Right Ventricular Dysfunction Predisposes to Inducible Ventricular Tachycardia at Electrophysiology Studies in Patients with Acute ST Elevation Myocardial Infarction and Reduced Left Ventricle Ejection Fraction

Running title: Thakkar et al.; RV dysfunction predisposes to inducible VT

Jay B. Thakkar, MBBS, MD, FRACP1,2; Sarah Zaman, FRACP1; Karen Byth, PhD1; Arun Narayan, RN1; Aravinda Thiagalingam, FRACP, PhD1,2; Clara Chow, FRACP, PhD1,2; Stuart P. Thomas, FRACP, PhD1,2; Gopal Sivagangabalan, FRACP, PhD1,2; David Farlow, FRACP, DDU1; Robert Barnett, MsC1; Pramesh Kovoor, MBBS, FRACP, PhD1,2

1Westmead Hospital, 2Sydney Medical School, University of Sydney, Sydney, Australia

Correspondence:
Associate Prof. Pramesh Kovoor
Department of Cardiology, Level 2
Westmead Hospital
Westmead, NSW, 2145
Australia
Tel: +61 2 9845 6030
Fax: +61 2 9845 8323
E-mail: pramesh.kovoor@sydney.edu.au

Abstract:

**Background** - Inducible Ventricular tachycardia (VT) is a strong predictor of spontaneous ventricular tachyarrhythmia following ST elevation myocardial infarction (STEMI). Reduced left ventricular ejection fraction (LVEF) predisposes patients to inducible ventricular tachycardia post STEMI. However, the role of right ventricular (RV) dysfunction in predisposing to inducible VT has not been described previously.

**Methods and Results** - Consecutive patients with STEMI treated with primary percutaneous coronary intervention (PPCI) underwent pre-discharge radionuclide gated heart pool scan (GHPS) to assess ventricular ejection fraction (EF). The study cohort included patients with reduced LVEF (LVEF ≤40%), that underwent electrophysiology study (EPS) (n=220) in an attempt to induce VT. We defined RV dysfunction as right ventricular ejection fraction (RVEF) ≤35%. The end point was sustained monomorphic VT (Cycle length CL ≥200ms). This was considered a positive study. No inducible arrhythmia, ventricular fibrillation or flutter (CL<200ms) was considered a negative study. Infarct region, infarct related artery, male gender and RVEF ≤35%, were univariable predictors of positive test. After multivariable analysis, RVEF ≤35% had the strongest association as an independent predictor of inducible VT at EPS (p<0.001, OR 5.8; 95% CI, 3.005 to 11.262).

**Conclusions** - RV dysfunction (RVEF ≤35%) predisposes to inducible VT at EPS in patients with impaired LVEF (≤ 40%) after acute STEMI treated with primary PCI.

**Key words:** right ventricle, electrophysiology, ventricular tachycardia, myocardial infarction, ejection fraction
Background

Ventricular arrhythmias remain an important cause of mortality in survivors of acute myocardial infarction (AMI). The rate of death including sudden cardiac death (SCD) remains highest in the first few weeks after STEMI. Identification of those at risk for arrhythmic death amongst survivors of AMI is a challenging problem.

After AMI, the infarct zone goes through series of stages with replacement of dead myocardium by collagenized scar tissue. Late remodelling involves myocyte hypertrophy and dense collagen scar formation. The collagen fibers disposition parallels that of infarcted myocardial fibers mirroring the anisotropic structure of myocardium. VT usually arises from these reentrant circuit that incorporates areas of adipose metaplasia and scarred myocardium with surrounding normal myocardium. While the myocardial scar composition does change with time, there is some evidence that the associated re-entrant circuit and associated substrate of VT stabilizes within the first week post-MI. The electroanatomic and electrophysiologic characteristics of inducible VT in subacute and chronic phase after AMI have been shown to be similar in an ovine model. This suggests that VT re-entrant circuits form early post infarct.

EPS can demonstrate the presence of an electrical substrate for re-entrant VT and consistently predicts arrhythmic risk in observational and randomised studies. Inducible VT at EPS in patients with left ventricular dysfunction post-MI is predictive of spontaneous ventricular arrhythmia.

