New Approaches to the Management of Ventricular Tachycardia

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Internal Medicine Grand Rounds

UT Southwestern Medical Center

August 3, 2012

This is to acknowledge that Richard Wu, M.D. has disclosed that he has disclosed research relationships with commercial concerns related directly or indirectly to this program. Dr. Wu will not be discussing off-label use of drugs or devices in his presentation.

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Purpose

The article is a discussion of current and developing treatment approaches to the management of ventricular tachycardia (VT) in patients with structural heart disease at high risk for sudden cardiac death. Most of the focus will be on patients with coronary artery disease (prior myocardial infarction) and systolic heart failure with recurrent VT refractory to standard medical therapy. Indications for antiarrhythmic drugs, internal cardioverter defibrillator, and catheter ablation will be discussed in detail.

Educational Objectives

After this presentation, the participant will have an increased knowledge of these principles:

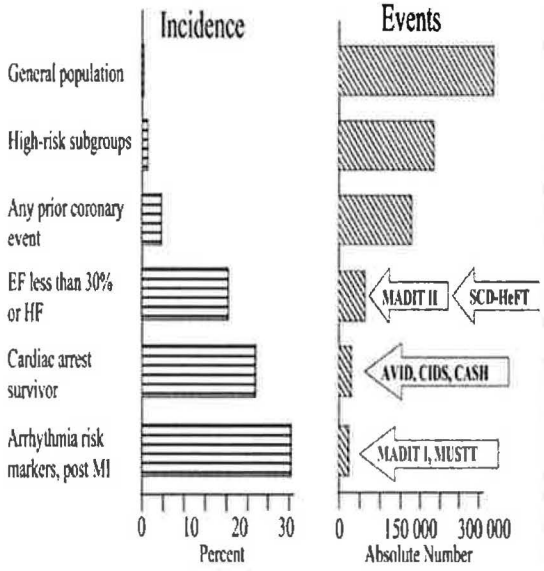
Describe the role of anti-arrhythmic drug therapy for the management of VT

Identify patients who are appropriate candidates for defibrillator or ICD therapies

When to recommend referral of patients for advanced therapies such as VT ablation

Introduction

Ventricular tachycardia (VT) is an arrhythmia characterized by a rapid rate (>100 beats per minute) with 3 or more consecutive beats originating from the ventricles.[1] In patients with underlying structural heart disease, VT is a major cause (>80%) of sudden cardiac death (SCD).[2] In the United States, the incidence of SCD accounts for ~300,000 deaths annually with an overall incidence 1 to 2 deaths per 1,000 adults (0.1 to 0.2%, figure 1).[3] Coronary artery disease (CAD) is the most common underlying cause (80%) of SCD.[4] Following a myocardial infarction, impaired left ventricular dysfunction and systolic heart failure are strong predictors for an increased risk of sudden death, with those with left ventricular ejection fraction of 30 per cent or less (LVEF<30%) carrying the greatest risk (~10% over 2 years, figure 2).[4, 5] For patients that experience an episode of sustained VT, the recurrence rates of VT can be as high as 63%.[6] Multiple treatments have been developed of the years including medical therapy, internal cardioverter defibrillators (ICD), and catheter ablation. This presentation will discuss the most current and developing treatment options for individuals with ventricular tachycardia in the setting of structural heart disease, particularly those with CAD.



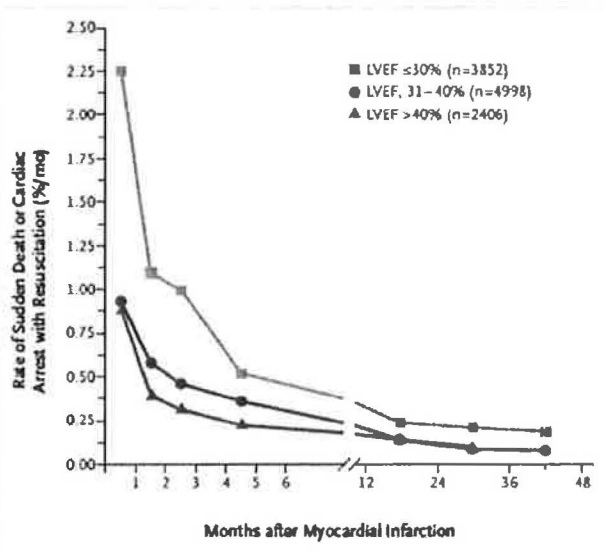


Figure 1 (left) Event rate of SCD in general population and subpopulation over 1 year[3, 7]

Figure 2 (right) Rate of Sudden Death or Cardiac Arrest With Resuscitation following Myocardial Infarction with risk stratification by Left Ventricular Ejection Fraction (LVEF).[5]

Ventricular Tachycardia (VT) Definitions and Mechanisms

Ventricular Tachycardia (VT) is an arrhythmia with three or more consecutive beats that originates from the ventricles (ventricular myocardium, His-Purkinje system, or in combination) independent of atrial or AV nodal conduction. The tachycardia rate is greater than 100 bpm. A

non-sustained VT terminates spontaneously within 30 seconds, and sustained VT is continuous > 30 seconds or requires and intervention for termination (such as cardioversion).

VT can be classified by 12-lead ECG morphology as monomorphic (similar QRS beat to beat), pleomorphic (more than one morphologically distinct monomorphic VT during the same episode of VT), or polymorphic (continuously changing QRS complex during the same episode of VT) - see figure 3.[1]

Monomorphic VTs in patients with structural heart disease (coronary artery disease – CAD or cardiomyopathy – CMP) are mostly due to reentry associated with areas of scar. Prior myocardial infarction (MI) is the most common cause.[3, 8] The damaged muscle is replaced by fibrotic tissue, however, surviving bundles of myocardium border the area of scar which have abnormally slow conduction and create regions of anatomical or functional conduction block facilitating reentry.[9] An isthmus of slow conduction is created when conductive myocardial tissue is bounded by 2 parallel barriers or scar (figure 4).[10] The QRS morphology of VT is determined by the location of the scar, the location of the reentry circuit, and the exit site from where the wavefront propagates away from the scar to depolarize the ventricles. Multiple VTs with different morphologies may originate from the same scar, use different circuits with the same isthmus but have different exits sites. The isthmus may span several centimeters.

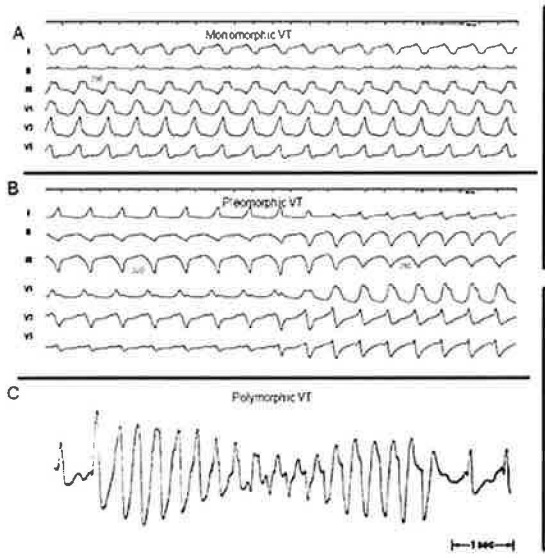


Figure 3 (left) Monomorphic, Pleomorphic, and Polymorphic VT

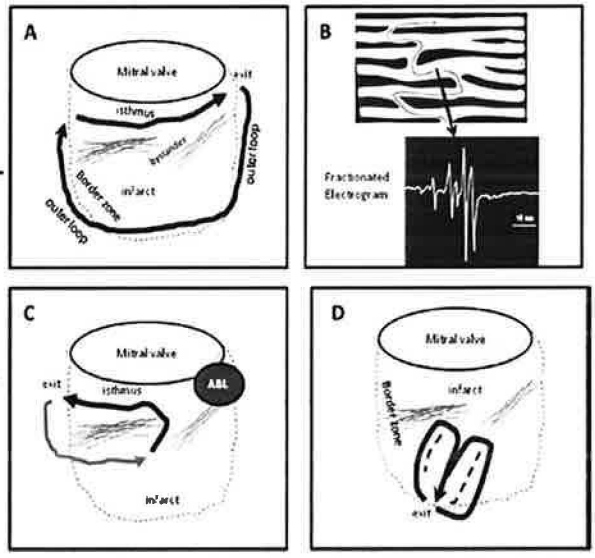


Figure 4 (right) Theoretical reentry circuits related to an inferior wall infarct scar.[1]

Monomorphic VT can occur in individuals without structural heart disease and are more commonly due to triggered activity or automaticity, and generally have a focal origin. Triggered activity arises from oscillations in the membrane potential and action potential, leading to depolarization of ventricular myocytes. Early afterdepolarizations and delayed afterdepolarizations induce triggered activity following intracellular calcium overload of

myocytes, more common after ischemic injury or adrenergic stimulation.[1, 11] Focal VT or frequent PVCs in patients with structural heart disease can initiate reentrant VT.

