

## Original Investigation

# Sudden Death in Patients With Cardiac Implantable Electronic Devices

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**IMPORTANCE** Interrogations and autopsies of sudden deaths with cardiac implantable electronic devices (CIEDs) are rarely performed. Therefore, causes of sudden deaths with these devices and the incidence of device failure are unknown.

**OBJECTIVE** To determine causes of death in individuals with CIEDs in a prospective autopsy study of all sudden deaths over 35 months as part of the San Francisco, California, Postmortem Systematic Investigation of Sudden Cardiac Death (POST SCD) study.

**DESIGN, SETTING, AND PARTICIPANTS** Full autopsy, toxicology, histology, and device interrogation were performed on incident sudden cardiac deaths with pacemakers or implantable cardioverter defibrillators (ICDs). The setting was the Office of the Chief Medical Examiner, City and County of San Francisco. Participants included all sudden deaths captured through active surveillance of all deaths reported to the medical examiner and San Francisco residents with an ICD (January 1, 2011, to November 30, 2013).

**MAIN OUTCOMES AND MEASURES** Identification of a device concern in sudden deaths with CIEDs, including hardware failures, device algorithm issues, device programming issues, and improper device selection. For the ICD population, outcomes were the cumulative incidence of death and sudden cardiac death and the proportion of deaths with an ICD concern.

**RESULTS** Twenty-two of 517 sudden deaths (4.3%) had CIEDs, and autopsy revealed a noncardiac cause of death in 6. Six of 14 pacemaker sudden deaths and 7 of 8 ICD sudden deaths died of ventricular tachycardia or ventricular fibrillation. Device concerns were identified in half (4 pacemakers and 7 ICDs), including 3 hardware failures contributing directly to death (1 rapid battery depletion with a sudden drop in pacing output and 2 lead fractures), 5 ICDs with ventricular fibrillation undersensing, 1 ICD with ventricular tachycardia missed due to programming, 1 improper device selection, and a pacemaker-dependent patient with pneumonia and concern about lead fracture. Of 712 San Francisco residents with an ICD during the study period, 109 died (15.3% cumulative 35-month incidence of death), and the 7 ICD concerns represent 6.4% of all ICD deaths.

**CONCLUSIONS AND RELEVANCE** Systematic interrogation and autopsy of sudden deaths in one city identified concerns about CIED function that might otherwise not have been observed. Current passive surveillance efforts may underestimate device malfunction. These methods can provide unbiased data regarding causes of sudden death in individuals with CIEDs and improve surveillance for CIED problems.

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More than 3 million people in the United States have a permanent pacemaker (PPM) or implantable cardioverter defibrillator (ICD).<sup>1</sup> These devices have saved the lives of innumerable patients and have improved the lives of many more. Currently, surveillance for cardiac implantable electronic device (CIED) malfunctions is based on the US Food and Drug Administration–mandated Manufacturer and User Facility Device Experience (MAUDE) database,<sup>2</sup> which is mandatory for manufacturers but voluntary for health care professionals. Food and Drug Administration adverse event reporting regulations require that manufacturers and facilities such as hospitals report when they learn that a device may have caused or contributed to a death or serious injury (21 CFR §803). Events in deceased patients are not excluded; however, because more than 90% of sudden cardiac deaths (SCDs) occur outside of the hospital<sup>3</sup> and because investigation after such sudden deaths is not a routine medical examiner or coroner practice, interrogations and autopsies of SCDs with pacemakers and ICDs are rarely performed.<sup>4</sup> Deaths in those with CIEDs that occur outside of care facilities or that are unknown to the manufacturer are at particularly high risk to be uninvestigated or unreported to the Food and Drug Administration MAUDE database, and rates of CIED failure that lead to sudden death are unknown. We sought to determine causes of SCDs with CIEDs in an ongoing prospective autopsy study of all incident SCDs in San Francisco, California.

## Methods

### Medical Examiner Evaluation

From January 1, 2011, to November 30, 2013, as part of the final month of the pilot study (January 1 to January 31, 2011) and the first 34 months of the San Francisco Postmortem Systematic Investigation of Sudden Cardiac Death (POST SCD) study (February 1, 2011, to November 30, 2013), autopsies were performed on 499 of 517 (96.5%) of all incident SCDs captured through active surveillance of out-of-hospital deaths, all of which are reported to the San Francisco medical examiner as mandated by state law. The 18 deaths that did not undergo full autopsy underwent external examination to exclude the presence of CIEDs. Sudden cardiac deaths were defined by World Health Organization criteria,<sup>5</sup> including sudden unexpected death within 1 hour of acute symptom onset (if witnessed) or within 24 hours of the last observation at baseline (if unwitnessed). Deaths in individuals with a known history of noncardiac chronic terminal illness (eg, terminal cancer) or an identifiable noncardiac etiology (eg, obvious recent drug use on the scene) were excluded. Sudden cardiac deaths in individuals younger than 18 years were also excluded.<sup>6</sup> We included all SCDs initially identified by the medical examiner as having an implanted PPM or ICD.