Reduced left ventricular ejection fraction (LVEF) is associated with increased risk of ventricular tachyarrhythmia. Optimal use of prognostic electrophysiology testing is achieved by using an LVEF of ≤ 40% as a preselector in infarct survivors. Survivors of STEMI with RV myocardial involvement have a worse prognosis than those who do not have RV involvement.
It is unclear if this is associated with increased arrhythmic risk. The role of RV dysfunction in predisposing to inducible VT has not been described previously. The purpose of the present study was to examine if RV dysfunction early after STEMI was associated with VT inducibility in patients with at least moderate LV dysfunction at EPS.

Methods

Study Population

Consecutive patients with STEMI treated with PPCI at a single tertiary centre from 2006 to 2012 were recruited. The study was approved by the Institutional review committee and the subjects gave their written informed consent. Patients presented directly to the primary percutaneous intervention centre, Westmead Hospital or were referred by three associated district hospitals. All patients in the study had angiographically confirmed coronary artery occlusion responsible for STEMI. STEMI was defined as the presence of typical chest pain and accompanying symptoms for duration of at least 30 minutes but less than 12 hours in the presence of ST-segment elevation ≥ 1 mm in at least 2 contiguous leads. Patients who underwent PPCI for LBBB, presumed new or undetermined duration; were not included. No patient received thrombolytic therapy because of the well established PPCI service at our hospital.

Patients were excluded if they were age <18 or > 85 years, underwent pre-discharge coronary artery bypass surgery (CABG), died prior to discharge from hospital or had ventricular ejection fraction estimated by transthoracic echocardiogram (TTE) only.

Following early revascularization, patients were commenced on optimal medical therapy including Aspirin, 2nd antiplatelet agent (Clopidogrel or Prasugrel) Beta-blocker (except sotalol), Angiotentison converying enzyme inhibitor (or Angiotensin receptor blocker) and statin.
Assessment of Ventricular Function

The study protocol required assessment of ventricular EF \( \geq 48 \) hours after PPCI with planar equilibrium radionuclide ventriculography. Radionuclide angiography was performed at rest in the supine position after “in-vivo” red cell labelling. Following 0.7 mg stannous pyrophosphate IV, 4 ml of heparinised autologous blood was labelled with 99mTc pertechnetate, and injected intravenously. Dynamic first pass (40x2 sec 64x64 matrix) in the RAO projection, and 16 bin zoomed gated images in MLAO, anterior and left lateral projections were acquired. A region of interest was precisely assigned to ventricular blood pool on the equilibrium gated images. To correct for activity originating from non-cardiac background structures, background region of interest was also assigned. Global ventricular EF was computed from composite time-activity curves using a computer algorithm incorporating background correction.

Electrophysiology Study

Patients with LVEF >40% were discharged; patients with LVEF \( \leq 40\)\% underwent pre-discharge EPS to assess risk of future spontaneous ventricular tachyarrhythmia. Reduced RVEF was not considered an indication for EPS.

All subjects gave informed written consent. Intravenous sedation using midazolam and fentanyl was used. Antiarrhythmic medications were avoided for at least 1 week prior to EPS when possible. Standard beta blocker therapy (except sotalol) was not interrupted. Programmed ventricular stimulation to induce VT was performed with a quadpolar catheter deployed in the right ventricular apex. Our institutional protocol for post-infarct EPS has been described previously \(^{15,20}\). A drive train (S1S1) of 8 beats at 400 ms was followed by up to four extrastimuli (ES) delivered one at a time. Our lab has previously published data supporting the role of up to 4 ES in an attempt to induce VT \(^{20}\). Stimuli were rectangular pulses of 2 ms duration.
at twice diastolic threshold with a 3 s delay between each drive train. The initial ES was delivered at a coupling interval of 300 ms and then decreased in 10 ms steps to ventricular refractoriness. If the earliest possible ES (e.g. S1S2) failed to induce VT, that ES was delivered 10 ms outside the ventricular effective refractory period and an additional ES added (e.g. S2S3) at a coupling interval of 300 ms. The additional ES was decreased in 10 ms steps in the same manner. Additional ES were added in a similar manner (always starting with coupling interval of 300 ms) until either VT or VF was induced or refractoriness of the fourth ES was reached. There was no set lower limit for the shortest permissible ES coupling interval. The endpoint for stimulation was sustained ventricular tachyarrhythmia. The induced tachyarrhythmia was terminated after 30 s if haemodynamically tolerated, or after 10 s if haemodynamically compromised. The primary study end point was inducible monomorphic VT at EPS. A sustained monomorphic VT with a CL ≥ 200 ms was considered a positive study. No inducible arrhythmia, ventricular fibrillation / flutter (CL < 200 ms) constituted a negative study. Stimulation was repeated a second time from the same site, using the same protocol, if the initial induction was negative for VT. Isoprenaline infusion was not utilized to facilitate VT induction. Additional stimulation from the RV outflow tract was not performed. If EPS was positive, pre-discharge implantable cardioverter defibrillator (ICD) implantation was recommended. If EPS was negative, discharge without an ICD was recommended.