Polymorphic VTs have changing or multiple sites of activation. These arrhythmias are associated with acute ischemia (depletion of intracellular adenosine ATP, anaerobic glycolysis, lactic acidosis, intracellular sodium and calcium overload), electrolyte abnormalities, drug-induced long QT syndrome, and inherited ion channelopathies.[1, 7] Polymorphic VTs are less likely to have a target for ablation therapies and warrant a different discussion in diagnosis and management.

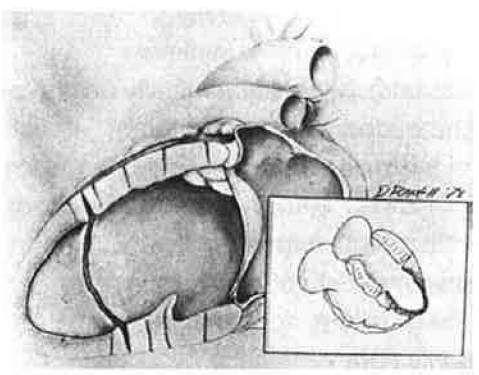
Ventricular tachycardia is also classified by clinical presentation and disease entity. Hemodynamically stable VT includes patients who are asymptomatic or have minimal symptoms of palpitations (pounding, racing, or skipped heartbeats). Hemodynamically unstable patients may report symptoms of presyncope (dizziness, lightheadedness, feeling faint), syncope (sudden loss of consciousness with loss of postural tone), sudden cardiac death (death form unexpected circulatory arrest, usually due to cardiac arrhythmia within an hour of the onset of symptoms) or sudden cardiac arrest (aborted sudden cardiac death where medical intervention such as defibrillation reverses event).[7] Patients with structural heart disease by definition have a clinical history of coronary artery disease, heart failure, congenital heart disease, cardiomyopathy (dilated, hypertrophic, arrhythmogenic right ventricular dysplasia, valvular), or hypertension with moderate to severe left ventricular hypertrophy.[7]

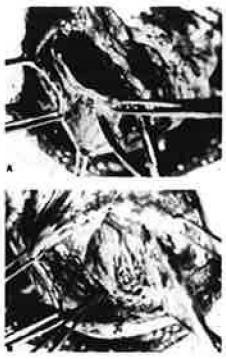
Surgical VT Ablation

Encircling endocardial ventriculotomy was first performed for treatment of life-threatening ventricular tachycardias resistant to medical treatment following myocardial infarction by Guiraudon and colleagues in 1978.[12] At that time, it was established that (1) myocardial necrosis is maximal at the center of the infracted area, becomes fibrosed, thin-walled, or aneurismal, (2) around the center border, there is a transition between normal muscle, ischemic muscle, and fibrotic tissue or a "twilight border zone," and (3) the ischemic lesions are essentially located in the subendocardial layer. Endocardial ventriculostomy was performed on 5 patients with history of prior MI between 1 month to 8 years, symptoms of syncope or angina, and ventricular tachycardia that could be induced by the extrastimulus technique which confirmed a reentrant mechanism. The ventricle was opened through the thinned zone of the infarct for direct visual examination, and transmural ventriculostomy was performed perpendicular to the wall along the border zone of diseased and healthy myocardium (see figure). The objective was to create a fibrous scar barrier around the diseased zone, separating it from healthy myocardium (figure 5). This was successful in preventing recurrent VT in all patients over an average of 1 year follow-up.[12]

Aneurysmectomy and endocardial excision was performed in 12 patients with refractory VT by Josephson and Harkin.[13] Using new techniques of pre- and intraoperative programmed

stimulation and mapping, Josephson demonstrated that simple aneurysmectomy alone did not abolish VT (11/12) patients had inducible VT after aneurysmectomy or ventriculotomy), but that resection of the diseased endocardium to the lateral border of the aneurysm in the area of VT origin was more effective (figure 6). This demonstrated that the reentrant circuit could be isolated to a smaller discrete area and that the procedure could be performed in patients with septal VTs, where aneurysmectomy is not always feasible. The surgical experience of VT ablation serves as the basis for catheter ablation of VT, where linear lesions are created and extend from dense scar to normal myocardium (figure 7).[14] Ablation of the scar border zone interrupts reentrant VT circuits or isthmuses, preventing initiation or propagation of VT.





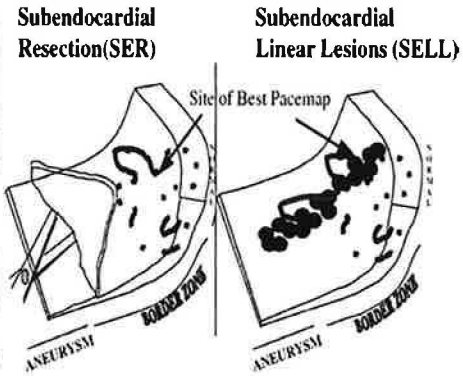


Figure 5 (left) Encircling Ventricuolotomy for Refractory Ventricular Tachycardia[12]

Figure 6 (middle) Endocardial Excision or resection for Ventricular Tachycardia[13]

Figure 7 (right) Contrast between surgical subendocardial resection and linear lesions created by catheter ablation extending from dense scare to normal myocardium. Placement of linear lesion is guided by pace mapping, much like subendocardial resection, to interrupt reentrant VT circuits.[14]

Although there is early and continually evolving experience with surgical VT ablation, it is not considered first-line therapy for management of refractory VT. Despite reported long-term arrhythmia control rate of 75-80%, peri-operative mortality can be as high as 10% and death from other causes remains high.[15-17] Percutaneous catheter ablation (including subxiphoid pericardial puncture) is generally attempted before surgical ablation. Surgical ablation is performed when catheter ablation fails, when venous or pericardial access to the heart is limited (venous thrombosis, adhesions due to previous cardiac surgery or pericardial inflammation), and electrophysiology (EP) study identifies an arrhythmia that is amenable to an open chest surgical procedure. VT refractory to catheter ablation is more commonly seen in non-ischemic cardiomyopathy.[15] VT circuits inaccessible to percutaneous ablation can be successfully targeted by surgical ablation in the operating room (under cardiopulmonary bypass -CPB) guided by pre-operative EP study or using a hybrid approach where surgical epicardial access (subxiphoid pericardial window or limited anterior thoracotomy performed without CBP) is performed prior to epicardial mapping or ablation in the EP lab.

Antiarrhythmic Drugs (AAD)

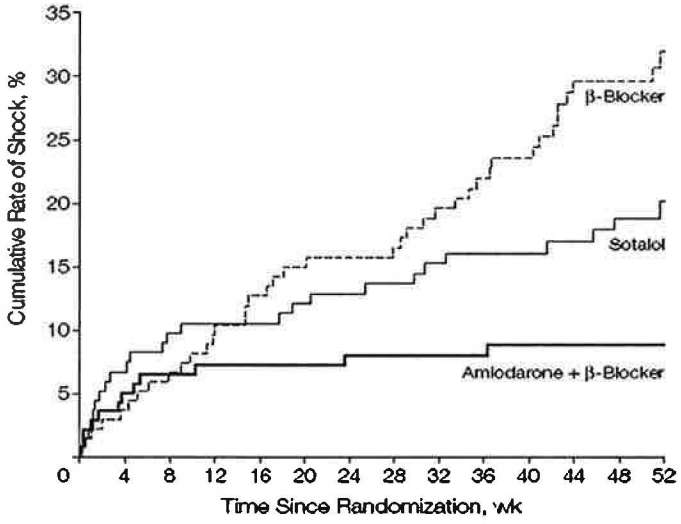
ICD therapy has been shown to be superior to anti-arrhythmic drug therapy in preventing SCD. However, the use of anti-arrhythmic drugs (AAD) is often necessary to prevent recurrent VT or frequent ICD shocks. In the AVID trial (Antiarrhythmics versus Implantable Defibrillator) trial ~20% of patients in the ICD arm had to start adjuvant AAD (amiodarone 42%, sotalol 21%, and mexiletine 20%).[18] The main benefit of AAD is to prevent episodes of VT and atrial tachyarrhythmias (which can induce VT or produce rapid ventricular rates) to reduce ICD shocks.[19] Additionally, AAD may stabilize or slow ventricular arrhythmias and make it more amenable to termination with pacing (anti-tachycardia pacing – ATP).