Evaluation for all cases included full autopsy, toxicology, histology with hematoxylin-eosin and trichrome staining, and detailed examination of the heart and cranial vault. Premortem medical records, medical examiner investigator reports, and emergency medical system documentation from the date of death were obtained. Device interrogation

was performed on SCDs with a PPM or an ICD. Terminal rhythm was determined from device interrogation, correlating events stored on the device with the precise timing of the arrest from emergency medical system documentation for witnessed deaths. A multidisciplinary committee consisting of 2 pathologists (P.C.U. and E.M.), 2 electrophysiologists (Z.H.T. and J.E.O.), and 1 neurologist reviewed all available data to adjudicate a final cause of death. *Sudden arrhythmic death* was defined as an SCD that met World Health Organization criteria and for which no obvious nonarrhythmic cause of death (eg, pericardial tamponade or myocardial rupture) was found.<sup>6</sup> Sudden deaths in individuals with CIEDs were further evaluated by an electrophysiologist (Z.H.T.), a cardiologist (R.M.H.), and a device technician (C.G.M.) to determine whether a device concern was present. Device concerns were subdivided into hardware failures, device algorithm issues, device programming issues, and improper device selection, which was adjudicated when a patient with an indication for an ICD based on published guidelines<sup>7</sup> died of documented ventricular tachycardia (VT) or ventricular fibrillation (VF) with a pacemaker in place.

### San Francisco ICD Population

As part of a separate ICD-only study, we identified all adult patients residing in San Francisco from the 5 cardiology practices that follow up patients with ICDs (California Pacific Medical Center Electrophysiology, Golden Gate Cardiology, Kaiser San Francisco, San Francisco Veterans Affairs Medical Center, and University of California, San Francisco) between January 1, 2011, and November 30, 2013. Death was determined by review of clinical records and by a death index search for those lost to follow-up. Sudden cardiac deaths were identified via the POST SCD study. We calculated the cumulative incidence of death, the cumulative incidence of SCD, and the proportion of deaths with an ICD concern.

The study was approved by the Institutional Review Board of the University of California, San Francisco. The ICD-only study was approved by the ethics boards of all 5 contributing cardiology practices. The research was carried out with a waiver of informed consent obtained from the ethics boards of the contributing cardiology practices to use deidentified data.

