Coronary artery reperfusion for ST elevation myocardial infarction is associated with shorter cycle length ventricular tachycardia and fewer spontaneous arrhythmias

Chrishan J. Nalliah, Sarah Zaman, Arun Narayan, Janice Sullivan, and Pramesh Kovoor*

Department of Cardiology, Westmead Hospital, Westmead, Sydney, NSW 2145, Australia

Received 24 May 2013; accepted after revision 4 September 2013; online publish-ahead-of-print 24 October 2013

Aims

Ventricular tachycardia (VT) induction at electrophysiological (EP) study early after ST elevation myocardial infarction (STEMI) has been a predictor of spontaneous ventricular arrhythmia. Reperfusion therapy for STEMI may have resulted in altered VT character. We attempted to determine differences in VT cycle length (CL) and VT recurrence rates, in patients who received early and late reperfusion treatment for STEMI.

Methods and results

Of 180 consecutive patients with left ventricular ejection fraction < 40%, 77 patients had positive EP studies. Forty-nine patients receiving early reperfusion treatment (group 1, n = 49) were compared with 28 patients who received late reperfusion (group 2; n = 28). Seventy-five patients had defibrillators implanted for primary prevention of sudden death. Patients were followed for up to 6 years to assess long-term rates of spontaneous ventricular tachyarrhythmia. Patients who received early reperfusion demonstrated shorter CL inducible VT (231 ± 43 ms vs. 252 ± 56 ms; P = 0.016). They also had fewer spontaneous arrhythmias (adjusted hazard ratio of 2.94, 95% confidence interval: 1.07–8.13; P = 0.03) with shorter CL spontaneous VT (266 ± 54 ms vs. 320 ± 80 ms; P = 0.02) at 53 ± 33 months. Ventricular tachycardia CL was the only independent predictor of spontaneous arrhythmia or sudden cardiac death (1.22, 1.07–1.47; P = 0.016).

Conclusions

Patients receiving early reperfusion for STEMI had faster inducible and spontaneous VT and fewer spontaneous recurrences. This may be due to changes in the myocardial substrate as a result of early coronary artery reperfusion.

Keywords

ST elevation myocardial infarction • Electrophysiology • Ventricular tachycardia • Acute myocardial infarction

Introduction

Sudden cardiac death (SCD) accounts for up to 50% mortality in acute myocardial infarction (AMI) survivors. Ventricular tachycardia (VT) is the commonest cause of SCD among AMI survivors, claiming the lives of approximately 30% of patients with impaired ventricular function within the first 2 years following AMI. Reperfusion with thrombolysis or primary angioplasty has significantly improved prognosis. Implantable cardiac defibrillators (ICDs) have been shown to improve mortality in patients with left ventricular ejection fraction (LVEF) < 30%.

Some studies have failed to show an overall mortality benefit of early ICD implantation, despite decreasing the risk of SCD. However, electrophysiological (EP) studies early after STEMI have been used in the pre-lytic era to detect re-entry circuits capable of maintaining VT, and in the reperfusion era to guide ICD implantation for primary prevention of SCD. In a sub-study of the Multicenter Automatic Defibrillator Implantation Trial II, patients with inducible monomorphic VT using up to three extrastimuli (ES) through the defibrillator, had a higher risk of spontaneous VT (29% vs. 19.3%). However, VT induction did not identify patients with a combined endpoint of VF and VT (29.4% vs. 25.5%). In contrast, other trials using invasive protocols with up to three ES have enjoyed greater success in stratifying patients at risk of SCD. Some studies suggest that VT induced using up to four ES confers up to a four-fold risk of spontaneous ventricular arrhythmia or SCD. Secondly,
arhythmic recurrence is highest amongst patients with inducible slow VT. However, fast VT (CL 200–230 ms) also identifies patients at significant risk of arhythmic recurrence. Protocols that exclude inducible fast monomorphic VT (CL 200–230 ms) or include inducible VF (CL < 200 ms) may have poor predictive value.

