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What is the optimal left ventricular ejection fraction cut-off for risk stratification for primary prevention of sudden cardiac death early after myocardial infarction?

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Aims The optimal left ventricular ejection fraction (LVEF) to select patients early post myocardial infarction (MI) for risk stratification for prevention of sudden cardiac death (SCD) in the era of primary percutaneous coronary intervention (PPCI) is unknown.

Methods and results Consecutive patients ($n = 1722$) treated with PPCI for ST-elevation MI underwent early (median 4 days) LVEF assessment. An electrophysiological study (EPS) was performed if LVEF $\leq$40% and a prophylactic implantable-cardioverter defibrillator (ICD) implanted for a positive [inducible monomorphic ventricular tachycardia (VT)], but not a negative, result. According to an early LVEF, a primary endpoint of inducible VT at EPS and a secondary endpoint of death or arrhythmia (SCD, resuscitated cardiac arrest or ECG-documented VT/ventricular fibrillation) were determined. The proportion of patients with early LVEF >40, 36–40, 31–35, and $\leq$30% were 75% ($n = 1286$), 7% ($n = 128$), 8% ($n = 136$), and 10% ($n = 172$), respectively. Inducible VT occurred in 22, 25, and 40% of patients with LVEF 36–40, 31–35, and $\leq$30%, respectively ($P = 0.014$). Three-year death or arrhythmia occurred in 6.6 $\pm$ 0.8, 8.1 $\pm$ 2.6, 18.0 $\pm$ 3.4, and 37.4 $\pm$ 3.9% of patients with LVEF >40, 36–40, 31–35, and $\leq$30%, respectively (overall $P < 0.001$; LVEF 36–40% vs. LVEF > 40% $P = 0.265$). The number of EPS-positive patients implanted with an ICD to treat one or more arrhythmic event (95% confidence interval) was 18.3 $\pm$ 2.4, 11.5 $\pm$ 3.0, and 4.2 $\pm$ 5.6 if LVEF is 36–40, 31–35, and $\leq$30%, respectively.

Conclusion A cut-off LVEF of $\leq$40% selects patients with a high incidence of inducible VT post-PPCI. Patients with LVEF $\leq$35% and inducible VT appear to derive a greater benefit from prophylactic ICD implantation due to their higher risk of death or arrhythmia.

Keywords Sudden cardiac death • Myocardial infarction • Left ventricular function • Electrophysiology study

Introduction

Impaired left ventricular ejection fraction (LVEF) is one of the strongest predictors of sudden death or arrhythmia following myocardial infarction (MI). However, LVEF alone has limited specificity when used to select patients for ICD implantation for primary prevention of sudden cardiac death (SCD). An electrophysiological study (EPS) demonstrates the presence of an electrical substrate for re-entrant ventricular tachyarrhythmia and consistently predicts arrhythmic risk in observational and randomized studies.[1–7] The EPS has been shown to be safe when performed in the early post-MI phase[8,9] with potential utility in guiding early ICD implantation.[8,10] Widespread implementation of primary percutaneous coronary intervention (PPCI) has resulted in declining overall mortality, yet

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What's new?

- This is the first study to assess the incidence of both inducible ventricular tachycardia and death or spontaneous arrhythmia according to an early left ventricular ejection fraction in contemporary ST-elevation MI patients treated with primary percutaneous coronary intervention
- This study provides valuable information on the risk of sudden death and arrhythmia in patients early after ST-elevation MI, a time where the risk of SCD is highest, yet primary prevention with ICD implantation remains controversial

SCD incidence remains 7- to 10-folds higher in the early post-MI phase.[11–13] Risk stratification for an ICD with non-invasive methods in this early phase has not yet been successful.[14,15] However, research into alternate risk stratification tests to identify patients early after MI who benefit from ICD implantation is ongoing. In order to progress in this area of early prevention of SCD, an appropriate LVEF cut-off must be established. The two major early prevention trials (DINAMIT and IRIS)[14,15] used a LVEF dichotomy of $\leq$35 and $\leq$40%, respectively, to select patients for further non-invasive risk stratification. We aimed to determine the optimal LVEF dichotomy limit to select patients early after PPCI for ST-elevation MI (STEMI) to undergo further risk stratification with EPS, for primary prevention of SCD. The secondary aim was to determine the long-term incidence of spontaneous tachyarrhythmia and overall prognosis based on an early LVEF post-STEMI.