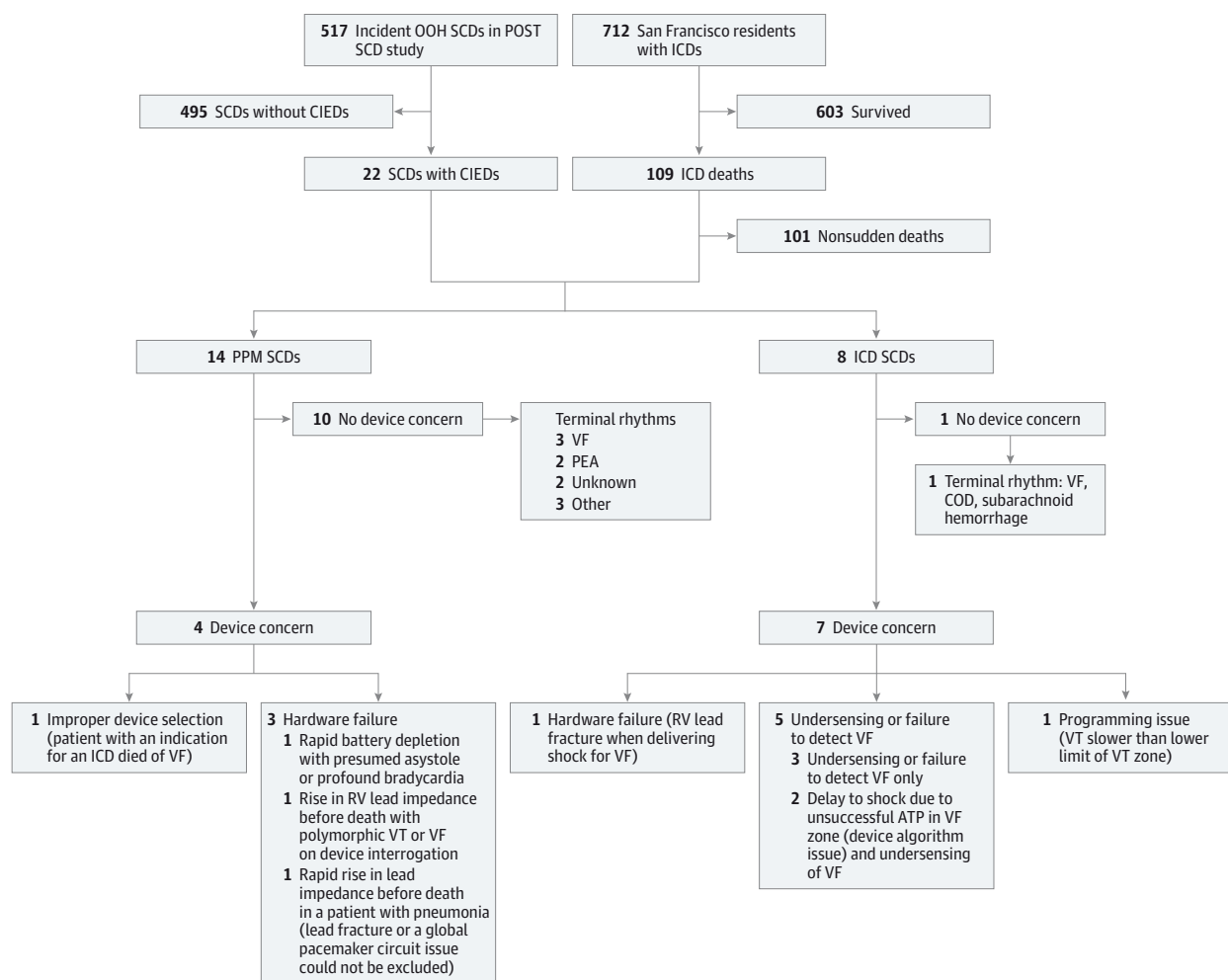
### Statistical Analysis

Continuous variables are presented as means (SDs), and categorical variables are presented as totals with percentages. Characteristics of SCDs with and without CIEDs were compared by unpaired *t* test with unequal variances for continuous variables and by Fisher exact test for categorical variables. Two-tailed *P* < .05 was considered statistically significant. Statistical analyses were performed using a software program (STATA SE, version 13.1; StataCorp LP).

## Results

Of 517 total incident SCDs in the study period, we identified 22 SCDs in individuals with CIEDs (Figure 1). Therefore, 4.3% of SCDs in San Francisco over a 35-month period occurred with

**Figure 1. Flowchart for Postmortem Investigation of Sudden Deaths With Cardiac Implantable Electronic Devices (CIEDs) in the First 35 Months of the Postmortem Systematic Investigation of Sudden Cardiac Death (POST SCD) Study and Deaths in the San Francisco Implantable Cardioverter Defibrillator (ICD) Population, 2011 to 2013**



Device concerns were identified after systematic autopsy and device interrogation of 22 consecutive sudden cardiac deaths (SCDs) with CIEDs via active medical examiner surveillance from January 1, 2011, to January 31, 2011 (pilot study), and from February 1, 2011, to November 30, 2013 (POST SCD study). Deaths in the San Francisco ICD population were from January 1, 2011, to

November 30, 2013. ATP indicates antitachycardia pacing; COD, cause of death; OOH, out of hospital; PEA, pulseless electrical activity; PPM, permanent pacemaker; RV, right ventricle; VF, ventricular fibrillation; and VT, ventricular tachycardia.

a PPM or an ICD. Individuals with devices were older than individuals without devices at the time of SCD (Table). Fourteen had PPMs, and one of these devices was a biventricular PPM (eTable in the Supplement). Eight individuals (6 with ischemic cardiomyopathy and 2 with dilated cardiomyopathy) had ICDs, and 2 of these devices were cardiac resynchronization therapy defibrillator devices.

Full autopsy, toxicology, and histology were performed on 19 individuals, device interrogation was performed for 21 individuals, and comprehensive review of medical records was performed in all 22 individuals. Device interrogation occurred at a median of 3.2 days (range, 13.5 hours to 23 days) after the time of emergency medical system dispatch. On toxicology screening, no lethal levels of any drugs were found in any of the deceased with CIEDs. Autopsy revealed a

noncardiac cause of sudden death in 6 individuals, including intracranial hemorrhage (2 cases), pneumonia (2 cases), massive pulmonary hemorrhage (eFigure 1 in the Supplement),<sup>8</sup> and blunt force trauma to the head and neck (eTable in the Supplement).

After full investigation, 8 of 14 SCDs with PPMs were found to have died of arrhythmic causes, including 6 VFs (cases 2, 4, 8, 9, 13, and 14), 1 presumed profound bradyarrhythmia or asystole (case 1), and 1 unknown rhythm (case 10). Device concerns were identified in 4 PPM individuals. One PPM-dependent individual (case 1 with hardware failure in eFigure 2 in the Supplement) had rapid battery depletion, resulting in clinically significant reduction in pacing output just before sudden death. One individual (case 2 with improper device selection in eFigure 3 in the Supplement)

**Table. Characteristics of Cardiac Implantable Electronic Device Sudden Cardiac Deaths (CIED SCDs), Non-CIED SCDs, and the San Francisco Implantable Cardioverter Defibrillator (ICD) Population, January 1, 2011, to November 30, 2013**

Variable	CIED SCDs (n = 22)	Non-CIED SCDs (n = 495)	P Value <sup>a</sup>	San Francisco ICD Population (n = 712)	P Value <sup>a</sup>
Age, mean (SD) [range], y	72 (16) [26-98]	63 (14) [18-92]	.004	69.8 (15) [22-103]	.50
Male sex, No. (%)	17 (77.3)	340 (68.7)	.63	524 (73.6)	.81
Race/ethnicity, No. (%)			.61		NA
White	15 (68.2)	269 (54.3)		357 (50.1)	
Black	1 (4.5)	74 (14.9)		92 (12.9)	
Asian	5 (22.7)	120 (24.2)		151 (21.2)	
Hispanic	1 (4.5)	25 (5.1)		38 (5.3)	
Other	0	7 (1.4)		29 (4.1)	
Unknown	0	0		45 (6.3)	
Coronary artery disease, No. (%)	10 (45.5)	93 (18.8)	.005	337 (47.3)	>.99
Congestive heart failure, No. (%)	10 (45.5)	62 (12.5)	<.001	372 (52.2)	.67
Diabetes mellitus, No. (%)	1 (4.5)	109 (22.0)	.06	142 (19.9)	.10
Hypertension, No. (%)	10 (45.5)	75 (15.2)	.001	319 (44.8)	.83

Abbreviation: NA, not applicable.

