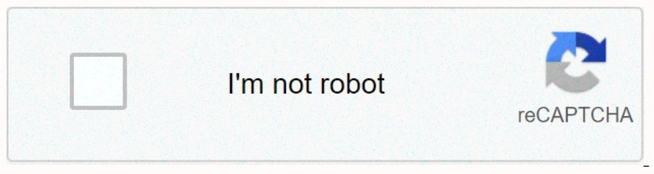

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2017 AHA/ACC/HRS Guidelines for the Management of Ventricular Rhythm Patients and Prevention of Sudden Cardiac Death: Summary: American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society report. 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 AND, Kay GN, Matlock DD, Myerburg RJ, Page RL. Al-Khatib SM, et al. Circulation. 2018 Sed 25;138(13):e210-e271. Doi: 10.1161/CIR.0000000000000548. Circulation. 2018. PMID: 29084733 Abstract is not available. 2017 AHA/ACC/HRS Guidance for the Treatment of Ventricular Rhythm Patients and Prevention of Sudden Cardiac Death: American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society report. 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 AND, Kay GN, Matlock DD, Myerburg RJ, Page RL. Al-Khatib SM, et al. Heart rhythm. 15.10.2018:e73-e189. doi: 10.1016/j.hrthm.2017.10.036. Epub 2017 October 30. The rhythm of the heart. 2018. PMID: 29097319 Review. Abstract is not available. Introduction e2741. Introduction e2751.1. Examination of methods and evidence e2751.2. Organisation of the Writing Committee e2761.3. Documentary review and approval e2761.4. Scope of guideline e2771.5 Abbreviations e2792. Epidemiology e2792.1. General concepts e2792.1.1. Premature chamber complexes and unsustainable VT e2792.1.2. VT and VF during ACS e2812.1.3. Continuing VT and VF are not related to ACS e2812.2. Sudden cardiac death e2812.2.1. Incidence of SCD e2812.2.2. Population subgroups and risk forecast e2823. Va e2833.1 mechanisms Cell mechanisms and substrates e2833.2. 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Improve. guidelines based on systematic methods for evaluating and classifying evidence provide an cornerstone for high-quality cardiovascular care. Acc and AHA sponsor the development and publication of guidelines without commercial support, and members of each organization voluntarily announce their time for writing and review efforts. The guidelines are the official policy of the ACC and the AHA. Intended use Policy guidelines make recommendations for patients who are or are at risk of cardiovascular disease. The focus is on medical practice in the United States, but the guidelines developed in collaboration with other organizations can have a global impact. Although the guidelines can be used to inform regulatory or payer decisions, they are designed to improve the quality of patient care and adapt to patients' interests. The guidelines are intended to define practices that are against patients' needs in most circumstances, but not in all circumstances, and should not replace clinical assessment. Clinical implementation The treatment recommended by the Guides is effective only if monitored by healthcare providers and patients. Compliance with the recommendations can be enhanced through joint decision-making by healthcare providers and patients, in which patients undertake to choose measures based on individual values, preferences and related conditions and infectious diseases. Methodology and modernisation The ACC/AHA Task Force will continuously review, update and change indicative methods based on published standards from organisations including the Institute of MedicineP-1,P-2 and an internal reassessment. Similarly, the presentation and delivery of the guidelines will be revalued and changed based on emerging technologies and other factors in order to facilitate the optimal dissemination of information at the treatment point to healthcare professionals. Toward this goal, this guide indicates the introduction of an advanced format that provides help recommendations and related text called a modular data block shape. Each modular block contains a table of related recommendations, a short summary, recommendation-specific support text, and flowcharts or additional tables, if necessary. There are references in the modular block itself to facilitate quick viewing. This format also makes it easier to seamlessly update your instructions with targeted updates when new evidence is released, and to mark content for a quick electronic search of relevant recommendations on a topic of interest. This evolved form emerged when this guideline was almost complete; Therefore, the current document represents the transition formatting that best fits the typed text. In future guidelines, this fully implemented, including provisions on limiting the amount of the text of the guideline. Identification Identification where appropriate and feasible, an analysis of the cost-value aspects set out in certain Guidelines may be analysed on the value of the medicinal product, device or measure in accordance with the ACC/AHA methodology. P-3 In order to keep the guideline recommendations up to date, new data will be continuously reviewed and all guidelines audits were commissioned over periods of approximately 6 years. New, potentially policy-changing, research results relevant to an existing or new medication, device or management strategy will be published, and the working group will assess, in the relevant guidance committee, whether a targeted update should be ordered. For more information and practices on how to develop guidelines, we encourage readers to consult the ACC/AHA Guidance Method ManualP-4 and other methodology articles. P-5–P-8 Election of the members of the Committee on Writing The working group aims to avoid bias by selecting experts from a wide range of backgrounds. The members of the Writing Committee represent different geographic areas, genders, ethnic groups, races, intellectual perspectives/biases, and the extent of clinical practice. The working group may also invite organisations and professional societies with related interests and expertise to participate as partners, partners or supporters. Relationships with industry and other entities ACC and AHA have strict policies and methods in place to ensure guidelines are developed without bias or inappropriate impact. Full relationships with industry and other entities (RWI) can be found online. Appendix 1 to the current document lists the relevant RWI of the members of the Writing Committee. In order to ensure full transparency, comprehensive disclosure information for the members of the Writing Committee is available online, as well as comprehensive disclosure information from the working group. Evidence assessment and evidence evaluation committees When developing recommendations, the Writing Committee shall use evidence-based methods based on all available information. P-4–P-7 Literature searches focus on randomized controlled trials (RCTs), but also include registers, non-breasted comparing and describing studies, case series, cohort studies, systematic evaluations and expert opinions. Only key references are mentioned. The Independent Evidence Review Committee (ERC) has been commissioned when there are ≥1 clinically very important issues that merit a formal systematic review. This systematic review aims to determine which patients are most likely to benefit from a strategy for testing, medication, equipment or treatment and to what extent. (a) the absence of a current authoritative systematic review; (b) benefit; it is possible to define a guideline in a timeframe consistent with the writing; (c) (c) the importance of a significant number of patients; and (d) the likelihood that the findings may be translated into enforceable recommendations. Members of the ERC may include meth evidence, epidemiologists, healthcare providers and biostatists. Once an official systematic review has been commissioned, the recommendations developed by the Writing Committee on the basis of a systematic review shall be marked with an SR. Guidance-controlled management and treatment The management and treatment (GDMT) guided by the guidelines includes clinical evaluation, diagnostic testing and pharmacological and processed treatments. For these and all recommended doses of medicinal products, the reader should confirm the dosage by reviewing the added material of the product and evaluate the treatment programme for contingencies and interactions. The recommendations are limited to medicinal products, devices and treatments approved for clinical use in the United States.The recommended category and the certification level Recommendation category (COR) demonstrates the strength of the recommendation, which covers the magnitude and certainty of the estimated efficiency in relation to the risk. The level of evidence (LOE) assesses scientific evidence supporting intervention based on the type, quantity and consistency of data from clinical trials and other sources (Table 1). P-4,P-6,P-8Table 1. Application of the recommended class and evidence level to clinical strategies, interventions, treatments or diagnostic tests in patient care* (updated August 2015)Glenn N. Levine, MD, FACC, FAHAChair, ACC/AHA Working Group on Clinical Practice Guidelines1. Introduction1.1. Methods and examination of evidence The recommendations listed in this Clinical Practice Guideline are based, as far as possible, on evidence. From April 2016 to September 2016, the first extensive screen review included literature related to the English and MEDLINE index (via PubMed), embase, Cochrane Library, Healthcare Research and Quality Agency and other selected databases relevant to this Guideline. Key keywords included: sudden cardiac death, ventricular tachycardia, ventricular fibrillation, premature ventricular contractions, implantable cardiooverter defibrillator, subcutaneous implantable cardiovascular defibrillator, wearable cardiooverter defibrillator and catheter ablation. The relevant additional studies published during the march 2017 guidance process were also taken into account by the Writing Committee and added to the evidence tables if necessary. The final evidence tables are included in the online data attachment and summarize the evidence used by the Writing Committee to formulate recommendations. In addition, the Writing Committee examined documents relating to: arrhythmias (VA) and sudden cardiac death (SCD), previously published by acc, AHA and Heart Rhythm Society (HRS). References selected and published in this document are representative and not all-inclusive references. As stated in the judgment of 10 December 2007, the independent ERC was commissioned to carry out a formal systematic review of 2 important clinical issues for which clear literature and the previous guideline consency were considered to be missing or