I'm not robot!

Adult Basic Life Support Algorithm for Healthcare Providers



1

Assess appropriateness for clinical condition. Heart rate typically ≥150/min if tachyarrhythmia.

2



(NOT synchronized)

Adenosine IV Dose:

First dose: 6 mg rapid IV push; follow with NS flush. Second dose: 12 mg if required.

Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia

Procainamide IV Dose:

20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose 17 mg/kg given. Maintenance infusion: 1-4 mg/min. Avoid if prolonged QT or CHF.

Amiodarone IV Dose:

First dose: 150 mg over 10 minutes. Repeat as needed if VT recurs. Follow by maintenance infusion of 1 mg/min for first 6 hours.

Sotalol IV Dose:

100 mg (1.5 mg/kg) over 5 minutes. Avoid if prolonged QT.



Pediatric Bradycardia With a Pulse Algorithm



What is it?	Preexcitation syndrome where an accessory pathway (Bundle of Kent) directly connects the atria and ventricles allowing electrical activity to bypass the AV node causing symptomatic arrhythmias. The WPW pattern can be seen in the absences of symptomatic arrhythmias.	
Diagnosis	ECG findings These may not be present in all patients; such as those with left-lateral bypass tracts	Short PR interval (<0.12 ms) Delta wave (slurred upstroke of QRS due to fusion between early myocardial activiation from accessory pathway and normal activation through the AV node) Widening of the QRS
	Delta wave	Delta wave
Complications	Arrythmias (AVRT 80% of WPW syndrome, atrial fibrillation 10-30% of WPW syndrome) which could lead to syncope or sudden cardiac death (risk ~0.13%/year)	
atment of acute complications	Antidromic AVRT (atrioventricular re-entrant tachycardia)	Vagal manuevers Procainamide Do NOT use nodal blockers such as adenosine beta-blockers, calcium channel blockers
	Atrial fibrillation (10-30% of WPW syndrome) Irregularly irregular rhythm with various QRS morphologies beat to beat. Can devolve into ventricular fibrillation	Cardioversion or Procainamide Do NOT use nodal blockers such as adenosine beta-blockers, calcium channel blockers
	@FOAMpodcast FOAMca	st.org

Aha guidelines for svt. Ventricular tachycardia treatment aha guidelines. Aha guidelines for ventricular fibrillation.