Statistical Methods

Continuous data are presented as either mean or median (lower-upper quartile). The categorical variables are presented as frequencies and percentage.

SPSS version 21 was used to analyze the data. Two-tailed tests with the significance level of 5% were used throughout. Chi-squared or Fisher’s exact tests as appropriate were used to test
for association between categorical variables. Mann-Whitney tests were used to compare the distribution of continuous variables between groups. One way analysis of variants was used to test for differences in RVEF by EPS results status.

For univariate analysis of variables, contingency table analysis of programmed ventricular stimulation outcomes (dichotomized into ‘inducible’ and ‘no inducible’ sustained ventricular arrhythmia) was performed for each of the variables. To determine independent predictors of programmed electrical stimulation outcome, stepwise logistic regression analysis was applied to variables. Selection of parameters into multivariate model was based on clinical judgment and univariate statistic significance.

The primary aim was to assess the association between the RVEF and inducible VT at electrophysiology study.

Results
Ventricular EF was estimated on consecutive STEMI patients (N=1733) who were recruited during April 2006 to Aug 2012. For the purpose of our study we selected those patients with EF determined on radionuclide ventriculography (N=1473) (Figure 1). Patients with LVEF > 40% (N=1126) were discharged. Patients with LVEF ≤ 40% (N=249) underwent diagnostic EPS to assess for inducible VT. Figure 1 outlines reasons for 29 patients who were excluded from the study (Figure 1). Final study cohort comprised of 220 patients. Ventricular EF was assessed at mean 6 ± 4 days after the infarct. We defined LV systolic function impairment as LVEF ≤ 40% and RV systolic function impairment as RVEF ≤ 35%. Impaired RV function in addition to impaired LV function i.e. Biventricular dysfunction (Group 1: RVEF i.e. ≤ 35% + LVEF ≤ 40%) was present in 80 (36%) patients, whereas 140 (64%) patients had preserved RV function but impaired LV systolic function (Group 2: (RVEF > 35% + LVEF ≤ 40%).
Table 1 summarizes baseline demographics and angiographic characteristics of the study cohort according to their RVEF. Mean age of study cohort was 57 ± 11 years, and most patients were men (86%). The baseline variables in the two groups were similar. However, the median LVEF in group 1 (biventricular dysfunction) was lower compared to group 2 (LV dysfunction only) (31%, range 27 -35 % versus 34%, range 29 to 37%; p=0.014).

STEMI was complicated with pulmonary edema in 43 (20%) of patients. As expected, mean LVEF was lower in patients with pulmonary edema (29± 7 % versus 32 ±6 %, p=0.018), but there was no difference in RVEF between patients with and without pulmonary edema (38±13% versus 38 ±13%, p=0.857).

Majority of patients had presented with anterior / antero-lateral infarct (84%). Pulmonary edema was observed in 37 (20%) of patients with anterior / antero-lateral infarction. RV dysfunction was observed in nearly a third of patients (n= 63, ie 34%) presenting with anterior / antero-lateral infarction. A small number of patients undergoing EPS had previous history of ischemic heart disease and percutaneous coronary intervention (PCI). However there was no statistic significant difference between the two groups (i.e. Biventricular dysfunction Vs Isolated LV dysfunction). When we analysed the indications for PCI, more number of patients in the group 2 (i.e. LVEF ≤40% but preserved RVEF > 35%) had suffered prior myocardial infarction (STEMI and NSTEMI) (38% vs 17%) with culprit vessel stent deployed in LAD (86%). However due to small numbers, a statistic analysis could not be interpreted with confidence.

