Sudden Cardiac Arrest

Meeting the Challenge

The Joint Commission

Contents

* Source: American Heart Association, Inc.

Foreword

About 295,000 people suffer sudden cardiac arrest each year in the United States. Only 7%–8.5% survive. This is more than the number who die from AIDS, Alzheimers, assault with firearms, breast cancer, colon cancer, fires, motor vehicle accidents, prostate cancer and suicides combined. In fact, the number of people who die each year from sudden cardiac arrest is seven times the number who die from breast cancer.[1]

What is Cardiac Arrest?

Cardiac arrest is the abrupt loss of heart function in a person who may or may not have diagnosed heart disease. The time and mode of death are unexpected. It occurs instantly or shortly after symptoms appear. Each year about 295,000 emergency medical services-treated out-of-hospital cardiac arrests occur in the United States.

Cardiac arrest is caused when the heart's electrical system malfunctions, causing arrhythmias. The most common arrhythmia in cardiac arrest is ventricular fibrillation. Cardiac arrest may be reversed if CPR (cardiopulmonary resuscitation) is performed and a defibrillator is used to shock the heart and restore a normal heart rhythm within a few minutes. Sudden cardiac arrest may be caused by almost any known heart condition. The most common causes are:

- Scarring from a prior heart attack or other causes: A heart that is scarred or enlarged from any cause is prone to develop life-threatening ventricular arrhythmias. The first six months after a heart attack is a particularly high-risk period for sudden cardiac arrest in patients with atherosclerotic heart disease.
- A thickened heart muscle (cardiomyopathy) from any cause (typically high blood pressure or valvular heart disease)—especially coupled with heart failure
- Heart medications: Under certain conditions, various heart medications can set the stage for arrhythmias that cause sudden cardiac arrest. Paradoxically, antiarrhythmic drugs used to treat arrhythmias can sometimes produce lethal ventricular arrhythmias even at normally prescribed doses (a "proarrhythmic" effect).
- Electrical abnormalities: Certain electrical abnormalities such as Wolff-Parkinson-White syndrome and long QT syndrome may cause sudden cardiac arrest in children and young people.
- Blood vessel abnormalities: Less often, inborn blood vessel abnormalities, particularly in the coronary arteries and aorta, may be present in young sudden death victims. Adrenaline released during intense physical or athletic activity often acts as a trigger for sudden cardiac arrest when these abnormalities are present.
- Recreational drug use: In people without organic heart disease, recreational drug use is an important cause of sudden cardiac arrest.[2]

Decreasing the number of those who die from cardiac arrest is a significant challenge, dependent on excellence in care at multiple levels:

Prevention. Prevention of arrest among those known to be at risk is a significant cornerstone in saving lives. Intended for physician reference, Part I of this

publication presents the condensed, evidence-based guidelines of the American College of Cardiology/ American Heart Association/European Society of Cardiology formulated in 2006, and the guidelines of the American College of Cardiology/American Heart Association/Heart Rhythm Society formulated in 2008.

Bystander care. Prompt care at the time arrest occurs, whether in the hospital or in the community, has significant impact on the outcome of sudden cardiac arrest. Placement of automated external defibrillators, and training in their use, has been shown to save lives. Part II of this publication offers examples of such programs.

Follow-up care. Those who are successfully resuscitated from sudden cardiac arrest require excellence in care from hospitals and from supportive entities in the community. Part III of this publication offers an example of these resources.

References

1.   Abstracted from the Sudden Cardiac Arrest Foundation Web site, http://www.sca-aware.org (accessed May 24, 2011).
2.   Abstracted from the American Heart Association Web site, http://www.heart.org (accessed May 24, 2011).

This publication is not intended to offer medical advice; it is intended as a reference and resource for health professionals.

Part I. Prevention

Sudden Cardiac Arrest Prevention

Note: This material was originally developed and published in the American College of Cardiology/American Heart Association/European Society of Cardiology. 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). It, in addition to excerpts from the ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities, is most closely and directly related to prevention of sudden cardiac arrest from among the abundant cardiac literature. Little has been added to the original content; additions may be found in the accompanying list of selected references.

Numerous other guidelines and protocols related to management of acute myocardial infarction (MI), management of heart failure, insertion and use of pacing devices, resuscitation and post-resuscitation care, and related fields have not been included in an effort to focus review and avoid an exhaustive reflection of cardiac care. We recognize that, in the general sense, all cardiac care has a goal of prevention of sudden cardiac arrest.

Sudden Cardiac Arrest (SCA). Cardiac arrest is characterized by an abrupt loss of effective blood flow, sufficient to cause immediate loss of consciousness, leading immediately to death if untreated. The most common electrical mechanisms for cardiac arrest are ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT), but substantial numbers of cardiac arrests begin as severe bradyarrhythmias, asystole, or pulseless electrical activity. Survival probabilities are better for victims presenting with VT/VF than for those with bradyarrhythmic or asystolic mechanisms. A rapid response time is the major determinant of survival.

Incidence. Geographic incidence of sudden cardiac death (SCD) varies as a function of the prevalence of coronary heart disease (HD) in geographic regions and, to some extent, the availability of emergency treatment and presence or absence of CPR training and automated external defibrillators (AEDs). Death estimates from SCD for the United States range from 200,000 to more than 450,000 annually, with the most widely used range of 300,000 to 350,000 SCDs annually. The variation is based, in part, on the inclusion criteria used in individual studies. The event rate for adults is 100 per 100,000 patient years.

SCD is not a reportable condition; fewer than 24 U.S. communities report survival rates from ventricular fibrillation, the most common rhythm associated with cardiac arrest. Selected examples range from 0% in Detroit and 3% in Chicago to 46% in Seattle and King County, Washington. Las Vegas casinos also report a high survival rate owing to the presence of monitors, AEDs, and trained personnel. Early defibrillation is key to survival; there is a 7%–10% decline in survival rate for each minute

without CPR/Defib. There are also recent reports of increased survival rates when a minimally interrupted cardiac resuscitation (MICR) protocol is used for out-of-hospital cardiac arrest.

Two studies published in the February 2002 *New England Journal of Medicine* demonstrated improved survival and neurological outcomes with induction of mild therapeutic hypothermia for comatose survivors of out-of-hospital cardiac arrest. The Hypothermia After Cardiac Arrest Study Group showed that, when applied to unconscious out-of-hospital cardiac arrest patients with return of spontaneous circulation (ROSC) (*n* = 274), mild hypothermia (cooling to 32–34ºC) provided significant improvement in functional recovery at hospital discharge (55% vs. 39%) (number needed to treat [NNT] = 6) and lower 6-month mortality rate when compared with patients who were not cooled (41% vs. 55%) (NNT = 7). The NNT is very low and comparable to other important emergent treatments such as cardiac catheterization for acute coronary syndrome. Bernard examined endpoint of survival to hospital discharge to home or a rehabilitation facility (good outcome) in 77 patients and demonstrated 49% in the hypothermia group compared with 26% in the normothermic group. Recent literature reports indicate that, increasingly, cities such as Boston, Seattle, and New York are requiring rescue personnel to take cardiac arrest victims to hospitals with cooling capabilities, rather than to the nearest hospital.

Populations: Subgroups and Risk Prediction. Overall incidence of SCD in the United States is 1–2 per 1,000 population. However, as the population is sub-grouped by known risk, the event incidence is greater but the number of events smaller. At least 50% of all SCDs due to coronary heart disease (CHD) occur as a first clinical event or among subgroups thought to be at relatively low risk.

Time-Dependent Risk. The risk of SCD is not linear as a function of time. Survival curves after major cardiovascular events demonstrate that the most rapid rate of attrition usually occurs in the first 6–18 months after the index event, such as recent MI or SCA survival. Mortality is highest in the first month after acute MI.

Age, Heredity, Gender, Race. The incidence of SCD parallels the increase in incidence of CHD with advancing age, but declines in the eighth decade due to competing causes of death. The incidence is 100-fold less in those 30 and younger than for those older than 35, but the proportion of coronary deaths and of all cardiac deaths that are sudden is highest in the younger age group.

Hereditary factors influencing plaque destabilization, thrombosis, and arrhythmogenesis have been identified. Studies suggest that SCD as a manifestation of CHD clusters in families. There is a preponderance of SCD in males in the young and early middle-age years, and among females the SCD risk increases proportionately with the post-menopausal state. The incidence of SCD at any age is greater in men than in women. Studies comparing risk of SCD among African-Americans and whites have yielded conflicting findings but some studies have demonstrated excess risk among African-Americans. SCD rates among Hispanics were lower.

Risk Profiles. Risk profiling for coronary artery disease is of some, but limited, value. Destabilization of lipid plaque, measured by markers such as C-reactive protein levels, has entered into risk factor equations, and familial clustering of SCD may lead to recognition of genetic factors that predispose toward SCD. Established risk factors for SCD are:
- Hypertension
- Left ventricular hypertrophy

- Left bundle branch block
- Cigarette smokers at 2- to 3-fold risk
- Survivors of SCA
- Obesity
- High resting heart rate with little change during exercise and recovery
- Varying associations between activity levels and SCD, but habitual exercise attenuates the risk
- Increase in social and economic stressors

Economic stressors, of themselves, may play a role in the incidence of SCA/SCD. Maintenance dose of cardiac medications may be skipped by those with limited economic resources, as access to care may be limited for the same population. Recent reports have also identified antidepressant use among women as another risk factor for both CHD and SCD, while another meta-analysis suggests anger and hostility associated with adverse outcomes of CHD.

Mechanisms and Substrates

Substrates for ventricular arrhythmias. Studies suggest that 75% of those with SCD have CHD, with a high percentage of those having 3- or 4-vessel disease. There are also acute changes in coronary plaques morphology, such as thrombus or plaque disruption or both, in more than 50% of SCD cases. Plaque rupture is frequently found, especially in older women.

The substrate will be different depending on the nature of the heart disease. Obesity, hypertension, lipid abnormalities, and diabetes are important risk factors. Right ventricular cardiomyopathy and hypertrophic cardiomyopathy are the major substrates found in the sudden deaths of pre-coronary age groups. The cumulative risk of SCD for those with aortic stenosis is 15% to 20%; reported rates in those with Wolff-Parkinson-White syndrome are 0.15%.

Genetic influences on the risk of SCD vary.
- Family members of those experiencing SCA and offspring of SCA patients exhibit higher risk of SCA.
- In 5% to 10% of cases, SCD occurs in the absence of CHD or cardiomyopathy.
- There exists a group of inherited abnormalities such as the long QT syndrome (LQTS), short QT syndrome (SQTS), Brugada syndrome, and catecholaminergic VT, which can precipitate SCD without overt structural changes in the heart.
- Other abnormalities in metabolism can disrupt the normal electrical processes of the heart to cause life-threatening ventricular arrhythmias.
- Some individuals can have inherited abnormalities that are not manifest until triggered by an external event. For example, autonomic modulation associated with certain types of activity, as well as drugs that affect cardiac repolarization, can convert a subclinical genetic abnormality to SCD.
- The most common genetic factors are DNA variants called "polymorphisms" that may be present in a large proportion of the population and create susceptibility for SCD. Single nucleotide polymorphisms (SNPs) are DNA variants that can be associated with a functional consequence. For example, a polymorphism identified in the alpha 2b adrenergic receptor is associated with an increased risk of MI and SCD.

Mechanisms of Sudden Cardiac Death. The rhythm most often recorded at the time of sudden cardiac arrest is VF. Previous studies suggest that 75% to 80% occur via this mechanism and 15% to 20% are attributed to bradyarrhythmias, including advanced atrioventricular (AV) block and asystole. While many studies have investigated EP mechanisms re-

sponsible for the onset of VT and VF and their continuation, no class I or III antiarrhythmic agent has clearly been demonstrated to reduce total and SCD mortality in patients at risk for SCD. In fact, it is the drugs without direct EP actions on cardiac muscle or specialized conducting tissue that have been shown effective for prevention of SCD. These drugs include beta blockers, ACE inhibitors, angiotensin receptor–blocking agents, lipid-lowering agents, spironolactone, and fibrinolytic and antithrombotic agents.

Clinical Presentations of Patients with Ventricular Arrhythmias and Sudden Cardiac Death

Asymptomatic: Ventricular arrhythmias may be detected as an incidental finding during ECG monitoring or physical examination or may also be uncovered during an attempt to further define prognosis in an individual with known heart disease. In general, treatment is indicated to prevent potential morbidity (e.g., "tachycardia-induced cardiomyopathy"), reduce symptom burden, or reduce the risk of SCD. There is no reason to treat asymptomatic ventricular arrhythmias in the absence of such potential benefit. The major determinants of risk of SCD are related more to the type and severity of associated cardiac disease and less to the frequency or classification of ventricular arrhythmia. Non-sustained ventricular tachycardia (NSVT) in the patient with previous MI and impaired left ventricular (LV) function indicates increased risk of SCD and the need for further evaluation or treatment. The contribution of asymptomatic ventricular arrhythmias to the patient's management is not well established for other cardiac diseases such as dilated cardiomyopathy or hypertensive cardiomyopathy.

Hemodynamically Stable Ventricular Tachycardia: Patients with slower, stable VT may be asymptomatic but more frequently present with a sensation of rapid heart beating possibly accompanied by dyspnea or chest discomfort. The stability or tolerance of VT is related to the rate of tachycardia, presence of retrograde conduction, ventricular function, and the integrity of peripheral compensatory mechanisms. A presentation with stable, relatively well-tolerated VT does not suggest the absence of heart disease and can be observed in patients with very poor LV function. Incessant VT, although hemodynamically stable, can be a cause of hemodynamic deterioration leading to heart failure (HF). In patients with an implantable cardioverter-defibrillator (ICD), the VT rate can fall below the lower rate of VT detection, causing under-detection of VT that can prevent arrhythmia termination. Immediate reinitiation of the VT following proper ICD therapy can also result in hemodynamic deterioration and early battery depletion.

Hemodynamically Unstable Ventricular Tachycardia: The term *hemodynamically unstable* has not been rigidly defined but connotes a tachycardia associated with hypotension and poor tissue perfusion that is considered to have the imminent potential to lead to cardiac arrest or shock if left untreated. Patients with normal ventricular function can have unstable VT or VF if the tachycardia is rapid enough, as in the LQTS and other abnormal repolarization syndromes.

Key

***** indicates that guidelines are available on companion documents

LOE: Level of Evidence of available guidelines:

Level of Evidence A: Data derived from multiple randomized clinical trials or meta-analyses.

Level of Evidence B: Data derived from a single randomized trial or nonrandomized studies.

Level of Evidence C: Only consensus opinion of experts, case studies, or standard-of-care.

Evaluation of Patients with Documented or Suspected Ventricular Arrhythmias

History and Physical: Palpitations, presyncope, and syncope are the three most important symptoms requiring further characterization in patients suspected of having ventricular arrhythmias. Other symptoms related to underlying structural heart disease may also be present, especially chest discomfort, dyspnea, and fatigue. A thorough drug history including dosages used must be included in the evaluation of patients suspected of having ventricular arrhythmias. A positive family history of SCD is a strong independent predictor of susceptibility to ventricular arrhythmias and SCD, as noted earlier. Physical examination is often unrevealing in patients suspected of having ventricular arrhythmias unless the arrhythmia occurs while the patient is being examined.

Noninvasive evaluation

Resting ECG   *****   LOE: A

A standard resting 12-lead electrocardiogram (ECG) allows not only identification of various congenital abnormalities associated with ventricular arrhythmias and SCD (e.g., LQTS, SQTS, Brugada syndrome, arrhythmogenic right ventricular cardiomyopathy [ARVC]) but also identification of various other ECG parameters, such as those due to electrolyte disturbances, or evidence suggesting underlying structural disease, such as bundle-branch block, AV block, ventricular hypertrophy, and Q waves indicative of ischemic heart disease or infiltrative cardiomyopathy. QRS duration and repolarization abnormalities are both independent predictors of SCD.

Exercise testing   *****   LOE: B,C

Exercise-ECG is commonly used in the evaluation of patients with ventricular arrhythmias. Its most common application is for detection of silent ischemia in patients suspected of having underlying CHD. In patients with known or silent CHD or cardiomyopathies, the presence of frequent premature ventricular contractions (PVCs) during or after exercise has been associated with greater risk for serious cardiovascular events but not specifically SCD. Exercise-induced PVCs in apparently normal individuals should not be used to dictate therapy unless associated with documented ischemia or sustained VT. With the exception of beta blockers, at the present time the use of antiarrhythmic drugs to abolish exercise-induced PVCs has not been proved to be effective in reducing SCD.

Although the safety of supervised exercise testing is well established, less data are available in patients at risk for serious ventricular arrhythmias. In one series, exercise testing in patients with life-threatening ventricular arrhythmias was associated with a 2.3% incidence of arrhythmias requiring cardioversion, intravenous drugs, or resuscitation. Such an exercise study may still be warranted because it is better to expose arrhythmias and risk under controlled circumstances. Exercise testing should be performed where resuscitation equipment and trained personnel are immediately available.

Ambulatory ECG   *****   LOE: A,B

The use of continuous or intermittent ambulatory recording techniques can be very helpful in diagnosing a suspected arrhythmia, establishing its frequency, and relating symptoms to the presence of the arrhythmia. Silent myocardial ischemic episodes may also be detected. A 24- to 48-hour continuous Holter recording is appropriate whenever the arrhythmia is known or suspected to occur at least once a day. For sporadic episodes producing palpitations, dizziness, or syncope, conventional event monitors are more appropriate because they can record over extended periods of time.

New implantable recorders are capable of monitoring the rhythm and can record on patient activation or automatically for prespecified criteria. Although these devices require surgical implantation, they have been shown to be extremely useful in diagnosing serious tachyarrhythmias and bradyarrhythmias in patients with life-threatening symptoms such as syncope.

ECG Techniques and Measurements *****
LOE: A,B

Only two modalities are currently approved by the U.S. Food and Drug Administration (FDA) for SCD risk: signal-average electrocardiogram (SAECG) and T-wave alternans (TWA). SAECG improves the signal-to-noise ratio of a surface ECG, permitting the identification of low-amplitude (microvolt level) signals at the end of the QRS complex referred to as "late potentials." Late potentials indicate regions of abnormal myocardium demonstrating slow conduction, a substrate abnormality that may allow for reentrant ventricular arrhythmias, and they are believed to serve as a marker for the presence of an electrophysiological (EP) substrate for reentrant ventricular tachyarrhythmias. The presence of an abnormal SAECG was shown to increase the risk of arrhythmic events by six- to eight-fold in a post-MI setting; however, there has been a noticeable reduction in the predictive power of this tool. SAECG in isolation, therefore, is no longer useful for the identification of post-MI patients at risk of ventricular arrhythmias. A high negative predictive value of 89% to 99% rendered the SAECG a useful tool with which to exclude a wide-complex tachycardia as a cause of unexplained syncope.

TWA, which is a fluctuation in the amplitude or morphology of the T-wave that alternates every other beat assessed during exercise testing or atrial pacing, has been shown to be an effective tool for identifying high-risk patients post-MI and in the presence of ischemic or nonischemic cardiomyopathy. This association appears to be independent of ejection fraction (EF) and equally strong in patients with ischemic and nonischemic cardiomyopathy.

Left Ventricular Function and Imaging *****
LOE: B,C

Echocardiograph: Echocardiography is the imaging technique that is most commonly used because it is inexpensive in comparison with other techniques such as MRI and cardiac CT, it is readily available, and it provides accurate diagnosis of myocardial, valvular, and congenital heart disorders associated with ventricular arrhythmias and SCD. In addition, LV systolic function and regional wall motion can be evaluated and, in a majority of patients, EF can be determined. Echocardiography is therefore indicated in patients with ventricular arrhythmias suspected of having structural heart disease and in the subset of patients at high risk for the development of serious ventricular arrhythmias or SCD, such as those with dilated, hypertrophic or RV cardiomyopathies, AMI survivors, or relatives of patients with inherited disorders associated with SCD. The combination of echocardiography with exercise or pharmacological stress (commonly known as "stress echo") is applicable to a selected group of patients who are suspected of having ventricular arrhythmias triggered by ischemia and who are unable to exercise or have resting ECG abnormalities that limit the accuracy of ECG for ischemia detection.

Cardiac MRI: This imaging technique to evaluate both the structure and function of the beating heart. The excellent image resolution obtained with current techniques allows for the accurate quantification of chamber volumes, LV mass, and ventricular function. This is of particular value to patients with

suspected arrhythmogenic RV cardiomyopathy (ARVC), in whom MRI provides excellent assessment of RV size, function, and regional wall motion and, importantly, may allow the detection of fatty infiltration within the RV myocardium.

Cardiac CT: These systems allow precise quantification of LV volumes, EF, and LV mass with results comparable to MRI but in addition provide segmental images of the coronary arteries from which the extent of calcification can be quantified. The majority of cardiac disorders associated with serious ventricular arrhythmias or SCD are assessed well with echocardiography. Cardiac CT can be used in selected patients in whom evaluation of cardiac structures is not feasible with echocardiography and MRI is not available.

Radionuclide tests: Myocardial perfusion SPECT using exercise or pharmacological agents is applicable for a selected group of patients who are suspected of having ventricular arrhythmias triggered by ischemia and who are unable to exercise or have resting ECG abnormalities that limit the accuracy of ECG for ischemia detection. Myocardial perfusion SPECT can also be used to assess viability in patients with LV dysfunction due to prior MI.

Coronary angiography: In patients with life-threatening ventricular arrhythmias or in survivors of SCD, coronary angiography plays an important diagnostic role in establishing or excluding the presence of significant obstructive coronary artery disease and it is commonly used as part of the workup.

Electrophysiological testing: EP testing is used to document the inducibility of VT, guide ablation, evaluate drug effects, assess the risks of recurrent VT or SCD, evaluate loss of consciousness in selected patients with arrhythmias suspected as a cause, and

assess the indications for ICD therapy. To evaluate patients with ventricular arrhythmias, most centers use 8 ventricular stimuli at drive cycle lengths between 600 and 400 ms at the RV apex, at twice diastolic threshold and the pulse duration of 0.5 to 2 ms, delivering 1 to 3 ventricular extrastimuli at baseline. This test may be repeated during isoproterenol infusion.

In patients with coronary heart disease *****
                                          LOE: B,C

In patients with CHD, asymptomatic NSVT, and an EF less than 40%, inducibility of sustained VT ranges between 20% and 40%. Inducibility conferred a worse prognosis. Inducibility identifies patients at high risk of subsequent VT and that the absence of inducibility indicated a low risk. However, these patients had a high rate of percutaneous revascularization. In CHD patients with a low EF (less than 30%), noninducibility does not portend a good prognosis. Persistent inducibility while receiving antiarrhythmic drugs predicts a worse prognosis. Patients in whom amiodarone suppressed VT inducibility or slowed VT to a mean cycle length of greater than 400 ms had 30% higher mortality compared with patients who did not respond to amiodarone and had an ICD placed instead. EP-guided antiarrhythmic drug effectiveness in patients with NSVT who had induced sustained VT conferred no benefit.