Reperfusion changes the extent, geometry, radiological, and histological appearance of infarcted scar tissue. We aimed to address differences in the characteristics of induced VT early after STEMI, with early and late reperfusion, and to correlate with spontaneous ventricular tachyarrhythmia at long-term follow up. We hypothesized that reperfusion is associated with faster inducible and spontaneous VT that are less prone to spontaneous recurrence.

Methods

Patients included in the study

One hundred and eighty consecutive patients with LVEF < 40% who underwent EP study for prognostic reasons following STEMI at Westmead Hospital over 4 years, from 2000 to 2004, were included in the study (Figure 1). The study protocol was approved by the Research and Human Ethics Review Committees at Westmead Hospital. A diagnosis of STEMI was determined by using standard clinical symptoms, electrocardiographic and cardiac enzyme criteria. Patients presented directly to the intervention-capable Westmead Hospital (tertiary centre, 957 beds) or were referred by three associated district hospitals (combined 685 beds). Primary percutaneous coronary intervention (PCI) was performed on all patients that presented to Westmead Hospital with STEMI with ongoing clinical symptoms. Patients who failed thrombolytic therapy at a regional centre (hemodynamically unstable or ongoing chest pain) were transferred to Westmead Hospital and underwent rescue PCI. Patients who did not clinically fail thrombolytic therapy, had a delayed presentation or a missed diagnosis at presentation, underwent delayed PCI, as part of the standard protocol after an ischemic cardiac event at our centre. Delayed angiography was defined as PCI >12 h after the onset of symptoms.

Early reperfusion was defined by resolution of ST segment elevation and thrombolysis in myocardial infarction (TIMI) 3 flow within 12 h of symptom onset. Patients having early reperfusion were categorized as having primary PCI or thrombolytic therapy. All other patients were considered to have late reperfusion (diagnosis was missed, the patient presented late to medical attention, a thrombolysed patient that was not successfully reperfused, primary PCI that failed to achieve TIMI 3 flow and successful rescue PCI >12 h after the onset of symptoms).

Revascularization by angioplasty or coronary artery bypass graft was performed in appropriate circumstances. Patients underwent EP study only after undergoing full revascularization and after commencement of optimal medical therapy based on established guidelines, provided there were no signs of cardiac ischemia, sustained VT or VF >48 h post AMI, cardiogenic shock or decompensated heart failure (Figure 1).

All patients underwent radionucide gated heart pool scan 3–7 days after full revascularization to determine LVEF. The value of an EP study early after AMI has been shown. Only patients with LVEF < 40% underwent EP study. The rationale for restricting EP studies to such patients has been described previously. Electrophysiological study was performed at 19 ± 24 days after AMI in patients with LVEF < 40%. Antiarrhythmic drugs were ceased 7 days prior to EP study. No patient was on Amiodorone or Sotolol at the time of the study. Beta blockers, Digoxin, and calcium channel blockers were continued.

Exclusion criteria

Patients were excluded from the study due to (1) normal coronary arteries on coronary angiogram; (2) ischemic symptoms despite early intervention; (3) clinical heart failure not controlled by standard medications; and (4) significant non-cardiac disease limiting life expectancy to less than 1 year.

Electrophysiological study

Electrophysiological study was performed under intravenous conscious sedation in the absence of anti-arrhythmic medication, except beta-blockers. Programmed ventricular stimulation was performed using a programmable cardiac stimulator (Micropace EPS 320 Cardiac Stimulator, Canterbury, Sydney, Australia) at twice diastolic threshold, applying rectangular 2 ms pulse stimuli, at the right ventricular apex, using an 8 beat drive train of 400 ms cycle length. A 3 s pause was inserted before the subsequent drive train. The initial coupling interval for ES was set at 300 ms and then decreased in 10 ms steps to ventricular refractoriness. If the earliest possible ES failed to induce VT, it was placed 10 ms outside the ventricular effective refractory period and an additional ES was added. The additional ES was used to scan diastole in the same manner. Up to
four ES were added until induction of VT, ventricular flutter or ventricular fibrillation or refractoriness of the fourth ES was reached. The value of the fourth ES in induction of VT has been demonstrated previously.\textsuperscript{17}

Ventricular tachycardia induction was attempted twice unless clinically contraindicated (more than three high energy shocks required for reversion or the patient was hemodynamically unstable after arrhythmia reversion). If VF was induced, direct current cardioversion was applied as soon as possible. When VT was induced, anti-tachycardia pacing (ATP) was attempted after 10 s. Direct current cardioversion was applied to terminate the arrhythmia if ATP failed.