<sup>a</sup> P value for comparison with CIED SCDs by t test.

with known hypertrophic cardiomyopathy, a septal thickness of 3.0 cm by transthoracic echocardiography, a history of syncope, and PPM implantation for heart block died of VF. Another patient (case 3 with possible hardware failure in eFigure 4 in the [Supplement](#)) with advanced atrioventricular block had a rapid rise in lead impedances from 650 to 1620  $\Omega$  in the ventricular lead in the week before death. Atrial lead impedance also increased, raising the possibility of changes due to acidosis or electrolyte disturbances, but atrial lead impedance had normalized on the day of death. On autopsy, pneumonia was present, and cause of death was adjudicated as pneumonia more likely than hardware failure, such as lead fracture or a global pacemaker circuit issue. A PPM-dependent patient (case 4 with hardware failure in eFigure 5 in the [Supplement](#)) had a rise in ventricular lead impedance 2 days before death and documented polymorphic VT or VF on device interrogation. Autopsy revealed cardiomegaly, single-vessel coronary artery disease, and severe interstitial and perivascular left ventricular fibrosis but no acute cause of death, including no evidence of acute myocardial infarction or acute heart failure that may have caused VT or VF. Cause of death was adjudicated as probable hardware failure via inconsistent ventricular capture that led to significant bradycardia and pause-dependent polymorphic VT or VF.

After comprehensive evaluation, 7 of 8 individuals with ICDs were found to have died of arrhythmic causes (6 VFs and 1 VT in cases 16-22). The final individual with an ICD (case 15 in eFigure 6 in the [Supplement](#)) died of massive subarachnoid hemorrhage and had a terminal rhythm of VF. Device concerns were identified in 7 ICD individuals. Undersensing or failure to detect VT or VF was found in 5 individuals (cases 16-20) ([Figure 2](#), [Figure 3](#), and eFigures 7, 8, and 9 in the [Supplement](#)). In addition, 2 of the individuals with undersensing (cases 16 and 20 with device algorithm issues) also had a delay to shock therapy due to unsuccessful antitachycardia pacing programmed in the VF zone ([Figure 2](#) and eFigure 9 in the

[Supplement](#)). In these 2 cases, an algorithm issue specific to this manufacturer's devices was also identified: these devices were not able to charge during antitachycardia pacing and as a result were unable to deliver therapy immediately after unsuccessful antitachycardia pacing, thus contributing to delay to shock. One individual (case 21 with a programming issue in eFigure 10 in the [Supplement](#)) had failure to rescue because the VT was slower than the lower limit of the VT detection zone. One individual (case 22 with hardware failure in [Figure 4](#)) had a right ventricular lead fracture when delivering shocks for a VF event.

