Patients treated with catheter ablation for ventricular tachycardia after an ICD shock have lower long-term rates of death and heart failure hospitalization than do patients treated with medical management only

T. Jared Bunch, MD,[*] J. Peter Weiss, MD,[*] Brian G. Crandall, MD,[*] John D. Day, MD, FHRS,[*] Heidi T. May, PhD, MSPH,[*] Tami L. Bair, RN,[*] Jeffrey S. Osborn, MD,[*] Charles Mallender, MD,[*] Avi Fischer, MD,[†] Kyle J. Brunner, MBA,[†] Srijoy Mahapatra, MD[†]

From the [*]Intermountain Heart Institute, Intermountain Medical Center, Murray, Utah, and [†]St Jude Medical Corporation, St. Paul, Minnesota.

BACKGROUND Ventricular arrhythmias in patients with implantable cardioverter-defibrillators (ICDs) adversely affect outcomes. Antiarrhythmic approaches to ventricular tachycardia (VT) have variable efficacy and may increase risk of ventricular arrhythmias, worsening cardiomyopathy, and death. Comparatively, VT ablation is an alternative approach that may favorably affect outcomes.

OBJECTIVE To further explore the effect on long-term outcomes after catheter ablation of VT, we compared patients with history of ICD shocks who did not undergo ablation, patients with a history of ICD shocks that underwent ablation, and patients with ICDs who had no history of ICD shocks.

METHODS A total of 102 consecutive patients with structural heart disease who underwent VT ablation for recurrent ICD shocks were compared with 2088 patients with ICDs and no history of appropriate shocks and 817 patients with ICDs and a history of appropriate shocks for VT or ventricular fibrillation. Outcomes considered were mortality, heart failure hospitalization, atrial fibrillation, and stroke/transient ischemic attack.

RESULTS The mean age of 3007 patients was 65.4 ± 13.9 years. Over long-term follow-up, 866 (28.8%) died, 681 (22.7%) had a heart failure admission, 706 (23.5%) developed new-onset atrial fibrillation, and 224 (7.5%) had a stroke. The multivariate-adjusted risks of deaths and heart failure hospitalizations were higher in patients with history of ICD shocks who were treated medically than in patients with ICDs and no history of shock (hazard ratio [HR] 1.45; $P < .0001$ vs HR 2.00; $P < .0001$, respectively). The multivariate-adjusted risks were attenuated after VT ablation with death and heart failure hospitalization rates similar to those of patients with no shock (HR 0.89; $P = .58$ vs HR 1.38; $P = .09$, respectively). A similar nonsignificant trend was seen with stroke/transient ischemic attack.

CONCLUSIONS Patients treated with VT ablation after an ICD shock have a significantly lower risk of death and heart failure hospitalization than did patients managed medically only. The adverse event rates after VT ablation were similar to those of patients with ICDs but without VT.

KEYWORDS Ventricular tachycardia; Catheter ablation; Heart failure; Mortality; Atrial fibrillation

ABBREVIATIONS **AF** = atrial fibrillation; **HR** = hazard ratio; **ICD** = implantable cardioverter-defibrillator; **ICD-9** = International Classification of Diseases, Ninth Revision; **IQR** = interquartile range; **TIA** = transient ischemic attack; **VF** = ventricular fibrillation; **VT** = ventricular tachycardia

(Heart Rhythm 2014;11:533–540)

Introduction

The wide use of implantable cardioverter-defibrillators (ICDs) for the primary and secondary prevention of ventricular fibrillation (VF) or ventricular tachycardia (VT) in patients with cardiomyopathy has improved survival.[1–5] As a consequence of favorable survival data and more patients surviving with advanced cardiomyopathies, the number of ICD implantations has increased significantly in the last decade. In general, the use of medications has declined owing to variable efficacy and proarrhythmia risk.[6] ICD therapies for new or recurrent arrhythmias are common, and

Dr Bunch is a consultant for St Jude Medical and Biosense Webster as well as an advisory board member with Boston Scientific. Dr Weiss is a consultant for Merit Medical, Stereotaxis, and Biosense Webster. Dr Crandall is a consultant and speaker for Merit Medical, St Jude Medical, and Boston Scientific. Dr Osborn is a consultant and speaker for Medtronic, St Jude Medical, Boston Scientific, and Cook. Dr Fischer, Dr Brunner, and Dr Mahapatra are employees of St Jude Medical. **Address reprint requests and correspondence:** Dr T. Jared Bunch, Intermountain Heart Rhythm Specialists, Intermountain Medical Center, Eccles Outpatient Care Center, 5169 Cottonwood St, Suite 510, Murray, UT 84107. E-mail address: Thomas.bunch@imail.org.

1547-5271/$-see front matter http://dx.doi.org/10.1016/j.hrthm.2013.12.014

antiarrhythmic drug therapy over time to reduce arrhythmic events in patients with ICDs for secondary prevention is often used.[7]

Although ICD shocks for rapid VT or VF can be lifesaving, the shocks can have deleterious consequences. The startle and pain associated with an ICD shock can lead to emotional distress and posttraumatic stress disorder.[8–10] In addition, ICD shocks of all types are associated with increased mortality despite termination of the acute event. In this regard, ventricular tachyarrhythmias are treated only if they are sustained and rapid enough to lead to probable cardiac instability or neurologic compromise.[11] Finally, minimizing the presence of rapid ventricular arrhythmias can reduce risk of presyncope or syncope, worsening cardiomyopathy in those patients with a high burden of events, and favorably reduce cardiovascular symptoms related to the arrhythmia itself.

