mV/cm standardization and Echocardiographic examinations were also performed to all patients and data recorded. **RESULTS**: The 30 patients included in this study were divided into three groups according to the QRS maximum duration: **(Group A)**: patients with QRS max less than 90 msec **(n =5). (Group B)**: patients with QRS max between 90 to 110 msec **(n=17). (Group C)**: Patients with QRS max more than 110 msec **(n=8)**. There was a positive correlation between the QRS duration and the (EF,EDD,ESD,STD score) and there was a statistically significant difference between the mean of the QRS max in patients with and patients without complications. **CONCLUSION**: In patients with STEMI the QRS duration is a useful indicator of left ventricular systolic function and dimensions and it is easily measured. Also QRS duration is a good predictor of outcome in patients with STEMI.

[Tarek Heussien El-Badawy; **Mohammed Mostafa Abd El-Salam Megahed**; and **Mahmoud Abd ELhamied Etman. The Prognostic Significance of QRS Complex Duration In Patients With ST Segment Elevation Myocardial Infarction Receiving Thrombolytic Therapy.** Report and Opinion 2012;4(3):39-48]. (ISSN: 1553-9873). http://www.sciencepub.net/report. 7

Keywords: prognostic; QRS Complex; Patients; ST Segment; Myocardial Infarction Thrombolytic Therapy

INTRODUCTION:

Coronary heart disease (CHD) is a worldwide health epidemic. Acute coronary syndrome (ACS) is a unifying term representing a common end result. Acute ischemia is usually, but not always, caused by atherosclerotic plaque rupture, fissuring, erosion, or a combination with superimposed intracoronary thrombosis, and is associated with an increased risk of cardiac death and myonecrosis.[1,2]

ST segment elevation MI (STEMI)represents the most lethal form of acute coronary syndrome, one in which a completely occlusive thrombus results in total cessation of coronary blood flow in the territory of the occluded artery and the resultant ST-segment elevation on the ECG..[3,4]

The classic World Health Organization criteria for an acute myocardial infarction requires that two of the following three elements be present: (1) a history suggestive of coronary ischemia for a prolonged period of time (>30 minutes), (2) evolutionary changes on serial ECGs suggestive of myocardial infarction, and (3) a rise and fall in serum cardiac markers consistent with myonecrosis. Only two of the three criteria are needed because of the wide variability in the pattern of patient presentation with acute myocardial infarction.[5]

Pathophysiology:

Coronary atherosclerotic disease is the underlying substrate in nearly all patients with acute MI. The initiating event is a crack or fissure in the diseased arterial wall, which occurs as a result of loss of integrity of the plaque cap (the fibrous tissue overlying the plaque and partitioning the atheroma from the arterial lumen). The fissure or even frank plaque rupture

leads to exposure of subendothelial matrix elements such as collagen, stimulating platelet activation and thrombus formation.[6]

Diagnosis of the STEMI:

Acute MI is a clinical syndrome for which a constellation of subjective and objective parameters needs to be assessed. The diagnosis must be obtained rapidly and accurately, and misdiagnosis can have catastrophic sequelae.[7]

Ventricular Activation and the QRS Complex:

Normal ventricular activation is a complex event that is dependent on interactions between the physiology and the anatomy of the specialized ventricular conducting system and the ventricular myocardium. The net result is the multiphasic normal QRS complex.

Ventricular activation is the product of two temporally overlapping events, endocardial activation and transmural activation. Endocardial activation is guided by the anatomical distribution and physiology of the His-Purkinje system. The broadly dispersed ramifications of this tree-like (fractal) system and the rapid conduction within it result in the simultaneous activation of multiple endocardial sites and the depolarization of most of the endocardial surfaces of both ventricles within several milliseconds.[8]

The upper normal value for QRS duration is traditionally given as shorter than 120 milliseconds (and often as shorter than 110 milliseconds) measured in the lead with the widest QRS duration. Women, on average, have somewhat smaller QRS durations than men (by about 5 to 8 milliseconds). [9]

Conduction abnormalities at the sub-AV nodal level:

The bundle of His and the proximal and distal parts of the bundle branches are perfused by the septal branches from the LAD. The posterior fascicle of the left bundle branch is frequently also supplied by the posterior descending coronary artery (which may come from the RCA or CX).[10]

Conduction disturbances in the His bundle and the bundle branch system occurring in the setting of anterior wall infarction indicate a very proximal occlusion in the LAD. The presence of conduction disturbances in or below His means that a large area of the left ventricle is in jeopardy.[10]

The prognostic significance and clinical application of the prolonged QRS duration:

The conduction disturbances are associated with poor short and long-term prognosis of patients with acute myocardial infarction.[11] It is known that left bundle branch block (LBBB) leads to delayed mechanical activation of the left ventricle and that this delay causes derangements of the left ventricular (LV) systolic and diastolic functions.[12]

Delayed mechanical activation of the left ventricle might lead to intraventricular asynchrony, which in turn could induce important hemodynamic changes, especially in patients with structural heart disease.[12]

In patients with LV dysfunction and prior myocardial infarction, various electrocardiographic scoring systems have been developed to estimate LV function [13], but their utility has been limited.[14] However the presence of a nonspecific prolonged QRS duration (0.10 s) on a standard resting 12-lead ECG in the absence of typical features of bundle branch block is indicative of decreased resting LV systolic function.[15]

AIM OF THE WORK:

The aim of this study was to evaluate the importance of the QRS complex duration and its relations to mechanical performance of the heart as well as its in hospital prognostic significance in patients presented to Critical Care Department in Alexandria Main University Hospital with STEMI who received thrombolytic therapy.

PATIENTS:

This prospective study included 30 adult patients of both sexes with STEMI who were admitted to Critical Care Department in Alexandria main university hospital.

Inclusion criteria:

1. Presence of ST segment elevation (at least 1 millivolt) in two contiguous leads anatomically related to certain artery.
2. Patients with typical chest pain of more than 30 minutes and within 6 hours from the onset of pain.
3. Absence of contraindications of thrombolytic according to practice guidelines published by the American College of Cardiology and American Heart Association

Exclusion criteria: Patients with left bundle branch block, Grade three atrioventricular block,

Pre-excitation syndrome, Electrolyte disturbance and Patients on temporary or permanent pacemaker were excluded.

METHODS:

1. On admission every patient had been subjected to: History taking, Clinical data recording, Routine laboratory data, Electrocardiogram, CK-MB and troponine measurement and plain X-ray chest film.
2. Monitoring: Patients had been monitored and followed up till discharge regarding: Vital signs, chest and heart examination, Electrocardiogram, Cardiac enzymes: CK, CK-MB, Troponine and Chest x-ray.
3. Electrocardiogram:
   - Standard resting 12-lead electrocardiograms was obtained at the time of admission, after receiving thrombolytic therapy, daily and before discharge at 25 mm /s and 50mm /s with 1 mV/cm standardization, the widest QRS complex, narrowest QRS complex and the QTc segment duration in standard leads was manually measured. Only leads without extreme ST segment deviation will be considered.
   - The (QRS max) is the duration of widest QRS complex in all ECG leads.
   - The (QRS min) is the duration of the narrowest QRS complex in all ECG leads.
4. Echocardiography: Echocardiographic examinations were performed twice one on admission and another on the third day before discharge.
5. In hospital complications have been documented, including cardiac arrhythmia, congestive heart failure, cardiogenic shock, cardiac arrest and death.

RESULTS:

The 30 patients included in this study were divided into three groups according to the QRS maximum duration:

- **(Group A)**: patients with QRS max less than 90 msec **(n =5).**
- **(Group B)**: patients with QRS max between 90 to 110 msec **(n=17).**
- **(Group C)**: patients with QRS max more than 110 msec **(n=8)**.

Relation between QRS max before thrombolysis and gender: (table 1)

number of male patients in (group A) was three (15.8%) while number of female patients was two(18.2%), in (group B) number of male patients was eleven (57.9%) while number of female patients was six (54.5%) and in (group C) number of male patients was five (26.3%) while number of female patients was three (27.3%).