In patients with dilated cardiomyopathy: In DCM, EP testing plays a minor role in the evaluation and management of VT.

In repolarization anomalies due to genetic arrhythmia syndromes:
   • *Long QT Syndrome:* EP testing has not proved useful in LQTS.

- *Brugada Syndrome:* The role of EP testing for risk stratification in Brugada syndrome is debated.
- *Hypertrophic Cardiomyopathy:* The value of EP testing in HCM has been controversial.
- *Arrhythmogenic Right Ventricular Cardiomyopathy:* The prognostic role of EP testing in patients presenting with isolated PVCs or NSVT is not known.

In patients with Outflow Tract Ventricular Tachycardia: EP testing for the evaluation of outflow tract VT is motivated by the need to establish precise diagnosis to guide curative catheter ablation.

In patients with syncope: ***** LOE: B
Syncope is a transient symptom that may be caused by an underlying rhythm disorder with or without an associated cardiac disease. EP testing is used to document or exclude the arrhythmic cause of syncope. It is most useful in patients with CHD and LV dysfunction.

When bradycardia is suspected: EP testing can be used to document or provoke bradyarrhythmias or AV block when other investigations have failed to provide conclusive information. The diagnostic yield varies greatly with the selected patient populations. EP testing is more useful in the presence of structural heart disease. The diagnostic yield in the absence of structural heart disease or abnormal ECG is low.

When supraventricular tachyarrhythmia is suspected: The role of EP testing is to document the type of tachyarrhythmia and to guide management of patients. In a mixed population, the diagnostic yield of EP testing was 5%.

When ventricular tachycardia is suspected: Syncope in patients with structural heart disease and, in particular, significant LV dysfunction is ominous. NSVT on Holter monitoring, syncope, and structural heart disease are highly sensitive for predicting the presence of inducible VT. Syncope associated with heart disease and reduced EF has high recurrence and death rates, even when EP testing results are negative. EP testing is useful in patients with LV dysfunction due to prior MI (EF less than 40%) but not sensitive in patients with nonischemic cardiomyopathy. Induction of polymorphic VT or VF, especially with aggressive stimulation techniques, is not specific. In CHD, the diagnostic yield may reach 50%. In HCM, EP testing is not diagnostic in the majority of patients.

Therapies for Ventricular Arrhythmias

General Management: The selection of appropriate therapy for the management of ventricular arrhythmias (PVCs, NSVT, sustained monomorphic and polymorphic VT, and ventricular flutter/VF) necessitates an understanding of the etiology and mechanism of the arrhythmia, an appreciation of the associated medical conditions that may contribute to and/or exacerbate the arrhythmia, the risk posed by the arrhythmia, and risk-to-benefit aspects of the selected therapy. Management of the manifest arrhythmia may involve discontinuation of offending proarrhythmic drugs, specific antiarrhythmic therapy with drugs, implantable devices, ablation, and surgery.

Drug Therapy: With the exception of beta blockers, the currently available antiarrhythmic drugs have not been shown in randomized clinical trials to be effective in the primary management of patients with life-threatening ventricular arrhythmias or in the prevention of SCD. As a general rule, antiarrhythmic agents may be effective as adjunctive therapy in the management of arrhythmia-prone patients under

special circumstances. Because of potential adverse side effects of the available antiarrhythmic drugs, these agents must be used with caution.

Many marketed cardiac and noncardiac drugs prolong ventricular repolarization and have the potential to precipitate life-threatening ventricular tachyarrhythmias. Once it is appreciated that a patient's ventricular arrhythmia may be due to QT prolongation from one or more prescribed medications, the possible offending therapies should be discontinued and appropriate follow-up monitoring of ventricular repolarization and cardiac rhythm should be carried out.

Antiarrhythmic Drugs
Value of Antiarrhythmics
Beta blockers: These drugs are effective in suppressing ventricular ectopic beats and arrhythmias as well as in reducing SCD in a spectrum of cardiac disorders in patients with and without HF. Beta blockers are safe and effective antiarrhythmic agents that can be considered the mainstay of antiarrhythmic drug therapy. The mechanism of antiarrhythmic efficacy of this class of drugs involves competitive adrenergic-receptor blockade of sympathetically mediated triggering mechanisms, slowing of the sinus rate, and possibly inhibition of excess calcium release by the ryanodine receptor.

Amiodarone and Sotalol: Amiodarone has a spectrum of actions that includes block of potassium repolarization currents that can inhibit or terminate ventricular arrhythmias by increasing the wavelength for reentry. The overall long-term survival benefit from amiodarone is controversial, with most studies showing no clear advantage over placebo. A few studies and one meta-analysis of several large studies have shown reduction in SCD using amiodarone for LV dysfunction due to prior MI and nonischemic DCM. But the SCD-HeFT trial showed no survival benefit from amiodarone compared with placebo. Sotalol, like amiodarone, is effective in suppressing ventricular arrhythmias, but it has greater proarrhythmic effects and has not been shown to provide a clear increase in survival; worsening ventricular arrhythmias occur in 2% to 4% of treated patients.

Efficacy of antiarrhythmic drugs: Overall, the available antiarrhythmic drugs other than beta blockers should not be used as primary therapy in the management of ventricular arrhythmias and the prevention of SCD. The efficacy of non-beta-blocker antiarrhythmic drugs is equivocal at best, and each drug has significant potential for adverse events including proarrhythmia.

Special Considerations Where Antiarrhythmic Drugs Might Be Indicated
Patients with v-tach who do not meet criteria for implantable cardioverter-defibrillator (ICD): Beta blockers are the first-line therapy, but if this therapy at full therapeutic dose is not effective, then amiodarone or sotalol can be tried with monitoring for adverse effects during administration.

Patients with ICD with frequent appropriate ICD Firing: This scenario, in its extreme, has been called defibrillator (tachycardia) storm, and it requires the addition of antiarrhythmic drugs and/or catheter ablation for control of the recurrent VT and associated ICD shocks. Sotalol is effective in suppressing atrial and ventricular arrhythmias; the combination of beta blockers and amiodarone is an alternative approach. Because many such patients have low EF and poor renal function, amiodarone and beta blockers rather than sotalol can be the first-line therapy for defibrillator storm.

Patients with ICD who have paroxysmal or chronic atrial fib with rapid rates and inappropriate ICD

firing: Combination therapy with a beta blocker and/or a calcium channel blocker is useful. Amiodarone has been used off-label for rate control if other therapies are contraindicated, not tolerated, or ineffective. Ablation of the AV node may be required when pharmacological therapy is not effective.

Nonantiarrhythmic Drugs

Electrolytes: Administration of potassium and magnesium, either as intravenously in the acute setting or orally for chronic augmentation in the blood levels of these electrolytes, can favorably influence the EP substrate involved in ventricular arrhythmias.

Antithrombins/antiplatelets: In a retrospective analysis of more than 6,700 patients participating in the Studies Of Left Ventricular Dysfunction (SOLVD) prevention and treatment trials, antithrombin therapy was associated with reduction in SCD.

n-3 fatty acids and lipids: Increasing experimental and clinical evidence suggests that n-3 fatty acids are antiarrhythmic and may prevent SCD in humans. However, data are conflicting. Findings indicate that statins reduce the occurrence of life-threatening ventricular arrhythmias in high-risk cardiac patients with electrical instability.

Implantable and External Cardioverter Devices

Implantable Cardioverter-Defibrillator: Several clinical trials have documented improved survival with ICD therapy in high-risk patients with LV dysfunction due to prior MI and nonischemic cardiomyopathy. ICD therapy, compared with conventional or traditional antiarrhythmic drug therapy, has been associated with mortality reductions from 23% to 55%, with the improvement in survival due almost exclusively to a reduction in SCD. Multiple publications contain guidelines for ICD implantation.

However, there are inconsistencies among guidelines regarding the EF cutoff used in the recommendations.

In a recent review of gender and ethnicity differences among the ICD population, findings were that ICD use among those eligible was:

> Black women: 28%
> White women: 29.8%
> Black men: 33.4%
> White men: 43.6%

Automated External Defibrillator: The automated external defibrillator (AED) saves lives when external defibrillation can be rendered within minutes of onset of VF. The AED represents an efficient method of delivering defibrillation to persons experiencing out-of-hospital cardiac arrest, and its use by both traditional and nontraditional first responders appears to be safe and effective. Eight percent of cardiac arrests occur in the home, and home placement may be of value for those with certain genetic arrhythmias.

Wearable Automatic Defibrillator: The wearable automatic defibrillator has been approved in the United States by the FDA for cardiac patients with a transient high risk for VF such as those awaiting cardiac transplantation, those at very high risk after a recent MI or an invasive cardiac procedure, or those requiring temporary removal of an infected implanted defibrillator for antibiotic therapy.

Ablation ***** LOE: B,C
Catheter Ablation-Background: RF ablation can be applied in the treatment of VT in patients with LV dysfunction due to prior MI, cardiomyopathy, bundle-branch reentry, and various forms of idiopathic VT.

No Apparent Structural Heart Disease: Patients with no overt structural heart disease account for a small percentage of patients with VT and are of particular interest for ablation therapy as this technique may be curative. These typically present as a single VT arising from the RV with a left bundle-branch block (LBBB) inferior axis morphology or from the LV with a right bundle-branch block (RBBB) morphology and, in general, are associated with a good prognosis.

Bundle-Branch Reentrant VT: Bundle-branch reentrant VT is often associated with cardiomyopathy. RF catheter ablation of the bundle branches is curative of the arrhythmia but not of the underlying structural abnormality. Because of the severity of underlying heart disease and the high prevalence of conduction abnormalities, adjunct device therapy should be strongly considered in these patients.

Structural Heart Disease: VT is a common complication of structural heart disease and carries significant risk for mortality in CHD patients with low EF. In those with extensive structural abnormalities, multiple morphologies of VT are often present. As a result, ablation of a single VT morphology can provide palliation but will not eliminate the need for device or antiarrhythmic therapy.

Additional Ablation Tools: Saline-irrigated cooled-tip catheters have been developed and used in VT ablation for those with deep lesions. Other techniques are considered highly experimental.

Surgery and Revascularization Procedures: Surgical therapy for the management of ventricular arrhythmias may involve ablation or surgical resection of an arrhythmogenic focus, cardiac sympathectomy, or aneurysm resection. Surgical or percutaneous coronary revascularization with improved coronary blood flow and reduction in myocardial ischemia has favorable antiarrhythmic effects.

Antiarrhythmic surgery: In patients with recurrent VT refractory to drugs, implanted defibrillators, and RF catheter ablation, direct surgical ablation or resection of the arrhythmogenic focus is an approach that continues to be used in experienced centers, but few reports are available to evaluate risk-to-benefit considerations in the current era in patients refractory to catheter ablation and implanted defibrillators.

Left cervicothoracic sympathetic ganglionectomy is associated with reduction in the frequency of arrhythmogenic syncope in this syndrome and may be useful as adjunctive therapy in high-risk LQTS patients who have recurrent syncope and/or aborted cardiac arrest despite combined ICD and beta-blocker therapy or in LQTS patients who cannot tolerate beta blockers.

Revascularization for Arrhythmia Management: In patients with ventricular arrhythmias, assessment for the presence of obstructive coronary disease and active ischemia is essential. Coronary revascularization involving either percutaneous balloon/stent angioplasty or bypass surgery is effective anti-ischemic therapy. No controlled trials have evaluated the effects of myocardial revascularization on VT or VF. In patients undergoing revascularization surgery following aborted cardiac arrest unrelated to an AMI, it is reasonable to implant a defibrillator after revascularization surgery in view of the assumed high-risk state. However, it is reasonable not to implant an ICD if there was direct, clear evidence of myocardial ischemia immediately preceding the onset of VF and there was no evidence for prior MI.

Acute Management of Specific Arrhythmias *****
                                                    LOE: A,B,C

Management of Cardiac Arrest: A decrease in cardiac arrest survival occurs at about 7% to 10% per minute if no CPR is initiated and at 3% to 4% per minute with bystander CPR. In contrast, when immediate defibrillation in highly protected environments is available, such as in monitored intensive care units and EP laboratories, where response times of less than 30 seconds are usually achievable, survival from VF is greater than 90%, the exception being patients with pathophysiological conditions that favor the persistence of this potentially fatal arrhythmia. Survivability falls off rapidly after the initial 2 minutes from the onset of cardiac arrest, so that by 4 to 5 minutes survivability may be 25% or less, and by 10 minutes it is less than 10%. Studies have suggested that while immediate defibrillation is the preferred method within 1 to 2 minutes after the onset of cardiac arrest, a brief period of CPR to provide oxygenation of the victim improves survivability when time to defibrillation is longer.

Advanced life support activities, other than those directly related to electrical methods for control of tachyarrhythmias, led to the generation of complex protocols to guide responders. The response algorithms to these various circumstances are complex and these documents are classified as Level of Evidence: C, but they are derived from a combination of varied studies and opinion from Levels of Evidence: A, B, or C. The general goals of advanced life support are to establish hemodynamically effective cardiac rhythm, to optimize ventilation, and to maintain and support the restored circulation. A 1-shock strategy is now recommended to minimize time between chest compressions and shock delivery and resumption of chest compressions. Epinephrine, 1 mg intravenously, is administered and followed by repeated defibrillation attempts at 360 J. Epineph-

rine may be repeated at 3- to 5-minute intervals with defibrillator shocks in-between doses

The approach to the patient with bradyarrhythmic or asystolic arrest or pulseless electrical activity differs from the approach to patients with tachyarrhythmic events of VT/VF. Once this form of cardiac arrest is recognized, efforts should focus first on establishing control of the cardiorespiratory status (i.e., continue CPR, intubate, and establish intravenous access), then on reconfirming the rhythm (in 2 leads if possible), and finally on taking actions that favor the emergence of a stable spontaneous rhythm or attempt to pace the heart. Possible reversible causes, particularly for bradyarrhythmia and asystole, should be considered and excluded (or treated) promptly. These include pulmonary embolus, AMI, hypovolemia, hypoxia, cardiac tamponade, tension pneumothorax, preexisting acidosis, drug overdose, hypothermia, and hyperkalemia.

Arrhythmia Associated with Acute Coronary Syndrome: Acute Coronary Syndrome (ACS) can give rise to a life-threatening arrhythmia that may be the first manifestation of ischemia. The mechanisms of these arrhythmias may be different from those seen in chronic stable ischemic heart disease. Arrhythmias during acute ischemia may be related to re-entry, abnormal automaticity, or triggered activity and are affected by a variety of endogenous factors such as potassium levels and autonomic states. These arrhythmias may cause many of the reported sudden deaths in patients with ischemic syndromes. VF or sustained VT has been reported in up to 20% of AMIs.

Use of prophylactic beta blockers in the setting of AMI reduces the incidence of VF, and this practice is encouraged when appropriate. Similarly, correction of hypomagnesemia and hypokalemia is encouraged

because of the potential contribution of electrolyte disturbances to VF. More recent data showed the benefit of the eplerenone, an aldosterone antagonist, in reducing the risk of SCD mortality.

Pulseless V-Tach/V-Fib: In the event of pulseless VT or VF in ACS, the standard ACLS protocol is initiated including unsynchronized electric shock following basic assessment of airway and initiation of CPR. Energy delivery consists of 1 or more monophasic shocks at 360 J or biphasic shocks at a dose range demonstrated by manufacturer to be effective. If not available, a dose of 200 J is recommended for the first shock and an equal or higher dose for subsequent shocks. The optimal dose for biphasic shocks has not been determined. If return to normal rhythm is not accomplished by defibrillation, the ACLS protocol for pulseless VT or VF is followed. This includes epinephrine (1 mg intravenously every 3 to 5 minutes) or vasopressin (40 U intravenously once only; 1 dose of vasopressin intravenously/intraosseously may replace either the first or second dose of epinephrine), and amiodarone (300-mg or 5-mg/kg intravenous push, with a possible repeat 150-mg intravenous push once only), or as a second tier, lidocaine (1.0 to 1.5 mg/kg with repeat dose of 0.5 to 0.75 mg intravenously/intraosseously up to a total dose of 3 mg/kg).

Idioventricular Rhythm and Nonsustained V-Tach: Neither idioventricular rhythm nor NSVT (lasting less than 30 seconds) occurring in the setting of ACS serves as a reliably predictive marker for early VF. In fact, accelerated idioventricular rhythm has been associated with reperfusion. As such, these arrhythmias do not warrant prophylactic antiarrhythmic therapy.

Unstable Sustained V-Tach: For recurrent VT, if VT is monomorphic and the EF is normal, either procainamide, sotalol, amiodarone, or lidocaine can be used. Alternately, if the EF is low, amiodarone or lidocaine is recommended (amiodarone 150 mg intravenously over 10 minutes or lidocaine 0.5 to 0.75 mg/kg intravenous push). If the VT is polymorphic and the baseline QT is normal, correction of underlying ischemia and electrolyte abnormalities is emphasized.

Bradycardia and Heart Block: Bradycardia and heart block can occur as a result of MI. The likelihood of developing complete heart block complicating MI increases in the presence of underlying conduction system disease. Occurrence of heart block as a result of MI has been associated with an increase in hospital mortality but does not predict long-term mortality in those surviving to discharge. While pacing has not been shown to increase long-term survival post-MI, it is still indicated in symptomatic bradyarrhythmias associated with AMI.

V-Tach Associated with Low Troponin Myocardial Infarction: *****   LOE: C
Prolonged episodes of sustained monomorphic VT may be associated with a rise in cardiac biomarkers due to myocardial metabolic demands exceeding supply, especially in patients with CHD. Such patients usually have a history of MI. It is reasonable to evaluate for myocardial ischemia in patients exhibiting these findings. When sustained VT is accompanied by a modest elevation in cardiac enzymes, it should not be assumed that a new MI occurred to cause the tachycardia. Patients experiencing sustained monomorphic VT are at risk for this arrhythmia in the same manner as are patients without biomarker release accompanying VT.

Sustained Monomorphic V-Tach: *****   LOE: B,C
Electrical cardioversion is always indicated for hemodynamically unstable tachycardia. Managing the patient presenting with well-tolerated, wide-QRS

tachycardia is facilitated by differentiating between VT, SVT with aberrant conduction, and preexcited tachycardia. A working diagnosis of VT is appropriate when the diagnosis is unclear because VT is more prevalent, especially in the patient with structural heart disease, and therapy directed inappropriately at SVT may have adverse consequences. Monomorphic VT is usually related to a structural abnormality such as MI scarring but is mechanistically heterogeneous.

Correction of potentially causative or aggravating conditions such as hypokalemia and ischemia is an early priority. Timely termination is usually desirable even if VT is well tolerated. This can be achieved with cardioversion, antiarrhythmic medications, or pacing techniques. DC cardioversion even at early stage or as "first line" is reasonable. Initial treatment often includes the administration of intravenous antiarrhythmic medication; only intravenous procainamide, lidocaine, and amiodarone are widely available. Intravenous amiodarone is not ideal for early conversion of stable monomorphic VT. Intravenous procainamide is more appropriate when early slowing of the VT rate and termination of monomorphic VT are desired. Close monitoring of blood pressure and cardiovascular status is recommended in the presence of congestive HF or severe LV dysfunction as intravenous procainamide can cause transient hypotension. Lidocaine is effective when VT is thought to be related to myocardial ischemia.

Repetitive Monomorphic V-Tach: ***** LOE: C
Repetitive monomorphic VT is characterized electrocardiographically by frequent ventricular ectopy and salvos of NSVT with intervening sinus rhythm. It typically occurs at rest and is self-terminating, although the arrhythmia can be present for much of the time. Although this terminology can refer to mechanistically diverse arrhythmias, it generally

refers to idiopathic VT, most frequently the RV outflow type. This tachycardia can cause palpitations or, rarely, tachycardia-related cardiomyopathy. Many patients have no symptoms related to the arrhythmia. In some patients, tachycardia is provoked by exercise. An electrocardiographically similar presentation is less frequent in patients with structural heart disease and, specifically, previous MI. Treatment is rarely required on an urgent basis, and chronic management should be based on symptoms and frequency of tachycardia.

Polymorphic V-Tach: ***** LOE: B,C
Polymorphic VT may be sustained, generally requiring urgent electrical cardioversion, or self-terminating with interludes of sinus rhythm. It is useful to distinguish polymorphic tachycardia associated with normal repolarization from that associated with abnormal repolarization (for example, prolonged QT interval). Both VTs may be similar with gross irregularity of rate and QRS morphology with phasic increase and decrease of QRS amplitude often described as "torsades de pointes."

Intravenous beta blockers are useful in this context and improve mortality in the setting of recurrent polymorphic VT with AMI. Intravenous loading with amiodarone is also useful. Urgent coronary angiography should be considered in the setting or recurrent polymorphic VT when ischemia is suspected or cannot be excluded. In all instances, treatment of HF and associated correctable conditions and repletion of potassium and magnesium should be done concurrently with the above.

Torsades de Pointes: ***** LOE: A,B,C
Marked QT interval prolongation and the morphologically distinctive polymorphic VT torsades de pointes occur in 3 common settings: in congenital LQTS, in a drug-associated form, and in patients

with advanced conduction system disease that has progressed to heart block. Torsades de pointes complicating heart block is managed with temporary pacing followed by permanent pacing. Other causes, such as severe electrolyte abnormalities alone or central nervous system injury, are less common.

Incessant V-Tach: ***** LOE: B,C
Clinical Features: The syndrome of very frequent episodes of VT requiring cardioversion has been termed "VT storm." Frequent appropriate ICD shocks represent another variant. While a definition of greater than 2 episodes in 24 hours has been used, much more frequent episodes can also occur. Hemodynamically stable VT lasting hours has been termed "incessant."

Severe underlying heart disease is frequently present. More rarely, VT storm can occur (e.g., in Brugada syndrome, LQTS, catecholaminergic VT, or in drug overdose) in patients who have a structurally normal heart. VT storm can be monomorphic or polymorphic. Polymorphic VT storm in a patient with coronary disease is strongly suggestive of acute myocardial ischemia; pauses may occur prior to polymorphic VT even in the absence of QT prolongation. Pause-dependent VT with marked QT prolongation should be managed as torsades de pointes.