**Definition of induced arrhythmias**

Monomorphic VT with CL $\leq$ 200 ms lasting at least 10 s or requiring cardioversion due to haemodynamic instability was considered a positive study. Completion of the protocol to refractoriness of the fourth ES without ventricular arrhythmia induction, ventricular flutter (CL $<$ 200 ms), or polymorphic VF were all considered a negative result.

**Implantable cardiac defibrillator implantation and programming**

Patients with inducible VT underwent ICD implantation. All devices were pre-or sub-pectoral systems with the manufacturer and type determined by the hospital device acquisition process. Whenever possible, VF and VT detection zones were set. VF detection required 18 out of 24 R–R intervals with CL $\leq$ 250 ms, and was treated with up to 6 shocks. Ventricular tachycardia required 16 consecutive beats of CL $> 250$ ms. Initially three sequences of ATP were applied. If this was unsuccessful subsequent therapies consisted of shocks.

**Follow-up**

Patients were followed up for at least 48 months through clinic visits at 1, 3, 6 months, then 6 monthly intervals thereafter, via telephone contact and through review of hospital medical records. Implantable cardiac defibrillator interrogation included analysis and recording of device detections and activations with electrogram verification of CLs of any arrhythmias, mode and success of therapy, and patient symptoms or complications. Implantable cardiac defibrillator activations were classified by two investigators while they were blinded to the group. Appropriate activation included sustained VT or VF. Inappropriate therapies were excluded.

**Study end points**

The primary end point was VT CL (induced and spontaneous), and the secondary end point was spontaneous arrhythmia recurrence. Cause of death was determined after clarification of all deaths made with the local Registry of Births, Deaths and Marriages. Cause of death was determined by two independent investigators blinded to the study group based on information collected from family members, death certificates, hospital records, and ICD interrogated data. Arrhythmic (sudden) death based on a modified Hinkle and Thayer system included witnessed instantaneous deaths, unwitnessed deaths with no clear cause identified, non-sudden deaths caused by incessant tachycardia, deaths considered to be a sequel to cardiac arrest, deaths resulting from pro-arrhythmic complications, and deaths caused by complications of ICD’s.

**Statistical analysis**

Categorical variables are presented as percentages and continuous variables are presented as mean $\pm$ standard deviation. Data that conformed to a normal distribution were evaluated using the student t-test (2 tailed). Comparison of groups with a non-parametric distribution was performed using the Wilcoxon-rank-sum test and correlation between different variables was calculated using the Spearman’s rho correlation. Cycle lengths were log transformed prior to analysis in order to stabilize the variance. To adjust for differences between the groups, a general linear model was fit to the log transform of cycle lengths. All variables $P < 0.2$ were included. Categorical variables with low patient numbers were compared using Fisher’s exact test. A Kaplan–Meier analysis with Cox regression analysis was used to determine the adjusted hazard ratios of spontaneous ventricular arrhythmia and death. For all comparisons, differences between groups were considered significant at the 5% level. Statistical analysis was performed with SPSS for Windows (version 12.0, SPSS Inc).

**Results**

A total of 180 consecutive patients (age 58 $\pm$ 11 years, male 151) underwent EP study following STEMI (late reperfusion: 67, lysis: 36, PCI: 77). Of 180 patients, 77 patients had inducible VT and were divided into group 1 ($n = 49$, early reperfusion) and group 2 ($n = 28$, late reperfusion). The two groups were similar, except for female gender, number of diabetics, and triple vessel disease (Table 1).