2017 AHA/ACC/HRS guideline 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 on Clinical Practice Guidelines and the Heart Rhythm Society. Al-Khatib SM, Stevenson WG, Ackerman MJ, Bryant WJ, Callans DJ, Curtis AB, Deal BJ, Dickfeld T, Field ME, Fonarow GC, Gillis AM, Granger CB, Hammill SC, Hlatky MA, Joglar JA, Kay GN, Matlock DD, Myerburg RJ, Page RL. Al-Khatib SM, et al. Heart Rhythm. 2018 Oct;15(10):e73-e189. doi: 10.1016/j.hrthm.2017.10.036. Epub 2017 Oct 30. Heart Rhythm. 2018. PMID: 29097319 Review. No abstract available. Guideline recommendations in slide format Top summary points of the guideline Read the latest news story The 2017 AHA/ACC/HRS Guideline for Management of Sudden Cardiac Death (SCD), seeks to provide contemporary recommendations for the management of adults who have VA or who are at risk for SCD, including diseases and syndromes associated with a risk of SCD from VA. This guideline includes indications for the treatment of VA and prevention of SCD, but it does not delve into details on individual device selection and programming, including considerations relevant to cardiac resynchronization therapy (CRT), bradycardia pacing, and hemodynamic monitoring, which have been covered elsewhere. This document is aimed at the adult population (>18 years of age), offering no specific recommendations for pediatric data were examined. Central Illustration: Indications for Implantable Cardioverter-Defibrillator (ICD) in Patients with Ischemic Heart Disease (IHD) to Prevent Sudden Cardiac Death (SCD) The American College of Cardiology (ACC)—along with collaborative societies—create Clinical Practice Guidelines, which become ACC policy at the time of publication. All Clinical Practice Guidelines undergo rigorous peer review that is independent of the Editors/editorial processes of the Journal of the American College of Cardiology (JACC). JACC Editors receive no compensation from the publication of the guidelines and Clinical Documents. Preamble e2741. Introduction e2751.1. Methodology and Evidence Review e2751.2. Organization of the Writing Committee e2761.3. Document Review and Approval e2792.1.1. Premature Ventricular Complexes and Nonsustained VT e2792.1.2. VT and VF During ACS e2812.1.3. Sustained VT and VF Not Associated With ACS e2812.2.1. Incidence of SCD e2812.2.2. Population Subgroups and Risk Prediction e2823.4. Reentry e2844. General Evaluation of Patients With Documented or Suspected VA e2844.1. History and Physical Examination e2864.2.3. Implanted Cardiac Monitors e2874.2.4. Noninvasive Evaluation e2864.2.1. 12-lead ECG and Exercise Testing e2864.2.3. Implanted Cardiac Monitors e2874.2.4. Noninvasive Evaluation e2864.2.3. Implanted Cardiac Monitors e2864.2.3. Implanted Cardiac Monitors e2874.2.4. Noninvasive Evaluation e2864.2.3. Implanted Cardiac Monitors e2864.2.3. Implanted Cardiac Monitors e2874.2.4. Noninvasive Evaluation e2864.2.3. Implanted Cardiac Monitors e2864.2.3. Implanted Cardiac Monitors e2864.2.3. Implanted Cardiac Monitors e2864.2.4. Noninvasive Evaluation e2864.2.4. Implanted Cardiac Monitors e2864.2.4. Implan Genetic Considerations in Arrhythmia Syndromes e2884.3. Invasive Testing e2894.3.1. Invasive Cardiac Imaging: Cardiac Catheterization or CT Angiography e2895.1. Medications With Prominent Sodium Channel Blockade e2905.1.2. Beta Blockers e2935.1.3. Amiodarone and Sotalol e2935.1.4. Calcium Channel Blockers e2945.1.5. Nonantiarrhythmic Medications e2955.4. Catheter Ablation e2955.4.1. General Considerations e2955.4.2. VA in Patients With No Apparent Structural Heart Disease e2965.4.3. Scar-Related VT e2965.5. Surgery and Revascularization Procedures in Patients With Ischemic Heart Disease e2965.5.1. Surgery for Arrhythmia Management of VA and SCD Risk Related to Specific Disease e3017.1.1. Ischemic Heart Disease e3017.1.2. Primary Prevention of SCD in Patients With Ischemic Heart Disease e3067.2. Nonischemic Cardiomyopathy e3087.2.1. Secondary Prevention of SCD in Patients With NICM e3087.2.2. Primary Prevention of SCD in Patients With NICM e3097.2.3. Treatment of Recurrent VA in Patients With NICM e3097.2.3. Treatment VA in Patients With N Sarcoidosis e3187.6.1. Other Infiltrative Cardiomyopathies e3207.7. Heart Failure e3217.7.1. HF With Reduced Ejection Fraction e3217.7.2. HF With Preserved Ejection Fraction e3217.7.3. Left Ventricular Assist Device e3227.7.4. ICD Use After Heart Transplantation e3227.8. Neuromuscular Disorders e3227.9. Cardiac Channelopathies e3247.9.1. Specific Cardiac Channelopathy Syndromes e3258. VA in the Structurally Normal Heart e3348.1. Outflow Tract and Atrioventricular Annular VA e3358.2. Papillary Muscle VA e3358.3. Interfascicular Reentrant VT (Belhassen Tachycardia) e3358.4. Idiopathic Polymorphic VT/VF e3369. PVC-Induced Cardiomyopathy e33710. VA and SCD Related to Specific Populations e33810.1. Athletes e33810.2. Pregnancy e33810.3. Older Patients With Comorbidities e33910.4. Chronic Kidney Disease e34010.5. Valvular Heart Transvenous ICDs e34711.1. Subcutaneous Implantable Cardioverter-Defibrillator e34912. Special Considerations for Catheter Ablation e34913. Postmortem Evaluation of SCD e35014. Terminal Care e35115. Shared Decision-Making e35116. Cost and Value Considerations e35217. Quality of Life e35318. Evidence Gaps and Future Research Needs e354Appendix 1: Author Relationships With Industry and Other Entities (Comprehensive) e389Since 1980, the American College of Cardiology (ACC) and American Heart Association (AHA) have translated scientific evidence into clinical practice quidelines, which are based on systematic methods to evaluate and classify evidence, provide a cornerstone for quality cardiovascular health. These quidelines, which are based on systematic methods to evaluate and classify evidence. of guidelines without commercial support, and members of each organization volunteer their time to the writing and review efforts. Guidelines provide recommendations applicable to patients with or at risk of developing cardiovascular disease. The focus is on medical practice in the United States, but guidelines developed in collaboration with other organizations may have a global impact. Although guidelines may be used to inform regulatory or payer decisions, their intent is to improve patients' quality of care and align with patients' interests. Guidelines are intended to define practices meeting the needs of patients in most, but not all, circumstances and should not replace clinical judgment. Clinical Implementation Guideline-recommended management is effective only when followed by shared decision-making between healthcare providers and patients, with patient engagement in selecting interventions based on individual values, preferences, and associated conditions and comorbidities. Methodology and Modernization The ACC/AHA Task Force on Clinical Practice Guidelines (Task Force) continuously reviews, updates, and modifies guideline methodology on the basis of published standards from organizations including the Institute of MedicineP-1,P-2 and on the basis of evolving technologies and other factors to facilitate optimal dissemination of information at the point of care to healthcare professionals. Toward this goal, this guideline heralds the introduction of an evolved format of presenting guideline recommendations, a brief synopsis, recommendation-specific supportive text and, when appropriate, flow diagrams or additional tables. References are provided within the modular chunk itself to facilitate quick review. This format also will facilitate seamless updating of guidelines with focused updates as new evidence is published, and content tagging for rapid electronic retrieval of related recommendations on a topic of interest. This evolved format was instituted when this guideline was near completion; therefore, the current document represents a transitional formatting that best suits the text as written. Future guidelines will fully implement this format, including provisions for limiting the amount of text in a guideline. Recognizing the importance of cost-value considerations in certain guidelines, when appropriate and feasible, an analysis of the value of a medication, device, or intervention may be performed in accordance with the ACC/AHA methodology.P-3To ensure that guideline revisions commissioned in approximately 6-year cycles. Publication of new, potentially practicechanging study results that are relevant to an existing