Management: Management guidelines for these syndromes rely on anecdotal evidence because they are rare, there are multiple potential underlying mechanisms, and no randomized trials have been conducted. Intravenous beta blockade should be considered for a polymorphic VT storm as it is the single most effective therapy. Revascularization procedures may be urgently needed. It is of utmost importance to try and understand the substrate of incessant arrhythmias, because if a diagnosis is established, a targeted treatment may be possible.

Monomorphic VT storm can be managed by intravenous antiarrhythmics (e.g., amiodarone, procainamide) to slow the rate but may aggravate the tachycardia by promoting frequent or incessant episodes. Ablation can also be effective. ICD therapy may eventually be needed.

Ventricular Arrhythmia and Sudden Cardiac Death Related to Specific Pathology ***** LOE: A,B,C
Patients with chronic CHD manifest 3 general types of ventricular tachyarrhythmias: NSVT (defined as 3 or more repetitive ventricular beats in a row lasting up to 30 seconds in duration at a rate greater than 100 beats per minute), sustained VT, and cardiac arrest resulting from VT or VF. The cardiac mortality of patients with all types of ventricular tachyarrhythmias is high. The high mortality results from nonsudden, as well as sudden, cardiac death. These arrhythmias may result from myocardial ischemia, or effects of HF, in addition to primary electrical abnormalities. Aggressive attempts should be made to treat HF and to search for and correct myocardial ischemia in patients with ventricular tachyarrhythmias. In some cases, appropriate treatment of ischemia and HF will abolish the arrhythmia (primarily polymorphic VT, VF, and NSVT). Even if specific antiarrhythmic therapy is necessary, the frequency and tolerance of arrhythmias may be improved with appropriate therapy for ischemia and HF.

Left Ventricular Dysfunction Due to Prior Myocardial Infarction

Nonsustained V-Tach: Most NSVT in patients with chronic CHD is brief and does not cause symptoms. There is no evidence that suppression of asymptomatic NSVT prolongs life. Thus, there is no indication to treat NSVT, except in the relatively uncommon circumstances where frequent (incessant) or very rapid episodes compromise

hemodynamic stability. When NSVT causes symptoms that require therapy, attempts should be made to characterize the NSVT electrocardiographically, in order to determine whether the NSVT is related to prior MI or arises by a distinct mechanism that may be especially amenable to RF catheter ablation, such as tachycardia arising from the ventricular outflow tract. Initial pharmacological therapy of symptomatic NSVT should consist of beta adrenergic-blocking agents, if they are not already being used at an adequate dosage. Pharmacological therapy in patients with symptomatic NSVT unresponsive to beta adrenergic–blocking agents would most appropriately be amiodarone or sotalol.

Sustained V-Tach: The treatment of sustained VT in patients with chronic CHD should be tempered by the clinical manifestations produced by the tachycardia, as well as the frequency of episodes. Patients who present with sustained monomorphic VT that does not precipitate cardiac arrest or cause severe hemodynamic instability are usually, but not always, at relatively low risk for SCD (2% yearly). If episodes are relatively infrequent, the ICD alone may be the most appropriate initial therapy, because antitachycardia pacing therapies or high-energy shock therapy may reduce the need for hospitalization and pharmacological antiarrhythmic therapy. Suitable adjunctive therapies include catheter ablation, surgical resection, and pharmacological therapy with agents such as sotalol or amiodarone. Curative therapy of sustained VT using either surgical resection or catheter ablation should be considered in patients with frequent recurrences of VT unresponsive to antiarrhythmic drugs. Patients in whom the tachycardia is hemodynamically stable may be considered for curative catheter ablation. Following correction of ischemia, patients who present with sustained VT that causes severe hemodynamic compromise may benefit from EP testing. Such testing will occasionally reveal

curable arrhythmias such as bundle-branch reentry. In addition, the results of testing often help in appropriate programming of implantable defibrillators. The ICD is the primary therapy for such patients.

Treatment of V-Fib and Cardiac Arrest Survivors: Patients experiencing cardiac arrest due to VF that does not occur within the first 24 to 48 hours of AMI may be at risk for recurrent cardiac arrest. As is the case for patients presenting with sustained VT, such patients should be evaluated and treated for myocardial ischemia. If there is direct, clear evidence of acute myocardial ischemia immediately preceding the onset of VF and there is no evidence of prior MI, the primary therapy should be complete coronary revascularization.

Primary Prevention of Sudden Cardiac Death: The risk for SCD in patients who do not have symptomatic arrhythmias, without prior MI, and those with prior MI whose LVEF is greater than 40% is sufficiently low that prophylactic therapy is not indicated at the present time. For patients with prior MI, multiple factors in addition to reduced EF have been demonstrated to contribute to the risk for SCD after MI; these include the presence of NSVT, symptomatic HF, and sustained monomorphic VT inducible by EP testing. The only specific antiarrhythmic treatment proved consistently effective to reduce risk of both SCD and total mortality is the ICD.

ICD therapy is indicated to reduce the risk of SCD in 2 patient groups: patients whose LVEF is less than or equal to 40% as a result of prior MI and who have spontaneous NSVT and sustained monomorphic VT inducible by EP testing, and patients whose LVEF is less than 30% as a result of an MI that occurred greater than or equal to 40 days earlier when HF (NYHA functional class II or III symptoms) is present. Evaluation of the need for an ICD and im-

plantation should be deferred until at least 3 months after revascularization procedures (i.e., surgical by-pass grafting or percutaneous angioplasty) to allow adequate time for recovery of ventricular function following revascularization. In general, ICD implantation should be deferred until at least 40 days after AMI in patients meeting the above criteria in order to allow time for recovery of ventricular function and because ICD therapy has not been demonstrated to improve survival when implanted within 40 days after MI.

Amiodarone should not be used routinely after MI but is probably the safest agent to use to suppress symptomatic arrhythmias.

Use of ICD for V-Tach in Patient with Normal or Near Normal Left Ventricular Ejection Fraction (LVEF): Recurrent sustained VT is usually treated by management of the underlying condition, prevention of predisposing and trigger factors, and the use of antiarrhythmic therapies such as class I and class III antiarrhythmic drugs. Increasingly, the ICD is being used effectively to treat these arrhythmias, which in themselves may not be life-threatening, in order to avoid the relative ineffectiveness and adverse complications of pharmaceutical therapy. In the case of monomorphic VT, antitachycardia pacing is often applied successfully without provocation of unto-ward symptoms. On the other hand, polymorphic VT or VF, whether or not related to antiarrhythmic drug treatment, may require shock therapy.

Valvular Heart Disease: *****   LOE: C
There is more knowledge on the risk for SCD in patients with aortic valve disease compared with other valvular lesions. Although the overall risk is small, sudden arrhythmic death appears to be more frequent in aortic stenosis than in other lesions: approximately 0.4% per year for aortic stenosis, less than 0.2% per year for regurgitation, and less than 0.2% per year for mitral valve disease.

The presence of a ventricular arrhythmia alone does not constitute an indication for valve repair or replacement. Most patients who die suddenly have been symptomatic from their valvular disease. Although recurrent NSVT may place a patient with severe aortic stenosis at risk for syncope, the management of such a patient is usually guided by the severity of the valvular lesion. Patients with mild valvular lesions who have no LV enlargement, LVH or depressed function should be managed as if they had no structural heart disease.

Congenital Heart Disease: *****   LOE: B,C
Although the short- and long-term survival of these patients is a matter of ongoing study, it is apparent that patients with certain defects have an increased risk of late sudden and total cardiac mortality. During infancy and childhood, greater than 75% of deaths in patients with congenital heart disease are in-hospital events, most occurring during the peri-operative period. The remaining deaths occur as out-of-hospital or emergency department events, often in patients with other congenital anomalies or sepsis. Therefore, the number of very young patients with congenital heart disease who are victims of arrhythmic SCD is quite small.

Beyond 20 years of age, there is a progressive increase in the incidence of sudden and total cardiac mortality in postoperative congenital heart disease patients. Hence, most studies of sudden death in congenital heart disease have evaluated adolescents and young adults. Five congenital heart defects have been associated with the greatest risks of late SCD: tetralogy of Fallot, D- and L-transposition of the great arteries, aortic stenosis, and functional single ventricle.

The largest number of late SCD studies in postoperative patients with congenital heart disease have been for tetralogy of Fallot. A meta-analysis of 39 studies including 4,627 patients showed that the combination of ventricular dysfunction and complex ventricular ectopy was the primary correlate of late SCD. Volume overload due to pulmonary insufficiency and QRS duration greater than 160 ms appear to be the additional factors most likely to be associated with an increased risk of SCD due to ventricular arrhythmias. The results of EP testing for risk stratification in these patients have been inconsistent. Postoperative patients with D-transposition of the great arteries appear to have differing risks for late SCD. A very high incidence of late atrial arrhythmias has been noted in patients following atrial switch procedures, complicated by profound sinus bradycardia. The mechanism of SCD appears to be atrial flutter with 1:1 AV conduction, followed by myocardial ischemia resulting in polymorphic VT or VF.

In general, postoperative patients with unexplained syncope should undergo both hemodynamic and EP evaluation. A high incidence of inducible sustained ventricular arrhythmias has been reported in syncopal postoperative patients who have complex ventricular ectopy. A positive response to EP testing, independent of the clinical indication, may identify patients with a high-risk of late SCD. In the absence of ventricular dysfunction or symptoms, isolated ventricular ectopy has minimal prognostic significance, and the risks of antiarrhythmic drug treatment can exceed any potential benefit.

Metabolic and Inflammatory Conditions: Although disorders in this category are important causes of life-threatening ventricular arrhythmias, the occurrence of VT/SCD is relatively rare.

Myocarditis, Rheumatic Disease, and Endocarditis: ***** LOE: C

Myocarditis: Myocarditis is an inflammatory process affecting the cardiac myocardium and is most often related to infection. However, other toxic exposures, such as exposure to radiation, chemicals, and other physical agents, can lead to cardiac inflammation. Cardiac arrhythmias associated with acute myocarditis can range from conduction abnormalities to difficult to suppress life-threatening ventricular arrhythmias. Death can occur related to HF and arrhythmias including heart block. Patients with arrhythmias or syncope may require antiarrhythmic drugs and/or device therapy. Temporary pacemaker insertion is indicated in patients with acute myocarditis who present with symptomatic heart block as it would be in other causes of acute symptomatic heart block. Pacing is indicated in patients with symptomatic sinus node dysfunction or AV block as a sequela of myocarditis as it would be in other causes of sinus or AV node dysfunction.

Rheumatic Disease: Acute rheumatic fever causes a pancarditis involving the pericardium, myocardium, and endocardium. Sinus tachycardia and PR prolongation are common. Bundle-branch block, nonspecific ST-T wave changes, and atrial and ventricular premature complexes may occur. Complete heart block and ventricular arrhythmias are rare. It has been associated with prolonged QT interval and torsades de pointes.

Endocarditis: Endocarditis of the aortic and mitral valves has been associated with rapid death owing to acute valvular disruption, emboli to the coronary arteries, or abscesses in the valvular rings or the septum. While these deaths are often rapid, they typically are not classified as sudden deaths. Uncommonly, endocarditis has been associated with SCD related to tamponade secondary to rupture. The de-

velopment of cardiac rhythm disturbances portends poorly in infective endocarditis. Antimicrobial therapy will be given as appropriate to the specific causative organism. Surgery is recommended in those with recurrent emboli or refractory HF or those who do not respond to antimicrobial therapy.

Infiltrative Cardiomyopathies: ***** LOE: C
Sarcoidosis: One quarter of patients with sarcoidosis have cardiac lesions, but there is a poor correlation between symptoms and myocardial involvement even in the most advanced cases, and hence SCD may be the first manifestation. In proven cases of cardiac sarcoidosis, supraventricular and ventricular arrhythmias occur frequently (73%) and bundle-branch block is present in about two thirds of patients. Approximately one quarter of these patients develop complete heart block; a similar proportion has congestive cardiac failure. The ECG and Holter monitor are not sensitive or specific enough for detecting myocardial involvement but can be useful for the identification of rhythm disturbances once the diagnosis has been confirmed by other means.

Corticosteroid therapy may reduce the number of premature ventricular complexes and episodes of tachycardia, rendering the arrhythmia easier to treat. The danger of SCD may also be diminished and an improvement may also be seen in conduction defects. The resolution of granulomas may leave a substrate for arrhythmogenesis. Spontaneous VT, severe LV dysfunction, and severe intraventricular conduction disturbance warrant ICD and/or pacemaker therapy as appropriate.

Amyloidosis: Cardiac involvement in amyloidosis, irrespective of the subtype or chemotherapeutic intervention, carries a very poor prognosis. In the AL subtype, the median survival is 6 months with a 6% 3-year survival rate. Progressive HF is usually the

mode of death, but bradyarrhythmia and especially VT may be the terminal event. The use of permanent pacemakers and ICD devices may not influence long-term outcome but in familial cases may be used as a bridge to transplantation.

Fabry Disease: Fabry cardiomyopathy has a prevalence of 3% to 6% in male patients with unexplained LVH. Although cardiac involvement causes a range of ECG abnormalities and conduction disturbances with AV block, ventricular arrhythmias and SCD appear to be very rare.

Hemochromatosis: Up to one third of homozygotes with hemochromatosis have cardiac involvement. Although the natural course of untreated cardiac involvement is progressive HF, ventricular arrhythmias have been reported, the incidence of SCD is unknown. Arrhythmias are managed conventionally.

Endocrine Disorders and Diabetes: *****
LOE: A,C
Endocrine disorders can induce VT/SCD by excess or insufficient hormone activity on myocardial receptors (for example, pheochromocytoma, hypothyroidism). The endocrinopathy can also cause myocardial changes (for example, acromegaly) or electrolyte disturbances produced by hormone excess (for example, hyperkalemia in Addison disease and hypokalemia in Conn syndrome), and certain endocrine disorders can accelerate the progression of conditions such as underlying structural heart disease secondary to dyslipidemia or hypertension, increasing the risk of serious arrhythmias.

Thyroid Disorders: Thyrotoxicosis commonly causes atrial arrhythmias; cases of VT/SCD are extremely uncommon but may occur with concomitant electrolyte disturbances. VT/SCD are more common in hypothyroidism, the basic underlying mechanism

being possibly related to prolongation of the QT interval. Thyroxin replacement therapy usually corrects this abnormality and prevents any further arrhythmias, but antiarrhythmic drugs, such as procainamide, have been used successfully in an emergency.

Pheochromocytosis: Pheochromocytoma may present with VT/SCD, but there are no data to quantify its incidence, best mode of management, or response to treatment.

Acromegaly: SCD is an established manifestation of acromegaly, and life-threatening arrhythmias are likely to be an important cause. Up to one half of all acromegalic patients have complex ventricular arrhythmias on 24-hour Holter recordings, and of these, approximately two thirds are repetitive. There is a strong correlation between these ventricular arrhythmias and LV mass and duration of the disease but not hormone levels. Appropriate surgical management of the pituitary tumor is paramount for improved long-term outcome. Somatostatin analogues such as octreotide and lanreotide have both been shown to reduce LVH and improve the ventricular arrhythmia profile.

Primary Aldosteronism, Addison Disease, Hyperparathyroidism, Hypoparathyroidism: Severe electrolyte disturbances form the basis of arrhythmogenesis and VT/SCD associated with the previously mentioned endocrinopathies; ECG changes including prolongation of QRS and QTc intervals can accompany the electrolyte disturbance. Electrolyte imbalance requires immediate attention before definitive treatment of the underlying cause.

Diabetes: Diabetes is a major risk factor for premature and accelerated atherosclerosis, resulting in an increased incidence of MI, stroke, and death compared with a similar age- and gender-matched population without diabetes. The management of atherosclerotic complications that predispose to ventricular arrhythmias and SCD in patients with diabetes is similar to that in patients without diabetes.

In addition to atherosclerosis and hyperglycemia that predispose the patient with diabetes to ventricular arrhythmias and SCD, autonomic neuropathy, transient hypoglycemic episodes that may occur with drug therapy, and target end-organ damage, such as renal failure, that results in hyperkalemia and occasionally hypokalemic episodes as a result of treatment, augment the risk of SCD. Restrictive cardiomyopathy may be a late complication in some patients with diabetes.

Hypoglycemic episodes increase sympathetic tone. The likelihood of ventricular arrhythmias is enhanced, particularly when they occur in a patient with autonomic neuropathy. Severe hypoglycemia is associated with ventricular repolarization abnormalities, prolongation of the QT interval, and ventricular arrhythmias. Beta blockers have been shown to reduce the magnitude of these abnormalities during experimental hypoglycemia. ACE inhibitors or angiotensin-2 blockers are recommended in all patients with vascular complications of diabetes if no contraindications exist.

End-Stage Renal Failure: ***** LOE: C
Cardiovascular causes account for at least 40% of deaths in patients with end-stage renal failure and 20% of these are sudden. Arrhythmias often occur during hemodialysis sessions and for at least 4 to 5 hours afterward. LQTS has been reported occasionally, sometimes related to therapy with sotalol. Risk factors predisposing to ventricular arrhythmias include LVH, hypertension, anemia, cardiac dysfunction, and underlying CHD. Of these, systolic blood

pressure and myocardial dysfunction have been suggested to be the more important determinants of complex arrhythmia. There are few data on how individuals at highest risk might be identified and treated.

Obesity, Dieting, and Anorexia: ***** LOE: C
Extreme disorders of eating, and overzealous methods of rectifying them quickly, are all associated with SCD. In overweight individuals, this risk is particularly evident in the severely obese with a 40 to 60 times higher incidence compared with that in the aged-matched general population. Some obese individuals have prolonged QTc intervals, but cardiomyopathy of obesity (for example, cardiomegaly, LV dilatation, and myocyte hypertrophy in the absence of interstitial fibrosis) is the most common association with SCD. Weight reduction strategies must be advocated in all obese patients at risk, but these must involve well-balanced, low-calorie diets. Prolonged, unbalanced, very low calorie, semistarvation diets (especially liquid protein diets) have been reported to cause cardiac arrhythmias and SCD by a variety of mechanisms.

Reported mortality rates in anorexia nervosa fluctuate from 5% to 20%, but the actual rate is likely to be around 6%. Up to one third of these deaths, including those occurring during re-feeding, are said to be due to cardiac causes but no precise data exist on SCD. Prolonged periods of starvation result in not only anatomical abnormalities such as cardiac muscle atrophy and pericardial effusions, but also ECG abnormalities, including sinus bradycardia and prolongation of the QTc interval. SCD is therefore a frequent cause of mortality in this cohort. Low weight, low body mass index, and rapid weight loss immediately preceding assessment are the most important independent predictors of QTc interval prolongation. Most of the cardiac manifestations of

anorexia nervosa are completely reversible by appropriate re-feeding.

Pericardial Diseases: ***** LOE: C
SCD can occur in the course of pericardial disease resulting from a variety of pathological processes; these include both constrictive and restrictive processes, resulting from trauma, inflammation, neoplastic, and infectious etiologies. There is no evidence linking specific ventricular arrhythmias with these diseases. Reports of SCD in patients with pericardial diseases suggest that primary hemodynamic processes (that is, acute tamponade, herniation of myocardium through pericardium) are responsible for the vast majority of SCD in such patients.

Pulmonary Arterial Hypertension: ***** LOE: C
SCD is responsible for 30% to 40% of mortality in patients with PAH. Patients experiencing SCD have lower partial pressure of oxygen than do those free of sudden death. SCD in patients with severe PAH appears to occur not only as a result of (presumed) ventricular arrhythmias but also as a result of pulmonary artery rupture or dissection. Cardiac arrhythmias may also result from ischemia. Marked dilatation of the main pulmonary artery has been reported to cause myocardial ischemia as a result of compression of the left main coronary artery. Cardiac catheterization is associated with increased risk of death, including documented VF in this population. In addition to patients with PAH, ventricular arrhythmias occur in persons with sleep disordered breathing and may be responsible for SCD in patients with sleep apnea.

Antiarrhythmic therapy is not indicated for prevention of SCD in patients with PAH or other pulmonary conditions. Good clinical judgment should be used in the management of asymptomatic arrhythmias in such patients.

Transient Arrhythmias of Reversible Cause: *****
LOE: B,C

The mortality of cardiac arrest survivors is high, even when the cause of the initial arrest appears to be a transient or correctable abnormality, and much of the mortality appears due to recurrent cardiac arrest. The most common putative reversible causes of arrest are acute ischemia and electrolyte imbalance.

The short-term (hospital) mortality of patients in whom primary VF complicates the acute phase of MI is high. However, patients who survive the initial hospitalization after Q-wave MI have survival virtually identical to patients without VF in the acute phase of infarction. Coronary artery spasm may increase the risk of ventricular arrhythmias and SCD.

Electrolyte abnormalities, including hypokalemia and hypomagnesemia, facilitate development of VT in predisposed patients receiving antiarrhythmic agents and other drugs associated with the LQTS. However, electrolyte abnormalities should not be assumed to be the cause of cardiac arrest, except in the presence of drug-induced LQTS.

In patients who develop polymorphic VT in association with drug-induced QT prolongation, withdrawal of the offending antiarrhythmic or other agent is usually sufficient to prevent arrhythmia recurrence. If ventricular function is normal, no therapy beyond drug withdrawal, avoidance of future drug exposure, and correction of electrolyte abnormalities is necessary. However, if ventricular function is abnormal, cardiac arrest or syncope should not be attributed solely to antiarrhythmic drugs, and evaluation and treatment should be similar to patients experiencing such events in the absence of antiarrhythmic drugs.

Ventricular Arrhythmias Associated with Cardiomyopathies

Dilated Cardiomyopathy (non-ischemic) *****
LOE: A,B,C

Risk Stratification: The 5-year mortality for DCM has been recently estimated at 20% with SCD accounting for approximately 30% (8% to 51%) of deaths. Ventricular arrhythmias, both symptomatic and asymptomatic, are common, but syncope and SCD are infrequent initial manifestations of the disease. The incidence of SCD is highest in patients with indicators of more advanced cardiac disease who are also at highest risk of all-cause mortality. Although VT and/or VF is considered the most common mechanism of SCD, bradycardia, pulmonary embolus, electromechanical dissociation and other causes account for up to 50% of SCDs in patients with advanced HF. Risk stratification is difficult in DCM. SCD occurs less frequently in patients with less advanced cardiac disease but the proportion of SCD to all-cause death is higher in this group. Predictors of overall outcome also predict SCD and generally reflect severity of disease. Induction of VT by EP testing has been shown to predict SCD but unfortunately failure to induce VT misses most individuals destined to die suddenly. Genetic information is not currently useful for risk stratification.

Electrophysiological Testing: In DCM, EP testing plays a minor role in the evaluation and management of VT. This is related to low inducibility, low reproducibility of EP testing, and low predictive value of induced VT.