limited (Table 2). The results of the ERC review were considered by the Writing Committee, which was included in this guideline. In the context of this process, the members of the Writing Committee evaluate other published information related to the guidelines. The findings of the ERC and the members of the Writing Committee were formally presented and discussed and guidelines were then developed. In connection with this Guide, a systematic review of the 2017 AHA/ACC/HRS Guidelines for the treatment of patients with ventricular arrhythmias and the prevention of sudden cardiac death will be published. S1.4-1Table 2. Systematic review questions about SCD PreventionQuestion NumberQuestionSection Number1 In patients with asymptomatic Brugada syndrome, What is the link between abnormal programmed ventricular stimulation study and scd and other arrhythmia endpoints?7.9.1.32In fact, the effect of ICD implantation on primary prevention in elderly patients and patients with significant involvement?10.3 ACC and AHA have recognised the importance of value in healthcare and required the possible development of the value level of clinical practice recommendations. S1.4-2 The available cost-effectiveness data were found to be sufficient to support 2 specific recommendations in this Guideline (see sections 7.1.1 and 7.1.2). As a result, these 2 recommendations provided a level of value on the basis of the ACC/AHA statement on cost/value methodology for clinical practice and performance measures, as considered in Table 3.S1.4-2 Fair Quality of Life (QoL) data to be insufficient to support the specific recommendations of this Guideline. Table 3. Proposed inclusion of value level in clinical practice guideline recommendations *High value of value: Better results at lower cost or ICER < $50,000 per QALY achieved QALYIntermediate value: $50,000 < $150,000 per QALY obtainedValue: $≥,150,000 per QALY obtainedUntacer value: but the data are not sufficient to draw conclusions because of studies, low-quality studies, conflicting studies, or previous studies that are no longer relevant Not evaluated: Value not evaluated by the Writing CommitteeThe proposed abbreviations for each value recommendation: Level of value: H to indicate high value; I, intermediate value; L, low value; U, uncertain value; and NA, has not been estimated on 1 February. Writing Committee OrganizationWriting Committee OrganizationWriting Committee electrocardiologists (including paediatric specialists), general adult and paediatric cardiologists (including critical treatment and acute coronary syndrome [ACS], specialist in genetic cardiology, heart failure and cost-effectiveness analyses), geriatria with expertise in terminal therapy and joint decision-making, and lay representative in addition to representatives of ACC, AHA, HRS and Heart Failure Society of America (HFSA)1.3. Review and approval of documentsInveinve of this document by two official evaluator appointed by ACC, AHA and HRS; 1 official lay assessor designated by the AHA; 1 organisation assessor appointed by the HFSA; and 28 individual content reviewers. The reviewers' RWI information was shared with the Writing Committee and will be published in this document (Appendix 2). This document was approved for publication by the governing bodies of the ACC, the AHA and the High Representative. and approved by the HFSA.1.4. Scope of the Guideline The purpose of this AHA/ACC/HRS document is to provide modern guidance for the treatment of adults with VA or at risk of mixed therapy, including diseases and syndromes associated with VA. This guide replaces the ACC/AHA/ESC 2006 guidelines for the treatment of patients with ventricular arrhythmias and the prevention of sudden cardiac death. S1.4-4 It is also replaced by some parts ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Heart Rhythm Abnormalities,S1.4-5 specifically those sections concerning the indications for implantable cardiooverter defibrillator (ICD); and it updates scd prevention recommendations in the 2011 ACCF/AHA Guidelines for the diagnosis and treatment of hypertrophic cardiomyopathy. S1.4-6 Some of the recommendations of the previous guidelines have been updated due to new evidence or a better understanding of existing evidence, and meaningless or overlapping recommendations were removed or amended. The current guideline defines sudden cardiac arrest (SCA) as a sudden cessation of cardiac function so that the victim does not respond, without normal breathing and signs of blood circulation. S1.4-7 If corrective measures are not taken quickly, this mode will progress to scd. Cardiac arrest is used to mark an event that can be reversed, usually by cardiac and pulmonary resuscitation (CPR), medication and/ or defibrillation or cardio version. SCA and SCD may be due to causes other than VA, such as bradyarrrhythmia, electromectic dissociation, pulmonary embolism, intracchial bleeding and aortic dissection; However, this document covers only SCA and SCD resulting from the VA. This programme contains references from ICD to VA treatment and SCD prevention, but does not delve into details of the selection and programming of individual devices, relevant factors for cardiac resynchronisation therapy (CRT), bradycardia t anxiety and hemodynamic monitoring. These important aspects of the management of the ICD have been addressed in the consensus opinion of the SG/A expert. S1.4-8 AHA Science Advisory discusses the use of wearable cardiovascular defibrillators. S1.4-9 The conclusions of the document were reviewed. However, recommendations on this subject have been developed regardless of the document. This guide contains indications of VA catheter ablation, but does not contain recommendations for specific techniques or ablation techniques that were not covered by this document. Interventional treatment recommendations, including ablation and implantation of devices, shall apply only if these treatments can be carried out by qualified doctors in such a way that, in accordance with the published literature, consistent results are a reasonable expectation. The Writing Committee agreed that high expertise is particularly important for va catheter ablation, and this point continues to be highlighted in the relevant sections. In addition, all IKKD-related recommendations require that relevant survival is expected during the year ; meaningful survival means that the patient has a reasonable quality of life and functional status. Although this document is intended for the adult population (≥18 years of age) and does not provide specific recommendations for paediat child patients, some literature on paediat child patients was examined. In some cases, the data of paediatric patients after their infancy helped inform about this guideline. The Writing Committee recognised the importance of shared decision-making and patient-centred care and, where possible, sought to draw up recommendations relevant to these important concepts. The importance of the joint decision-making process, in which patients, families and doctors discuss the risks and benefits of diagnostic and treatment options and reflect on patients' personal preferences, is emphasised (see paragraph 15). In developing this guidance, the Writing Committee reviewed previously published guidelines and related opinions. Table 4 lists the guidelines and statements that are considered relevant to this writing effort and are intended to be used as a resource, preventing the current Help recommendations from being repeated. Table 4. Related instructions and statementsTitleOrganizationPublication Year (Ref)Guidelines SyncopeACC/AHA/HRS2017S1.4-10 Heart failureACCF/AHA2017S1..4-11 2016,S1.4-12 and 2013S1.4-13 Valvular Heart DiseaseAHA/ACC2017S1.4-1 4 and 2014S1.4-15 Supraventricular tachycardiaACC/AHA/HRS2015S1.4-16 Ventricular arrhythmias and heart Prevention of sudden death of originESC2015S1.4-17 Guidelines for cardiopupul resuscitation and cardiovascular emergency servicesAHA2015S1.4-18 Acute coronary artery disease coronary artery disease of cardiovascular riskACC/AHA2013S1.4-21 ST-elevation myocardial infarctionACCF/AHA2013S1.4-22 Acute myocardial infarction in patients presenting with ST-segment elevationESC2012S1.4-23 Device-based therapies for cardiac rhythm abnormalitiesACCF/AHA/HRS2012S1.4-24 Coronary artery bypass graft surgeryACCF/AHA2011S1.4-25 Hypertrophic cardiomyopathyACCF/AHA2011S1.4-6 Percutaneous coronary interventionACCF/AHA/SCAI2011S1.4-26 Secondary prevention and risk reduction therapy for patients with coronary and other atherosclerotic vascular diseaseAHA/ACCF2011S1.4-27Scientific Statements Wearable cardioverter-defibrillator therapy for the prevention of sudden cardiac deathAHA2016S1.4-9 Optimal implantable cardioverter defibrillator programming and testingHRS/EHRA/APHRS/SOLAECE2016S1.4-8 Treatment of cardiac arrest: current status and future directions: strategies to improve cardiac arrest survivalIOM2015S1.4-28 Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalitiesACC/AHA2015S1.4-29 Ventricular arrhythmiasEHRA/HRS/APHRS2014S1.4-30 Arrhythmias in adult congenital heart diseasePACES/HRS2014S1.4-31 Implantable cardioverter-defibrillator therapy in patients who are not included or not well represented in clinical trialsHRS/ACC/AHA2014S1.4-32 Cardiac sarcoidosisHRS2014S1.4-33 Inherited primary arrhythmia syndromesHRS/EHRA/APHRS2013S1.4-34During final production review of the guidelines , useita suosituksia tarkennettiin vastaamaan paremmin tietoja ja nykyistä suositeltua lääketieteellistä käytäntöä. Nämä parannukset tarkasteltiin ja hyväksyttiin kirjoituskomiteassa, työryhmässä sekä ACC:n, AHA:n ja HRS:n organisaatiojohdossa. Nämä suositukset olivat:7.1.1 kohta, suositus 1Jakso 7.1.3., suositus 2Section 7.2.1., suositus 1Jakso 7.9.1.4., suositus 2Jakso 10.8., suositus 6Lukijat viittaavat näihin jaksoihin päivitetyssä tekstissä.1.5. LyhenteetAbbreviationMeaning/PhraseACSacute sepelvaltimo-oireyhtymätAED automaattinen ulkoinen defibrillaattoriAMIacute sydäninfarktiBNPB-tyyppinen natriureettinen peptidiCABGcoronary valtimo ohitusleikkaus graftCKDchronic munuaissairausCPRcardiopulmonaarinen elvytysCRTcardiac uudelleensynkronointihoitoCTtietokonellinen tomografiaECGelectrocardiogramERCevidence arviointikomiteaESRDend-vaiheen verisuonisairausGDMTguideliinin ohjaama hoito ja hoitoHCMhypertrophic cardi Heitto-osuusICDimplantable cardioverter-defibrillatorLVleft ventricularLVADleft ventricular assist deviceLVEFleft ventricular ejection