- There was statistically insignificant difference between the three groups as regards the gender of patients (p = 1.000).

Table (1): Relation between QRS max before thrombolysis and gender

	Gender				Test of sig.
	Male		Female		
	No.	%	No.	%	
QRS max before thrombolysis					
<90	3	15.8	2	18.2	
90-110	11	57.9	6	54.5	MCp = 1.000
>110	5	26.3	3	27.3	

MCp: p for Monte Carlo test

Comparison between the site of the infarction and the QRS max and the QRS min before thrombolysis revealed that: (table 2)

- There was statistically insignificant relation between the site of the infarction and leads that shows maximum duration of the QRS complex in ECG before thrombolysis (p=0.372).
- And also there was statistically insignificant relation between the site of the infarction and leads that shows minimum duration of the QRS complex in ECG before thrombolysis (p=0.795).

Table (2): Relation between site of the infarction and the QRS max and QRSmin.

	Site						MCp
	Anterior		Inferior		Lateral		
	No.	%	No.	%	No.	%	
QRS max.							
V1 – V6	19	100.0	6	85.7	4	100.0	
II, III, AVF	0	0.0	1	14.3	0	0.0	0.372
I, AVL	0	0.0	0	0.0	0	0.0	
QRS min.							
V1 – V6	3	15.8	0	0.0	0	0.0	
II, III, AVF	11	57.9	4	57.1	2	50.0	0.795
I, AVL	5	26.3	3	42.9	2	50.0	

MCp: p for Monte Carlo test
*: Statistically significant at $p \leq 0.05$

Relation between the success of the thrombolysis and the change of the QRS max before thrombolysis and before discharge. (Table 3)

The duration of the QRS max before thrombolytic therapy in patients with failed thrombolysis ranged from 88.0 to 118.0 msec with a mean of 106.14±10.64 msec and before discharge ranged from 88.0 to 118.0 msec with a mean 105.79±10.64 msec**.**

The duration of the QRS max before thrombolytic therapy in patients with successful thrombolysis ranged from 85.0 to 118.0 msec with a mean of 98.75 ± 11.02 msec and before discharge ranged from 85.0 to 116.0 msec with a mean 98.25±10.40 msec.

Comparison between the success of the thrombolysis and the change of the QRS max before thrombolysis and before discharge revealed that: (table3)

- There was statistically significant difference between the QRS max before thrombolysis and before discharge in patients with successful thrombolysis (p=0.041). The shortening of the QRS duration in patients with successful thrombolysis is statistically significant.
- There was statistically insignificant difference between the QRS max before thrombolysis and before discharge in patients with failed thrombolysis (p=0.174).

Table (3): Relation between the success of the thrombolysis and the change of the QRS max before thrombolysis and before discharge.

		Success	
		No	Yes
QRS max	**Before thrombolysis**		
	Range	88.0 – 118.0	85.0 – 118.0
	Mean ± SD	106.14 ± 10.52	98.75 ± 11.02
	Before discharge		
	Range	88.0 – 118.0	85.0 – 116.0
	Mean ± SD	105.79 ± 10.64	98.25 ± 10.40
	t (p)	1.439 (0.174)	2.236 .041)

- t: Paired t-test *: Statistically significant at $p \leq 0.05$

Relation between the three groups as regard the ECG parameters before thrombolytic therapy: Table (4)

Heart rate ranged from 60.0 to 98.0 b/sec with a mean of 79.60 ± 15.19 in (group A) ,it ranged from 60.0 to 95.0 b/sec with a mean of 81.18 ± 10.68 in (group B) while in (group C) it ranged from 95 to 113 b/sec with a mean of 110.63 ± 12.08 b/sec.

- Correlation test was done and showed a positive correlation between the QRS max and heart rate and was statistically significant (r= 0.656 p<0.001).