Inferior infarct was more likely associated with RV dysfunction (p=0.022) compared to anterior infarct. LV dysfunction defined as LVEF ≤40% was observed in 33 (15%) patients who had inferior / infero-lateral infarct.
When RCA was the culprit vessel patients were more likely to have lower mean RVEF compared to other coronary arteries (p=0.022). This is in keeping with prior observation that inferior infarct is often result of dominant RCA occlusion which is more likely to be associated with RV dysfunction.

Baseline TIMI flow was comparable between the two groups. Procedural success with TIMI III flow was similar in both groups (group 1: 96.25% Vs group 2: 96.42%)

Results of programmed ventricular stimulation (Table 2):

Sustained VT (CL ≥ 200ms) was induced in 60 (27.3 %) patients and was considered positive EPS. Ventricular fibrillation / ventricular flutter (CL < 200 ms) was induced in 40 (18.2%) patients and was considered negative EPS. No arrhythmia was inducible in 120 (54.5%) patients and was considered negative EPS. There were no complications or deaths associated with EPS.

VT with a LBBB morphology (suggesting origin from interventricular septum or right ventricle free wall) was induced in 50% of patients with biventricular dysfunction (Group 1) and 35% of patients with isolated left ventricular dysfunction (Group 2). This difference was not statistically significant.

More extrastimuli were required for VT induction in patients with biventricular dysfunction compared to those with mainly left ventricular dysfunction (mean: 3.43 Vs 2.75, p=0.001). The ventricular arrhythmias were terminated with anti-tachycardia pacing or DC shock in all patients.

Relationship of RV dysfunction to programmed ventricular stimulation outcome:

Significantly higher rates of inducible sustained VT (positive EPS result) were observed in presence of reduced RVEF ≤ 35% (50% versus 14.3%, p <0.001).

The mean RVEF in patients with inducible VT (30.52% ±10.96%) was significantly
lower when compared to patients with no inducible arrhythmia 41.65 (±12.05%) (p <0.001). The mean RVEF in patients with inducible ventricular fibrillation or flutter (40 ± 12.27%) was similar to those with no inducible arrhythmia (41.65 ±12.05%). (See Figure 2).

RV dysfunction in anterior infarct (p<0.001), but not inferior infarct (p=0.579) was associated with VT inducibility (Table 3). However, the Test of interaction between the effects of RVEF <35% and infarct region in those with LVEF <40% was not sig (p= 0.097). The power to detect significant interaction is low here because of small individuals with inferior infarct.

Multivariate analysis of predictors for inducible sustained VT:

Stepwise logistic regression was applied to the significant univariate predictors (Table 4) of inducible VT (p≤0.1). These variables were entered as ‘regressors’ after appropriate coding. The presence or absence of inducible VT was entered as the ‘dependent’ variable. Variables assessed were Male Gender, RVEF ≤35%, LVEF value, Infarct region, Infarct artery, symptom to reperfusion time and percentage ST resolution at 90 min.

Variables found to be independent predictors for inducible VT were RVEF (p <0.001, OR 5.817, 95% CI 3.005-11.262), LVEF (p=0.044, OR 0.948, 95% CI 0.900 – 0.999) and Male gender (p=0.076, OR 2.651 95% CI 0.904- 7.772).

RVEF (Figure 3) was found to be predictive of inducible VT in patients with moderate (LVEF 26-40%) as well as severe LV systolic dysfunction (LVEF ≤25%). (p=0.039, OR 2.416, 95% CI 1.047 – 5.575).

Discussion

To our knowledge, this is the first study to systematically evaluate association between RV function and inducible VT at programmed ventricular stimulation in patients with LV systolic dysfunction following STEMI. The major and unique finding of the study was that RV
dysfunction had independent predictive value in identifying a subgroup of patients who were highly susceptible to inducible VT.

