DOI: https://doi.org/10.3329/cardio.v15i1.61908

Original Article

Relationship between QRS duration on ECG and Left Ventricular Systolic Function by Echocardiography in patients with Non-ST elevated Myocardial Infarction

Md. Zillur Rahman[1], Abdul Kader Akanda[2], Mohammad Ullah[3], Md. Shariful Alam[3], Md. Gulam Mostofa[2], Mohammad Mamoon Islam[4], Md. Iqbal Hossain[5], Md. Ahsanul Haque[3]

[1]Upazilla Health Complex, Gaforgaon, Mymensingh, [2]Department of Cardiology, Sir Salimullah Medical College, Dhaka, [3]Department of Cardiology, Dhaka Medical College, Dhaka, [4]Department of Cardiology, Mugda Medical College, Dhaka, [5]100 bedded Sadar Hospital, Shariatpur

Key Words : *Coronary artery disease, QRS duration, non-ST elevated myocardial infraction, Left Ventricular Systolic Function.*

Abstract:

Background: *Early risk stratification of patients with myocardial infarction is critical to determine optimum treatment strategies and enhance outcomes. The present study was therefore undertaken to determine the relationship between QRS duration (QRSd) on admission ECG and left ventricular ejection fraction (LVEF) as a measure of left ventricular function in non-ST elevated myocardial infarction (NSTEMI) patients.*

Methods: *This observational study was carried out from January to December 2020 with total of 120 patients with a history of NSTEMI. Based on the cut-off value of QRS duration 100 msec, the patients were divided into two groups – one group with QRS duration d" 100 msec (normal QRS) and another group with QRS duration > 100 msec (prolonged QRS). Left ventricular systolic function was considered preserved, if it was e" 52% and reduced if it was < 52%. The association and correlation between QRS duration and LVEF was then observed.*

Results: *The prevalence of reduced LVEF in patients with prolonged QRS duration (> 100 msec) was double (38%) than that of preserved (19.5%). The risk of having LV dysfunction in patients with prolonged QRS duration was 2.5 (95% CI = 1.1 – 6.2) times higher than that in patients normal QRS duration (d" 100 msec) (p = 0.039). The QRS duration and LVEF bear a significantly inverse relationship (r = -0.341, p < 0.001). The sensitivity of prolonged QRS duration (> 100 msec) in correctly detecting LV dysfunction was inappreciably low (38%), although its specificity in excluding those who did not have LV dysfunction was optimum (80.5%) with overall diagnostic accuracy being 52.5%.*

Conclusion: *Prolonged QRS duration on a standard 12-lead ECG is associated with reduced echocardiographic LVEF. However, QRS duration in predicting LV dysfunction is much less sensitive, although its specificity is optimum indicating that QRS duration is not a good predictor of LV dysfunction (reduced LVEF), but it can dependably predict those who do not have LV dysfunction (preserved LVEF).*

(Cardiovasc j 2022; 15(1): 36-41)

Introduction

Cardiovascular diseases (CVDs) are known to be the leading causes of death worldwide. In 2015, nearly 20 million CVD deaths occurred (equivalent to one-third of total global deaths) and 423 million people had prevalent CVD (1 in 17 of the global population).[1] In Bangladesh death due chronic diseases, especially the 'fatal four' i.e. Cardiovascular disease (CVD), cancer, chronic respiratory disease and diabetes is increasing at galloping pace.[2]

Acute coronary syndromes appeared as the leading cause (3.7%) of death across 504 public hospitals

Address of correspondence: Dr. Md. Zillur Rahman, Upazilla Health Complex, Gaforgaon, Mymensingh, Bangladesh.

in Bangladesh in 2012 as reported by the "Health Bulletin 2013".[3] Of them Non-ST segment elevation myocardial infarction (NSTEMI) is the commonest form of acute coronary syndrome (ACS) and is a leading global cause of premature morbidity and mortality.[4] The electrocardiogram, due to its wide availability, low cost and simplicity, has emerged as an essential tool for diagnostic and prognostic stratification of NSTEMI.[5]

ST segment depression or transient ST segment elevation and T wave changes occur in up to 50% of patients with NSTEMI. New ST-segment deviation (≥0.1 mV) is a useful measure of ischemia and prognosis. Transient (i.e., <20 minutes) ST elevation, which occurs in approximately 10% of patients with UA/NSTEMI, portends a high risk of future cardiac events. T wave changes are sensitive but not specific for acute ischemia unless they are marked (>0.3 mV).[6]