Class IC AADs can be detrimental in patients with structural heart disease of CAD. The Cardiac Arrhythmia Suppression Trial (CAST) examined the effect of encainide and flecainide on mortality in a randomized trial of arrhythmia suppression after myocardial infarction. [20] Although these drugs were effective in suppressing ventricular arrhythmias in asymptomatic patients, overall mortality (RR 2.5) and non-fatal cardiac arrests were higher in the group receiving treatment with IC drugs. The Cardiac Arrest Hamburg (CASH) trial randomized cardiac arrest survivor patients between implantable cardioverter-defibrillator (ICD) versus antiarrhythmic drug therapy (amiodarone, propafenone, or metoprolol). The propafenone arm was discontinued before completion of the study after interim analysis showed 61% higher all-cause mortality than in ICD patients, most likely because of pro-arrhythmic effects.[21] In the CASH trial, crude death rates were 36.4% in the ICD arm compared to 44.4% in the amiodarone/metoprolol arms with a 23% (non-significant) reduction in all-cause mortality and 61% reduction in sudden cardiac death in patients receiving ICD therapy compared to drugs.[21]

Beta-blocker therapy is standard therapy for the treatment of coronary artery disease and systolic heart failure.[22] In particular, carvedilol and metoprolol, have been shown to reduce all cause mortality or SCD, but have not been specifically studied for the treatment of VT. Most clinical trials for ICDs, for example, compare standard medical therapy (including treatment with a beta-blocker) to device therapy in addition to medical therapy, and not placebo. In a retrospective analysis of the MADIT-II trial, patients receiving beta-blocker had a significant reduction in risk of recurrent VT/VF requiring ICD therapy as compared with patients not receiving beta-blocker (HR, 0.48; p=0.02).[23] Small clinical trials have suggested that sotalol, a class III AAD with beta-blocker properties, reduce frequency of ICD shocks due to any cause compared to placebo or metoprolol, but have no significant effect on survival or even increased mortality compared to metoprolol.[24-26]

Amiodarone has been studied and compared to the efficacy of beta-blocker and sotalol in the prevention of ICD shocks. In the Optimal Pharmacological Therapy in Cardioverter Defibrillator Patients (OPTIC) trial, patients in the amiodarone plus beta-blocker was more effective than beta blocker alone (HR 0.27) in preventing both appropriate (VT/VF) and inappropriate (AT/AF) shocks (figure 8). There was a trend toward better efficacy of sotalol compared with beta-blocker alone but did not reach statistical significance. Mortality rate did not differ among the 3 groups. However, rates of study drug discontinuation were high in the

amiodarone group (18.2%) and sotalol group (23.5%) compared to the beta-blocker alone group (5.3%) Adverse side effects (pulmonary, thyroid toxicity, bradycardic events) were common in the group randomized to amiodarone.[27] A meta-analysis of 15 randomized controlled trials examining the use of amiodarone vs placebo or control for the prevention of sudden cardiac death found that amiodarone (average daily maintenance dose 200mg) reduced the risk of SCD by 29% but was neutral with respect to all-cause mortality and associated with 2-5X risk of pulmonary and thyroid toxicity.[28]



No. at Risk

β-Blocker	138	119	109	91	42
Sotalol	134	118	108	94	35
Amiodarone + β-Blocker	140	124	115	106	56

Figure 8 Cumulative risk of ICD shock in patients randomized to 3 anti-arrhythmic drug treatment groups from OPTIC [27] Amiodarone plus beta-blocker reduces the risk of shock (HR 0.27) compared to beta blocker (without change in mortality rate).

Drug induced pro-arrhythmia, particularly Torsades de Pointes, are a rare but potential adverse side effect with class III AAD. Extracardiac side effects of amiodarone are a limitation to long term use.

According to the **ACC/AHA Guidelines for Management of Patients with Ventricular Arrhythmias and Prevention of Sudden Cardiac Death**[7], use of antiarrhythmic drugs in patients with prior MI are summarized as follows:

Class IIa (Conditions for which there is conflicting evidence and/or divergence of opinion about the usefulness/efficacy of a procedure or treatment. Weight of evidence/opinion is in favor of useful).

1. Amiodarone, often in combination with beta blockers, can be useful for patients with LV dysfunction due to prior MI unresponsive to beta-blocker agents.

2. Sotalol is reasonable therapy to reduce symptoms resulting from VT for patients with LV dysfunction due to prior MI unresponsive to beta-blocker agents.
3. Amiodarone is reasonable therapy to reduce symptoms due to recurrent, hemodynamically stable VT for patients with LV dysfunction due to prior MI who cannot or refuse to have an ICD implanted.

Class III (Conditions for which there is evidence that a procedure/treatment is not useful/effective and in some cases may be harmful).

1. Prophylactic antiarrhythmic drug therapy is not indicated to reduce mortality in patients with asymptomatic nonsustained ventricular arrhythmias.
2. Class IC antiarrhythmic drugs in patients with a past history of MI should not be used.

Defibrillators and Implantable Cardioverter Defibrillators (ICD)

The principal treatment of VT in structural heart disease relies on the ICD for the prevention of sudden death. An ICD does not prevent VT but attempts to terminate the arrhythmia. ICD therapy compared with conventional or antiarrhythmic drug therapy has been associated with mortality reduction from 23 to 55% depending on the risk group of the trial.[7] The trial are categorized into either 1) primary prevention (prophylactic treatment) in which subjects have not experienced a life-threatening ventricular arrhythmia or syncope, or 2) secondary prevention trials, where subjects have been resuscitated from cardiac arrest, experienced hemodynamically unstable sustained ventricular arrhythmias, or suffered from unexplained syncope with a work-up suggesting ventricular arrhythmias as the cause of syncope.

Secondary Prevention ICD Trials

The first and largest of the "secondary prevention" of sudden cardiac death trials was the Antiarrhythmics Versus Implantable Defibrillators (AVID) trial which compared treatment with antiarrhythmic medications (primarily amiodarone) to implantation of an ICD in patients who had been resuscitated from a ventricular fibrillation (VF) arrest or who underwent cardioversion from sustained ventricular tachycardia. After a mean follow up of 18 months, the mortality rates were 15.8% in the ICD group and 24% in the antiarrhythmic drug (AAD) arm.[29] Two other trials, the Canadian Implantable Defibrillator Study (CIDS) and the Cardiac Arrest Study Hamburg (CASH), also randomized patients who survived cardiac arrest to ICD versus antiarrhythmic medications and both showed ~20% reduction in mortality from ICD therapy compared to drug, but neither reached statistical significance (see table).[21, 30] Shortly after the publication of the CIDS trial, the primary investigators for the AVID, CASH, and CIDS trials published a meta-analysis of the data accumulated in the 1866 patients enrolled in those three trials. After pooling the primary data from the three trials, the hazard ratio for all-cause mortality was 0.72 (P= 0.0006) for the ICD group compared to the amiodarone group.[17,31] The mortality benefit was most prominent in patients with an EF<35%, and was not apparent in patients with an EF

>35% when these subgroups were analyzed using the pooled data. The secondary prevention trials are summarized in Table 1.

Trial	Patients	Trial design	All-cause mortality
AVID	1016	EF<40% ICD or optimal drug therapy (amiodarone/sotalol)	Overall survival: 75.4% vs. 64.1%, p=<0.02 31% reduction in death at 3 yrs with ICD
CIDS	659	EF<40% ICD or amiodarone	20% reduction in all cause mortality (3 yrs) with ICD (p=ns vs. amiodarone)
CASH	288	ICD, amiodarone, metoprolol or propafenone	23% reduction all-cause mortality (9 yrs) with ICD (p=ns vs. drug treatment)

Table 1. Summary of secondary prevention ICD trials

Many studies support the use of ICD for primary prevention of SCD in patients with ischemic and non-ischemic cardiomyopathies. Reviewing the trials in detail is beyond the scope of this presentation and the results are summarized in Table 2. The Multicenter Automatic Defibrillator Implantation Trial (MADIT) demonstrated efficacy of ICD therapy in patients with ischemic cardiomyopathy (prior MI), congestive heart failure, LVEF $\leq$ 35%, and non-sustained VT risk stratified by electrophysiology study (EPS).[32] During EPS, if sustained VT was induced, patients were randomized to ICD therapy versus usual medical therapy. The MADIT II trial used history of MI, NYHA class I-III congestive heart failure, and LVEF $\leq$30% for enrollment.[33] The Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) is the only primary prevention trial to enroll patients with either ischemic or nonischemic cardiomyopathy, and proved that amiodarone was not as effective as an ICD in preventing sudden death.[34] This trial enrolled 2521 patients with LVEF$\leq$ 35%, and NYHA class 2 or 3 heart failure to conventional medical therapy plus placebo, conventional medical therapy plus amiodarone or conventional medical therapy plus ICD implantation. The primary endpoint, like the other primary prevention trials, was all-cause mortality. After a median follow up of 45 months, the hazard ratio for ICD therapy was 0.77 (P=0.006) compared to the placebo group and the hazard ratio for the amiodarone group was 1.06 (P=0.53). At 5 years, the absolute risk reduction in all cause mortality in the ICD group was 7.2%. ICD therapy appeared to be beneficial regardless of the etiology of the cardiomyopathy.