STDS (ST segment deviation score) ranged from 10.0 to 15.0 with a mean of 12.0 ± 1.87 in (group A), it ranged from 5.0 to 18.0 with a mean of 13.53 ± 3.84 in (group B) while in (group C) it ranged from 16.0 to 23.0 with a mean of 19.38 ± 2.33.

- Correlation test was done and showed a positive correlation between the QRS max and STDS and was statistically significant (r= 0.712 p<0.001).

The corrected QT interval (QTc) ranged from 0.40 to 0.44 sec with a mean of 0.41 ± 0.02sec in (group A), it ranged from 0.41 to 0.44 sec with a mean of 0.42 ± 0.01sec in (group B) while in (group C) it ranged from 0.40 to 0.43 sec with a mean of 0.42 ± 0.01sec.

- Correlation test was done and showed a slight positive correlation between the QRS max and QTc and was statistically insignificant (r= 0.133 p=0.551)

Table (4): Relation between QRS max and ECG parameters before thrombolysis

	QRS max before thrombolysis			r (p)
	<90	**90 -110**	**>110**	
STDS				
Range	10.0 – 15.0	5.0 – 18.0	16.0 – 23.0	0.712*
Mean ± SD	12.0 ± 1.87	13.53 ± 3.84	19.38 ± 2.33	(<0.001)
F (p)	10.967* (<0.001)			
p_1		0.366	<0.001*	
p_2			<0.001*	
HR				
Range	60.0 – 98.0	60.0 – 95.0	95.0 – 130.0	0.656*
Mean ± SD	79.60 ± 15.19	81.18 ± 10.68	110.63 ± 12.08	(<0.001)
F (p)	18.691* (<0.001)			
p_1		0.795	<0.001*	
p_2			<0.001*	
QTc				
Range	0.40 – 0.44	0.41 – 0.44	0.40 – 0.43	0.113 (0.551)
Mean ± SD	0.41 ± 0.02	0.42 ± 0.01	0.42 ± 0.01	
F (p)	1.008 (0.378)			
p_1		0.168	0.372	
p_2			0.640	

F: F test (ANOVA) p_1: p value of LSD test between <90 and other stages p_2: p value of LSD test between 90-100 and >110 r: Pearson coefficient *: Statistically significant at $p \leq 0.05$

Relation of QRS max before thrombolysis and Echo on admission. Table (5):

Ejection fraction in (group A) range from 50 to 60% with a mean 56.60 ± 4.45 in (group B) ranged from 40 to 62% with a mean 51.76 ± 7.41 and in (group C) EF ranged from 24 to 48% with a mean 36.38 ± 6.72.

There was a negative correlation between the QRS max and EF and was statistically significant (r=- 0.879 p<0.001)

ESD in (group A) range from 2.4 to 4.8cm with a mean 3.68 ± 0.86cm .In (group B) ranged from 2.6 to 5.4cm with a mean 3.85 ± 0.79cm and in (group C) ESD ranged from 4.1 to 6.1cm with a mean 4.98 ± 0.63cm.

- Correlation test was done showed a positive correlation between the QRS max and ESD and was statistically significant (r= 0.629 p<0.001)

EDD in (group A) range from 4.0 to 6.4cm with a mean 5.34 ± 0.90cm .In (group B) ranged from 4.3 to 6.9cm with a mean 5.59 ± 0.75cm and in (group C) EDD ranged from 5.5 to 6.8cm with a mean 6.15 ± 0.48cm.

- Correlation test was done and showed a positive correlation between the QRS max and EDD and was statistically significant (r= 0.473 p=0.008)

Left atrial diameter in (group A) range from 31 to 37mm with a 34.60 ± 2.51mm.In (group B) ranged from 33 to 43mm with a mean 36.65 ± 2.67mm and in (group C) ranged from 39 to 45mm with a mean 41.75 ± 2.25mm.

- Correlation test was done and showed a slight positive correlation between the QRS max and left atrial diameter and was statistically insignificant (r= 0.189 p=0.316).

Table (5):Relation between QRS max before thrombolysis and Echo on admission.