Management: The treatment of DCM is often based on individual patient presentation and local physician experience. Pharmaceuticals that have improved overall mortality in patients with HF, such as beta blockers and ACE inhibitors, have also reduced

SCD. Amiodarone is generally preferred to treat patients with symptomatic arrhythmias. The ICD has been shown to be superior to amiodarone for secondary prevention of VT and VF in studies where the majority of patients had CHD, but the role of the ICD in primary prophylaxis has been controversial.

Genetic Analysis: Based on current knowledge, genetic analysis does not contribute to further risk stratification in DCM.

Hypertrophic Cardiomyopathy: ***** LOE: B,C
Risk Stratification: Most individuals with HCM are asymptomatic and the first manifestation may be SCD. SCD is usually related to ventricular arrhythmia with varying contribution of triggers such as ischemia, outflow obstruction, or AF. SCD is less frequently due to bradycardia. Features suggesting higher risk of SCD have been derived from observational studies and include septal wall hypertrophy and LV wall thickness >30 mm. Athletes with HCM should not participate in most competitive sports with the possible exception of sports of low dynamic and low static intensity.

Cardiac MRI and CT have been suggested to be helpful in assessing extent of disease and predicting SCD. A history of SCD in one or more family members has been considered to signify higher risk. Other features associated with higher risk of SCD include syncope, flat or hypotensive response to upright or supine exercise testing in patients younger than 40, presence of VT on Holter monitoring, and VT induced in the EP laboratory. The positive predictive value of any single risk factor is limited. Risk stratification based on incorporation of multiple risk factors would likely improve positive predictive accuracy.

Electrophysiological Testing: The value of EP testing in HCM has been controversial.

Management: The mainstay of pharmacological management for the symptomatic patient has been beta blockers or verapamil, which probably exert their effect by reducing heart rate and decreasing contractility. Amiodarone is widely used and considered the most effective antiarrhythmic agent, although large controlled comparative trials are not available. Medical therapy has not been proved to be beneficial in the prevention of disease progression in the asymptomatic individual and is generally not indicated.

The ICD is not indicated in the majority of asymptomatic patients with HCM, who will have a relatively benign course. Its role is individualized in the patient considered to be at high risk for SCD. Although precise risk stratification has not been validated, patients with multiple risk factors (especially severe septal hypertrophy, greater than or equal to 30 mm) and those with SCD (especially multiple SCDs) in close relatives appear to be at sufficiently high risk to merit consideration of ICD therapy.

Genetic Analysis: Genetic analysis may contribute to risk stratification in selected circumstances where familial patterns are suspected.

Arrhythmogenic Right Ventricular Cardiomyopathy: ***** LOE: B,C

Risk Stratification: ARVC ("dysplasia") is suspected in patients, typically a young man, with RV arrhythmias or in relatives of individuals with known ARVC. Syncope, presyncope, and, less frequently, biventricular failure are also observed. The ventricular arrhythmias have LBBB morphology that spans the spectrum of simple ventricular ectopy, sustained

and NSVT, or VF. The ECG in ARVC frequently shows precordial T-wave inversion, usually over V1 to V3, and QRS duration greater than 110 ms.

SCD is frequently the first manifestation of the disease. The annual incidence of SCD has varied, ranging from 0.08% to 9%. SCD occurs relatively frequently during exercise or during stress, but SCD with no apparent provocation is not uncommon. Although SCD usually occurs in individuals with grossly visible RV abnormalities, it can occur in those with only microscopic abnormalities and no obvious RV enlargement. RV dilation, precordial repolarization abnormalities, and LV involvement have been associated with risk of sudden death.

Electrophysiological Testing: EP testing, in general, is used to reproduce clinical VT and to guide ablation.

Management: The treatment of ARVC is often based on individual patient presentation and local physician experience. The ICD has been used in patients with unexplained syncope, sustained VT, or VF with a high incidence of appropriate shocks. ICD treatment in individuals with a known family history of SCD or unexplained syncope is intuitively compelling but not rigidly proved. The impact of medical therapy on mortality is not established. RF ablation has been used in selected patients for VT in medically refractory patients. Elimination of 1 or more clinical tachycardias by RF ablation is useful for management of symptoms but may not be sufficient to prevent SCD. Operative therapy in the form of total electrical RV disconnection has proved successful in medically refractory patients with normal LV function but does carry a risk of postoperative right HF. Heart transplantation and ventricular assist devices are an option in patients with biventricular failure.

Genetic Analysis: Genetic analysis is useful in families with RV cardiomyopathy.

Neuromuscular Disorders: *****   LOE: A,B
The inherited neuromuscular disorders may predispose to atrial arrhythmias, conduction defects, advanced AV block, monomorphic VT, polymorphic VT, and SCD. The clinical presentation, indicating the potential substrate for SCD, is quite variable, ranging from asymptomatic to the symptoms of syncope, lightheadedness, and palpitations. There are no large series of asymptomatic patients treated with devices and the timing of pacemaker/ICD implantation is not clear. SCD is a well-recognized complication of some of the neuromuscular diseases and progression of the conduction abnormalities may be unpredictable. Once cardiac involvement occurs, particularly with the muscular dystrophies, the clinician should maintain a low threshold for investigating symptoms or ECG findings to determine the need for pacemaker insertion, invasive EP studies, or ICD implantation.

Indications for pharmacological or device therapy in patients with myasthenia gravis, Guillain-Barre syndrome, or an acute cerebrovascular event are quite different than those for other inherited neuromuscular disorders. Treatment is often temporary to manage the acute event and not usually required on a long-term basis.

Heart Failure: *****   LOE: A,B,C
Ventricular arrhythmias and SCD are common in patients with symptomatic acute and chronic HF and LV systolic dysfunction. The cause of HF likely influences the mechanisms and types of ventricular arrhythmias. The guidelines and comments in this section refer to patients with symptomatic HF, not just abnormal LVEF.

Evaluation of arrhythmias in the setting of acute HF necessitates a search for correctable mechanical problems such as catheters placed for hemodynamic monitoring that are causing ventricular or supraventricular arrhythmias. In addition, meticulous attention needs to be given to such factors as pharmacological agents used in the management of acute heart failure and electrolyte and oxygen status. The use of intravenous amiodarone for the management of life-threatening arrhythmias during acute HF has gained widespread acceptance.

In the acutely ill patient with HF, SVT and AF or atrial flutter may impose hemodynamic decompensation, and aggressive therapy may be needed. Vagotonic measures rarely work in the setting of acute HF. Poorly tolerated SVT may be better treated acutely by synchronous cardioversion. Intravenous amiodarone may be more effective at rate control of AF or atrial flutter and may restore sinus rhythm. In HF patients, amiodarone either alone or with electrical cardioversion is effective at slowing the heart rate and achieving cardioversion. Ventricular arrhythmias may be especially poorly tolerated and early cardioversion should be performed, rather than attempting pharmacological termination of arrhythmia.

NSVT can be documented on 24-hour ambulatory ECG monitoring in 30% to 80% of chronic HF patients without arrhythmia symptoms, but there is not a link between NSVT and SCD. Asymptomatic NSVT should not be treated by antiarrhythmic medication. If NSVT causes symptoms that require therapy, amiodarone is probably the safest agent.

SCD accounts for approximately 50% of deaths in patients with HF. However, there is little evidence that empiric antiarrhythmic therapy can reduce the risk of SCD. ICD in combination with biventricular pacing may improve survival and improve symptoms

of patients with advanced HF (NYHA functional class III and IV) over short-term follow-up (1 to 2 years). Biventricular pacing may be used to synchronize the contraction of the LV in patients with abnormal ventricular activation. Cardiac resynchronization therapy has been shown to improve hemodynamics, increase LVEF, extend exercise tolerance, and improve quality of life. The value of biventricular pacing without additional ICD support for the reduction of sudden death remains controversial.

Genetic Arrhythmia Syndromes

General Concepts for Risk Stratification: These diseases share genetically determined susceptibility to VT and SCD in the absence of recognizable structural abnormalities of the heart. These syndromes are by definition rare diseases, because they have an estimated prevalence below 5 in 10,000. In general:

- A family history of SCD has not proved useful in stratifying risk in affected patients.
- Because these diseases are characterized by electrical abnormalities occurring in the structurally intact heart, the use of the ICD is always indicated with a class I indication in the secondary prevention of cardiac arrest. Its use in primary prevention is more debated.
- The severity of the ECG phenotype is generally a marker of increased risk of SCD in most of these diseases.
- Most of the data available for these conditions derive from large registries that have followed patients over time, recording outcome information. No randomized studies are available.
- Avoidance of competitive sports is recommended by some, but not others, for all patients affected by inherited arrhythmogenic disorders even when physical activity is not considered to be the trigger for arrhythmic episodes.

Long QT Syndrome: *****   LOE: A,B
The LQTS is an inherited disease characterized by prolonged ventricular repolarization (QT interval) and by ventricular tachyarrhythmias that may manifest as syncopal events. Cardiac arrhythmias are often elicited by stress and emotion, although in some cases they may also occur at rest or during sleep. QT interval duration was identified as the strongest predictor of risk for cardiac events (syncope, SCD) in LQTS. A family history of SCD has not proved to be a risk factor for SCD.

Symptoms in LQTS range from SCD to syncope and near syncope. Patients resuscitated from SCD have an especially ominous prognosis, with a relative risk of 12.9% of experiencing another cardiac arrest. In addition, affected patients may be identified because of QT prolongation detected incidentally or because they are relatives of affected individuals and are found to be mutation carriers in genetic screening. The mean age for first manifestation of the disease is 12 years old, but there is a wide range from the first year of life to as late as the fifth through sixth decades. Documentation of the arrhythmia during cardiac events is relatively uncommon in LQTS: when arrhythmias are recorded, the characteristic polymorphic VT, "torsades de pointes," is identified; SCD may be the first manifestation of the disease.

It is recommended that all patients affected by LQTS avoid competitive sports activity. For LQT1 patients, swimming should be specifically limited or performed under supervision. LQT2 patients should avoid exposure to acoustic stimuli especially during sleep (avoidance of telephone and alarm clock on the night stand). All patients with LQTS should avoid drugs known to prolong the QT interval and those that deplete potassium and magnesium.

Genetic analysis is very important for identifying all mutation carriers within an LQTS family. Once identified, silent carriers of LQTS genetic defects may be treated with beta blockers for prophylaxis of life-threatening arrhythmias.

Short QT Syndrome: First described in 2000, at present, it is still undefined whether the diagnosis of SQTS should be based on QT or QTc and which is the sensitivity and specificity of different QT/QTc interval cutoff values. Morphological T-wave abnormalities accompany the abbreviated repolarization in SQTS. Several patients have tall and peaked T waves or asymmetrical T waves with a normal ascending phase and a very rapid descending limb. Clinical parameters for diagnosis are not yet known, so genetic analysis seems useful to confirm diagnosis in suspected cases.

Up to now, only 23 cases of SQTS from 6 different families have been reported and the present experience suggests that the disease may be highly lethal. It is interesting to note that all 3 SQTS genes (KCNH2, KCNQ1, and KCNJ2) also cause LQTS. The ECG phenotypes depend on the opposite biophysical consequences of the underlying mutations, with loss-of-function mutations being associated with LQTS and gain-of-function mutation being the cause of SQTS.

EP investigations have shown that both atrial and ventricular effective refractory periods are shortened in SQTS and programmed electrical stimulation usually induces ventricular tachyarrhythmias. The management of patients with SQTS is still poorly defined; genetic analysis does not contribute to risk stratification.

Brugada syndrome: *****   LOE: C
The Brugada syndrome is associated with a character-

istically abnormal ECG and a high risk of SCD in individuals with a structurally normal heart. The Brugada pattern ECG shows J-point segment elevation in leads V1 to V3 and RBBB in some patients; the ECG pattern can be present always or intermittently.

The disease is transmitted with an autosomal dominant pattern of inheritance. The clinical expression of the phenotype is modified by gender as 90% of the affected individuals with a diagnostic ECG are male. Cardiac events (syncope or cardiac arrest) occur predominantly in males in the third and fourth decades of life, although presentation with cardiac arrest in neonates or children have been reported. Fever is a predisposing factor for cardiac arrest in the Brugada syndrome. Because implantation of an ICD is the only prophylactic measure able to prevent SCD, risk stratification is of major importance in these patients. As with LQTS, there are no data showing that family history predicts cardiac events among family members. Patients with history of syncope and the ECG pattern of spontaneous ST-segment elevation have a 6-fold higher risk of cardiac arrest than patients without syncope and the spontaneous ECG pattern.

The role of EP testing for risk stratification is debated. Patients with Brugada syndrome usually do not have ventricular extrasystoles or nonsustained runs of VT at Holter recording. Therefore, the therapeutic approach for these patients is centered on the prevention of cardiac arrest. Basic science studies and clinical studies suggest a role for block of the transient outward potassium current by quinidine in reducing arrhythmia frequency.

Genetic analysis may help identify silent carriers of Brugada syndrome-related mutations so that they can remain under clinical monitoring to detect early manifestations of the syndrome. Furthermore, once identified, silent mutation carriers should receive genetic counseling and discussion of the risk of transmitting the disease to offspring. Based on current knowledge, genetic analysis does not contribute to risk stratification.

Catecholaminergic Polymorphic V-Tach: *****
LOE: C

CPVT is characterized by ventricular tachyarrhythmias that develop during physical activity or acute emotion in the presence of an unremarkable resting ECG. The first episodes often manifest during childhood, although late-onset cases have been described. The disease can be transmitted as an autosomal dominant as well as an autosomal recessive trait. Half of the autosomal dominant cases are caused by mutations in the gene encoding the cardiac ryanodine receptor (RyR2), responsible for calcium release from the stores of the sarcoplasmic reticulum.

Too few patients with CPVT have been reported to allow the definition of a risk stratification scheme. Beta blockers appear to be effective. Patients who have had an episode of VF are considered at higher risk and are usually implanted with an ICD along with beta-blocker therapy. The recurrence of sustained VT or of hemodynamically nontolerated VT while receiving beta blockers is usually considered a marker of higher risk and an ICD is often recommended in these patients. EP testing is not useful for management and risk stratification because CPVT patients are usually not inducible.

Supraventricular and ventricular arrhythmias are usually reproducibly induced by exercise stress when the heart rate reaches a threshold of 120 to 130 beats per minute. Isolated PVCs usually develop first and are followed shortly after by short runs of NSVT. If the patient continues to exercise, the duration of VT runs progressively increase and VT may become sus-

tained. A beat-to-beat alternating QRS axis that rotates by 180°, "bidirectional VT," is the typical pattern of CPVT-related arrhythmias.

Genetic analysis may help identify silent carriers of catecholaminergic VT-related mutations; once identified silent carriers may be treated with beta blockers to reduce the risk of cardiac events and may receive appropriate genetic counseling to assess the risk of transmitting the disease to offspring. Based on current knowledge, genetic analysis does not contribute to risk stratification.

Arrhythmias in Structurally Normal Hearts

Idiopathic Ventricular Tachycardia: *****
                                    LOE: B,C
Demographics of Outflow Tract V-Tach: VT arising from the RV is the most common form of VT in apparently healthy people and is associated with a good prognosis in those without overt structural heart disease. This VT usually has a left bundle-branch, inferior-axis morphology and often presents as non-ischemic exercise-induced and/or repetitive monomorphic VT. Symptoms tend to be mild and syncope is rare. Left ventricular outflow tract (LVOT) VT can arise in the absence of overt structural abnormalities and accounts for a small percentage of the overall cases of VT.

The ECG recorded during sinus rhythm in patients with RVOT tachycardia helps distinguish it from the more serious condition of RV dysplasia where the ECG is more often abnormal. RVOT has long been thought to be idiopathic in nature, but this characterization has relied on conventional imaging and diagnostic techniques. More recently, MRI has been applied to the evaluation of patients with VT arising from the RV in the absence of defined abnormalities on conventional testing particularly to exclude

ARVC. LVOT VT can be classified by site of origin to either an endocardial origin; coronary cusp origin; or epicardial origin. VT arising from the LVOT and from the LV septum typically presents in the third to fifth decades of life. LVOT VT is more common in men than in women. This VT may be incessant and may be provoked by exercise.

Mechanisms: The most common form of RVOT VT is related to triggered activity arising from delayed afterdepolarizations and is thought to be dependent on intracellular calcium overload and cyclic adenosine monophosphate. RVOT VT is frequently adenosine sensitive, may terminate with vagal maneuvers, and is facilitated by catecholamines. As such, it is often not easily inducible at baseline EP testing and may require rapid burst pacing or stimulation by isoproterenol.

LV VT arising from the outflow tract may be reentrant but can also result from enhanced automaticity. Incessant LV VT has been related to a triggered mechanism associated with delayed afterdepolarizations. Idiopathic LV tachycardia can be verapamil sensitive, adenosine sensitive, and propranolol sensitive.

Electrophysiological Testing: EP is motivated by the need to establish precise diagnosis to guide curative catheter ablation. Outflow tract VT in the absence of concomitant cardiac disease does not carry an adverse prognosis, although syncope can occur. The prognostic value of inducibility of ventricular arrhythmias has not been systematically evaluated. Induction of LVOT VT by EP testing is not consistent, although the arrhythmia may be provoked. It can be provoked during isoproterenol infusion.

Management: Clinical treatment of RVOT or LVOT VT often involves beta and calcium channel block-

ers. Type IC antiarrhythmic drugs have been found to be useful in RVOT VT. In patients who remain symptomatic or for whom drug therapy fails, catheter ablation of the arrhythmia focus in the RVOT should be considered. Acute success rates for RVOT ablation have been reported in excess of 90%. However, long-term success varies and may depend on the degree or presence of other abnormalities.

Demographics of Other Outflow Tract V-Tach: So-called idiopathic LV VT can arise from the LVOT or from the fascicles of the specialized conduction system. Fascicular VT can be classified into 3 types according to origin and QRS morphology during VT. Left posterior fascicular VT typically has an RBBB and superior axis morphology and is the more common form of fascicular VT. VT arising from the left anterior fascicle has an RBBB and right-axis deviation configuration and is less common. Rarely, fascicular VT will arise from fascicular location high in the septum and has a narrow QRS and normal axis configuration. This VT presents in the third to fifth decades of life and is equally distributed between the sexes.

Mechanisms and Treatment: Left fascicular VT typically is reentrant and may respond to beta or calcium channel blockers. However, in patients who do not tolerate medical treatment or for whom medical treatment has failed, ablation can be considered. Ablation of posterior fascicular VT is guided by a recording made either during sinus rhythm or in VT demonstrating a discrete potential preceding the earliest ventricular electrogram. Newer 3-dimensional mapping devices that do not require the presence of sustained VT can facilitate ablation in these patients.

Electrolyte Disturbances: ***** LOE: B,C
Although changes in extracellular potassium, extra-

cellular and intracellular magnesium (especially with associated hypokalemia), and intracellular calcium are all associated with EP changes that are arrhythmogenic, life-threatening ventricular arrhythmias in patients with structural heart disease should not be attributed solely to changes in these ionic concentrations. Changes in potassium concentration may occur after cardiac arrest or may accompany certain disease states such as periodic paralysis.

A rapid rise in extracellular potassium, hypokalemia (less than 3.5 mM), and hypomagnesemia are all associated with ventricular arrhythmias and SCD in patients with structurally normal hearts (some of whom may have underlying channelopathies) and in an AMI setting. Hypomagnesemia is classically associated with polymorphic VT or torsades de pointes, which together with ventricular arrhythmias in an AMI setting may respond to intravenous magnesium. Hypokalemia with or without hypomagnesemia may be responsible for ventricular arrhythmias in subjects with hypertension and congestive cardiac failure (precipitated by the use of thiazide and loop diuretics, acute starvation, acute alcohol toxicity/withdrawal, and those with ventricular arrhythmias associated with digoxin and other Vaughan Williams class I antiarrhythmic drugs. Significant hypocalcemia can prolong the QT interval.

Changes in the extracellular ionic concentrations of calcium required to produce EP changes that may contribute to ventricular arrhythmias are not encountered in clinical practice. Occasionally, hyperparathyroidism can cause important elevations in serum calcium concentrations. Intracellular fluctuations in calcium concentration influenced by drugs (for example, digitalis glycosides), exercise (for example, catecholamines), and reperfusion following myocardial ischemia, however, can trigger EP changes that may lead to life-threatening arrhythmias. The

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protective effects of beta blockade in the latter settings may in part be due to the inhibition of calcium influx into myocytes.

Physical and Toxic Agents

Alcohol: *****   LOE: C

The relationship between alcohol ingestion and VT/SCD is indisputable; what is controversial however, is its exact nature. A number of studies claim a J-shaped relationship with risk lowest in individuals with low alcohol intake (that is, 2 to 6 drinks per week) compared with those who rarely or never consume alcohol and those with a high alcohol intake (that is, more than 3 to 5 drinks per day) and binge drinking habits, the "holiday heart syndrome." Alcohol ingestion may reduce the incidence of VT/ SCD due to coronary events, but its effect on life-threatening arrhythmias correlates directly with the amount and duration of alcohol intake and even small quantities may be significant in susceptible individuals.

The mechanisms associated with alcohol-induced VT/SCD are complex and not entirely related to the presence of alcohol-induced cardiomyopathy. Alcohol has a negative inotropic effect mediated by direct interaction with cardiac muscle cells, although this action is often masked by the indirect actions from enhanced release of catecholamines. EP studies have shown alcohol to induce various arrhythmias including VT in patients with and without cardiomyopathy. LVH and remodeling is an early response to heavy drinking; one third of alcoholics demonstrate diastolic dysfunction correlating with consumption and 20% to 26% develop DCM within 5 years. In these patients, myocyte and nuclear hypertrophy, interstitial fibrosis, and myocyte necrosis provide the substrate for arrhythmogenesis. QTc is prolonged in patients with proved alcoholic liver disease in the absence of electrolyte disturbances and may act as the trigger to life-threatening arrhythmias.

Smoking: *****   LOE: B

Cigarette smoking is an independent risk factor for SCD regardless of underlying CHD. The vast majority of these deaths are arrhythmic. In females who smoke 25 or more cigarettes per day, the risk of ventricular arrhythmia and SCD is increased 4-fold, similar to that conferred by a history of MI. It is a long-term risk factor and continues to be so in survivors of out-of-hospital cardiac arrest who fail to give up smoking. Cessation of smoking significantly reduces risk of SCD. There are no data available to allow identification of individuals at greatest risk.