All antiarrhythmic drugs used to reduce ventricular arrhythmias in patients with structural heart disease have potential adverse reactions. These include risk of proarrhythmia, a potential negative effect on the defibrillation threshold, lowering of a VT rate below that of the detection window of the ICD resulting in delayed diagnosis and treatment, and worsening of cardiomyopathy. In addition to these cardiovascular adverse reactions, there are many drug-related side effects that prompt discontinuation. In trials examining their efficacy in patients with ICDs, discontinuation rates varied from 18% to 35%.[12,13]

With the limitations noted with antiarrhythmic drugs for VT suppression or treatment in patients with structural heart disease, percutaneous catheter ablation of VT has emerged to become a standard of care to prevent medically refractory ICD shocks.[14–17] Since a catheter ablation for VT, if successful, can minimize long-term exposure to antiarrhythmic drugs or substantially reduce their dose requirement, it may result in better long-term outcomes such as death and heart failure. Of additional interest is the long-term risk of atrial arrhythmias and stroke after ablation since these rhythms can be suppressed by the antiarrhythmic drugs used to treat VT. In this regard, their risk may increase but the increased risk may be attenuated by minimizing the negative effects of antiarrhythmic agents on cardiac function. To further explore the effect on long-term outcomes after catheter ablation of VT, we compared patients with history of ICD shocks who did not undergo ablation and patients with ICDs and no history of ICD shocks.

Methods

Patient populations

We studied patients with structural heart disease who underwent ICD implantation for VT at an Intermountain Healthcare hospital (LDS Hospital and Intermountain Medical Center, Salt Lake City, UT; McKay Dee Hospital, Ogden, UT; Utah Valley Regional Medical Center, Provo, UT; and Dixie Regional Medical Center, St. George, UT) and had 3 years or greater in follow-up care after their ICD was implanted. The study was approved by the institutional board review of the Intermountain Medical Center.

The patient population was stratified into 3 groups: patients who received no follow-up shock after ICD implantation (n = 2088), patients who received an appropriate follow-up shock for VT or VF (n = 817) and did not receive an ablation procedure, and patients who underwent catheter ablation for VT after ICD implantation (n = 102). Patients were treated by their electrophysiologists, cardiologists, or primary care physicians, who made individual choices regarding an ablation approach, antiarrhythmic medications, and/or anticoagulation. The VT ablation procedure provided was individualized and varied by the operator who performed it. This study characteristic provided a general sense of outcomes across multiple procedural approaches, but it also compromised our ability to look at procedural characteristics and outcomes. In general, noninducibility of the clinical VT after ablation was the goal as documented by postprocedural and discharge summary notes. Rates of epicardial access among centers ranged from 0% to 16%. An epicardial approach was undertaken only in those patients who had failed endocardial ablation. At the Intermountain Medical Center, more procedural variables were available. CARTO electroanatomic mapping was used to determine scar ($\leq$1.5 mV) and, if possible, activation mapping. If activation mapping was possible, the critical region on slow conduction was targeted. In patients in whom activation mapping was not possible owing to hemodynamic stability, scar substrate modification was performed to homogenize scar borders and target diastolic potentials within the scar.

After VT ablation, patients are typically seen at 1 month and every 3 months thereafter for the first year. The frequency of subsequent care with them varies, depending on the patient and the involvement of his or her cardiologist. ICD programming reflected the preferences of the primary electrophysiologist. During the study period, the most common empiric ICD programming involved a VF, shock only zone of $<$320 ms, 1 attempt at antitachycardia pacing in a fast VT zone (240–320 ms), and then a VT zone (320–360 ms) in which antitachycardia pacing was applied for at least 3 attempts, with supraventricular tachycardia discriminators programmed on. We do not have details regarding programming changes made after the first ICD shock or VT ablation.

All-cause mortality was considered the primary study end point. Deaths were determined by hospital records and Utah State Health Department records (death certificates) and were verified through Social Security death records. We performed a telephone survey of 1000 patients previously and found a 99% accuracy of mortality detection when using these 2 data sets. Patients not listed as deceased in any registry were considered to be alive.

Heart failure hospitalization was determined by searching the *International Classification of Diseases, Ninth Revision* [*ICD-9*]) codes 398.91, 402.01, 402.11, 402.91, 404.01, 404.03, 404.11, 404.13, 404.91, 404.93, and 428.0 for primary diagnosis only. Patients' atrial fibrillation (AF)

status over time was determined by searching the hospital discharge summary for diagnostic codes for AF (*ICD-9* code: 427.31; *ICD-10* codes: I480, I481, I482, and I4891) at index and previous admissions to Intermountain Healthcare hospitals (Salt Lake City, UT, and its surrounding areas) and by searching the electrocardiographic database of all Intermountain Healthcare hospitals, which is maintained on electronic records. The electrocardiographic database includes electrocardiograms, ambulatory monitor reports, and symptom- and auto-triggered event monitor reports from all Intermountain Healthcare hospitals. These databases are updated daily with completion of the dictated medical reports and physician reviews of the ordered electrocardiograms. Stroke and transient ischemic attack (TIA) were determined by using *ICD-9* codes: 433_1 and 434_1 (the underscore denotes any one character), and 435, respectively.