		QRS max before thrombolysis			r (p)
		<90 (n=5)	90-100 (n=17)	>110 (n=8)	
Echo on admission	**EF**				
	Range	50.0 – 60.0	40.0 – 62.0	24.0 – 48.0	-0.879* (<0.001)
	Mean ± SD	56.60 ± 4.45	51.76 ± 7.41	36.38 ± 6.72	
	F (p)	17.857* (<0.001)			
	p_1		0.178	<0.001*	
	p_2			<0.001*	
	ESD				
	Range	2.40 – 4.80	2.60 – 5.40	4.10 – 6.10	0.629* (<0.001)
	Mean ± SD	3.68 ± 0.86	3.85 ± 0.79	4.98 ± 0.63	
	F (p)	6.912* (0.004)			
	p_1		0.671	0.006*	
	p_2			0.002*	
	EDD				
	Range	4.0 – 6.40	4.30 – 6.90	5.50 – 6.80	0.473* (0.008)
	Mean ± SD	5.34 ± 0.90	5.59 ± 0.75	6.15 ± 0.48	
	F (p)	2.398 (0.110)			
	p_1		0.491	0.057	
	p_2			0.081	
	Left atrium				
	Range	36.0 – 40.0	35.0 – 40.0	34.0 – 42.0	0.189 (0.316)
	Mean ± SD	38.0 ± 1.58	36.71 ± 1.57	38.0 ± 2.78	
	F (p)	1.609 (0.219)			
	p_1		0.205	1.000	
	p_2			0.135	

F: F test (ANOVA) p_1: p value of LSD test between <90 and other stages
p_2: p value of LSD test between 90-100 and >110 .r: Pearson coefficient
Statistically significant at $p \leq 0.05$

Relation of QRS max before discharge and Echo before discharge: (table 6)

Another Echo was done on the third day revealing no significant different result compared to the first Echo, the result is shown in the following table.

Comparison between the three groups before thrombolysis as regards the incidence of complications revealed that: (table 7)

- There was statistically significant difference between the three groups as regards the incidence of arrhythmias (p=0.001), the signs of heart failure (p<0.001) and the incidence of developing pulmonary edema (p=0.003).
- There was statistically insignificant difference between the three groups as regards the incidence of shock (p=0.093) and mortality (p=0.084).

And with doing a comparison there was a statistically significant difference between the mean of the QRS max in patients with and patients without complications with p value (<0.001), as the incidence of complications increases with the increase in the QRS max (table 8).

Table (6): Relation of QRS max before discharge and Echo on the third day.

		QRS max before discharge			r (p)
		<90 (n = 5)	90-100(n = 17)	>110 (n = 8)	
Echo on the third day.	**EF**				
	Range	50.0 – 60.0	42.0 – 62.0	24.0 – 48.0	-0.702* (<0.001)
	Mean ± SD	56.60 ± 4.45	52.41 ± 6.64	38.0 ± 6.89	
	F (p)	17.550* (<0.001)			
	p_1		0.212	<0.001*	
	p_2			<0.001*	
	ESD				
	Range	2.40 – 4.80	2.60 – 5.40	4.10 – 6.10	0.495* (0.005)
	Mean ± SD	3.68 ± 0.86	3.79 ± 0.77	4.88 ± 0.66	
	F (p)	6.367* (0.005)			
	p_1		0.781	0.010*	
	p_2			0.002*	
	EDD				
	Range	4.0 – 6.40	4.30 – 6.90	5.50 – 6.80	0.379* (0.039)
	Mean ± SD	5.34 ± 0.90	5.59 ± 0.75	6.16 ± 0.50	
	F (p)	2.458 (0.105)			
	p_1		0.494	0.055	
	p_2			0.077	
	Left atrium				
	Range	36.0 – 40.0	35.0 – 40.0	34.0 – 42.0	0.129
	Mean ± SD	37.80 ± 1.48	37.12 ± 1.36	37.88 ± 2.70	(0.496)
	F (p)	0.593 (0.560)			
	p_1		0.468	0.943	
	p_2			0.340	

F: F test (ANOVA) p_1: p value of LSD test between <90 and other stages
p_2: p value of LSD test between 90-100 and >110
r: Pearson coefficient *: Statistically significant at $p \leq 0.05$

Table (7): Relation between QRS max before thrombolysis and complications.

