

A Study on Systolic Dyssynchrony Index on Predicting Arrhythmias in Heart Diseases

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ABSTRACT

Cardiac disorders are the most common cause of death. The condition with its chronicity can bring the patient back for consultation with the manifestation as arrhythmias. Myocardial infarction and cardiomyopathy are amongst the cause of highest arrhythmogenic potential in heart conditions. This study focuses on easing the prediction value of arrhythmias in patients post MI and DCMP. Systolic Dyssynchrony Index or SDI addresses the lacuna of discharging patients with an anti-arrhythmic at the OPD or emergency set-up before or during the presentation of arrhythmia.

Keywords- “MI-Myocardial Infarction”, “DCMP-Dilated Cardiomyopathy”, “SDI-Systolic Dyssynchrony Index-SDI”, “LVMD-Left Ventricular Mechanical Dyssynchrony”, “LVH-Left Ventricular Hypertrophy”, “HOCM-Hypertrophic Obstructive Cardiomyopathy”, “LVD-Left Ventricular Dysplasia”, “CRT-Cardiac Resynchronisation Therapy”, “ISI-Isolated Systolic Index”

INTRODUCTION

Cardiac disorders are common in the developing world and also in the West. This condition with its chronicity can eventually terminate in arrhythmias. Hence with the starting point as Myocardial Infarction, the ischemic types of cardiomyopathy⁵ are making heads turn. The investigation of choice for the same being ECHO 3D heart takes time for evaluation. Echocardiographic assessment of left ventricular mechanical dyssynchrony (LVMD)² status post MI or LVH or HOCM or LVD received great interest with the appearance of Cardiac resynchronization therapy ever since the first successful implants. Recent guidelines still keep QRS duration as the main selection criterion for diagnosing the presence of LVMD. However, measurement of QRS duration, which is an electrical phenomenon, seems to provide only a crude estimate on myocardial activation and is poorly correlated with the presence of LVMD². Echocardiography seems to be a more reliable tool for correctly identifying candidates for CRT and thus reducing the number of clinical non-responders.

Recently LVMD was found to be associated with other cardiac and noncardiac diseases. Therefore, echocardiographic assessment of LVMD will always remain of importance. The lacuna here is that a formula by ECHO and ECG parameters to calculate SDI is not available.

Patients with end-stage heart failure status post MI manifesting with a wide QRS complex in ECG are considered candidates for cardiac resynchronization therapy (CRT). However, 20% to 30% of patients do not respond to CRT. Presence of left ventricular dyssynchrony may explain the nonresponse.¹ From 30% to 40% of heart failure patients with QRS duration >120 ms exhibit left ventricular dyssynchrony, which may explain the nonresponse to CRT. Echocardiography has played an essential role in Cardiac resynchronization therapy (CRT) ever since the first successful implants whether it were for the estimation of left ventricular ejection fraction (LVEF), LV volumes, grade of mitral regurgitation, etiology of heart failure (HF), or the assessment of left ventricular mechanical dyssynchrony (LVMD). In recent guidelines, QRS duration still remains the main selection criterion for diagnosing the presence of LVMD in clinical practice. This is mainly because the large prospective, randomized trials solely relied on the QRS width as the only marker for dyssynchrony.¹ However, measurement

of QRS duration, which is an electrical phenomenon, seems to provide only a crude estimate on myocardial activation and is poorly correlated with the presence of mechanical dyssynchrony.²

In contrast, echocardiography allows the severity of mechanical dyssynchrony and its impact on cardiac hemodynamics to be assessed in a quantitative manner. It therefore seems to be a more reliable tool for correctly identifying candidates for CRT and thus reducing the number of clinical non-responders.

At first glance, echocardiographic assessment of LVMD seems to be time consuming, requiring special expertise, and the optimal protocol for dyssynchrony assessment has not yet been defined. However, important information about the presence and severity of dyssynchrony can be obtained from conventional echocardiographic techniques and dyssynchrony assessment can be easily integrated into daily routine practice.¹

The role of echocardiography in assessing LVMD in CRT patients remains controversial to date. The Predictors of Response to Cardiac Resynchronization Therapy (PROSPECT) trial examined the predictive value of many echocardiographic parameters of dyssynchrony (Doppler, M-mode, tissue Doppler imaging [TDI], and delayed longitudinal contraction) on LV reverse remodeling and composite clinical score. It concluded that given the modest sensitivity and specificity in this multicenter setting despite training and central analysis, no single echocardiographic measure of dyssynchrony may be recommended to improve patient selection for CRT beyond current guidelines. However, another study from three experienced centers in dyssynchrony assessment reproduced a positive role for TDI. In that study, three parameters derived from 12 LV segments and septal-to-lateral wall delay predicted LV reverse remodeling and improvement of LV ejection fraction.³