fraction MImyokardiaalinen infarktiNICMnonischemic kardiomyopatiaNSVTnonsustained kammiotakykardiaPETpositronipäästötmalliPCIperkutaaninen sepelvaltimon interventioPVCpremature kammiokompleksiQoLquality of kontrolloitu trialRVright ventricularRVOTright kammion ulosvirtaus tractSCAsudden sydämenpysähdysSCDsudden sydänkuolemaSVTsupraventrikulaarinen takykardiaTOFtetralogia FallotVAventricular rytmihäiriöVTventrikulaarinen takykardia2. Epidemiologia2.1. Yleiset käsitteetTable 5. Taulukko yleisesti käytettyjen termien määritelmistä tässä asiakirjassaTermFinition tai DescriptionVentrikulaariset takykardiatS2.2.2-2 Kammioista peräisin olevien ≥3 peräkkäisen kompleksin rytmihäiriöt nopeudella >100 000 bpm (syklin <600 ms).= types= of= vt:sustained:= vt=>pituus: 30 s tai hemodynaamisen kompromissin <30 s.nonsustained/unsustained:= ≥3= beats,= terminating= spontaneously.monomorphic:= stable= single= qrs= morphology= from= beat= to= beat.polymorphic:= changing= or= multiform= qrs= morphology= from= beat= to= beat.bidirectional:= vt= with= a= beat-to-beat= alternation= in= the= qrs= frontal= plane= axis,= often= seen= in= the= setting= of= digitalis= toxicity= or= catecholaminergic= polymorphic= vtmonomorphic= vtpolymorphic= vtbidirectional= vttorsades= de= pointess2.2.2-2torsades= de= pointes= is= polymorphic= vt= that= occurs= in= the= setting= of= a= long= qt= interval= and= is= characterized= by= a= waxing= and= waning= qrs= amplitude.= it= often= has= a= long-short= initiating= sequence= with= a= long= coupling= interval= to= the= first= vt= beat= and= may= present= with= salvos= of= nsvt.= the= twisting= of= the= points ,= although= characteristic,= may= not= always= be= seen,= especially= if= the= episode= is= nonsustained= or= if= only= a= limited= number= of= leads= are= available.= torsades= de= pointes= can= result= from= bradycardia= including= high-grade= av= block= that= leads= to= a= long-short= sequence= initiating= torsades= de= pointes.ventricular= flutters2.2.2-2a= regular= va= ≈300= bpm= (cycle= length:= 200= ms)= with= a= sinusoidal,= monomorphic= appearance;= no= isoelectric= interval= between= successive= qrs= complexes.ventricular= fibrillations2.2.2-2rapid,= grossly= irregular= electrical= activity= with= marked= variability= in= electrocardiographic= waveform,= ventricular= rate= usually=>vuoksi 300 000 km/h (syklin pituus: 30 s tai hemodynaamisen kompromissin vuoksi 300 000 km/h (syklin pituus: <200 ms). Sudden cardiac arrestS2.2.2-2SCA is the sudden cessation of cardiac activity such that the victim becomes unresponsive, with either persisting gasping respirations or absence of any respiratory movements, and no signs of circulation as manifest by the absence of a perceptible pulse. An arrest is presumed to be of cardiac etiology unless it is known or likely to have been caused by trauma, drowning, respiratory failure or asphyxia, electrocution, drug overdose, or any other noncardiac cause. Sudden cardiac deathS2.2.2-2Sudden and unexpected death occurring within an hour of the onset of symptoms, or occurring in patients dead within 24 h of being asymptomatic and presumably due to a cardiac arrhythmia or hemodynamic catastrophe. VT/VF stormS2.2.2-3VT/VF storm (electrical storm or arrhythmic storm) refers to a state of cardiac electrical instability that is defined by ≥3 episodes of ms).sudden= cardiac= arrests2.2.2-2sca= is= the= sudden= cessation= of= cardiac= activity= such= that= the= victim= becomes= unresponsive,= with= either= persisting= gasping= respirations= or= absence= of= any= respiratory= movements,= and= no= signs= of= circulation= as= manifest= by= the= absence= of= a= perceptible= pulse.= an= arrest= is= presumed= to= be= of= cardiac= etiology= unless= it= is= known= or= likely= to= have= been= caused= by= trauma,= drowning,= respiratory= failure= or= asphyxia,= electrocution,= drug= overdose,= or= any= other= noncardiac= cause.sudden= cardiac= deaths2.2.2-2sudden= and= unexpected= death= occurring= within= an= hour= of= the= onset= of= symptoms,= or= occurring= in= patients= found= dead= within= 24= h= of= being= asymptomatic= and= presumably= due= to= a= cardiac= arrhythmia= or= hemodynamic= catastrophe.vt/vf= storms2.2.2-3vt/vf= storm= (electrical= storm= or= arrhythmic= storm)= refers= to= a= state= of= cardiac= electrical= instability= that= is= defined= by= ≥3= episodes= of=></200 ms). Sudden cardiac arrestS2.2.2-2SCA is the sudden cessation of cardiac activity such that the victim becomes unresponsive, with either persisting gasping respirations or absence of any respiratory movements, and no signs of circulation as manifest by the absence of a perceptible pulse. An arrest is presumed to be of cardiac etiology unless it is known or likely to have been caused by trauma, drowning, respiratory failure or asphyxia, electrocution, drug overdose, or any other noncardiac cause. Sudden cardiac deathS2.2.2-2Sudden and unexpected death occurring within an hour of the onset of symptoms, or occurring in patients found dead within 24 h of being asymptomatic and presumably due to a cardiac arrhythmia or hemodynamic catastrophe. VT/VF stormS2.2.2-3VT/VF storm (electrical storm or arrhythmic storm) refers to a state of cardiac electrical instability that is defined by ≥3 episodes of > </30> </600> </600> Appropriate impacts caused by VT, VF or ICD within 24 hours. Primary prevention ICDS2.2.2-2ICD placement with the aim of preventing SCD in patients who have not had VT or SCA but have an increased risk for these events. Secondary contraception ICDS2.2.2-2ICD in patients with a history of SCA, long-term VT or VA-induced fainting. Structural heart disease*This term covers IHD, all types of cardiomyopathy, controlled heart disease and congenital heart disease in adults. Cardiac channelopathyS2.2.2-4 Arrhythmia disease due to a genetic abnormality that leads to cardiac ion channel dysfunction (e.g. long QT syndrome, calycolimin-related polymorphic VT). VA contains a spectrum ranging from premature ventricular complex (PVC) to ventricular fibrillar (VF), the clinical presentation of which ranges from a complete lack of symptoms to cardiac arrest. Most life-threatening heart disease is associated with ischemical heart disease, especially in older patients. The risks of S2.2.2-1 Va. and SCD vary in certain populations with different underlying heart diseases and have a specific family history and genetic variants, and this variation has significant implications for the research and application of treatments.2.1.1. Premature ventricular complexes and unsustainable VTPVCs are common and multiply with age. Although PVC was found in a healthy military population of only 0.6 <20 years= of= age= and= 2.7%= of= those=>% among 12-lead ECGs aged 2.2.2 to 5 in the 50s, longer-term monitoring shows that PVC's are in about 50% of all heart disease or without heart disease. S2. 2.2-6 2-6 2-minute follow-up of middle-aged patients in ARIC (Atherosclerosis Risk In Communities) was associated with an increased risk of developing both sechemical heart disease and mortality with or without general sechemical heart disease. S2.2.2-7,S2.2.2-8 PVCcs frequently used in the general population, defined as at least PVC 12-lead ECG or >30 PVC per hour, are associated with increased cardiovascular risk and increased mortality. S2. 2.2-9 In a study from Taiwan in patients without long-term VT or structural heart disease and having 24-hour Holter monitoring for clinical evaluation, multifocal PVC compounds were associated with an increased risk of death and non-fatal cardiovascular adverse reactions. S2. 2.2-10 In the same population, unsustainable ventricular tachycardia (NSVT) was independently associated with an increased risk of death and other cardiovascular adverse reactions such as stroke. S2.2.2-11 In the ARIC population there was also a link to PVC, which had an increased risk of stroke. S2.2.2-8If some studies have shown that: adverse reactions, detection of PVC,</20> especially if it is multifocal and frequent, is generally considered a risk factor for adverse reactions and such patients are generally assessed ensure that they do not have underlying diseases (e.g. ischemical heart disease, left ventricular [LV] dysfunction) that require further treatment to reduce the risk. PVC and NSVT in patients with cardiovascular diseases are common and have been associated with side effects. S2. 2. 2-12, S2. In 2.2-13 CAST studies (cardiac arrhythmia suppression studies), treatment of patients with post-infarction infarction (e. g. flecainide, encainide, bridesin) increased the risk of death despite va suppression. S2.2.2-14,S2.2.2-15 Antiarrhythmia drugs for PVC treatment have not been shown to reduce mortality and in the post-caracass population class I sodium tract inhibition therapy (e. g. quiniidine, flecainide) increases the risk of death. S2.2.2-15,S2.2.2-16 Similarly, patients with reduced LVEF class I sodium channel inhibitors and d-sotalol increase the risk of death. S2.2.2-16,S2.2.2-17 Beta-blockers, nondihydropyridines calcium channel inhibitors and some antiarrhythmia medications can alleviate the symptoms of palpitations. S2.2.2-18PVC, which occurs during the exercise test, carries a higher risk of death. S2.2.2-19 PVC's occurring during recovery in one study predict death more strongly than PVC's, which occur only during exercise. However, PVC's are common in trained athletes treating palpitations, where the risk of death does not appear to have increased based on studies in small athletes, at least not in those without other cardiovascular abnormalities. S2.2.2-21,S2.2.2-22 Complex PVCcs do not necessarily represent a benign observation of endurance athletes. Electrophysiological examination may be required to assess the risk of arrhythmia in patients. S2. 