Early Reperfusion Therapy Affects Inducibility, Cycle Length, and Occurrence of Ventricular Tachycardia Late After Myocardial Infarction

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Background—This study aimed to evaluate the impact of early reperfusion during acute myocardial infarction (MI) on ventricular tachycardia (VT) inducibility, inducible VT cycle length (CL), and occurrence of spontaneous VT late after MI.

Methods and Results—Five hundred six patients (440 men; age, 63±11 years) with prior MI who underwent electrophysiology study before implantation of an implantable cardioverter-defibrillator for primary or secondary prevention were assessed. Patients were classified according to the reperfusion strategy (reperfusion: thrombolysis, n=44, or percutaneous coronary intervention, n=65, versus no reperfusion, n=397) during acute MI. Monomorphic sustained VT was inducible in 351 (69%) patients. Inducibility in reperfused and nonreperfused patients was similar in primary prevention patients (56% versus 58%) but significantly higher for nonreperfused patients in secondary prevention patients (56% versus 79%, P=0.001). Induced VTCL was shorter (247±40 versus 287±63, P<0.001) and very fast VT (CL ≤250 ms) was more often induced in reperfused patients (71% versus 47%, P=0.001). In primary prevention patients, nonreperfusion was associated with a doubled risk for first spontaneous VT during follow-up.

Conclusions—There are important differences in VT inducibility, induced VTCL, and occurrence of spontaneous VT in the chronic infarct healing phase between patients with and those without successful reperfusion during acute MI. These findings suggest differences in the chronic arrhythmogenic substrate. (Circ Arrhythm Electrophysiol. 2011;4:195-201.)

Key Words: implantable cardioverter-defibrillator ■ myocardial infarction ■ ischemic heart disease ■ ventricular arrhythmia ■ reperfusion

Survivors of acute myocardial infarction (MI) may be at risk for reentrant ventricular tachycardia (VT) originating from infarct scar.1

Clinical Perspective on p 201

In the past decades, early reperfusion therapies such as thrombolysis and primary percutaneous coronary intervention (PCI) have significantly improved outcome after acute MI.2 Early reperfusion during MI results in myocardial salvage and improved ventricular function but also influences size, transmurality, and geometry of myocardial fibrosis, which may function as a substrate for ventricular arrhythmias.3–7 A fixed substrate may lead to reentrant tachycardias producible induced during electrophysiological study (EPS). In 1986, Prystowsky et al could demonstrate that in >90% of patients presenting with sustained VT late after MI, the VT could be induced during EPS.8 However, these data were derived before the widespread availability of reperfusion therapy, and the role for EPS in patients late after reperfused MI is less clear. Mapping studies demonstrated that the 3-dimensional geometry of the infarcted area may determine VT reentry circuit characteristics.9–12 In a small group of patients referred for treatment of VT late after MI, we previously demonstrated that early reperfusion affects the electroanatomic VT substrate.13 In addition, the cycle length (CL) of spontaneous and induced VTs was shorter in patients who underwent early reperfusion as compared with nonreperfused patients.

The aim of the present study was to evaluate the affect of early reperfusion during acute MI on VT inducibility, induced VTCL, and the occurrence of spontaneous VT during follow-up in a larger population of patients with prior MI in 1 coronary territory who underwent EPS before implantable cardioverter-defibrillator (ICD) treatment.

Methods

Patients Since 1996, information on all patients who received an ICD at the Leiden University Medical Center was prospectively collected in the
Myocardial Infarction

The presence of prior MI was assessed using the definition for MI, as defined by the Task Force for the Redefinition of Myocardial Infarction. Either acute MI had to be documented, or evidence of prior MI had to be present, based on any of the following criteria: (1) development of new pathological Q-waves with or without symptoms; (2) imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a nonischemic cause; or (3) pathological findings of a healed or healing MI. MI was considered to be present in >1 coronary artery region if the criteria for acute or prior MI were met for >1 of the regions provided by the 3 main coronary arteries.