	QRS max before thrombolysis						MCp
	<90 (n = 5)		90-110 (n = 17)		>110 (n = 8)		
	No.	%	No.	%	No.	%	
Arrhythmia	0	0.0	3	17.6	7	87.5	0.001*
Signs of HF	0	0.0	5	29.4	8	100.0	<0.001*
Shock	0	0.0	0	0.0	2	25.0	0.093
Pulmonary edema	0	0.0	0	0.0	4	50.0	0.003*
Mortality	0	0.0	0	0.0	2	25.0	0.084

MCp: p for Monte Carlo test Statistically significant at $p \leq 0.05$

Table (8): Relation between QRS max before thrombolysis and complications

		QRS max before thrombolysis		t(p)
		Absent	Present	

Complications	**Arrythmias**			
	Range	85.0 – 118.0	100.0 – 18.0	3.914* (0.001)
	Mean ± SD	97.55 ± 10.03	111.50 ± 7.15	
	Sings of HF			
	Range	85.0 – 105.0	100.0 – 118.0	8.464*
	Mean ± SD	94.0 ± 6.71	112.92 ± 5.09	(<0.001)
	Shock			
	Range	85.0 – 118.0	116.0 – 117.0	7.204*
	Mean ± SD	101.18 ± 10.94	116.50 ± 0.71	(<0.001)
	Pulm edema			
	Range	85.0 – 118.0	116.0 – 118.0	8.413*
	Mean ± SD	99.88 ± 10.24	117.25 ± 0.96	(<0.001)
	Mortality			
	Range	85.0 – 118.0	116.0 – 117.0	7.204*
	Mean ± SD	101.18 ± 10.94	116.50 ± 0.71	(<0.001)

t: Student t-test : Statistically significant at $p \leq 0.05$

DISCUSSION:

This study was conducted on 30 patients presented to the critical care department in Alexandria main university hospital with the diagnosis of ST-segment elevation myocardial infarction (STEMI) within 6 hours of chest pain and received thrombolytic therapy.

Effect of thrombolytic therapy on the QRS max:

In the present study comparing the ECG before discharge with the ECG on admission in our study, there was a significant change of the QRS duration (shortening) in patients with successful thrombolysis while there was no significant difference between the QRS duration before thrombolytic therapy and before discharge in patients with failed thrombolytic therapy.

Orhan et al, [16] in a study to show the effect of successful reperfusion on QRS duration showed that there was significantly longer QRS duration on admission and significantly less change in QRS duration in the 60th-minute ECG in impaired reperfusion group compared to reperfusion group and he related that to the idea of dynamic nature of QRS changes during ischemia and reperfusion,.[16]

Kengo et al, [17] in a study to show the clinical implications of intermediate QRS prolongation in the absence of bundle-branch block in patients with ST-segment-elevation acute myocardial infarction, found that the resolution of intermediate QRS prolongation within 24 h of successful reperfusion tended to be associated with lower 6-month mortality than did persistent intermediate QRS prolongation.[17]

The relation of the QRS and the extent of the myocardial infarction:

In the present study there was a significant difference between the three groups as regards the ST deviation score as patients in (group C) had the highest score, a correlation test was done and there was a positive correlation between the QRS max and the ST deviation score.

The importance of STD score was shown in a multivariate analysis from the GUSTO-I database of 41,021 patients it was found that the number of ECG leads showing ST segment deviation and the ST segment deviation score (using the sum of ST segment deviation in all 12 leads) are markers for the extent of the ischemic area in acute coronary syndromes, and they found association of prolonged QRS duration with increased mortality and it also reflects extensive infarcts and involvement of the interventricular conduction system.[18]

Relation of the QRS Max with Echocardiography:

In the present study there was a significant difference between the three groups as regards the EF, EDD, ESD.

