Sudden Cardiac Death in Patients With Ischemic Heart Failure Undergoing Coronary Artery Bypass Grafting

Results From the STICH Randomized Clinical Trial (Surgical Treatment for Ischemic Heart Failure)

BACKGROUND: The risk of sudden cardiac death (SCD) in patients with heart failure after coronary artery bypass graft surgery (CABG) has not been examined in a contemporary clinical trial of surgical revascularization. This analysis describes the incidence, timing, and clinical predictors of SCD after CABG.

METHODS: Patients enrolled in the STICH trial (Surgical Treatment of Ischemic Heart Failure) who underwent CABG with or without surgical ventricular reconstruction were included. We excluded patients with prior implantable cardioverter-defibrillator and those randomized only to medical therapy. The primary outcome was SCD as adjudicated by a blinded committee. A Cox model was used to examine and identify predictors of SCD. The Fine and Gray method was used to estimate the incidence of SCD accounting for the competing risk of other deaths.

RESULTS: Over a median follow-up of 46 months, 113 of 1411 patients who received CABG without (n = 934) or with (n = 477) surgical ventricular reconstruction had SCD; 311 died of other causes. The mean left ventricular ejection fraction at enrollment was 28±9%. The 5-year cumulative incidence of SCD was 8.5%. Patients who had SCD and those who did not die were younger and had fewer comorbid conditions than did those who died of causes other than SCD. In the first 30 days after CABG, SCD (n=5) accounted for 7% of all deaths. The numerically greatest monthly rate of SCD was in the 31- to 90-day time period. In a multivariable analysis including baseline demographics, risk factors, coronary anatomy, and left ventricular function, end-systolic volume index and B-type natriuretic peptide were most strongly associated with SCD.

CONCLUSIONS: The monthly risk of SCD shortly after CABG among patients with a low left ventricular ejection fraction is highest between the first and third months, suggesting that risk stratification for SCD should occur early in the postoperative period, particularly in patients with increased preoperative end-systolic volume index or B-type natriuretic peptide.

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Meena P. Rao, MD, MPH
Sana M. Al-Khatib, MD, MHS
Sean D. Pokorney, MD, MBA
Lilin She, PhD
Alexander Romanov, MD, PhD
Jose C. Nicolau, MD, PhD
Kerry L. Lee, PhD
Peter Carson, MD
Craig H. Selzman, MD
Janina Stepinska, MD
John G.F. Cleland, MD, PhD
Wiwun Tungsubutra, MD
Patrice M. Desvigne-Nickens, MD
Carla A. Sueta, MD
Matthias Siepe, MD
Irene Lang, MD
Arthur M. Feldman, MD
Michael Yii, MD
Jean L. Rouleau, MD
Eric J. Velazquez, MD
On behalf of the STICH Trial Investigators

Correspondence to: Eric J. Velazquez, MD, Duke Clinical Research Institute, PO Box 17969, Durham, NC 27715. E-mail eric.velazquez@duke.edu

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Clinical Perspective

What Is New?

- The timing of sudden cardiac death in patients with heart failure and coronary disease was found to be highest during the first and third months after coronary artery bypass graft surgery.
- In this analysis, several clinical, echocardiographic, and biochemical factors were found to be significantly associated with the risk of sudden cardiac death, particularly preoperative end-systolic volume index and B-type natriuretic peptide.

What Are the Clinical Implications?

- Patients with ischemic cardiomyopathy who undergo coronary artery bypass graft surgery may need to be risk stratified before 90 days after coronary artery bypass graft surgery to help prevent sudden cardiac death.
- Preoperative clinical predictors can help inform early sudden cardiac death risk stratification.

Patients with coronary artery disease and those with left ventricular (LV) dysfunction are thought to be at increased risk for sudden cardiac death (SCD).[1] Although the implantable cardioverter-defibrillator (ICD) reduces SCD in patients with coronary artery disease and a moderately depressed LV ejection fraction (LVEF),[2–5] ICD implantation at the time of coronary artery bypass graft surgery (CABG) did not improve survival in a trial conducted in the 1990s.[1,6] Although surgical revascularization may improve LVEF in a subset of patients,[7] strategies to predict LVEF improvement after CABG are not validated in the context of current guideline-directed medical therapy. Improvement in LVEF after revascularization might render ICDs unnecessary; however, patients who undergo CABG with or without improvement in LVEF may remain at risk for SCD.[8] In addition, there may be a delay between CABG and improvement in LVEF during which patients may be at risk for SCD.