Methods

Consecutive patients with STEMI treated with PPCI at a single tertiary centre from 2004–11 were prospectively recruited. The study complies with the Declaration of Helsinki, the research protocol was approved by the hospital appointed ethics committee, and all patients gave their written informed consent. Patients presented directly to the intervention-capable Westmead Hospital or were referred by three associated district hospitals. ST-elevation myocardial infarction was defined as persistent ST elevation or presumed new left bundle branch block with either characteristic symptoms of myocardial ischaemia and/or subsequent release of biomarkers of myocardial necrosis. All patients were taken to the cardiac catheterization laboratory with intent for PPCI with no patients receiving thrombolytic therapy. Patients underwent inpatient assessment of LV function at Days 3–5 post-MI with gated heart pool scan (GHPS) or transthoracic echocardiogram (TTE) where GHPS was not available. Following early revascularization, patients with LV dysfunction were commenced on optimal medical therapy including beta-blockers, ACE-I, statins, and anti-platelet therapy.

Electrophysiological study

The EPS was performed after full revascularization in patients with LVEF $\leq$40% as part of a primary prevention strategy for prevention of SCD. This centre's post-MI SCD prevention approach has been described in detail previously.[8,10,16] Programmed ventricular stimulation (PVS) was performed at the right ventricular apex (single site) using a drive train of eight beats at 400 ms cycle length (CL) followed by up to four extrastimuli. Extrastimuli were added, each starting with a coupling interval of 300 ms, until ventricular refractoriness. If ventricular fibrillation (VF)/flutter were induced then cardioversion was performed and that PVS was stopped. If the first PVS was negative a repeat PVS would be performed from the same site, utilizing the same protocol of up to four extrastimuli. The sustained monomorphic ventricular tachycardia (VT) CL $\geq$ 200 ms induced by four or less extra stimuli was considered a positive EPS result. A negative EPS result was no inducible arrhythmia or inducible VF/flutter CL $<$ 200 ms after completion of two PVS protocols. Pre-discharge ICD implantation was recommended for EPS-positive patients while EPS-negative patients were discharged without an ICD irrespective of LVEF.

Endpoints and follow-up

The primary endpoint was inducible VT at EPS early after MI. The secondary endpoint was a combined endpoint of death (all-cause) and/or arrhythmia (arrhythmia defined as SCD, resuscitated cardiac arrest, and ECG-documented sustained VT or ventricular fibrillation). The cause of death was determined by two 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. Sudden cardiac death was strictly defined based on a modified Hinkle and Thaler system[17] 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 h 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 pro-arrhythmia of anti-arrhythmic drugs. Resuscitated cardiac arrest was defined as a sudden circulatory arrest requiring cardiopulmonary resuscitation (with or without documented VT or VF) from which the patient regains consciousness. Ventricular tachyarrhythmia was defined as ECG-documented VT or VF in patients without an ICD, or ICD-detected VT or VF which required treatment to terminate (anti-tachycardia pacing or shock). Cardiac mortality included both sudden and non-SCDs with non-SCDs defined as death due to MI, heart failure (HF), or another cardiovascular cause. Heart failure was defined as symptoms or signs consistent with congestive HF (either clinical or radiographic evidence) requiring treatment with decongestive therapy (diuretics or inotropes), intra-aortic balloon pump, or invasive/non-invasive ventilation. Only HF during the index STEMI admission was assessed. All patients were followed by the study investigators throughout their time in hospital and by telephone contact at 1, 3, and 6 months after discharge with 6-monthly intervals thereafter. Patients with an ICD were also followed in the ICD clinic with electrograms of device detections or activations analysed by the study investigators.