Widening of the QRS complex is related to slower spread of ventricular depolarization, either due to disease of the His-Purkinje network and/or reliance on slower, muscle-to- muscle spread of depolarization. Shape of an abnormal QRS complex varies from almost normal to wide and bizarre and/or slurred and notched. Causes of a widened QRS complex include right or left BBB, pacemaker, hyperkalemia, ventricular pre-excitation as is seen in Wolf-Parkinson-White pattern, and a ventricular rhythm The QRS duration determined by electrocardiography (ECG) is related to ventricular dysfunction. Prolonged QRS duration can lead to ventricular dysfunction in long-term and also it can be a direct result of ventricular dysfunction. Additionally, it was found that prolonged QRS duration was related to poor prognosis in anterior AMI.[7] In previous studies, it was shown that prolonged QRS on admission was related to cardiac adverse events.[8,9] Many studies documented the prognostic value of ECG with LV systolic function by echocardiography, where prolonged QRS duration of acute STEMI patients demonstrated significant association with left ventricular systolic dysfunction.[10] Another study showed that patients with prolongation of QRS duration had increased ventricular volume, decreased left ventricular ejection fraction (EF), and higher incidence of sudden cardiac death.[11]

But there are limited studies to show the correlation between QRS duration on ECG and LV systolic function by echocardiography in patients with NSTEMI in Bangladesh. The present study was, therefore designed to see the relationship between QRS duration on surface ECG and left ventricular systolic function in patients with Non-ST elevated Myocardial Infarction.

Methods:

This study was designed as an observational study at Department of Cardiology, Sir Salimullah Medical College & Mitford Hospital, Dhaka. This study was conducted from January 2020 to December 2020. A total number of 120 patients who fulfilled inclusion and exclusion criteria were selected for the study as the sample population. The samples were collected by purposive sampling method. Patients with Previous history of myocardial infarction, known non-ischemic causes which can cause prolonged QRS duration (WPW syndrome, drugs, electrolyte imbalance), LBBB or RBBB, Pacemaker rhythm, known valvular heart disease, congenital heart disease and cardiomyopathy, major non-coronary disorders which cause elevation of troponin-I such as CKD, myocarditis, acute pulmonary embolism were excluded from this study.

Informed written consent was taken from each patient before enrollment. Meticulous history was taken and detailed clinical examination was performed. Risk factors profile including smoking, hypertension, dyslipidemia and family history of myocardial infarction were noted. Necessary physical examinations were done including pulse, blood pressure, jugular venous pressure, basal crepitation, auscultation for any cardiac murmur. Some primary investigations were done including serum troponin value, random blood sugar, serum creatinine, serum electrolytes, lipid profile on the day of admission. Resting ECG of all patients was done at a paper speed of 25 mm/s and 10 mm standardization at admission using Fukuda ECG machine (Model: FX -2111) Denshi Co Ltd Japan. QRS duration was manually measured and calculated from the beginning of the first appearing Q or R wave to the end of the S wave. All measurements were taken from the precordial leads (V1 to V6). Average of measurements from

all precordial leads was considered. Based on the cut-off value of QRS duration 100, the patients were divided into two groups – one group with QRS duration ≤100 msec (normal QRS, 82 patients) and another group with QRS duration > 100 msec (prolonged QRS, 38 patients). Trans thoracic echocardiography was done preferably within 24 hours of admission, left ventricular systolic function was measured in terms of left ventricular ejection fraction (LVEF). LVEF was measured with the help of modified Simpson's method. Accordingly, LV systolic function was considered normal, if left ventricular ejection fraction was ≥52%. ECHO done by GE Vivid E95 Machine. Collected data were processed and analyzed using SPSS (Statistical Package for Social Science), version 23.0

Results:

This observational analytical study aimed at finding the relationship between QRS duration and left ventricular systolic function in patients with NSTEMI. The exposure or the independent variable (QRS duration on admission ECG) was divided into ≤100 msec and > 100 msec, while the outcome or dependent variable, LVEF was divided into < 52% (reduced, n = 79) and ≥52% (normal, n = 41). The association & correlation between QRS duration and LVEF was then observed. The findings obtained from data analyses are presented below:

Nearly half (49.2%) of the patients had family history of CAD, 44.2% were hypertensive, 30.8% diabetic, 38.3% smoker and 10% were dyslipidemia (Fig. 1).