The risk of SCD and how that risk might change over time after CABG are uncertain. Although most randomized clinical trials of primary prevention ICDs required a 90-day waiting period after CABG before implantation of an ICD, the 2008 practice guidelines on device-based rhythm management did not mandate any waiting period after surgical revascularization by CABG.[9] Nonetheless, many major payers, including the Centers for Medicare & Medicaid Services, do not reimburse hospitals and healthcare providers for primary prevention ICDs implanted within 90 days after CABG, and therefore, in the United States, ICD therapy is usually deferred. Therefore, it is important to determine the magnitude of SCD risk in patients with heart failure recovering from CABG.

This analysis describes the incidence, timing, and clinical predictors of SCD after CABG in patients with coronary artery disease and a low LVEF.

METHODS

Study Population

This study includes patients enrolled in the STICH trial (Surgical Treatment of Ischemic Heart Failure) with an LVEF ≤35% who underwent CABG or CABG plus surgical ventricular reconstruction (SVR). The STICH study design and results of the surgical revascularization and SVR components have been previously described.[10–13] Briefly, the study enrolled patients who were candidates for at least 2 of the 3 possible therapeutic options: medical therapy alone, medical therapy plus CABG, or medical therapy plus CABG and SVR. Of the 2136 patients randomized, we excluded patients who were randomized to medical therapy only (n=602), those randomized to CABG but did not receive CABG (n=74), those with ICD or pacemakers for resynchronization before randomization (n=44), and those who received an ICD or pacemaker after randomization but before CABG (n=5), leaving 1411 patients eligible for this analysis. The study complied with the Declaration of Helsinki, and the locally appointed ethics committee approved the research protocol. Informed consent was obtained from the subjects or their legally authorized representatives.

Study Outcomes

A blinded committee adjudicated all deaths using prespecified definitions of causes of death. SCD was defined by this committee as death that occurred suddenly and unexpectedly and was judged to be due to a cardiovascular cause as previously described.[14]

Statistical Analysis

Baseline or preoperative characteristics of patients are presented as means and standard deviations for continuous variables and as frequencies and percentages for categorical variables. Data are presented for 3 groups of patients: patients who had SCD, patients who died of causes other than SCD, and patients who did not die by the end of the study follow-up. Group comparisons with respect to baseline clinical characteristics were performed with the Kruskal-Wallis test for continuous variables and the conventional χ^2 test for categorical variables.

Cox proportional hazard models were used to examine individual relationships of candidate clinical characteristics and SCD, censoring other deaths at the time of death.[15] Clinical characteristics included in the model were baseline patient demographics, medical history, prior cardiac procedures, presenting characteristics of heart failure (New York Heart Association heart failure class), Canadian Cardiovascular Society angina, baseline laboratory measures, baseline medications, coronary anatomy, LV function measures and volumes, 6-minute walk distance, and surgical details. Candidate variables also include biomarkers (such as BNP [B-type natriuretic peptide] and tumor necrosis factor receptor 1) and echocardiographic diastolic function variables such as E/A ratio, e velocity, deceleration time, and right ventricular dysfunction. Of note, LVEF,

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end-systolic volume index (ESVI), and other echocardiographic variables as above were independently measured in a blinded fashion by STICH core laboratories as previously described.[16]

Only a few of the included variables had >2% missing data. The magnitude of missing data for the biomarkers and diastolic function parameters was high at 15% to 35%; this was addressed by the multiple imputation method, in which 25 multiply imputed data sets (with missing values imputed) were created. Imputed data sets were used in the univariable and multivariable model analyses, in which the results from the 25 data sets were appropriately combined. Variables with a significant (P<0.05) or marginally significant (P<0.10) relationship with SCD in the univariable models were included in the multivariable models. Backward elimination was used to determine factors that were statistically significant (P<0.05) in a multivariable model for SCD.