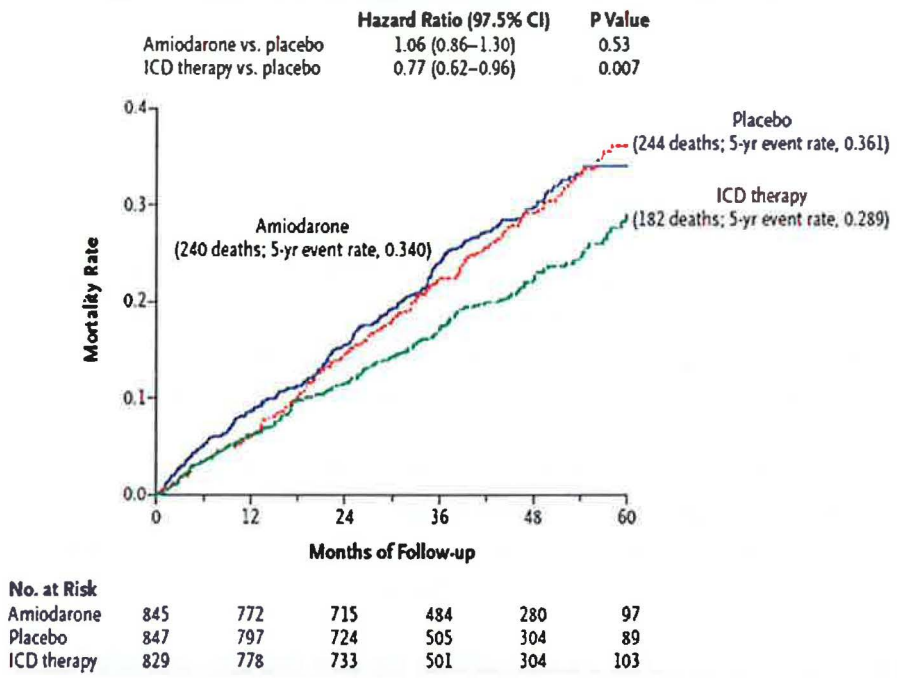


Figure 9 SCD-HeFT Trial Kaplan-Meir Estimates of Death from Any Cause. No survival benefit of amiodarone compared to placebo (conventional CHF therapy).[34] ICD reduced overall mortality (23% relative risk reduction) compared to amiodarone or placebo.

Trial	Year	Patients (n)	LVEF	Additional Study Features	Hazard Ratio*	95% CI	p
MADIT I	1996	196	≤ 35%	NSVT and EP+	0.46	(0.26-0.82)	p=0.009
MADIT II	2002	1232	≤ 30%	Prior MI	0.69	(0.51-0.93)	p=0.016
CABG-Patch	1997	900	≤ 36%	+SAECG and CABG	1.07	(0.81-1.42)	p=0.64
DEFINITE	2004	485	≤ 35%	NICM, PVCs or NSVT	0.65	(0.40-1.06)	p=0.08
DINAMIT	2004	674	≤ 35%	6-40 days post-MI and Impaired HRV	1.08	(0.76-1.55)	p=0.66
SCD-HeFT	2006	1676	≤ 35%	Prior MI of NICM	0.77	(0.62-0.96)	p=0.007

Table 2. Summary of primary prevention ICD trials

According to the **ACC/AHA Guidelines for Management of Patients with Ventricular Arrhythmias and Prevention of Sudden Cardiac Death**[7], use of ICDs are recommended as follows for both ischemic and non-ischemic cardiomyopathies*:

Class I (Conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective).

1. ICD therapy is recommended for **primary prevention to reduce total mortality** by a reducing in SCD in patients with LV dysfunction due to prior MI are at least 40 d post MI and LVEF less than or equal to 30-40%, and are NYHA functional class II or III.
2. ICD is effective to reduce mortality by a reduction in SCD with LV dysfunction due to prior MI who present with hemodynamically **unstable sustained VT**, and are receiving chronic optimal medical therapy.
3. In ICD should be implanted in patients with non-ischemic dilated cardiomyopathy and significant LV dysfunction who have **sustained VT or VF**.
4. ICD is recommended for primary prevention to reduce morality in patients with dilated cardiomyopathy and LVEF $\leq$35%, NYHA functional Class II or III.

Recommendations apply to patients receiving chronic optimal medical therapy, and who have reasonable expectation of survival with a good functional status more than 1 yr.

Class II (Conditions for which there is conflicting evidence and/or divergence of opinion about the usefulness/efficacy of a procedure or treatment. Weight of evidence/opinion is in favor of useful).

1. Ischemic cardiomyopathy with LVEF < 30% and class I symptoms.
2. Ischemic (or non-ischemic) cardiomyopathy, recurrent, sustained VT, and normal or near normal LVEF.
3. Patients with unexplained syncope, significant LV dysfunction, and dilated cardiomyopathy.

For more details on other indications (hypertrophic cardiomyopathy, congenital heart disease, channelopathies, etc.) and contraindications for ICD implant, please refer to the guidelines.[7]

ICD Therapies

Although ICD shocks are often lifesaving, patients may receive frequent shocks after implantation. ICDs do not prevent ventricular tachycardia but terminate the arrhythmia by halting the activation wavefront in at least one critical region of ventricular myocardium preventing reentry.[35] This occurs either by DC shock (cardioversion or defibrillation) or anti-tachycardia pacing.[36, 37] ICD shocks may occur in 33-50% of those who receive a device implant.[27, 38] Repeated ICD shocks for VT within a short period (>3 separate arrhythmia episodes leading to ICD therapies within 24 hours), known as "electrical or VT storm" occur in 10 to 24% of patients.[39, 40] Electrical storm is potentially life-threatening and has poor short and long-term prognosis. In the SCD-HeFT trial, 20% of ICD patients still died from sudden death.[41]

Both appropriate ICD shocks (for VT or VF) and inappropriate shocks (for SVT or lead malfunction) increase mortality.[38] Data from the SCD-HeFT trial showed that an appropriate shoc was associated with a significant increase in subsequent death from all causes (HR 5.68). Prevention of appropriate ICD shocks for VT, either by programming ICDs for anti-tachycardia pacing[42] or VT ablation may improve mortality.[43]

Ventricular Tachycardia (VT) Ablation

Catheter ablation of ventricular tachycardia in patients with underlying heart disease was introduced in the early 1980's but only progressed after the development of radiofrequency (RF) current ablation and mapping techniques developed in the early 1990's.[44, 45] Initially, two limitations of catheter ablation were that it relied on conventional RF catheters and that it was performed in patients with hemodynamically tolerated VT, such that "activation mapping" or identification of the potential ablation sites was performed mostly during sustained episodes of VT.[46] However, only 10-50% of VTs are considered "mappable."[47] The introduction of electroanatomical mapping systems using computers to create a three-dimensional (3D) geometric model of the heart chamber to track catheter location with corresponding voltage facilitated the development of "substrate mapping." [14, 48, 49] VT ablation using substrate modification relies on identifying areas of low voltage corresponding with scar from previous infarction (figure). More contemporary approaches rely on delivering ablation lesions to these low-voltage areas (scar with surviving bundle of myocardium) to eliminate potential reentrant circuits (VT isthmuses) from within the scar.[50] Despite delivery of multiple ablation lesion, this technique does not significantly affect LV function when lesion are created in diseased or previously damaged low-voltage areas of myocardium.[47, 51]

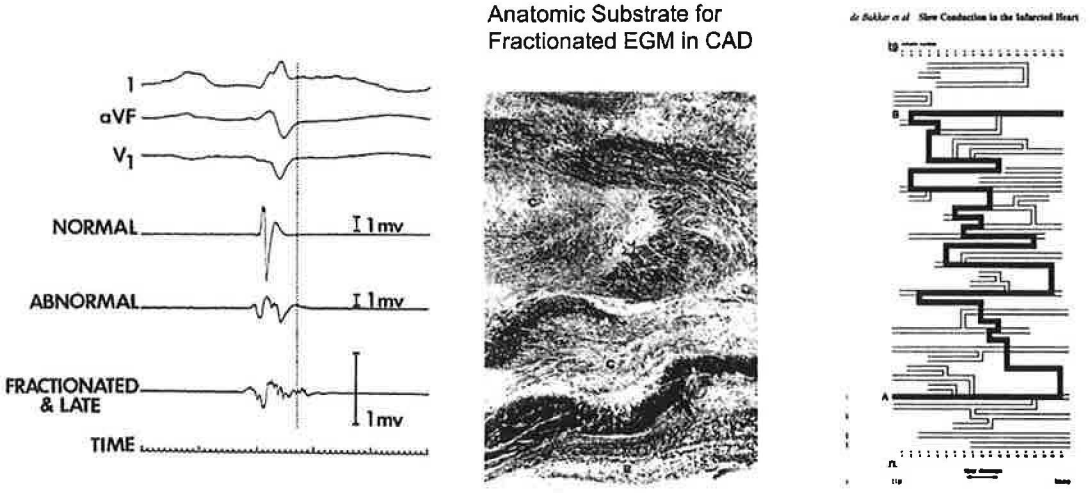


Figure 11 (left) Anatomic Substrate for Fractionated EGM in CAD[52]

