Significance of Inducible Very Fast Ventricular Tachycardia (Cycle Length 200–230 ms) After Early Reperfusion for ST-Segment–Elevation Myocardial Infarction

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Background—Electrophysiological study (EPS) after myocardial infarction may have a role in identifying patients at risk of sudden cardiac death. It has been shown previously that inducible very fast ventricular tachycardia (VT; cycle length [CL], 200–230 ms) is predictive of arrhythmia recurrence; however, its significance early after reperfusion in ST-segment–elevation myocardial infarction is unknown.

Methods and Results—Consecutive patients with ST-segment–elevation myocardial infarction treated with primary percutaneous coronary intervention with a left ventricular ejection fraction ≤40% underwent early EPS with an implantable-cardioverter defibrillator implanted for inducible VT, but not for a negative EPS. The end point was the cumulative incidence of death or first arrhythmic event (defined as resuscitated cardiac arrest or spontaneous ventricular tachyarrhythmia). A total of 1721 patients with ST-segment–elevation myocardial infarction underwent early left ventricular ejection fraction assessment (median, 4 days after myocardial infarction) with a left ventricular ejection fraction ≤40% in 24%. EPS was performed in 290 eligible patients with no arrhythmia or ventricular fibrillation/flutter (CL<200 ms) induced in 203 patients (EPS negative, group 1), monomorphic VT induced in 87 patients, consisting of very fast VT in 67% (group 2; n=58), and standard VT (CL>230 ms) in 33% (group 3; n=29). Kaplan–Meier 4-year cumulative incidence of death or arrhythmia was 8.2±2.3%, 33.1±7.1%, and 37.0±10.2% in groups 1, 2, and 3, respectively (P<0.001).

Conclusions—The majority of inducible VT in patients who have been reperfused early after ST-segment–elevation myocardial infarction is very fast VT (CL, 200–230 ms). This very fast VT incurs at least a similar risk of arrhythmia or death as inducible standard VT (CL>230 ms) and a significantly higher risk than patients with a negative EPS. **(*Circ Arrhythm Electrophysiol*. 2013;6:884-890.)**

Key Words: arrhythmias, cardiac ■ cardioverter-defibrillators, implantable ■ death, sudden, cardiac ■ electrophysiology ■ myocardial infarction ■ tachycardia, ventricular

Inducible ventricular tachycardia (VT) in patients with coronary artery disease and left ventricular (LV) dysfunction has been found to be predictive of spontaneous ventricular arrhythmia.[1–4] Inducible VT (cycle length [CL], 200–230 ms) has been historically classified as ventricular flutter or a non-predictive electrophysiological study (EPS) result. In the landmark MUSTT (Multicenter Unsustained Tachycardia Trial)[2] trial, patients with this arrhythmia induced by ≥2 extrastimuli were excluded from receiving a primary prevention implantable-cardioverter defibrillator (ICD). However, more recently, this inducible very fast VT has been shown to be of prognostic significance in patients with myocardial infarction (MI) and reduced LV function.[5] The predictive value of inducible very fast VT in patients who have been reperfused early with primary percutaneous coronary intervention (PPCI) is unknown. We aimed to assess the incidence and predictive value of inducible very fast VT (CL, 200–230 ms) at EPS performed in patients who have been revascularized for ST-segment–elevation myocardial infarction (STEMI).

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Methods

Consecutive patients with STEMI referred for PPCI from 2004 to 2011 were recruited. The study was approved by the Hospital Ethics Committee, and all patients gave their informed consent. Patients presented directly to the intervention-capable Westmead Hospital or were referred by 3 associated district hospitals. Patients with STEMI underwent inpatient assessment of LV function beyond day 3 post-MI with gated heart pool scan or transthoracic echocardiogram. Those with LV ejection fraction (LVEF) ≤40% underwent inpatient EPS. Previous studies published from this institution assessing the predictive value of EPS have prospectively recruited patients post-MI from 1999 to 2007[4] and to 2008.[6] This analysis looking specifically at the

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predictive value of inducible VT (CL, 200–230 ms) recruited only patients with STEMI referred for PPCI.