Statistical analysis

The Student *t* test and the χ^2 statistic were used to evaluate baseline and clinical characteristics among patients with ICDs on the basis of the presence of shocks and therapies. Multivariable Cox hazard regression analysis (SPSS, version 21.0) was used to evaluate the association of shocks and VT ablation with the incidence of the study end points. For each specific outcome (total mortality, heart failure hospitalization, stroke, TIA, and atrial arrhythmias), a model was developed that only included confounding (10% change in hazard ratio [HR]) and significant ($P < .05$) covariables. Covariables included baseline risk factors identified through *ICD-9* codes and physician reports that were documented either at or before index (baseline) date (Table 1), which was defined as ICD implantation. Two-tailed *P* values of $<.05$ were designated to be nominally significant. We estimated survival free rates by using the Kaplan-Meier method and log-rank test for all-cause mortality. Survival rates were compared between all 3 cohorts described previously.

Results

Of the 3007 patients, the mean age was 65.4 ± 13.9 years. The median days of follow-up after ICD implantation was 1243.5 days (interquartile range [IQR] 534.0–2162.0 days) (no shock: 1125.0 [IQR 451.0–1940.5]; shock, no ablation: 1546 [792.5–2518.0]; shock, ablation: 2112.0 [1086.5–3074.3]; $P < .0001$).

The median days from ICD implantation to shock and from ICD implantation to ablation were 412.0 (84.0–1204.0) and 686.5 (215.3–1806.8), respectively. The basic demographic characteristics of the 3 study groups are summarized in Table 1. Patients who did not experience a shock were older, were less often men, and had more hypertension and worse renal function. The ablation group had a higher average ejection fraction. The most common antiarrhythmic

Table 1 Baseline characteristics of the patient populations at the time of ICD implantation

Characteristic	No shock (n = 2088)	Shock, no ablation (n = 817)	Shock, ablation (n = 102)	*P*
Age (y)	66.2 ± 13.9	63.7 ± 14.0	62.1 ± 12.8	<.0001
Sex: male	73.2%	76.0%	90.2%	<.0001
Hypertension	80.7%	74.4%	66.7%	<.0001
Hyperlipidemia	73.4%	71.4%	65.7%	.07
Diabetes	41.8%	38.8%	22.5%	<.0001
Heart failure (NYHA class ≥2)	89.2%	82.3%	74.5%	<.0001
Renal failure (creatinine >2.0)	21.1%	20.8%	12.7%	.13
Coronary artery disease	79.3%	78.7%	75.5%	.63
Prior MI	30.4%	31.0%	29.4%	.93
CVA/TIA	5.3%	4.7%	3.9%	.66
Valve history	45.3%	40.5%	44.1%	.07
Atrial fibrillation or flutter history	15.5%	12.6%	18.6%	.08
$CHADS_2$ score				<.0001
0	2.9%	6.5%	10.5%	
1	14.0%	19.2%	27.4%	
2	27.7%	26.4%	33.1%	
3	35.5%	34.0%	15.3%	
4	13.0%	8.4%	9.7%	
≥5	6.9%	5.4%	4.0%	
EF (%)	32.2 ± 12.6	32.8 ± 14.2	37.6 ± 14.8	<.0001
ACE inhibitor	55.2%	56.4%	55.6%	.85
Antiplatelet	61.6%	61.0%	73.7%	.04
Angiotensin receptor blocker	16.0%	12.7%	11.1%	.05
Aspirin	58.4%	58.5%	72.7%	.02
β-Blocker	79.3%	75.1%	76.8%	.05
Calcium channel blocker	5.8%	5.4%	9.1%	.34
Diuretic	60.2%	55.7%	48.5%	.01
Statin	60.5%	55.1%	56.6%	.03
Warfarin	23.0%	21.6%	23.5%	.72

ACE = angiotensin-converting enzyme; CVA/TIA = cerebrovascular accident/transient ischemic attack; EF = ejection fraction; ICD = implantable cardioverter-defibrillator; MI = myocardial infraction; NYHA = New York Heart Association.

Table 2 Time-dependent death rates (A) and hazard ratios for death (B) in patients with an ICD who have no history of a shock (referent), a history of a shock treated medically only, and a history of a shock treated with catheter ablation

	No shock	Shock, no ablation	Shock, ablation	*P*
		A: Time-dependent death rate		
Death	23.4% (488/2088)	43.2% (353/817)	24.5% (25/102)	<.0001
3 y	19.2% (284/1478)	22.5% (163/725)	9.0% (8/89)	.006
5 y	28.2% (288/1021)	34.8% (204/586)	17.6% (13/74)	.001
		B: Hazard ratios for death		
	Shock, no ablation vs no shock		Shock, ablation vs no shock	
	Univariable	Multivariable	Univariable	Multivariable
Death	HR 1.40; $P < .0001$	HR 1.56; $P < .0001$	HR 0.68; $P = .07$	HR 0.89; $P = .58$
3 y	HR 1.27; $P = .01$	HR 1.34; $P = .003$	HR 0.55; $P = .06$	HR 0.67; $P = .21$
5 y	HR 1.39; $P < .0001$	HR 1.51; $P < .0001$	HR 0.63; $P = .07$	HR 0.80; $P = .37$

HR = hazard ratio; ICD = implantable cardioverter-defibrillator.

drug used after ICD shock was amiodarone (shock, ablation: 37.4% vs shock, no ablation: 24.5%; $P < .0001$)

Over long-term follow-up, 866 (28.8%) patients died. Table 2A lists the absolute numbers of deaths per group. In the shock, no ablation group, the rates were consistently higher at all the time strata studied. Death rates were the lowest in the shock, ablation group. The univariable- and multivariate-adjusted HRs showing these risks are presented in Table 2B. The Kaplan-Meier survival curve free of death analyzed by the 3 studied groups is shown in Figure 1.