or new medication, device, or management strategy will prompt evaluation by the Task Force, in consultation with the relevant guideline development, we encourage readers to consult the ACC/AHA guideline methodology manualP-4 and other methodology articles.P-5-P-8Selection of Writing committee Members represent different geographic regions, sexes, ethnicities, races, intellectual perspectives/biases, and scopes of clinical practice. The Task Force may also invite organizations and professional societies with related interests and expertise to participate as partners, collaborators, or endorsers. Relationships With Industry and Other Entities The ACC and AHA have rigorous policies and methods to ensure that guidelines are developed without bias or improper influence. The complete relationships with industry and other entities (RWI) policy can be found online. Appendix 1 of the current document lists writing committee members' relevant RWI. For the purposes of full transparency, writing committee members' comprehensive disclosure information is available online, as is the comprehensive disclosure information for the Task Force. Evidence Review and Evidence Review Committee uses evidence-based methodologies that are based on all available data. P-4-P-7 Literature searches focus on randomized controlled trials (RCTs) but also include registries, nonrandomized comparative and descriptive studies, case series, cohort studies, systematic reviews, and expert opinion. Only key references are cited. An independent evidence review committee (ERC) is commissioned when there are ≥ 1 questions deemed of utmost clinical importance that merit formal systematic review. This systematic review will strive to determine which patients are most likely to benefit from a test, medication, device, or treatment strategy and to what degree. Criteria for commissioning an ERC and formal systematic review; b) the feasibility of defining the benefit and risk in a time. frame consistent with the writing of a guideline; c) the relevance to a substantial number of patients; and d) the likelihood that the findings can be translated into actionable recommendations. ERC members may include methodologists, healthcare providers, and biostatisticians. When a formal systematic review has been commissioned, the recommendations developed by the writing committee on the basis of the systematic review are marked with "SR." Guideline-Directed Management and TherapyThe term guideline-Directed Management and therapy (GDMT) encompasses clinical evaluation, diagnostic testing, and pharmacological and procedural treatments. For these and all recommended medications, devices, and treatments approved for clinical use in the United States. Class of Recommendation and Level of Evidence The Class of Recommendation (COR) indicates the strength of the recommendation, encompassing the estimated magnitude and certainty of benefit in proportion to risk. The Level of Evidence (LOE) rates the strength of the type, quantity, and consistency of data from clinical trials and other sources (Table 1).P-4,P-6,P-8Table 1. Applying Class of Recommendation and Level of Evidence to Clinical Strategies, Interventions, Treatments, or Diagnostic Testing in Patient Care* (Updated August 2015)Glenn N. Levine, MD, FACC, FAHAChair, ACC/AHA Task Force on Clinical Practice Guidelines1. Introduction1.1. Methodology and Evidence ReviewThe recommendations listed in this clinical practice guideline are, whenever possible, evidence review, which included literature derived from research involving human subjects, published in English, and indexed in MEDLINE (through PubMed). EMBASE, the Cochrane Library, the Agency for Healthcare Research and Quality, and other selected databases relevant to this guideline, was conducted from April 2016 to September 2016. Key search words included, but were not limited, to the following: sudden cardiac death, ventricular tachycardia, ventricular fibrillation, premature ventricular contractions, implantable cardioverter-defibrillator, subcutaneous implantable cardioverter-defibrillator, and catheter ablation. Additional relevant studies published through March 2017, during the guideline writing process, were also considered by the writing committee, and added to the evidence tables when appropriate. The final evidence tables are included in the Online Data Supplement and summarize the evidence used by the writing committee reviewed documents related to ventricular arrhythmias (VA) and sudden cardiac death (SCD) previously published by the ACC, AHA, and the Heart Rhythm Society (HRS). References selected and published in this document are representative and not all-inclusive. As noted in the Preamble, an independent ERC was commissioned to perform a formal systematic review of 2 important clinical guestions for which clear literature and prior guideline consensus were felt to be lacking or limited (Table 2). The results of the ERC review were considered by the writing committee for incorporation into this guideline. Concurrent with this process, writing committee members were formally presented and discussed, then guideline recommendations were developed. The "Systematic Review for the 2017 AHA/ACC/HRS Guideline for Management of Patients With Ventricular Arrhythmias and the Prevention Questions on SCD Prevention Question NumberQuestionSection Number1For asymptomatic patients with Brugada syndrome, what is the association between an abnormal programmed ventricular stimulation for primary prevention in older patients with significant comorbidities? 10.3The ACC and AHA have acknowledged the importance of value in health care and have called for eventual development of a Level of Value for clinical practice recommendations. S1.4-2 Available cost-effectiveness data were determined to be sufficient to support 2 specific recommendations. S1.4-2 Available cost-effectiveness data were determined to be sufficient to support 2 specific recommendations. result, a Level of Value was assigned to those 2 recommendations on the basis of the "ACC/AHA Statement on Cost/Value Methodology in Clinical Practice Guidelines and Performance Measures," as shown in Table 3.S1.4-2 Available guality of life (OoL) data were deemed to be insufficient to support specific recommendations in this guideline. Table 3. Proposed Integration of Level of Value Into Clinical Practice Guideline Recommendations*Level of ValueHigh value: Better outcomes at lower cost or ICER 100 bpm (cycle length: 30 PVCs per hour, are associated with increased cardiovascular risk and increased mortality.S2.2.2-9 In a study from Taiwan of patients without sustained VT or structural heart disease who had 24-hour Holter monitoring for clinical evaluation, multifocal PVCs were associated with increased risk of death and nonfatal cardiovascular adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death and nonfatal cardiovascular adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death and nonfatal cardiovascular adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death and nonfatal cardiovascular adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death and nonfatal cardiovascular adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death and nonfatal cardiovascular adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death and nonfatal cardiovascular adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death and nonfatal cardiovascular adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with increased risk of death adverse outcomes.S2.2.2-10 In the same population, multifocal PVCs were associated with adverse outcomes.S2.2.2-10 In the same population, mult was independently associated with increased risk of death and other cardiovascular adverse outcomes, including stroke. S2.2.2-11 An association of PVCs, particularly if multifocal and frequent, is generally considered a risk factor for adverse cardiovascular outcomes, and such patients are generally evaluated to ensure they do not have underlying conditions (eg, ischemic heart disease, left ventricular [LV] dysfunction) that warrant further treatment to reduce risk. PVC and NSVT in patients with cardiovascular disease are common and have been associated with adverse outcomes.S2.2.2-12,S2.2.2-13 In CAST (Cardiac Arrhythmia Suppression of VA.S2.2.2-14,S2 Treatment of PVCs with antiarrhythmic medications has not been shown to reduce mortality and, in the post-MI