Given the competing risk of SCD and other modes of death, cumulative incidence rates for SCD and other death were estimated with the Fine and Gray method when SCD and other death rates were reported.[17] The conditional SCD rates per month for different time intervals during follow-up were calculated from the cumulative incidence rate of SCD. For each time interval, the SCD rate per month is based on the difference between the cumulative incidence SCD rates at the end and the beginning of the time interval divided by the number of months for the time interval, under the condition that the patients survived to the beginning of the time interval. Confidence intervals for the SCD rates per month were obtained with bootstrapping methods.

SAS statistical software (version 9.2 or higher) was used in all analyses (SAS Institute, Cary, NC). All statistical tests were 2 sided and performed at a level of significance of α=0.05.

RESULTS

Of the 1411 patients included in this analysis, 934 patients received CABG, and 477 received CABG plus SVR. During the median follow-up period of 46 months, 234 patients (16.6%) received an ICD. There were 113 (8.0%) and 311 (22.0%) patients who died of SCD and other causes of death, respectively. Baseline or preoperative characteristics comparing patients who died of SCD, had other causes of death, and did not die are shown in Table 1. Patients who had SCD were younger and had fewer comorbid conditions and lower New York Heart Association heart failure class than those who died of other causes. Patients who died of SCD had higher diastolic blood pressure and used more digoxin. Diuretics were used more frequently in all patients who died compared with those who lived. Patients who died of causes other than SCD were more likely to have 3-vessel disease compared with patients who died suddenly. Although LVEF was similar in patients who died of SCD and those who died of other causes, ESVI was higher in patients who died suddenly.

Cumulative incidence rates of SCD and other death over the 5-year follow-up period after CABG are shown in Table 2. The 1-, 3-, and 5-year cumulative incidence of SCD after CABG in this patient population with low LVEF is 2.8%, 6.1%, and 8.5%, respectively. The conditional risk of SCD per month over different time intervals after revascularization is shown in Figure 1. In the first 30 days after CABG, the risk of SCD was 0.35% (95% confidence interval, 0.15–0.85). The risk of SCD per month was numerically highest during the 31- to 90-day window (0.43%; 95% confidence interval, 0.21–0.67), although it was only slightly higher than the risk during the first month. After 6 months, the risk per month decreased to 0.14% (95% confidence interval, 0.07–0.24) and remained relatively stable thereafter.

The results of the univariable analyses of the relationship between baseline characteristics and SCD are shown in Table 3. Variables that have significant (P<0.05) or marginally significant (P<0.10) association with SCD in the univariable models are listed in Table 3 by the magnitude of their univariable χ^2 statistics. Of all the variables studied, baseline BNP (χ^2=16.85, P<0.001) and ESVI (χ^2=16.53, P<0.001) are the most strongly associated with SCD. Other factors found to be associated with increased SCD risk included renal function, LVEF, right ventricular dysfunction, diuretic use, coronary artery disease index, history of atrial fibrillation/flutter, mitral regurgitation, stroke, and intra-aortic balloon pump use. Statin use, hyperlipidemia, sodium level, and having CABG plus SVR were found to be associated with lower SCD.

Variables found to be independently and significantly associated with SCD in the multivariable model are listed in Table 4. In the final multivariable model, ESVI was the strongest factor associated with SCD, followed closely by BNP. Other factors associated with SCD are statin use, Duke Coronary Artery Disease Index, baseline sodium level, history of atrial fibrillation, and having received CABG plus SVR surgery. Figure 2 shows that as the ESVI increases, the 5-year risk of SCD also increases (12.4% versus 5.7% for those in the fourth versus first quartile of ESVI). Higher BNP value, high Duke Coronary Artery Disease Index, lower sodium level, and history of atrial fibrillation were also associated with increased risk of SCD, whereas statin use and having received CABG with SVR appeared to be associated with a lower risk of SCD. None of the factors in the final multivariable model were highly discriminatory between SCD and death resulting from other causes. The c index for the final selected multivariable model for SCD was 0.70.