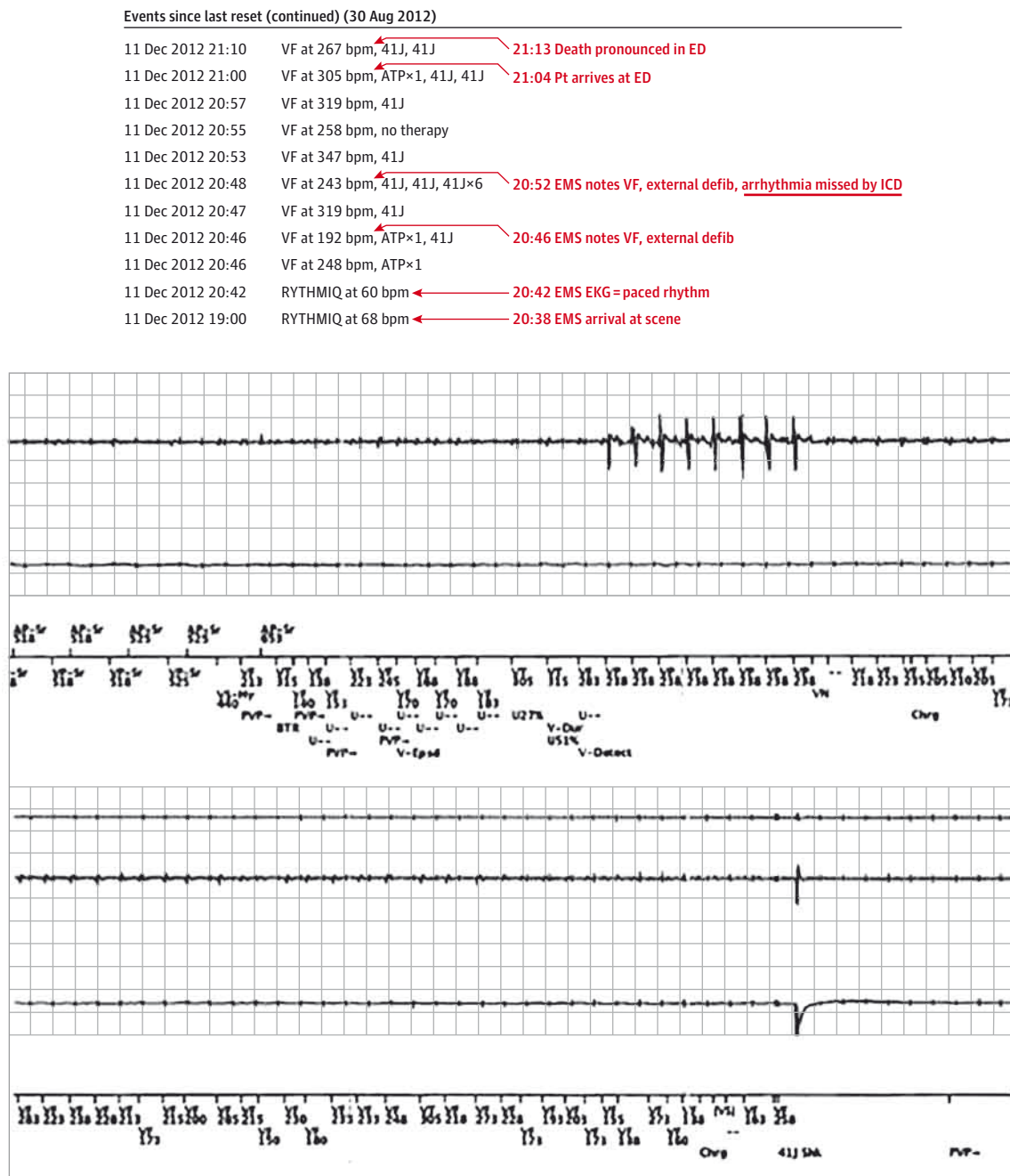
In total, 712 San Francisco residents had an ICD during the study period (Table), and 109 of these patients died (15.3% cumulative 35-month incidence of death). Sudden deaths and sudden deaths with a device concern accounted for 7.3% (8 of 109) and 6.4% (7 of 109) of total ICD mortality, respectively.

## Discussion

This study used prospective medical examiner surveillance to identify sudden deaths with CIEDs in San Francisco over a 35-month period. The major findings were as follows: (1) 4.3% (22 of 517) of sudden deaths in San Francisco occurred with a pacemaker or ICD; (2) half of the sudden deaths with CIEDs had a device concern; (3) VT or VF was the most common cause of death in PPM SCDs; (4) for patients with ICDs, the cumulative 35-month incidence of death was 15.3% (109 of 712), and 6.4% (7 of 109) of these deaths were SCDs with a device concern; and (5) VT or VF was the underlying cause of death in almost every SCD with an ICD.

Although multiple randomized trials<sup>9-11</sup> have demonstrated the survival benefits of ICDs in patients at high risk for sudden arrhythmic death, rates of sudden death have generally not been reported. Two trials<sup>12,13</sup> reported rates of arrhythmic death, but this end point was adjudicated based on clini-

Figure 2. Case 16 Device Interrogation



Top: Early episodes of ventricular fibrillation (VF) during emergency medical system (EMS) rescue were missed by the device and required external shocks for rescue. The clocks from the device programmer used to care for this patient (Pt) and the EMS were synchronized for this analysis. Bottom: Strips are continuous. Delay to shock was due to antitachycardia pacing (ATP) programmed in the VF zone. Ventricular fibrillation recurred 16 seconds after the shock and was successfully treated with another shock of 41 J (41J). On

autopsy, the patient had a scar from a remote myocardial infarction and 99% stenosis of the left anterior descending coronary artery but no acute myocardial infarction. bpm indicates beats per minute; defib, defibrillation; ED, emergency department; EKG, electrocardiogram; ICD, implantable cardioverter defibrillator; RYTHMIQ, a pacing algorithm (the event indicates that the device switched to a dual-chamber pacing mode); x1, 1 time; and x6, 6 times.