Lipids: *****   LOE: A,B

The association of high total, very low-density lipoprotein (VLDL), or low-density lipoprotein (LDL) cholesterol levels, a low HDL cholesterol level together with high triglyceride and apolipoprotein B levels with increased risk of VT/SCD is almost entirely due to concurrent CHD. Appropriate lipid management strategies, especially the use of statins, reduces the risk of SCD by preventing recurrent fatal MI and ventricular arrhythmia. The effect of lipid lowering on SCD in primary prevention has not been addressed, but a relative risk reduction of 30% to 40% would be expected in parallel with the reduction in the risk of CHD death.

In one study of patients with an ICD and a recent episode of ventricular arrhythmia, there was a trend toward a higher incidence of VT/VF in patients randomized to fish oil, a trend that correlated with n-3 PUFA levels. An actuarial analysis of time to recurrent events showed significantly more events in patients randomized to fish oil.

Ventricular Arrhythmias and Sudden Cardiac Death Related to Specific Populations

Athletes: *****   LOE: B,C

Screening and Management

Screening: The major causes of SCD in athletes are HCM (36%), coronary artery anomalies (19%), ARVC, and myocarditis. In Italy, the incidence of the former as a cause of SCD has been reduced considerably due to an ECG and echocardiographic screening program. It is generally accepted that preparticipation screening for medical conditions should be a requirement for clearance to participate in competitive athletics, but there are no uniformly accepted standards for screening. Because the risk of SCD among athletes appears to exceed the risk in comparably aged populations, attention to cardiovascular screening is of special importance.

Preparticipation cardiovascular screening focuses in general on a young population group (aged less than 30 years), among whom most anomalies will be congenital, although some might be acquired disorders. Special consideration is required in athletes who are middle-aged and older.

Screening of athletes is a difficult task. The low incidence of anomalies makes screening not very cost effective, although one study has suggested that ECG screening is more cost effective than echocardiographic screening. Routine physical examination might not reveal clinically significant anomalies, and personal or family histories have limited value. The resting ECG can disclose rhythm disturbances, abnormal repolarization syndromes such as the LQTS, the Brugada syndrome, the WPW syndrome, and the depolarization and repolarization abnormalities associated with HCM. However, nonspecific variations commonly observed on ECGs recorded from adolescents and young athletes may be confounding. Echocardiography may show structural anomalies but will not disclose anomalies of the coronary arteries.

It is recommended that all candidates undergo screening tests, such as ECG and, when appropriate, echocardiography (for example, abnormal ECG, family history), beyond the history and physical examination.

Management of Arrhythmias, Cardiac Arrest, and Syncope: In athletes, risk factors might be aggravated or attenuated but not abolished by regular physical activity. For legal and ethical reasons, athletes receiving cardiovascular drugs and devices such as pacemakers and ICDs are generally not allowed to participate in high-grade competition. Athletes presenting with syncope or presyncope should not participate in competitive sports until the cause is determined to be both benign and treatable. Increase of PVCs during exercise requires careful evaluation. Athletes with nonsustained and asymptomatic exercise-induced ventricular arrhythmias may participate in low-intensity competitive sports provided that no structural heart disease has been demonstrated. Athletes presenting with rhythm disorders, cardiac anomalies, or syncope should be treated as any other patients.

Gender and Pregnancy: *****   LOE: B,C
QT Interval: Typically, women have longer QT intervals than do men, and this difference is more pronounced at slower heart rates, but by age 50, gender differences in QT intervals have largely equalized. A similar shortening of the QT interval at puberty has been noted in males genotypically characterized with LQTSs. These observations strongly support a hormonal effect on QT and hence arrhythmia susceptibility. In women with the congenital LQTS, the risk of cardiac arrest is greater during the postpartum period compared with before or during pregnancy. The relative tachycardia seen during pregnancy may serve to shorten the QT interval and be protective.

Beta blockers have a major benefit during the post-partum period when the heart rate naturally falls. Beta blockers can generally be used safely during pregnancy. Most are excreted in breast milk. Use during pregnancy is generally well tolerated by both the mother and the fetus, although a decrease in fetal heart rate can be seen. Several studies have demonstrated an increased susceptibility in women to torsades de pointes, likely related to the longer baseline QT interval and perhaps to differences in drug pharmacodynamics. The incidence of both congenital and acquired forms of long QT intervals and resultant torsades de pointes is higher in women than in men.

In the Long QT Registry, 70% of the subjects and 58% of affected family members are women. Until puberty, males in the registry were found to be more likely than females to have cardiac arrests or syncope, but subsequently, the incidence of these potentially fatal events predominated in females. Several studies have shown that drug-induced torsades de pointes is more common in women than in men. ICD therapy should be strongly considered in patients with long-term QT syndromes who are drug-resistant and those with marked potential for life-threatening arrhythmias.

Pregnancy and Postpartum: Palpitations are extremely common during pregnancy, and several studies have shown an increase in the symptoms of SVT during pregnancy. While most palpitations are benign during pregnancy, new-onset VT is of concern. Although the presence of structural heart disease should be sought in these women, often VT occurs in the absence of overt structural heart disease and may be related to elevated catecholamines. As such, these arrhythmias may be beta-blocker sensitive.

In women presenting with new-onset VT during the last 6 weeks of pregnancy or in the early postpartum period, the possibility of postpartum cardiomyopathy should be ruled out. In women with non-long QT-related sustained VT during pregnancy, antiarrhythmic therapy may be indicated with intravenous lidocaine acutely or procainamide long term. Amiodarone can have deleterious effects on the fetus, including hypothyroidism, growth retardation, and premature birth. Prophylactic therapy with a cardioselective beta blocker may be effective. Sotalol can be considered if beta-blocker therapy is ineffective.

For women with known structural heart disease, pregnancy may present significant risk. Pulmonary edema, stroke, or cardiac death can occur in up to 13% of such pregnancies. Independent predictors of risk in women with heart disease include prior history of arrhythmias, cyanosis, poor functional class, LV systolic dysfunction, and LV outflow obstruction. Potentially life-threatening ventricular tachyarrhythmias should be terminated by electrical cardioversion. Beta 1-selective beta blockers alone, amiodarone alone (noting cautions about birth defects above), or in combination may be used, and ICD may be needed as its presence does not contraindicate future pregnancies.

Special Concerns for Specific Arrhythmias: WPW syndrome and orthodromic AV reciprocating tachycardia are more common in men than in women. In addition, in patients with WPW syndrome manifest pathways are more common in men. Conversely, antidromic AV ventricular reciprocating tachycardia is more common in women than in men. However, AF degenerating to VF is more common in men than in women. For symptomatic WPW syndrome, the treatment of choice is RF ablation. The outcomes are similar in both sexes. Management of sympto-

matic WPW during pregnancy may require initiation of antiarrhythmic drugs to block the accessory pathway and, in some, long-term monitoring.

Classic predictors such as obesity, LVH, hyperlipidemia, and tobacco use are associated with CHD and VT more in men than in women. For women, hyperglycemia, elevated hematocrit, and decreased vital capacity are more important predictors for CHD and VT. The impact of diabetes is seen in both sexes but is much more pronounced in women. While NSVT and PVCs have been associated with increased risk of sudden death in men with or without CHD, no such association has been seen in women. Similarly, while PVCs post-MI in men have been associated with increased mortality, this does not hold true for women.

Elderly Patients: *****   LOE: A,C
Epidemiology: Ventricular arrhythmias are common in elderly populations, and the incidence increases in the presence of structural heart disease. It must be noted that the elderly are a heterogeneous group. In different studies, elderly patients are defined anywhere from greater than 60 years to greater than 85 years of age. This lack of uniformity raises concerns regarding the applicability of study results to the entire elderly population.

Ventricular arrhythmias can be found in 70% to 80% of persons over the age of 60 and complex ventricular ectopy is common in this age group, although many such persons are often asymptomatic. Complex ventricular arrhythmias often presage new major coronary events and SCD in patients with CHD and other types of structural heart disease. The incidence of SCD increases with advancing age. In elderly patients with CHD, the proportion of cardiac deaths that are sudden decreases, whereas the proportion of "out of hospital"

SCD increases progressively with advancing age. Although greater than 80% of patients who die suddenly from cardiac causes have CHD, elderly patients with DCM and valvular heart disease are also at risk, as are those with HCM, ARVC, and surgically repaired tetralogy of Fallot. Brugada syndrome and congenital LQTS are uncommon causes of SCD in elderly patients.

Pharmacological Therapy: The management of ventricular arrhythmias and the prevention of SCD in elderly patients do not differ appreciably from those recommended for the general population. One must take into account the physiological changes that occur with advancing age and adjust drug regimens accordingly; drug therapy should be initiated at lower than the usual dose and titration of the drug should take place at longer intervals and smaller doses.

The empiric use of most antiarrhythmic drugs to treat NSVT and other complex ventricular ectopy has been shown to be ineffective in preventing SCD and is even deleterious under certain circumstances. Amiodarone is the only antiarrhythmic drug shown to improve prognosis in survivors of cardiac arrest. Beta blockers, along with several agents that do not possess classic antiarrhythmic properties (e.g., ACE inhibitors, angiotensin receptor blockers, statins), have been shown in many studies to reduce all-cause mortality and SCD after AMI in all age groups, including the elderly. The combination of beta blockers and amiodarone may reduce all-cause mortality and SCD to a greater extent than amiodarone alone, but studies demonstrate that beta blockers are underused in the elderly.

Device Therapy: Several randomized, prospective trials have demonstrated the efficacy of ICDs in reducing SCD in patients with CHD at high risk for SCD

(primary prevention) and in patients resuscitated from SCD (secondary prevention) compared with antiarrhythmic drug therapy, and all studies have included patients over 65 years. Data comparing the efficacy and complications of ICD therapy in older and younger patients are sparse. Very elderly patients with multiple comorbidities and limited life expectancy may not be appropriate candidates for ICD therapy even if they meet standard criteria. In such circumstances, the clinical judgment of the primary treating physician and the desires of the patient and/or his or her family take precedence over general guideline recommendations.

Pediatric Patients: *****   LOE: C
The incidence of SCD due to cardiovascular disease is significantly less in pediatric than in adult patients. Current estimates are that deaths due to cardiovascular disease in individuals younger than 25 years of age account for less than 1% of all cardiac mortality, with an event rate between 1.3 and 4 deaths per 100,000 patient years. A definite or probable cardiac cause has been estimated in 70% of young, unexpected sudden death victims. Several groups of young patients have been identified who are at an increased risk of SCD compared with the general population. These include patients with congenital heart disease, coronary artery anomalies, cardiomyopathies, and primary arrhythmic diagnoses such as the LQTSs.

SCD in pediatric patients with WPW syndrome is uncommon and occurs primarily in patients with prior syncope, multiple accessory pathways, or short refractory periods. Therefore, in selected patients, an EP study may be indicated and ablation performed if the patient is symptomatic or the refractory period of the accessory pathway is equal to or less than 240 ms.

Isolated PVCs are common in infants, with 15% of all newborns reported as having some ventricular ectopy during 24-hour ambulatory ECG monitoring. The prevalence of ventricular ectopy decreases to less than 5% in children but then increases to 10% by 10 years of age and 25% during late adolescence and early adulthood. For the vast majority of young patients with ventricular ectopy, the primary objective is to exclude any associated functional or structural heart disease.

Sustained ventricular arrhythmias may also occur in infants, most commonly, it is an accelerated idioventricular rhythm. This arrhythmia typically resolves spontaneously during the first months of life. This is in contrast to the rare infant with incessant VT, which may be due to discrete myocardial tumors or cardiomyopathy. VF and SCD have been reported in these infants, most often following the administration of intravenous digoxin or verapamil for a presumptive diagnosis of SVT. These ventricular arrhythmias may respond to antiarrhythmic treatment or be amenable to surgical resection. Sustained VT in infants may also be caused by hyperkalemia or associated with one of the LQTSs, particularly those forms with AV block or digital syndactyly.

RVOT and LVOT tachycardia and LV septal tachycardia may be diagnosed during childhood or adolescence; the general prognosis for these arrhythmias is mostly benign. The role and benefit of ICD implantation for the prevention of SCD in young children with advanced ventricular dysfunction have not been defined.

The treatment of potentially life-threatening ventricular arrhythmias in children is disease specific (e.g., beta blockade for LQTSs, catheter or surgical ablation for focal VTs, and heart transplantation for end-stage cardiomyopathies). When indicated, ICDs

with transvenous lead systems are generally feasible in children older than 10 years.

Patients with Implantable Cardioverter-Defibrillators (ICDs): ***** LOE: B,C

Supraventricular Tachyarrhythmias: SVT may trigger ICD action due to fulfilling programmed ventricular or SVT detection criteria. The effect of atrial tachyarrhythmia on ventricular rate response is crucial. As long as the ventricular rate fits within the tachycardia detection window, meaningful programming of the detection algorithms may prevent device action for VT. If ventricular rate falls within the VF detection window, appropriate therapy should not be withheld. Beta blockade is also a valuable therapy that will prevent many unwanted device interventions due to supraventricular arrhythmias. Additional investigations such as Holter recordings, patient-activated loop recorders, and EP studies might be required to guide the management of these arrhythmias.

Supraventricular Tachycardia in Patients with Ventricular ICDs: AF is the most frequent culprit of arrhythmia. Rapid ventricular rate during SVTs may provoke ventricular antitachycardia pacing. Device action may be proarrhythmic, as inappropriate antitachycardia pacing may cause VT or VF. Careful analysis of detected episodes, the effects of antitachycardia pacing on the cycle length intervals and the mode of termination or acceleration are important for classification of the detected tachycardia.

Dual-Chamber ICDs: Dual-chamber ICDs provide improved atrial diagnostic features with recording of local atrial electrograms, regularity of atrial signals, and cycle lengths. This may provide additional features to avoid inappropriate VT/VF therapies, but inappropriate ventricular tachyarrhythmia sensing still occurs in 10% to 15% of cases. Oversensing of

far-field signals by the atrial electrodes may prompt inappropriate therapies for SVTs, such as antitachycardia pacing or automatic cardioversion. In case of programmed internal atrial cardioversion therapies, even low-energy shocks may be painful and compromise quality of life. High-rate atrial antitachycardia pacing may induce (transient) AF. Efficacy of advanced atrial pacing or cardioversion therapies varies greatly in function of episode duration, atrial cycle length, and atrial tachycardia mechanism.

Arrhythmia Storm in ICD Patients: The term arrhythmia storm refers to a situation when numerous device discharges occur due to recurrent repetitive arrhythmias. A vicious cycle between device action and cardiac dysfunction may lead to further deterioration. The management must address all aspects to correct the situation.

Drug-Induced Arrhythmias: Specific syndromes of drug-induced arrhythmias, with diverse mechanisms and management strategies, are described in the sections that follow. Treatment guidelines focus on avoiding drug treatment in high-risk patients, recognizing the syndromes of drug-induced arrhythmia and withdrawal of the offending agent(s).

Interactions can occur when a drug is eliminated by a single pathway and that pathway is susceptible to inhibition by the administration of a second drug. Interactions can reduce plasma concentrations of antiarrhythmic drugs and thereby exacerbate the arrhythmia being treated. Additive pharmacological effects may also result in arrhythmias.

Digitalis Toxicity: ***** LOE: A,C

Clinical Presentation: Certain arrhythmias are typical: enhanced atrial, junctional, or ventricular automaticity (with ectopic beats or tachycardia) often

combined with AV block. Overdose of digitalis causes severe hyperkalemia and cardiac standstill. The diagnosis is established by the combination of characteristic rhythm disturbances, ancillary symptoms (for example, visual disturbances, nausea, changes in mentation), and elevated serum concentrations. Contributing factors may include hypothyroidism, hypokalemia, or renal dysfunction.

Specific Management: In mild cases, management includes discontinuing the drug, monitoring rhythm and maintaining normal serum potassium. Occasionally, temporary pacing may be needed. For more severe intoxication (serum digoxin concentration greater than 4 to 5 ng/mL, and with serious arrhythmias), the treatment of choice is digoxin-specific Fab antibody.

Drug-Induced Long QT Syndrome: *****
LOE: A,B,C
Clinical Features: Marked QT prolongation, often accompanied by polymorphic VT torsades de pointes, occurs in 1% to 10% of patients receiving QT-prolonging antiarrhythmic drugs and much more rarely in patients receiving "noncardiovascular" drugs with QT-prolonging potential. Most cases of drug-induced torsades de pointes display a "short-long-short" series of cycle length changes prior to initiation of tachycardia. QT intervals, uncorrected for rate, are generally greater than 500 ms, prominent U waves are common, and marked QTU prolongation may be evident only on postpause beats.

Presentations of drug-induced QT prolongation include incidental detection in an asymptomatic patient, palpitations due to frequent extrasystoles and nonsustained ventricular arrhythmias, syncope due to prolonged episodes of torsades de pointes, or SCD.

Management: Monitoring high-risk patients during initiation of QT-prolonging antiarrhythmic drugs and recognition of the syndrome when it occurs are the first steps. Maintaining serum potassium between 4.5 and 5 mEq/L shortens QT. Intravenous magnesium can suppress episodes of torsades de pointes without necessarily shortening QT. Magnesium toxicity (areflexia progressing to respiratory depression) can occur when concentrations reach 6 to 8 mEq/L but is a very small risk with the doses usually used in torsades de pointes, 1 to 2 g intravenously. Temporary pacing is highly effective in managing torsades de pointes that is recurrent after potassium repletion and magnesium supplementation. Isoproterenol can also be used to increase heart rate and abolish postectopic pauses.

Sodium Channel Blocker Related Toxicity: *****
LOE: A,C
Clinical Features: Antiarrhythmic drugs are the most common precipitants, although other agents, notably tricyclic antidepressants and cocaine, may produce some of their toxicities through these mechanisms. Sodium channel-blocking drugs with slower rates of dissociation tend to generate these adverse effects more commonly; these include agents such as flecainide, propafenone, and quinidine that (as a consequence of the slow dissociation rate) tend to prolong QRS durations even at normal heart rates and therapeutic dosages.

In patients treated for sustained VT, these agents may provoke more frequent, and often more difficult to cardiovert, episodes of sustained VT. While the drugs generally slow the rate of VT, occasionally the arrhythmia becomes disorganized and may be resistant to cardioversion; deaths have resulted. Sodium channel-blocking drugs increase defibrillation energy requirement and pacing thresholds; as a consequence, patients may require reprogramming

or revision of pacing or ICD systems or changes in their drug regimens. Sodium channel blockers can "convert" AF to slow atrial flutter, which can show 1:1 AV conduction with wide-QRS complexes. This drug-induced arrhythmia can be confused with VT.

Sodium channel blockers can occasionally precipitate the typical Brugada syndrome ECG. This has been reported not only with antiarrhythmic drugs but also with tricyclic antidepressants and cocaine.

Management: Sodium channel-blocking drugs should not be used in patients with MI or sustained VT due to structural heart disease. The major indication for these drugs is atrial arrhythmias in patients without structural heart disease. When used for AF, AV nodal-blocking drugs should be coadministered to prevent rapid ventricular rates should atrial flutter occur; amiodarone may be an exception. Patients presenting with atrial flutter and rapid rates (and in whom VT is not a consideration) should be treated by slowing of AV conduction with drugs such as intravenous diltiazem. Ablation of the atrial flutter and continuation of the antiarrhythmic drug may be an option for long-term therapy.

Tricyclic Antidepressant Overdose
Clinical Features: Tricyclic antidepressants are second only to analgesics as a cause of serious overdose toxicity. Typical cardiac manifestations include sinus tachycardia, PR and QRS prolongation, and occasionally a Brugada syndrome-like ECG. Hypotension, fever, and coma are other common manifestations of serious toxicity.

Management: QRS duration can be shortened in experimental animals and in humans by administration of $NaHCO_3$ or NaCl boluses. Antiarrhythmic drugs, including beta blockers, are generally avoided. Supportive measures, such as pressors, activated charcoal, and extracorporeal circulation, may be required.

Sudden Cardiac Death and Psychiatric or Neurological Disease: The incidence of SCD is increased in patients with seizure disorders and schizophrenia. It is uncertain whether this reflects specific abnormalities, such as autonomic dysfunction or an unusually high prevalence of cardiovascular disease, or the therapies used to treat the disease. Drug interactions may also contribute. Antipsychotic agents well known to produce marked QT prolongation and torsades de pointes include thioridazine and haloperidol. Another group of generally newer antipsychotic drugs also prolong the QT interval.

Other Drug-Induced Toxicity: *****   LOE: B,C
Anthracycline cardiotoxicity is dose dependent, with intermittent high doses and higher cumulative doses increasing the risk of cardiomyopathy and lethal arrhythmias. Risk factors include younger age, female gender, and use of *trastuzimab*. This form of cardiomyopathy can occur acutely soon after treatment, within a few months of treatment (the so-called subacute form), or many years later. There is an increase in ventricular ectopy in patients receiving *doxorubicin* during the acute infusion period, but this is very rarely of any significance. There is, however, little evidence of reversibility in the *anthracycline*-induced myopathic process.

5-Fluorouracil causes lethal and potentially fatal arrhythmias irrespective of underlying coronary disease during the acute infusion period, the vast majority occurring during the first administration. Cardiac monitoring during the infusion period, especially the first, is recommended for all patients receiving 5-fluorouracil therapy. Symptoms, with or without corresponding ECG changes compatible with cardiac ischemia, should lead to an immediate discon-

tinuation of the infusion. Ischemia should be treated conservatively or conventionally with anticoagulants, nitrates, and calcium channel and beta blockade as required. Although this cardiotoxicity is reversible, 5-fluorouracil sensitizes individuals and should be avoided in the future. Cesium, well-recognized to produce torsades de pointes in animal models, has also been used as "alternate therapy" for malignancy and when torsades de pointes has been reported.

Herbal compounds including *foxglove tea* and *toad venom*, an ingredient of some traditional Chinese medicines, produces clinical toxicity resembling that of digoxin, and in animal models, digoxin-specific antibodies are successful in reversing the toxicity.

Cocaine has both slow offset sodium channel-blocking and QT-prolonging properties. Arrhythmias associated with cocaine ingestion include wide-complex tachycardias suggestive of sodium channel block (and responding to sodium infusion) as well as torsades de pointes. Cocaine also causes other cardiovascular complications that can lead to arrhythmias, notably myocarditis, and coronary spasm.

Coronary spasm has been reported with ephedra and multiple other medications and can present as VF: certain anticancer drugs (for example, *5-fluorouracil, capecitabine, triptans*) used in the treatment of migraines, recreational agents (such as *ecstasy, cocaine*), inadvertent vascular administration of pressor catecholamines, and anaphylaxis due to any one of a wide range of drugs may also have this effect.