DISCUSSION

This article has 3 main findings. First, in patients with a depressed LVEF and coronary artery disease undergoing CABG with or without SVR, the 5-year cumulative incidence of SCD was 8.5%. Second, the monthly risk of SCD was greatest between the first and third months af-

Table 1. Baseline Characteristics of Patients Who, After Coronary Artery Bypass Graft Surgery With or Without Surgical Ventricular Reconstruction, Underwent Sudden Cardiac Death Compared With Those Who Died of Other Causes and Those Who Did Not Die

Characteristics*	All Patients (N=1411)	SCD (n=113)	Other Deaths (n=311)	No Death (n=987)	*P* Value
Age, y	61±10	61±10	65±9	60±9	<0.001
Male	1212 (86)	99 (88)	268 (86)	845 (86)	0.836
White	1153 (82)	93 (82)	270 (87)	790 (80)	0.026
Medical history					
Stroke	94 (7)	11 (10)	35 (11)	48 (5)	<0.001
Diabetes mellitus	518 (37)	44 (38)	140 (45)	335 (34)	0.002
Hypertension	839 (59)	61 (54)	197 (63)	581 (59)	0.174
Hyperlipidemia	947 (67)	65 (58)	199 (64)	683 (69)	0.017
Peripheral vascular disease	208 (15)	20 (18)	64 (21)	124 (13)	0.002
Chronic kidney disease	108 (8)	15 (13)	45 (15)	48 (5)	<0.001
Atrial flutter/fibrillation	161 (11)	18 (16)	59 (19)	84 (9)	<0.001
Prior CABG	36 (3)	2 (2)	16 (5)	18 (2)	0.005
NYHA heart failure class					<0.001
I	132 (9)	9 (8)	22 (7)	101 (10)	
II	655 (46)	57 (50)	124 (40)	474 (48)	
III	560 (40)	41 (36)	134 (43)	385 (39)	
IV	64 (5)	6 (5)	31 (10)	27 (3)	
Diastolic blood pressure	74±11	75±10	73±12	75±11	0.052
3-Vessel coronary disease	909 (64)	70 (62)	218 (70)	621 (63)	0.060
LVEF, %	28±9	26±9	26±8	29±9	<0.001
ESVI	83±32	93±37	89±35	79±31	<0.001
EDVI	116±40	127±42	124±44	112±38	<0.001
BNP	442±501	622±609	568±664	381±403	<0.001
Mitral regurgitation					<0.001
None or trace	513 (37)	36 (32)	90 (29)	387 (39)	
Mild (≤2+)	655 (47)	51 (46)	153 (50)	451 (46)	
Moderate (3+)	188 (13)	19 (17)	46 (15)	123 (13)	
Severe (4+)	46 (3)	6 (5)	20 (7)	20 (2)	
Medications					
β-Blocker	1202 (85)	96 (85)	247 (79)	859 (87)	0.004
Digoxin	230 (16)	25 (22)	61 (20)	144 (15)	0.024
ACE-I/ARB	1249 (89)	95 (84)	277 (89)	877 (89)	0.301
Antiarrhythmic drug use	165 (12)	17 (15)	48 (15)	100 (10)	0.021
Statin	1090 (77)	78 (69)	228 (73)	784 (79)	0.008
Diuretic (loop/thiazide)	861 (61)	84 (74)	224 (72)	553 (56)	<0.001
Diuretic (potassium sparing)	566 (40)	45 (40)	154 (50)	367 (37)	<0.001

ACE-I indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BNP, B-type natriuretic peptide; CABG, coronary artery bypass graft; EDVI, end-diastolic volume index; ESVI, end-systolic volume index; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; and SCD, sudden cardiac death.

*Continuous variables are presented as mean +/- standard deviation. Categorical variables are presented as frequency (%).

Table 2. Cumulative Number of Events and Cumulative Incidence Rate of Sudden Cardiac Death and Other Death for Different Time Points After Surgery Among 1411 Patients

Time After Surgery	SCDs		Other Deaths	
	Cumulative Deaths, n	Cumulative Incidence Rate, % (95% CI)	Cumulative Deaths, n	Cumulative Incidence Rate, % (95% CI)
30 d	5	0.4 (0.2–0.9)	65	4.6 (3.7–5.7)
90 d	17	1.2 (0.7–2.0)	90	6.4 (5.2–7.7)
6 mo	28	2.0 (1.4–2.8)	114	8.1 (6.8–9.6)
1 y	40	2.8 (2.1–3.8)	148	10.5 (9.1–12.0)
3 y	86	6.1 (4.9–7.6)	231	16.4 (14.6–18.5)
5 y*	107	8.5 (7.2–10.1)	300	25.3 (22.9–28.0)