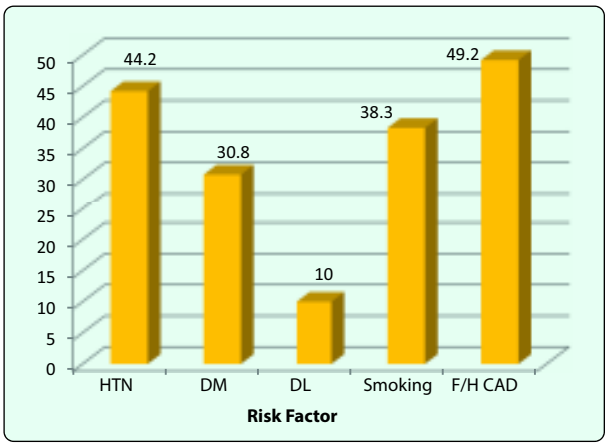

Fig.-1 : *Distribution of risk factors among the sampled population.*

The mean age of the NSTEMI patients with prolonged QRSd was significantly lower than that of the patients with normal QRSd (p = 0.008). However, sex distribution was almost identical between the groups (p = 0.497). BMI was no different between the groups (p = 0.224). None of the conventional risk factors of coronary artery diseases were significantly different between the groups (p > 0.05) (table I).

Table-I

Distribution of baseline characteristics between prolonged and normal QRS duration.

Baseline characteristics	QRSd (msec)		p value
	Prolonged (n = 38)	Normal (n = 82)	
Male	30(78.9%)	60(73.2%)	0.497
Age (yrs.)	53.7 ± 9.2	58.9 ± 10.7	0.008
BMI (kg/m2)	27.9 ± 4.1	27.1 ± 3.3	0.224
Hypertension	15(39.5%)	38(46.3%)	0.481
Diabetes Mellitus	11(28.9%)	26(31.7%)	0.761
Dyslipidemia	5(13.2%)	7(8.5%)	0.647
Smoking habit	18(47.4%)	28(34.1%)	0.166
Family history of CAD	21(55.3%)	38(46.3%)	0.363

Figures in the parentheses indicate corresponding %; Chi-squared Test was done to analyze the qualitative data. Quantitative data were analyzed using unpaired t-Test and were presented as mean ± SD.

None of the co-morbidities or factors (hypertension, diabetes mellitus, dyslipidemia, smoking habit and family history CAD) shown in table II was significantly associated with LV dysfunction (reduced LVEF). However, smoking habit was considerably higher in patients with reduced LVEF (p = 0.062).

Table-II

Association between co-morbidities or risk factors and LVEF.

Risk factors*	LVEF (%)		p value
	< 52(n = 79)	≥52(n = 41)	
Hypertension	33(41.8)	20(48.8)	0.463
Diabetes	22(27.8)	15(36.6)	0.326
Dyslipidemia	6(7.6)	6(14.6)	0.369
Smoking habit	35(44.3)	11(26.8)	0.062
Family history of CAD	41(51.9)	18(43.9)	0.406

Figures in the parentheses indicate corresponding %; *Chi-squared Test was done to analyze the data.

The prevalence of reduced LVEF (< 52%) with prolonged QRS duration was double (38%) than that of preserved LVEF (≥52%) (19.5%). The risk of having LV dysfunction in patients with prolonged QRS duration was 2.5 (95% CI = 1.1 – 6.2) times higher than that in patients normal QRS duration (≤100 msec) (p=0.039) (Table III).

Table-III

Association between prolonged QRS duration and LVEF.

QRS duration (msec)	LVEF (%)		(95% CI of OR)	*p-value
	< 52 (n = 79)	≥52 (n = 41)		
> 100	30 (38.0%)	8 (19.5%)	2.5 (1.1 - 6.2)	0.039
≤100	49 (62.0%)	33 (80.5%)		

Figures in the parentheses indicate corresponding %; *Chi-squared Test (?2) was done to analyzed the data.

Correlation between QRS duration and LVEF depicts that as QRS duration increases LVEF decreases. The two variables bear a significantly inverse relationship (r = - 0.341, p < 0.001) (Fig.2).