Bradyarrhythmias are common (and desired) pharmacological effects of *digoxin, verapamil, diltiazem,* and *beta blockers*. Severe bradyarrhythmias may occur with usual doses in sensitized individuals, particularly those receiving combinations, or in suicidal or accidental overdose. Marked sinus bradycardia is also common with *clonidine.* ■
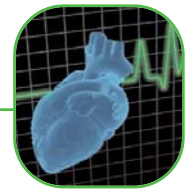
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Part II. Bystander Care

Saving Lives with School-Based CPR/AED Programs: Awareness, Education, Planning, and Partnerships

Introduction

Sudden cardiac death (SCD) in children and adolescents is the leading cause of death in young persons during sports and exercise and is devastating for all involved as well as the community at large. Most episodes of SCD occur in young people who may have no antecedent signs or symptoms of a potentially life-threatening cardiac abnormality. Despite preparticipation screenings of these individuals with history check, physical exam, and/or noninvasive testing, the first presentation in fact may be SCD. This underscores the need for effective secondary prevention strategies including bystander cardiopulmonary resuscitation (CPR) and automatic external defibrillators (AED).

This paper discusses the incidence and etiologies of SCD in children and adolescents, reviews current out-of-hospital cardiac arrest (OHCA) outcomes data, and presents detailed information of unique school-based public access defibrillation programs in Wisconsin (Project ADAM) and Georgia (Project S.A.V.E.). The paper also discusses the methods and challenges of initiating and implementing such programs as well as the efforts of establishing affiliate programs in other states. Finally, and perhaps most encouragingly, we present existing outcomes of sudden cardiac arrest (SCA) resuscitation that has occurred in schools in association with CPR/AED programs both in Wisconsin and Georgia as well as throughout the United States.

Cardiac Disorders Predisposing to Pediatric and Adolescent SCA

SCA in children and adolescents occurs due to a variety of disorders, roughly half of which are genetic and therefore may affect multiple family members (see Table 1, page 48).[1]

In general, causes of pediatric SCA can be either (a) structural or functional (which could be expected to be identified by echocardiography or at autopsy); (b) primary cardiac electrical disorders (most commonly associated with a structurally and functionally normal heart); or (c) others, including the use of illicit drugs and stimulants (for example, cocaine or ephedra) or certain prescription medication (for example, erythromycin, ketoconazole, Tegretol, and so on).

Models of School-Based CPR/AED Programs: Project ADAM and Project S.A.V.E.

Emergency response planning for SCA and placement of AEDs in schools provides an important means of achieving early defibrillation and improving survival in victims of SCA on school grounds. Project ADAM and Project S.A.V.E. have made great efforts—with significant success—in implementing public access defibrillator programs in schools. A review of the background, methods, results, and survival outcomes from these programs is presented in this paper.

Table 1: Differential Diagnosis of Pediatric and Young Adult SCA

Structural/Functional	Electrical
Hypertrophic cardiomyopathy (HCM)*	Long QT syndrome (LQTS)*
Coronary artery anomalies	Wolff-Parkinson-White syndrome (WPW)
Aortic rupture/Marfan syndrome*	Brugada syndrome*
Dilated cardiomyopathy (DCM)	Catecholaminergic polymorphic ventricular tachycardia (CPVT)*
Myocarditis	Short QT syndrome*
Left ventricular outflow tract obstruction	Complete heart block (CHB)
Mitral valve prolapse (MVP)	Other
Coronary artery atherosclerotic disease*	Drugs and stimulants
Arrhythmogenic right ventricular cardiomyopathy (ARVC)*	Commotio cordis
Postoperative congenital heart disease	Primary pulmonary hypertension (PPH)*

* Familial or genetic.

Project Descriptions

Project ADAM: http://www.chw.org/display/ PPF/DocID/26050/Nav/1/router.asp

Project ADAM (Automated Defibrillators in Adam's Memory) is a not-for-profit program of Herma Heart Center at Children's Hospital of Wisconsin, whose mission is to serve children and adolescents through education and deployment of lifesaving programs that help prevent SCA.

Project ADAM began in 1999 after a series of sudden deaths among high school athletes in southeastern Wisconsin. The project began after Adam Lemel, a 17-year-old high school student, collapsed and died while playing basketball. Adam's parents, Patty and Joe Lemel, along with David Ellis, a childhood friend of Adam's, collaborated with Children's Hospital of Wisconsin to create this program in Adam's memory.

Project ADAM is committed to making AEDs universally available to all children and adolescents by being a national resource for implementing public access defibrillation programs in schools as well as working toward eradicating SCD in children through research, education, and prevention initiatives. The three specific goals of Project ADAM include (1) advocacy and education for the issues related to SCD, (2) advocacy for CPR education for all students prior to graduation from high school, and (3) placement of CPR/AED programs in all Wisconsin schools.

Project ADAM has endeavored to provide access to early defibrillation to both young people and adult staff members and visitors to schools in a systematic and efficient way. In order to do so, pediatric health care professionals from Children's Hospital of Wisconsin help schools in the following ways:

- Identify a project coordinator at the school.
- Implement the program.
- Obtain information about acquiring CPR/AED training.
- Develop a school crisis team.
- Recognize risk factors and symptoms of SCA.
- Connect with other schools served by Project ADAM to share best practices.

Resources that are made available to schools include the following:

- The step-by-step guide (now in its third edition) "Public Access Defibrillation (PAD) Implementation Manual: AEDs in Schools"
- The Project ADAM quarterly e-newsletter, which reaches a national audience of more than 4,000 subscribers
- A multipart video resource that is designed to educate viewers about SCA and PAD initiatives. The video presentation is available on the Project ADAM Web site at http://www.projectadam.com and in print (by request).
- Various templates and worksheets to help schools create policies and procedures in regard to public access defibrillation, establish budgets, and maintain their equipment
- Directional signage to identify AED location schools
- A listing of potential training grants to help defray the cost of CPR/AED training for students and staff
- Access to health care professionals via phone, in-person consultation, or e-mail for questions related to SCA or establishing school-based CPR/AED programs

Project ADAM schools agree to use any funds they receive from Project ADAM for a PAD training program, to refer to Project ADAM when promoting their program, to report each year on their progress to Project ADAM staff, and to inform Project ADAM of any event of SCA or during which an AED was used.

In 2005, with the assistance of Project ADAM and generous donations from local partners, the Milwaukee Public School District (MPS) was able to make an initial purchase of 55 AEDs and begin the process of training groups of responders in the schools. In 2006 an MPS school nurse was assigned to develop and implement the program.

The time when defibrillators were actually deployed was preceded by a lengthy discernment period within MPS regarding the long-term commitment that a school-based PAD program requires. Issues related to budget, resource allocation, maintenance, staff training, safety, and liability concerns all had to be appropriately vetted. Ongoing communication focusing on SCA and AED education with stakeholders (district leadership, school administrators, facilities and maintenance, and others), technology demonstrations, and Q&A sessions, ultimately resulted in a decision to go forward.

The goal of the program was to equip all 177 MPS sites with AEDs and to train staff as first responders. A district-wide policy and procedure manual was developed along with site-specific cardiac emergency response plans that were to be clearly documented, communicated, and practiced throughout the year. The location and number of units installed in the MPS was determined by these factors:

- The risk of SCA based on student age (high schools were equipped first, then middle, then elementary)
- Site surveys accounting for building size, dimensions, and barriers to ensure that the unit could be obtained and brought to the victim within three minutes
- Number of students

Students and staff are in-serviced annually with regard to their particular site's response plan (also referred to as "CODE BLUE AED Emergency"). This plan instructs staff and students how to react if an unresponsive victim is found, thereby ensuring that

911 is called and CPR and defibrillation are performed within five minutes of onset of SCA. In order to assist responders in maintaining a high level of skill retention, quarterly defibrillator drills are run at each site, allowing responders to test their skills and cardiac response plans.

Continued financial support from community partners has enabled the purchase of additional AEDs. Currently, there are 219 AEDs installed at 168 MPS sites including schools, offices, football stadiums, community centers, and facilities/maintenance buildings. As mentioned, people at all MPS schools have been trained in CPR and the use of an AED, and all sites have at least one AED.

In Wisconsin, 850 schools (25% of schools in the state) to date have CPR/AED programs. In addition, Wisconsin Senator Russ Feingold and Maine Senator Susan Collins have embraced this innovative program by sponsoring the ADAM Act, highlighting the need for placing AEDs in schools across the nation and calling for establishment of a national clearinghouse. Though the bill was signed into law in 2003, it is awaiting funding/appropriations from the United States Congress.

Project ADAM also provides support and guidance to affiliate sites in other states. Through its affiliate program, Project ADAM helps other hospitals and health care organizations become a comprehensive resource for schools developing PAD programs. This has translated into mobilizing the necessary local, regional, and statewide resources necessary to implement a seamless and sustainable program for PAD placement in all school settings.

The definition of a Project ADAM affiliate is an organization (such as a children's hospital) that has committed itself to providing resources to schools wishing to implement PAD programs in their buildings. It is an organization that is pursuing this commitment through its partnership with Project ADAM at Children's Hospital of Wisconsin (CHW), with other states and affiliate organizations across the nation, and with the Project ADAM administrative staff at CHW. Project ADAM affiliates receive the following:

- Strategic planning assistance
- Affiliate operations training
- All printed material templates
- National research opportunities regarding SCA in youth and AEDs in schools
- Expert advisory council guidance
- Regularly scheduled affiliate meetings (via conference call, video conferencing, or in person)

Project ADAM currently has six affiliate sites in the United States: Project S.A.V.E. at Children's Healthcare of Atlanta; Florida Children's Hospital in Orlando; Children's Hospital of Philadelphia; Children's Hospital of Alabama; Midwest Heart Foundation in Illinois; and Sacred Heart Medical Center in Spokane, Washington. It is anticipated that as affiliate sites are added, there will be additional opportunities for research and continued establishment of best practices/guidelines on a national level.

Finally, new American Heart Association guidelines for CPR were recommended in 2005.[2] The "new CPR" was an evidence-based attempt to improve circulation during CPR. The details of the new regulations are published[2] and were incorporated into Project ADAM and Project SAVE as appropriate. These new recommendations included changes in the ventilation-to-compression ratio, changes in the number of breaths per minute, concentration on chest compression depth (quality of CPR), and minimal interruption of chest compressions.

Project S.A.V.E. (**S**udden Cardiac Death: **A**wareness, **V**ision for Prevention, and **E**ducation) began at Children's Healthcare of Atlanta in August 2004 and became the first state affiliate of Project ADAM in 2005, sharing the above goals. Project S.A.V.E., like Project ADAM, was initiated in response to several student-athlete sudden deaths that occurred in metropolitan Atlanta in 2003. Initial funding of Project S.A.V.E. occurred via the Children's Miracle Network.

The Project S.A.V.E. team typically meets with the school nurse, a school administrator and an individual from the physical education department in order to review the details of a comprehensive school CPR/AED program. Project S.A.V.E. also provides tools for education and awareness as well as fundraising. Project S.A.V.E. does not provide schools with the actual AEDs, but it has provided training grants and assist in training school nurses to function as CPR instructors. Project S.A.V.E. employs a standard manual, forms, a training DVD, and a checklist that schools can use as a roadmap in the process of AED program implementation. Typically, the entire school faculty and staff participate in the educational process. At that point in time, the school is recognized as a Project S.A.V.E. "HeartSafe" school. Project S.A.V.E. HeartSafe schools, similar to Project ADAM schools, file a report whenever CPR or an AED is employed.

The Project S.A.V.E. team at Children's Healthcare of Atlanta has contacts with school nurses throughout the state of Georgia. School administrators, athletic directors, parents, and teachers/coaches help facilitate the development of local school programs. Thus far, Project S.A.V.E. information has been shared with all 180 school districts in Georgia, re-

sulting in a tremendous increase in comprehensive school AED programs in Georgia over the last five years. In addition, a 2008 state law mandates that all high schools with interscholastic sports programs have an AED on the premises.

Project Outcomes
Project ADAM

In Wisconsin, 850 schools (25% of schools in the state) to date have CPR/AED programs and all schools in the MPS system have CPR/AED programs. A cohort study of Wisconsin high schools[3] investigated the prevalence of AEDs and emergency preparedness for responding to SCA. Data and information about past AED utilization for SCA were also collected as part of the study. Seventy percent of Wisconsin high schools responded to the survey. Nearly 95% of responding high schools indicated they already have an AED on school grounds, and 73% reported having two or more. This is the highest reported prevalence of AEDs in high schools to date, and it surpasses what was reported in NCAA Division I institutions (91%).[4] This would seem to indicate that publicly accessible AEDs in the school setting are rapidly becoming the norm—and likely attributable to the advocacy efforts of Project ADAM. The majority of Wisconsin high schools (80%) were also likely to have an emergency action plan (EAP) to respond to medical emergencies, including SCA.

The study identified eight cases of SCA in Wisconsin high schools during the study period (2005–2008), including one case in a 16-year-old male athlete.[3] Collapse was witnessed in all eight cases. CPR was initiated in seven out of eight (87.5%) cases of SCA, and an on-site AED was accessed and applied in all instances. A shock was deployed in six out of seven cases (86%), with two victims receiving two shocks. Three out of eight cases (37.5%) survived to hospital discharge, including the

16-year-old student. Only two cases of SCA had EMS arrival times $\leq$ 3 minutes (range 1.5–3 minutes). Six out of eight cases (75%) had EMS response times $\geq$ 4.5 minutes (mean = 8.6 minutes, range 4.5–13 minutes). All three successful resuscitations had a collapse-to-shock time < 3 minutes.

Since the inception of the MPS system defibrillator program in September 2006, there have been five incidents of on-site SCA: two staff members, one parent, and two children aged 13 and 12. In four of the five incidents, bystander CPR and AED deployment occurred within five minutes of onset of SCA. In all four cases, a viable rhythm was restored. In the fifth case, SCA was not recognized, no CPR or AED was utilized, and the victim died at the scene.

Since the start of the program in 1999, the lives of at least 16 individuals who experienced SCA have been saved in Project ADAM Wisconsin schools. These survivors include 6 adolescents (aged 11 to 16) and 10 adults. These individuals include students, visitors to schools, and school staff.

Project S.A.V.E.

A 2008 survey by the Georgia Department of Education documented that 65% of all school districts had AEDs in at least high school and middle schools. In addition, a comprehensive Project S.A.V.E. HeartSafe School program has been recognized in 728 schools (32% of the schools). Between October 2004 and May 2010, there have been 49 (26 students and 23 adults) sudden cardiac events reported in Georgia schools. The first reported school save was in December 2007 and was a result of improvements to school CPR/AED programs and emergency response plans. Since that time, 22 SCA victims (45%)—10 students and 12 adults—have survived to hospital discharge. Just as in Wisconsin, the SCAs occurred at all levels and not just in high school athletes (2 elementary school, 6 middle school, and 18 high school students).

Cost-Effectiveness

A study to determine the cost-effectiveness of Project ADAM implementation in the MPS system was reported in 2004.[5] This analysis showed that with a societal willingness to pay $100,000 per life per year saved, Project ADAM is cost-effective if one life per year is saved. This study concluded that Project ADAM implementation in schools in the United States is associated with a favorable incremental cost-effectiveness ratio.

Discussion

According to the American Heart Association, on any given school day as much as 20% of the combined U.S. adult and child population can be found in schools. Schools are the primary daytime location for children, serve as community gathering sites for sporting and other events, and at times are densely populated with adults and other visitors on campus. Given that SCA is the leading cause of death in the United States and the leading cause of death in young athletes on the playing field, there are compelling reasons to implement effective strategies to prevent SCD.

Every school should have an emergency response plan for SCA with written policies and procedures to ensure an efficient and structured response to a cardiac emergency.[6,7]

In recent years, significant attention has been raised about ensuring that there are proper emergency preparations at youth sporting practices and competitions. SCA in children and young athletes is a devastating event with tremendous impact on a school and local community. As demonstrated by Project ADAM and Project S.A.V.E., school-based

CPR/AED programs are a critical component to protecting young people who are competing in sports or exercising from a catastrophic outcome in the event of a cardiac emergency. Prompt recognition of SCA is the first step to an efficient emergency response. Potential first responders to SCA in a child or athlete, such as coaches, teachers, and athletic trainers, must maintain a high index of suspicion for SCA in any collapsed and unresponsive student. Resuscitation can be delayed because SCA is mistaken for a seizure, agonal respirations are mistaken for normal breathing, or rescuers inaccurately assess the presence of a pulse. Brief seizurelike activity has been reported in more than 50% of young athletes with SCA.[8] To avoid potentially fatal delays in resuscitation, a collapsed and unresponsive athlete or student should be treated as having had a cardiac arrest and an AED applied as soon as possible for rhythm analysis and defibrillation if indicated.[5]

OHCA is uncommon in children compared to adults, with incidence rates ranging from 2.6 to 19.7 cases per 100,000 annually in a systematic review by Donoghue and colleagues.[9] Although the physiology of cardiac arrest in children is different from that in adults, the survival outcomes for OHCA are similarly dismal: only 9.4% of pediatric patients receive bystander CPR, and only 4.7% of those who do not receive bystander CPR survive to hospital discharge.[7] Survival following exercise-related SCA also has been historically low. Drezner et al. reported a seven-year analysis of survival trends in the United States following exercise-related SCA in youth. From 2000 to 2006, 486 total cases of exercise-related SCA were identified in individuals aged 5 to 22; the overall survival rate was only 11% (with a range of 4% to 21%) per year.[10] Two studies suggest that the frequency of SCA in adolescents and young adults may actually be increasing.[11,12]

The prevalence of documented ventricular fibrillation (VF) in the pediatric OHCA population is less than that in adults, but recent data suggest pediatric patients with OHCA have an initial cardiac rhythm amenable to defibrillation (VF or pulseless ventricular tachycardia) 10% to 19% of the time.[13,14] It is likely that a larger percentage of pediatric victims have VF or rapid ventricular tachycardia at the time of collapse but that the rhythm has already deteriorated to asystole before the first rhythm analysis.

In young persons with exercise-related SCA, the likelihood of VF arrest is substantially higher. In high school athletes with SCA, 93% had a shockable rhythm when the SCA was witnessed, and defibrillation occurred within a mean of 3.6 minutes (median 2.4 minutes) from collapse.[8]

This would suggest pediatric patients suffering OHCA could expect to benefit from publicly accessible defibrillators to reduce time to defibrillation, similar to the survival benefits seen in adults.

Witnessed cardiac arrest, early bystander CPR, and early defibrillation have been associated with an improved likelihood of survival and represent important links in the "chain of survival."[15] Of these, the single most important factor influencing survival following OHCA is the time from arrest to defibrillation.[16] A consistent call-to-shock time interval of less than five minutes cannot be reliably achieved in many EMS systems. Public access defibrillation, therefore, often provides the greatest opportunity to defibrillate victims of sudden cardiac arrest within a collapse-to-shock interval of three to five minutes as recommended by the American Heart Association.[6,7,16] In an early study of AED use by first responders, Weaver and colleagues found that survival rates improved 30% when trained firefighters administered AED-delivered shocks to VF/VT victims

prior to EMS paramedic arrival—compared to only 19% when only CPR was administered prior to the arrival of the paramedics.[17] Since then there have been several prospective studies demonstrating the increased effectiveness of publicly accessible AEDs for the treatment of SCA in public settings such as airports, airlines, and casinos, with survival rates ranging from 41% to 74%.[18–21] The PAD Trial was a recent multicenter, randomized trial comparing survival from OHCA in community centers with and without on-site AEDs. The PAD Trial confirmed that victims of SCA were twice as likely to survive if AEDs were publicly available.[19] Limited published data is available for employment of publicly accessible defibrillators for treatment of SCA in younger populations. Initial reports raised questions about whether defibrillation with AEDs in this population would provide the same survival benefit seen in older patients who predominantly have coronary artery disease. Drezner and colleagues[4] reported details of nine cases of SCA in intercollegiate athletes with SCA who received bystander CPR and AED defibrillation, yet only 11% (one out of nine) survived to hospital discharge. However, recognition of SCA and subsequent resuscitation procedures may have been delayed in several of these cases due to the reported presence of seizure-like activity, mistaking agonal gasping for normal breathing, and falsely assessing the presence of a pulse. In addition, four of the nine individuals received defibrillation from responding EMS, suggesting that the reported response times may have been underestimated.

More recently, an investigation of 1,710 U.S. high schools suggests that SCA victims, when treated with early CPR and defibrillation, receive significant survival benefit with consistent use of on-site school-based AEDs. In 14 cases of SCA in high school athletes (aged 14 to 17 years), the rate of survival to hospital discharge was 64%; an on-site AED was employed in 79% of cases (11 of 14).[8]

Summary and Conclusions

SCD remains a leading cause of mortality in children and adolescents and is likely more common than represented by initial estimates. Schools are a strategic location for serving large concentrations of people at risk for SCA, and school-based CPR/AED programs have tremendous potential to improve survival in both children and adults who suffer SCA on school grounds. Survival data from school AED programs is encouraging and far exceeds the dismal outcomes typically found for OHCA. School-based CPR/AED programs establish a community of first responders, markedly improve the likelihood of survival for students and nonstudents with SCA, and should be encouraged in all schools. Project ADAM and Project S.A.V.E. provide two successful models schools can use to create their own CPR/AED programs and further improve survival from OHCA in school and athletic settings.

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Development of an Integrated Community-Based Program to Treat Sudden Cardiac Arrest

Background: Successful resuscitation of victims of out-of-hospital sudden cardiac arrest (SCA), which is usually precipitated by ventricular fibrillation/tachycardia (VF/VT), remains a major challenge. While coronary artery disease is the most frequent underlying cause of SCA in adults, acquired and congenital heart disease can effect SCA in younger individuals as well. Advances in cardiopulmonary resuscitation (CPR) and the greater prevalence of automated external defibrillators (AEDs), when combined with post-resuscitation strategies such as hypothermia to ameliorate anoxic brain injury and expeditious coronary and cerebral artery reperfusion when indicated, improve survival and reduce functional disabilities. Minimizing SCA morbidity and mortality requires a systems approach that integrates rapid bystander-administered CPR and defibrillation, prompt emergency medical services (EMS) response times, the induction of therapeutic hypothermia, and expeditious transport and rapid reperfusion.

Methods: A working group (the Cardiac-Stroke Committee of the Hillsborough County Emergency Medical Planning Council [EMPC]) composed of representatives from all four Advanced Life Support (ALS) EMS, nine hospitals, and other community stakeholders has developed an integrated care approach to ST-elevation myocardial infarction (STEMI), acute stroke, therapeutic hypothermia, and SCA care based in part on the trauma model. The group meets quarterly to coordinate and integrate the programs in those areas as well as to develop and monitor performance improvement data collection inclusive of outcome measures.