Statistical analysis

SPSS for Windows (release 21.0) was used to analyse the results. Two-tailed tests with a significance level of 5% were used throughout. χ^2 or Fisher's exact tests as appropriate were used to test for association between categorical variables. Analyses of variants or Kruskal–Wallis equivalent were 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 the primary and secondary endpoints 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 endpoint of death or arrhythmia. 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 STEMI patients treated with PPCI were recruited. Of these patients, 188 (9.8%) did not undergo early LVEF assessment. The reasons for this included inpatient death prior to LVEF assessment ($n = 94$, 50%), patient refusal or discharge prior to LVEF assessment ($n = 54$, 29%), transfer back to peripheral hospital ($n = 20$, 11%), and transfer to another treating specialty ($n = 20$, 11%). Early LVEF assessment was performed in 1722 patients at a median of 4 days post-STEMI by GHPS in 87% ($n = 1506$) and echocardiogram in 13% ($n = 216$). Patients with LVEF >40% made up 75% ($n = 1286$) while the remaining 25% of patients post-STEMI had an early LVEF of ≤40% ($n = 436$). Of the total cohort of STEMI patients, 7% had an LVEF of 36–40% ($n = 128$), 8% had an LVEF of 31–35% ($n = 136$), and 10% had an LVEF of ≤30% ($n = 172$). The baseline characteristics according to an early LVEF are shown in *Table 1*.

Inducible ventricular tachycardia at electrophysiological study

The EPS was performed at a median 8 (IQR 6–11) days post-STEMI in 290 patients with LVEF ≤40%. This included 91 (72%) patients with LVEF 36–40%, 96 (71%) with LVEF 31–35%, and 103 (60%) with LVEF ≤ 30%. It was not performed in all patients with impaired LVEF due to either in-hospital death prior to EPS ($n = 35$, 28%), secondary indication for ICD ($n = 4$, 3%), treating cardiologists' decision to reassess LVEF due to borderline result of 38–40% ($n = 68$, 55%), patient refusal ($n = 3$, 2%) or patient deemed inappropriate for primary prevention of SCD due to limited life-expectancy (old age, significant co-morbidities or malignancy, $n = 14$, 11%). The EPS was negative in 70% ($n = 203$) and positive in 30% ($n = 87$) with the proportion of patients with inducible VT differing significantly ($P = 0.014$) according to an early LVEF (*Figure 1*). Negative EPS consisted of no inducible arrhythmia in 49% and inducible VF/flutter in 51%. The mean CL of

Table 1 Baseline characteristics according to an early LVEF following STEMI

Characteristic	LVEF≤30% ($n = 172$)	LVEF 31–35% ($n = 136$)	LVEF 36–40% ($n = 128$)	LVEF >40% ($n = 1286$)	*P* value
Age (years), mean ± SD	62 ± 13	61 ± 13	60 ± 13	59 ± 12	0.027
Male gender	79%	74%	85%	80%	0.122
Background history:					
Prior IHD	32%	33%	27%	22%	0.001
Hypercholesteraemia	56%	58%	63%	57%	0.647
Hypertension	56%	49%	55%	54%	0.672
Diabetes mellitus	30%	30%	24%	23%	0.270
Smoker, past, or current	67%	62%	67%	68%	0.278
Anterior STEMI	77%	80%	73%	34%	<0.001
Number of coronary vessels with ≥50% stenosis:					<0.001
Single vessel	35%	56%	45%	48%	
Double vessel	21%	15%	28%	30%	
Triple vessel or left main	44%	30%	27%	22%	
STEMI treatment:					0.022
PPCI	91%	93%	93%	96%	
CABG	6%	2%	5%	2%	
Medical management only	3%	5%	2%	2%	
Post-procedure TIMI III flow	91%	94%	95%	96%	0.065
HF during STEMI admission	58%	38%	23%	10%	<0.001
Discharge pharmacotherapy:					
ACE-I or ARB	83%	89%	77%	88%	0.102
Beta-blocker	91%	89%	84%	86%	0.537
Statin	92%	98%	95%	99%	0.281
Aspirin	100%	100%	100%	100%	–
Clopidogrel or prasugrel	94%	97%	100%	100%	0.180
Anti-arrhythmic	6%	2%	2%	0	0.422

IHD, ischaemic heart disease; STEMI, ST-elevation myocardial infarction; PPCI, primary percutaneous coronary intervention; CABG, coronary artery bypass grafting; TIMI, thrombolysis in myocardial infarction flow score; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; anti-arrhythmic not inclusive of digoxin (amiodarone and sotalol were the only anti-arrhythmics used).