**Subsequent treatment**

Patients having late reperfusion had later EP study after the index AMI, due to a need for revascularization. There were no significant differences between groups in terms of the number of patients receiving antiarrhythmic therapy (AAD), beta-adrenergic blockade or

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Baseline patient characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>Group 2</td>
</tr>
<tr>
<td>Early reperfusion ($n = 49$)</td>
<td>Late reperfusion ($n = 28$)</td>
</tr>
<tr>
<td>Male gender, $n$ (%)</td>
<td>46 (94)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>55 $\pm$ 11</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>30 $\pm$ 5</td>
</tr>
<tr>
<td>Site of MI, $n$ (%)</td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>41 (84)</td>
</tr>
<tr>
<td>Inferior</td>
<td>5 (10)</td>
</tr>
<tr>
<td>Other</td>
<td>3 (6)</td>
</tr>
<tr>
<td>Coronary artery disease at angiogram, $n$ (%)</td>
<td></td>
</tr>
<tr>
<td>1 Vessel CAD</td>
<td>22 (45)</td>
</tr>
<tr>
<td>2 Vessel CAD</td>
<td>15 (31)</td>
</tr>
<tr>
<td>3 Vessel CAD</td>
<td>9 (18)</td>
</tr>
<tr>
<td>No result available</td>
<td>3 (6)</td>
</tr>
<tr>
<td>Risk factors, $n$ (%)</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>14 (28)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>9 (18)</td>
</tr>
<tr>
<td>Smoking</td>
<td>21 (42)</td>
</tr>
<tr>
<td>Family history</td>
<td>23 (46)</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>25 (50)</td>
</tr>
</tbody>
</table>

ATP, anti-tachycardia pacing; CAD, coronary artery disease; DC Shock, direct current shock; LVEF, left ventricular ejection fraction; MI, myocardial infarction.
angiotensin converting enzyme (ACE) blockade. There were also no differences in medical therapy between the groups among patients with spontaneous recurrence [AAD 1 out of 6 (group 1) vs. 6 out of 12 (group 2); \(P = 0.32\), beta-blocker 4 vs. 8; \(P = 1.0\) and ACE inhibitor 3 vs. 9; \(P = 0.34\)]. Four years following AMI, six (12%) patients from group 1 and five (18%) patients from group 2 underwent coronary artery bypass grafting. Elective angioplasty was performed in two (4%) patients from group 1 and in three (11%) from group 2 (Table 2).

**Total mortality**

Eleven patients died (one sudden death, five cardiac death, five non-cardiac) during follow-up (Table 2). There was no difference in mortality between those who received or did not receive reperfusion therapy [Adjusted Hazard Ratio (AHR) of 1.06, 95% confidence interval (CI): 0.31–3.60; \(P = 0.93\)].

**Implantable cardiac defibrillator implantation**

Forty-eight patients in group 1 (96%) and 27 in group 2 (98%) underwent ICD implantation. Following implantation, one patient suffered a surgical infection and one patient experienced bleeding from the surgical site. Both complications were effectively treated. Two patients (one in group 1 vs. one in group 2) did not have ICDs implanted based on patient and treating physician preferences.

**Cycle length and spontaneous arrhythmia**

There were no complications related to the EP study. The incidence of inducible VT at EP testing was similar in group 1 and group 2 (42% vs. 43%; \(P = 0.40\)). Patients who had reperfusion treatment had shorter CL inducible VT compared with patients who did not (231 ± 43 ms vs. 252 ± 56 ms; \(P = 0.016\)) (Figure 2A).

After follow-up of 53 ± 33 months (group 1: 52 ± 27 months vs. group 2: 53 ± 23 months; \(P = 0.91\)), ICD interrogation showed 56 appropriate activations in 18 patients (group 1: 16 activations in six patients vs. group 2: 40 activations in 12 patients). Two patients

![Figure 2](image-url)
from group 1 underwent cardiac transplantation and had the ICD removed.

Spontaneous arrhythmia was significantly faster in patients that received early reperfusion (266 ± 54 ms vs. 320 ± 80 ms; \( P = 0.02 \)) (Figure 2B). Spontaneous VT was faster than induced VT, but not significantly so (group 1 mean difference: 5.75, 95% CI 0.02) \( P = 0.90 \) and group 2 mean difference: 50, 95% CI 0.35). Similarly, time from AMI till EP study was not a univariable predictor of spontaneous arrhythmia, and did not impact (AHR 1.00, 95% CI: 0.99–1.02; \( P = 0.70 \)). Their inclusion in the multivariable model did not significantly influence the results.