Inpatient Management

All patients in the study were taken to the cardiac catheterization laboratory with angiographically confirmed STEMI and intention for PPCI. Patients with LV dysfunction post-MI were commenced on optimal medical therapy, including β-blockers and angiotensin-converting enzyme inhibitor. Exclusion criteria for EPS included ongoing myocardial ischemia, decompensated heart failure or cardiogenic shock, and patient refusal and life expectancy limited to <1 year (advanced age or comorbidities).

Electrophysiological Study

Programmed stimulation was performed at twice diastolic threshold at the right ventricular apex (single site) using a programmable stimulator. A drive train (S1S1) of 8 beats at 400 ms was followed by ≤4 extrastimuli (ES). The initial ES was delivered at a coupling interval of 300 ms and then decreased in 10 ms steps to ventricular refractoriness. A second induction was performed if the initial induction did not induce VT. Patients with sustained monomorphic VT (CL≥200 ms) with ≤4 extrastimuli were considered inducible (positive EPS). Inducible VT had to be ≥30 seconds in duration if hemodynamically tolerated or ≥10 seconds in duration if hemodynamically unstable. Patients with no arrhythmia induced or inducible ventricular fibrillation (VF) or ventricular flutter (CL<200 ms) were considered to have a negative EPS. The predictive value of this approach has been discussed in detail previously.[4,6–10] Predischarge ICD implantation was recommended for EPS-positive patients. EPS-negative patients were discharged without an ICD and according to study protocol did not undergo ICD implantation >40 days post-MI irrespective of persistent LV dysfunction.

ICD Implantation and Programming

All devices were pre- or subpectoral systems with the manufacturer and type determined by the hospital device acquisition process. Device detection and therapy were programmed as follows: VF required 18 of 24 R-R intervals with CL <250 ms. Therapy was ATP during charging and shock. VT required 16 consecutive beats with CL 251 to 360 ms and 12 consecutive beats with CL 251 to 360 ms for redetection. Therapy consisted of ATP followed by shock. Ventricular arrhythmia that did not reach the set number of detection intervals was classified as nonsustained. Discriminators for supraventricular tachycardia were standardized based on arrhythmia onset, stability, and ventriculoatrial dissociation.

End Points and Follow-Up

The combined primary end point was the occurrence of a first arrhythmic event (defined as resuscitated cardiac arrest, spontaneous VT or VF, and ICD-detected VT or VF) or death. Secondary end points included arrhythmic events, sudden cardiac death (SCD), type of tachyarrhythmia event, and method of termination. Cause of death was determined by 2 local investigators based on information obtained from witnesses, family members, death certificates provided by the state registry of births and deaths, hospital medical records, rhythm strips, and autopsy reports. A third independent investigator adjudicated if opinion differed. SCD was strictly defined based on a modified Hinkle and Thaler system[11] as death that occurred suddenly and unexpectedly in a patient in otherwise stable condition, inclusive of witnessed instantaneous death (with or without documentation of arrhythmia), unwitnessed death if the patient had been seen within 24 hours before death (in the absence of another clear cause of death), death caused by incessant ventricular tachyarrhythmia, deaths considered a sequel of cardiac arrest, and death resulting from proarrhythmia of antiarrhythmic drugs. Resuscitated cardiac arrest was defined as a sudden circulatory arrest requiring cardiopulmonary resuscitation with the most likely cause a tachyarrhythmia (with or without documented VT or VF) from which the patient regained consciousness. Ventricular tachyarrhythmia was defined as rhythm-documented VT or VF in patients without an ICD or ICD-detected VT or VF requiring treatment (antitachycardia pacing or shock). All patients were followed up by the study investigators throughout their time in hospital and by telephone contact at 1, 3, and 6 months with 6-monthly intervals thereafter. Patients with an ICD were also followed up in the ICD clinic with electrograms of device detections analyzed by the study investigators blinded to the EPS result.