The treatment of prior MI was assessed using the patients’ medical charts. Patients were categorized as reperfused when TIMI flow grade 3 was achieved within 9 hours after onset of symptoms. Reperfused patients were subdivided into patients who had undergone primary PCI (defined as angioplasty and/or stenting without prior or concomitant fibrinolytic therapy) and patients who had received thrombolytic therapy. All other patients were categorized as nonreperfused.

Electrophysiological Study

EPS to test the inducibility of VT were performed according to the current international guidelines. Patients were studied in the postabsorptive, nonsedated state. Antiarrhythmic drugs were discontinued for 5 half-lives, with the exception of amiodarone. EPS consisted of up to 3 basic drive cycle lengths (600, 500, and 400 ms) with up to 3 ventricular extrastimuli and burst pacing from the right ventricular apex and right ventricular outflow tract. The positive end point of EPS was reproducible induction of a sustained monomorphic VT (SMVT). Arrhythmias induced by antichardycardia pacing (ATP) to terminate the induced VT were not included in the analysis.

Ventricular Arrhythmia During EPS

Ventricular arrhythmias were categorized according to the EHRA/HRS consensus document. SMVT was defined as continuous VT with a similar QRS configuration from beat to beat lasting for >30 seconds or requiring an intervention for termination. Separately scored arrhythmia not considered diagnostic for the presence of fixed reentry circuits and therefore aspecific were polymorphic VT, defined as VT with continuously changing QRS configuration from beat to beat and VF.

For each patient, the mean CL of all induced SMVTs was calculated. Patients with missing data on 1 or more CLs were excluded from all analyses involving VTCL. Monomorphic VTs were categorized into the following 4 predefined subgroups according to CL: ≥250, 251 to 286, 287 to 320, and ≥320 ms (corresponding to a rate of ≥240, 240 to 210, 210 to 188, and <188 bpm, respectively). A very fast VT was defined as a VT with CL ≥250 ms.

ICD Settings and Follow-Up

Defibrillators were programmed as follows: A ventricular arrhythmia monitor zone was programmed in all patients (150 to 188 bpm). Ventricular arrhythmias faster than 188 bpm were initially attempted to be terminated with 2 bursts of ATP followed by a defibrillator shock, if appropriate. In the case of a ventricular arrhythmia faster than 210 bpm, device shocks were the initial therapy. Atrial arrhythmia detection was set to >170 bpm with supraventricular tachycardia discriminators enabled. Settings were adapted, only when clinically indicated (ie, hemodynamically well-tolerated VT at a high rate; VT in the monitored zone).

Patient follow-up was scheduled every 3 to 6 months. Device interrogation printouts were checked for appropriate and inappropriate ICD therapy (ATP or shocks). Therapies were classified as appropriate when they occurred in response to VT or VF and as inappropriate when triggered by sinus or supraventricular tachycardia, T-wave oversensing, or electrode dysfunction.

In both primary and secondary prevention ICD recipients, the correlation between treatment of acute MI and the occurrence of first spontaneous monomorphic VT during follow-up was assessed.

Statistical Analysis

Dichotomous and categorical data are displayed as numbers and percentages. Continuous data are expressed as mean±SD or median and interquartile ranges where appropriate. Patients were grouped according to acute MI treatment. Different groups were compared for (1) inducibility of SMVT, (2) mean CL of induced SMVTs, (3) inducibility of very fast VT (CL ≤250 ms), and (4) the occurrence of first spontaneous VT during follow-up in primary and secondary prevention ICD patients. Data were analyzed by means of the χ² test, Student t test, or the Mann-Whitney U test, as appropriate. For the Student t test, equal variances were assumed if the Levene test for equality of variances showed a probability value >0.05. Univariate relationships between baseline parameters and the mean CL of induced VTs were analyzed with linear regression analysis. For each variable, the effect on the mean CL with a 95% confidence interval (95% CI) was calculated. Variables with a probability value <0.10 were further evaluated in a multivariate model, using backward stepwise selection. At each step, the least significant variable was discarded from the model until all variables in the model reached a probability value <0.25.