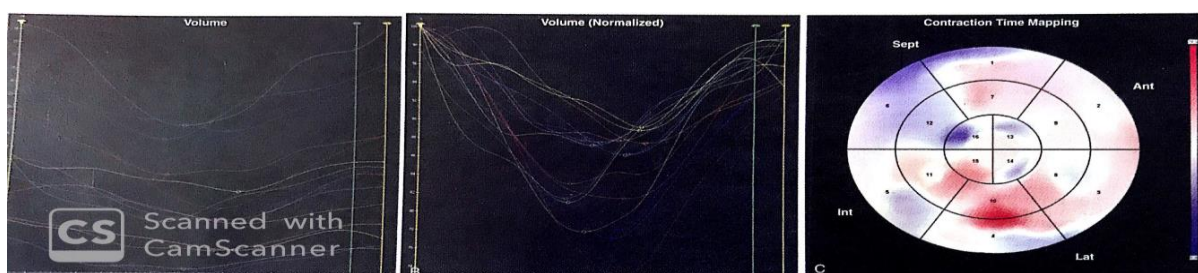
Three different levels of dyssynchrony can be distinguished by echocardiography: (1) Atrioventricular dyssynchrony: delayed ventricular activation in relation to the atria owing to prolongation of the PR interval. (2) Interventricular dyssynchrony: delayed onset and end of LV systole due to delayed LV electrical activation in comparison to the RV. (3) Intraventricular dyssynchrony: delayed activation of some LV segments with prolonged contraction after aortic valve closure.⁷

The first two levels can be easily identified by conventional echocardiography. Tissue Doppler tissue imaging (TDI) is currently regarded as the most sensitive technique for quantification of intraventricular dyssynchrony.⁴

METHODS

Patients with valvular lesions and cardiomyopathy status post angina/myocardial infarction were observed in the ward over a duration of three days. Serial ECG's and 2nd day ECHO of 36 patients was observed without taking age and sex as confounding factors. This was an cross sectional randomised control trial with a prospective motive. With respect to easing the cost-effectiveness of the procedure i.e. devise formulae to calculate SDI or ISI of left ventricle, a linear measurement of an average of height difference between qrs waves w.r.t. blood volume between the left ventricle and right ventricle by ECG and ECHO was necessary so as to ease the prognostication in arrhythmogenic heart. Patients included were those with acute coronary syndromes, acute valvular lesions, clients with decompensated heart failure. Stroke and burns patients were excluded from the study. Those included had wide or narrow QRS or those previously on cardiac pacemakers.

RESULTS AND DISCUSSION



Parameters of Heart Rate(bpm/ms), QT prolongation(ms), PR interval and PP interval (ms) by ECG were studied in this prospective trial. Also Stroke volume and Ejection fraction was studied by ECHO. Cardiac Output was calculated by the formula $HR \times SV$ (ml/min).

1.SDI/ISI = $\{CO / (HR-EF \text{ difference for } 90 \text{ ml})\} \times 1/100$ by ECHO & ECG IN NORMAL HEART

2.Sdi/isi = $\{Co / (hr-ef \text{ diff})\} / 10 - SV/HR$ by echo and ECG in angina