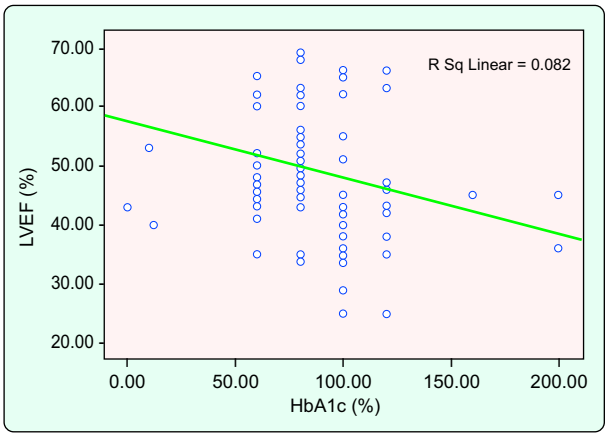

Fig 2: *Correlation between QRS duration and LVEF.*

Table IV shows the accuracy of QRS duration as a non-invasive test in differentiating NSTEMI patients with LV dysfunction from normal LV function. The sensitivity of prolonged QRS duration (> 100 msec) in correctly detecting LV dysfunction from the normal ones was 30/79×100 = 38%, while the specificity of the test in correctly excluding those who did not have LV dysfunction was 33/41×100 = 80.5%. The positive and negative predictive values of the test were 30/38 × 100 = 78.9% and 33/82×100 = 40.2% respectively. The percentages of false positive and false negative as yielded by the test were 8/38×100 = 21.1% and 49/82×100 = 59.8% respectively. The overall diagnostic accuracy of the test was (30 + 33)/100×100 = 52.5%.

Table-IV

Accuracy of QRS duration in detecting LV dysfunction.

QRS duration (msec)	LV Dysfunction		Total
	Yes (LVEF < 52%)	No (LVEF ≥52%)	
> 100	30	8	38
≤100	49	33	82
Total	79	41	120

Sensitivity = 38%, Specificity = 80.5%, Positive predictive value of the test (PPV) = 78.9%, Negative predictive value of the test (NPV) =40.2%, Percentage of false positive = 21.1%, Percentage of false negative = 59.8%

Discussion:

Despite decreasing mortality trends of coronary artery disease (CAD) in many developed countries, increasing number is noticed in developing countries.[12] The ECG, due to its wide availability, low cost and simplicity, is an essential tool for the diagnosis and prognostic stratification of STEMI.[13] Whether, the same tool can be used in the prediction of left ventricular systolic dysfunction in patients with NSTEMI has been tested in the present study

In this study prolonged QRS duration (> 100 msec) on a standard 12-lead ECG was associated with reduced LVEF, as determined by echocardiography. The high specificity of the 12-lead ECG for the prediction of abnormal LV systolic function suggests that in patients with QRS duration > 100 msec, the resting LVEF is more likely to be abnormal. The resting 12-lead ECG is readily available for all patients with suspected or proven cardiac disease. Although valuable information to diagnose the rhythm and presence or absence of acute or remote myocardial infarction and left ventricular hypertrophy is evident in the resting ECG, the utility of the QRS duration as a predictor of LV systolic function has

long been ignored. Previous studies indicated that a normal 12-lead resting ECG is associated with normal LV function in 92-95% of cases.[14-17]

QRS scores incorporating several ECG variables have been devised by several investigators. Palmeri et al. used a 29-point system based on the duration of Q and R waves and on the ratios of R-to-Q amplitude and R- to-S amplitude. Palmeri showed certain QRS scores to be proportional to the severity of wall–motion abnormalities (determined by radionuclide gated blood pool scanning) and to have inverse correlations with measured LVEF.[18] Roubin et al. using the same scoring system showed that a QRS score of ≥7 had a specificity of 97% and a sensitivity of 59% for predicting a reduced LVEF of 45%.[19] However, the utility of such scoring systems has been questioned by other investigators. Fiorettiet al. showed that QRS score of Wagner et al. was of little use in estimating LVEF.[20,21]