CI indicates confidence interval; and SCD, sudden cardiac death.
*Six SCDs and 11 other deaths happened during the follow-up period that were >5 years after the surgery.

ter CABG and decreased subsequently, remaining nearly constant after 6 months. Third, we identified several factors that are significantly associated with the risk of SCD, including baseline ESVI, BNP, statin use, the Duke coronary artery disease index, serum sodium concentration, history of atrial fibrillation/flutter, and having received CABG with SVR. Of these variables, baseline ESVI and BNP were most strongly associated with SCD. Although baseline or preoperative ESVI was also associated with death resulting from other causes, it was more strongly associated with SCD than LVEF and therefore may be useful for determining which post-CABG patients might benefit from early interventions to reduce the risk of SCD.

In the STICH trial, CABG with medical therapy compared with medical therapy alone significantly reduced the risk of SCD, pump failure death, and fatal MI death, with the protective effect of CABG being most prominent beyond 2 years after CABG.[11,13,14] This analysis shows the monthly risk of SCD to be greatest between the first and third months after CABG. Although overall SCD numbers are a low proportion of overall deaths, this observation is important because it challenges the current practice in the United States of postponing risk stratification for SCD and supports the pursuit of identifying those patients at high risk for SCD early after CABG.

The best method for risk stratifying post-CABG patients for SCD is yet to be determined; however, data from this analysis may help inform this process. We identified several clinical, echocardiographic, and blood test factors that are significantly associated with SCD. Unique to our model is the incorporation of echocardiographic data, biomarker data, and medications. It is interesting to note that β-blocker use was not significantly associated with SCD, presumably as a result of the very

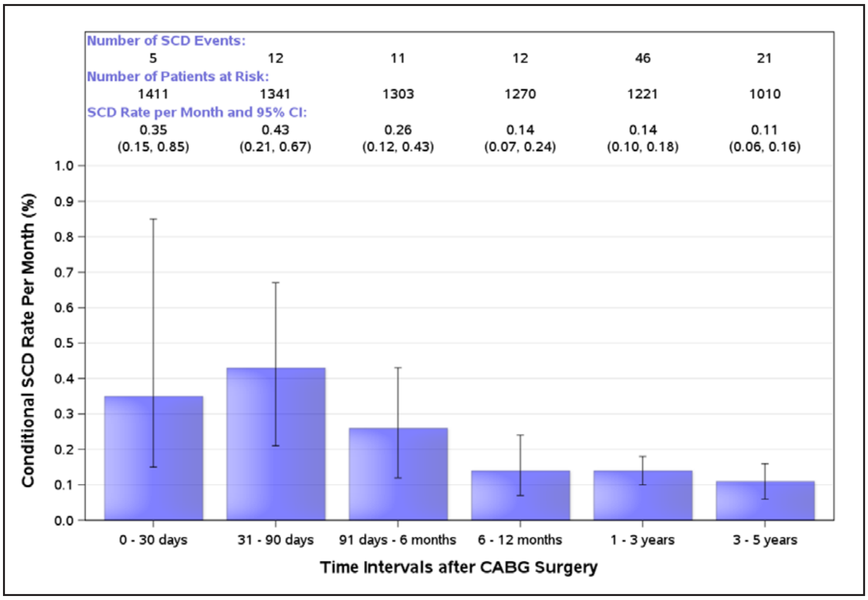

Figure 1. Conditional risk of sudden cardiac death (SCD) per month for different time intervals after coronary artery bypass graft surgery (CABG). CI indicates confidence interval.