Regarding the other outcomes of interest, 681 (22.7%) patients had a heart failure admission, 706 (23.5%) developed AF, and 224 (7.5%) had a stroke. Heart failure admissions were highest in the shock, no ablation group (n = 303 [37.1%]) vs the other 2 groups (no shock: n = 345 [16.5%]; shock, ablation: n = 33 [32.4%]; $P < .0001$). AF was highest in the shock, ablation group (n = 44 [43.1%]) than in the other 2 groups (no shock: n = 424 [20.3%]; shock, no ablation: n = 238 [29.1%]; $P < .0001$). After multivariate adjustment of baseline risk factors, the risk of AF in the shock, ablation group compared with the other groups became nonsignificant. The multivariable-adjusted HRs for each of these end points are listed in Table 3.

Figure 2 displays the Kaplan-Meier survival curves based on ejection fraction. Survival was highest in the no shock and shock, ablation groups over time in all ejection fraction groups studied except those with ejection fraction ≥0.50.

Table 4 lists the absolute event rates of death, atrial arrhythmias, heart failure hospitalizations, and cerebral

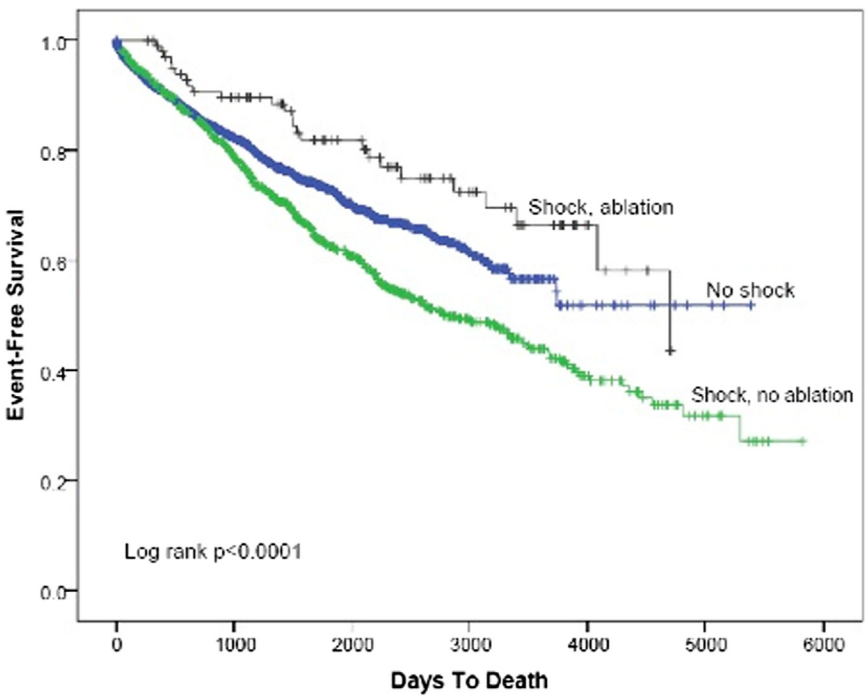

Figure 1 The multivariate-adjusted Kaplan-Meier survival curve obtained after ICD implantation for freedom of all-cause mortality. Patients with ICDs are compared in 3 groups: no shock; shock, no ablation; and shock, ablation. Survival was significantly better in the no shock and shock, ablation groups over time (log-rank $P < .0001$). ICD = implantable cardioverter-defibrillator.

Table 3 Multivariable-adjusted hazard ratios of patients with an ICD who have no history of a shock, a history of a shock treated medically only, and a history of a shock treated with catheter ablation

	Shock, no ablation vs no shock	Shock, ablation vs no shock	Shock, no ablation vs shock, ablation
Atrial fibrillation	HR 1.17; $P = .06$	HR 1.49; $P = .02$	HR 0.79; $P = .17$
Atrial flutter	HR 1.32; $P = .20$	HR 1.53; $P = .33$	HR 0.86; $P = .74$
Cerebrovascular accident	HR 1.22; $P = .31$	HR 1.09; $P = .84$	HR 1.12; $P = .80$
Transient ischemic attack	HR 1.34; $P = .16$	HR 1.22; $P = .68$	HR 1.11; $P = .83$
Heart failure hospitalization	HR 2.00; $P < .0001$	HR 1.45; $P = .05$	HR 1.38; $P = .09$

HR = hazard ratio; ICD = implantable cardioverter-defibrillator.

ischemic based on cardiomyopathy status (ischemic and nonischemic) at 3 years, 5 years, and long-term. Compared with the general analysis, this subanalysis was similar with regard to lower mortality and heart failure hospitalization rates in the no shock and shock, ablation groups. AF and cerebral ischemic event rates remained the highest in the shock, ablation group than in the other groups.

Finally, Figure 3 displays the Kaplan-Meier survival curve based on freedom from a recurrent appropriate ICD shock after catheter ablation vs initiation of an antiarrhythmic drug. There was an early benefit that persisted with regard to a lower risk of an ICD shock in the shock, ablation group than in the shock, no ablation group ($P < .0001$).

