Right Ventricular Dysfunction Predisposes to Inducible Ventricular Tachycardia at Electrophysiology Studies in Patients With Acute ST-Segment–Elevation Myocardial Infarction and Reduced Left Ventricular Ejection Fraction

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

Methods and Results—Consecutive patients with ST-segment–elevation myocardial infarction treated with primary percutaneous coronary intervention underwent predischarge radionuclide gated heart pool scan to assess ventricular EF. The study cohort included patients with reduced left ventricular EF (left ventricular EF ≤40%) who underwent electrophysiology study (n=220) in an attempt to induce VT. We defined RV dysfunction as RVEF ≤35%. The end point was sustained monomorphic VT (cycle length ≥200 ms). This was considered a positive study. No inducible arrhythmia, ventricular fibrillation, or flutter (cycle length <200 ms) was considered a negative study. Infarct region, infarct-related artery, male sex, 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 electrophysiology study (P<0.001; odds ratio, 5.8; 95% confidence interval, 3.005–11.262).

Conclusions—RV dysfunction (RVEF ≤35%) predisposed to inducible VT at electrophysiology study in patients with impaired left ventricular EF (≤40%) after acute ST-segment–elevation myocardial infarction treated with primary percutaneous coronary intervention. (Circ Arrhythm Electrophysiol. 2014;7:898-905.)

Key Words: electrophysiology ■ heart ventricles ■ myocardial infarction ■ stroke volume ■ tachycardia, ventricular

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 ST-segment–elevation MI (STEMI).1,2 Identification of those at risk for arrhythmic death among survivors of AMI is a challenging problem.3,4

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After AMI, the infarct zone goes through series of stages with replacement of dead myocardium by collagenized scar tissue.5 Late remodeling involves myocyte hypertrophy and dense collagen scar formation. The collagen fibers disposition parallels that of infarcted myocardial fibers mirroring the anisotropic structure of myocardium.6 Ventricular tachycardia (VT) usually arises from these re-entrant circuit that incorporates areas of adipose metaplasia7 and scarred myocardium with surrounding normal myocardium.8 Although the myocardial scar composition does change with time, there is some evidence that the associated re-entrant circuit and associated substrate of VT stabilize within the first week post-MI.9 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 postinfarct.

Electrophysiology study (EPS) can demonstrate the presence of an electric substrate for re-entrant VT and consistently predicts arrhythmic risk in observational and randomized studies.10,11 Inducible VT at EPS in patients with left ventricular (LV) dysfunction post-MI is predictive of spontaneous ventricular arrhythmia.12-15
Reduced LV 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. It is unclear whether 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 whether 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 primary percutaneous coronary intervention (PCI) at a single tertiary center 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 center, Westmead Hospital, or were referred by 3 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 ≥30 minutes but <12 hours in the presence of ST-segment elevation ≥1 mm in ≥2 contiguous leads. Patients who underwent primary PCI for left bundle branch block, presumed new vs. old MI, or developed acute heart failure during primary PCI were excluded. Patients who presented >12 hours after symptom onset were eligible if they had hemodynamic instability. Patients who presented ≤12 hours after symptom onset were eligible if they had new Q waves or elevated cardiac biomarkers. Patients were enrolled from 2006 to 2012. The study was approved by the institutional review board. Consent. Patients presented directly to the primary percutaneous intervention center, Westmead Hospital, or were referred by 3 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 ≥30 minutes but <12 hours in the presence of ST-segment elevation ≥1 mm in ≥2 contiguous leads. Patients who underwent primary PCI for left bundle branch block, presumed new vs. old MI, or developed acute heart failure during primary PCI were excluded. Patients who presented >12 hours after symptom onset were eligible if they had hemodynamic instability. Patients who presented ≤12 hours after symptom onset were eligible if they had new Q waves or elevated cardiac biomarkers. Patients were enrolled from 2006 to 2012. The study was approved by the institutional review board.