Results: Over 1,000 AEDs have been placed in Hillsborough County, Florida. In order to develop a large cadre of people capable of performing bystander CPR/AED to SCA victims, a program is being initiated to educate all Hillsborough County Public Schools ninth-grade students in CPR/AED based on the American Heart Association (AHA) CPR Anytime™ syllabus in the Health Opportunities through Physical Education (H.O.P.E.) course, a graduation requirement. Subsequent plans are under way to expand CPR/AED training to the students' family members and other residents of the county. The largest EMS provider employs in-ambulance hypothermia protocols and transports SCA survivors to the four area hospitals capable of performing primary percutaneous coronary intervention (PCI) and administering therapeutic hypothermia and other treatments to minimize anoxic brain injury.

Conclusions: The Hillsborough County system integrates all phases of prehospital and hospital care for SCA victims. This approach was designed to increase the number of residents trained in EMS activation, CPR, and AED use and to integrate the performance of bystander CPR with EMS and hospital-based protocols. The ultimate objective is to increase the survival rates of SCA victims and optimize their long-term neurologic and functional outcomes.

Statement of Evidence Supporting Practice

More than 295,000 Americans suffer an out-of-hospital sudden cardiac arrest (SCA) yearly.[1] Successful resuscitation of victims of SCA, which is usually precipitated by ventricular fibrillation/tachycardia (VF/VT), remains a major challenge, with reported median survival rates to hospital discharge that range from 4.6%–7.9%.[1,2] While coronary artery disease is the most frequent underlying cause of SCA in adults, acquired and congenital heart disease and other conditions (for example, drowning or trauma) can effect SCA in younger individuals as well. Advances in cardiopulmonary resuscitation (CPR) and the greater prevalence of automated external defibrillators (AEDs), when combined with post-resuscitation strategies such as therapeutic hypothermia to ameliorate anoxic brain injury and expeditious coronary artery reperfusion when indicated, improve survival and reduce functional disabilities.[3]

Objective

Maximizing SCA survivorship requires a systems approach based on the "chain of survival" algorithm (*see* Figure 1, below), which integrates rapid bystander-administered CPR, early defibrillation, prompt emergency medical service (EMS) response times, rapid transportation to an appropriate hospital, and the administration of therapeutic hypothermia and reperfusion protocols[4,5] that have been incorporated in the most recent revision of the American Heart Association (AHA) guidelines.[6] In order to meet the demands of the "chain of survival" approach, a program has been initiated throughout Hillsborough County, Florida, with the following goals:

- Increase the number of residents capable of and confident in performing compression-only CPR and operating an AED.
- Increase the availability of AEDs.
- Integrate bystander CPR/AED and EMS response.
- Accelerate the induction of therapeutic hypothermia after return of spontaneous circulation (ROSC) to preserve cerebral function.
- Implement destination protocols to transport patients to the most appropriate hospital.
- Increase public awareness of the need to become proficient in CPR and use of an AED; that is, to get involved in the care of an SCA victim.

Background

Hillsborough County comprises 1,072 square miles of west-central Florida and has a population of approximately 1.2 million residents.[7] Though the city of Tampa is the county seat, unincorporated suburban and rural areas comprise 84% of the county.[7] Hillsborough County Public Schools (HCPS), the only public school system in the county, educates 191,192 students.[8]

Figure 1: The Chain of Survival (modified by HCFR Division Chief David Travis)

Early Access → Early CPR → Early Defibrillation → Early Advanced Care → Comprehensive Cardiac Care

The county is served by four Advanced Life Support (ALS) EMS agencies and nine acute care hospitals, four of which are percutaneous coronary interventional (PCI) capable. A working group, the Cardiac-Stroke Committee, a subcommittee of the Hillsborough County Emergency Medical Planning Council (EMPC), is a volunteer organization composed of representatives from all of the county's EMS services and hospitals plus other stakeholders. The group has developed an integrated care approach to ST-elevation myocardial infarction (STEMI), acute stroke, hypothermic therapy, and SCA based in part on the trauma model, and it meets quarterly to coordinate and integrate the programs in those areas as well as to develop and monitor performance improvement data collection inclusive of outcome measures. Its findings are reported to the EMPC and ultimately to the Hillsborough County Commission.

The Cardiac-Stroke Committee's program for out-of-hospital sudden cardiac arrests focuses on the following objectives:

1. Increase the number of residents capable of performing CPR and operating an AED. The entry point for this effort is the Hillsborough County Public Schools Health Opportunities through Physical Education (H.O.P.E.) course, which is taken by all ninth-grade students and required for graduation. The material is taught at points where instruction in compression-only CPR and AED operation, based on the AHA CPR Anytime™ program, has been incorporated into the syllabus. The students then are assigned to disseminate their knowledge to their relatives.
2. Increase public awareness of the benefits and simplicity of bystander-administered compression-only CPR and AED use by public service announcements.

3. Place a large number of AEDs throughout the county.
4. Initiate EMS-administered post-resuscitation hypothermia induction.
5. Transport survivors to hospitals providing therapeutic hypothermia.

Methods/Results

Student Education. In 2008 Hillsborough County Fire Rescue (HCFR) Chief David Travis and Drs. Charles Sand and Joel Strom, under the auspices of the Hillsborough County EMPC, proposed developing a curriculum to educate HCPS students in the techniques of bystander CPR and AED use. This proposal led to a meeting in August 2009 with Ken Otero, deputy superintendent of HCPS, and to a subsequent meeting with Steve Vanoer, supervisor, K–12 physical education and health. Over the course of the next year, a curriculum was developed to incorporate this program into the H.O.P.E. course, which is taken by approximately 14,000 ninth-grade students a year. In a pilot program supported by the AHA, physical education teachers from six middle and high schools introduced their students to the CPR Anytime kits during the 2009–2010 school year.

The full program was initiated at the beginning of the 2010–2011 school year. During a professional study day, held on August 18, 2010, and supported by a grant from the AHA, all high school physical education and health teachers received CPR Anytime program training. These teachers are now using the H.O.P.E. course curriculum (*see* Figure 2, page 60) to provide instruction on basic bystander CPR/AED techniques. Students receive anywhere from two to eight hours of instruction and evaluation, and they are given the homework assignment to educate friends and family members about basic lifesaving skills. Student performance will be assessed

Figure 2: Outline of the Curriculum for the CPR/AED Unit for the H.O.P.E. Course

WEEK 31

Focus: CPR/AED and Heimlich (HE Ch. 28)
Definition: CPR Anytime™ Program
Training for students, ABCs of CPR,
Steps to adult Heimlich procedures

Class activities: 1. Hands-on practice
2. Lecture, guided notes, HE/PF textbook, supplemental materials (CPR Anytime Kit)

Outside activities: Continue hands-on practice

Source: Hillsborough County Public Schools

and tracked after course completion using the HCPS Achievement Series software. Refresher sessions are currently in development.

In order to further increase the number of people in the county who are competent in the use of CPR/AED, plans are being developed to expand this program to include private school students and other Hillsborough County residents. An innovative approach currently in development is the incorporation of CPR/AED education into *The Amazing You* exhibit at the Tampa Museum of Science and Industry (MOSI), the sixth largest science center in the United States. Each year the museum hosts 800,000 visitors. Many visitors are students, their parents, and other residents of the region who would be ideal candidates for this type of educational program.

Dissemination of CPR and AED Information. With the support of Hillsborough County Commissioner Rose Ferlita and her staff, a community information program was initiated as the HCPS curriculum was

being developed and implemented. The program includes public service announcements, the first of which was rolled out in October 2010; it can be viewed at http://www.youtube.com/watch?v=5klvmm_Wj-8.

Automated External Defibrillator Placement. Over 1,000 AEDs have been placed in the county, particularly in highly frequented areas such as airports, sports complexes, and office buildings. An Emergency Preparedness Grant allowed the HCPS to purchase 314 AEDs and to provide CPR/AED and first aid training for more than 2,800 district staff. To date, at least four known "saves" have been credited to this program. This illustrates the important point that the workforce is confident in dealing with the daily emergencies of a school district and the larger community.

*Post-Resuscitation Therapeutic Hypothermia (***see** *Figure 3, page 61).* Hypothermic therapy for SCA victims is now a major component of post-resuscitation care in Hillsborough County. The paradigm for the therapeutic hypothermia therapy model was developed as a result of direct facilitated communications among the many stakeholders who had encountered an SCA survivor. These communications required both finesse and determination to overcome the real and perceived obstacles posited by the multiple disciplines involved in the patient care continuum.

The HCFR first implemented post-resuscitation field-initiated hypothermic therapy in 2006. While hypothermia was initially induced by the placement of external cold packs on the neck and axillae, this approach was supplemented by the I.V. administration of chilled saline that is currently recommended by the AHA.[9] To facilitate this technique, refrigerators were installed in ambulances. Upon ROSC,

Figure 3: Comparison of Protocols for EMS Agencies in Hillsborough County

VT/NF	Field Therapeutic Hypothermia	Destination Protocol for ROSC and VT/VF Patients to PCI Center
HCFR	Yes	Yes
TFR	No	Yes
TTFD	In development	Yes
PCFR	In development	No

HCFR, Hillsborough County Fire Rescue; TFR, Tampa Fire Rescue; TTFR, Temple Terrace Fire Department; PCFR, Plant City Fire Rescue; ROSC, return of spontaneous circulation.

paramedics immediately begin rapid infusion of a liter of chilled saline through an intravenous or intraosseous line coupled to a pressure infuser to decrease the patient's core temperature by a rate of 1.9ºC/hour. The target temperature is 33ºC. Patients are sedated with midazolam and either veccuronium or rocuronium to prevent shivering. The bags of chilled saline are labeled "Medication Added" to alert emergency department (ED) staff that this treatment is under way. A key objective of the hypothermia initiative was to build relationships between the specialists in the units and the ED physicians to ensure the continuum of hypothermic therapy.

Currently, the HCFR is the only EMS agency in the county that administers prehospital therapeutic hypothermia. The HCFR serves the largest region of the county, and its transportation times from SCA scene to hospital can be as long as 40 minutes. However, two other EMS agencies (Temple Terrace Fire Department and Plant City Fire Rescue) are establishing field-initiated therapeutic hypothermia protocols based on the HCFR model. Tampa Fire

Rescue has no plans to offer therapeutic hypothermia at this time due to its short transport times. Almost concurrent with HCFR's initiation of prehospital therapeutic hypothermia—and stimulated by media reports of noteworthy cardiac arrest saves that emphasized hypothermia as a key component—Hillsborough County hospitals began ED and intensive care unit (ICU) therapeutic hypothermia programs. The hospitals have made significant investments in the technology to accomplish the initiation of cooling in the hospital or the continuance of field-initiated cooling efforts. Currently, all Hillsborough County hospitals provide therapeutic hypothermia for SCA patients with ROSC. While some use nonmechanical methods, they are all in the process of acquiring the technology for device-administered hypothermia. A number of devices are employed, among them the Arctic Sun Temperature Management System™ (Medivance, Inc., Louisville, Colorado) and the ZOLL Intravascular Temperature Management™ system (ZOLL Medical Corp., Chelmsford, Massachusetts).

Destination protocols are in place to optimize the transportation of SCA survivors to hospitals performing therapeutic hypothermia. The Temple Terrace Fire Department transports all patients with ROSC as well as VF/VT patients to PCI-capable hospitals. The HCFR and Tampa Fire Rescue screen all patients with ROSC after SCA for STEMI. Those with STEMI are taken only to PCI-capable hospitals. All others are transported to the closest appropriate hospital depending on the patient's clinical status. Plant City Fire Rescue transports survivors to the nearest hospital, but it is currently reevaluating its protocol, according to Division Chief James Wilson.

Finally, in order to coordinate all of the above programs, integrate them with the countywide ST-elevation myocardial infarction and stroke pro-

grams, and foster collaboration among institutions, the Cardiac-Stroke Committee and the EMPC have recurring agenda items relating to the performance of these programs. Quarterly updates and discussion of issues have been useful to maintain network cohesion and to develop novel therapeutic and educational strategies.

Discussion

Improvement in the dismal survival outcomes reported after out-of-hospital SCA requires new paradigms employing system engineering approaches (for example, reperfusion and transportation to the most appropriate hospital) to achieve timely resuscitation, early intervention with cerebral preservation techniques, and rapid identification and correction of the underlying cause. It is imperative that there be close collaboration among all participants—particularly between bystanders and EMS personnel as most episodes occur at home, work, or school. Four major advances have driven the Hillsborough County effort. First is the recognition that the more people trained in CPR/AED use, the greater the probability that a trained bystander will be near an SCA victim and that the victim will receive appropriate therapy with concomitant improvement in survival.[3,10] This has led to the design and implementation of compression-only CPR and AED instruction as part of the public school health education curriculum, an innovative component of this program. A number of recent studies have demonstrated that the outcomes of compression-only CPR administered by those with no or limited training are equivalent to the outcomes of standard CPR,[11,12] even for those between 1 and 17 years of age with SCA due to cardiac causes.[13] This approach is supported by the most recent AHA guidelines.[14] Compression-only CPR will encourage better compliance among untrained individuals and others who may be reluctant to perform rescue breathing.

Instruction does not have to be extensive to achieve competence. Lynch et al.[15] reported that people aged 40–70 can be trained in CPR with 30 minutes of self instruction. Roppolo et al.[16] reported that a 30-minute CPR/AED course was as effective both immediately and six months later compared to standard courses. Young age is not a barrier to acquiring these skills. Fleischhackl et al.[17] reported that children as young as 9 years old could learn and retain CPR skills including operation of an AED. Physical strength is a concern as children under the age of 13 often lack the physical strength to perform effective chest compressions. However, by age 13 or 14, the average age for ninth graders, 45% of children have the strength to perform effective CPR.[18] Thus, we can expect that ninth-grade students have the cognitive capacity to learn and retain the skills required to perform CPR and operate an AED. While only half will initially have the physical strength to perform effective chest compressions, they will be able to achieve that capacity within one to two years.

Based on animal data that early induction of hypothermia following ROSC reduces neurologic injury,[19] therapeutic hypothermia protocols were initiated for comatose SCA survivors. Most protocols expose a comatose survivor to temperatures of 32ºC–34ºC for 12 to 24 hours followed by gradual rewarming. A number of studies have reported the efficacy of this approach.[20,21] The strongest evidence for the effectiveness of therapeutic hypothermia is the ROSC in comatose patients who have suffered SCA due to VF/VT, and this indication carries the strongest recommendation in the most recent AHA guidelines.[9] Initially, therapeutic hypothermia was induced upon admission to the ED or ICU. Based on recent randomized trials demonstrating the feasibility and safety of starting therapeutic hypothermia in the field either before or after ROSC,[22–24] some EMS agencies (for example, those in Richmond,

Virginia, and Raleigh, North Carolina) have integrated this practice into their protocols. In a recently presented abstract, Cabanas and colleagues reported an adjusted odds ratio of 8.6 for survival in those patients with VF/VT with ROSC who received prehospital hypothermic therapy with cold saline compared to those who did not receive hypothermia.[25] Survival benefit was also observed for those with pulseless electrical activity and asystole. While prehospital induction of hypothermia achieves more rapid cooling, a recent study failed to demonstrate a survival benefit of prehospital compared to hospital-initiated hypothermia.[26] The time intervals between the onset of SCA, restoration of circulation, and lowering of core temperature may affect the magnitude of benefit. As the HCFR often has the longest transit distances of the four Hillsborough County EMS agencies, its initiation of prehospital hypothermia may prove to be the most efficacious course of action. Similarly, two other EMS agencies that also serve suburban and rural areas of the county are implementing prehospital hypothermia protocols. Finally, the transport of the patient with ROSC or VF/VT to a hospital capable of rapidly diagnosing correctable causes for the SCA and administering reperfusion therapy is extremely important for optimal outcomes.

In summary, improvement in the survival of SCA victims will require a coordinated, community-based systems engineering approach involving all aspects of diagnosis and care with the goals of rapid recognition, circulation support to preserve vital organ function, restoration of effective spontaneous cardiopulmonary function, and correction of the underlying cause for the SCA. Systems that include therapeutic hypothermia are being developed in a number of regions.[27] The multifaceted program developed in Hillsborough County features a central steering committee that coordinates the integration of state-of-the art care and novel educational initiatives.

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CARES: The Cardiac Arrest Registry to Enhance Survival

System Attributes of an Out-of-Hospital Cardiac Arrest Surveillance Registry

Statement of Evidence Supporting Practice

More than 295,000 cases of out-of-hospital cardiac arrest (OHCA) occur each year in the United States, approximately 60% of which are treated by emergency medical services (EMS).[1] Most are caused by a lethal heart rhythm disturbance called ventricular fibrillation (VF). VF occurs twice as often in men as in women. The odds of surviving OHCA can be increased dramatically with the following four time-sensitive interventions, collectively termed the "chain of survival"[2]:

- Rapid activation of EMS via 911
- Early initiation of cardiopulmonary resuscitation (CPR)
- Early defibrillation
- Rapid delivery of definitive care

Although consensus guidelines for treatment do exist, rates of OHCA survival vary widely from one community to another. A recent study found up to fivefold differences between participating communities.[3]

In 2004 the American Heart Association (AHA) issued a call for an integrated method of OHCA data collection from hospitals, EMS, and communities that would identify opportunities to improve care and enable valid comparisons of performance across systems.[4] To meet this need, the Centers for Disease Control and Prevention (CDC) and the Department of Emergency Medicine at the Emory University School of Medicine developed the Cardiac Arrest Registry to Enhance Survival (CARES). This report (*see* Table 1 on page 66) summarizes the initial development and expansion of CARES.

Objective

Although the benefits of systematic data collection for public health surveillance and performance improvement are well known, few communities currently monitor EMS treatment and outcomes of OHCA.[5] In 2006 the Institute of Medicine (IOM) noted that OHCA may be a valuable "sentinel condition" for assessing the overall quality of EMS care. This is based on the knowledge that successful treatment of OHCA requires rapid delivery of a sequence of time-critical actions—the aforementioned "chain of survival." Communities that achieve high rates of OHCA survival probably do equally well with other time-critical emergencies.[6]

CARES was created to provide EMS jurisdictions of every size with a simple, standardized manner for collecting OHCA data while maintaining patient privacy. Communities currently participating in CARES represent an estimated 20 million people. By collecting the minimum number of data elements in a flexible yet consistent manner, CARES allows communities to benchmark performance without the burden of lengthy, time-consuming data collection. The data are used to pinpoint opportunities for

Table 1: Demographic and Survival Characteristics of OHCA
October 1, 2005–December 31, 2010*

Characteristic	National (*N* = 31,894)
Age	***N* = 31,831**
Mean (SD)	63.9 (18.3)
Median	65
Gender (%)	***N* = 31,882**
Female	12,380 (38.8)
Male	19,502 (61.2)
Race (%)	***N* = 31,811**
American Indian/Alaskan	177 (0.6)
Asian	463 (1.5)
Black/African-American	8,625 (27.1)
Hispanic/Latino	1,703 (5.3)
Native Hawaiian/Pacific Islander	176 (0.6)
White	12,357 (38.8)
Unknown	8,310 (26.1)
Location of Arrest (%)	***N* = 31,894**
Educational Institution	80 (0.3)
Farm	4 (0.01)
Health Care Facility	623 (2.0)
Home/Residence	21,165 (66.4)
Hospital	117 (0.4)
Industrial Place	222 (0.7)
Jail	135 (0.4)
Nursing Home	4,276 (13.4)
Other	872 (2.7)
Place of Recreation	426 (1.3)
Public/Commercial Building	1,885 (5.9)
Residence/Institution	407 (1.3)
Street/Highway	1,544 (4.8)
Transport Center	138 (0.4)
Type of Initial Rhythm (%)	***N* = 31,849**
V-fib/V-tach	5,993 (18.8)
Asystole	14,328 (45.0)
Idioventricular/PEA	6,182 (19.4)
Unknown Shockable Rhythm	1,609 (5.1)
Unknown Unshockable Rhythm	3,737 (11.7)

Table 1: Demographic and Survival Characteristics of OHCA October 1, 2005–December 31, 2010* (continued)

Characteristic	National (*N* = 31,894)
Arrest Witnessed (%)	***N* = 31,888**
No	16,772 (52.6)
Yes	15,116 (47.4)
Arrest After Arrival of EMS or FR? (%)	***N* = 31,892**
No	28,483 (89.3)
Yes	3,409 (10.7)
Who Initiated CPR (%)	***N* = 31,853**
Not Applicable	23 (0.1)
Total Bystanders (LP+LPFM+LPMP)[†]	10,603 (33.3)
First Responder	11,342 (35.6)
Emergency Medical Services (EMS)	9,885 (31.0)
Who First Applied Automated External Defibrillator (%)	***N* = 31,879**
Not Applicable	95 (0.3)
Total Bystanders (LP+LPFM+LPMP)[†]	1,174 (3.7)
First Responder	12,217 (38.3)
Emergency Medical Services (EMS)	18,393 (57.7)
Return of Spontaneous Circulation in Field (%)	***N* = 31,886**
No	20,857 (65.4)
Yes	11,029 (34.6)
Pre-hospital Outcome (%)	***N* = 31,894**
Dead in Field	6,831 (21.4)
Ongoing Resuscitation in ED	17,268 (54.2)
Pronounced in ED	7,795 (24.4)
Overall Survival (%)	***N* = 31,894**
Overall Survival to Hospital Admission	8,359 (26.2)
Overall Survival to Hospital Discharge	3,039 (9.5)
With Good or Moderate Cerebral Performance	2,198 (6.9)
Utstein Survival (%)	***N* = 4,249**
Witnessed by bystander and found in shockable rhythm	1,278 (30.1)
Utstein Bystander Survival (%)	***N* = 1,491**
Witnessed by bystander, found in shockable rhythm, and received some bystander intervention (CPR by bystander and/or AED applied by bystander)	703 (47.1)

* Excludes unworked arrests and arrests of non-cardiac etiology.

[†] LP, layperson; LPFM, layperson family member; LPMP, layperson medical provider.

improvement, such as streamlining 911 call handling, enhancing community-based CPR training, and repositioning EMS units to reduce response times.

CARES is a secure, Web-based data management system that allows participating communities to easily enter OHCA data and generate summary reports. Established in 2005, CARES was pilot-tested in Atlanta and then replicated in a six-county area of metropolitan Atlanta; it was expanded on a national level in 2006. CARES provides its participating 911 centers, EMS agencies, and communities with confidential online access to their statistics, which they can then compare to anonymous aggregate data at the local, state, or national level. Standard reports automatically calculate local 911 response intervals, critical community interventions (bystander CPR and public access defibrillation [PAD] use), and community survival rates. Trends are displayed as well.