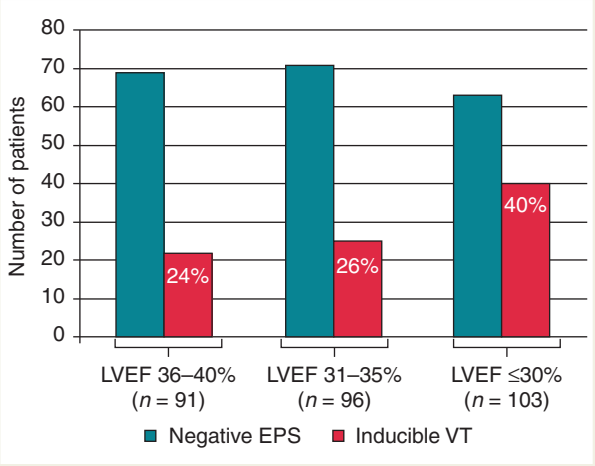

Figure 1 Proportion of patients with inducible VT at EP study in each LVEF group ($P = 0.014$ for LVEF ≤ 30% vs. others).

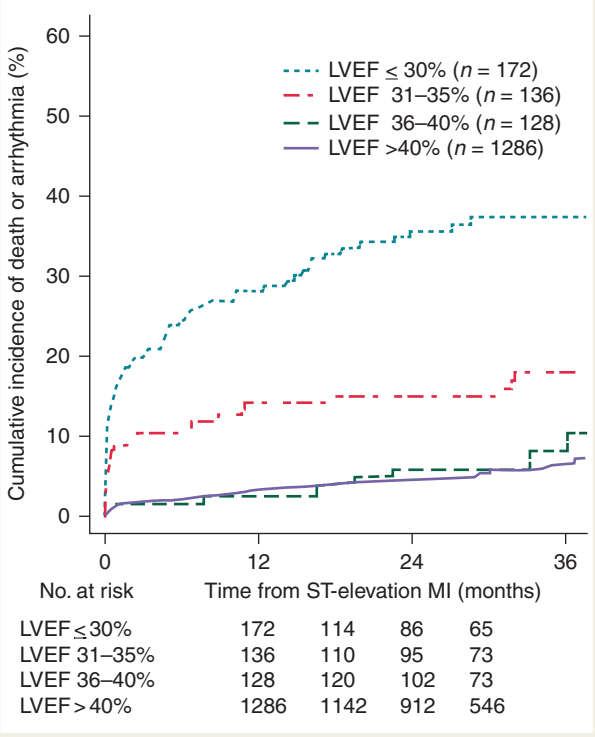

Figure 2 Kaplan–Meier estimated cumulative death or arrhythmia according to an early LVEF following STEMI.

inducible VT was 217 ± 18, 227 ± 28, and 233 ± 38 in patients with LVEF 36–40, 31–35, and ≤30%, respectively ($P = 0.141$). There were no deaths or serious complications associated with early EPS.

Secondary outcomes

The overall median follow-up was 32 (IQR 24–50) months. The Kaplan–Meier estimated cumulative incidence of death or arrhythmia (*Figure 2*) was significantly higher in patients with LVEF ≤30% and LVEF 31–35% compared with patients with LVEF >40% ($P < 0.001$). Patients with LVEF 36–40% had a comparable incidence of death or arrhythmia as patients with preserved LVEF >40% ($P = 0.265$). During the median follow-up, arrhythmic events occurred in 2.4% ($n = 41$) of patients consisting of 0.5, 3, 5, and 12.8% of patients with LVEF >40, 36–40, 31–35 and ≤30%, respectively. The baseline characteristics of the patients according to whether they experienced an arrhythmic event, died from a cause other than SCD, or survived free of death or arrhythmia are shown in *Table 2*. The multivariable model for the predictors of the endpoint of death or arrhythmia is shown in *Table 3*. Amongst patients with LVEF ≤40% who were eligible for an EPS ($n = 290$), the only independent predictor of death or arrhythmia was inducible VT at EPS (hazard ratio 4.1, confidence interval 2.2–7.9; $P < 0.001$).