The cumulative incidence of first spontaneous VT during follow-up in primary and secondary prevention ICD patients was analyzed by method of Kaplan-Meier. The effect of acute MI treatment on the risk of spontaneous VT was assessed in a Cox regression model. As with linear regression analysis, first, univariate analyses were performed and variables with a probability value <0.10 were further evaluated in a multivariate model, using backward stepwise selection. At each step, the least significant variable was discarded from the model until all variables in the model reached a probability value <0.25.

All analyses were performed with SPSS for Windows, version 17.0 (SPSS, Chicago, IL). Probability values are all 2-sided, and for all tests, a probability value <0.05 was considered statistically significant.

Results

Patients

Since 1996, 996 patients with prior MI in 1 coronary artery region received an ICD at the Leiden University Medical Center. Of these patients, 511 had a documented reperfusion strategy and underwent EPS before ICD implantation and after exclusion of reversible ischemia. Five patients (1%) were excluded because of the lack of late patency of the infarct-related artery. The remaining 506 patients (87% men; age, 63±11 years) comprised the studied sample. The presenting and documented arrhythmia was SMVT in 221 patients (44%) and VF in 113 (22%); 172 patients (34%) had no prior sustained arrhythmia. The majority of patients had a depressed LVEF (36±13%). Mean QRS duration was
115±29 ms and mean creatinine clearance was 79±39 mL/min. Medication during EPS included amiodarone in 22% and angiotensin-converting enzyme or angiotensin II antagonists in 75% of patients. Baseline characteristics are summarized in Table 1.

### MI Treatment

One hundred nine (22%) of 506 patients were treated with reperfusion therapy during the acute MI and 397 (78%) were not reperfused. Of the 109 reperfused patients, 65 (13%) underwent primary PCI and 44 (9%) received thrombolytic therapy. Before electrophysiological evaluation, the infarct-related artery was patent in 97 (89%), collaterally perfused in 11 (10%), and bypassed in 1 (1%) of the initially reperfused patients. In nonreperfused patients, the infarct-related artery was patent in 140 (35%), occluded in 116 (29%), collaterally perfused in 88 (22%), and bypassed in 53 (13%). Patients who were reperfused were younger than nonreperfused patients (59±11 versus 65±10, P<0.001), presented less often with SMVT (22% versus 49%, P<0.001), and had a shorter interval between MI and electrophysiological evaluation (median, 1.6; interquartile range [IQR], 0.2 to 5.9 versus median, 10.5; IQR 2.1 to 17.5, P<0.001). In addition, reperfused patients had an anterior MI more frequently (72% versus 52%, P<0.001), a shorter QRS duration (median, 100; IQR, 90 to 120 versus median, 110; IQR, 100 to 138; P=0.001), were less often treated with amiodarone (12% versus 25%, P=0.001) but were more likely to receive β-blockers (53% versus 40%, P=0.014).

### Inducibility of Ventricular Arrhythmia

In 422 patients (83%), at least 1 ventricular arrhythmia was inducible: SMVT was induced in 351 (69%) patients, 45 of them were also inducible for polymorphic VT or VF. In 71 (14%) patients, only polymorphic VT or VF was induced. Inducibility of SMVT consisted of only one VT morphology in 222 (63%) and 129 (37%) patients. Inducibility for SMVT was similar in reperfused and nonreperfused patients without prior documentation of sustained VT or VF (56% versus 58%, P=NS). However, in the secondary prevention group, inducibility for SMVT was significantly lower in reperfused patients as compared with nonreperfused patients (56% versus 79%, P=0.001).