Discussion

This study has several important findings. First, over long-term follow-up, mortality rates after VT ablation for medically refractive ICD shocks are similar to those of patients

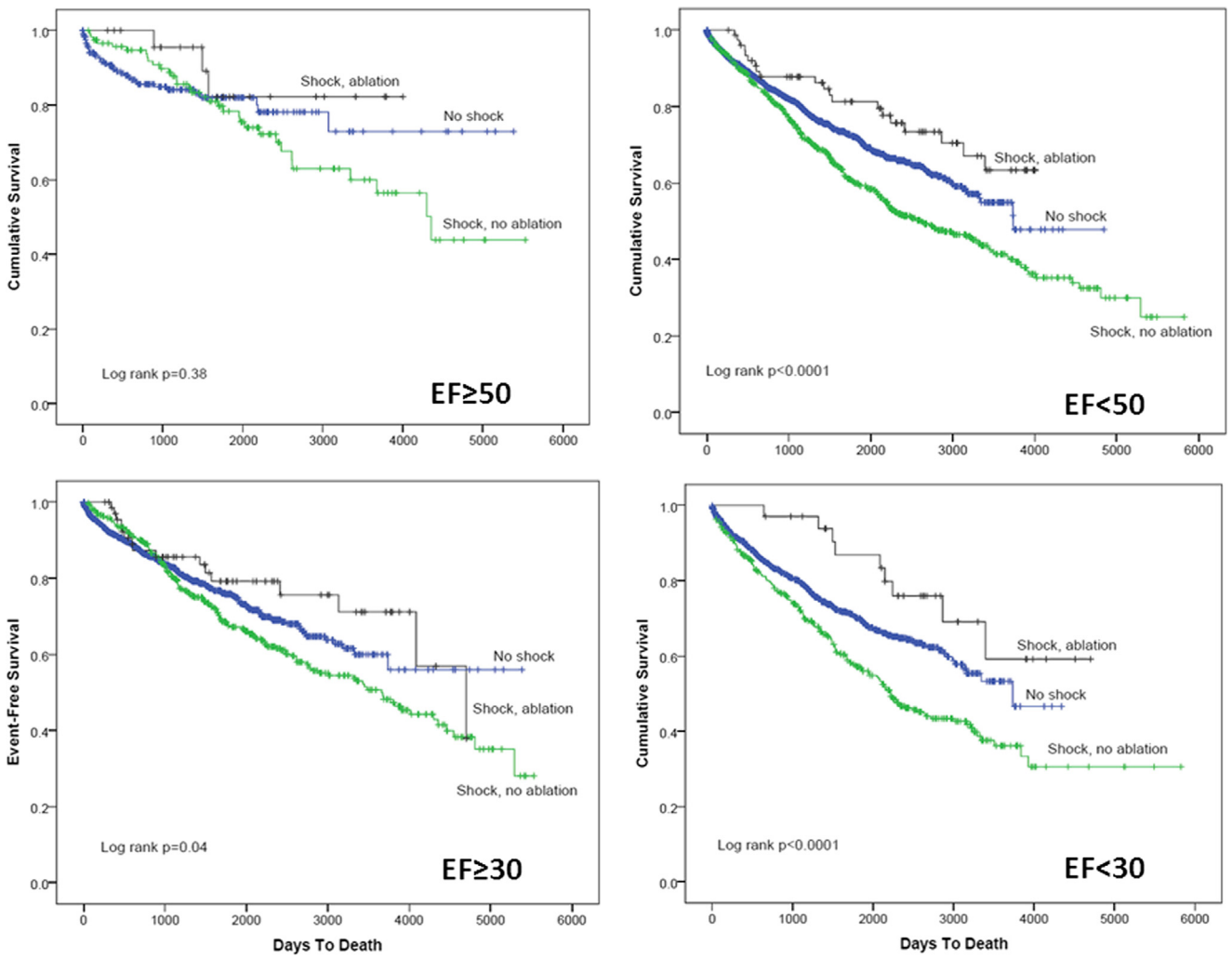

Figure 2 The multivariate-adjusted Kaplan-Meier survival curves obtained after ICD implantation for freedom of all-cause mortality. Patients with ICDs are compared in 3 groups: no shock; shock, no ablation; and shock, ablation. Graphs are stratified by ejection fraction. Survival was significantly better in the no shock and shock, ablation groups over time (log-rank $P < .0001$) in all ejection fraction groups studied except those patients with ejection fraction ≥ 0.50. EF = ejection fraction; ICD = implantable cardioverter-defibrillator.