The Kaplan–Meier cumulative incidence of arrhythmia in patients with LVEF ≤40% with a positive EPS is shown in *Figure 3*. Patients with a positive EPS had significantly higher rates of arrhythmia than patients with a negative EPS in all three impaired LVEF groups ($P = 0.017$, $P < 0.001$, and $P < 0.001$ for EPS-negative patients vs. EPS-positive patients with LVEF 36–40, 31–35 and ≤30%, respectively). During the follow-up period, one SCD occurred in the group with LVEF ≤40% and a negative EPS (1/203, <1%). By 3 years the number of patients who were EPS positive who received an ICD in order to treat one or more arrhythmic event was 18.3 ± 2.4, 11.5 ± 3.0, and 4.2 ± 5.6 (95% confidence interval) according to if LVEF was 36–40, 31–35, or ≤30%, respectively (*Figure 4*).

Discussion

Patients in the current study were selected for risk stratification with EPS based on an early LVEF dichotomy of ≤40%. We found that significantly higher proportions of patients with severe LV dysfunction (EF ≤ 30%) had inducible VT compared with only a quarter of patients with moderate LV dysfunction (EF 31–40%). All patients in our cohort were STEMI patients treated with PPCI. In comparison, STEMI patients treated with thrombolysis have been found to have an incidence of inducible VT of 38%.[18] Chong *et al.*[18] demonstrated that patients with LVEF ≤40% had similar susceptibility to inducible VT irrespective of treatment with thrombolysis or PPCI. Early LVEF can overestimate long-term LV impairment given the phenomenon of myocardial stunning,[19] particularly after PPCI, where there is a greater proportion with patent arteries compared with lysis.[20] We found that patients who suffered an arrhythmic event had significantly lower rates of TIMI III flow post-PPCI with corresponding higher rates of inducible VT. The pathophysiological basis for development of re-entrant ventricular tachyarrhythmia after MI is multifactorial. The scar provides the underlying substrate with prolonged refractory periods within the infarct core and border zone.[21] Re-entrant VT circuits have been seen to form and stabilize within the first week post-infarction, with early inducibility of VT at EPS at Day 8 post-MI concordant with inducibility of VT at Day 100 in an ovine model.[22]

The VALIANT trial demonstrated that post-MI patients with LVEF ≤30% had the highest rate of sudden death or arrhythmia however; they also found that almost half of the absolute events still occurred in those with preserved EF (31–40%).[11] Our findings in patients treated exclusively with PPCI are consistent with this landmark trial with a

Table 2 Baseline characteristics according to the outcome

Characteristic	Arrhythmic event: SCD, cardiac arrest, VT/VF ($n = 41$)	Death from cause other than SCD ($n = 171$)	*P* value*	Survival free of arrhythmia or death ($n = 1516$)	*P* value†
Age (years), mean ± SD	61 ± 12	70 ± 12	0.001	59 ± 12	0.358
Male gender	93%	68%	<0.001	81%	0.033
Background history:					
Prior IHD	30%	39%	0.345	22%	0.163
Hypercholesteraemia	63%	50%	0.148	58%	0.353
Hypertension	58%	72%	0.087	52%	0.312
Diabetes mellitus	37%	31%	0.553	24%	0.096
Smoker, past, or current	80%	72%	0.018	67%	0.191
Anterior STEMI territory	68%	46%	0.016	44%	<0.001
Number of coronary vessels with ≥50% stenosis:			0.200		<0.001
Single vessel	33%	34%		49%	
Double vessel	30%	17%		28%	
Triple vessel or left main	37%	49%		23%	
Post-procedure TIMI III flow	76%	90%	0.016	96%	<0.001
Clinical HF at study entry	54%	47%	0.464	13%	<0.001
Proportion of patients with an EPS who had inducible monomorphic VT, % (number)	83% (19/23)	38% (8/21)	0.003	25% (61/248)	<0.001
LVEF, mean ± SD	31 ± 13	42 ± 14	<0.001	50 ± 12	<0.001

SCD, sudden cardiac death; VT, ventricular tachycardia; VF, ventricular fibrillation; IHD, ischaemic heart disease; STEMI, ST elevation myocardial infarction; TIMI, thrombolysis in myocardial infarction flow score; EPS, electrophysiological study; LVEF, left ventricular ejection fraction.
**P* value comparison between patients' with an arrhythmic event and patients' with death from cause other than SCD.
†*P* value comparison between patients' with an arrhythmic event and patients' survival free of arrhythmia or death.