Figure 12 (right) Slow Conduction in Infarcted Human Heart. Zigzag course of activation.[9]

VT Ablation outcomes in patients with previous myocardial infarction

In most published series of catheter ablation for ventricular tachycardia, the procedure is performed in patients with ischemic heart disease (prior MI) after receiving multiple ICD interventions, including patients with incessant VT. The largest contemporary (Thermocool) trial conducted in the US included 231 patients (mean LVEF 25%) with recurrent monomorphic VT (median 11 episodes) both mappable and unmappable (69% of patients), 70% of which failed treatment with amiodarone.[47] After ablation, ~50% of patients were free of VT at 6 months, and ~2/3 patients had at least a 75% reduction in in VT events. The procedure mortality rate was 3%, however, the 1 year morality rate was 18% in this population (73% deaths due to arrhythmias or heart failure).[47]

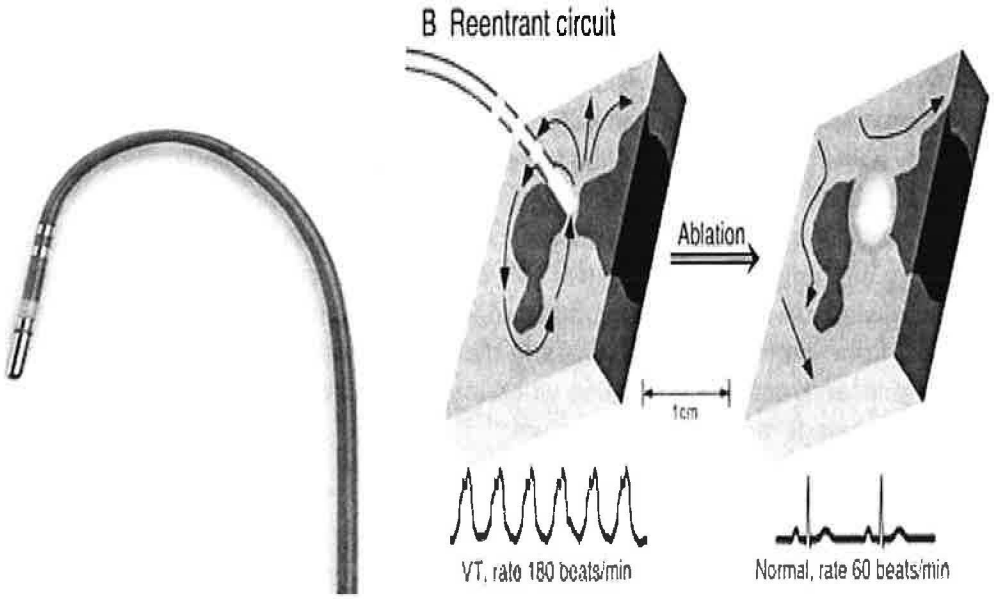


Figure 13 Radiofrequency Energy Ablation Catheter and Ischemic VT ablation. Radiofrequency energy (alternating electrical current) heats muscle and produces a necrotic lesion. When applied to the myocardium at a potential reentrant isthmus, the RF lesion interrupts the VT circuit.[45, 53]

A limitation of catheter ablation in ICD patients is that antiarrhythmic drugs are not routinely or systematically stopped after ablation.[47, 54] In the Thermocool trial, AAD were continued without change in 55%, reduced in 26%, and increased in 19%. At 6 month follow-up, only 35% were no longer taking AAD.[47] In contrast to arrhythmias such as atrial fibrillation, freedom from AAD therapy may not be a reasonable goal given the progressive nature of cardiomyopathies or ischemic heart disease, and VT recurrence may increase over longer follow-up.[55] It is generally recommended that patients continue the same AAD they receive before catheter ablation, however, reduction in drugs can be performed during follow-up. Major complications of VT ablation occur in 5-10% of patients and include a risk stroke, complete AV block, tamponade, valve injury, and myocardial infarction.[55, 56]

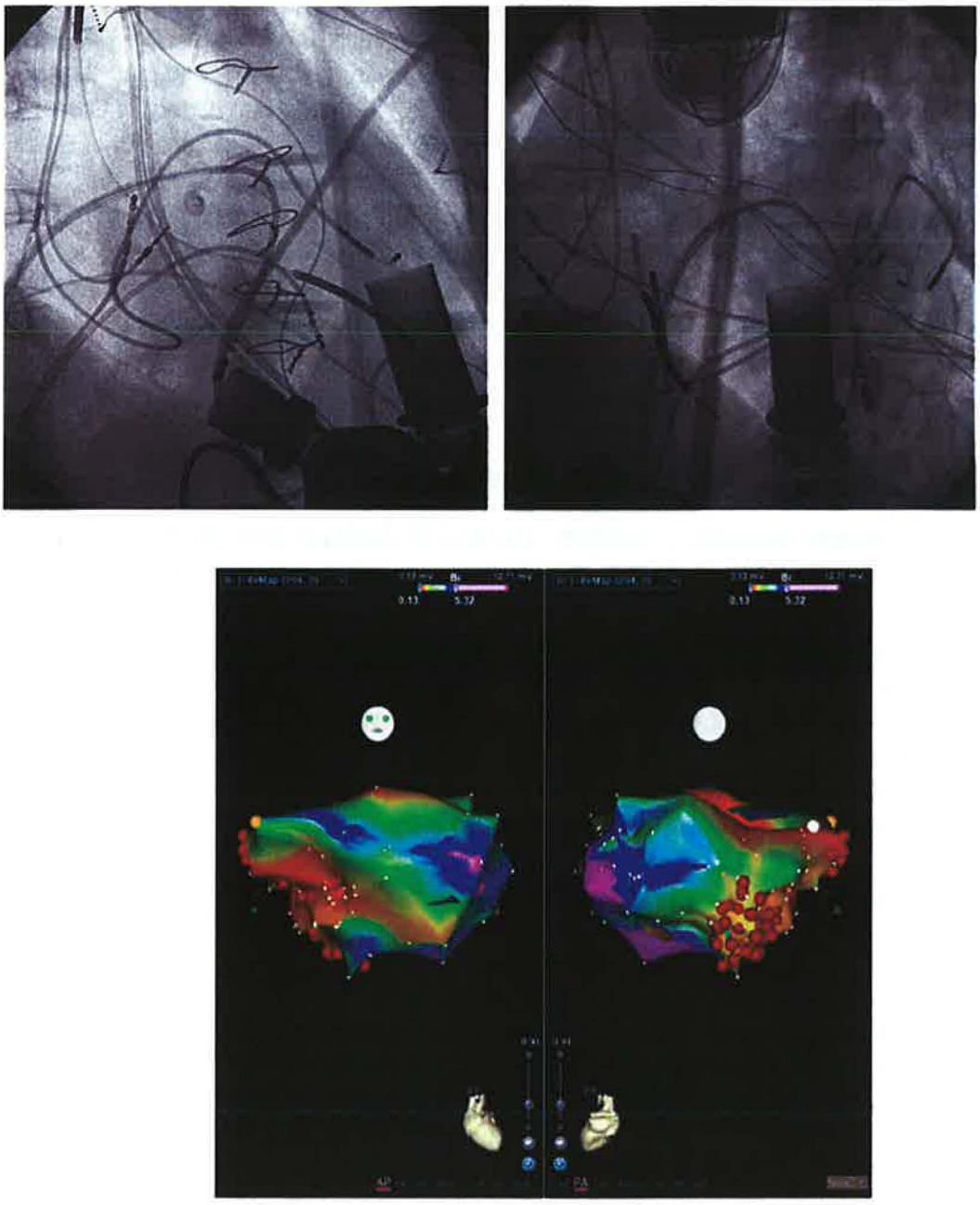


Figure 14 Fluoroscopic (X-ray image) of electrophysiology catheters (above) in patient with LVAD and electroantomical 3D voltage map (computerized reconstruction) of left ventricle (below). Ablation (tagged red dots) is performed around an area of scar (low voltage) where pacemapping mimics VT morphology. Color scale represents voltage (red-orange is lowest and purple is normal).