Through multivariate analysis we identified, RV dysfunction, infarct related artery (LAD) and infarct region were significantly associated with inducible VT. Previous studies have identified LAD disease and anterior segment wall motion (Table 4)\textsuperscript{21} as predictors of VT inducibility at EPS. However, our data have identified strong and highly significant association between VT inducibility and presence of RV dysfunction (p<0.001). No other study has examined this association yet. Mehta et al in a metanalysis found that RV myocardial involvement in patients with acute inferior myocardial infarction had a 2.7x increase in the risk of spontaneous sustained ventricular tachycardia and ventricular fibrillation supporting our finding that infarcted RV may be potentially arrhythmogenic. Their study has shown that adverse outcomes in patients with RV myocardial involvement are not simply due to more extensive LV dysfunction but rather due to direct RV involvement. Our findings are also consistent with MUSTT, a large randomized clinical trial in which EPS was performed in patient cohort with LVEF ≤ 40%. They found similar rates of VT inducibility in patients with LVEF < 30% and those with EF ≥ 30% but ≤ 40%\textsuperscript{22}. This suggests VT inducibility is more complex and not simply a function of degree of LV impairment. In our study we found presence of RV dysfunction was strongly predictive of inducible VT in cohort with moderate as well as more severe LV dysfunction, suggesting that RV impairment confers arrhythmogenicity at all degrees of LV impairment (figure 3).

RV function has also been shown to have independent prognostic value in patients with LV dysfunction after AMI\textsuperscript{23}. Preserved RVEF predicts exercise capacity and survival in advanced heart failure\textsuperscript{24}. While the existing literature supports that patients with RV dysfunction
have increased mortality\textsuperscript{25-27}, the mechanisms underlying this are not very clear. It is not clear if increased mortality is a result of progressive pump failure or increased incidence of ventricular arrhythmias. Our study has shown presence of underlying arrhythmogenic substrate is more common in presence of biventricular dysfunction compared to isolated LV systolic dysfunction.

In survivors of AMI, ventricular arrhythmia remains an important cause of SCD. Strategies for primary prevention of SCD in this cohort remain imperfect. The current guidelines for implantation of ICD for primary prevention of SCD after myocardial infarction are based solely on LVEF\textsuperscript{28}. According to one report nearly 37\% of patients after experiencing myocardial infarction will meet the 2006 ACC/AHA/ESC criteria for implantation of ICD for primary prevention of SCD\textsuperscript{29}. Despite guideline recommendation widespread use of AICD is not generally practised, primarily because of the cost of therapy and associated morbidity. Judicious use of ICD in these patients is justified as sensitivity and specificity of LVEF in isolation to predict risk of SCD is limited\textsuperscript{30}. While it is clear that a subgroup of STEMI patients with LV dysfunction remains at high risk of arrhythmic complications after STEMI, we are limited by not being able to accurately identify them. This necessitates further efforts to better risk stratify patients after AMI\textsuperscript{31}. We feel that adding RVEF assessment to the equation may help identify particularly vulnerable patients who may benefit from primary prevention strategies. However, more work needs to be done in this area and there is a need for longitudinal study assessing incidence of spontaneous arrhythmia in the patients with RV dysfunction in addition to LV dysfunction.

During our study, we found more extrastimuli were required to induce VT in patients with biventricular dysfunction compared to those with preserved RV and impaired LV dysfunction (3.43 vs 2.75, p=0.001). It is unclear whether the need for more extrastimuli for
induction of VT in patients with biventricular dysfunction is related to the presence of scarring adjacent to the pacing site (RV apex). Our protocol involves 4 ES with single site pacing from RV apex. We are unsure if additional pacing from the RV outflow would have induced VT with fewer ES. If programmed ventricular stimulation was limited to use of three extrastimuli, a small proportion of inducible VT could have been missed. We have shown previously that the prognostic yield of four ES is similar to that of VT induced by less than or equal to three ES in patients with post-MI LV dysfunction.20

We found an interesting propensity for LBBB morphology VT in patients with RV dysfunction in addition to left ventricular dysfunction, when compared to those with isolated LV dysfunction (50% versus 35%). This difference was not statistically significant probably because our study cohort was too small. VT of LBBB morphology usually suggests origin of VT from the interventricular septum or right ventricular free wall. Based on our observation we hypothesize that VT induced in patients with RV dysfunction from STEMI has a greater possibility of originating from the RV free wall or more likely interventricular septum, compared to patients without significant RV dysfunction. However we did not undertake detailed mapping of the VT during the EPS to confirm this hypothesis.