population, treatment with class I sodium channel-blocking medications and d-sotalol increase the risk of death.S2.2.2-16,S2.2.2-17 Beta blockers, and some antiarrhythmic medications may relieve symptoms of palpitations.S2.2.2-18 PVCs that occur during an exercise test are associated with a higher risk of death.S2.2.2-19 In 1 study, PVCs that occur during recovery are a stronger predictor of death than PVCs occurring only during exercise.S2.2.2-20 However, PVCs are common in trained athletes who have palpitations, in whom there does not appear to be increased risk of death based on studies of small numbers of athletes, at least in those without other cardiovascular abnormalities.S2.2.2-21,S2.2.2-22 Complex PVCs may not represent a benign finding in endurance athletes. An electrophysiological study may be needed to assess patients' arrhythmogenic risk.S2.2.2-22 Very frequent PVCs, >10 000 to 20 000 a day, can be associated with depressed LV function in some patients that is reversible with control of the PVCs, and has been referred to as PVCinduced cardiomyopathy. S2.2.2-23, S2.2.2-24 (See also Section 8.5. PVC-Induced Cardiomyopathy.) Very rarely, idiopathic PVCs from the outflow tract may trigger malignant VA in patients with out-of-hospital cardiac arrest with the first rhythm identified as VF and who survive to hospital admission have evidence of acute MI (AMI).S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary are significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary are significant coronary angiography.S2.2.2-27 Of all out-of-hospital cardiac arrests, >50% will have Significant coronary are significant 5% will have VF or sustained VT after hospital arrival, most within 48 hours of admission. A study of patients, with 60% of those events within 48 hours of admission. S2.2.2-28 Accelerated idioventricular rhythm is a common arrhythmia in patients with acute MI, including patients with ST-segment elevation MI undergoing primary percutaneous coronary intervention (PCI). Accelerated idioventricular rhythm is more closely related to the extent of infarction than to reperfusion itself. S2.2.2-29Sustained VA that occurs in the setting of an ACS is more often polymorphic VT or VF than monomorphic VT. Risk factors for VT/VF include prior history of hypertension, prior MI, ST-segment changes at presentation, and chronic obstructive pulmonary disease. S2.2.2-30 A nationwide Danish study found that 11.6% of patients with ST-segment elevation MI who underwent PCI had VF prior to the PCI, and that VF was associated with alcohol consumption, preinfarction angina, anterior infarct location, and complete coronary occlusion at the time of coronary angiography.S2.2.2-31 In a select group of patients undergoing primary PCI in a clinical trial, 5.7% developed sustained VT or VF, with two thirds of these events occurring prior to the end of the catheterization, and 90% within 48 hours from the procedure. VT or VF after primary PCI was associated with lower blood pressure, higher risk of death within a substantially higher risk of death within 90 days. Late VT or VF (after 48 hours of hospital presentation). S2.2.2-332.1.3. Sustained VT and VF. Sustained VT that is not associated with an ACS is often monomorphic as it is usually due to scar-related reentry, but it may degenerate to VF.S2.2.2-34 The risk and predictors of VT in patients with structural heart disease depend on the type, severity, and duration of structural heart disease. symptomatic HF. Monomorphic VT occurring in the absence of structural heart disease is commonly referred to as idiopathic VT and is often due to an automatic focus in a characteristic location, giving rise to typical electrocardiographic appearances. due to a cardiac channelopathy, S2.2.2-35, S2.2.2-36 medication-induced long QT syndrome, S2.2.2-36 or they may be idiopathic. S2.2.2-37, S2.2.2-382.2. Sudden Cardiac Death 2.2.1. Incidence of SCDSCA and its most common consequence, SCD, constitute major public health problems, accounting for approximately 50% of all cardiovascular deaths, S2.2.2-1, S2.2.2-39 with at least 25% being first symptomatic cardiac events. S2.2.2-41 In addition, analyses of the magnitude of SCD are limited, in part because of the broad range of estimates of the risk based on different epidemiological methods. S2.2.2-42 During the past 20 to 30 years, SCD accounted for approximately 230 000 to 350 000 deaths per year in the United States, with a range of 450 000, depending on epidemiological methods, data sources, and inclusion criteria.S2.2.2-43 The lowest of these extremes came from national extrapolation of data from specific local programs, while the highest rates included noncardiac causes of sudden death such as pulmonary embolism or intracranial bleeding. The mid-range numbers were largely based on death certificate studies that required a code inclusive of ischemic heart disease. The 2017 update of cardiovascular statistics from the AHA estimated the total annual burden of out-of-hospital cardiac arrest at 356 500. S2. 2. 2. 44 An additional 209 000 inhospital cardiac arrests occur annually.S2.2.2-45 Among the out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, approximately 357 000 events trigger emergency rescue response, with 97% occurring in adults >18 years of age. The survival statistics for out-of-hospital cardiac arrest group, a the subgroup of 70% of out-of-hospital cardiac arrests that occur in the home, survival is 6%. The best reported outcomes are from location of cardiac arrest, bystander witnesses willing to provide CPR, first responders arriving quickly, shockable rhythm at initial contact, availability of automated external defibrillators (AEDs), and possibly a benefit from telecommunication-directed CPR.S2.2.2-46, S2.2.2-48 In all settings, survival statistics appear to be better when rhythms recorded by responders are shockable (VF, pulseless VT), compared with pulseless electrical activity or asystole.S2.2.2.49 Although the apparent increase in the incidence of shockable rhythm has also been attributed, in part, to improvements in diagnosis and treatment of structural heart disease.S2.2.2-402.2.2. Population Subgroups and Risk Prediction Risk prediction and individual risk prediction.S2.2.2-41,S2.2.2-50 Conventional epidemiological markers provide insight into probabilities for the development of ischemic heart disease within a general class of subjects, but adequately tested and validated profiles for SCA risk in individuals derives from a population model characterized by large numbers of events diluted into a very large denominator (Figure 1). The overall population can be subgrouped into categories based on integration of small, high-risk subgroups within the large denominator general population. Increasing age is a strong predictor of risk for SCA, but it is not linear. Risk in the general population, over time, beginning at 35 years of age has been estimated at 1 per 1000 population per year, increasing from a risk 75 years of age.S2.2.2-51 The data also suggested that SCD is uniformly more common in men than in women at all age groups. In contrast, the population of children, adolescents, and young adults has an overall annual risk of 1 per 100 000, and there is somewhat a higher risk of SCD at the younger end of that age range. S2.2.2-41 An age-associated transition range, from the mid-20s to 35 to 40 years of age, is characterized by a steep increase in risk from that of the adolescent group to the middle-aged group, corresponding to the emergence of ischemic heart disease. Although ischemic heart disease remains the most common underlying substrate associated with SCD, the incidence of ischemic heart disease-related SCD appears to be decreasing. S2.2.2-53 In addition, a trend over time has suggested that out-of-hospital cardiac arrest patients who are admitted alive to a hospital are becoming more likely to have high-risk clinical profiles, as opposed to manifest disease.S2.2.54 The younger population—children, adolescents, and young adults—is affected by a series of disorders that manifest earlier in life, including the genetic structural disorders and cardiac channelopathies, myocarditis, congenital heart disease, and other rare disorders.S2.2.43 During the transition range, from the mid-30s, causes of SCA and SCD include a lower proportion of inherited diseases and increasing proportion of ischemic heart disease (>40% of