Table 3. Baseline Variables Predicting Sudden Cardiac Death in Univariate Analyses

Variable	Wald χ^2	HR	95% CI	*P* Value
BNP in 100-pg/mL increments*	16.85	1.21	1.10–1.33	<0.001
ESVI in 20-mL/m^2 increments	16.53	1.22	1.11–1.35	<0.001
Creatinine in mg/dL	13.02	1.58	1.23–2.03	<0.001
LVEF in 5% increments	10.75	0.83	0.74–0.93	0.001
Duke CAD index in 10-point increments	10.62	1.15	1.06–1.26	0.001
Chronic renal insufficiency	9.63	2.37	1.37–4.08	0.002
TNF receptor in 200-pg/mL increments*	9.54			0.009
1000–1800		1.16	1.00–1.34	
>1800 in 200		1.02	1.00–1.04	
Diuretics (loop/thiazide or potassium sparing)	8.34	2.01	1.25–3.23	0.004
Statin	6.66	0.59	0.40–0.88	0.010
RV dysfunction*	6.22	1.33	1.06–1.65	0.013
New-onset ventricular arrhythmia	6.15	5.97	1.45–24.53	0.013
Hyperlipidemia	5.91	0.63	0.43–0.91	0.015
Atrial flutter/fibrillation	5.60	1.84	1.11–3.04	0.018
Mitral regurgitation*	5.31	1.31	1.04–1.64	0.021
E/A ratio overall in 0.1 increments*	5.20			0.074
1.6–2.0		1.19	1.03–1.39	
>2.0		0.96	0.90–1.02	
Sodium in mEq/L	4.40	0.95	0.91–1.00	0.036
E velocity in m/s*	4.29	2.29	1.04–5.04	0.039
Deceleration time in 10-ms increments*	4.06	0.91	0.84–1.00	0.044
Stroke	3.53	1.82	0.97–3.38	0.060
Received mitral valve procedure	3.46	1.54	0.98–2.44	0.063
Received CABG+SVR	3.43	0.66	0.42–1.02	0.064
IABP for low cardiac output	3.03	1.97	0.92–4.24	0.082

BNP indicates B-type natriuretic peptide; CABG, coronary artery bypass graft; CAD, coronary artery disease; CI, confidence interval; ESVI, end-systolic volume index; HR, hazard ratio; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; RV, right ventricle; SVR, surgical ventricular reconstruction; and TNF, tumor necrosis factor.

*Variables with ≥10 or more patients with missing data. The number and percentage of patients with missing data are as follows: BNP, 493 (34.9%); TNF receptor 1, 493 (34.9%); deceleration time, 434 (30.8%); E/A ratio, 402 (28.5%); E velocity, 337 (23.9%); and RV dysfunction, 209 (14.8%).

high rates of use in this patient population. The observation in this population that baseline ESVI appeared to be more strongly associated with SCD than LVEF is interesting and probably due in part to the universally depressed baseline LVEF mandated by protocol in this cohort. Our analysis did not incorporate follow-up postoperative LVEF data including at the time of the SCD event, so any conclusions about relationships between post-CABG LVEF and SCD would be largely speculative.[7] Future studies should focus on the relationship between follow-up LVEF and risk of SCD after CABG and should explore the role of other echocardiographic parameters and other imaging modalities in the risk stratification of post-CABG patients for SCD.

Even if accurate risk stratification of post-CABG patients for SCD is achievable, there is still great uncertainty as to how one would reduce this risk shortly after CABG. The CABG-Patch trial showed no benefit from an ICD implanted at the time of CABG.[1] It may be that ICD implantation at the time of CABG is beneficial only in high-risk patients. It is also possible that the wearable defibrillator is a beneficial strategy early on with a plan to pursue an ICD if the LVEF does not improve to >35% at >90 days after CABG.[18] The advantage of the wearable defibrillator is that it is noninvasive and does not commit patients to a lifelong therapy like the ICD, especially if their LVEF becomes normal or nearly normal. Although data on the efficacy of the wearable

Table 4. Baseline Variables Predictive of Sudden Cardiac Death in a Multivariable Model

Variable	Wald χ^2	HR	95% CI	*P* Value
ESVI in 20-mL/m² increments	9.59	1.18	1.06–1.31	0.002
BNP in 100-pg/mL increments up to 750	9.51	1.16	1.06–1.28	0.002
Statin use	7.52	0.57	0.38–0.85	0.006
Duke CAD index in 10-point increments	5.71	1.11	1.02–1.22	0.017
Sodium in mEq/L	5.21	0.95	0.90–0.99	0.023
History of atrial fibrillation/flutter	5.01	1.79	1.08–2.98	0.025
Received CABG+SVR	4.02	0.64	0.41–0.99	0.045