Statistical Analysis

SPSS for Windows (release 21.0) was used to analyze the results. Two-tailed tests with a significance level of 5% were used throughout. χ^2 or Fisher exact tests as appropriate were used to test for association between categorical variables. ANOVA or Kruskal–Wallis equivalent was used to test for differences in the distribution of continuous variables between the groups. Kaplan–Meier curves were used to illustrate the cumulative distribution of death or arrhythmia by time post infarction. Log-rank tests were used to look for differences between the groups. Multiple Cox-regression analysis with backward stepwise variable selection was used to identify the independent predictors of the primary end point. Candidate variables for entry into the model were those associated with the outcome with $P<0.1$. The strategy for retention of variables in the model was a P value of 0.05 and for removal was $P=0.1$.

Results

A total 1910 patients with STEMI underwent primary PCI. LVEF assessment was not performed in 9.9% because of inpatient death before LVEF assessment (n=94; 50%), patient refusal or discharge (n=54; 29%), transfer back to a peripheral hospital (n=21; 11%), and transfer to another treating specialty (n=20; 11%). Early LVEF assessment was performed in 1721 patients at a median of 4 days post-MI with a LVEF ≤40% in 24% (n=414) and a LVEF ≤30% in 9% (n=162).

EPS Results

EPS was performed at median 8 (interquartile range, 6–11) days post-STEMI in 290 patients with LVEF ≤40%. It was not performed in all patients with impaired LVEF because of

Table 1. Characteristics of Electrophysiological Study Result According to Study Group

Variable	EP_{pos} Inducible VT (CL, 200–230 ms; n=58)	EP_{pos} Inducible VT (CL>230 ms; n=29)	*P* Value
CL of first induced VT, ms (mean±SD)	211±8	264±32	0.001
Proportion of VT induced with			<0.001
≤2 extrastimuli	10%	24%	
Third extrastimulus	43%	45%	
Fourth extrastimulus	47%	31%	
Morphology of VT			0.022
LBBB	52%	35%	
RBBB	48%	65%	
Duration, s (mean±SD)	23±10	26±12	0.171
Mode of termination			<0.001
ATP	12%	58%	
Cardioversion	78%	35%	
Spontaneous	10%	7%	

CL indicates cycle length; EP, electrophysiological; LBBB, left bundle-branch block; RBBB, right bundle-branch block; and VT, ventricular tachycardia.

in-hospital death before EPS (n=35; 28%), secondary indication for ICD (n=4; 3%), treating cardiologists' decision to reassess LVEF because of borderline LVEF 38% to 40% (n=68; 55%), patient refusal (n=3; 2%), or patient deemed inappropriate for primary prevention of SCD because of limited life expectancy (old age, significant comorbidities, or malignancy; n=14; 11%). The EPS was negative in 70% (group 1; n=203) with no arrhythmia induced (n=101) or inducible VF/flutter CL <200 ms (n=102). The EPS was positive for inducible monomorphic VT (CL>200 ms) in 30% (n=87), consisting of very fast VT (CL, 200–230 ms) in 67% (group 2; n=58), and standard VT (CL>230 ms) in 33% (group 3; n=29). The characteristics of inducible VT in patients with a positive EPS are shown in Table 1. Patients who had inducible very fast VT (CL, 200–230 ms) were significantly more likely to require 3 or 4 extrastimuli to induce it, have VT of left bundle-branch block morphology, and require cardioversion to terminate it.

Baseline Characteristics

The baseline characteristics for the 3 groups are presented in Table 2. Patients with a negative EPS compared with patients with a positive EPS were significantly more likely to be on a statin at discharge, to not be on antiarrhythmic therapy, and have a higher LVEF. Patients with inducible slower VT (CL>230 ms) were significantly more likely to have a previous history of ischemic heart disease and have a lower LVEF.