cases).S2.2.2-43Despite the small progress that has been made in risk prediction of SCA and SCD, the greatest challenge is to identify the relatively small, high-risk subgroups concealed within the large general population who have no identified disease but are at risk of SCA as their first cardiac event (Figure 1).S2.2.2-50Figure 1A. SCD incidence and total events.S2.2.2-1 EF indicates ejection fraction; and SCD, sudden cardiac death.Figure 1B. SCD and clinical subsets.S2.2.2-1 SCD indicates sudden cardiac death.3. Mechanisms of VA3.1. Cellular Mechanisms and SubstratesMechanisms of VA3.1. Cellular Mechanisms and SubstratesMechanisms and SubstratesMechanisms of VA3.1. Cellular Mechanisms and SubstratesMechanisms of VA3.1. Cellular Mechanisms and SubstratesMechanisms of VA3.1. Cellular Mechanisms and SubstratesMechanisms and SubstratesMechanisms and SubstratesMechanisms of VA3.1. Cellular Mechanisms and SubstratesMechanisms late afterdepolarizations, and reentry.S3.4-1-S3.4-3 Reentry requires a trigger to initiate the arrhythmia and a substrate may be structural remodeling secondary to an underlying disease process, and often includes a scar secondary to a prior MI or surgical repair, or patchy fibrosis in the setting of cardiomyopathy or hypertrophy. Changes in ion channel or transporter function and/or expression and cell to cell coupling secondary to the underlying pathology may alter the initiation or propagation of the cardiac action potential. The electrophysiological substrate is dynamically influenced by a variety of factors including cardiac metabolism, electrolytes, signaling pathways and autonomic effects. Enhanced automaticity or abnormal automaticity results from phase 4 spontaneous depolarization of the transmembrane action potential arising from a normal resting potential, reaching threshold and initiating an action potential.S3.4-3 An initiating current (If) is responsible for spontaneous phase 4 depolarization, the slope and initiating current (If) is responsible for spontaneous phase 4 depolarization, the slope and initiating current (If) is responsible for spontaneous phase 4 depolarization in the sinus node. of phase 4 depolarization, and the threshold potential. In contrast, abnormal automaticity arises from a partially depolarized membrane.S3.4-1,S3.4-3 In the acute phase of an MI or during transient ischemia, increased extracellular potassium causes partial depolarization of the resting membrane potential creating injury currents between the infarcted/ischemic tissue and healthy myocardium. These injury currents may initiate spontaneous activity. In ischemia, abnormal automaticity in Purkinje fibers in the ischemic zone.3.3. Triggered ActivityEarly afterdepolarizations occur during late phase 3 of the action potential, S3.4-3-S3.4-5 usually in the setting of action poten or a decrease in repolarizing potassium currents. Under these conditions, early afterdepolarizations may be initiated when reactivation of the inward L-type calcium channel occurs before the membrane has returned to a more negative potential than that required for calcium channel reactivation. reticulum may also result in activation of a depolarizing sodium/calcium exchange current. Early afterdepolarizations or other acquired factors or due to mutations of ion channels causing the long QT syndrome. In these cases, it is possible that the early afterdepolarization/triggered activity sequence is the trigger that culminates in polymorphic VT/VF.Delayed afterdepolarizations of intracellular calcium overload. Factors contributing to elevated intracellular calcium load include tachycardia, catecholamines hypokalemia, digoxin toxicity, cardiac hypertrophy, and HF.S3.4-6,S3.4-7 Elevated sarcoplasmic calcium content or increased sensitivity of the ryanodine receptor can initiate spontaneous calcium release, which activates a transient inward current driven predominantly by the sodium-calcium exchange current. If the membrane depolarization is sufficiently large, the inward sodium current is activated resulting in a triggered action potential. Delayed afterdepolarizations are the underlying mechanism for VT in the setting of digoxin toxicity, catecholaminergic polymorphic VT, and idiopathic outflow tract VA. Delayed afterdepolarizations are the underlying mechanism for VT in the setting of digoxin toxicity, catecholaminergic polymorphic VT, and idiopathic outflow tract VA. the setting of HF. Purkinje cells are more susceptible to spontaneous sarcoplasmic reticulum calcium release than ventricular myocytes suggesting that delayed afterdepolarizations may be an important mechanism for most sustained VA in the presence of structural heart disease. S3.4-1-S3.4-3, S3.4-10-S3.4-12 Reentry may occur around a fixed anatomical obstacle, such as scar after an MI or surgically repaired congenital heart disease. In this setting, an excitable gap separates the excitation wavefront from its tail of refractoriness. The existence of structural reentrant substrates provide the rationale for VT ablation in scar-related VTs.S3.4-11,S3.4-12Functional reentry around areas of functional block without anatomical obstacles can also occur. Two main models of functional reentry is driven by a rotor with a curved wavefront and wavetail pivoting around an excitable but unexcited core. There remains much debate about the precise mechanisms may be operational in different phases of VF.S3.4-10Phase 2 reentry may occur due to heterogeneity of ventricular repolarization. Electrotonic currents may flow from endocardial sites with longer action potential durations to the epicardium with shorter action potential durations which can result in reexcitation when these sites have recovered from refractoriness. This is believed to be one potential durations to the epicardium with shorter action potential durations which can result in reexcitation when these sites have recovered from refractoriness. during ischemia.4. General Evaluation of Patients With Documented or Suspected VA4.1. History and Physical ExaminationSynopsisVA can produce a wide spectrum of symptoms, and the severity of symptoms does not necessarily reflect the extent of structural heart disease or the potential risk of SCD. Symptoms of VA include palpitations, either skipped or extra beats or sustained palpitations, shortness of breath, chest pain, dizziness, near syncope, and syncope, and syncope, and syncope, and syncope, and syncope includes VA but also inclu other etiologies. Nonetheless, more dramatic symptoms, particularly in patients with known or discovered structural or electrical heart disease should prompt focused investigation for possible association with VA (Table 6. Important Considerations in the Evaluation of Patients With Known or Suspected VAComponentAssessment and Findings Relevant for VA and/or SCD RiskHistory1. Symptoms related to underlying heart disease: Dyspnea at rest or on exertion, orthopnea, paroxysmal nocturnal dyspnea, chest pain, edema3. Precipitating factors: Exercise, emotional stress4. Known heart disease: Coronary, valvular (eg, mitral valve prolapse), congenital heart disease; other5. Risk factors for heart disease; other5. Risk factors factors for heart disease; other5. Risk factors for heart disease; othe with potential to provoke or aggravate VA Stimulants including cocaine and amphetamines Supplements including anabolic steroids Medication-medication interaction that could cause QT prolongation and torsades de pointes7. Past medical history Thyroid disease Acute kidney injury, chronic kidney disease, or electrolyte abnormalities Stroke or embolic events Lung disease Epilepsy (arrhythmic syncope can be misdiagnosed as epilepsy) Alcohol or illicit drug use Use of over-the-counter medications that could cause QT prolongation and torsades de pointes Unexplained drowning in a first-degree relative2. SIDS or repetitive spontaneous pregnancy losses given their potential association with cardiac channelopathies: Long QT, Brugada, Short QT, CPVT Arrhythmias Conduction disorders, pacemakers/ICDs4. Neuromuscular disease associated with cardiomyopathies Muscular dystrophy5. EpilepsyExamination1. Heart rate and regularity, blood pressure3. Murmurs4. Pulses and bruits5. Edema6. Sternotomy scarsThe elucidation of precipitating factors, such as exertional or emotional stress, concurrent medications or illness, and alleviating factors is a concurrent medication of precipitating factors is concurrent medications or illness. important. The presence of a family history of SCD, ischemic heart disease, valvular heart disease, va and torsades de pointes (www.crediblemeds.org)S4.1-8; some medications can also induce Brugada type I electrocardiographic pattern and VF (www.brugadadrugs.org)S4.1-9,S4.1-10Patients with bigeminy and trigeminy can pressure. Effective bradycardia from PVCs can result in inaccurate estimation of the heart rate. Although premature beats on auscultation of the heart can be detected, the physical examination is focused largely on finding evidence of structural heart disease. with ischemic heart