3.Sdi/isi in angina = $(qt + pp) / 10 - t \text{ wave amplitude in MV}$ by ecg

= $\{CO / (RR-EF \text{ DIFF}) - T \text{ wave amp} / 100$ by echo

BIGEMINI = $\{Co / (HR-EF \text{ DIFF})\} / 100 + SV/HR$

$\{(QT + PP) - PR\} / 100$

Trigemini with veb = $\{3QT + 2PP\} - PR$

Normal values = 1.1-1.3

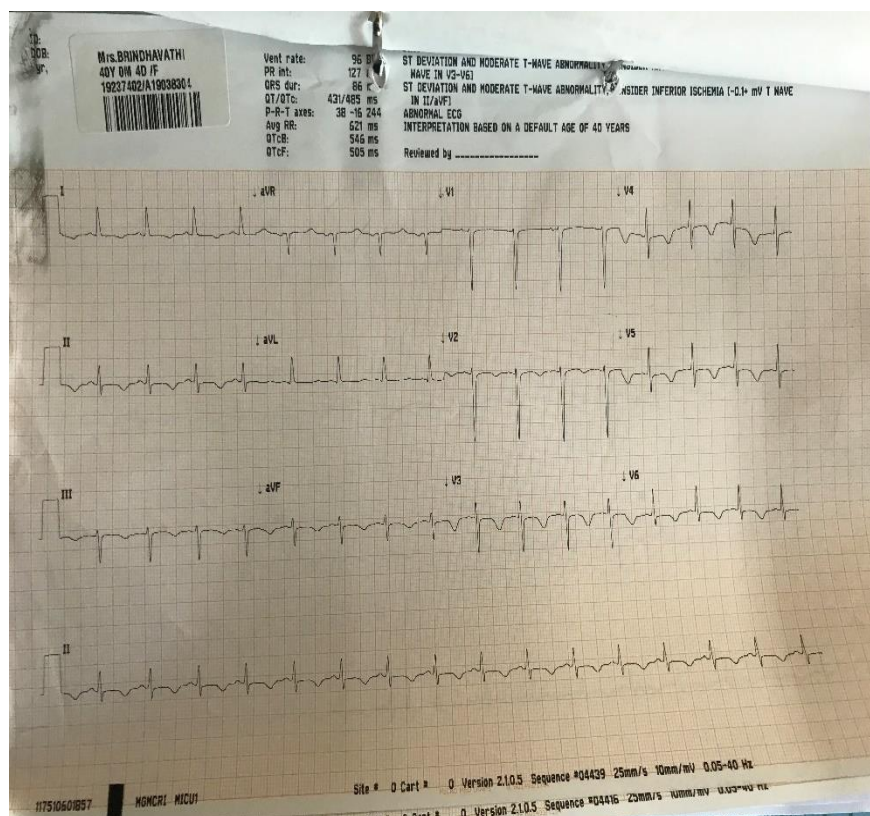
Elevated in arrhythmias = 5.4-7.3(EDV/EDD)

Values in between show ongoing pathology of the hypertrophic/dilated heart.

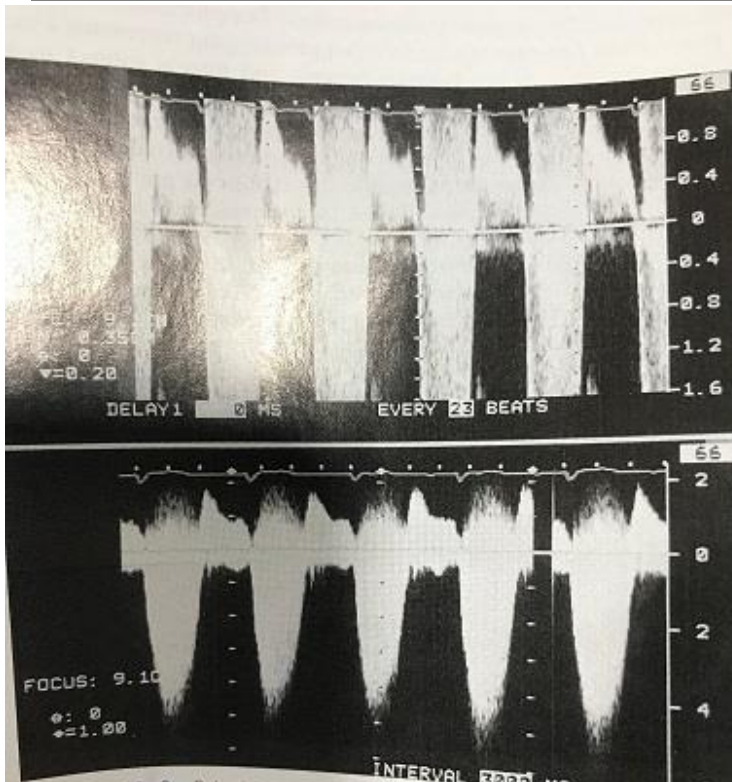
In the above excerpt, we can discuss that SDI by calculation via the abv formule show arrythmogenic potential of the diseased heart, say after MI when the value of SDI/ISI is between 5.4 and 7.3.A heart with no arrythmogenic poetential showed values below 1.3 on follow-up.

Values in between 1.3 and 6.4 show HOCM properties.

Patient with chronic unstable angina/NSTEMI profile-ECG-narrow QRS (poor CRT creiterion)



Patient with Mitral Regurgitation-3D ECHO



Patient profile	HR	SV	EF diff	QRS
Patient X	90bpm	100ml	77%	77ms
Patient Y	52bpm	20ml	18%	86ms

CONCLUSIONS

The hypothesis of echocardiogenic heart post MI or HOCM or DCM can show systolic dyssynchrony when selection of cardiac pacemaker remains a mystery. The calculation of SDI in a heart with arrhythmogenic potential using the above formulae after jotting down required ECG/ECHO parameters proves noteworthy in patients with LVMD.

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