Catheter Ablation for Electrical Storm

Electrical storm (ES), characterized by frequent arrhythmia episodes (3 or more VT/VF episodes in 24 hours) resulting in appropriate ICD shocks, is associated with poor short- and long-term prognosis with mortality ranging from 27-54% over 2 years.[39, 40] Patients with history of CAD and those treated with amiodarone are more likely to have VT instead of VF as a cause of ES.[39] Catheter ablation (with sometimes multiple attempts required) can significantly reduce the number ICD intervention in patients with ES. In a large series of patients that underwent VT ablation for ES, 66% were free of VT recurrence over 22 months follow-up. Interestingly, even with inducible clinical VT at the end of the procedure not be suppressed by ablation, recurrent VT storm was suppressed in all for at least a week before hospital discharge. However, patients who acutely fail VT ablation appear to have a poor prognosis. [57]

Catheter Ablation of VT/VF before ICD interventions

Catheter ablation has been shown to significantly reduce ICD shocks in patients after they receive multiple ICD interventions or shocks. As noted above, ICD shocks are associated with increased mortality compared with patients that receive no shocks. To date, there are only two randomized controlled clinical trials which have addressed the impact of prophylactic VT ablation performed in patients with ischemic cardiomyopathy and a history of VT. The SMASH-VT trial examined whether catheter ablation in patients with a prior MI would reduce ICD therapies.[42] Enrolled patients had either a history of unstable VT/VF, syncope with inducible VT, or had recent ICD therapy for VT after a recent implant (within 6 months). The patients did not receive AAD and were randomized to ICD or ICD with catheter ablation. The catheter ablation (intervention) arm had a 73% reduction of ICD shocks compared to those who received standard therapy. There was a trend towards fewer deaths in the ablation group.

The VTACH trial randomized patients with hemodynamically stable VT and prior MI to ablation or conventional therapy (without ADD) prior to ICD implant.[58] By intention to treat analysis, patients in the ablation group had a longer time to first ICD therapy (18.6 vs 5.9 months) and greater freedom from VT or VF (47% vs 29%) at 2 years. Most of the benefit was seen in patients with LVEF > 30%. Importantly, catheter ablation reduced the median number of appropriate ICD interventions by 93% per patient, reduced the incidence of shocks by43% and reduced hospitalizations. The study was not sufficiently powered to detect a difference in mortality.

In both prophylactic VT ablation trials, the incidence of death due to an ablation procedure was 0% and major complication rate was < 5%.[42, 58]

Study	Patients, #n	EF (%)	Treatment	Acute Success, (%)	FU (mo)	Long-term success (%)	Mortality (%)
VTACH (Kuck) 2010	107 Active 52	34±10	VT Ablation+ICD vs	60%	22.5	47%	10%
	Control 55		ICD only			29%	7%
SMASH VT (Reddy) 2007	128 Active 64	32±10	VT Ablation+ICD vs	NA	22.5	88%	9%
	Control 64		ICD only			67%	17%
Thermocool (Stevenson) 2008	231	25	VT Ablation	49%	6	53%	18%
Cooled RF (Calkins) 2000	146	31±13	VT Ablation	41%	8	46%	25%
Tanner 2009	63	30±13	VT Ablation	81%	12	51%	9%

Table 3 Prospective randomized multi-center trials on catheter ablation of VT in patients with ischemic cardiomyopathy.

ACC/AHA Recommendations for Ventricular Tachycardia (VT) Ablation[7]

Class I (Conditions for which there is evidence and/or general agreement that a given procedure or treatment is beneficial, useful, and effective).

1. Ablation is indicated in patients who are otherwise at low risk for SCD and have sustained predominantly monomorphic VT that is drug resistant, who are drug intolerant, or who do not wish long-term drug therapy.
2. Ablation is indicated in patients with bundle-branch reentrant VT.
3. Ablation is indicated as adjunctive therapy in patients with an ICD who are receiving multiple shocks as a result of sustained VT that is not manageable by reprogramming or changing drug therapy or who do not wish long-term therapy.

Class IIa (Conditions for which there is conflicting evidence and/or divergence of opinion about the usefulness/efficacy of a procedure or treatment. Weight of evidence/opinion is in favor of usefull).

4. Ablation can be useful therapy in patients who are otherwise at low risk for SCD and have symptomatic nonsustained monomorphic VT that is drug resistant, who are drug intolerant, or who do not wish long-term drug therapy.
5. Ablation can be useful in patients who are otherwise low risk for SCD and have frequent symptomatic predominantly monomorphic PVCs that are drug resistant, who are drug intolerant, or do not wish long-term drug therapy.

Class IIb (Usefullness/efficacy is less well established by evidence or opinion).

1. Ablation of Purkinge fiber potentials may be considered in patient with ventricular arrhythmia storm consistently provoked by PVCs of similar morphology.
2. Ablation of asymptomatic PVCs may be considered when the PVCs are very frequent to avoid or treat tachycardia-induced cardiomyopathy.

Class III (Conditions for which there is evidence that a procedure/treatment is not useful/effective and in some cases may be harmful).

Ablation of asymptomatic relatively infrequent PVCs is not indicated.

Summary

Ventricular tachycardia (VT) remains a significant cause of morbidity and mortality, particularly in patients with ischemic and structural heart disease. Treatment for VT has progressed and evolved over 30 years and includes surgical, pharmacological, implantable cardioverter defibrillator (ICD) and catheter ablation therapies. Antiarrhythmic drugs have been shown to reduce the likelihood of VT occurrence or ICD shocks, but have either neutral or adverse effects on overall mortality. ICDs have become standard therapy for reducing mortality in patients with a history of ventricular arrhythmias or sudden death and are recommended for patients with systolic heart failure receiving optimal medical therapy. ICD therapy does not prevent the occurrence of VT, and ICD shocks are common in patients who undergo implant of these devices. ICD shocks from any cause are associated with an increase in mortality. Catheter ablation reduces VT recurrences and ICD shocks in patients who have a history of VT and appears to improve prognosis when successful.

References

1. Aliot EM, Stevenson WG, Almendral-Garrote JM, Bogun F, Calkins CH, Delacretaz E, Bella PD, Hindricks G, Jais P, Josephson ME, Kautzner J, Kay GN, Kuck KH, Lerman BB, Marchlinski F, Reddy V, Schalij MJ, Schilling R, Soejima K, Wilber D. 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 : European pacing, arrhythmias, and cardiac electrophysiology : journal of the working groups on cardiac pacing, arrhythmias, and cardiac cellular electrophysiology of the European Society of Cardiology*. 2009;11:771-817
2. Bayes de Luna A, Coumel P, Leclercq JF. Ambulatory sudden cardiac death: Mechanisms of production of fatal arrhythmia on the basis of data from 157 cases. *American heart journal*. 1989;117:151-159
3. Myerburg RJ. Sudden cardiac death: Exploring the limits of our knowledge. *Journal of cardiovascular electrophysiology*. 2001;12:369-381
4. Huikuri HV, Castellanos A, Myerburg RJ. Sudden death due to cardiac arrhythmias. *The New England journal of medicine*. 2001;345:1473-1482
5. Solomon SD, Zelenkofske S, McMurray JJ, Finn PV, Velazquez E, Ertl G, Harsanyi A, Rouleau JL, Maggioni A, Kober L, White H, Van de Werf F, Pieper K, Califf RM, Pfeffer MA. Sudden death in patients with myocardial infarction and left ventricular dysfunction, heart failure, or both. *The New England journal of medicine*. 2005;352:2581-2588
6. Klein RC, Raitt MH, Wilkoff BL, Beckman KJ, Coromilas J, Wyse DG, Friedman PL, Martins JB, Epstein AE, Hallstrom AP, Ledingham RB, Belco KM, Greene HL. Analysis of implantable cardioverter defibrillator therapy in the antiarrhythmics versus implantable defibrillators (avid) trial. *Journal of cardiovascular electrophysiology*. 2003;14:940-948
7. Zipes DP, Camm AJ, Borggrefe M, Buxton AE, Chaitman B, Fromer M, Gregoratos G, Klein G, Moss AJ, Myerburg RJ, Priori SG, Quinones MA, Roden DM, Silka MJ, Tracy C, Smith SC, Jr., Jacobs AK, Adams CD, Antman EM, Anderson JL, Hunt SA, Halperin JL, Nishimura R, Ornato JP, Page RL, Riegel B, Blanc JJ, Budaj A, Dean V, Deckers JW, Despres C, Dickstein K, Lekakis J, McGregor K, Metra M, Morais J, Osterspey A, Tamargo JL, Zamorano JL. Acc/aha/esc 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: A report of the american college of cardiology/american heart association task force and the european society of cardiology committee for practice guidelines (writing committee to develop guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death). *Journal of the American College of Cardiology*. 2006;48:e247-346
8. Josephson ME, Horowitz LN, Farshidi A, Kastor JA. Recurrent sustained ventricular tachycardia. 1. Mechanisms. *Circulation*. 1978;57:431-440
9. de Bakker JM, van Capelle FJ, Janse MJ, Tasseron S, Vermeulen JT, de Jonge N, Lahpor JR. Slow conduction in the infarcted human heart. 'Zigzag' course of activation. *Circulation*. 1993;88:915-926
10. de Chillou C, Lacroix D, Klug D, Magnin-Poull I, Marquie C, Messier M, Andronache M, Kouakam C, Sadoul N, Chen J, Aliot E, Kacet S. Isthmus characteristics of reentrant ventricular tachycardia after myocardial infarction. *Circulation*. 2002;105:726-731
11. Di Diego JM, Antzelevitch C. Ischemic ventricular arrhythmias: Experimental models and their clinical relevance. *Heart rhythm : the official journal of the Heart Rhythm Society*. 2011;8:1963-1968
12. Guiraudon G, Fontaine G, Frank R, Escande G, Etievent P, Cabrol C. Encircling endocardial ventriculotomy: A new surgical treatment for life-threatening ventricular tachycardias resistant to medical treatment following myocardial infarction. *The Annals of thoracic surgery*. 1978;26:438-444
13. Josephson ME, Harken AH, Horowitz LN. Endocardial excision: A new surgical technique for the treatment of recurrent ventricular tachycardia. *Circulation*. 1979;60:1430-1439
14. Marchlinski FE, Callans DJ, Gottlieb CD, Zado E. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. *Circulation*. 2000;101:1288-1296