Table 4 Subanalysis of outcomes based on ischemic vs nonischemic cardiomyopathy

	No shock	Shock, no ablation	Shock, ablation	*P*
		Ischemic heart disease		
Death				
3 y	21.7% (255/1174)	25.7% (148/576)	10.3% (7/68)	.008
5 y	32.6% (269/825)	39.7% (186/469)	21.4% (12/56)	.004
Long-term	27.0% (447/1656)	49.5% (318/643)	27.3% (21/77)	<.0001
Atrial fibrillation				
3 y	36.7% (431/1174)	39.8% (229/576)	38.2% (26/68)	.47
5 y	42.2% (348/825)	49.7% (233/469)	37.5% (21/56)	.02
Long-term	21.1% (349/1656)	30.2% (194/643)	39.0% (30/77)	<.0001
Cerebrovascular accident				
3 y	2.6% (30/1174)	3.6% (21/576)	0% (0/68)	—
5 y	4.2% (35/825)	4.7% (22/469)	0% (0/56)	—
Long-term	3.7% (61/1656)	5.8% (37/643)	6.5% (5/77)	.06
Transient ischemic attack				
3 y	2.5% (29/1174)	3.8% (22/576)	1.5% (1/68)	.23
5 y	3.9% (32/825)	5.3% (25/469)	3.6% (2/56)	.46
Long-term	3.2% (53/1656)	5.1% (33/643)	3.9% (3/77)	.10
Heart failure hospitalization				
3 y	16.5% (194/1174)	25.7% (148/576)	11.8% (8/68)	<.0001
5 y	20.6% (170/825)	32.2% (151/469)	25.0% (14/56)	<.0001
Long-term	18.7% (310/1656)	37.3% (240/643)	32.5% (25/77)	<.0001
		Nonischemic heart disease		
Death				
3 y	9.5% (29/304)	10.1% (15/149)	4.8% (1/21)	.74
5 y	9.7% (19/196)	15.4% (18/117)	5.6% (1/18)	.22
Long-term	9.5% (41/432)	20.1% (35/174)	16.0% (4/25)	.002
Atrial fibrillation				
3 y	25.3% (77/304)	32.2% (48/149)	28.6% (6/21)	.30
5 y	31.6% (62/196)	38.5% (45/117)	61.1% (11/18)	.03
Long-term	29.4% (127/432)	50.6% (88/174)	72.0% (18/25)	<.0001
Cerebrovascular accident				
3 y	2.3% (7/304)	2.0% (3/149)	0 (0/21)	—
5 y	1.0% (2/196)	2.6% (3/117)	5.6% (1/18)	.36
Long-term	2.5% (11/432)	4.0% (7/174)	4.0% (1/25)	.62
Transient ischemic attack				
3 y	1.3% (4/304)	3.4% (5/149)	9.5% (2/21)	.08
5 y	2.0% (4/196)	4.3% (5/117)	11.1% (2/18)	.17
Long-term	1.2% (5/432)	3.4% (6/174)	8.0% (2/25)	.05
Heart failure hospitalization				
3 y	6.9% (21/304)	27.5% (41/149)	23.8% (5/21)	<.0001
5 y	9.2% (18/196)	30.8% (36/117)	22.2% (4/18)	<.0001
Long-term	8.1% (35/432)	36.2% (63/174)	32.0% (8/25)	<.0001

with ICDs who do not experience shocks. In addition, over time, patients treated with ablation experience fewer appropriate ICD shocks than those treated with medication only. ICD shocks can result in myocardial injury and have been associated with an increased risk of mortality.[18–21] Furthermore, efforts to reduce ICD shocks have been shown to reduce mortality.[11] Despite ICD programming changes, many patients with ICDs receive an appropriate shock that is often lifesaving. To minimize additional shocks, antiarrhythmic drugs are often used, but as discussed previously, these therapies are associated with many potential adverse side effects including some that drive mortality risk.

Catheter ablation is a means to accomplish many potentially favorable objectives. First is to reduce or prevent additional ICD therapies. Second is to minimize exposure to long-term antiarrhythmic therapies or improve their efficacy. In contrast to antiarrhythmic medications that are typically required long-term, ablation, if successful, is a single point in time therapy. The composite of achieving these objectives can result in favorable long-term benefits. In the prophylactic catheter ablation for the prevention of defibrillator therapy trial (SMASH-VT trial), a mortality benefit was observed. Although not significant, mortality was lower in the ablation group than in the control group (9% vs 17%; $P = .29$).[22] In pooled analysis of 5 studies, one of which was the SMASH-VT trial, there was a nonsignificant trend in mortality reduction after catheter ablation (HR 0.76; $P = .541$). Our data also show consistently lower mortality rates in patients with ICDs after catheter ablation for VT across both ischemic and nonischemic cardiomyopathy subtypes as well as over diverse baseline ejection fraction profiles than in patients with ICDs and medical management of VT alone. The stronger mortality benefit observation in our study was likely due to several factors. First, the SMASH-VT trial was a