2.2-22 Very frequent VCCs, >10,000 to 20,000 per day, may be associated with depression of LV function in some patients, which is reversible in PVC control and has been used as pvc-induced cardiomyopathy. S2.2.2-23,S2.2.2-24 (See also section 8.5. PVC-induced cardiomyopathy.) Very rarely, idiopathic VCCs from the outflow channel can trigger malignant gastrointestinal tract in patients without structural heart disease. S2.2.2-25,S2.2.2-262.1.2. VT and VF during treatment with ACSApproximately in half of patients with out-of-hospital cardiac arrest whose first rhythm has been identified as VF and who survive hospital admissions show signs of acute cardiac infectator (AMI). S2.2.2-27 Of all outside the hospital >50% have significant coronary artery damage in acute coronary artery angiography. S2. Of the 2.2-27 patients treated with AMI, between 5% and 10% have VF or long VT prior to hospitalisation, and 5% have VF or long VT after hospital arrival, most within 48 hours In the study, patients with non-ST height ACS who underwent cardiac encatment within 48 hours were diagnosed with VT/ VF in 7.6% of patients, and 60% of these events within 48 hours Accelerated idioventricular rhythm is a common arrhythmia in patients with acute cardiac infection, including patients with ST segment height cardiac infection who undergo primary perkutan coronary artery disease (PCI). Accelerated idioventricular rhythm is more closely related to the expansion of the infarction than to the reperfusion itself. S2.2.2-29Sustained VA in acs insertion is more often polymorphic VT or VF than monomorphic VT. Risk factors for VT/ VF include previous hypertension, a history of myoestry, changes in the ST segment during inhibition and chronic obstructive pulmonary disease. S2.2.2-30 A nationwide Danish study found that 11.6% of patients with ST segment height mi who underwent PCI had VF prior to PCI, and that VF was associated with alcohol consumption, preinfarctiangina pectoris, frontal infarction location and complete coronary artery blockage during coronary artery angiography. S2. 2.2-31 In a given group of patients undergoing primary PCI in a clinical trial, 5.7% developed long-term VT or VF, and two-thirds of these events occurred before the end of catheterization and 90% within 48 hours of the procedure. VT or VF after primary PCI was associated with lower blood pressure, higher heart rate, poor coronary artery flow at the end of the procedure and incomplete resolution of st altitude. S2.2.2-32 Most importantly, and unlike some previous studies, VT or VF was at any time associated with a significantly higher risk of death within 90 days. Late VT or VF (after 48 hours of hospital presentation) was associated with a higher risk of death than early VT or VF (within 48 hours of hospital introduction). S2.2.2-332.1.3. Long-lasting VT and VF not associated with ACS diseases have an increased risk of developing long-lasting VT and VF diseases. Long-lasting VT, unrelated to ACS, is often monomorphic, since it is usually caused by scar-related reassing, but it can degenerate into VF. The risk and predictors of S2.2.2-34 VT in patients with structural heart disease depend on the type, severity and duration of heart disease, which increases due to the severity of ventricular dysfunction and the presence of symptomatic HF. Polymorphic VT and VF, which occur without structural heart disease, are rare and may be caused by cardiac channelopathy, S2.2.2-35, S2.2.2-36 long QT syndrome caused by medication, S2. 2. S2.2.2-37,S2.2.2-382.2. Sudden cardiac death2.2.1. The prevalence of SCDSCA and its most common consequence, SCD, are major public health problems, accounting for about 50% of all cardiovascular deaths, S2.2.2-1,S2.2.2-39 and at least 25% is the first symptomatic heart In addition, analyses of the magnitude of SCD are limited, partly due to a wide range of risk assessments based on different epidemiological methods. S2.2.2-42 ScD has accounted for approximately 230,000 to 350,000 deaths per year in the United States in the last 20 to 30 years, and <170,000 to=>it ranges from 450,000, depending on epidemiological methods, data sources and inclusion criteria. S2.2.2-41,S2.2.2-43 The smallest of these extremes came from the national extrapolation of data from certain local programs, while the highest figures included non-carnage-related causes of non-poking death, such as pulmonary embolic or intracranial bleeding. The mid-range figures were largely based on death certificate studies, which required a code that includes sechemistry heart disease. An update of AHA cardiovascular statistics in 2017 estimated the total annual cardiac arrest rate outside the hospital at 356,500.S2.2.2-44 In addition, 209,000 cardiac arrests occur annually at the hospital. Among the cardiac arrest team outside the S2.2.2-45 Hospital, some 357,000 events trigger an emergency rescue response, with 97 percent of them occurring among adults >18 years old. The survival statistics for cardiac arrest left outside the hospital remain disappointing, with an estimated overall survival of 10%. S2.2.2-44 Among the subgroups that account for 70% of out-of-hospital cardiac arrest at home, survival is 6%. The best-reported results are from places with advanced and publicly visible emergency rescue, as well as a combination of cardiac arrest public location, bystander witnesses willing to give CPR, first responders arriving quickly, from a shocking rhythm in first contact, the availability of automated external defibrillators, and possibly benefit from telecommunications-driven resuscitation. S2.2.2-46,S2.2.2-47 After the discharge of the hospital, survival in the hospital is estimated to be 24%. S2.2.2-48 In all circumstances, survival statistics appear to be better when the rhythm recorded by respondents is shock (VF, pulseless VT) compared to pulse-free electrical activity or asystole. S2.2.2-49 Although the apparent increase in pulse-free electrical activity or asystolen prevalence may be due to the subsequent arrival of medical care, the reduction in shock rhythm has also been partly due to the diagnosis and treatment of structural heart disease. S2.2.2-402.2.2. Population subgroups and risk prediction Risk forecast for SCA and SCD is complex. The risk analysis is divided into two general categories: population risk prediction and individual risk prediction. S2.2.2-41,S2.2.2-50 Conventional epidemiological markers give an idea of the probabilities of sechemistry development in the general test subject category, but adequately tested and validated profiles for individuals' SCA risk stratifiers </170 000> 000> there is currently no population. In individuals defining the SCA risk, the challenge is due to a demographic model characterized by a large number of events diluted to a very large denominator (Figure 1). The entire population can be grouped into groups based on age, occurrence and disease scale integration, as well as the identification of small and risky subgroups of the large nominating population. Increasing age strongly predicts the risk of SCA, but it is not linear. Over time, the population risk has been estimated at 35-year-olds per 1,000 inhabitants per year, which increases to <1000 at= the= younger= end= of= that= spectrum= to= a= higher= risk= in= the= elderly.s2.2.2-41 = however,= an= analysis= of= lifetime= risk= of= scd,= derived= from= the= framingham= data,= suggested= that= the= incidence= of= scd= decreases= in= later= years,= especially= in= people=>75 years of age. S2.2.2-51 The data also suggested that SCD is uniformly more common among men than women in all age groups. By contrast, the total annual risk for the population of children, adolescents and young adults is 1/100,000, and the risk of SCD is somewhat higher in younger contexts in this age range. S2.2.2-41 The age-related transition interval between the ages of 20 and 35-40 is characterised by a sharp increase in the risk of a group of young people in the middle-aged group, corresponding to the emergence of sechemical heart disease. Although ischemical heart disease remains the most common scd-related substrate, the intrenity of sepelm-related heart disease-related SCD appears to be decreasing, S2. 2.2-52, where myocardium fibrosis and LV hypertrophy are associated with different forms of cardiomyopathy. S2.2.2-53 In addition, over time there has been a trend that clinical profiles of cardiac arrest patients living outside the hospital are increasingly likely to be at risk than emerging diseases. S2.2.2-54 The younger population – children, adolescents and young adults – is affected by a number of disorders that occurred earlier in life, such as genetic structural disorders and cardiac channelopathies, myocarditis, congenital heart disease and other rare disorders. S2.2.2-43 During the transition period from the mid-20s to the mid-30s, the causes of SCA and SCD include a lower proportion of hereditary diseases and a growing proportion of sechemical heart disease (>40% of cases). S2.2.2-43Spoistating the small progress made in SCA and SCD risk forecasts, the biggest challenge is to identify relatively small, high-risk subgroups hidden in large populations that do not have an identified disease but are at risk of SCA as the first cardiac event (Figure 1). S2.2.2-50Figure 1A. SCD prevalence and overall events. S2.2.2-1 EF means an ejection fraction; and </1000>Sudden cardiac death. Figure 1B. SCD and clinical sub-series. S2.2.2-1 SCD refers to sudden cardiac death.3. Mechanisms of VA3.1 Cellular mechanisms and substratesA mechanisms Include improved normal automaticity, abnormal automaticity, activity and rebirth caused by early or late surgery. S3.4-1–S3.4-3 Reentry requires a trigger to trigger the arrhythmia and maintain a substrate to maintain it. The trigger can be PVC, which can be caused by automaticity. The substrate can be a structural overhaul that is secondary to the underlying disease process and often contains a scar secondary to previous heart or surgical repair, or in the setting of scattered fibrosis cardiomyopathy or hypertrophy. Changes in the switch between the ion channel or transfer function and/or the expression and cell and cell and cell in the secondary to the underlying pathology may alter the initiation or spread of the heart's action potential. Electrophysiological substrate is dynamically influenced by many factors, such as cardiac metabolism, electrolytes, signalling pathways and autonomous effects. Subjugation of the his-Purkinje system or ventricular heart cardirium may cause improved automaticity or abnormal automaticity causing VA. AutomaticityNormal automaticity is due to spontaneous depolarisation of the transmembrane action potential of phase 4 due to normal rest potential, threshold achievement and determination of functional potential. S3.4-1,S3.4-3 Alullepanitive current (If) is responsible for spontaneous phase 4 depolarization in the sinus node. The speed is determined by the integration of the greatest diastovic potential at the end of repolarization, the slope and threshold potential of phase 4 depolarization. In contrast, abnormal automaticity is partly due to depolarized membrane potential, which is usually close to the activation potential of calcium channels in the cell