Askenazi et al. demonstrated that the sum of the R-waves in leads aVL, aVF, and v1 to v6 correlated with the LVEF, and an R-wave sum of 4 mV was the best predictor of a decreased EF.[22] Young et al. found that the correlation between the modified QRS score of Wagner et al. and LVEF to be only fair, and the sum of R-wave voltage criterion of Askenazi et al. to correlate poorly with LVEF.[23, 22] Although depressed LVEF is associated with a prolonged QRSd, it is also possible that prolonged QRS duration (even in the normal ranges) could directly contribute to worsen the prognosis by causing ventricular asynchrony, functional mitral regurgitation and left ventricular dysfunction.[24]

In the present study, the correlation between QRS duration and resting LVEF was tested outright and QRS duration was found to be negatively correlated with resting LVEF, that is, left ventricular systolic function. However, as accuracy of prolonged QRS duration (> 100 msec) in predicting LV dysfunction was tested, its sensitivity was found to be inappreciably low (38%), although its specificity was optimum indicating that QRS duration is not a good predictor of LV dysfunction (reduced LVEF) but it can dependably predict those who do not have LV dysfunction (preserved LVEF). Murkofsky et al. also found a prolonged QRS (> 100 msec) to be highly specific, but relatively insensitive, for predicting LV dysfunction.[25] Thus, although a QRS duration > 100 msec was highly associated with an abnormal resting LVEF, a normal QRS duration of d" 100 msec did not reliably rule out reduced LVEF. Furthermore, correlation between QRS duration and LVEF suggests that more the prolonged QRS duration the greater the worsening of LVEF (the severe the LV dysfunction). However, every scientific study might be associated with some inherent biases and limitations. Likewise, there were several limitations of the present study, which deserve mention.

Limitations

The main limitation of our study is the manually measurement of QRS duration. This might have reduced the accuracy. The present study did not use other described ECG scoring systems that had been proven useful in the determination of infarct size and estimation of LV function shortly after a myocardial infarction.

Conclusion:

Prolonged QRS duration on a standard 12-lead ECG is associated with reduced echocardiographic LVEF. The QRS duration bears negative correlation with resting LVEF suggesting that as QRS duration increase LVEF decreases. However, QRS duration in predicting LV dysfunction is much less sensitive, although its specificity is optimum indicating that QRS duration is not a good predictor of LV dysfunction, but it can dependably predict those who do not have LV dysfunction.

Conflict of Interest - None.

References:

1. Roth GA, Johnson C, Abajobir A, et al. Global, Regional, and National Burden of Cardiovascular Diseases for 10 Causes, 1990 to 2015. J Am Coll Cardiol. 2017; 70(1):1-25. doi:10.1016/j.jacc.2017.04.052
2. Islam A, Majumder A. Coronary artery disease in Bangladesh: A review. Indian Heart J. 2013;65(4):424-435. doi:10.1016/j.ihj.2013.06.004
3. Al Mamun M, Rumana N, Pervin K et al. Emerging Burden of Cardiovascular Diseases in Bangladesh. J Atheroscler Thromb. 2016;23(4):365-375. doi:10.5551/jat.30445
4. Carrick D, Oldroyd KG, McEntegart M, et al. A randomized trial of deferred stenting versus immediate stenting to prevent no- or slow-reflow in acute ST-segment elevation myocardial infarction (DEFER-STEMI). J Am Coll Cardiol. 2014; 63(20):2088-2098. doi:10.1016/j.jacc.2014.02.530

5. de Luna AB, Zareba W, Fiol M, et al. Negative T wave in ischemic heart disease: a consensus article. Ann Noninvasive Electrocardiol. 2014; 19(5):426-441. doi:10.1111/anec.12193

6. Lilly, L.S. and Braunwald, E., 2012. Braunwald's heart disease: a textbook of cardiovascular medicine (Vol. 2). Elsevier Health Sciences.

7. Roe MT, Ohman EM, Maas AC, et al. Shifting the open-artery hypothesis downstream: the quest for optimal reperfusion. J Am Coll Cardiol. 2001; 37(1):9-18. doi:10.1016/s0735-1097(00)01101-3

8. Açil T, Türköz R, Açil M, et al. Value of prolonged QRS duration as a predictor of low cardiac output syndrome in patients with impaired left ventricular systolic function who undergo isolated coronary artery bypass grafting. Am J Cardiol. 2006; 98(10):1357-1362. doi:10.1016/j.amjcard.2006.06.031

9. Abdelnoor M, Nitter-Hauge S, Risum O, Svennevig JL, Amlie JP. Duration of preoperative electrocardiographic QRS complex and the incidence of heart arrest after aorto coronary bypass surgery. Scand Cardiovasc J. 2000; 34(2):186-191. doi:10.1080/14017430050142233

10. Patwary MSR, Reza AQM, Mohibullah AKM. Prolonged QRS duration in AMI patients, Indicative of Left Ventricular Systolic Dysfunction. Chest and Heart journal 2007; 31(1):36-40.