In a similar study done by **Rachel et al,**[19] conducted on 270 consecutive patients, who were referred to the Nuclear Cardiology Laboratory for radionuclide exercise ventriculography , a standard resting 12-lead ECG was obtained for all the patients. The QRS duration in patients with an abnormal EF was significantly longer compared with that in patients with normal EF, In addition to EF, a prolonged QRS duration was associated with significantly increased LV ESD and LV EDD.[19]

On the other hand In the sight of the Valsartan in acute myocardial infarction (**VALIANT**) trial,[20] one of the largest long-term

study of survivors of acute MI; the trial enrolled 14,703 patients between 0.5 and 10 days following MI.[20]

A Baseline QRS duration was measured offline from single-lead electrocardiographic (ECG) tracings read from the echocardiograms. The VALIANT echocardiography cohort was divided into quartiles of baseline QRS duration (< 75 ms, 75-88 ms, 89-108 ms, > 108 ms), and these groups were related to clinical outcomes, as well as to changes in LV size and function.

Lakshminarayan et al,[21] found that a prolonged QRS duration at baseline was associated with larger ventricular volumes and reduced systolic function. However, following adjustment for other variables, including age, ejection fraction, and Killip class, the association of prolonged QRS duration for adverse cardiovascular outcomes lost significance. In addition, there were no appreciable differences in ventricular remodeling between the baseline QRS duration groups.[20-21]

The QRS duration and complications:

Comparison between the three groups before thrombolysis as regards the incidence of complications during the ICU stay revealed this:

There was statistically significant difference between the three groups as regards the incidence of arrhythmias, signs of heart failure and developing pulmonary edema, while there was statistically insignificant difference between the three groups as regards the incidence of developing shock and mortality.

Pudilb et al,[22] in a study to show the significance of intermediate QRS prolongation in patients with acute myocardial infarction done.[22]

The patients were divided according to QRS complex duration on admission 12-lead standard electrocardiogram to three groups: <0.09 s, 0.09–0.11 s and >0.11 s.

In hospital complications, including asystole, second and third degree atrioventricular block, ventricular tachycardia and fibrillation, paroxysmal atrial fibrillation, congestive heart failure and cardiogenic shock, were more common among patients with prolonged QRS duration, this difference reached statistical significance for asystole, congestive heart failure and cardiogenic shock.[22]

Another study was done by **elhendy et al**,[23] to assess the relation between QRS duration and mortality in patients with known or suspected coronary artery disease, the QRS duration was positively associated with an increased risk of death. This risk was observed throughout the entire range of QRS duration.[23]

Also **Lakshminarayan et al**.[21] in the sight of the valiant trial found that a lengthening QRS duration was associated with an increase risk of heart failure, sudden death, and cardiovascular death, respectively.[21]

CONCLUSION:

- In patients with STEMI the QRS duration is a useful indicator of left ventricular systolic function and dimensions and it is easily measured.
- QRS duration is a good predictor of outcome in patients with STEMI.
- Shortening of a prolonged QRS complex is a useful parameter for identifying successful thrombolysis in patients with acute STEMI.

***REFERENCES*:**

1. The American Heart Association. 2004 Heart and Stroke Statistical Update. Dallas, TX: American Heart Association, 2004.
2. Fuster V, Moreno PR, Fayad ZA, Corti R, Badimon JS. Atherothrombosis and high-risk plaque: part 1: evolving concepts. J Am Coll Cardiol 2005;46:937–54.
3. Myocardial infarction redefined-a consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol 2000;36:959–69.
4. Pedoe TH, Kuulasmaa K, Amouyel P, Davidson M, Mendis S, Tolonen H, et al. Myocardial infarction and coronary deaths in the World Health Organization MONICA Project. Circulation 1994; 90:583–612.
5. Canto JG, Every NR, Magid DJ, Rogers WJ, Malmgren JA, Frederick PD, et al. National Registry of Myocardial Infarction 2 Investigators. N Engl J Med 2000;342:1573–80.
6. Libby P. Molecular basis of the acute coronary syndromes. Circulation 1995;91:2844-50.
7. Pfeffer MA, Moye LA, Braunwald E, Basta L, Brown EJ, Cuddy TE, et al. Selection bias in the use of fibrinolytic

therapy in acute myocardial infarction. JAMA 1991;266:528-32.