membrane. S3.4-1,S3.4-3 In the acute phase of myocardial infarction or during transient ischaemia, elevated extra cell potassium causes partial depolarisation of the potential of the resting membrane, causing an injury flow between infarction/cardiac acardium. These injury flows can trigger spontaneous action. In ischemia, abnormal automaticity can occur in both ventricular myocytes and Purkinje fibres, and can also improve the normal automaticity of canned fibres in the ischemical zone.3.3. Triggered activityEarly afterdepolarizations occurs late stage 2 or early 3 with operational potential, S3.4-3-S3.4-5 usually setting the operating potential extension due to increased inward flow (late sodium flow, inward calcium flow or sodium calcium AC) or decrease potassium flows. Under these conditions, early dedepolarisations may be initiated when the inward L-type calcium channel is reactivated. Again. before the membrane has returned to negative potential as required for the reactivation of the calcium channel. Spontaneous calcium release from sarcoplasmic retica may also lead to the activation of depolarizing sodium/ calcium ac power. Early death reproduction triggers VT indicators associated with QT extension, either by drugs or other acquired factors or by mutations in the ion channels that caused long QT syndrome. In these cases, it is possible that the early afterdepolarization/triggered activity sequence is a trigger that culminates in polymorphic VT/VF. Delayed postoperation occurs after complete membrane repolarisation and develops under conditions of in-cell calcium overload. Factors affecting elevated in-cell calcium load include tachycardia, calycolamines, hypocaemia, digoxin toxicity, cardiac hypertrophy and HF. S3. 4-6, S3. Elevated sarcoplasm calcium levels or increased sensitivity of the ryanodine receptor may trigger spontaneous calcium release, activating a transient inward current based primarily on sodium and calcium AC. If the depolarisation of the membrane is large enough, the inward sodium flow is activated, resulting in triggering action potential. Delayed afterdepolarizations are the mechanism behind VT for the setting of digoxin toxicity, caecolamine-gic polymorphic VT and idiopathic outflow channel VA. Delayed dedupolarizations are also considered an important trigger for VA in setting HF. S3.4-3,S3.4-8,S3.4-93.4. ReentryReentry is the basic mechanism for the most sustainable VA disease in the presence of structural heart disease. S3.4-1–S3.4-3,S3.4-10–S3.4-12 A re-attempt may occur around a fixed anatomical barrier such as heartbreak or surgically repaired congenital heart disease. Here, the arousing gap in the regulation is distinguished by aroused wave front from its fire resistance. The existence of structural reentrant substrates provides justification for VT ablation in scar-related VTs. S3.4-11,S3.4-12Funcal retraining around functional block areas without anatomical barriers can also occur. Two main models of functional reinhab have been proposed. S3.4-2,S3.4-3 The leading circle model has a functionally refractory core and no a si000 aperture. The re-attempt at the spiral wave is controlled by a rotor with a curved wavelength and wave turn around a rousing but uninspiratored core. There is still a lot of discussion about the exact mechanisms of VF (rotor vs. multiple wavelength re-attempt). Both mechanisms may be operational at different stages of VF. S3.4-10Phase 2 re-attempt may occur the heterogeneity of ventricular repolarization. Electrotinic flow currents can flow from endocardial objects with longer operational potential to a shorter duration of epicardium action potential, which can lead to re-implantation once these areas have recovered from fire resistance. This is believed to be one possible VT/ VF mechanism in Brugida syndromeS3. General assessment of patients with documented or suspected VA4.1. History and physical researchSynopsisVA can produce a wide range of symptoms, and the severity of the symptoms may not reflect the extent of the structural heart disease or the potential risk of SCD. Symptoms of VA include palpitations, either missed or excess beats or persistent palpitations, shortness of breath, chest pain, dizziness, near fainting and fainting. S4.1-5,S4.1-6 Palpitations may correlate with VA, but it is often reported during normal rhythm. S4.1-7 Exercise tolerance, chest pain, shortness of breath, pre-synchronisation and fainting include VA, but also includes other ethicologies. However, more dramatic symptoms, especially in patients with known or observed structural or electrical heart disease, should lead to targeted research into a possible link to VA treatment (Table 6). Table 6. Important considerations for evaluating patients with known or suspected VAComponentAssessment and findings relevant to va and/ or scd risk history1. Symptoms/events associated with arrhythmia: Palpitations, fainting, fainting, fainting, shortness of breath, chest pain, cardiac arrest2. Symptoms associated with underlying heart disease: Shortness of breath at rest or during exertion, orthopaedics, paroxysmal nocturnal shortness of breath, chest pain, swelling3. Sasing factors: Exercise, emotional stress4. Known heart disease: Coronary artery disease, valvulaarin (e.g. mitral valve prolapse), congenital heart disease, others5. Risk factors for heart disease: hypertension, diabetes mellitus, hyperlipidemia and smoking6. Medicines Antiarrhythmics Other drugs with the potential for QT-time extension and vääntimines de points Medications That can cause or exacerbate VA stimulants, including cocaine and amphetamine Supplements including anabolic steroids Medication-medication interaction that can cause QT to lengthen and torsades de pointes7. Previous medical histories Thyroid disease Acute kidney damage, chronic kidney disease or electrolyte abnormalities Stroke or embolic events Lung disease Epilepsy (arrhythmia fainting can be misdiagnosed as epilepsy) Use of alcohol or illegal drugs Over-medication use that can cause QT time to be extended and torsion de pointes Unexplained motor vehicle overturns Family history1. SCD, SCA or drowning in a first-degree relative2. SIDS or recurrent spontaneous pregnancy losses, as they may channelopathies3. Heart disease IHD Cardiomyopathy: Hypertrophic, dila enlarged, ARVC Congenital heart disease Cardiac channelopathies: Long QT, Brugada, Short QT, CPVT Arrhythmias Lead disorders, pacemaker/ ICD 4. Neuromuscular disease associated with cardiomyopathy Muscular dystrophy5. EpilepsyExamination1. Heart rate and regularity, blood pressure2. Carotid artery venin pressure3. Growe4. Pulses and bruits5. Swelling 6. Hemortage scars Unsolved of saostam factors such as stress or emotional stress, concomitant medications or diseases, and alleviation factors is important. The presence of SCD, ischemistry heart disease, controlled heart disease, nonchemical cardiomyopathy (NICM) or HF raises concerns about any of these VA-related disorders. Getting a complete history of medication is important. Various antiarrhythmia drugs and other medicines may cause QT extensions and torsion (www.crediblemeds.org)S4.1-8; some drugs can also cause Brugada type I electrocardiogram pattern and VF (www.brugadadrugs.org). S4.1-9,S4.1-10 Patients with bigeminy and trigeminy can cause effective bradykardia, apical-radial pulse deficit and relative hypertension with extensive pulse pressure. Effective bradykardia of PVC can lead to an inaccurate assessment of the heart rate. Although premature beatings can be observed in the auscultation of the heart, physical research focuses largely on finding evidence of structural heart disease. Carotid artery brutes or reduced peripheral pulses may be indicators of ischemic atherosclerotic disease. Carotid artery enlargement, rales, gallops and peripheral oedema provide evidence of HF. Midsystolic clicking may refer to the prolapse of the mitral valve, which can be connected to the VA. S4.1-11–S4.1-13 Many VA's are asymptomatic and are detected only in ECG or telemetry. Such cases highlight the need to look for evidence of underlying heart disease. Support text per recommendation1. A synchrotist or SCA may be the first manifestation of structural or electrical heart disease, S4.1-14, and some SCA victims have previous periods of sentinel fainting. S4.1-15 Syncope or its early warning of dizziness, shortness or near syncing can be a risk factor for SCA and SCD. S4.1-2 The initial assessment focuses at any age on the detection or exclusion of heart disease. Fainting during exercise should lead to a thorough assessment to absent heart problems. Heart evaluation naatine monitoring and exercise testing may be justified according to the clinical data provided. S4.1-3,S4.1-4 Synkope heart conditions include continuous VT, high-level atrioventricular lobes or severe sinus bradycardia or prolonged sinus diseases, supraventricular tachycardia (SVT), pacestine dysfunction, cardiac channelopathies or heart disease symptomatics such as hypertrophic cardiomyopathy (HCM) or congenital heart disease. S4.1-3, S4.1-4, S4.1-16 Cardiac channelopathies and HCM are especially important to consider in young and young adults. Synkope arrhythmias are often associated with very short symptoms of hunching or palpitations and known existing existing friendships, especially low LVEF or HF. S4. For 1-1 cardiac rhythmless reasons, account should be taken of the heart muscle crisis, severe aortic htauma, HCM, HF and prosthetic valve malfunctions, pulmonary embolism, medicines and illicit drug use. S4.1-34.2. Non-invasive assessment4.2.1. 