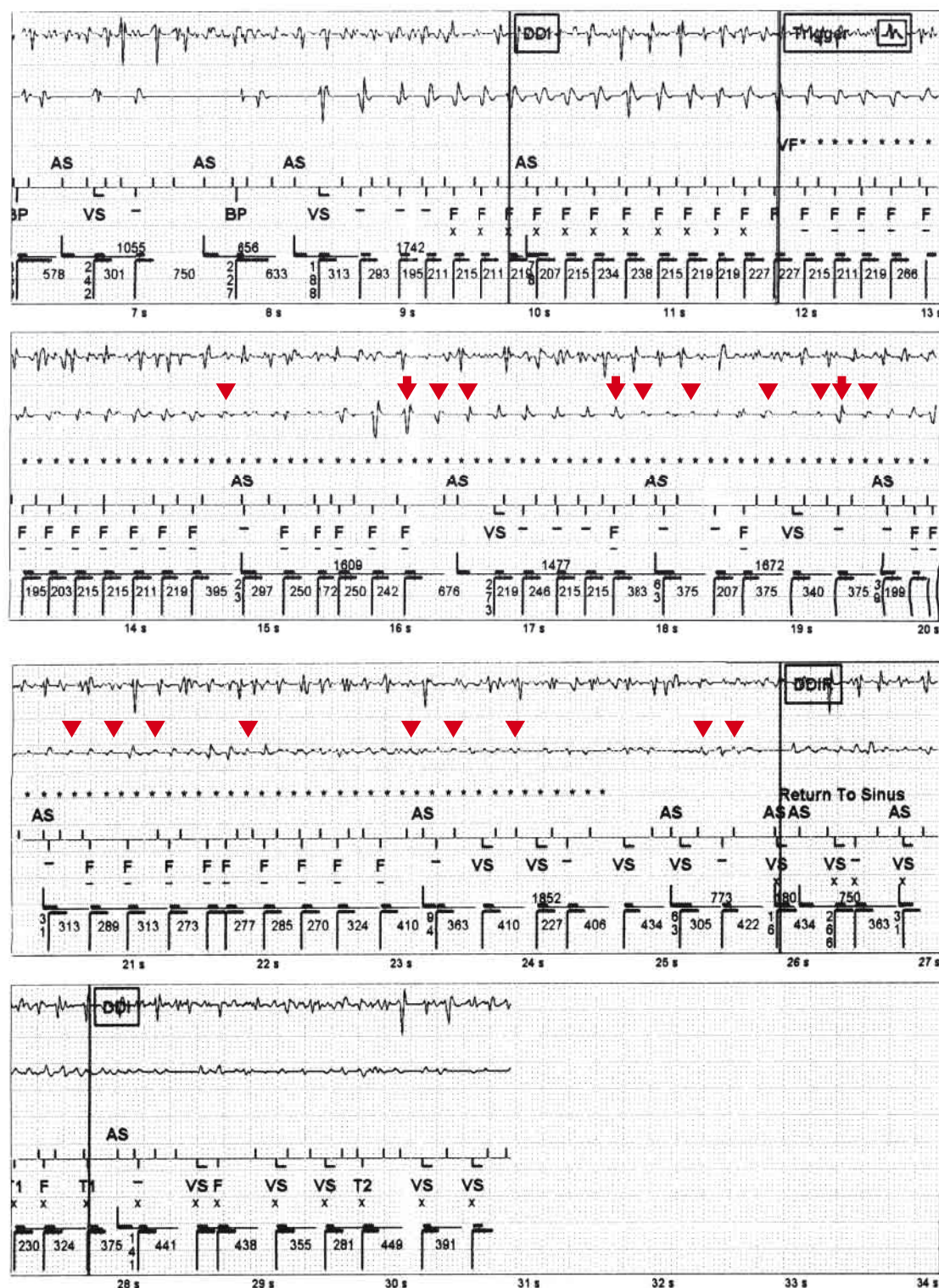
cal criteria, and postmortem device interrogation is not mentioned and thus likely was not performed.

Two large cohort studies<sup>14,15</sup> of patients with ICDs have found ICD lead failure rates of 2.5% to 15% at 5 years. Eckstein and colleagues<sup>14</sup> reported a 5-year cumulative incidence of lead

failure of 2.5% and a 5-year overall mortality of 22.8%. Over a median follow-up of 77 months, 38 lead failures and 315 deaths occurred in 1317 patients. Death was treated as a competing rather than censoring event and thus regarded as not involving device failure. Results from an ICD cohort study for actu-



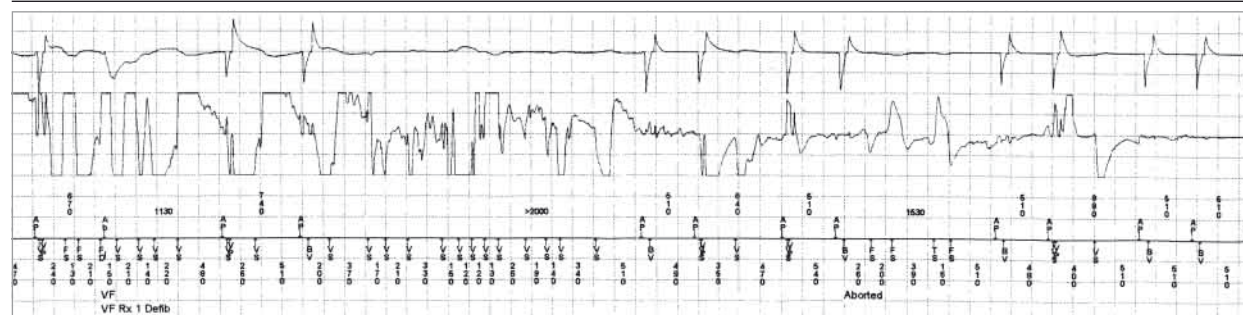
Figure 3. Case 17 Device Interrogation



Top: Atrial electrogram. Middle: Ventricular electrogram. Bottom: Device markers. Strips are continuous. Device interrogation demonstrates ventricular fibrillation (VF) that was undersensed, with the device interpreting a return to sinus rhythm (\*) and no shock delivered. The undersensed events (arrowheads) commonly occur after larger ventricular electrograms (arrows), which are due to a delay decay algorithm. Specific to this manufacturer, decay delay is a

nonprogrammable refractory period intended to allow sensing of the next ventricular event without double-counting the T-wave in detecting tachyarrhythmia. However, in this case causes undersensing of VF. AS indicates atrial sense; BP, biventricular pace; DDI, DDI mode of cardiac pacing; F, fibrillation; and VS, ventricular sense.