Assessment of Ventricular Function
The study protocol required assessment of ventricular EF ≥48 hours after primary PCI with planar equilibrium radionuclide ventriculography. Radionuclide angiography was performed at rest in the supine position after in vitro red cell labeling. After 0.7 mg stannous pyrophosphate IV, 4 mL of heparinized autologous blood was labeled with 99mTc pertechnetate and injected intravenously. Dynamic first pass (40×2 s; 64×64 matrix) in the right anterior oblique projection and 16-bin zoomed gated images in modified left anterior oblique, 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 noncardiac 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 ≤40% underwent predischarge 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 ≥1 week before EPS when possible. Standard β-blocker therapy (except sotalol) was not interrupted. Programmed ventricular stimulation to induce VT was performed with a quadpolar catheter deployed in the RV apex. Our institutional protocol for postinfarct EPS has been described previously. A drive train (S1S1) of 8 beats at 400 ms was followed by ≤4 extrastimuli delivered one at a time. Our laboratory has previously published data supporting the role of ≤4 extrastimuli in an attempt to induce VT. Stimuli were rectangular pulses of 2-ms duration at twice diastolic threshold with a 3-5 s delay between each drive train. The initial extrastimuli was delivered at a coupling interval of 300 ms and then decreased in 10-ms steps to ventricular refractoriness. If the earliest possible extrastimuli (eg, S1S2) failed to induce VT, that extrastimuli was delivered 10 ms outside the ventricular effective refractory period and an additional extrastimuli added (eg, S2S3) at a coupling interval of 300 ms. The additional extrastimuli was decreased in 10-ms steps in the same manner. Additional extrastimuli were added in a similar manner (always starting with coupling interval of 300 ms) until either VT or ventricular fibrillation was induced or refractoriness of the fourth extrastimuli was reached. There was no set lower limit for the shortest permissible extrastimuli coupling interval. The end point for stimulation was sustained ventricular tachyarrhythmia. The induced tachyarrhythmia was terminated after 30 s if hemodynamically tolerated, or after 10 s if hemodynamically compromised. The primary study end point was inducible monomorphic VT at EPS. A sustained monomorphic VT with a cycle length ≥200 ms was considered a positive study. No inducible arrhythmia, ventricular fibrillation/flutter (cycle length <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 used to facilitate VT induction. Additional stimulation from the RV outflow tract was not performed. If EPS was positive, predischarge 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. χ2 or Fisher 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 ANOVA 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 electric stimulation outcome, stepwise logistic regression analysis was applied to variables. Selection of parameters into multivariate model was based on clinical judgment and univariate statistical significance.

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

Results
Ventricular EF was estimated on consecutive patients with STEMI (N=1733) who were recruited during April 2006 to August 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 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, that is, biventricular dysfunction (group 1: RVEF, ie, ≤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 2 groups were similar. However, the median LVEF in group 1 (biventricular dysfunction) was lower compared with group 2 (LV dysfunction only; 31% [range, 27%–35%] versus 34% [range, 29%–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 PCI. However, there was no statistically significant difference between the 2 groups (ie, biventricular dysfunction versus isolated LV dysfunction). When we analyzed the indications for PCI, more number of patients in the group 2 (ie, LVEF ≤40% but preserved RVEF >35%) had prior MI (STEMI and non-STEMI; 38% versus 17%) with culprit vessel stent deployed in left anterior descending artery (86%). However because of small numbers, a statistical analysis could not be interpreted with confidence.

Inferior infarct was more likely associated with RV dysfunction (P=0.022) compared with anterior infarct. LV dysfunction defined as LVEF ≤40% was observed in 33 (15%) patients who had inferior/inferolateral infarct.

When right coronary artery was the culprit vessel, patients were more likely to have lower mean RVEF compared with other coronary arteries (P=0.022). This is in keeping with prior observation that inferior infarct is often result of dominant right coronary artery occlusion which is more likely to be associated with RV dysfunction.

Baseline thrombolysis in acute myocardial infarction flow was comparable between the 2 groups. Procedural success with thrombolysis in acute myocardial infarction III flow was similar in both groups (group 1: 96.25% versus group 2: 96.42%).

**Results of Programmed Ventricular Stimulation**

Sustained VT (cycle length, 200 ms) was induced in 60 (27.3%) patients and was considered positive EPS (Table 2). Ventricular fibrillation/ventricular flutter (cycle length <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 left bundle branch block morphology (suggesting origin from interventricular septum or RV free wall) was induced in 50% of patients with biventricular dysfunction (group 1) and 35% of patients with isolated LV
More extrastimuli were required for VT induction in patients with biventricular dysfunction compared with those with mainly LV dysfunction (mean: 3.43 versus 2.75; P=0.001). The ventricular arrhythmias were terminated with antitachycardia pacing or direct current 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%; P<0.001).