12-lead ECG and exercise testingRecommendation specific support text1. A 12-lead ECG during tachycardia is the first diagnostic test to be carried out on any patient found to be in stable wide QRS complex tachycardia monitor. VT is a diagnosis in most adults with extensive complex tachycardia and underlying structural heart disease. S4.2.1-3 VT is based on AV dissociation, QRS complex >0,14 s, single-phase R wave in aVR, certain QRS morphologies (e.g. positively or negatively capacitant QRS complexes in pre-condensation wires), absence of RS complex in all prexordial wires, and RS spam >100 ms in at least one precondensial cable. S4.2.1-2 Exceptions occur especially in patients with advanced heart disease who take certain antiarrhythmics. S4.2.1-1 In patients with a history of a bundle branch block, comparing QRS morphology during ponyery to extensive complex tachycardia is often relevant.2.Due to stress-related arrhythmias, exercise in the controlled environment can reproduce symptoms and/or related arrhythmias, allowing diagnosis. Exercise testing is especially important when caecolimin polymorphic ventricular tachycardia is possible. However, stress-related symptoms and findings may not be reliably reproducible through exercise testing, and long-term cardiac electrocardiogram monitoring of external or implantable recorders may be necessary.3.A A 12-lead ECG may indicate the presence of structural heart disease such as prior cardiac repair or ventricular enlargement, increasing the likelihood that the patient's symptoms may be caused by VA, or may provide evidence for documented VA. The ECG can also reveal hereditary arrhythmias such as long QT syndrome, Brugida syndrome and correct ventricular cardiomyopathy. In patients with structural heart disease, QRS duration and leadership disorders provide predicted data. S4.2.1-7-S4.2.1-14 Data on the use of microvolt T wave current locks and signal average ECG are not convincing as such tests are not routinely used in clinical practicesS4.2.1-15-S4.2.1-19; the only exception is the possible use of mean ecg signal in patients with arrhythmia-genic correct ventricular cardiomyopathy (see section 7.3).4. Ambulatory electrocardiogram Recommendation-specific support text1. Ambulatory electrocardiography monitoring is often used to assess the effectiveness of arrhythmia suppression treatments, but more robust information is needed on the clinical use of this practice. Continuous or intermittent ambulatory electrocardiogram recording with a Holter monitor or events recorder helps diagnose suspected arrhythmias, their frequency, attach them to symptoms and assess the response to treatment. Although the return on these tests is relatively low, VT is sometimes documented. S4.2.2-4 24-hour continuous Holter recording is appropriate when symptoms occur at least once a day or when it comes to defining PVC/NSVT to evaluate the function of any depressed chamber associated with the VA. For occasional symptoms, event or loop displays are more appropriate because they can be activated for long periods of time and increase diagnostic yield. S4.2.2-2,S4.2.2-3 Glue patch electrocardiogram monitors can be stored for weeks and allow continuous short-term 1-lead monitoring and patient activation due to symptoms. Studies have shown that the patient is in satisfactory compliance and that arrhythmias are detected; However, arrhythmias detected by some monitors will not be discovered until the patch is returned for analysis. S4.2.2-1,S4.2.2-4 Serial assessments with exercise testing and/or 24-hour therapy monitoring are also used to assess the rhythm and VAS response. In particular, the monitors to be implanted are discussed in section 4.2.3. Importantly, when a patient's VA suspicion is high, monitoring outpatient care is not appropriate, since rapid diagnosis and va prevention are justified. It is important to accurately correlate the symptoms with arrhythmias detected by ampbulatory ECG monitoring.4.2.3. Implanted cardiac monitorsSysensitical supporting texts1. Implanted heart monitors provide continuous rhythm monitoring and recorded electrogram recordings based on patient activation or preset parameter, allowing for a few years of long follow-up period. These devices require a small invasive procedure with local anaesthesia for implantation. In patients with occasional symptoms, including fainting, the implantable recorders are severe takyarrhymia (including They are usually reserved for patients who do not carry out other ambulatory monitoring due to the rareness of events. The additional yield of 25% of the diagnosis is described after an unpublished external ambulatory monitor. S4.2.3-5 In a study in synchropic patients, the diagnostic yield of the monitor implanted was higher than conventional testing through external monitoring, tilt table testing and electrophysiological examination. S4.2.3-2 A systematic review of synchropic patients concluded that the use of these devices provides a greater diagnosis and trend to reduce relapse after diagnosis compared to conventional treatment. S4. 2.3-3 Prospective study of patients after study support, LVEF <40%, demonstrated= nsvt= (=>16 wins long) in 13%, VT (>30 s) in 3% and VF in 3% of patients. S4.2.3-1 It is important to correlate the symptoms accurately with arrhythmias detected by implanted heart monitors.4.2.4. Non-invasive heart description Recommendation-specific support text1. Assessment of global and regional myocardial function, controlled structure and function, and assessment of congenital heart disease in adults is necessary in patients at high risk of heart or second-second renal disease, including cardiomyopathy patients, HF, previous myocardial infarction, cardiomyopathy or SCD or hereditary structural heart disease. Echocardiography is an easily accessible and most commonly used imaging technology. S4.2.4-1, S4.2.4-2 LVEF is a strong, independent predictor of SCD and cardiovascular mortality and determinant of the eligibility of ICD implantation in the primary prevention of SCD. S4. 2.4-1 SCD-HeFT (sudden death from heart failure) S4. 2. In clinical practice, echocardiography may be unnecessary if cardiac CTS4. This imaging recommendation differs from the 2017 ACC/AHA/HRS fainting guidelineS4.2.4-4, which applies to patients who may not have VA.2.VA or SCA, may have the first manifestation of ischemia heart disease, cardiomyopathic processes or heart clotting. Cardiac CT and cardiac MRI scans allow for the assessment of structural heart disease and the assessment of the function of LV and the motorhome, including the mass and volume of LVEF, LV, the quantification of the controlled structure and coronary anatomy, including abnormal coronary artery origin. An MRI scan of the heart can be useful in assessing myocardia and infiltration processes, which manifests itself as a late addition to gadolinium. S4.2.4-5-S4.2.4-9 Cardiac MRI also provides a high-quality assessment of the function, size and fibrosis of LV and campervan, and is especially useful in arrhythmiagenic right ventricular cardiomyopathy and HCM.4.2.5. </40%,>Support text1.Elevated levels of natriuretic peptides – type B natriuretic peptide (BNP) or N-terminal BNP – are associated with an increased risk of SCA and appropriate ICDs even after adjustment of LVEF and other risk factors. S4.2.5-1-S4.2.5-4 These biomarkers also predict non-udd cardiovascular mortality and are therefore not specific to scd risk alone. Natriuretic peptides have also been evaluated to predict SCD in the general population. S4.2.5-5,S4.2.5-6 In the Nurses' Health Survey, the elevated N-terminal pro-BNP was an independent risk sign for SCD in supposedly healthy women. S4. 2.5-5 In the older adult population, SCD was associated with higher baseline levels from terminal N against BNP over a 16-year follow-up period. S4.2.5-6 These biomarkers may also play a potential role in facilitating the identification of individuals at increased risk of SCD and VA across the population, especially those at medium or high risk of developing sechemical heart disease, but further research is needed. Biomarkers have not been shown to be useful in selecting patients for ICD. In a study in which 4,431 patients found high-susceptible troponin predicted only poorly for a second cost. However, S4.2.5-7 There is no information on whether high-sensitivity troponin can improve existing SCD forecast algorithms.4.2.6. Genetic considerations in arrhythmia syndromesSynopsis The diagnosis of most hereditary arrhythmia syndromes is based on clinical characteristics and family history. The availability of genetic testing of inherited arrhythmia syndromes may: 1) provide an opportunity to confirm a suspected clinical diagnosis and sometimes provide proselytized information to probandi, and (2) provide cascading screening for potentially affected family members when a disease-causing mutation is identified in the proband. The yield of genetic testing varies depending on the diseases. The verification of the pathogenicity of suspected mutations is an evolving field, and the sequencing of exosoms has detected an increasing number of variants of uncertain significance in the population. S4.2.6-1-S4.2.6-5 Genotyping may have a therapeutic effect on some arrhythmia gene phenotypes, as for long periods of QT syndrome and Fabry disease, S4.2.6-S4.2.6-9, where a clearly identified monogenic pathogenic mutation has been identified, the mutation has been extensively investigated and effective treatment relevant to the mutation can be introduced. In other diseases, such as Brugida syndrome, the role of a mutation causing a clear monogenic disease is less certain, and the genotype does not provide therapeutic or predictive information to probandi. S4.2.6-5,S4.2.6-10-S4.2.6-12 Arrhythmiagenic right ventricular cardiomyopathy for some desmosomal mutation positive individuals does not develop diseases, which that additional mutations and environmental interactions are likely to have an impact on the clinical development of the disease. S4.2.6-13-S4.2.6-16 It is important that the absence of a genetic mutation causing the identifiable disease does not exclude the presence of the disease, so continuous monitoring and decision-making is made on the basis of clinical phenova. Genotyping is often most useful when a pathogenic mutation is identified in the proband, allowing screening to be applied to relatives who are in the preclinical phase, where positive gene mutation can be monitored through lifestyle changes, therapy or continuous monitoring. S4.2.6-7 See section 7. In young patients (<40 years of age) without structural heart disease with unexplained cardiac arrest, unexplained near drowning or repeated stress synclops, genetic testing may be important to identify hereditary arrhythmia syndrome as a probable cause. S4.2.6-17–S4.2.6-23Sextive support text1. The decision to continue genetic testing requires discussion on the clinical use of genetic information for both proband and family members, as well as taking into account important psychological, financial, employment, disability and life insurance effects of the use of positive genotypes. S4.2.6-17,S4.2.6-18,S4.2.6-20,S4.2.6-24 Balance the privacy of proband health care information with the right of family members to be informed, and the ability to provide appropriate data transfer to all potential family members can be challenging on many levels, including family