ICD Implantation

A predischarge ICD was implanted early after MI in 79 of 87 patients with a positive EPS. An ICD was not implanted in 8 patients with a positive EPS because of patient refusal (n=2), patient not covered by health insurance (n=3), and EP result considered borderline (inducible VT [CL, 200–205 ms]) by treating physician (protocol violation; n=3). A predischarge ICD was implanted in 8 patients with a negative EPS (protocol violations) by treating physicians based on impaired LVEF alone (LVEF≤35%). An ICD was implanted late after discharge in 1 patient with a positive EPS (VT [CL, 200–230 ms]) after a resuscitated cardiac arrest and in 5 patients with a negative EPS: in 3 patients because of repeat STEMI with second EPS and in 2 patients for secondary

Table 2. Baseline Characteristics of Patients With LVEF ≤40% After STEMI According to Electrophysiology Study (N=290)

Variable	EP_{neg} VF/Flutter or No VT (n=203)	EP_{pos} Inducible VT (CL, 200–230 ms; n=58)	EP_{pos} Inducible VT (CL>230 ms; n=29)	*P* Value
Age, y (mean±SD)	57.2±11.4	56.9±10.6	57.8±11.9	0.942
Men	80.3%	91.4%	86.2%	0.118
Background history				
Previous IHD	21.5%	27.6%	42.9%	0.043
Hypercholesterolemia	52.0%	67.2%	64.3%	0.079
Hypertension	42.6%	48.3%	64.3%	0.089
Diabetes mellitus	20.6%	20.7%	39.3%	0.116
Smoker, past or current	68.7%	72.4%	75%	0.236
STEMI territory: anterior	84.7%	79.3%	65.5%	0.062
STEMI treatment				0.109
Intent for PPCI	100%	100%	100%	
PCI	97.5%	93.2%	96.6%	
CABG	1.5%	5.1%	3.4%	
Medical management only	1.0%	1.7%	0	
Post-procedure TIMI III flow	97.5%	93.0%	86.2%	0.065
Discharge medications				
ACE-I or ARB	85.3%	78.2%	80.8%	0.420
β-Blocker	90.6%	81.8%	84.6%	0.172
Statin	98.4%	85.5%	88.5%	<0.001
Aspirin	100%	100%	100%	…
Clopidogrel or prasugrel	97.4%	94.5%	96.2%	0.082
Antiarrhythmic*	0	5.2%	7.1%	0.042
Median LVEF (IQR)	34.0 (29.0–37.0)	32.0 (27.0–37.0)	30.0 (22.2–32.8)	0.001
Proportion patients with LVEF ≤30%	30.5%	39.7%	51.7%	0.052

ACE-I or ARB indicates angiotensin-converting enzyme inhibitor or angiotensin receptor blocker; CABG, coronary artery bypass grafting; CL, cycle length; EP, electrophysiological; IHD, ischemic heart disease; IQR, interquartile range; LVEF, left ventricular ejection fraction; PPCI, primary percutaneous coronary intervention; STEMI, ST-segment–elevation myocardial infarction; TIMI, thrombolysis in myocardial infarction; VF, ventricular fibrillation; and VT, ventricular tachycardia.

*Antiarrhythmic not inclusive of digoxin (amiodarone and sotalol were only antiarrhythmics used).