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**Table 1. Baseline Characteristics**

<table>
<thead>
<tr>
<th></th>
<th>All Patients (n=506)</th>
<th>Nonreperfused (n=397)</th>
<th>Reperfused (n=109)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>440 (87%)</td>
<td>341 (86%)</td>
<td>99 (91%)</td>
<td>0.176</td>
</tr>
<tr>
<td>Age, y</td>
<td>65 (57–71)</td>
<td>66 (58–73)</td>
<td>60 (51–68)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Presenting arrhythmia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sustained VT</td>
<td>221 (44%)</td>
<td>197 (50%)</td>
<td>24 (22%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
<td>113 (22%)</td>
<td>94 (24%)</td>
<td>19 (17%)</td>
<td></td>
</tr>
<tr>
<td>No sustained arrhythmia</td>
<td>172 (34%)</td>
<td>106 (27%)</td>
<td>66 (61%)</td>
<td></td>
</tr>
<tr>
<td>Location of MI</td>
<td></td>
<td></td>
<td></td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Anterior</td>
<td>285 (56%)</td>
<td>206 (52%)</td>
<td>79 (72%)</td>
<td></td>
</tr>
<tr>
<td>Nonanterior</td>
<td>221 (44%)</td>
<td>191 (48%)</td>
<td>30 (28%)</td>
<td></td>
</tr>
<tr>
<td>Years from MI to EPS</td>
<td>7.5 (0.9–15.0)</td>
<td>10.5 (2.1–17.5)</td>
<td>1.6 (0.2–5.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>36±13</td>
<td>35±13</td>
<td>37±13</td>
<td>0.180</td>
</tr>
<tr>
<td>NYHA classification</td>
<td></td>
<td></td>
<td></td>
<td>0.555</td>
</tr>
<tr>
<td>I</td>
<td>227 (45%)</td>
<td>172 (43%)</td>
<td>55 (50%)</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>201 (40%)</td>
<td>164 (41%)</td>
<td>37 (34%)</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>72 (14%)</td>
<td>56 (14%)</td>
<td>16 (15%)</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>6 (1%)</td>
<td>5 (1%)</td>
<td>1 (1%)</td>
<td></td>
</tr>
<tr>
<td>QRS duration, ms</td>
<td>110 (94–130)</td>
<td>110 (100–138)</td>
<td>100 (90–120)</td>
<td>0.001</td>
</tr>
<tr>
<td>Renal clearance, mL/min*</td>
<td>79±39</td>
<td>77±41</td>
<td>85±27</td>
<td>0.066</td>
</tr>
<tr>
<td>Documented AF or atrial flutter</td>
<td>89 (18%)</td>
<td>69 (17%)</td>
<td>20 (18%)</td>
<td>0.823</td>
</tr>
<tr>
<td>Current smoking</td>
<td>126 (25%)</td>
<td>94 (24%)</td>
<td>32 (29%)</td>
<td>0.253</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>111 (22%)</td>
<td>98 (25%)</td>
<td>13 (12%)</td>
<td>0.004</td>
</tr>
<tr>
<td>β-blocker</td>
<td>217 (43%)</td>
<td>159 (40%)</td>
<td>58 (53%)</td>
<td>0.014</td>
</tr>
<tr>
<td>ACE inhibitor or ARB</td>
<td>380 (75%)</td>
<td>291 (73%)</td>
<td>89 (82%)</td>
<td>0.070</td>
</tr>
</tbody>
</table>

ACE indicates angiotensin-converting enzyme; AF, atrial fibrillation; ARB, angiotensin receptor blocker; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NYHA, New York Heart Association; EPS, electrophysiological study; and VT ventricular tachycardia.

Data are expressed as No. (percentages), mean±SD, or median (interquartile range). 