disease. Jugular venous distention, rales, gallops, and peripheral edema provide evidence of HF. Auscultation may reveal cardiac murmurs consistent with valvular heart disease, such as aortic stenosis or mitral regurgitation, and may be associated with HF and VA. A midsystolic click may indicate mitral valve prolapse that can be associated with VA.S4.1-11-S4.1-13 Many VA are asymptomatic and detected only on an ECG or telemetry. Such cases highlight the need to search for evidence of underlying heart disease. Recommendation-Specific Supportive Text1. Rapid, sustained VT may result in syncope secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction in cardiac output, followed by spontaneous secondary to marked reduction. recovery if VT terminates, or SCA if VT persists and is not treated promptly. Syncope or SCA may be the first manifestation of structural or electrical heart disease, S4.1-15 Syncope, or its forewarnings of dizziness, lightheadedness, or near-syncope, may constitute a risk factor for SCA and SCD.S4.1-2 The initial evaluation at any age focuses on detection or exclusion of heart disease. Syncope during exercise should prompt thorough evaluation with echocardiography, ambulatory monitoring, and exercise testing may be warranted depending on the clinical information elicited.S4.1-3,S4.1-4 Cardiac causes of syncope include sustained VT, high-grade atrioventricular block or severe sinus bradycardia or prolonged sinus pauses, supraventricular tachycardia (SVT), malfunction of pacemakers, VA from cardiac channelopathies or structural heart disease syndromes, such as hypertrophic cardiomyopathy (HCM) or congenital heart disease.S4.1-3,S4.1-4,S4.1-16 Cardiac channelopathies and HCM are particularly important to consider in adolescents and young adults. Arrhythmic causes of syncope are often associated with very short periods of premonitory symptoms, or palpitations, and known preexisting heart disease, especially a history of a low LVEF or HF.S4.1-1 Among nonarrhythmic cardiac causes, considerations, and illicit drug use.S4.1-34.2. Noninvasive Evaluation4.2.1. 12-lead ECG and Exercise TestingRecommendation-Specific Supportive Text1.A 12-lead ECG during tachycardia is the first diagnostic test that should be done in any patient found to be in a stable wide QRS complex tachycardia and underlying structural heart disease. S4.2.1-3 Criteria that support a diagnosis of VT include AV dissociation, a QRS complex >0.14 s, monophasic R wave in aVR, specific QRS morphologies (eg, positively or negatively concordant QRS complexes in the precordial leads), the absence of an RS complex in all precordial leads and an RS interval >100 ms in at least 1 precordial leads), the absence of an RS complexes in the precordial leads), the absence of an RS complex in all precordial leads and an RS interval >100 ms in at least 1 precordial leads). of certain antiarrhythmic medications.S4.2.1-1 For patients with preexisting bundle branch block, comparison of the QRS morphology during sinus rhythmic symptoms, exercise in a monitored setting may reproduce the symptoms and/or the related arrhythmia, allowing for diagnosis. Exercise testing is particularly important when catecholaminergic polymorphic ventricular tachycardia is a possibility. However, exertion-related symptoms and findings may not be reliably reproducible with exercise testing, and long-term electrocardiographic monitoring with external or implantable recorders may be necessary.3.A 12-lead ECG may indicate the presence of structural heart disease such as prior MI or chamber enlargement that would increase the likelihood that a patient's symptoms might be due to VA, or it may provide evidence of the underlying substrate for documented VA. An ECG may also reveal evidence of inherited arrhythmia disorders such as long QT syndrome, Brugada syndrome, and arrhythmogenic right ventricular cardiomyopathy. In patients with structural heart disease, QRS duration and the presence of conduction abnormalities provide prognostic information.S4.2.1-7-S4.2.1-14 Data on the use of microvolt T wave alternans and the signal averaged ECG are inconclusive, as such these tests are not routinely used in clinical practiceS4.2.1-15-S4.2.1-19; the one exception is the potential use of signal averaged ECG in patients with arrhythmogenic right ventricular cardiographycecommendation-Specific Supportive Text1. Ambulatory electrocardiographic monitoring is often used to assess the effectiveness of treatments to suppress arrhythmias, but more robust data are needed on the clinical use of this practice. Continuous or intermittent ambulatory electrocardiographic recording with a Holter monitor or an event recorder is helpful in diagnosing suspected arrhythmias, establishing their frequency relating them to symptoms, and assessing the response to therapy. Although the yield of these tests is relatively low, VT is occasionally documented.S4.2.2-4 A 24-hour continuous Holter recording is appropriate when symptoms occur at least once a day or when quantitation of PVCs/NSVT is desired to assess possible VA-related depressed ventricular function. For sporadic symptoms, event or "looping" monitors are more appropriate because they can be activated over extended periods of time and increase diagnostic yield.S4.2.2-2,S4.2.2-3 Adhesive patch electrocardiographic monitors can record for weeks and allow for continuous short-term 1-lead monitoring and patient activation for symptoms. Studies have shown satisfactory patient compliance, and arrhythmia detection; however, with some monitors, detected arrhythmias are not discovered until the patch is returned for analysis.S4.2.2-4 Serial evaluations with exercise testing and/or 24-hour ambulatory monitoring are also used to assess rhythm burden and response of VA to therapy. Notably, implantable monitors are covered in Section 4.2.3. Importantly, when the suspicion of VA are warranted. It is important to accurately correlate the symptoms with the arrhythmias detected by ambulatory ECG monitoring.4.2.3. Implanted Cardiac Monitors Recommendation-Specific Supportive Text1. Implanted cardiac monitoring and stored recordings of electrograms based on patient activation or preset parameters, allowing a prolonged monitoring cardiac monitors provide continuous rhythm monitoring and stored recordings of electrograms based on patient activation or preset parameters, allowing a prolonged monitoring period of a few years. with local anesthesia for implantation. In patients with sporadic symptoms, including syncope, implantable recorders are useful in diagnosing serious tachyarrhythmias. S4.2.3-4 They are generally reserved for patients in whom other ambulatory monitoring is nonrevealing due to the infrequency of events. A 25% added yield in diagnosis has been described after an unrevealing external ambulatory monitor. S4.2.3-5 In a study of patients with syncope, the implantable monitoring, tilt table testing and electrophysiological study. S4.2.3-2 A systematic review in patients with syncope concluded that use of these devices provide a higher rate of diagnosis and a trend toward reduction in syncope relapse after MI, with LVEF 16 beats long) in 13%, VT (>30 s) in 3% and VF in 3% of patients.S4.2.3-1 It is important to accurately correlate the symptoms with the arrhythmias detected by implanted cardiac monitors.4.2.4. Noninvasive Cardiac ImagingRecommendation, along with assessment of global and regional myocardial function, valvular structure and function, along with assessment of global and regional myocardial function, valvular structure and function, along with assessment of global and regional myocardial function, valvular structure and function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function, along with assessment of global and regional myocardial function. or at high risk for VA or SCD, including patients with cardiomyopathy, HF, prior MI, family history of cardiomyopathy or SCD, or an inherited structural heart disease associated with SCD. Echocardiography is the most readily available and commonly used imaging technique.S4.2.4-1,S4.2.4-2 LVEF is a strong, independent predictor of SCD and cardiovascular mortality and a determinant of eligibility for ICD implantation for primary prevention of SCD.S4.2.4-1 In SCD-HeFT (the Sudden Cardiac Death in Heart Failure Trial), S4.2.4-2 the benefit of the ICD was not dependent on the modality (ie, echocardiography, radionuclide angiography, or contrast angiograms) by which the LVEF was assessed. In clinical practice, if cardiac CTS4.2.4-3 or cardiac MRI has been performed and provides sufficient evaluation, echocardiography may be unnecessary. This recommendation for imaging differs from that of the 2017 ACC/AHA/HRS syncope guidelineS4.2.4-4 that applies to patients who may not have VA.2.VA or SCA can be an initial manifestation of ischemic heart disease, cardiomyopathic processes, or myocarditis. Cardiac CT and cardiac MRI allow for evaluation of structure and coronary anatomy including anomalous coronary origins. Cardiac MRI can be useful in the evaluation