Figure 4. Case 22 Device Interrogation



Top: Atrial electrogram. Middle: Right ventricular electrogram. Bottom: Marker channels. The patient had ventricular fibrillation (VF) with successful defibrillation (3 times, not shown). After the fourth shock, right ventricular lead

noise with terminal underlying rhythm of VF is observed (shown) and no further shocks. Rx 1 Defib indicates the attempted therapy (defibrillation) for the episode of ventricular fibrillation.

atrial lead survival cannot be directly compared with those from our cross-sectional population study of the burden of out-of-hospital SCD. However, the device concern rate in our analysis would be in addition to the rate reported by these studies because the possibility of device failure leading to death was not included in their estimates. In San Francisco, we found that 6.4% (7 of 109 ICD deaths) of patients with ICDs who died had SCD with a device concern over a 35-month period. If 14.0% of the deaths over 77 months in the study by Eckstein and colleagues<sup>14</sup> were related to device concerns (an estimated 44 deaths), this would have more than doubled their rate of device concerns, and these missed cases would represent the most serious type of device concern (ie, those resulting in death).

One cohort study<sup>16</sup> that evaluated for arrhythmic deaths in ICD patients identified 16 out-of-hospital arrhythmic deaths in 822 ICD patients, but 15 of these patients did not undergo postmortem ICD interrogation. Few studies of pacemaker failure have been performed. Therefore, it is difficult to estimate the proportion of missed device concerns without postmortem evaluation of pacemaker sudden deaths.

We identified multiple device concerns, including the following: rapid battery depletion with a significant reduction in pacing output in a PPM-dependent patient, a rise in ventricular lead impedance 2 days before death and documented VT on device interrogation, undersensing of VF (5 cases), improper device selection, and programming-related issues (3 cases). In the cases of antitachycardia pacing programming in the VF zone, this programming extended the episode of VF and allowed for degradation of the signal and undersensing, thus further extending the interval and possibly directly leading to subsequent VF episodes due to ischemia or refractory VF. In these 2 cases, we also identified a device algorithm issue: the delay to shock was specific to this manufacturer's device, which is unable to deliver shock immediately after unsuccessful antitachycardia pacing, instead requiring redetection of VF and thus contributing to delay of therapy.

Our findings raise the possibility of undersensing of some VF episodes in delayed-style ICD programming, including antitachycardia pacing in the VF zone or prolonged detection intervals for VF. Longer detection intervals for VF have been re-

cently examined, with the objective of decreasing inappropriate shocks, and have demonstrated lower rates of shocks compared with standard programming.<sup>17,18</sup> Although these studies found no increase in mortality, they were powered for the number of shocks rather than mortality. Deaths were rare because the median follow-up was only 12 to 17 months, and causes of death were not reported. Because we found undersensing and prolongation of VF episodes in most ICD SCDs, delayed-style programming strategies, while improving patient outcomes by reducing unnecessary shocks, may result in an as-yet unmeasured increase in sudden deaths during longer follow-up, especially because patients may live up to 10 years with an ICD.<sup>19</sup>

Because ICD first-shock success rates for spontaneous VT or VF exceed 90%,<sup>20</sup> patients with functional ICDs might be expected to die of nonarrhythmic causes. The present study also illustrates the fact that, for some patients with sudden death, VF can be the final rhythm event for deaths from noncardiac causes, illustrated by the individual who developed VF due to subarachnoid hemorrhage and died, representing so-called neurocardiogenic VF.<sup>21</sup> Therefore, despite ICD documentation of VF episodes that resulted in rescue by the ICD, the patient experienced a sudden neurological death.<sup>22</sup> Without full autopsy examination, including opening the cranial vault, which is rarely performed in medical examiner or coroner cases, some of these deaths would have been incorrectly attributed to failure of the device to rescue from an arrhythmia. Moreover, case 20 died of VF but had acute thrombus in the right coronary artery on autopsy. Without autopsy, death would have been attributed exclusively to a device failure to rescue rather than acute myocardial infarction. Therefore, while systematic interrogation after death is necessary to uncover potential device problems, without concomitant autopsy the actual culpability of these concerns for SCD is less clear.

Our findings also demonstrate that corroborating information in addition to device interrogation is critical for complete evaluation of device function because devices can undersense or miss events. In case 16, the ICD log showed no arrhythmias during a 5-minute window when emergency medical system documentation showed administration of exter-



nal shocks to rescue VF. It is possible that even our in-depth interrogation missed other instances of undersensing during the study period. However, it would also be expected that a less robust surveillance system like the MAUDE database would be even more likely to miss these events.