11. Yerra L, Anavekar N, Skali H, et al. Association of QRS duration and outcomes after myocardial infarction: the VALIANT trial. Heart Rhythm. 2006; 3(3):313-316. doi:10.1016/j.hrthm.2005.11.024

12. Bhatnagar P, Wickramasinghe K, Williams J, Rayner M, Townsend N. The epidemiology of cardiovascular disease in the UK 2014. Heart. 2015; 101 (15):1182-1189. doi:10.1136/heartjnl-2015-307516

13. Guía de práctica clínica de la ESC 2013 sobre estimulación cardiaca y terapia de resincronización cardiaca. Revista Española de Cardiología. 2014; 67 (1):58.e1-58.e60. doi:10.1016/j.recesp.2013.12.001

14. Swartz MH, Pichard AD, Meller J, Teichholz LE, Herman MV. The normal electrocardiogram as a predictor of left ventricular function in patients with coronary artery disease. Br Heart J. 1977; 39 (2):208-211. doi:10.1136/hrt.39.2.208

15. O'Keefe JH Jr, Zinsmeister AR, Gibbons RJ. Value of normal electrocardiographic findings in predicting resting left ventricular function in patients with chest pain and suspected coronary artery disease. Am J Med. 1989;86(6 Pt 1):658-662. doi:10.1016/0002-9343(89)90439-7

16. Rihal CS, Davis KB, Kennedy JW, Gersh BJ. The utility of clinical, electrocardiographic, and roentgenographic variables in the prediction of left ventricular function. Am J Cardiol. 1995;75(4):220-223. doi:10.1016/0002-9149(95)80023-l

17. Christian TF, Chareonthaitawee P, Miller TD, Hodge DO, Gibbons RJ. Is itnecessary to measure resting left ventricular function in patients with normal resting electrocardiograms? Circulation 1996; 94: 11-20.

18. Palmeri S, Harrison D, Cobb F et al. A QRS Scoring System for Assessing Left Ventricular Function after Myocardial Infarction. New England Journal of Medicine. 1982; 306(1):4-9. doi:10.1056/nejm198201073060102

19. Roubin GS, Shen WF, Kelly DT, Harris PJ. The QRS scoring system for estimating myocardial infarct size: clinical, angiographic and prognostic correlations. J Am Coll Cardiol. 1983; 2(1):38-44. doi:10.1016/s0735-1097(83)80374-x

20. Fioretti P, Brower R, Lazzeroni E et al. Limitations of a QRS scoring system to assess left ventricular function and prognosis at hospital discharge after myocardial infarction. Heart. 1985; 53(3):248-252. doi:10.1136/hrt.53.3.248

21. Wagner G, Freye C, Palmeri S et al. Evaluation of a QRS scoring system for estimating myocardial infarct size. I. Specificity and observer agreement. Circulation. 1982; 65(2):342-347. doi:10.1161/01.cir.65.2.342

22. Askenazi J, Parisi AF, Cohn PF, Freedman WB, Braunwald E. Value of the QRS complex in assessing left ventricular ejection fraction. Am J Cardiol. 1978; 41(3):494-499. doi:10.1016/0002-9149(78)90005-x

23. Young SG, Abouantoun S, Savvides M, Madsen EB, Froelicher V. Limitations of electrocardiographic scoring systems for estimation of left ventricular function. J Am Coll Cardiol. 1983; 1(6):1479-1488. doi:10.1016/s0735-1097(83)80052-7

24. Rouleau F, Merheb M, Geffroy S, et al. Echocardiographic assessment of the interventricular delay of activation and correlation to the QRS width in dilated cardiomyopathy. Pacing Clin Electrophysiol. 2001; 24(10):1500-1506. doi:10.1046/j.1460-9592.2001.01500.x

25. 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 [see comment]. J Am Coll Cardiol. 1998; 32(2):476-482. doi:10.1016/s0735-1097(98)00242-3