BNP indicates B-type natriuretic peptide; CABG, coronary artery bypass graft; CAD, coronary artery disease; CI, confidence interval; ESVI, end-systolic volume index; HR, hazard ratio; and SVR, surgical ventricular reconstruction.

defibrillator in this setting are lacking, a science advisory from the American Heart Association endorsed by the Heart Rhythm Society included a Class IIa recommendation on the use of wearable defibrillators after revascularization.[19]

As a post hoc analysis, our observations must be interpreted with caution; however, this is to date the most comprehensive analysis of SCD in a post-CABG patient population. Patients enrolled in STICH may not be representative of patients seen in routine clinical practice. This analysis includes patients who received CABG and CABG plus SVR, although the vast majority of patients in clinical practice receive CABG only. We pursued this approach because the overall mortality in these 2 groups was not significantly different, with a hazard ratio for all-cause mortality of 1.0 among those randomized to CABG or CABG plus SVR.[12] Although the rates of SCD were higher among those who received CABG than those patients who underwent CABG plus SVR, the increase in SCD was offset by a corresponding and equalizing increase in non-SCD (Table 5). Whether SVR partially mitigates SCD risk relative to CABG is consistent with the construct posited in prior reports that exclusion of LV scar may diminish the burden of ventricular tachycardia as a substrate for SCD but is of unclear clinical impact in the era of ICD availability and the results of the STICH SVR study.[12,20] By affecting ESVI[21] and hemodynamics, SVR may have an antiarrhythmic effect; alternatively, SVR may result in scar tissue at the surgical incisions, creating a potentially arrhythmogenic substrate. In STICH, the patients receiving SVR were not followed up long term; therefore, we looked at SCD in a 5-year window as opposed to a 10-year window. Overall arrhythmic deaths in this analysis are a small proportion of all deaths, and although SCD was centrally adjudicated with prespecified and well-accepted criteria, not all SCD events could be attributed to a ventricular arrhythmia, and such granular data were not available. Unfortunately, we did not have data on ICD shocks or aborted sudden cardiac arrest during follow-up. Last, our model included some baseline

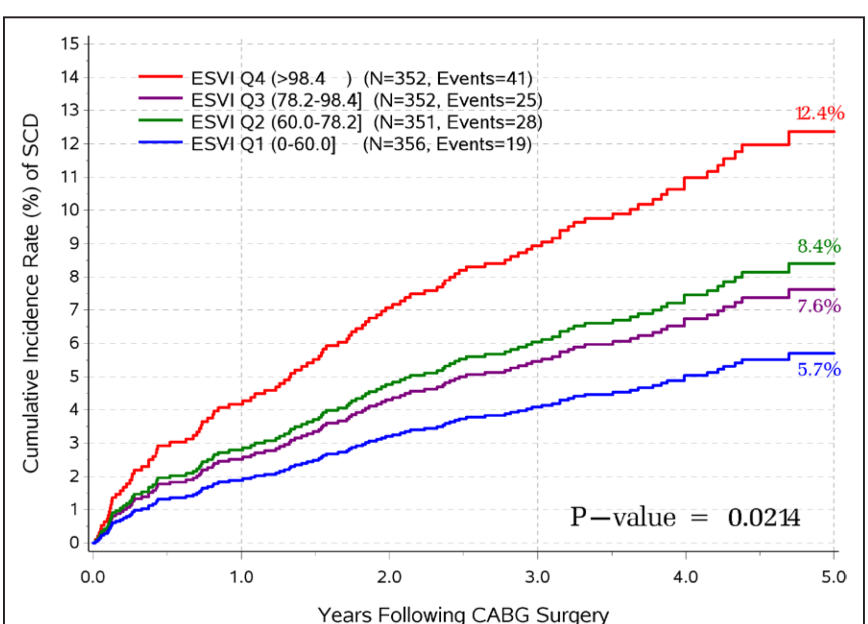

Figure 2. Cumulative incidence rates of sudden cardiac death (SCD) after coronary artery bypass grafting (CABG) by end-systolic volume index (ESVI) quartiles.
Q indicates quartile.