8. Ramanathan C, Jia P, Ghanem R, Ryu K, Rudy Y. Activation and repolarization of the normal human heart under complete physiological condition. Proc Natl Acad Sci U S A 2006; 103:6309.
9. MacAlpin RN: In search of left septal fascicular block. Am Heart J 2002; 144:948.
10. Lie KI, Wellens HJ, Schuilenburg RM.. The conduction system of the heart, Philadelphia, Lea and Febiger 1976; 663-72.
11. Harpaz D, Behar S, Gottlieb S, Boyko V, Kishonsprint Y. Complete atrioventricular block complicating acute myocardial infarction in the thrombolytic era. J Am Coll Cardiol 1999; 34: 1721–8.
12. Xiao HB, Brecker SJ, Gibson DG. Effects of abnormal activation on the time course of the left ventricular pressure pulse in dilated cardiomyopathy. Br Heart J 1992; 68: 403–7.
13. Palmeri ST, Harrison DG, Cobb FR, Morris KG, Harrell FE, Ideker RE, et al. A QRS scoring system forassessing left ventricular function after myocardial infarction. N Engl J Med 1982; 306:4-9.
14. Fioretti P, Brower RW, Lazzeroni E, Simoons ML, Wijns W, Reiber JH, et al. Limitations of a QRS scoring system to assess left ventricular function and prognosis at hospital discharge after myocardial infarction. Br Heart J 1985; 53:248 –52.
15. Murkofsky RL, Dangas G, Diamond JA, Mehta D, Schaffer A, Ambrose JA. A prolonged QRS duration on surface electrocardiogram is a specific indicator of left ventricular dysfunction. J Am Coll Cardiol 1998; 32: 476-82.
16. Orhan M, Kacmaz F, Alyan O, Atak R, Senen K, Balbay Y, et al. A new electrocardiographic marker of successful myocardial reperfusion in patients undergoing primary angioplasty: resolution of QRS prolongation. Journal of Electrocardiology 2007; 40(4); 51.
17. Tsukahara K, Kimura K, Kosuge M, Shimizu T, Sugano T, Hibi K, et al. Clinical Implications of Intermediate QRS Prolongation in the Absence of Bundle-Branch Block in Patients With ST-Segment-Elevation Acute Myocardial Infarction. Circ J 2005; 69: 29 –34.
18. Zabel K, Pieper K, Hathaway W, Peterson E, Wagner G, Granger C, et al. For the GUSTO-I Investigators. Prognostic significance of the initial electrocardiogram in patients with acute myocardial infarction. JAMA 1998; 279:387.
19. Rachel L. Murkofsky R, George D, Joseph A., Davendra M, Abraham S, and John A. prolonged QRS duration on surface electrocardiogram is a specific indicator of left ventricular dysfunction. J Am Coll Cardiol 1998; 32;476-82.
20. Pfeffer M, McMurray J, Velazquez E, Rouleau J, Køber L, Maggioni A, et al. For the Valsartan in Acute Myocardial Infarction Trial Investigators. N Engl J Med 2003; 349:1893-906.
21. Lakshminarayan Y, Anavekar NS, Velazquez E, McMurray JJ, Skali H, Califf R, et al. Association of QRS duration and outcomes after myocardial infarction: The VALIANT trial. Circulation 2004; 110: III-725
22. Pudilb R, Feinberga MS, Hanoch H, Boykoa V, Mandelzweiga L. The prognostic significance of intermediate QRS prolongation in acute myocardial infarction. International Journal of Cardiology 2001; 78: 233–9.
23. Elhendy A, Stephen HC, Douglas MW, Patricia PA. Relation of QRS Duration on the Surface 12-Lead Electrocardiogram With Mortality in Patients With Known or Suspected Coronary Artery Disease. Am J Cardiol 2005; 96: 1082–8.

2/1/2012