This study also highlights the importance of proper CIED selection in patients with cardiac rhythm disorders. Case 2 had an indication for a primary prevention ICD based on clinical data before death: ICD implantation is a IIa recommendation in the most recent American College of Cardiology, American Heart Association, and Heart Rhythm Society guidelines<sup>7</sup> for patients with hypertrophic cardiomyopathy with these risk factors. It is recognized that the clinical decision for implantation of an ICD is not always straightforward and includes weighing psychosocial factors, patient wishes, comorbidities, and other factors that may be difficult to glean from a postmortem investigation. However, rigorous and systematic postmortem data provide an opportunity for clinical practice improvement.

This study has several limitations. Despite thorough investigation, the exact rhythm at the time of death is not always known because of uncertainty regarding the exact time of death, because the terminal rhythm did not meet criteria for device recording, or because the CIED had reached the elective replacement indicator and electrograms were not stored. In addition, despite the fact that this study has evaluated more comprehensive data per SCD than any other study to date, the exact cause of death sometimes remains unclear and is subject to interpretation. For example, we adjudicated 2 PPM-dependent SCDs with right ventricular lead concern due to premortem doubling of lead impedance not seen in PPM SCDs without lead concern, but these SCDs also had pneumonia (case 3) or cardiomegaly (case 4) and thus produced uncertainty regarding the true cause of death.

Also, when VF persists for prolonged periods, electrogram amplitude decreases and undersensing are potential problems.<sup>23</sup> Whether appropriate sensing and additional shocks could have saved these patients who had recurrent VF, despite multiple appropriate shocks, is not known. In addition, programming changes to increase detection of VF by preventing dropout have the potential to increase inappropriate shocks.<sup>24</sup> Furthermore, epidemiological studies<sup>6,25</sup> of SCD vary on whether to include patients with end-stage renal disease. We included CIED SCDs with end-stage renal disease (case 19 in this study) because these patients were included in the ICD-only study, and the goal was to evaluate all patients with active devices. However, patients with end-stage renal disease are excluded from the POST SCD study, and removing the one CIED SCD case with end-stage renal disease from the rate analysis gives a rate of 4.1% (21 of 517) over 35 months, which is virtually unchanged from the main estimate of 4.3% (22 of 517). Finally, the numbers of patients with PPMs in San Francisco and nationally are not precisely known, and further study in this area is needed to determine actuarial rates of PPM failure that lead to sudden death.

## Conclusions

In summary, over a 3-year period in one city, systematic interrogation and autopsy of SCDs with CIEDs, which is not part of standard practice, identified concerns about CIED function that might otherwise not have been observed. Current passive surveillance efforts may underestimate device malfunction. A prospective, systematic approach incorporating these methods can provide unbiased data regarding what may lead to sudden death in individuals with CIEDs and improve surveillance for CIED problems.

## ARTICLE INFORMATION

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**Study concept and design:** Tseng, Olgin, Hart.

**Acquisition, analysis, or interpretation of data:**

Tseng, Hayward, Clark, Mulvanny, Colburn, Ursell, Olgin.

**Drafting of the manuscript:** Tseng, Hayward, Clark, Colburn, Olgin.

**Critical revision of the manuscript for important intellectual content:** Tseng, Hayward, Mulvanny, Ursell, Olgin, Hart, Moffatt.

**Statistical analysis:** Tseng, Hayward, Clark, Mulvanny.

**Obtained funding:** Tseng.

**Administrative, technical, or material support:** Hayward, Clark, Mulvanny, Colburn, Olgin, Hart, Moffatt.

**Study supervision:** Tseng, Colburn, Ursell, Olgin, Hart, Moffatt.

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(University of California, San Francisco), assisted with preparation of the tables and figures. Nikolaos Lemos, PhD (Office of the Chief Medical Examiner, City and County of San Francisco), performed all toxicological analyses for sudden cardiac deaths in the Postmortem Systematic Investigation of Sudden Cardiac Death (POST SCD) study.

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## Editor's Note

## Strengthening Medical Device Postmarket Safety Surveillance

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**The system** by which the US Food and Drug Administration engages in medical device postmarket safety surveillance needs strengthening.<sup>1-4</sup> Efforts are limited by reliance on passively aggregated adverse events through the Manufacturer



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and User Facility Device Experience (MAUDE) database, investigated adverse events at select clinical sites within the Medical Product Safety Network (MedSun), and select active surveillance efforts using Post-Approval Studies and Postmarket Surveillance Studies (also known as the 522 Postmarket Surveillance Studies Program). While these efforts have successfully detected potential safety issues and contributed to reassessments of the benefits and risks, these systems are likely to identify only a small proportion of the totality of adverse events that occur. Passive surveillance is

undermined by the frequent reporting of incomplete, inaccurate, untimely, unverified, or biased data, whereas active surveillance is expensive, often narrow in scope, and impaired by the frequent delays in completion of requested studies. Efforts will be enhanced by the planned addition of a Unique Device Identification System integrated within electronic health care data, such as administrative claims data collected by health insurance payers for billing purposes, which would allow large-scale, proactive assessments of medical device safety.<sup>4</sup>

Without a better functioning system, postmarket surveillance of medical devices has largely been driven by experiences at select institutions, where individuals identify and publicize potential safety issues that the Food and Drug Administration subsequently investigates, such as the recent class I recall of implantable cardioverter defibrillator leads.<sup>5</sup>