Another interesting clinical observation was high incidence of RV dysfunction (34%) in patients with anterior infarcts and LV dysfunction. MRI studies have found similar high incidence of RV involvement in AMI. Jensen and colleagues32 performed cardiac MRI at mean of 2.9 days after primary PCI for acute myocardial infarction. According to their MRI criteria RV involvement was present in 65% of patients with anterior myocardial infarction. TTE failed to identify RV involvement in 77% of anterior myocardial infarction, implying that RV dysfunction is perhaps more commoner in anterior infarcts than generally appreciated. It might
be under recognised since RV is a difficult chamber to image with the TTE, the most used technique for assessment of ventricular function. The mechanisms underlying RV dysfunction in association with anterior STEMI are not well understood. Left anterior descending artery provides a number of septal perforators and often wraps around the apex to supply the posterior aspect of apical septum. Hence it is not surprising that LAD occlusion results in septal necrosis and impaired septal contractility with consequential RV dysfunction. One alternative mechanism could be, depressed RV function simply due to mechanical reasons secondary to increased afterload after LV dysfunction from anterior infarction. While elevated pulmonary pressures does remain a logical explanation animal models have shown this is not essential.

**Limitations**

Data was obtained from a single-centre observational study, but was collected prospectively. We preselected subgroup of patients with LVEF ≤40% undergoing EPS and hence cannot comment on patients with RVEF ≤ 35% in presence of preserved LVEF (>40%). We have not yet analysed clinical outcomes and hence cannot determine from this data whether inducible VT in patients with RV dysfunction is related to arrhythmia risk. We used radionuclide derived ejection fraction for assessing RV function. The gold standard for measurement of RVEF is probably cardiac MRI. However this technique is limited by cost, availability and technical difficulties (claustrophobic patients, breath holding, good heart rate control and prolonged procedural times). By comparison gated blood scan is inexpensive, widely available and well tolerated by patients. While transthoracic echocardiography (TTE) is the most widely available technique, an accurate estimate of RVEF is difficult often due to poor windows and inability to define the endocardial borders accurately. The accuracy of radionuclide parameters in assessing RVEF is somewhat intermediate between MRI and TTE. Despite inherent limitations, radionuclide RVEF continues
to be a powerful parameter for cardiac survival prediction.\textsuperscript{27}

\textbf{Conclusion}

Right ventricular dysfunction predisposed patients with acute ST elevation myocardial infarction and reduced left ventricle ejection fraction to inducible VT at EPS. It is not known if this finding is a marker of sudden death in this population. Measuring RVEF in addition to LVEF may have an incremental value in identifying patients at increased risk of inducible ventricular tachycardia and guiding targeted primary prevention strategies in survivors of AMI.

\textbf{Acknowledgments:} We thank all the cardiologists at Westmead Hospital for their tremendous support for this study.

\textbf{Conflict of Interest Disclosures:} None.

\textbf{References:}


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Table 1: Baseline demographics and angiographic characteristics according to presence or absence of right ventricular dysfunction