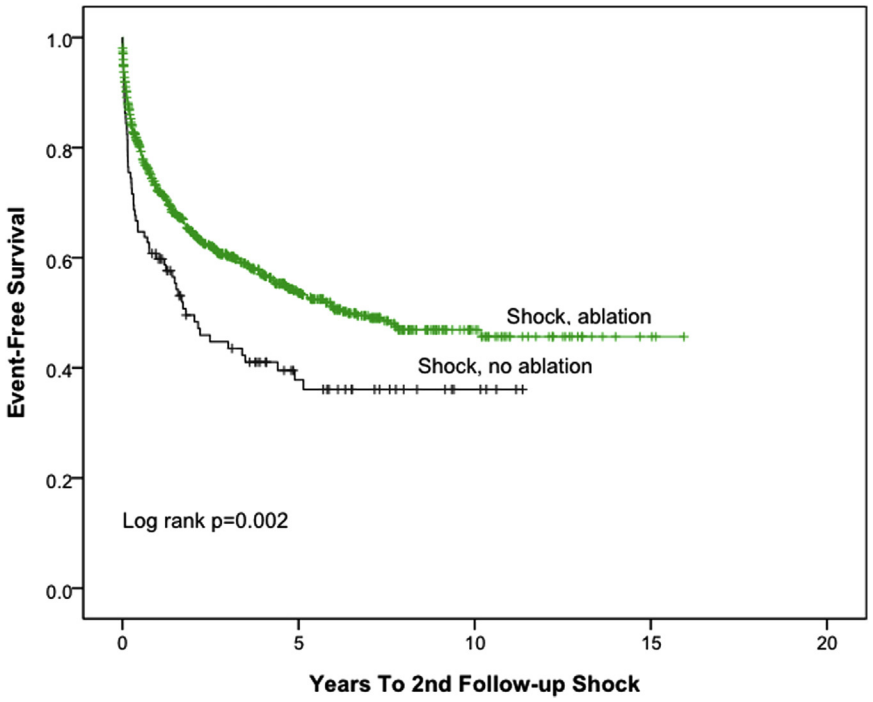

Figure 3 The multivariate-adjusted Kaplan-Meier survival curve obtained for patients with ICDs for freedom of a subsequent appropriate ICD shock after receiving an appropriate ICD shock with comparison based on therapy. Comparison is made between patients treated with ablation vs those treated medically. The ablation group had an early and sustained ICD shock reduction benefit (log-rank $P < .0001$). ICD = implantable cardioverter-defibrillator.

prospective study in which both groups had to meet inclusion criteria for trial participation. This lessens the possibility of a procedural-based selection bias that would select a relatively healthier population for the procedure compared with those who did not undergo the procedure. Next, our study includes both ischemic and nonischemic heart disease and as such the temporal mortality patterns and appropriate ICD shock risk may be different from those of the studies in which ischemic disease was largely studied. However, these data reflect outcomes in a large community that spans distinct practice styles and approaches and as such likely approximate real life results and outcomes obtained when ICD implantation and subsequent long-term management of arrhythmias is undertaken.

Antiarrhythmic medications can affect cardiac function and minimize the ability to titrate or use other medical therapies that affect heart function such as β-blockers and renin-aldosterone inhibitors. The effect of therapeutic strategies on heart function was evaluated in the Ventricular Tachycardia Ablation in Coronary Heart Disease study, a trial comparing ICD implantation with and without VT ablation in patients with stable VT, a previous myocardial infarction, and left ventricular dysfunction.[23] The 24-month survival free from cardiac hospitalization for cardiac reasons (HR 0.45; $P = .013$) was significantly reduced in those patients treated with catheter ablation than in those treated with an ICD and medical management only. Our outcome data are supportive of these data in that we found a significant decreased risk of heart failure hospitalization after catheter ablation with both ischemic and nonischemic cardiomyopathy in patients with ICDs and VT.

Clearly, there are significant risks with VT ablation. In an analysis of 5 catheter ablation for VT studies, complication rates included 1% risk of death, 1% risk of cardiac perforation, and 1.6% risk of complete heart block. Some of these significant complications affected mortality outcomes in the included studies.[24] One other potential problem with discontinuing antiarrhythmic drugs in patients with structural heart disease, if the ablation is successful, is that these agents also treat atrial arrhythmias. As heart failure advances, the incidence of AF can be as high as 50%.[25] Removal of these drugs may result in higher risks of AF over time. Also, catheter ablation of VT in the epicardium (~10% of the study population) can cause postprocedural AF, but this procedural aspect should not be a long-term effect.[26] We found that new-onset AF was increased in the group that underwent ablation. The majority of risk was accounted for in multivariate analysis that suggests it may have been driven by coexistent disease. However, future studies of AF risk after VT ablation are required to understand if this finding represents a unique risk in the heart failure population or if it reflects the characteristics of the VT ablation group observed in this retrospective study. However, there were also slightly higher rates of stroke and TIA long-term after ablation, which likely reflected concurrent higher rates of atrial arrhythmias at baseline. In multivariate analysis to account for atrial arrhythmias in the shock, ablation group, the higher risk of cerebral ischemic events was no longer significant. We cannot exclude the possibility of atrial arrhythmias that become manifest after discontinuing antiarrhythmic drugs may affect morbidity long-term.

Study limitations

Our study has several limitations. It is an epidemiologic study from a large health care database that we have used to identify associations but not to establish causality or mechanisms. As such, the study relies on physicians to make and document the disease states. The treatment of patients is individualized, and this may directly affect the risks of morbidity and mortality. The presence of patient comorbidities can affect referral for a VT ablation procedure that can also drive some of the mortality curves observed. The presence of a significant survival benefit in the sickest groups supports our conclusions as well as the lack of benefit in those with ejection fraction ≥ 0.50. There are unique complications with catheter ablation that require consideration in understanding the effect of this invasive therapy, such a vascular access injury, perforation, and iatrogenic ICD lead injury or dislodgement. In our system-wide analysis, we were not confident that all these complications were accurately searchable and reported and as such we have not included them in this analysis. Finally, ICD programming can affect risk of appropriate and inappropriate ICD therapies. We do not have detailed information regarding ICD programming in general or after ablation. This is an important limitation to consider when interpreting these study data.

Conclusions

Patients treated with VT ablation after an ICD shock have a significantly lower risk of death and heart failure hospitalization compared with patients managed medically only, independent of ejection fraction of underlying cardiomyopathy type. AF was highest in the shock, ablation group, which was in part due to preexisting comorbidities including more AF before ICD implantation and could also represent in part lower rates of antiarrhythmic drug use long-term that would have been used to treat VT.