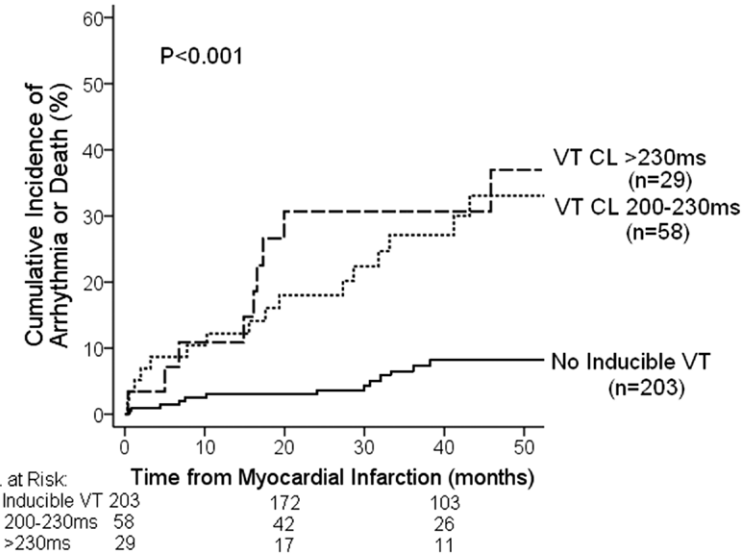

Figure. Kaplan–Meier estimates of arrhythmia or death according to electrophysiological study group. CL indicates cycle length; and VT, ventricular tachycardia.

prevention indications). There were no deaths associated with ICD implantation.

Primary and Secondary End Points

The Kaplan–Meier (Figure) cumulative incidence of first arrhythmia or death differed significantly among the 3 groups (log-rank, $P<0.001$). The 4-year combined event rate was 8.2±2.3% in patients with a negative EPS (no inducible VT or VF/flutter) compared with 34.4±5.8% in EP-positive patients. The 4-year combined event rate in patients with inducible very fast VT (CL, 200–230 ms) at 33.1±7.1% was similar to that of patients with slow inducible VT (CL>230 ms) at 37.0±10.2% (log-rank, $P=0.880$). All independent predictors of the combined end point of arrhythmia or death are shown in Table 3.

Arrhythmic event or death rates during the follow-up are shown in Table 4. The details for each individual patient's arrhythmic event are shown in Table 5. Three patients with a negative EPS had an arrhythmic event. Patient 1 had no arrhythmia induced at EPS and died after an out-of-hospital cardiac arrest with no arrhythmia documented but classified as SCD. Patient 2 had ventricular flutter (CL, 175 ms) induced by 3 extrastimuli (EPS terminated after 3 extrastimuli on both inductions) and represented with spontaneous conscious VT requiring cardioversion. Patient 3 had ventricular flutter (CL, 180 ms) induced by 2 extrastimuli (EPS terminated after 2 extrastimuli on both inductions) and represented with conscious VT, which self terminated. Repeat EPS performed on patient 3 demonstrated inducible VT (CL, 210 ms) with the third extrastimulus. Fifteen patients with inducible very fast VT had an arrhythmic event; because of ICD-treated activations in 13, a SCD and a resuscitated cardiac arrest occurred in 2 patients without an ICD. Five patients with standard inducible VT had an arrhythmic event consisting of 4 with ICD-treated activations and 1 patient who refused an ICD who died of incessant VT. Of the EP-positive patients who experienced an ICD activation (n=17), 100% were alive at 6 months postactivation, and of the 75% (n=12) who completed 12-month follow-up postactivation, 100% were still alive.

Table 3. Significant Predictors of Arrhythmia or Death

	Univariable		Multivariable	
	HR (95% CI)	*P* Value	HR (95% CI)	*P* Value
Inducible very fast VT (CL, 200–230 ms)	4.6 (2.0–10.4)	<0.001	4.2 (2.2–8.3)	<0.001
Inducible standard VT (CL>230 ms)	4.5 (2.3–8.9)	<0.001	3.8 (1.7–8.7)	0.002
LVEF ≤30%	2.7 (1.5–5.0)	0.001	2.3 (1.3–4.3)	0.007

CI indicates confidence interval; CL, cycle length; HR, hazard ratio; LVEF, left ventricular ejection fraction; and VT, ventricular tachycardia.