<table>
<thead>
<tr>
<th></th>
<th>Group 1: RVEF ≤35% + LVEF ≤40% (N = 80)</th>
<th>Group 2: RVEF &gt; 35% + LVEF ≤40% (N = 140)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs) Median</td>
<td>56.5 (50-63.5)</td>
<td>56 (49-65)</td>
<td>0.588</td>
</tr>
<tr>
<td>Male</td>
<td>85 %</td>
<td>86 %</td>
<td>0.885</td>
</tr>
<tr>
<td>Hypertension</td>
<td>48%</td>
<td>54%</td>
<td>0.386</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>23%</td>
<td>24%</td>
<td>0.764</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>51.2%</td>
<td>55%</td>
<td>0.592</td>
</tr>
<tr>
<td>Family history of IHD</td>
<td>46%</td>
<td>51%</td>
<td>0.459</td>
</tr>
<tr>
<td>Current smoker</td>
<td>55 %</td>
<td>51%</td>
<td>0.610</td>
</tr>
<tr>
<td>Lung Disease</td>
<td>10%</td>
<td>12%</td>
<td>0.689</td>
</tr>
<tr>
<td>Pulmonary edema on admission</td>
<td></td>
<td></td>
<td>0.630</td>
</tr>
<tr>
<td>Previous IHD</td>
<td>28%</td>
<td>25%</td>
<td>0.620</td>
</tr>
<tr>
<td>Previous PCI</td>
<td>15%</td>
<td>14%</td>
<td>0.885</td>
</tr>
<tr>
<td>Previous CVA/TIA</td>
<td>4%</td>
<td>1%</td>
<td>0.138</td>
</tr>
<tr>
<td>LVEF% Median</td>
<td>31 (27-35)</td>
<td>34 (29-37)</td>
<td>0.014</td>
</tr>
<tr>
<td>Infarct region</td>
<td></td>
<td></td>
<td>0.050</td>
</tr>
<tr>
<td>Anterior AMI</td>
<td>78.8%</td>
<td>88.6%</td>
<td></td>
</tr>
<tr>
<td>Inferior AMI</td>
<td>21.3%</td>
<td>11.4%</td>
<td></td>
</tr>
<tr>
<td>Infarct artery</td>
<td></td>
<td></td>
<td>0.022</td>
</tr>
<tr>
<td>LAD culprit</td>
<td>73.8%</td>
<td>85.7%</td>
<td></td>
</tr>
<tr>
<td>RCA Culprit</td>
<td>17.5%</td>
<td>7.9%</td>
<td></td>
</tr>
<tr>
<td>LCX</td>
<td>3.8%</td>
<td>5.7%</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>5%</td>
<td>0.7%</td>
<td></td>
</tr>
<tr>
<td>Pre TIMI 0/1 flow</td>
<td>89%</td>
<td>84%</td>
<td>0.484</td>
</tr>
<tr>
<td>Post PCI TIMI 3 flow</td>
<td>96%</td>
<td>96%</td>
<td>0.360</td>
</tr>
</tbody>
</table>

LVEF= Left ventricular ejection fraction; RVEF=Right ventricular ejection fraction; IHD=Ischemic heart disease; PCI=Percutaneous coronary intervention; AMI=Acute myocardial infarction; LAD=Left anterior descending artery; RCA=Right coronary artery; LCX=Left circumflex artery; TIMI=Thrombolysis in acute myocardial infarction score
### Table 2: Result of the electrophysiology study according to RVEF group

<table>
<thead>
<tr>
<th></th>
<th>Group 1: RVEF ≤35% (N = 80)</th>
<th>Group 2: RVEF &gt; 35% (N = 140)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT induced</td>
<td>50%</td>
<td>14%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VT induced at first induction attempt</td>
<td>75%</td>
<td>67%</td>
<td>0.795</td>
</tr>
<tr>
<td>EPS Result (Type of arrhythmia induced)</td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VT Induced</td>
<td>50.0%</td>
<td>14.3%</td>
<td></td>
</tr>
<tr>
<td>VFib / Vflutter</td>
<td>13.8%</td>
<td>20.7%</td>
<td></td>
</tr>
<tr>
<td>No arrhythmia</td>
<td>36.2%</td>
<td>65.0%</td>
<td></td>
</tr>
<tr>
<td>VT Morphology</td>
<td></td>
<td></td>
<td>0.279</td>
</tr>
<tr>
<td>LBBB</td>
<td>18 (50%)</td>
<td>7 (35%)</td>
<td></td>
</tr>
<tr>
<td>RBBB</td>
<td>18 (50%)</td>
<td>13 (65%)</td>
<td></td>
</tr>
<tr>
<td>Mean number of Extra stimuli that induced VT</td>
<td>3.43</td>
<td>2.75</td>
<td>0.001</td>
</tr>
<tr>
<td>Median Cycle Length of the VT (msec)</td>
<td>220 (210-245)</td>
<td>215 (206-250)</td>
<td>0.554</td>
</tr>
</tbody>
</table>

VT= Ventricular tachycardia; EPS=Electrophysiology study; VFib=Ventricular fibrillation; LBBB=Left bundle branch block; RBBB=Right bundle branch block

### Table 3: Subgroup analysis for effects of infarct location and RV function on VT inducibility at EPS in patients with LVEF ≤40%.