15. Anter E, Hutchinson MD, Deo R, Haqqani HM, Callans DJ, Gerstenfeld EP, Garcia FC, Bala R, Lin D, Riley MP, Litt HI, Woo JY, Acker MA, Szeto WY, Zado ES, Marchlinski FE, Dixit S. Surgical ablation of refractory ventricular tachycardia in patients with nonischemic cardiomyopathy. *Circulation. Arrhythmia and electrophysiology*. 2011;4:494-500
16. Michowitz Y, Mathuria N, Tung R, Esmailian F, Kwon M, Nakahara S, Bourke T, Boyle NG, Mahajan A, Shivkumar K. Hybrid procedures for epicardial catheter ablation of ventricular tachycardia: Value of surgical access. *Heart rhythm : the official journal of the Heart Rhythm Society*. 2010;7:1635-1643
17. Haines DE, Lerman BB, Kron IL, DiMarco JP. Surgical ablation of ventricular tachycardia with sequential map-guided subendocardial resection: Electrophysiologic assessment and long-term follow-up. *Circulation*. 1988;77:131-141
18. Steinberg JS, Martins J, Sadanandan S, Goldner B, Menchavez E, Domanski M, Russo A, Tullo N, Hallstrom A. Antiarrhythmic drug use in the implantable defibrillator arm of the antiarrhythmics versus implantable defibrillators (avid) study. *American heart journal*. 2001;142:520-529
19. Patel C, Yan GX, Kocovic D, Kowey PR. Should catheter ablation be the preferred therapy for reducing icd shocks?: Ventricular tachycardia ablation versus drugs for preventing icd shocks: Role of adjuvant antiarrhythmic drug therapy. *Circulation. Arrhythmia and electrophysiology*. 2009;2:705-711; discussion 712
20. Preliminary report: Effect of encainide and flecainide on mortality in a randomized trial of arrhythmia suppression after myocardial infarction. The cardiac arrhythmia suppression trial (cast) investigators. *The New England journal of medicine*. 1989;321:406-412
21. Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest : The cardiac arrest study hamburg (cash). *Circulation*. 2000;102:748-754
22. Jessup M, Abraham WT, Casey DE, Feldman AM, Francis GS, Ganiats TG, Konstam MA, Mancini DM, Rahko PS, Silver MA, Stevenson LW, Yancy CW. 2009 focused update: Accf/aha guidelines for the diagnosis and management of heart failure in adults: A report of the american college of cardiology foundation/american heart association task force on practice guidelines: Developed in collaboration with the international society for heart and lung transplantation. *Circulation*. 2009;119:1977-2016
23. Brodine WN, Tung RT, Lee JK, Hockstad ES, Moss AJ, Zareba W, Hall WJ, Andrews M, McNitt S, Daubert JP. Effects of beta-blockers on implantable cardioverter defibrillator therapy and survival in the patients with ischemic cardiomyopathy (from the multicenter automatic defibrillator implantation trial-ii). *The American journal of cardiology*. 2005;96:691-695
24. Pacifico A, Hohnloser SH, Williams JH, Tao B, Saksena S, Henry PD, Prystowsky EN. Prevention of implantable-defibrillator shocks by treatment with sotalol. D,l-sotalol implantable cardioverter-defibrillator study group. *The New England journal of medicine*. 1999;340:1855-1862
25. Seidl K, Hauer B, Schwick NG, Zahn R, Senges J. Comparison of metoprolol and sotalol in preventing ventricular tachyarrhythmias after the implantation of a cardioverter/defibrillator. *The American journal of cardiology*. 1998;82:744-748
26. Kettering K, Mewis C, Dornberger V, Vonthein R, Bosch RF, Kuhlkamp V. Efficacy of metoprolol and sotalol in the prevention of recurrences of sustained ventricular tachyarrhythmias in patients with an implantable cardioverter defibrillator. *Pacing and clinical electrophysiology : PACE*. 2002;25:1571-1576
27. Connolly SJ, Dorian P, Roberts RS, Gent M, Bailin S, Fain ES, Thorpe K, Champagne J, Talajic M, Coutu B, Gronefeld GC, Hohnloser SH. 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 : the journal of the American Medical Association*. 2006;295:165-171
28. Piccini JP, Berger JS, O'Connor CM. Amiodarone for the prevention of sudden cardiac death: A meta-analysis of randomized controlled trials. *European heart journal*. 2009;30:1245-1253
29. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. The antiarrhythmics versus implantable defibrillators (avid) investigators. *The New England journal of medicine*. 1997;337:1576-1583
30. Connolly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon RS, Mitchell LB, Green MS, Klein GJ, O'Brien B. Canadian implantable defibrillator study (cids) : A randomized trial of the implantable cardioverter defibrillator against amiodarone. *Circulation*. 2000;101:1297-1302

31. Connolly SJ, Hallstrom AP, Cappato R, Schron EB, Kuck KH, Zipes DP, Greene HL, Boczor S, Domanski M, Follmann D, Gent M, Roberts RS. Meta-analysis of the implantable cardioverter defibrillator secondary prevention trials. Avid, cash and cids studies. Antiarrhythmics vs implantable defibrillator study. Cardiac arrest study hamburg . Canadian implantable defibrillator study. *European heart journal*. 2000;21:2071-2078
32. Moss AJ, Hall WJ, Cannom DS, Daubert JP, Higgins SL, Klein H, Levine JH, Saksena S, Waldo AL, Wilber D, Brown MW, Heo M. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. Multicenter automatic defibrillator implantation trial investigators. *The New England journal of medicine*. 1996;335:1933-1940
33. Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, Daubert JP, Higgins SL, Brown MW, Andrews ML. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *The New England journal of medicine*. 2002;346:877-883
34. Bardy GH, Lee KL, Mark DB, Poole JE, Packer DL, Boineau R, Domanski M, Troutman C, Anderson J, Johnson G, McNulty SE, Clapp-Channing N, Davidson-Ray LD, Fraulo ES, Fishbein DP, Luceri RM, Ip JH. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. *The New England journal of medicine*. 2005;352:225-237
35. Witkowski FX, Penkoske PA, Plonsey R. Mechanism of cardiac defibrillation in open-chest dogs with unipolar dc-coupled simultaneous activation and shock potential recordings. *Circulation*. 1990;82:244-260
36. Weber M, Block M, Bansch D, Castrucci M, Gradaus R, Schriever C, Breithardt G, Bocker D. Antitachycardia pacing for rapid vt during icd charging: A method to prevent icd shocks. *Pacing and clinical electrophysiology : PACE*. 2001;24:345-351
37. Mirowski M, Reid PR, Mower MM, Watkins L, Gott VL, Schauble JF, Langer A, Heilman MS, Kolenik SA, Fischell RE, Weisfeldt ML. Termination of malignant ventricular arrhythmias with an implanted automatic defibrillator in human beings. *The New England journal of medicine*. 1980;303:322-324
38. Poole JE, Johnson GW, Hellkamp AS, Anderson J, Callans DJ, Raitt MH, Reddy RK, Marchlinski FE, Yee R, Guarnieri T, Talajic M, Wilber DJ, Fishbein DP, Packer DL, Mark DB, Lee KL, Bardy GH. Prognostic importance of defibrillator shocks in patients with heart failure. *The New England journal of medicine*. 2008;359:1009-1017
39. Verma A, Kilicaslan F, Marrouche NF, Minor S, Khan M, Wazni O, Burkhardt JD, Belden WA, Cummings JE, Abdul-Karim A, Saliba W, Schweikert RA, Tchou PJ, Martin DO, Natale A. Prevalence, predictors, and mortality significance of the causative arrhythmia in patients with electrical storm. *Journal of cardiovascular electrophysiology*. 2004;15:1265-1270
40. Hohnloser SH, Al-Khalidi HR, Pratt CM, Brum JM, Tatla DS, Tchou P, Dorian P. Electrical storm in patients with an implantable defibrillator: Incidence, features, and preventive therapy: Insights from a randomized trial. *European heart journal*. 2006;27:3027-3032
41. Packer DL, Prutkin JM, Hellkamp AS, Mitchell LB, Bernstein RC, Wood F, Boehmer JP, Carlson MD, Frantz RP, McNulty SE, Rogers JG, Anderson J, Johnson GW, Walsh MN, Poole JE, Mark DB, Lee KL, Bardy GH. Impact of implantable cardioverter-defibrillator, amiodarone, and placebo on the mode of death in stable patients with heart failure: Analysis from the sudden cardiac death in heart failure trial. *Circulation*. 2009;120:2170-2176
42. Reddy VY, Reynolds MR, Neuzil P, Richardson AW, Taborsky M, Jongnarangsin K, Kralovec S, Sediva L, Ruskin JN, Josephson ME. Prophylactic catheter ablation for the prevention of defibrillator therapy. *The New England journal of medicine*. 2007;357:2657-2665
43. Sweeney MO, Sherfesee L, DeGroot PJ, Wathen MS, Wilkoff BL. Differences in effects of electrical therapy type for ventricular arrhythmias on mortality in implantable cardioverter-defibrillator patients. *Heart rhythm : the official journal of the Heart Rhythm Society*. 2010;7:353-360
44. Morady F, Harvey M, Kalbfleisch SJ, el-Atassi R, Calkins H, Langberg JJ. Radiofrequency catheter ablation of ventricular tachycardia in patients with coronary artery disease. *Circulation*. 1993;87:363-372
45. Stevenson WG, Khan H, Sager P, Saxon LA, Middlekauff HR, Natterson PD, Wiener I. Identification of reentry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. *Circulation*. 1993;88:1647-1670