Table 3 Predictors of the combined endpoint of death or arrhythmia

	Univariable model		Multivariable model	
	Unadjusted HR (95% CI)	*P* value	Adjusted[a] HR (95% CI)	*P* value
Inducible VT at EPS (positive EPS)	4.4 (2.4–8.2)	<0.001	4.1 (2.2–7.9)	**<0.001**
LVEF ≤30%	1.8 (1.6–2.0)	<0.001	1.6 (0.8–3.3)	0.082
HF	5.5 (4.2–7.2)	<0.001	1.7 (0.9–3.1)	0.090
Post-PCI TIMI flow ≤ 2	2.7 (1.8–4.0)	<0.001	1.5 (0.5–4.4)	0.517
Age (decade increase)	1.8 (1.6–2.0)	<0.001	1.1 (0.8–1.4)	0.807
Anterior STEMI	1.2 (0.9–1.6)	0.120	1.3 (0.5–3.0)	0.558

HR, hazard ratio; CI, confidence interval; VT, ventricular tachycardia; LVEF, left ventricular ejection fraction; PCI, percutaneous coronary intervention; TIMI, thrombolysis in MI flow score.
[a]Each HR has been adjusted for all other variables in the model.

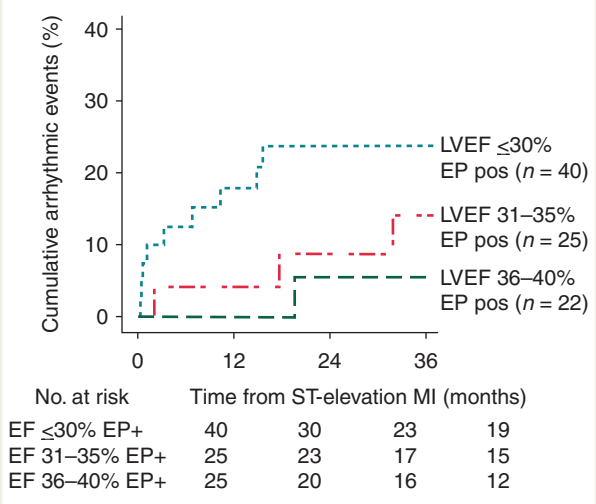

Figure 3 Kaplan–Meier cumulative arrhythmic events (SCD, cardiac arrest, VT/VF) in EPS positive patients with LVEF ≤40% who were stratified to receive an ICD.

significant proportion of arrhythmia or SCD (~40%) occurring in patients with LVEF 31–40%. We found that patients who were free of death or arrhythmia were more likely to have single vessel coronary artery disease, TIMI III flow post-PPCI, less HF, and a higher LVEF (*Table 2*). In contrast, the presence of an anterior STEMI, poor TIMI flow, impaired LVEF and HF were all associated

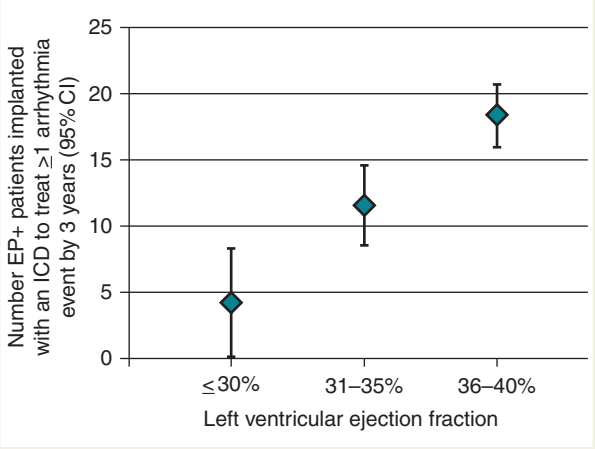

Figure 4 Number of ICDs implanted by 3 years in order to treat one or more arrhythmic event in patients with a positive EPS according to the LVEF group.