*Renal clearance was determined with the formula of Cockroft-Gault.
VTs: no reperfusion, QRS

Table 2. Difference of Mean Cycle Length of Induced Monomorphic Ventricular Tachycardias by t Tests

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Mean CL</th>
<th>Difference* (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>314</td>
<td>280±61</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Nonreperfused</td>
<td>258</td>
<td>287±63</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Reperfused</td>
<td>56</td>
<td>247±40</td>
<td>40 (27–53)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thrombolysis</td>
<td>23</td>
<td>260±37</td>
<td>27 (2–53)</td>
<td>0.038</td>
</tr>
<tr>
<td>Primary PCI</td>
<td>33</td>
<td>238±40</td>
<td>49 (27–70)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

CI indicates confidence interval; CL, cycle length; NA, not applicable; and PCI, percutaneous coronary intervention.

*When compared with nonreperfused patients.

Ventricular Tachycardia Cycle Length

In 37 (11%) of 351 patients with inducible SMVT, 1 or more induced VTCL was unknown; these patients were excluded from VTCL analyses. In the remaining 314 patients, the mean CL was 280±61 ms (Table 2). In 161 (51%) patients, at least 1 monomorphic fast Vt (CL ≤250) was induced.

Reperfused patients had a shorter mean CL of the induced Vt than nonreperfused patients (247±40 ms versus 287±63 ms, P<0.001, Table 2). This difference was even more pronounced when patients who underwent primary PCI were compared with nonreperfused patients (238±40 ms versus 287±63 ms, P<0.001). Patients treated with thrombolysis also had a significantly shorter mean CL than nonreperfused patients, but the difference was smaller (260±37 ms versus 287±63 ms, P=0.038). After exclusion of patients on amiodarone during EPS, the induced VTCL remained significantly shorter in reperfused patients (244±40 ms versus 274±58 ms, P<0.001). Of importance, reperfused patients were more often inducible for very fast Vt (CL ≤250 ms) than nonreperfused patients (71% versus 47%, P=0.001, Figure 1). The difference was mainly attributable to patients treated with primary PCI, whereas patients treated with thrombolysis had a similar percentage of very fast Vt, as compared with nonreperfused patients. Univariate and subsequent multivariate analysis identified the following 5 variables as independently affecting the mean CL of induced VTs: no reperfusion, QRS >120 ms, presenting arrhythmia, use of amiodarone, and age. After adjustment, no reperfusion at MI was correlated with 17 ms (95% CI, 0 to 34 ms; P=0.048) increase in Vt CL at EPS (Figure 2).

Spontaneous VT

The 172 primary prevention ICD recipients were followed for a median of 28 months (IQR, 11 to 45). During follow-up, spontaneous Vt triggering appropriate ICD therapy was observed in 51 (30%) patients. The cumulative incidence of first appropriate therapy was 13% (95% CI, 8% to 18%) at 1 year, 22% (95% CI, 15% to 28%) at 2 years, and 30% (95% CI, 22% to 39%) at 3 years of follow-up.

The 3-year cumulative incidence of first appropriate device therapy was 36% (95% CI, 26% to 47%) in nonreperfused patients and 18% (95% CI, 7% to 29%) in reperfused patients (Figure 3). Cox regression modeling demonstrated that nonreperfused patients exhibited a more than doubled risk for spontaneous Vt (hazard ratio, 2.2; 95% CI, 1.2 to 4.3; P=0.010).

The 334 secondary prevention ICD recipients were followed for a median of 37 months (IQR, 9 to 68). Spontaneous Vt occurred in 170 (51%) patients. The 3-year cumulative incidence of first appropriate device therapy was 38% (95% CI, 32% to 44%) in nonreperfused patients and 26% (95% CI, 12% to 39%) in reperfused patients (Figure 4). In this population with prior sustained ventricular arrhythmia, MI treatment did not significantly correlate with the occurrence of spontaneous Vt during follow-up (hazard ratio, 1.4; 95% CI, 0.9 to 2.3).