Reperfusion status and induced VT CL were the only predictors of appropriate defibrillator activation and sudden death. Spontaneous arrhythmia occurred in 6 out of 49 (12%) patients who received early reperfusion vs. 12 out of 28 (43%) patients that received late reperfusion (Figure 3; Log Rank test: \( P = 0.028 \)). Patients who received late reperfusion were almost three times as likely to experience appropriate defibrillator activation or SCD compared with patients that received early reperfusion based on univariable analysis (AHR 2.94, 95% CI: 1.07–8.13; \( P = 0.03 \)).

Cycle length at EP study was also predictive of appropriate defibrillator activation during follow-up. Every 20 ms increase in induced VT CL conferred 25% increased risk in appropriate activation (AHR (for every 20 ms increase CL) 1.25, 95% CI: 1.07–1.47; \( P = 0.004 \)). Induced VT CL in patients with appropriate defibrillator activation was significantly longer than in patients without recurrence (268 ± 65 vs. 231 ± 40 ms; \( P = 0.03 \)) (Figure 4).

Univariable analysis did not demonstrate that LVEF was a predictor of VT inducibility or spontaneous arrhythmic recurrence (AHR 0.96, 95% CI: 0.89–1.04; \( P = 0.35 \)). Similarly, time from AMI till EP study was not a univariable predictor of spontaneous arrhythmia, and did not impact (AHR 1.00, 95% CI: 0.99–1.02; \( P = 0.70 \)). Their inclusion in the multivariable model did not significantly influence the results.

Multivariable analysis was performed to determine independent predictors of arrhythmia recurrence (Table 3). Ventricular tachycardia CL was the only independent predictor of spontaneous recurrence. Reperfusion group demonstrated a strong trend towards significance with a two-and-a-half-fold increase in risk of arrhythmia recurrence among patients with late vs. early reperfusion.

**Discussion**

The main findings of this study are (1) early reperfusion therapy for STEMI is associated with shorter CL inducible VT early after AMI; (2) spontaneous ventricular tachyarrhythmia in patients with early reperfusion are faster than in patients with late reperfusion; and (3) early reperfusion has a lower rate of spontaneous ventricular arrhythmia and SCD.

Past studies demonstrate that VT is maintained by areas of patchy myocardium at the scar border zone. Richards et al. performed intracardiac voltage mapping and cardiac biopsies in patients referred for VT ablation. Short CL VT correlated well with surviving muscle bundles and fragmented voltage maps at the infarct border zone.25 Piers et al. demonstrated shorter VT CL in secondary prevention patients with effective reperfusion after myocardial infarction.26 Kumar et al. showed that delayed reperfusion increased the risk of spontaneous VT up to three-fold.27

**Figure 3** Kaplan–Meier survival curve comparing freedom from appropriate ICD activation of patients with early and late reperfusion during 48 months follow up. \( P \) value is based on univariable analysis.

**Figure 4** Cycle length of induced VT for patients with and without spontaneous recurrence. Whiskers indicate the usual range of observations. Circles mark outliers.
Previous studies have shown less inducible and spontaneous VT among AMI patients receiving thrombolysis. Magnetic resonance imaging studies of infarcted hearts imply that delayed or failed reperfusion results in increased infarct size and greater degrees of microvascular occlusion that can increase VT inducibility. Presumably, early reperfusion results in small areas of surviving myocardium at the infarct border zone allowing for smaller re-entry circuits capable of sustaining shorter CL VT. Re-entry circuits that are too small may not be capable of maintaining VT, as the depolarizing wave front encounters refractory myocardium. This may provide a rationale for shorter CL VT up to 4 years following an infarct.

Our findings are consistent with past studies concerning VT character following reperfusion. However, there are little data surrounding VT CL at EP study. VT CL during spontaneous recurrences and long-term outcomes in patients with early and late reperfusion. We were able to show shorter CL VT (inducible and spontaneous) and fewer arrhythmic recurrences in primary prevention patients receiving early reperfusion for AMI. These results provide further evidence suggesting that early reperfusion in AMI changes VT character. This may reflect a change in the arrhythmogenic substrate.