The methods used to enter, link, and display 911, EMS, and hospital outcome data (https://mycares.net) are described in detail elsewhere.[7] The CARES database is maintained by the Sansio Corporation (Duluth, Minnesota) through a contract with the Emory University School of Medicine.

Background/Methods

CARES is a surveillance registry with a quality improvement purpose that can best be described using system attributes found in the CDC document "Evaluating Public Health Surveillance Systems."[8] These attributes, discussed below, include simplicity, flexibility, data quality, acceptability, sensitivity, representativeness, timeliness, and stability.

Simplicity. CARES was designed explicitly to facilitate the timely collection, analysis, and reporting of

OHCA data for use in improving emergency cardiac care at the local level. Only data elements that are essential to this goal are collected. Because EMS personnel work in a time-pressured, stressful, and resource-constrained environment, CARES was designed with simplicity as a fundamental attribute.

Flexibility. Although CARES specifies essential data elements that all agencies must report, it accommodates a variety of input methods (for example, scanned paper form, uploaded data file, online entry). Additional supplemental fields may be collected at an agency's discretion to address local interests or concerns. Automated links and reminders have been incorporated into the data-entry process to reduce the burden of participation in an effort to make the program widely acceptable and, ultimately, sustainable as an ongoing surveillance registry.

Data quality. A data dictionary provides users with clear and concise definitions of each variable in the registry. CARES software includes imbedded error checks and limits that highlight outlier values to improve quality. All entries are double-checked by a CARES data analyst before a record is stripped of individual identifiers and permanently entered in the registry.

Acceptability. CARES is currently used by numerous 911 centers, 54 EMS agencies, and 340 hospitals in 39 communities representing 22 states. None have withdrawn from the registry during the first four years of operation.

Sensitivity. CARES captures all OHCA events of presumed cardiac etiology during which resuscitative efforts were initiated (CPR and/or defibrillation). A case is excluded if (1) EMS personnel determine that the arrest was due to a noncardiac etiology (for example, trauma, overdose, drowning, or respiratory

arrest) or (2) out-of-hospital resuscitation was not attempted based on local protocols (for example, obvious signs of death such as dependent lividity, rigor mortis, or decomposition). Based on initial audits by CARES site directors in the first 15 communities to use it, the registry is capturing 95% of eligible cases. With the exception of the victim's race, a social construct typically determined by self-report,[9] all essential elements of the data set are being consistently reported. Missing data ranges from 25% for victim race to a low of < 1% for patient name (used to link records prior to de-identification). Hospitals are reporting outcome data in 99% of cases.

Representativeness. CARES does not represent a random sample of communities. Rather, it is being offered to those that express a desire to participate in the system. However, the initial output of data from the registry is wholly in line with the published experiences of other communities with regard to OHCA (data shown below).

Timeliness. The typical time from arrest to EMS data submission is one week (daily in sites with electronic patient care records). At the end of each month, delinquent reports and missing data fields are requested from each agency by the CARES data analyst who provides oversight of the data collection and auditing processes. This regularly scheduled data collection and review process ensures compliance with reporting deadlines and allows for longitudinal benchmarking of key performance indicators. A nationwide report for external benchmarking purposes is distributed to participating agencies and communities six times a year.

Stability. During its first four years of operation, CARES has expanded from a single 911 center, an EMS service, and 30 hospitals to a nationwide registry that currently covers a population base of 20 million people. A number of communities are waiting to join the program. Considering the scope of the program, the budget for CARES is extremely modest. Participation requires little in the way of cost or effort at the local level.

Results

The full registry includes 40,273 cases submitted to CARES between October 1, 2005, and December 31, 2010. Of these cases, 8,379 were excluded because the arrest was due to a non-cardiac etiology. The remaining 31,894 cases of presumed cardiac etiology were included in Table 1, which displays the demographic, clinical, and EMS characteristics of the dataset. Descriptive data were categorized based on patient demographics (age, sex, race/ethnicity), aspects of the event (for example, witnessed, unwitnessed, bystander intervention), and incident location (such as home, street, airport, and so on). Retention of incident location allows for the geographic information system (GIS) mapping of events. This enables EMS services to examine neighborhood characteristics as well as individual factors and system issues that might influence the likelihood of surviving OHCA. To preserve patient confidentiality, individual identifiers were stripped from each record after a CARES analyst confirmed the completeness and accuracy of each record.

The overall survival to hospital discharge of the remaining cases was 9.5% (*n* = 3,039/31,894). The survival rate among patients witnessed to have collapsed and found in VF (the group most likely to benefit from optimal EMS care) was 30.1%. Bystander performance of CPR and usage of automated external defibrillators (AEDs) was 33.3% and 3.7% respectively. A total of 6,831 (21.4%) were pronounced dead in the out-of-hospital setting, based on EMS agency protocols.

Discussion

CARES was designed to help communities determine how well they are achieving key links in the "chain of survival" and identify opportunities to improve performance and boost outcomes. For example, CARES data may provide insights into streamlined call handling at 911 centers, enhanced community-based CPR training, strategic placement of AEDs, or reallocation of ambulances to decrease response time intervals. CARES data have already been used to guide placement of public access AEDs in Fulton County, Georgia, and have prompted efforts to improve the handling of 911 calls to suspected cases of OHCA.[10] Despite 30 years of progress in the science and treatment of OHCA, survival rates remain low. The objective of CARES is to improve community survival rates by producing local data for local action.

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Part III. Follow-up Care

The National Sudden Cardiac Arrest Survivor Network™

A Program of the Sudden Cardiac Arrest Foundation

Mary M. Newman, M.S.; Jeremy Whitehead; Bobby V. Khan, M.D., Ph.D., Sudden Cardiac Arrest Foundation

Statement of Evidence Supporting Practice

More than 295,000 people suffer out-of-hospital cardiac arrest (OHCA) each year in the United States.[1] Research suggests that on average, only 5%–7% (approximately 14,750–21,000) victims survive. However, survival rates of 38% or more have been achieved in communities with strong systems of emergency response.[2] If this rate of survival could be achieved nationwide, as many as 112,000 lives could be saved each year. If the mean national survival rate increased to even 20%, nearly 60,000 lives could be saved annually.

Promising new research suggests that the odds of survival may improve significantly in the next decade. Contributing factors include the following:

- The American Heart Association (AHA) has recommended that untrained lay rescuers use "hands-only" cardiopulmonary resuscitation (CPR).[3] Laypersons may now be more inclined to get involved when sudden cardiac emergencies occur because the technique can be learned through brief exposure to information (without lengthy, expensive training courses) and mouth-to-mouth breathing is no longer recommended for untrained rescuers.
- Efforts are under way to develop a national registry of automated external defibrillators (AEDs) to facilitate rapid identification of AED locations and rapid deployment. This will help not only first responders and other emergency medical services (EMS) responders but also the general public.[4]
- Mobile phone "apps" that provide real-time coaching in emergencies and, in some cases, AED locations, may improve the effectiveness of bystander intervention.[5]
- The 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care emphasize the importance of post-resuscitation care—including mild therapeutic hypothermia, which has been shown to preserve neurological function.[3]

As the population of OHCA survivors grows, there will be an increasing demand for support services for survivors and their loved ones—plus a growing army of champions for "the cause." The National SCA [sudden cardiac arrest] Survivor Network™ was established to address these needs.

Objective

The National SCA Survivor Network (http://www.sca-aware.org/sca-survivor-network) is the first online community of OHCA survivors in the United States. The Network gives SCA survivors and their families opportunities to do the following:

- Connect with others who have experienced similar life-changing events.
- Share experiences and help one another with the healing process.
- Participate in research and awareness initiatives designed to help save more lives.

The Network's survivor stories have become required reading for various organizations in the field (http://www.sca-aware.org/survivor-stories).

Research suggests that most survivors leave the hospital without severe neurological disabilities and continue to have a reasonable quality of life. However, a growing body of research suggests that survivors frequently suffer memory loss (particularly surrounding the time of the event), anxiety, depression, survivor guilt, fear of mortality, and severe distress about implantable cardioverter defibrillator (ICD) shocks and recalls. In addition, the psychological impact of OHCA survival among family members is profound. At the same time, survivors are interested in participating in survey research and advocacy initiatives.[5–25]

Background

The Sudden Cardiac Arrest Foundation is a national nonprofit 501(c)(3) organization based in Pittsburgh. Our mission is to serve as an information clearinghouse dedicated to raising awareness about SCA and stimulating attitudinal and behavioral changes that will help save more lives. Simply put, our mission is "to raise awareness and support programs that give 'ordinary people' the power to save a life." Our vision is that by 2015, the following changes will have taken place:

- One hundred million Americans will be aware that SCA is a major public health problem.
- The number of at-risk people who are identified and treated to prevent sudden death will double.
- Ten million people will be prepared to save a life.
- Rates of bystander intervention and survival will double.

We aim to raise awareness by advancing these key messages:

1. SCA is a public health crisis.
2. SCA is not the same as heart attack.
3. SCA affects people of all ages—even children.
4. Many high-risk individuals can be identified before SCA occurs (for example, through heart screening in schools), and can be treated prophylactically with ICD therapy.
5. AEDs should be considered safety—not medical—devices, and they should be deployed much more widely.
6. Unfounded legal liability concerns related to AED deployment should be addressed in such a way that entities recognize that AED deployment and management minimizes legal liability risks.
7. Survival from SCA depends largely upon immediate intervention by bystanders who call 911, provide CPR, and use AEDs.
8. If the public were to understand the importance of early bystander intervention and to act confidently, and if AEDs were more widely deployed, survival rates could skyrocket.
9. Laypersons should be prepared to provide CPR and use AEDs.
10. Every student should learn CPR and AED use before graduation from high school. These skills should be a requirement for individuals seeking drivers' licenses.
11. Mild therapeutic hypothermia should become routine post-arrest treatment for SCA survivors.

These are our goals:

1. Develop, collect, provide, and promote credible, reliable information resources on the prevention and treatment of SCA.
2. Motivate individuals at risk for SCA to explore prevention strategies.
3. Motivate laypersons to be prepared to intervene when SCA occurs.
4. Motivate communities to improve SCA response systems.
5. Foster communications among those interested in improving survival from SCA, including those personally affected by SCA and other advocates.
6. Further develop and promote the National SCA Survivor Network.
7. Facilitate and conduct research among SCA survivors.
8. Become recognized as the premier information clearinghouse on SCA in the United States, with the nation's most robust registry of SCA survivors and most active online community.

Methods

The SCA Foundation has developed a searchable database of approximately 300 SCA survivors from nearly every state and territory in the United States by using a Drupal content management system. Survivors register online and have the opportunity to share their experiences, connect with their peers, seek support, and get involved in research and awareness initiatives. Survivors may write about their experiences or opt to be interviewed. For this analysis, we reviewed self-reported information from 246 OHCA survivors. It is important to note that these self-reports are, by definition, based on "hearsay."

Results

An analysis of survivors in our database as of May 2010 yielded the information described below.

Respondents were asked to complete responses for as many questions as possible; this accounts for the variability in response rate.
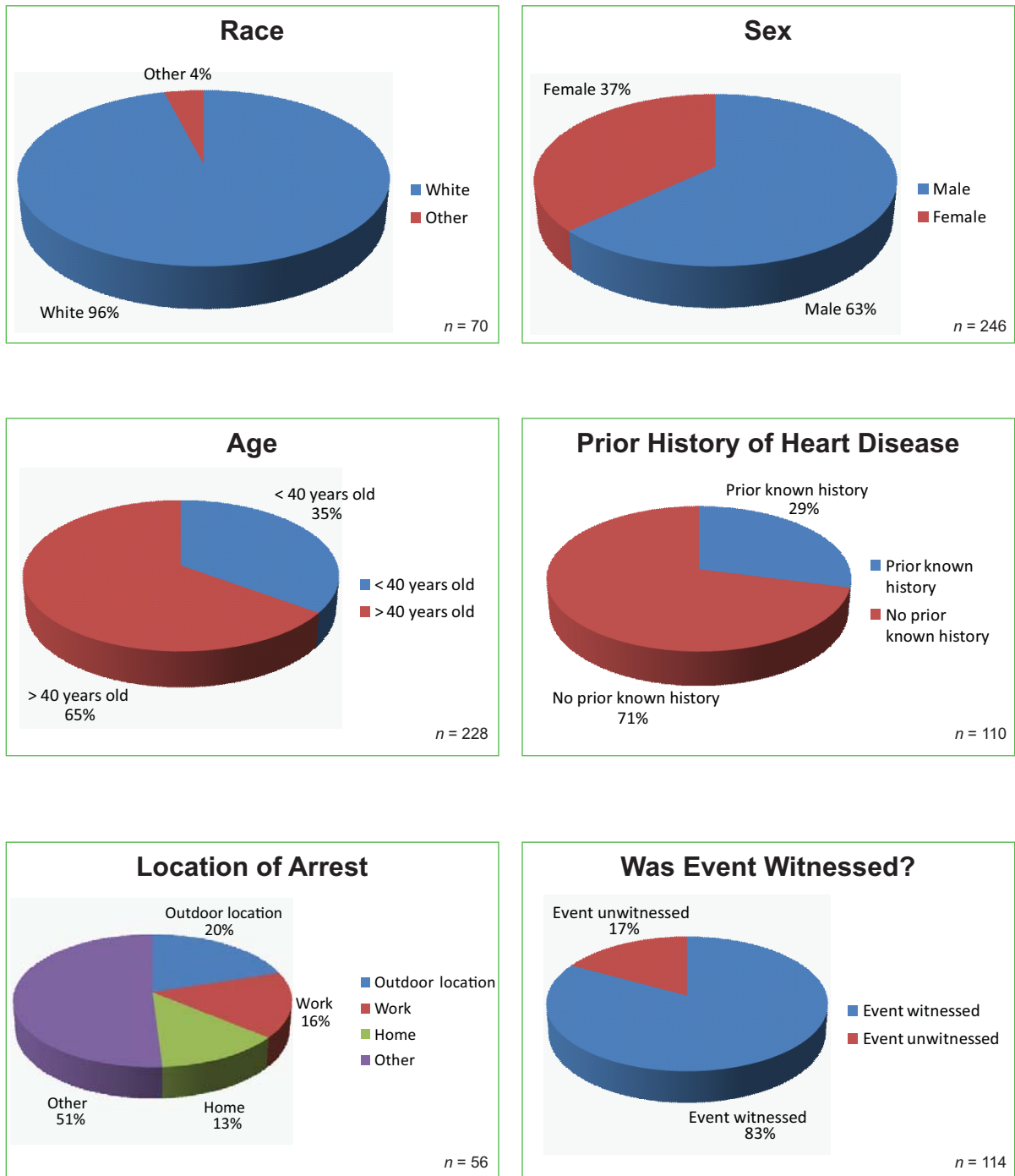
Database Review

- Most cases were witnessed (83%, n = 114).
- The most common locations of arrest were parks/golf courses/sports fields/other outdoor locations (20%), followed by work (16%) and home (13%); (n = 56).
- Most survivors are white (96%, n = 70).
- Two thirds are male (63%, n = 246).
- The mean age is 45.3; 35% are less than 40 years old (n = 228).
- Most victims had no known history of a heart condition (71%, n = 110).
- Most received CPR (73%, n = 115). Those providing CPR were friends or family members (30%), strangers (29%), or coworkers (15%); (n = 49).
- Twenty-eight percent report they were treated with a defibrillator (n = 116).
- Less than one third were treated with mild therapeutic hypothermia (28%, n = 114).
- Most have ICDs (72%, n = 114).
- Most say they have returned to their previous level of functioning (71%, n = 79) or to their previous level of functioning with some limitations (10%, n = 79) (*see* Figure 1, pages 78–79).

Conclusion

Characteristics of survivors in this database generally reflect the literature and best practice recommendations, though there are some exceptions. The average age of survivors in the National SCA Survivor Network is younger than reported elsewhere (45 vs. 65 years old). This may be explained by the fact that Network members represent a self-selected sample of web-savvy individuals. Another anomaly is the fact
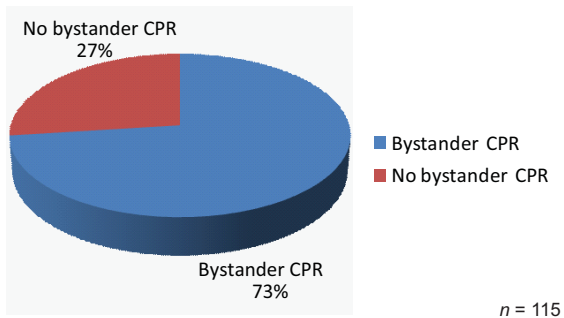
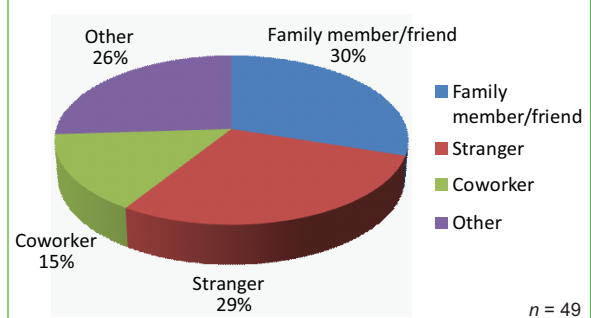## Figure 1. Characteristics of Members of National SCA Survivor Network™

Race

Other 4%

White 96%

- White
- Other

n = 70

Sex

Female 37%

Male 63%

- Male
- Female

n = 246

Age

< 40 years old
35%

> 40 years old
65%

- < 40 years old
- > 40 years old

n = 228

Prior History of Heart Disease

Prior known history
29%

No prior known history
71%

- Prior known history
- No prior known history

n = 110

Location of Arrest

Outdoor location
20%

Work
16%

Home
13%

Other
51%

- Outdoor location
- Work
- Home
- Other

n = 56

Was Event Witnessed?

Event unwitnessed
17%

Event witnessed
83%

- Event witnessed
- Event unwitnessed

n = 114

78

Figure 1. Characteristics of Members of National SCA Survivor Network™ (continued)

Did Patient Receive Bystander CPR?
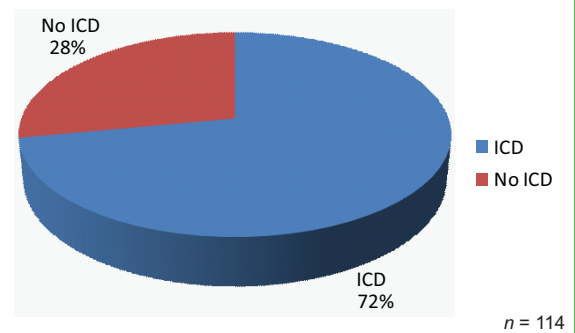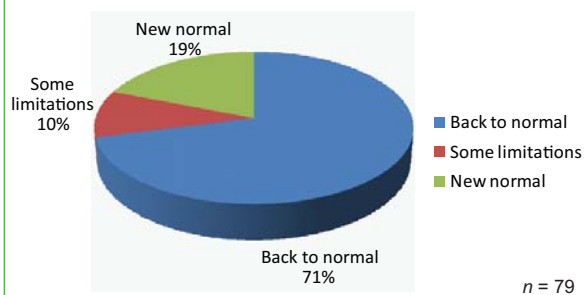
No bystander CPR
27%

- Bystander CPR
- No bystander CPR

Bystander CPR
73%

n = 115

If Yes, Who Gave Bystander CPR?

Other
26%

Family member/friend
30%

- Family member/friend
- Stranger
- Coworker
- Other

Coworker
15%

Stranger
29%

n = 49

Was Patient Treated with Mild Therapeutic Hypothermia?

Hypothermia
28%

- Hypothermia
- No hypothermia

No hypothermia
72%

n = 114
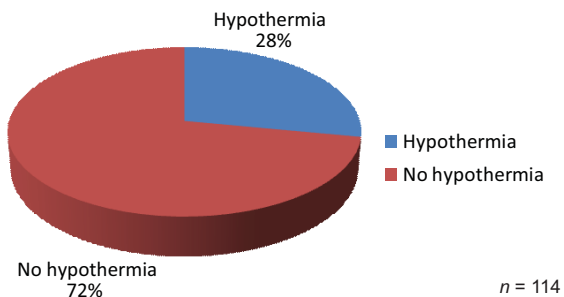
Did Patient Undergo ICD Therapy?

No ICD
28%

- ICD
- No ICD

ICD
72%

n = 114

Did Patient Return to Previous Level of Functioning?

New normal
19%

Some limitations
10%

- Back to normal
- Some limitations
- New normal

Back to normal
71%

n = 79

that most survivors reported that they were not treated with a defibrillator—a surprising finding that suggests individuals may not have been fully aware of the treatment they received. ∎

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Web Resources

American Heart Association
http://www.heart.org
The Association is a national voluntary health agency whose mission is to reduce disability and death from cardiovascular diseases and stroke.

Theheart.org
http://www.theheart.org/
Theheart.org provides information on caring for people with disorders of the heart and circulation, and on preventing such disorders.

The Joint Commission's Sudden Cardiac Arrest Initiatives
http://www.jointcommission.org/sudden_cardiac_arrest_initiatives/
The Joint Commission is developing a set of measures for inpatients and a monograph for community-based effective practices, both with a focus on prevention and treatment of sudden cardiac arrest.

The National SCA Survivor Network
http://www.sca-aware.org/sca-survivor-network
The National SCA Survivor Network is the first on-line community of OHCA survivors in the United States. The Network gives SCA survivors and their families opportunities to connect and share with others who have experienced similar life-changing events, as well as participate in research and awareness initiatives designed to help save more lives.

Project ADAM
http://www.chw.org/display/PPF/DocID/26050/Nav/1/router.asp
Project ADAM (Automated Defibrillators in Adam's Memory) is a not-for-profit program of Herma Heart Center at Children's Hospital of Wisconsin, whose mission is to serve children and adolescents through education and deployment of lifesaving programs that help prevent sudden cardiac arrest.

Project S.A.V.E.
http://www.choa.org/childrens-hospital-services/cardiac/for-professionals/project-save
Project S.A.V.E. (Sudden Cardiac Death: Awareness, Vision for Prevention, and Education) began at Children's Healthcare of Atlanta in August 2004 and became the first state affiliate of Project ADAM in 2005, sharing the above goals. Project S.A.V.E., like Project ADAM, was initiated in response to several student-athlete sudden deaths that occurred in the metropolitan Atlanta area in 2003.

Sudden Cardiac Arrest Foundation
http://www.sca-aware.org/
The mission of the Sudden Cardiac Arrest Foundation is to prevent death and disability from sudden cardiac arrest (SCA). The vision of the Foundation is to increase awareness about sudden cardiac arrest and influence attitudinal and behavioral changes that will reduce mortality and morbidity from SCA.