The mean RVEF in patients with inducible VT (30.52±10.96%) was significantly lower when compared with 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%; 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 significant (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 sex, RVEF ≤35%, LVEF value, infarct region, infarct artery, symptom to reperfusion time, and percentage ST-segment resolution at 90 minutes.

### 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 Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, 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>21%</td>
<td>19%</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>0.022</td>
</tr>
<tr>
<td>Inferior AMI</td>
<td>21.3%</td>
<td>11.4%</td>
<td>0.022</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>0.022</td>
</tr>
<tr>
<td>RCA culprit</td>
<td>17.5%</td>
<td>7.9%</td>
<td>0.022</td>
</tr>
<tr>
<td>LCX</td>
<td>3.8%</td>
<td>5.7%</td>
<td>0.022</td>
</tr>
<tr>
<td>Other</td>
<td>5%</td>
<td>0.7%</td>
<td>0.022</td>
</tr>
<tr>
<td>Pre-TIMI 0/1 flow</td>
<td>89%</td>
<td>84%</td>
<td>0.484</td>
</tr>
<tr>
<td>Post-PHI TIMI 3 flow</td>
<td>96%</td>
<td>96%</td>
<td>0.360</td>
</tr>
</tbody>
</table>

AMI indicates acute myocardial infarction; CVA, cerebrovascular accident; IHD, ischemic heart disease; LAD, left anterior descending artery; LCX, left circumflex artery; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; RCA, right coronary artery; RVEF, right ventricular ejection fraction; TIA, transient ischemic attack; and TIMI, thrombolysis in acute myocardial infarction score.

### Table 2. Result of the EPS 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 Value</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></td>
<td></td>
<td></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 extrastimuli that induced VT</td>
<td>3.43</td>
<td>2.75</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean cycle length of the VT, ms</td>
<td>220 (210–245)</td>
<td>215 (206–250)</td>
<td>0.554</td>
</tr>
</tbody>
</table>

EPS indicates electrophysiology study; LBBB, left bundle branch block; RBBB, right bundle branch block; RVEF, right ventricular ejection fraction; VFib, ventricular fibrillation; and VT, ventricular tachycardia.
Table 3. Subgroup Analysis for Effects of Infarct Location and RV Function on VT Inducibility at Electrophysiology Study in Patients With Left Ventricular EF ≤40%

<table>
<thead>
<tr>
<th>Infarct Subgroup</th>
<th>Total, N</th>
<th>VT Positive, n</th>
<th>Percent VT Positive</th>
<th>χ² P Value</th>
<th>Interaction P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVEF ≤35</td>
<td>63</td>
<td>32</td>
<td>50.8%</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>RVEF &gt;35</td>
<td>124</td>
<td>14</td>
<td>11.3%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior</td>
<td></td>
<td></td>
<td></td>
<td>0.097</td>
<td></td>
</tr>
<tr>
<td>RVEF ≤35</td>
<td>17</td>
<td>8</td>
<td>47.1%</td>
<td>0.579</td>
<td></td>
</tr>
<tr>
<td>RVEF &gt;35</td>
<td>16</td>
<td>6</td>
<td>37.5%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

RVEF indicates right ventricular ejection fraction; and VT, ventricular tachycardia.

Variables found to be independent predictors for inducible VT were RVEF (P<0.001; odds ratio, 5.817; 95% confidence interval, 3.005–11.262), LVEF (P=0.044; odds ratio, 0.948; 95% confidence interval, 0.900–0.999), and male sex (P=0.076; odds ratio, 2.651; 95% confidence interval, 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; odds ratio, 2.416; 95% confidence interval, 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 after 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 that RV dysfunction, infarct-related artery (left anterior descending artery), and infarct region were significantly associated with inducible VT. Previous studies have identified left anterior descending artery disease and anterior segment wall motion (Table 4) 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 meta-analysis found that RV myocardial involvement in patients with acute inferior MI had a 2.7 times increase in the risk of spontaneous sustained VT 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 because of more extensive LV dysfunction but rather because of direct RV involvement.