46. Soejima K, Delacretaz E, Suzuki M, Brunckhorst CB, Maisel WH, Friedman PL, Stevenson WG. Saline-cooled versus standard radiofrequency catheter ablation for infarct-related ventricular tachycardias. *Circulation*. 2001;103:1858-1862
47. Stevenson WG, Wilber DJ, Natale A, Jackman WM, Marchlinski FE, Talbert T, Gonzalez MD, Worley SJ, Daoud EG, Hwang C, Schuger C, Bump TE, Jazayeri M, Tomassoni GF, Kopelman HA, Soejima K, Nakagawa H. Irrigated radiofrequency catheter ablation guided by electroanatomic mapping for recurrent ventricular tachycardia after myocardial infarction: The multicenter thermocool ventricular tachycardia ablation trial. *Circulation*. 2008;118:2773-2782
48. Delacretaz E, Stevenson WG. Catheter ablation of ventricular tachycardia in patients with coronary heart disease. Part ii: Clinical aspects, limitations, and recent developments. *Pacing and clinical electrophysiology : PACE*. 2001;24:1403-1411
49. Delacretaz E, Stevenson WG. Catheter ablation of ventricular tachycardia in patients with coronary heart disease: Part i: Mapping. *Pacing and clinical electrophysiology : PACE*. 2001;24:1261-1277
50. Soejima K, Suzuki M, Maisel WH, Brunckhorst CB, Delacretaz E, Blier L, Tung S, Khan H, Stevenson WG. Catheter ablation in patients with multiple and unstable ventricular tachycardias after myocardial infarction: Short ablation lines guided by reentry circuit isthmuses and sinus rhythm mapping. *Circulation*. 2001;104:664-669
51. Sacher F, Tedrow UB, Field ME, Raymond JM, Koplan BA, Epstein LM, Stevenson WG. Ventricular tachycardia ablation: Evolution of patients and procedures over 8 years. *Circulation. Arrhythmia and electrophysiology*. 2008;1:153-161
52. Josephson ME. Electrophysiology of ventricular tachycardia: An historical perspective. *Journal of cardiovascular electrophysiology*. 2003;14:1134-1148
53. Stevenson WG, Tedrow UB. Ablation for ventricular tachycardia during stable sinus rhythm. *Circulation*. 2012;125:2175-2177
54. Calkins H, Epstein A, Packer D, Arria AM, Hummel J, Gilligan DM, Trusso J, Carlson M, Luceri R, Kopelman H, Wilber D, Wharton JM, Stevenson W. Catheter ablation of ventricular tachycardia in patients with structural heart disease using cooled radiofrequency energy: Results of a prospective multicenter study. Cooled rf multi center investigators group. *Journal of the American College of Cardiology*. 2000;35:1905-1914
55. Natale A, Raviele A, Al-Ahmad A, Alfieri O, Aliot E, Almendral J, Breithardt G, Brugada J, Calkins H, Callans D, Cappato R, Camm JA, Della Bella P, Guiraudon GM, Haissaguerre M, Hindricks G, Ho SY, Kuck KH, Marchlinski F, Packer DL, Prystowsky EN, Reddy VY, Ruskin JN, Scanavacca M, Shivkumar K, Soejima K, Stevenson WJ, Themistoclakis S, Verma A, Wilber D. Venice chart international consensus document on ventricular tachycardia/ventricular fibrillation ablation. *Journal of cardiovascular electrophysiology*. 2010;21:339-379
56. Tanner H, Hindricks G, Volkmer M, Furniss S, Kuhlkamp V, Lacroix D, C DEC, Almendral J, Caponi D, Kuck KH, Kottkamp H. Catheter ablation of recurrent scar-related ventricular tachycardia using electroanatomical mapping and irrigated ablation technology: Results of the prospective multicenter euro-vt-study. *Journal of cardiovascular electrophysiology*. 2010;21:47-53
57. Carbucicchio C, Santamaria M, Trevisi N, Maccabelli G, Giraldi F, Fassini G, Riva S, Moltrasio M, Cireddu M, Veglia F, Della Bella P. Catheter ablation for the treatment of electrical storm in patients with implantable cardioverter-defibrillators: Short- and long-term outcomes in a prospective single-center study. *Circulation*. 2008;117:462-469
58. Kuck KH, Schaumann A, Eckardt L, Willems S, Ventura R, Delacretaz E, Pitschner HF, Kautzner J, Schumacher B, Hansen PS. Catheter ablation of stable ventricular tachycardia before defibrillator implantation in patients with coronary heart disease (vtach): A multicentre randomised controlled trial. *Lancet*. 2010;375:31-40
59. Wilkoff BL, Williamson BD, Stern RS, Moore SL, Lu F, Lee SW, Birgersdotter-Green UM, Wathen MS, Van Gelder IC, Heubner BM, Brown ML, Holloman KK. Strategic programming of detection and therapy parameters in implantable cardioverter-defibrillators reduces shocks in primary prevention patients: Results from the prepare (primary prevention parameters evaluation) study. *Journal of the American College of Cardiology*. 2008;52:541-550

True/False Questions

1. Amiodarone reduces the risk of death in patients with heart failure and ventricular tachycardia.

2. ICD shocks may occur in 1/3 patients who receive a device and ICD shocks from any cause are associated with an increased risk in overall mortality.

3. VT ablation may reduce the risk of ICD shocks when performed in ischemic cardiomyopathy patients with history of VT even before an ICD is implanted.

True/False Answers

1. False. Amiodarone reduces the risk of sudden cardiac death ~30% but has a neutral effect on all-cause mortality. ICD therapy reduces overall mortality by ~ 25% compared to amiodarone or standard therapy.[28, 34]

2. True. In the SCD-HeFT trial, over a median follow-up of 45 months, 33.2% of patients with heart failure randomly assigned to ICD therapy received at least 1 shock. Both appropriate (sustained VT or VF) and inappropriate ICD shocks (non-sustained VT, SVT or device malfunction) are associated with higher mortality (HR is 5.6 for appropriate shocks, 2.0 for inappropriate, and 11.3 for both shocks).[38, 59]

3. True. Clinical trials have shown VT ablation reduces the likelihood of ICD shocks ~75% in post MI patients who have sustained VT prior to or shortly after receiving ICD implant compared to those who receive only an ICD.[42, 58] Although, this is currently not recommended by the ACC/AHA (2006) guidelines for management of VT, ablation should be considered early in selected ischemic cardiomyopathy patients who are receiving an ICD for ventricular tachycardia.