Discussion

Early reperfusion therapy for acute MI has dramatically increased over the last decenniums and is likely to influence the chronic arrhythmogenic substrate for reentrant tachycardias after infarct healing.

The current study evaluates the effects of early reperfusion during acute MI on Vt inducibility, induced VtCL, and occurrence of spontaneous Vt late after the index MI. Reperfused patients who presented with sustained Vt or VF were less likely inducible for monomorphic Vt as compared with nonreperfused patients. Reperfused and nonreperfused patients without prior sustained arrhythmia had comparable Vt induction rates; however, reperfused patients were less likely to have spontaneous Vt...
during follow-up. Among inducible patients, VTCL of induced VTs was significantly shorter and inducible VT was more often very fast VT (CL $< 250$ ms) in reperfused patients. These differences were even more pronounced in patients treated with primary PCI as compared with thrombolytic therapy. After adjustment for potential confounders, treatment of the index MI appeared to have an independently significant effect on the mean CL of induced VTs.

**Early Reperfusion During MI and Inducibility of VT**

Former studies performed before the widespread availability of reperfusion therapy have demonstrated that in patients with coronary artery disease VT was inducible in 92% of patients who presented with sustained VT and in 83% of patients who presented with cardiac arrest due to VF. Accordingly, 79% of the nonreperfused patients in our study who presented with sustained VT or VF were inducible for monomorphic VT. In contrast, in only 56% of reperfused patients with documented VT or VF, monomorphic VT was inducible. Although all patients had a chronic substrate as spontaneous arrhythmias occurred in the absence of a reversible cause, the sensitivity of an EPS for substrate assessment after reperfusion therapy seems to be low. The fact that in reperfused patients who present with monomorphic VT and VF the arrhythmia is less likely inducible during EPS as compared with nonreperfused patients has clinical implications, in particular if EPS is performed to evaluate a potential arrhythmogenic cause of syncope; or if noninducibility of monomorphic VT during radiofrequency catheter ablation is regarded as a procedural endpoint.

**Figure 2.** Forest plot of the effect of baseline parameters on the mean cycle length of induced monomorphic ventricular tachycardias (VT). For example, in the multivariate analysis, patients who did not receive reperfusion therapy at the index myocardial infarction on average had a 17-ms (95% confidence interval [CI], 0 to 34 ms) longer VT cycle length (ie, a slower VT), as compared with patients who did receive reperfusion therapy. AF indicates atrial fibrillation; LVEF, left ventricular ejection fraction; EPS, electrophysiology study; VA, ventricular arrhythmia; and VF, ventricular arrhythmia.

**Figure 3.** Kaplan-Meier curve for the occurrence of first appropriate implantable cardioverter-defibrillator therapy in patients with primary-prevention implantable cardioverter-defibrillator with reperfusion versus no reperfusion at prior acute myocardial infarction.

**Figure 4.** Kaplan-Meier curve for the occurrence of first appropriate implantable cardioverter-defibrillator therapy in patients with secondary-prevention implantable cardioverter-defibrillator with reperfusion versus no reperfusion at prior acute myocardial infarction.
Acute reperfusion has resulted in a lower prevalence of spontaneous and induced ventricular arrhythmias in the acute phase of MI. In small series, 9% to 48% of patients receiving thrombolysis were inducible for ventricular arrhythmias as compared with 88% to 100% of patients without reperfusion therapy. However, the arrhythmogenic substrate causing late ventricular arrhythmias may develop over time. In our series, EPS was performed in the chronic healing phase of MI. Of interest, nonreperfused and reperfused patients without prior spontaneous arrhythmia showed high inducibility rates of 58% and 56%, respectively. However, during follow-up, the 3-year cumulative incidence of appropriate device therapy was 36% in nonreperfused patients and only 17% in reperfused patients. This finding suggests that despite the presence of an arrhythmogenic substrate, the value of EPS to predict VT occurrence is lower in reperfused patients as compared with nonreperfused patients.