Our findings are also consistent with MUSTT (Multicentre Unsustained Tachycardia Trial), 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%. This suggests that VT inducibility is more complex and not simply a function of degree of LV impairment. In our study, we found that 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. Preserved RVEF predicts exercise capacity and survival in advanced heart failure. Although the existing literature supports that patients with RV dysfunction have increased mortality, the mechanisms underlying this are not clear. It is not clear whether increased mortality is a result of progressive pump failure or increased incidence of ventricular arrhythmias. Our study has shown that presence of underlying arrhythmogenic substrate is more common in presence of biventricular dysfunction compared with 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 MI are based solely on LVEF. According to 1 report, nearly 37% of patients after experiencing MI will meet the 2006 American College of Cardiology/American Heart Association/European Society of Cardiology criteria for implantation of ICD for primary prevention of SCD. Despite guideline recommendation, widespread use of automated implantable cardioverter-defibrillator is not generally practiced, 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. Although it is clear that a subgroup of patients with STEMI 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. 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 that more extrastimuli were required to induce VT in patients with biventricular dysfunction compared with those with preserved RV and impaired LV dysfunction (3.43 versus 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 extrastimuli with single-site pacing from RV apex. We are unsure whether additional pacing from the RV outflow would have induced VT with fewer extrastimuli. If programmed ventricular stimulation was limited to use of 3 extrastimuli, a small proportion of inducible VT could have been missed. We have shown previously that the prognostic yield of 4 extrastimuli is similar to that of VT induced by \( \leq 3 \) extrastimuli in patients with post-MI LV dysfunction.20

We found an interesting propensity for left bundle branch block morphology VT in patients with RV dysfunction in addition to LV dysfunction, when compared with those with isolated LV dysfunction (50% versus 35%). This difference was not statistically significant probably because our study cohort was too small. VT of left bundle branch block morphology usually suggests origin of VT from the interventricular septum or RV 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 with 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 et al performed cardiac MRI at mean of 2.9 days after primary PCI for AMI. According to their MRI criteria, RV involvement was present in 65% of patients with anterior MI. TTE failed to identify RV involvement in 77% of anterior MI, implying that RV dysfunction is perhaps more commoner in anterior infarcts than generally appreciated. It might be underrecognized because 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 several septal perforators and often wraps around the apex to supply the posterior aspect of apical septum. Hence, it is not surprising that left anterior descending artery occlusion results in septal necrosis and impaired septal contractility with consequential RV dysfunction.32 One alternative mechanism could be depressed RV function simply attributable to mechanical reasons secondary to increased afterload after LV dysfunction from anterior infarction.33 Although elevated pulmonary pressures do remain a logical explanation, animal models have shown that this is not essential.34

**Limitations**

Data were obtained from a single-center observational study, but was collected prospectively. We preselected subgroup of patients with LVEF \( \leq 40\% \) undergoing EPS and hence cannot comment on patients with RVEF \( \leq 35\% \) in presence of preserved LVEF (>40%). We have not yet analyzed 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 EF 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. Although transthoracic echocardiography (TTE) is the most widely available technique, an accurate estimate of RVEF is difficult often attributable 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.27

Conclusions
RV dysfunction predisposed patients with acute STEMI and reduced LVEF to inducible VT at EPS. It is not known whether 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 VT and guiding targeted primary prevention strategies in survivors of AMI.

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Disclosures
None.

References
CLINICAL PERSPECTIVE

Inducible ventricular tachycardia predicts arrhythmia risk in survivors of ST-segment–elevation myocardial infarction. Reduced left ventricular ejection fraction is a marker for increased risk of arrhythmic death, but its predictive value is limited. This study assesses the relation of right ventricular dysfunction after ST-segment–elevation myocardial infarction to ventricular tachycardia inducibility. Consecutive patients treated with primary percutaneous coronary intervention for acute ST-segment–elevation myocardial infarction who had left ventricular ejection fraction <40% underwent electrophysiology study. Right ventricular dysfunction as detected on gated heart pool scan and defined as right ventricular ejection fraction ≤35% showed a strong independent association with inducible ventricular tachycardia (P<0.001; odds ratio, 5.8; 95% confidence interval, 3.005–11.262). The nature of the pathophysiologic link and assessment of whether right ventricular dysfunction is a marker for spontaneous arrhythmias and arrhythmic death warrant further investigations.
Right Ventricular Dysfunction Predisposes to Inducible Ventricular Tachycardia at Electrophysiology Studies in Patients With Acute ST-Segment–Elevation Myocardial Infarction and Reduced Left Ventricular Ejection Fraction


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