dynamics, geographical proximity and access to healthcare. S4.2.6-25 For these reasons, genetic counselling usually takes place before the continuation of genetic testing, and from the patient's point of view, genetic counsellors, if available, are optimally available in cooperation with doctors. S4.2.6-26,S4.2.6-27 The combination of genetic counselling and medical guidance can properly balance the decision on whether genetic testing would be beneficial individually.4.3. Invasive testing4.3.1. Invasive heart description: Heart encapsulation or CT angiographyAd-specific support text1. Although randomized studies are not available, coronary angiography plays an important role in determining or excluding the occurrence of significant obstructive isctroke heart disease in patients with SCA or in patients with life-threatening va. S4.3.1-1–S4.3.1-4 Repeated polymorphic VT or VF may be caused by persistent myocardial ischemia, which was corrected by coronary revascularisation. ST elevation in preresusation or early post-resuscitation ECG indicates ischemia and possible ACS, justifying urgent angiography and revascularization. The ST height of S4.3.1-5 may also be due to coronary artery spasms or DC shocks. Absence of ST height after cardiac arrest does not rule out coronary artery damage. A coronary angiogram may not be justified if a non-ischemismal cause of sca has been established. Coronary artery disease and CT angiography also play an important role, with the exception of the abnormal origin of the coronary arteries, which can cause SCD.4.3.2. Electrophysiological examination for VasynopsisElectrophysiological can be used to induce long-lasting VA in patients known or suspected of having va. Due to the product of ICD and its proven benefit, there are fewer indications in primary and secondary scd prevention to stimulate programmed stimulation. Patients with HF and LVEF ≤35% usually have an indication of ICD and specific induction of VT/ VF prior to implantation. Patients with LVEF >35% and unexplained fainting or near fainting may benefit from an electrophysiological study to determine whether VT/ VF is the cause of the symptoms and to direct further treatment. Induction of VT/VF is often attempted prior to a catheter ablation of the arrhythmia substrate to control the procedure and determine the success of the procedure after ablation. Electrophysiological research can be used to determine an extensive complex mechanism of takycardia. Recommendations for electrophysiological research for certain disease states are set out in points 7.3, 7.4, 7.6, 7.9.1.3 and 10.8. Support text per recommendation1. S4. In a prospective cohort of patients with 2-17,180 ischemia or NICM and fainting, induction of VT or VF in an electrophysiological study correlates with heart mortality only in patients with ischemical heart disease. In NICM patients, heart mortality correlates with LVEF, but not with the inductiveness of electrophysiological research. S4.3.2-182.In that meet the criteria for ICD implantation (i.e. ICD implantation). HF and LVEF ≤35%), the data do not support the routine use of electrophysiological studies only through risk partitioning, as such patients have been shown to benefit from survival from ICD. However, S4.3.2-8-S4.3.2-11 Electrophysiological examination may be useful in selected patients suspected of having preventive or supraventricular arrhythmias caused by symptoms or extensive complex tachycardia patients requiring final diagnosis and treatment. SVT leading to VT/ VF or abnormally performed SVT may also be suspected in younger or patients with surviving LVEF. Induction of SVT and ablation can then be healing, and no ICD is required. In such cases, VT/VF cannot be achieved after the removal of the SVT substrate.3.Channelopathy risk stratation is usually based on symptoms, EKG,S4.3.2-13,S4.3.2-19–S4.3.2-24 exercise running mating, S4.3.2-25–S4.3.2-27 and during genetic testing. S4.3.2-28-S4.3.2-32 Electrophysiological examination (i.e. programmed ventricular stimulation) has no predictable risk for stratification in patients with these cardiac channelopathy. S4.3.2-12–S4.3.2-155. Treatments for the treatment or prevention of VA5.1. Medication Treatment With the exception of beta-blockers (e. g. metoprolol succinate, veistonilol), there is no evidence from RCT that antiarrhythmia drugs for VA improve survival when administered for primary or secondary prevention of one second. However, the use of these drugs is necessary in some patients to control arrhythmias and improve symptoms. The use of medication for va is discussed, and any recommendations are listed in the following sections. In addition, proarrhymia caused by medication is discussed in section 10.7.Antiarrhythmia drugs are often classified according to Vaughan Williams' 4-degree structure (Class I: rapid sodium channel blockers; Class II: beta-blockers; Class III: potassium flow inhibitors; Class IV: nondihydropyridines calcium channel inhibitors). S5.1.5.2-1 This system does not deal with the complexity of antiarrhythmic drugs, since almost every substance has several effects. Table 7 shows the uses of antiarrhythmics, electrophysiological effects, pharmacological effects and general adverse reactions. Table 7. Pharmacological properties of available antiarrhythmic medicinal products VAAntiarrhythmic Medication (class) and doseUs va/SCATargetElectrophysiological EffectsPharmacological CharacteristicsCommon Adverse reactionsAcebutololPO 200-1200 mg daily or up to 600 mg bidVT, PVCsBeta 1, Mild Inherent Sympostomytic ActivityIncrease Rate DecelerationAV Nodal Refractory Increased Active Metabolite t1/ 2:8–13 hpProlonged with Renal Impairment)Metab: HExcr: F 60%, U 40%Heart: Bradycardia, Hypotension, HF, AVBTother: Dizziness, fatigue, anxiety, impotence, hyper/ hypoesesthesisAmiodaron (III)IV: 300 mg bolus VF / pulseless VT arrest; 150 mg bolus for stable VT; 1 mg/min x 6 h, then 0,5 mg/min x 18 hPO: 400 mg* q 8-12 h for 1-2 wk, then 300-400 mg daily; reduce the dose to 200 mg daily if possibleVT, VF, PVC, INa, ICa, IKr, IK1, IKs, Ito, Beta receptor, Alpha receptor core T3reseptorSinus degree slowed downQRS prolongedQTc prolongedAV-nodal refractory refractory resistance; increased DFTT1/2: 26-107 dMetab: HExcr: FCardiac: Hypotension, bradycardia, AVB, TdP, slows VT below programmed ICD detection rate, increases defibrillation thresholdInformation: Corneal microdepostits, thyroid abnormalities, attic, nausea, vomiting, constipation, photosensitivity, skin discoloration, attax, dizziness, peripheral neuropathy, tremors, hepatitis, cirrhosis, pulmonary fibrosis or pneumonitisAtenolol (II)PO: 25-100 mg qd or PVC, ARVC, LQTSBeta 1Sinus rate slowedAV nodal refractoriness increasedt1/2: 6–7 h (pitkittynyt munuaisten vajaatoimintaan) Metab: Metab: 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 4.5–12 h, extended release 12 h, and severe hepatic impairment 14–16 hMetab: HExcr : UCardiac: Hypotension, swelling, HF, AVB, bradykardia, worsening of HFrEFOther: headache, rash, constipationEsmolol (II)IV: 0,5 mg/kg bolus, 0,05 mg/kg/minVTBeta 1 receptorSinus rate decelerationAV nodal fire resistance increased by 1/2:9 minMetab: RBC esterasesExcr: UCardiac: Bradykardia, hypotension, HF, AVBTother: Dizziness, nauseaFlecainide (IC) PO: 50-200 mg q 12 hVT, PVC (without structural heart disease). Plays a role in treating patients with CPVTINA, IKr, IKurPR prolongedQRS prolonged; increased DFTt1/2: 7-22 hMetab: HExcr: UCardiac: Sinus node malfunction, AVB, drug-induced Brugada syndrome, monomorphic intravenous throlysis in patients with myocardia anthrope, exacerbation of HFrEFOther: dizziness, tremors, visual disturbances, shortness of breath, nauseaLidocain (IB) IV: 1 mg per bolus kilogram, 1-3 mg/ min1 to 1.5 mg/ kg. Repeat 0,5 to 0,75 mg/kg of bolus for 5 to 10 minutes 10 minutes between 0,5 and 0,75 mg/kg (maximum cumulative dose 3 mg/kg). Maintenance infusion is 1-4 mg/ min, although it can be started at a dose of 0.5 mg/ minVT, VFINaNo has a significant effect on most intervals; QTc may slightly shorteninitial t1/2 7–30 min; terminal 90–120 min. Long-term HF, liver disease, shock, severe kidney disease Metab: HExcr: UCardiac: Bradykardia, hemodynamic collapse, AVB, sinus arrestToi: Delirium, psychosis, seizure, nausea, tinnitus, shortness of breath, bronchospasmMetoprolol (II)IV: 5 mg q 5 min up to 3 dosesPO: 25-100 mg Prolonged release qd or q 12 hp, PVCBeta 1 receptorSinus speed slowed downAV nodal fire resistance increased1/2:3-4 hMetab: HExcr: UCard bradycardia, hypotension, AVBTother: Dizziness, fatigue, diarrhoea depression, shortness of breathMexetin (IB)PO: 150-300 mg q 8 h or q 12 hT, VF, PVC, affects patients with AQT3INaNo a significant impact on most intervals; QTc may slightly shorten1/2: 10-14 hMetab: HExcr: UCardiac: HF, AVBTother: Lightheaded, tremor, attax, paresques, nausea, blood dyscrasiasNadolol (II)PO: mg dailyVT, PVC, LQTS, CPVTBeta 1 and 2 receptorsSinus rate slowedav nodal fire resistance increased1/2:20-24 hMetab: noneExcr: UCardiac: UCardiac: UCardiac: hypotension, HF, AVBTother: Swelling, dizziness, cold limbs, bronchospasmProkainamide (IA)IV: saturation dose 10-17 mg/kg at 20-50 mg/minIncreasing dose: 1-4 mg/minPO (SR preparation): 500-1250 mg q 6 hVTINa, IKrQRS prolongedQTced; increased DFTMetab: Ht1/2: 2-5 h; NAPA 6-8 ht1/2 prolonged treatment disorders. Anephric: proc 11 h and NAPA 42 hExcr: UCardiac: TdP; AVB, hypotension and worsening of HFrEFOther: Lupus symptoms, Diarrhoea, nausea, blood dyscrasiasPropafenone (IC)PO: Immediate release 150-300 mg q 8 hExtended release 225-425 mg q 12 hVT, PVC (without structural heart disease)INa, IKr, IKur, Beta receptor, AlfareceptoriPR prolongedQRS prolongedQRS prolongedQRS prolonged increased DFTt1/2: 2-10 h or 10-32 ht1/2: extensive metabolisers 2-10 h; poor metabolisers 10-32 h Metab: HExcr: UCardiac: HF, AVB, drug-induced Brugada syndrome Other: dizziness, fatigue, nausea, diarrhoea, electroscostomy, tremor, blurred visionPropranolol (II)IV: 1-3 mg q 5 min total 5 mgPO: Immediate release 10-40 mg q 6 h; Extended release 60-160 mg q 12 hp, PVC, LQTSBeta 1 and 2 receptors, INaSinus speed slowedAV nodal fire resistance increased1/2: Immediate release 3-6 hExtended release 8-10 hMetab: HExcr: UCardiac: Bradykardia, hypotension, HF, AVBOther: Sleep disorder, dizziness, nightmares, hyperglycaemia, Diarrhoea, bronchospasmiQuine (IA)PO: sulphate salt 200-600 mg q 6 h –q 12 hglukonate salt 324-648 mg q 8 h –q 12 hIV: saturation dose: 800 mg in 50 ml infused at 50 