**Effect of Early Reperfusion and Inducible VTCL**

Early reperfusion during MI results in myocardial salvage and reduced mortality during follow-up. Histological studies in patients and animal models of acute MI have shown that the duration of coronary artery occlusion is proportionally correlated to the size and transmural extent of myocardial scar. Scar size and geometry are important determinants for the reentrant circuit geometry and may contribute to occurrence and CL of VT.

We recently demonstrated that reperfused patients referred for ablation of recurrent VT late after MI appeared to have smaller and less confluent electroanatomic scars with thick layers of surviving myocardium found at histology. Interestingly, the CL of spontaneous and induced VTs was shorter in reperfused than in nonreperfused patients, probably because of the observed differences in scar geometry after reperfusion therapy. The association between acute reperfusion therapy and shorter inducible VTCL was confirmed and extended, as the present study was conducted in a large population of post-MI patients, also including patients without previously documented ventricular arrhythmia. In addition, we adjusted for all baseline characteristics that affect induced VTCL.

**Limitations**

Our study was performed in a selected population of ICD recipients with prior MI and low LVEF, who are not representative for the general population with prior MI. In addition, our cutoff of 9 hours for early reperfusion is arbitrarily chosen. Furthermore, we studied induced VTs, and our findings do not necessarily apply to spontaneous VTs. However, in the previous study by Wijnmaalen et al., spontaneous VTs were taken into account and similar differences were found, as compared with induced VTs. Finally, therapy for acute MI was not randomly assigned and time from MI to EPS differs between groups, which could have resulted in selection bias. However, because primary PCI has been shown to be the preferred treatment for acute MI, a study in which patients would be randomly assigned to primary PCI, thrombolysis, or conservative treatment is ethically unacceptable, and therefore selection bias is now inevitable in studying the effects of reperfusion. To correct for this, we adjusted for baseline characteristics using linear regression and could demonstrate that reperfusion independently affects the CL of induced VTs.

**Conclusions**

There are important differences in VT inducibility, induced VTCL, and occurrence of spontaneous VT in the chronic phase of MI between patients with and without successful reperfusion during acute MI. Reperfused patients who present with sustained VT/VF are less likely to be inducible for monomorphic VT. Despite similar VT induction rates in patients without prior documented sustained arrhythmias, reperfused patients are at lower risk for spontaneous VT. However, early reperfusion is associated with faster induced VT. These findings suggest important differences in the chronic arrhythmogenic substrate after reperfusion, which gives rise to faster VT and might be less reliably assessable by EPS.

**Disclosures**

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**References**

Early reperfusion during acute myocardial infarction (MI) results in myocardial salvage and improved ventricular function, but it also influences the specific dimensions and geometry of myocardial fibrosis and thus the propensity for ventricular arrhythmia occurring late after MI. Recently, it has been demonstrated that reperfusion is associated with smaller, less confluent myocardial scars that appear to give rise to faster spontaneous and induced ventricular tachycardias (VTs) in patients referred for VT ablation late after MI. The present study comprised a large population of post-MI patients referred for electrophysiological evaluation and implantation of defibrillators, without previously documented ventricular arrhythmia. In this population, reperfusion during MI affects inducibility, spontaneous occurrence, and cycle length of VTs. Monomorphic VT was inducible in only 56% of reperfused patients as compared with 79% of nonreperfused patients with documented VT or ventricular fibrillation. Repерfused and nonreperfused patients without prior sustained arrhythmia had comparable VT induction rates, but reperfused patients were less likely to have spontaneous VT during follow-up, suggesting that the value of electrophysiological study to predict VT occurrence is lower in reperfused patients as compared with nonreperfused patients. Finally, induced VTs were faster in reperfused patients. These findings probably reflect a different chronic arrhythmogenic substrate in reperfused patients, manifest by faster VTs and less reliably assessed by electrophysiological study.
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