Discussion

The majority of inducible VT in patients with LVEF ≤40% early after revascularization with PPCI for STEMI is very fast VT (CL, 200–230 ms). This inducible very fast VT incurs a similar risk of tachyarrhythmia or death as inducible standard definition VT (CL>230 ms). Patients early after MI with inducible monomorphic VT at EPS have a significantly higher rate of arrhythmia or death than patients with a negative EPS.

This study adds to previous work demonstrating that induced very fast monomorphic VT, previously termed ventricular flutter, can no longer be considered a nonspecific finding at EPS.[5,12–14] Similar to a study by Kumar et al[5] conducted at our center and published in the *Journal of Cardiovascular Electrophysiology*, we have demonstrated that this induced arrhythmia is strongly predictive of future spontaneous tachyarrhythmia or SCD. The current study differs from this earlier one in that induced VT at EPS was assessed in a homogenous group of patients, early after STEMI, who had all been reperfused with primary PCI. Unlike that seen in Kumar et al, we found that inducible very fast VT was just as strong a predictor of arrhythmia or death than standard slower VT (CL>230 ms).

We demonstrated that very fast VT comprised the majority of inducible VT in patients early after MI (67% of inducible VT in the current study was very fast VT [CL, 200–230 ms]). In previous studies where patients with MI were treated with medical therapy or late reperfusion, the proportion of

Table 4. Primary and Secondary Outcomes According to Electrophysiology Study Groups

Outcome	EP_{neg} No VT or VF/Flutter (n=203)	EP_{pos} Inducible VT (CL, 200–230 ms; n=58)	EP_{pos} Inducible VT (CL>230 ms; n=29)	*P* Value
Proportion of patients with ≥1 arrhythmic event	1.5%	25.9%	17.2%	…
ICD implanted predischarge	3.9%	87.9%	96.6%	<0.001
Median days post-STEMI (IQR)	15.2 (8.5–21.0)	13.0 (10.0–20.0)	11.0 (9.0–17.0)	
ICD implanted later date	2.4%	1.7%	0	…
Median months post-STEMI (IQR)	8.3 (1.0–36.3)	41.0	…	
Proportion of patients with ≥1 appropriate ICD activation because of	0.5%	22.4%	13.7%	0.001
VT	100%	69.2%	75.0%	
VF	…	30.8%	25.0%	
Terminated by				0.069
ATP	100%	69.2%	75.0%	
Shock	…	30.8%	25.0%	
Proportion of patients without a predischarge ICD with ≥1 arrhythmic event	1% (n=2/195)	29% (n=2/7)	100% (n=1/1)	…
Total mortality, comprising	6.9%	8.6%	17.2%	0.166
Sudden cardiac death	0.5%	1.7%	3.4%	
Non–sudden cardiac death	2.5%	3.4%	0	
Non–cardiac death	3.9%	3.5%	13.8%	
Median follow-up, months (IQR)	41.8 (24.2–60.7)	44.0 (26.0–61.0)	38.3 (17.5–65.5)	0.910

CL indicates cycle length; EP, electrophysiological; ICD, implantable cardioverter-defibrillator; IQR, interquartile range; STEMI, ST-segment–elevation myocardial infarction; VF, ventricular fibrillation; and VT, ventricular tachycardia.

inducible very fast VT was much lower, ranging from 9% to 30%.[10,13,14] The higher occurrence of faster inducible VT in our cohort likely reflects the differing substrate, with patients who are revascularized having smaller, less dense infarcts. We found that patients with slower inducible VT (CL>230 ms) had a significantly lower LVEF ($P<0.01$) and a higher incidence of previous MI ($P=0.04$), likely a reflection of a larger infarcted area of myocardium. Wijnmaalen et al[15] published important findings in *Circulation* of the influence of early reperfusion on the arrhythmogenic substrate for inducible VT. They found that early reperfusion was associated with faster VTs and consequent less confluent scars on histology. In comparison, the larger, denser scars seen in patients without reperfusion produced slower VT CT. The induction of very fast VT has become more relevant in the contemporary era of early and more effective reperfusion with primary PCI, where in our study it can now be seen to make up the majority of inducible VT post-MI.