with the combined endpoint of death or arrhythmia. Consistent with the VALIANT trial, we found no difference in the occurrence of HF between patients with an arrhythmic event and patients with non-sudden death. Following adjustment for all variables, the only independent predictor of the combined death/arrhythmia endpoint was inducible VT at EP study. While non-invasive methods may be ideal to identify high-risk patients, randomized trials have not yet demonstrated a mortality benefit when used to guide early ICD implantation. The DINAMIT and IRIS trials, which utilized autonomic dysfunction combined with LVEF, found that early ICD implantation reduced SCD but this was offset by a high occurrence of non-SCDs.[14,15] In contrast, the EPS has the potential to identify patients at risk of predominant arrhythmic mortality who may also survive long-term after ICD treatment for arrhythmia.[23]

The evidence supporting late post-MI ICD implantation for primary prevention has largely come from randomized trials utilizing LVEF as the sole selection tool.[24,25] We found that LVEF was significantly lower in patients who died of both arrhythmic and non-arrhythmic deaths; however, it was not an independent predictor of death and arrhythmia when all other variables were adjusted for. The major concerns with LVEF alone to select for an ICD are its low specificity for arrhythmic vs. non-arrhythmic deaths[26] and limited sensitivity whereby a large proportion of SCDs occur above a single LVEF cut-off.[11,27] The MADIT-II and SCD-HeFT trials which used LVEF alone found that 11 and 14 patients required treatment with a defibrillator to save one life.[28] In comparison, the MUSTT and MADIT trials where the EPS was used in addition to LVEF required four and five patients treated with an ICD to save one life.[2] We cannot directly compare the current study with these randomized trials as it is well documented that ICD-detected VT or VF can overestimate SCD by two- to four-folds.[29,30] However, we can demonstrate that the EPS in addition to an early LVEF dichotomy of ≤30 or 31–35% required approximately 4 and 12 patients to receive an ICD to treat at least one arrhythmic event by 3 years, respectively. In contrast, the benefit of ICD implantation in patients with LVEF 36–40%, even in the presence of inducible VT, is uncertain.

The majority of patients with LVEF ≥35% are at low overall risk of death or arrhythmia. However, as shown in previous studies[11,27] a reasonable number of total SCDs still occur in this cohort. Further elucidation of arrhythmia risk assessment in patients with preserved ejection fraction is required, in order to guide ICD implantation in a manner that remains cost-effective.

Limitations

The main limitation of this study was its observational nature. However, LVEF was systematically measured, EPS considered in all patients with LVEF ≤40%, and patients uniformly and prospectively followed, which enabled the discriminatory analysis between the LVEF categories. A higher number of ICDs were implanted in patients with lower LVEF and a positive EPS which would have resulted in a bias in the detection of the secondary arrhythmia outcome. While a LVEF dichotomy of ≤40% appears to be reasonable for selecting patients to undergo EPS, a randomized study is necessary to determine the benefit of ICD implantation in patients with inducible VT and varying levels of LV dysfunction. The LVEF dichotomy assessed in this study was solely to utilize the EPS for risk stratification to determine the need for an ICD. It is unlikely that a single LVEF dichotomy limit will ever be completely satisfactory in selecting patients for ICD treatment, and we did not assess LVEF cut-off for other risk stratification modalities.

Conflict of interest: none declared.

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Dual implantable electronic device therapy

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Dual-implantable electronic device (IED) therapy is challenging since potential device interaction might occur resulting in inappropriate ICD therapy. A 57-year-old male was referred to our implantable cardioverter defibrillator (ICD) clinic for interrogation of his ICD before, during and after implantation of a device for gastric electrical stimulation (GES). Implantation of the device for GES (*Figure*) was scheduled for the treatment of the patient's severe chronic intractable nausea secondary to diabetic gastroparesis.

We investigated the safety and efficacy of the GES device in this ICD patient up to 3 months after GES device implantation. During follow-up, no interactions of the dual-IED therapy were detected with the applied device programming. The GES device provided symptom relief and the proper functioning of each of the dual IEDs was assured by device interrogation.

The full-length version of this report can be viewed at: http://www.escardio.org/communities/EHRA/publications/ep-case-reports/Documents/Dual-implantable-electronic-device-therapy.pdf.