References

1. Bardy GH, Lee KL, Mark DB, et al. Sudden Cardiac Death in Heart Failure Trial I. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. N Engl J Med 2005;352:225–237.
2. Bigger JT Jr. Coronary Artery Bypass Graft (CABG) Patch Trial Investigators. Prophylactic use of implanted cardiac defibrillators in patients at high risk for ventricular arrhythmias after coronary-artery bypass graft surgery. N Engl J Med 1997;337:1569–1575.
3. Buxton AE, Lee KL, Fisher JD, et al. Multicenter Unsustained Tachycardia Trial Investigators. A randomized study of the prevention of sudden death in patients with coronary artery disease. N Engl J Med 1999;341:1882–1890.
4. Moss AJ, Hall WJ, Cannom DS, et al. Multicenter Automatic Defibrillator Implantation Trial Investigators. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. N Engl J Med 1996;335:1933–1940.
5. Moss AJ, Zareba W, Hall WJ, et al. Multicenter Automatic Defibrillator Implantation Trial III Investigators. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med 2002;346:877–883.
6. Al-Khatib SM, LaPointe NM, Curtis LH, et al. Outpatient prescribing of antiarrhythmic drugs from 1995 to 2000. Am J Cardiol 2003;91:91–94.
7. The CASCADE Investigators. Randomized antiarrhythmic drug therapy in survivors of cardiac arrest (the CASCADE Study). Am J Cardiol 1993;72:280–287.
8. Ahmad M, Bloomstein L, Roelke M, Bernstein AD, Parsonnet V. Patients' attitudes toward implanted defibrillator shocks. Pacing Clin Electrophysiol 2000;23:934–938.
9. Sears SF, Matchett M, Conti JB. Effective management of ICD patient psychosocial issues and patient critical events. J Cardiovasc Electrophysiol 2009;20:1297–1304.
10. Sears SF, Hauf JD, Kirian K, Hazelton G, Conti JB. Posttraumatic stress and the implantable cardioverter-defibrillator patient: what the electrophysiologist needs to know. Circ Arrhythm Electrophysiol 2011;4:242–250.
11. Moss AJ, Schuger C, Beck CA, et al. Reduction in inappropriate therapy and mortality through ICD programming. N Engl J Med 2012;367:2275–2283.
12. Connolly SJ, Dorian P, Roberts RS, et al. Comparison of beta-blockers, amiodarone plus beta-blockers, or sotalol for prevention of shocks from implantable cardioverter defibrillators: the OPTIC Study: a randomized trial. JAMA 2006;295:165–171.
13. Pacifico A, Hohnloser SH, Williams JH, et al. d,l-Sotalol Implantable Cardioverter-Defibrillator Study Group. Prevention of implantable-defibrillator shocks by treatment with sotalol. N Engl J Med 1999;340:1855–1862.
14. Aliot EM, Stevenson WG, Almendral-Garrote JM, et al. EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias: developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). Europace 2009;11:771–817.
15. Stevenson WG, Friedman PL, Kocovic D, Sager PT, Saxon LA, Pavri B. Radiofrequency catheter ablation of ventricular tachycardia after myocardial infarction. Circulation 1998;98:308–314.
16. Callans DJ, Zado E, Sarter BH, Schwartzman D, Gottlieb CD, Marchlinski FE. Efficacy of radiofrequency catheter ablation for ventricular tachycardia in healed myocardial infarction. Am J Cardiol 1998;82:429–432.
17. Miller MA, Dukkipati SR, Mittnacht AJ, et al. Activation and entrainment mapping of hemodynamically unstable ventricular tachycardia using a percutaneous left ventricular assist device. J Am Coll Cardiol 2011;58:1363–1371.
18. Barker-Voelz MA, Van Vleet JF, Tacker WA Jr, Bourland JD, Geddes LA, Schollmeyer MP. Alterations induced by a single defibrillating shock applied through a chronically implanted catheter electrode. J Electrocardiol 1983;16:167–179.
19. Tereshchenko LG, Faddis MN, Fetics BJ, Zelik KE, Efimov IR, Berger RD. Transient local injury current in right ventricular electrogram after implantable cardioverter-defibrillator shock predicts heart failure progression. J Am Coll Cardiol 2009;54:822–828.
20. Pacifico A, Ferlic LL, Cedillo-Salazar FR, Nasir N Jr, Doyle TK, Henry PD. Shocks as predictors of survival in patients with implantable cardioverter-defibrillators. J Am Coll Cardiol 1999;34:204–210.
21. Poole JE, Johnson GW, Hellkamp AS, et al. Prognostic importance of defibrillator shocks in patients with heart failure. N Engl J Med 2008;359:1009–1017.
22. Reddy VY, Reynolds MR, Neuzil P, et al. Prophylactic catheter ablation for the prevention of defibrillator therapy. N Engl J Med 2007;357:2657–2665.
23. Delacretaz E, Brenner R, Schaumann A, et al. Catheter ablation of stable ventricular tachycardia before defibrillator implantation in patients with coronary heart disease (VTACH): an on-treatment analysis. J Cardiovasc Electrophysiol 2013;24:525–529.
24. Mallidi J, Nadkarni GN, Berger RD, Calkins H, Nazarian S. Meta-analysis of catheter ablation as an adjunct to medical therapy for treatment of ventricular tachycardia in patients with structural heart disease. Heart Rhythm 2011;8:503–510.
25. Morrison BT, Bunch TJ, Gersh BJ. Pathophysiology of concomitant atrial fibrillation and heart failure: implications for management. Nat Clin Pract Cardiovasc Med 2009;6:46–56.
26. Mahapatra S, LaPar DJ, Bhamidipati CM, et al. Incidence, risk factors, and consequences of new-onset atrial fibrillation following epicardial ablation for ventricular tachycardia. Europace 2011;13:548–554.