for myocardial scar and infiltrative processes evident as late gadolinium enhancement.S4.2.4-9 Cardiac MRI also provides high-quality assessment of LV and RV function, size, and degree of fibrosis and is particularly useful in arrhythmogenic right ventricular cardiomyopathy and HCM.4.2.5. BiomarkersRecommendation-Specific Supportive Text1. Elevated levels of natriuretic peptides—B-type natriuretic peptide (BNP) or N-terminal pro-BNP—are associated with increased risk of SCA and appropriate ICD therapies, even after adjustment of LVEF and other risk factors. S4.2.5-1-S4.2.5-4 These biomarkers are also predictive of nonsudden cardiovascular mortality and thus are not specific to SCD risk alone. Natriuretic peptides have also been evaluated for predicting SCD in the general population.S4.2.5-5,S4.2.5-6 In the Nurses' Health Study, an elevated N-terminal pro-BNP was an independent risk marker for SCD in the general population.S4.2.5-5,S4.2.5-6 In the Nurses' Health Study, an elevated N-terminal pro-BNP was an independent risk marker for SCD in the general population.S4.2.5-6 In the Nurses' Health Study, an elevated N-terminal pro-BNP was an independent risk marker for SCD in the general population.S4.2.5-6 In the Nurses' Health Study, an elevated N-terminal pro-BNP was an independent risk marker for SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the general population.S4.2.5-6 In the Nurses' Health Study, and thus are not specific to SCD in the sp higher baseline levels of N-terminal pro-BNP were associated with SCD over a 16-year follow-up period.S4.2.5-6 These biomarkers may also have a potential role in facilitating the identification of individuals at increased risk of SCD and VA in the general population, particularly in those at intermediate or high risk of ischemic heart disease, but further studies are needed. Use of biomarkers has not been shown to be useful for selecting patients for ICDs. A study of 4431 patients found high-sensitivity troponin can improve the current SCD prediction algorithms.4.2.6. Genetic Considerations in Arrhythmia SyndromesSynopsisThe diagnosis of most inherited arrhythmia syndromes can: 1) provide opportunity to confirm a suspected clinical diagnosis and sometimes provide prognostic information for the proband and 2) offer cascade screening of potentially affected family members when a disease-causing mutation is identified in the proband. The yield of genetic testing varies by disease. The verification of pathogenicity of suspected mutations is an evolving field, and exome sequencing has identified an increasing number of variants of uncertain significance in the general population.S4.2.6-1-S4.2.6-5 Genotyping can have therapeutic implications for some arrhythmogenic pathogenic mutation has been clearly identified, the risk to mutation positive individuals has been extensively studied, and effective therapy relevant to the mutation can be instituted. In other diseases, such as Brugada syndrome, the role of a clear monogenic disease-causing mutation is less certain, and the genotype does not provide therapeutic or prognostic information for the proband.S4.2.6-5,S4.2.6-10-S4.2.6-12 In arrhythmogenic right ventricular cardiomyopathy. some desmosomal mutation positive individuals do not develop disease, indicating that additional mutations and environmental interactions likely influence the clinical development of disease, and as such, ongoing monitoring and decision-making are done based on the clinical phenotype. Genotyping is frequently most useful when a pathogenic mutation is identified in the proband, such that screening can be applied to relatives who are gene mutation positive.S4.2.6-7 Refer to Section 7.9 for disease-specific recommendations. In young patients (35% and unexplained syncope or near-syncope may benefit from an electrophysiological study to determine if VT/VF is the cause of symptoms and to guide further therapy. Induction of VT/VF is often attempted before catheter ablation of the arrhythmia substrate to guide the procedure and to determine the success of the intervention after ablation is performed. An electrophysiological study can be used to determine the mechanism of a wide complex tachycardia. See Sections 7.3, 7.4, 7.6, 7.9.1.3, and 10.8 for recommendations regarding electrophysiological study for specific disease states. Recommendation-Specific Supportive Text1. A study of electrophysiological testing in patients with symptomatic NICM found inducible VT/VF in 28% of patients with ischemic or NICM and syncope, induction of VT or VF at electrophysiological study correlated with cardiac mortality only in patients who meet criteria for ICD implantation (ie, HF and LVEF ≤ 35%), data do not support the routine use of electrophysiological study solely for risk stratification, as such patients have been shown to derive survival benefit from the ICD.S4.3.2-11 An electrophysiological study may be helpful, however, in selected patients suspected to have preexcitation or supraventricular arrhythmias as the cause of symptoms or wide complex tachycardias that warrant definitive diagnosis and management. SVT leading to VT/VF or aberrantly conducted SVT may also be suspected in younger patients or those with a preserved LVEF. Induction of SVT and ablation may then be curative, with no need for an ICD. In such cases, failure to induce VT/VF after elimination of the substrate for SVT would be expected.3.Risk stratification for channelopathies is generally made on the basis of symptoms, the ECG,S4.3.2-19-S4.3.2-28-S4.3.2-29-S4.3.2-27 and the results of genetic testing.S4.3.2-29-S4.3.2-27 and the results of genetic testing.S4.3.2-29-S4.3.2stratification in patients with these cardiac channelopathies.S4.3.2-12-S4.3.2-155. Therapies for Treatment or Prevention of VA5.1. Medication TherapyWith the exception of beta blockers (eg, metoprolol succinate, carvedilol), there is no evidence from RCTs that antiarrhythmic medications for VA improve survival when given for the primary or secondary prevention of SCD. However, the use of these medications is essential in some patients to control arrhythmias and improve symptoms. Medication-induced proarrhythmia is addressed in Section 10.7. Antiarrhythmic medications are often categorized by the Vaughan Williams 4-level schema (class I: fast sodium channel blockers; class II: beta blockers; class III: repolarization potassium current blockers; class III: repolarization potassium current blockers; class III: beta blockers; class III: repolarization potassium current blockers; class II multiple effects. Table 7 shows uses, electrophysiological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects, and common adverse effects of Available Antiarrhythmic Medications. Table 7. Pharmacological effects of Available An EffectsPharmacological CharacteristicsCommon Adverse EffectsAcebutololPO 200-1200 mg dailyor up to 600 mg bidVT, PVCsBeta 1, Mild intrinsic sympathomimetic activitySinus rate slowedAV nodal refractoriness increasedActive metabolite t1/2: 8-13 hpProlonged with renal impairment)Metab: HExcr: F 60%, U 40%Cardiac: Bradycardia hypotension, HF, AVBOther: Dizziness, fatigue, anxiety, impotence, hyper/hypoesthesiaAmiodarone (III)IV: 300 mg bolus for VF/pulseless VT arrest; 150-mg bolus f Ito, Beta receptor, Alpha receptornuclear T3receptorSinus rate slowedQRS prolongedQTc prolongedAV nodal refractoriness increased; increased DFTt1/2: 26-107 dMetab: HExcr: FCardiac: Hypotension, bradycardia, AVB, TdP, slows VT below programmed ICD detection rate, increased defibrillation thresholdOther: Corneal microdeposits, thyroid abnormalities, ataxia, nausea, emesis, constipation, photosensitivity, skin discoloration, ataxia, dizziness, peripheral neuropathy, tremor, hepatitis, cirrhosis, pulmonary fibrosis or pneumonitisAtenolol (II)PO: 25-100 mg qd or bidVT, PVC, ARVC, LQTSBeta 1Sinus rate slowedAV nodal refractoriness increasedt1/2: 6-7 h(prolonged with renal impairment) Metab: HExcr: F 50%, U 40%Cardiac: Bradycardia, hypotension, HF, AVBOther: Dizziness, fatigue, depression, impotenceBisoprolol (II)PO: 2.5-10 mg once dailyVT, PVCBeta 1 receptorSinus rate slowedAV nodal refractoriness increasedt1/2: 9-12 hMetab: HExcr: UCardiac: Chest pain, bradycardia, AVB Other: Fatigue, insomnia, diarrheaCarvedilol (II)PO: 3.125-25 mg q 12 hVT, PVCBeta 1 and 2 receptors, AlphaSinus rate slowedAV nodal refractoriness increasedt1/2: 7-10 hMetab: HExcr: FCardiac: Bradycardia, hypotension, AVB, edema, syncopeOther: Hyperglycemia, dizziness, fatigue, diarrheaDiltiazem (IV)IV: 5-10 mgqd: 15-30 minExtended release: PO: 120-360 mg/dayVT specificallyRVOT, idiopathic LVTICa-LSinus rate slowedPR prolongedAV nodal conduction slowedt1/2: Injection 2-5 h, immediate release 12 h, and severe hepatic impairment 14-16 hMetab: HExcr: UCardiac: Hypotension, edema, HF, AVB, bradycardia, exacerbation of HFrEFOther: Headache, rash, constipationEsmolol (II)IV: 0.5 mg/kg bolus, 0.05 mg/kg/minVTBeta 1 receptorSinus rate slowedAV nodal refractoriness increasedt1/2: 9 minMetab: RBC esterasesExcr: UCardiac: Bradycardia, hypotension, HF, AVBOther: Dizziness, nauseaFlecainide (IC) PO: 50-200 mg g 12 hVT, PVC (in the absence of structural heart disease). Has a role in treating patients with CPVTINa, IKr, IKurPR prolongedQRS prolonged; increased DFTt1/2: 7-22 hMetab: HExcr: UCardiac: Sinus node dysfunction, AVB, drug-induced Brugada syndrome, monomorphic VT in patients with a myocardial scar, exacerbation of HFrEFOther: Dizziness, tremor, vision disturbance, dyspnea, nauseaLidocaine (IB)IV: 1 mg/kg bolus, 1-3 mg/min1-1.5 mg/kg. Repeat 0.5-0.75 mg/kg bolus every 5-10 min (max cumulative dose 3 mg/kg). Maintenance infusion is 1-4 mg/min although one could start at 0.5 mg/kg. Repeat 0.5-0.75 mg/kg bolus every 5-10 min (max cumulative dose 3 mg/kg). Maintenance infusion is 1-4 mg/min although one could start at 0.5 mg/kg. Metab: HExcr: UCardiac: Bradycardia, hemodynamic collapse, AVB, sinus arrestOther: Delirium, psychosis, seizure, nausea, tinnitus, dyspnea, bronchospasmMetoprolol (II)IV: 5 mg q 5 min up to 3 dosesPO: 25-100 mg Extended release gd or q 12 hVT, PVCBeta 1 receptorSinus rate slowedAV nodal refractoriness increasedt1/2: 3-4 hMetab: HExcr: UCardiac: Bradycardia, hypotension, AVBOther: Dizziness, fatigue, diarrhea, depression, dyspneaMexiletine (IB)PO: 150-300 mg q 8 h or q 12 hT, VF, PVC, has a role in patients with LQT3INaNo marked effect on most intervals; QTc can slightly shortent1/2: 10-14 hMetab: HExcr: UCardiac: HF, AVBOther: Lightheaded, tremor, ataxia, paresthesias, nausea, blood dyscrasiasNadolol (II)PO: 40-320 mg dailyVT, PVC, LQTS, CPVTBeta 1 and 2 receptorsSinus rate slowedAV nodal refractoriness increasedt1/2: 20-24 hMetab: noneExcr: UCardiac: Bradycardia, hypotension, HF, AVBOther: Edema, dizziness, cold extremities, bronchospasmProcainamide (IA)IV: loading dose 10-17 mg/kg at 20-50 mg/minMaintenance dose: 1-4 mg/minPO (SR preparation): 500-1250 mg q 6 hVTINa, IKrQRS prolongedQTc prolonged; increased DFTMetab: Ht1/2: 2-5 h; NAPA 6-8 ht1/2 prolonged; increased DFTMetab: Ht1/2: 2-5 h; NAPA 6-8 ht1/2: 2-5 h; NA blood dyscrasiasPropafenone (IC)PO: Immediate release 150-300 mg q 8 hExtended release 225-425 mg q 12 hVT, PVC (in the absence of structural heart disease)INa, IKr, IKur, Beta receptor PR prolongedQRS prolonged; increased DFTt1/2: 2-10 h or 10-32 ht1/2: extensive metabolizers 2-10 h; poor metabolizers 10-32 h.Metab: HExcr: UCardiac: HF, AVB, drug-induced Brugada syndromeOther: Dizziness, fatigue, nausea, diarrhea, xerostomia, tremor, blurred visionPropranolol (II)IV: 1-3 mg q 5 min to a total of 5 mgPO: Immediate release 10-40 mg q 6 h; Extended release 60-160 mg q 12 hVT, PVC, LQTSBeta 1 and 2 receptors, INaSinus rate slowedAV nodal refractoriness increasedt1/2: Immediate release 3-6 hExtended release 8-10 hMetab: HExcr: UCardiac: Bradycardia, hypotension, HF, AVBOther: Sleep disorder, dizziness, nightmares, hyperglycemia, diarrhea, bronchospasmQuinidine (IA)PO: sulfate salt 200-600 mg q 6 h to q 12 hgluconate salt 324-648 mg q 8 h to q at 50 mg/minT, VF, (including short QT syndrome, Brugada)INa, Ito, IKr, M, Alpha receptorQRS prolongedQTc prolongedQTc prolongedQTc prolongedQTc prolonged, increased DFT1/2: 6-8 h longer in HF, liver cirrhosis, and with older ageMetab: HExcr: UCardiac: Syncope, TdP, AVBOther: Dizziness, diarrhea, nausea, esophagitis, emesis, tinnitus, blurred vision, rash, weakness, tremor, blood dyscrasiasRanolazine (not classified)PO: 500-1000 mg q 12 hVTINa, IKrSinus rate slowedTc prolongedt1/2: 7 hMetab: HExcr: U 75%, F 25%Cardiac: Bradycardia, hypotensionOther: Headache, dizziness, syncope, nausea, dyspneaSotalol (III)IV: 75 mg q 12 hPO: 80-120 mg q 12 h, may increase dose every 3 d; max 320 mg/dVT, VF, PVCIKr, Beta 1 and 2 receptorSinus rate slowedQTc prolongedAV nodal refractoriness increased; decreased DFTt1/2: 12 hMetab: noneExcr: UCardiac: Bradycardia, hypotension, nausea, diarrheaVerapamil (IV)IV: 2.5-5 mg q 15-30 minSustained release PO: 240-480 mg/dVT (specifically RVOT, verapamil-sensitive idiopathic LVT)ICa-LSinus rate slowedPR prolongedAV nodal conduction slowedt1/2: 3-7 hMetab: HExcr: UCardiac: Hypotension, edema, HF, AVB, bradycardia, exacerbation of HFrEFOther: Headache, rash, gingival hyperplasia, constipation, dyspepsia5.1.1. Medications With Prominent Sodium Channel BlockadeExcept in specific circumstances, sodium channel blockers (Vaughn-Williams class I agents) have a limited role in the prevention of VT/SCD; this is based on a lack of survival benefit and increased mortality observed during chronic therapy in patients with ischemic heart disease (see Section 10.7). Specific circumstances where sodium channel blockers have been used to treat VT/SCA include: intravenous lidocaine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; and flecainide for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quinidine for patients with refractory VT/cardiac arrest (especially witnessed)S5.1.5.2-4; quint arrest (especially witnessed)S5.1.5.2-4; quint arrest (especially witnessed)S5.1.5.2-4; quint arrest (especially witnessed)S5.1.5.2-4; quint arres tachycardia.S5.1.5.2-5 These medications could also be used in ICD patients with drug- and ablation-refractory VT. One newer medication, developed and FDA-approved as an antianginal agent, provides relatively specific late sodium channel current blockade in addition to less potent blockade of the phase 3 repolarizing potassium current; that is, the rapid delayed rectifier potassium current; IKr. The potential for clinical data are scant. In a study of 12 patients, ranolazine reduced ICD shocks in otherwise medicationresistant VT/VF in 11 patients.S5.1.5.2-7 In MERLIN TIMI-36 (Metabolic Efficiency With Ranolazine for Less Ischemia in Non-ST-Elevation Acute Coronary Syndromes-Thrombolysis In Myocardial Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis In Myocardial Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis In Myocardial Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis In Myocardial Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis In Myocardial Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis In Myocardial Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis In Myocardial Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis In Myocardial Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis In Myocardial Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment elevation Acute Coronary Syndromes-Thrombolysis Infarction 36), ranolazine did not reduce VT in the first few days after a non-ST-segment ICD patients with ischemic or NICM were randomly assigned to ranolazine 1000 mg twice a day versus placebo.S5.1.5.2-9 High risk was defined as: 1) having a primary prevention ICD without a history of documented VT/VF and with one of the following conditions: BUN ≥ 26 mg/dL, QRS > 120 msec, atrial fibrillation, or NSVT or > 500 VPBs on 24hour Holter recording; 2) having a primary prevention ICD with a history of documented VT/VF appropriately treated with ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD therapy or untreated VT/VF appropriately treated with ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriately treated with ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriately treated with ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriately treated with ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriate ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriate ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriate ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriate ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriate ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriate ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriate ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriate ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented VT/VF appropriate ICD therapy or untreated NSVT; or 3) having a secondary prevention ICD with a history of documented NSVT; or 3) having a secondary prevention ICD with a history of documented NSVT; or 3) having a secondary prevention ICD with a history of documented NSVT; or 3) having a secondary prevention ICD with a history of documented NSVT; or 3) hav death. In a prespecified secondary analysis, ranolazine was associated with a significant reduction in VT events treated with anti-tachycardia pacing.S5.1.5.2-95.1.2. Beta Blockers are often first-line antiarrhythmic therapy.S5.1.5.2-95.1.2. 10,S5.1.5.2-11 Their antiarrhythmic efficacy is related to the effects of adrenergic-receptor blockade on sympathetically mediated triggering mechanisms, slowing of the sinus rate, and possibly inhibition of excess calcium release by the ryanodine receptor.S5.1.5.2-12Beta blockers reduce all-cause mortality and SCD in patients with HF with reduced EF (HFrEF).S5.1.5.2-13-S5.1.5.2-15 Although beta blockers have long been proven to reduce mortality after MI,S5.1.5.2-16 registry data confirm that early beta blocker use in patients with MI and risk factors for shock (>70 years of age, symptoms

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