Induced VT in past studies had longer CL’s compared with the present one. However, CL was only evaluated in patients referred for secondary prevention of spontaneous VT. Therefore, patients with short CL VT that could have caused earlier fatality may have been missed. Secondly, they did not compare changes of both induced with spontaneous VT CL. Our results demonstrate that reperfusion is associated with shorter inducible and spontaneous VT CL up to 4 years following an infarct.

Although not significant, spontaneous VT was slower than induced VT. This may be due to a number of reasons. Firstly, although shorter CL VT is clinically significant, it is less likely to recur spontaneously than long CL VT. Myocardial remodeling following infarction through mechanisms of ventricular dilatation, myocyte hypertrophy, and interstitial fibrosis can lead to infarct expansion that can result in longer CL VT. Pharmacotherapy may modulate these mechanisms, but was utilized equally by both groups in this study.

Our study did not demonstrate higher rates of inducible VT among patients with late reperfusion. All patients in the study had a LVEF. Hence the study population was not large enough to demonstrate relationships between reperfusion group and frequency of inducible VT. Past comparisons of STEMI patients who were treated with lysis vs. primary PCI failed to show differences in the incidence of inducible VT. Our overall VT rates (induced and spontaneous) are consistent with The Multicenter Unsustained Tachycardia Trial that consisted of a composite of early and late reperfused patients, with an activation rate of 21%. This is consistent with the spontaneous activation rate in this study of 23% (early reperfusion 12% vs. late reperfusion 43%).

There was no difference in the number of ES used to induce VT between the two groups. However, the number to ES used was inversely proportional to VT CL. Greater numbers of ES potentiate greater degrees of conduction delay and block around a short re-entry circuit, preventing the depolarizing wave front from encountering refractory tissue.

### Table 3 Univariable and multivariable predictors of arrhythmia recurrence

<table>
<thead>
<tr>
<th>Predictors of recurrence</th>
<th>n</th>
<th>Univariable analysis</th>
<th>Multivariable analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT CL (per 20 ms increase)</td>
<td>77</td>
<td>1.25 (1.07–1.47) 0.004</td>
<td>1.22 (1.04–1.44) 0.016</td>
</tr>
<tr>
<td>Reperfusion group (late vs. early)</td>
<td>77</td>
<td>2.94 (1.07–8.13) 0.03</td>
<td>2.50 (0.88–6.91) 0.07</td>
</tr>
<tr>
<td>Family history</td>
<td>77</td>
<td>0.44 (0.15–0.28) 0.13</td>
<td>0.47 (0.16–1.39) 0.17</td>
</tr>
<tr>
<td>Male</td>
<td>77</td>
<td>1.22 (0.39–4.31) 0.76</td>
<td>– –</td>
</tr>
<tr>
<td>Diabetes</td>
<td>75</td>
<td>1.22 (0.39–3.80) 0.74</td>
<td>– –</td>
</tr>
<tr>
<td>CAD (single vs. multi-vessel)</td>
<td>70</td>
<td>0.90 (0.31–2.61) 0.85</td>
<td>– –</td>
</tr>
<tr>
<td>LVEF (%) (per 1% increase)</td>
<td>77</td>
<td>0.96 (0.89–1.04) 0.35</td>
<td>– –</td>
</tr>
<tr>
<td>Days till EP study (per day)</td>
<td>77</td>
<td>1.00 (0.99–1.02) 0.70</td>
<td>– –</td>
</tr>
</tbody>
</table>

VT, ventricular tachycardia; CL, cycle length; CAD, coronary artery disease; LVEF, left ventricular ejection fraction.
Patients that received early reperfusion also required more aggressive forms of arrhythmia termination (direct current shock vs. ATP or spontaneous termination).\(^2\) Faster VT’s required more shocks because of poor efficacy of ATP and earlier hemodynamic compromise. This is consistent with previous studies (PainFREE Trial) demonstrating that fast VT are less responsive to ATP, frequently requiring shocks.\(^3\)