Table 5. Cumulative Incidence Rate of Sudden Cardiac Death and Other Death for Different Time Points After Surgery, by Treatment Received

Time After Surgery	Cumulative Incidence Rate, % (95% CI)			
	SCD		Other Death	
	CABG (n=934)	CABG+SVR (n=477)	CABG (n=934)	CABG+SVR (n=477)
30 d	0.4 (0.2–1.0)	0.3 (0.1–0.6)	4.4 (3.5–5.6)	5.0 (3.9–6.4)
90 d	1.4 (0.8–2.2)	0.9 (0.5–1.6)	6.1 (5.0–7.4)	6.9 (5.3–9.0)
6 mo	2.3 (1.6–3.3)	1.4 (0.9–2.4)	7.7 (6.4–9.4)	8.7 (6.7–11.3)
1 y	3.2 (2.4–4.4)	2.0 (1.3–3.1)	10.1 (8.6–11.8)	11.3 (9.2–14.0)
3 y	7.0 (5.5–8.8)	4.4 (3.0–6.6)	15.8 (13.9–17.9)	17.7 (14.8– 21.1)
5 y	9.6 (7.8–11.8)	6.1 (4.1–9.1)	24.4 (21.6–27.6)	27.2 (23.1–32.0)

CABG indicates coronary artery bypass graft; CI, confidence interval; SCD, sudden cardiac death; and SVR, surgical ventricular reconstruction.

characteristics that might be affected by CABG, and our analysis does not account for changes that may have resulted from CABG.

CONCLUSIONS

Among patients with a low LVEF undergoing CABG with or without SVR, the overall risk of SCD was low relative to all deaths. The risk of SCD peaked during the first and third months after CABG and subsequently declined and remained stable after 6 months. This observation suggests that patients with ischemic cardiomyopathy who undergo CABG may need to be risk stratified and potentially treated to prevent SCD before 90 days after CABG. Several preoperative clinical, echocardiographic, and biochemical factors are significantly associated with the risk of SCD that could inform SCD risk stratification. Future studies should examine the relationship between follow-up LV characteristics and risk of SCD after CABG and define the best strategies for preventing SCD early after CABG.

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AFFILIATIONS

From Duke Clinical Research Institute (M.P.R., S.M.A.-K., S.D.P., L.S., K.L.L., E.J.V.) and Departments of Biostatistics and Bioinformatics (K.L.L.) and Medicine (S.M.A.-K., E.J.V.), Duke University School of Medicine, Durham, NC; State Research Institute of Circulation Pathology, Novosibirsk, Russia (A.R.); Heart Institute, University of São Paulo Medical School, Brazil (J.C.N.); Washington DC Veterans Affairs Medical Center (P.C.); Division of Cardiothoracic Surgery, University of Utah, Salt Lake City (C.H.S.); Institute of Cardiology, Warsaw, Poland (J.S.); National Heart and Lung Institute, Royal Brompton & Harefield Hospitals, Imperial College, London, UK (J.G.F.C.); Siriraj Hospital, Mahidol University, Bangkok, Thailand (W.T.); Division of Cardiovascular Sciences, National Heart, Lung, and Blood Institute, Bethesda, MD (P.M.D.-N.); University of North Carolina at Chapel Hill (C.A.S.); University Heart Center Freiburg-Bad Krozingen, Germany (M.S.); Medical University of Vienna, Austria (I.L.); Department of Medicine, Temple University School of Medicine, Philadelphia, PA (A.M.F.); St. Vincent's Hospital, University of Melbourne, Fitzroy, Australia (M.Y.); and Department of Medicine, Montreal Heart Institute, University of Montreal, Quebec, Canada (J.L.R.).

FOOTNOTES

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Meena P. Rao, Sana M. Al-Khatib, Sean D. Pokorney, Lilin She, Alexander Romanov, Jose C. Nicolau, Kerry L. Lee, Peter Carson, Craig H. Selzman, Janina Stepinska, John G.F. Cleland, Wiwun Tungsubutra, Patrice M. Desvigne-Nickens, Carla A. Sueta, Matthias Siepe, Irene Lang, Arthur M. Feldman, Michael Yii, Jean L. Rouleau and Eric J. Velazquez
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