<table>
<thead>
<tr>
<th>Infarct subgroup</th>
<th>VT positive (n)</th>
<th>Percent VT positive</th>
<th>chi-sq</th>
<th>Interaction p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVEF&lt;=35</td>
<td>32</td>
<td>50.8%</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>RVEF&gt;35</td>
<td>14</td>
<td>11.3%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVEF&lt;=35</td>
<td>8</td>
<td>47.1%</td>
<td>0.579</td>
<td></td>
</tr>
<tr>
<td>RVEF&gt;35</td>
<td>6</td>
<td>37.5%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

VT= Ventricular Tachycardia, RVEF= Right ventricle ejection fraction
**Table 4:** Predictors of inducible VT electrophysiology study in patients with LVEF ≤ 40%

<table>
<thead>
<tr>
<th></th>
<th>VT Negative</th>
<th>VT Positive</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age Yrs (Median)</td>
<td>56 (49-65)</td>
<td>57 (53-64)</td>
<td>0.374</td>
</tr>
<tr>
<td>Male</td>
<td>83 %</td>
<td>92 %</td>
<td>0.110</td>
</tr>
<tr>
<td>RVEF ≤ 35%</td>
<td>25%</td>
<td>67%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Infarct Region</td>
<td></td>
<td></td>
<td>0.034</td>
</tr>
<tr>
<td>Anterior</td>
<td>88%</td>
<td>77%</td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td>12%</td>
<td>23%</td>
<td></td>
</tr>
<tr>
<td>Infarct Related Artery</td>
<td></td>
<td></td>
<td>0.038</td>
</tr>
<tr>
<td>LAD</td>
<td>86%</td>
<td>70%</td>
<td></td>
</tr>
<tr>
<td>RCA</td>
<td>9%</td>
<td>17%</td>
<td></td>
</tr>
<tr>
<td>Symptom – Reperfusion time min (median)</td>
<td></td>
<td></td>
<td>0.174</td>
</tr>
<tr>
<td>90 Min ST resolution left %</td>
<td></td>
<td></td>
<td>0.363</td>
</tr>
<tr>
<td>&lt;= 50</td>
<td>34%</td>
<td>26%</td>
<td></td>
</tr>
<tr>
<td>51-70%</td>
<td>29%</td>
<td>26%</td>
<td></td>
</tr>
<tr>
<td>&gt;70%</td>
<td>37%</td>
<td>48%</td>
<td></td>
</tr>
</tbody>
</table>

VT=Ventricular tachycardia; RVEF=Right ventricular ejection fraction; LAD=Left anterior descending; RCA=Right coronary artery.

**Figure Legends:**

**Figure 1:** Study Cohort

**Figure 2:** Mean RVEF based on EPS result. Lower mean RVEF was noted in patients with inducible ventricular tachycardia compared to those with inducible Ventricular fibrillation / flutter or no arrhythmia
Figure 3: Reduced RVEF \( \leq 35\% \) was independent predictor of inducible VT in patients with moderate LV dysfunction (LVEF 25 to \( \leq 40\% \)) as well as severe LV dysfunction (LVEF \( \leq 25\% \)).

\( P<0.001, \text{ OR } 6.2, 95\% \text{ CI } 3.20-12.03 \)
Protocol violation due to treating clinician decision to reassess LVEF in those with borderline LVEF.
Mean RVEF
VT: 31%
VFibrillation / V flutter: 40%
No Arrhythmia: 42%
Right Ventricular Dysfunction Predisposes to Inducible Ventricular Tachycardia at Electrophysiology Studies in Patients with Acute ST Elevation Myocardial Infarction and Reduced Left Ventricle Ejection Fraction


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