**Limitations**

This was not a randomized controlled study. However, all consecutive patients presenting or referred to Westmead Hospital during the relevant time period were prospectively included. Randomization was not possible because of the strong correlation of reperfusion with mortality\(^4\) and the resultant ethical difficulty in randomizing patients to a no-reperfusion arm. Furthermore, due to the nature of the study only small numbers of patients could be recruited. A long duration of follow-up was utilized to correlate early inducibility with late spontaneous occurrence. We could not establish an association between LVEF and inducible or spontaneous arrhythmia. This was also secondary to small patient numbers. Studies with larger patient numbers are required to establish reperfusion time as an independent predictor of recurrence. Secondly, characteristics between groups were different (females, diabetics, and triple vessel disease) which may have influenced the results. There is evidence that arrhythmic events and sudden death may be less common among women meeting criteria for ICD therapy.\(^5\) However, adjustment for baseline characteristics showed that the only independent predictors of spontaneous arrhythmias were VT CL and reperfusion status. Despite the late reperfusion group having more females in this study, this group still had more arrhythmic events than the group that received early reperfusion. There was also a difference in time taken from the index event till the EP study between the two groups. There are little data on the evolution of the VT substrate following infarction. However, it is unlikely that this influenced the result, as both induced and spontaneous VT CL were shorter in the early reperfusion group. Since this study, evidence for longer ICD detection intervals has emerged resulting in fewer VT treatments.\(^6\)\(^7\) Despite the use of standard ICD settings for the relevant time period, this study may over-represent the number of clinically relevant arrhythmias. Larger studies comparing early and late reperfusion are required to verify our results, but could be difficult in the current era of effective primary angioplasty.

**Conclusions**

Our results demonstrate that early coronary artery reperfusion in STEMI is associated with shorter CL VT (inducible and spontaneous) and fewer spontaneous recurrences. The risk of arrhythmic recurrence increases independently with longer CL VT. This suggests that early reperfusion therapy may change the substrate responsible for maintaining VT and that these changes can persist for years.

**Acknowledgements**

We are indebted to the contribution of all the cardiologists and technicians at Westmead Hospital.

**Conflict of interest:** none declared.

**References**


Ventricular fibrillation associated with multi-vessel coronary spasms following radiofrequency ablation of atrial fibrillation and atrial flutter

Ryudo Fujiwara*, Akihiro Yoshida, and Ken-ichi Hirata

Department of internal medicine, Division of cardiovascular medicine, Kobe University Graduate School of Medicine, Kobe 650-0017, Japan

* Corresponding author. Tel: +078 382 5111; fax: +078 382 5859, Email: snowreveries119@yahoo.co.jp

A 62-year-old man was admitted for radiofrequency (RF) ablation of symptomatic paroxysmal atrial fibrillation and typical atrial flutter refractory to medical treatment. He had a history of syncope diagnosed as vasospastic angina pectoralis. Upon admission to our hospital for RF ablation, an echocardiogram revealed a structurally normal heart. Sedation was maintained with the intravenous administration of dexmedetomidine hydrochloride during the ablation procedure. All four pulmonary veins were successfully isolated and bidirectional cavotricuspid isthmus block was confirmed. On the way to the medical ward after the procedure, he suffered a cardiopulmonary arrest. Cardiopulmonary resuscitation was started. Ventricular fibrillation was confirmed and terminated with a 300 J shock. Immediately after the ablation, the 12-lead electrocardiogram showed sinus tachycardia without any ST elevation, but 10 min later, it revealed ST elevation in Leads I, II, III, aVF, aVL, and V3–6. The intravenous administration of nitrates was started and the ST elevation improved. Urgent coronary angiography was performed and it revealed severe stenosis of the right coronary artery and left circumflex artery. An intracoronary bolus of nitroglycerin induced vasodilation of the coronary artery (Figure). He recovered without any neurological sequelae and received an implantable cardioverter-defibrillator for secondary prevention.


Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2014. For permissions please email: journals.permissions@oup.com.