We found that patients with a positive EPS had a high rate of arrhythmia or SCD consistent with previous work conducted at our institution[4,6,8] and other centers.[2,16–18] Of the 8 patients in the EP-positive group who violated protocol and did not receive an ICD, more than one third went on to die from an arrhythmic death. The predictive value of EPS differs between studies, likely a consequence of differences in the VT induction protocol. The Multicenter Automatic Defibrillator Implantation Trial II investigators found inducible monomorphic VT predicted future VT, but not VF.[17] The more recent CARISMA (Cardiac arrhythmias and risk stratification after acute myocardial infarction) study demonstrated that although EPS was significantly predictive of arrhythmia, it was inferior to heart rate variability.[16] Both studies did not include very fast VT in their definition of monomorphic VT. Given our findings that the majority of inducible VT early after MI is now very fast VT, the exclusion of this would result in many high-risk patients being classified as having a negative or nonpredictive EP result. In a previous study, we have also shown that the prognostic significance of VT induced by the fourth extrastimulus is similar to that of VT induced by ≤3 extrastimuli in patients post-MI.[19] Limitation of the VT induction protocol to 3 extrastimuli would have resulted in one third of patients at high arrhythmic risk being classified as EP negative. After a mean follow-up of ≈4 years, we found that 98% of patients with a negative EPS were free of arrhythmia or sudden death, despite a one third of these patients having a LVEF ≤30% and only 6% (who violated study protocol) protected by an ICD. This is consistent with our previous results, with Kumar et al[6] demonstrating a 96% 2-year arrhythmia free survival in EP-negative patients and Zaman et al[4] showing no arrhythmic deaths in EP-negative patients with LVEF ≤30% after 2-year follow-up. The 2 patients with a negative EPS in the current study, who went on to have spontaneous VT, did not complete the full EP protocol of ≤4 extrastimuli. This was because of the occurrence of VF on the second or third extrastimulus, requiring cardioversion and cessation of the test. Hence, the negative predictive value of our EP protocol may be limited in a small number of patients in whom the full VT induction cannot be completed.

The DINAMIT (Defibrillator IN Acute Myocardial Infarction Trial) investigators implanted defibrillators 6 to 40 days after MI in patients with abnormal heart rate variability and LVEF ≤35%.[20] The IRIS (Immediate Risk Stratification

Table 5. Details of Patients Who Experienced an Arrhythmic Event

	Early LVEF%	Heart Failure*	Arrhythmic Event (Cycle Length, Terminated By)	Months Post-MI
EP_{neg} (n=203)				
Patient 1	23	No	SCD	7.5
Patient 2	29	Yes	VT (CL, 350; self-reverted)	30.0
Patient 3	35	No	VT (CL, 290; shock)	0.5
Mean±SD or median (IQR)	29±6		320±42	7.5 (0.5–7.5)
EP_{pos} VT (CL, 200–230 ms; n=58)				
Patient 1	14	No	VT (CL, 330; ATP)	27.3
Patient 2	19	Yes	VF (CL, 220; ATP)	15.6
Patient 3	21	No	VT (CL, 270; shock)	0.53
Patient 4	24	No	VT (CL, 330; ATP)	3.2
Patient 5	25	No	VT (CL, 300; ATP)	1.2
Patient 6	26	Yes	SCD	0.3
Patient 7	27	Yes	VF (CL, 190; shock)	53.2
Patient 8	27	No	VT (CL, 350; ATP)	10.3
Patient 9	30	Yes	Resuscitated cardiac arrest	43.2
Patient 10	31	Yes	VT (CL, 260; ATP)	31.8
Patient 11	32	Yes	VT (CL, 350; ATP)	2.0
Patient 12	33	No	VF (CL, 180; shock)	41.2
Patient 13	33	Yes	VT (CL, 340; ATP)	17.6
Patient 14	36	No	VT (CL, 290; ATP)	54.1
Patient 15	40	No	VF (CL, 170; shock)	19.3
Mean±SD or median (IQR)	28±7		275±66	17.6 (2.0–41.2)
EP_{pos} VT (CL, >230 ms; n=29)				
Patient 1	17	No	VF (CL, 210; shock)	6.8
Patient 2	20	No	VT (CL, 250; ATP)	14.9
Patient 3	27	No	VT (CL, 280; ATP)	45.8
Patient 4	30	No	VT (CL, 390; ATP)	5.0
Patient 5	30	No	VT (CL, 260; shock) and SCD	0.4
Mean±SD or median (IQR)	25±6		278±68	6.8 (2.7–30.4)

CL indicates cycle length; EP, electrophysiological; IQR, interquartile range; LVEF, left ventricular ejection fraction; SCD, sudden cardiac death; STEMI, ST-segment–elevation myocardial infarction; VF, ventricular fibrillation; and VT, ventricular tachycardia.

*Only inclusive of heart failure occurring during the index STEMI admission with clinical or radiographic evidence requiring treatment with decongestive therapy (intravenous diuretics, intravenous nesiritide, intravenous inotropes), invasive or noninvasive ventilation, or intra-aortic balloon pump. All CLs are in ms.

Improves Survival) trial also looked at early ICD implantation selecting patients based on impaired LVEF and elevated resting heart rate.[21] In both trials, the rate of SCD was significantly reduced; however, this was counterbalanced by an increase in the rate of non–sudden death. The use of low heart rate variability and resting tachycardia seemed to identify patients at increased risk of cardiac mortality, rather than discriminating between arrhythmic and nonarrhythmic causes of death.[22] In our current study, of the patients with inducible VT and an ICD who experienced ICD treatment, 100% of these patients were still alive at 6 months post-treatment. It seems that EPS clearly identifies patients at risk of arrhythmia, who benefit from an ICD, who do not go on to die of non-SCD.

Limitations

The main limitation of this study was its observational nature. As ICDs were not implanted in EP-negative patients, there was an unavoidable bias in the detection of arrhythmic events. ICD-detected VT/VF overestimates SCD by 2- to 4-fold,[23] which limits any comparison in arrhythmic events of overall EP-positive with overall EP-negative patients (although the very low SCD rate of <1% in the EP-negative group is reassuring). However, the primary aim of this study to compare inducible very fast VT to slower VT was not influenced by this bias, as both these groups received ICDs as per our study protocol.

Disclosures

None.

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CLINICAL PERSPECTIVE

The presence of inducible ventricular tachycardia (VT) at electrophysiological study (EPS) identifies patients at risk of spontaneous ventricular tachyarrhythmia. EPS may play a role in risk stratification for prevention of sudden cardiac death early after myocardial infarction. However, the use of EPS in identifying high-risk patients seems critically dependent on the classification of inducible VT. Standard inducible VT cycle length >230 ms is commonly classified as a positive result, whereas inducible very fast VT (cycle length, 200–230 ms) has been historically termed ventricular flutter and a negative EPS result. In the current study, 290 consecutive patients with ST-segment–elevation myocardial infarction with left ventricular ejection fraction ≤40% underwent early EPS with inducible monomorphic VT (cycle length, ≥200 ms) classified as a positive result. We found that one third of patients early after ST-segment–elevation myocardial infarction had inducible VT according to this definition, with the majority comprising very fast VT. Inducible very fast VT was just as strong a predictor of tachyarrhythmia or death as standard slower inducible VT. Our findings suggest that inducible very fast VT cannot be considered a nonspecific finding at EPS in contemporary patients with ST-segment–elevation myocardial infarction. Although the use of EPS early after myocardial infarction remains controversial, this study has important implications for future trials assessing EPS as a risk stratification tool for primary prevention of sudden cardiac death.

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