mg/ mint, VF (including short QT syndrome, Brugada)INa, Ito, IKr, M, Alpha receptorQRS prolongedQTc; increased DFT1/ 2:6-8 h longer in HF, cirrhosis of the liver and older ageMetab: HExcr: UCardiac: Syncope, TdP, AVBTother: Dizziness, diarrhoea, nausea, oesophageal inflammation, vomiting, tinnitus, blurred vision, rash, weakness, tremors; blood dyscrasiasRanolatsin (not classified)PO: 500-1000 mg q 12 hVTINa, IKrSinus speed slowed downTc prolonged by 1/2: 7 hMetab: HExcr: U 75%, F 25%Heart: Bradykardia, hypotensionToi: Headache, dizziness, fainting, nausea, shortness of breathSotalol (III)IV: 75 mg q 12 hPO: 80-120 mg q 12 h, may increase the dose by 3 d over a 12-hour 12-hour sinusation; max 320 mg/dVT, VF, PVCIKr, Beta 1 and 2 receptorSinus rate decelerationQTc prolongedAV nodal fire resistance increased; decreased DFTt1/2: 12 hMetab: noneExcr: UCardiac: Bradykardia, hypotension, HF, fainting, TdPTother: Fatigue, dizziness, weakness, shortness of breath, bronchitis, Depression, nausea, diarrhoeaVerapamil (IV)IV: 2.5-5 mg q 15-30 min Unsustainable release PO: 240-480 mg/dVT (especially RVOT, verapamil sensitivity i diodepathic LVT)ICa-LSinus rate decelerationPR prolongedAV-nodal conductivity slowed down by 1/2:3-7 hMetab: HExcr: UCardiac: Hypo attention, swelling, HF, AVB, bradycardia, worsening of HFrEFOther: rash, gingenic hyperplasia, constipation, dyspepsia5. Medicinal products with a significant sodium channel blockadeExcept in a specific sodium channel inhibitors (Vaughn-Williams Class I substances) are limited in VT/SCD prevention; This is based on a lack of survival benefit and an increase in mortality in heart disease with ischemia observed during chronic treatment (see section 10.7). The specific conditions under which sodium channel inhibitors have been used to treat VT/SCA are: intravenous lidococin for patients with refractory VT/ cardiac arrest (specially proven)S5.1.5.2-3; oral mexlentine with congenital long QT syndrome S5. 1. 5. kinidine in patients with Brugada syndrome; and flecainide in patients with caecolamine-rged polymorphic ventricular tachycardia. S5.1.5.2-5 These medicines may also be used in ICD patients with VT taking the medicine and ablation medicine. One new medication that can benefit is based on very limited data, is ranolatsin. This drug, developed and FDA-approved antianginal agent, provides a relatively specific late sodium channel flow blockade in addition to a less intense blockade of phase 3 repolarizing potassium flow; i.e. a rapidly delayed straightener potassium flow; IKr. The potential of the efficacy of clinical arrhythmia is supported by basic studies and experimental models. S5.1.5.2-6 Clinical data are limited. In a study of 12 patients, ranolazin reduced ICD shocks with otherwise drug-resistant VT/ VF in 11 patients. In S5.1.5.2-7 Merlin TIMI-36 (metabolic efficacy with ranolatsine less ischemic in the thrombolysis of non-ST elevation acute coronary syndromes in myocardial infarction 36), ranolatin did not reduce SCD, but reduced VT in the first days after the elevation of the non-ST segment ACS. S5. 1. 5. With 2-8 1 RCT, high-risk ICD patients with iscino or NICM were randomly given ranolazin 1000 mg twice daily against placebo. S5.1.5.2-9 High risk was defined as: 1) primary prevention ICD without documented VT/VF and one of the following conditions: BUN ≥26 mg/dL, QRS >120 msec, atrial fibrillal fibrillace or NSVT or >500 VPBs for 24-hour Holter recording; (2) primary prevention ICD with previously documented VT/ VF, which has been properly treated with ICD or untreated NSVT; or (3) have secondary prevention of ICD documented VT/VF or cardiac arrest. Ranolazin did not significantly reduce the primary endpoint of VT/ VF requiring appropriate ICD or death. In a predetermined secondary analysis, ranolatin was associated with a significant reduction in VT events treated with takycardia pacemakers. S5.1.5.2-95.1.2. Beta-anta-tasalpaajat Beta-anta-tabers are often first-line antiarrhythmics because beta-estors are excellent due to their safety profile and effectiveness. and reducing the risk of SCD. S5.1.5.2-10,S5.1.5.2-11 Their antiarrhythmia effectiveness is associated with the effects of the adrenergic receptor blockade on sympathetically adapted trigger mechanisms, the sinus rate slows down, slows down, possible inhibition of calcium release of the ryanodine receptor. S5. 1. 5. 2-12Beta inhibitors reduce overall mortality and seconds in patients with reduced EF (HFrEF). S5.1.5.2-13-S5.1.5.2-15 Although beta-catchers have long been shown to reduce mortality based on MI,S5.1.5.2-16 registry data, confirm that early use of beta-predators in patients with cardiac infeaeto and risk factors for shock (>70 years, symptoms <12 hours= [st-elevation= mi= patients],= systolic= blood= pressure=></12> <120 mm= hg,= and= heart= rate=>110 beats/min during presentation) are associated with an increased risk of shock or death. S5.1.5.2-17 When setting polymorphic VT after the MI, beta-catchers reduce mortality. S5.1.5.2-18 Beta workshops dampen VA inhibitors in some patients with structurally normal hearts. S5.1.5.2-19 When beta-antalysists are used in combination with a membrane-stabilizing antiarrhythmic medicine, it can improve the effectiveness of antiarrhythmic drugs. S5.1.5.2- 20 Beta catches (e. g. nadolol, propranolol) are also first-line treatments for some cardiac channelopathies (e. g. long QT syndrome, caemorphine polymorphic polymorphic tachycardia).5. Amiodaron and SotalolAmiodaron have a wide range of activities, including a blockade of beta receptors and sodium, calcium and potassium flows (i.e. multichannel blocking). Its long-term overall impact on survival is controversial, and most studies do not show a clear advantage against placebo. A few studies and meta-analysis from several large studies have shown that, using Amiodarone, a decrease in mixing in patients with LV dysfunction due to a prior error and NICM, S5. 1. S5.1.5.2- 24 Secondary analysis of SCD-HeFT showed an increased risk of amiodaron mortality in patients with New York Heart Association (NYHA) Class III symptoms. S5.1.5.2-25 Systematic review of literature for at-risk patients <40%, with or without coronary disease), concluded that, for primary prevention, amiodarone, compared with no treatment or placebo, decreased the risk of SCD (Risk ratio: 0.76; 95% CI: 0.66–0.88) and all-quality of the supporting evidence was very low. S5.1.5.2-26 For secondary prevention of SCD, the same systematic review identified neither risk nor benefit with amiodarone. S5.1.5.2-26 Compared with beta-blocker therapy and other antiarrhythmic medications (including sotalol), amiodarone appears to reduce the risk of SCD and all-cause mortality. S5.1.5.2-26 Intravenous amiodarone has a role in reducing recurrent VF/VF during resuscitation. S5.1.5.2-3,S5.1.5.2-27–S5.1.5.2-29Chronic administration of amiodarone is associated with complex interactions and a host of adverse effects involving the lung, liver, thyroid, skin, and nervous system. As a general rule, the longer the therapy and the with= or= without= coronary= disease),= concluded= that,= for= primary= prevention,= amiodarone,= compared= with= no= treatment= or= placebo,= decreased= the= risk= of= scd= (risk= ratio:= 0.76;= 95%= ci:= 0.66–0.88)= and=

all-cause= mortality= (risk= ratio:= 0.88;= 95%= ci:= 0.78–1.00),= but= the= quality= of= the= supporting= evidence= was= very= low.s5.1.5.2-26= for= secondary= prevention= of= scd,= the= same= systematic= review= identified= neither= risk= nor= benefit= with= amiodarone.s5.1.5.2-26= compared= with= beta-blocker= therapy= and= other= antiarrhythmic= medications= (including= sotalol),= amiodarone= appears= to= reduce= the= risk= of= scd= and= all-cause= mortality.s5.1.5.2-26= intravenous= amiodarone= has= a= role= in= reducing= recurrent= vf/vf= during= resuscitation.s5.1.5.2-3,s5.1.5.2-27–s5.1.5.2-29chronic= administration= of= amiodarone= is= associated= with= complex= medication= interactions= and= a= host= of= adverse= effects= involving= the= lung ,= liver,= thyroid,= skin,= and= nervous= system.= as= a= general= rule,= the= longer= the= therapy= and= the=></40%, with or without coronary disease), concluded that, for primary prevention, amiodarone, compared with no treatment or placebo, decreased the risk of SCD (Risk ratio: 0.76; 95% CI: 0.66–0.88) and all-cause mortality (Risk ratio: 0.88; 95% CI: 0.78–1.00), but the quality of the supporting evidence was very low. S5.1.5.2-26 For secondary prevention of SCD, the same systematic review identified neither risk nor benefit with amiodarone. S5.1.5.2-26 Compared with beta-blocker therapy and other antiarrhythmic medications (including sotalol), amiodarone appears to reduce the risk of SCD and all-cause mortality. S5.1.5.2-26 Intravenous amiodarone has a role in reducing recurrent VF/VF during resuscitation. S5.1.5.2-3,S5.1.5.2-27–S5.1.5.2-29Chronic administration of amiodarone is associated with complex medication interactions and a host of adverse effects involving the lung, liver, thyroid, skin, and nervous system. As a general rule, the longer the therapy and the > (LVEF</120> (LVEF</120> dose of amiodarone, the greater the likelihood of adverse reactions requiring discontinuation of the drug. S5.1.5.2-26 For this reason, chronic treatment of young amiodarone patients should be reserved as a bridge to final treatment options such as catheter ablation. The baseline assessment of patients may include ECG, liver function tests, thyroid function tests, chest X-rays and lung function tests (including pulmonary diffnus capacity for carbon monoxide). Monitoring toxicity usually includes seasonal history and physical examination, as well as evaluation of ECG, chest X-ray and thyroid, liver and lung function. High-sestered chest containers are usually reserved for suspected pulmonary toxity. S5. 1. 5. 2-30Ether sotalol has some effectiveness in suppressing VA, has significant proarrrytic effects and has not been shown to improve survival. S5. 1. 5. Survival With Oral d-Sotalol (SWORD) was shown to increase the risk of death in heart failure patients. S5.1.5.2-32 Unlike amiodarone and many other arrhythmia agents, sotalol appears to lower the threshold for defibrillation. S5. 1. 5. The use of 2-33 Sotalol may also lead to the decompensation of HF, so its use in LVEF patients <20% is generally avoided. Calcium channel inhibitors The treatment of most calcium channel inhibitors of hydrogen hydropyridines is not relevant. In fact, intravenous verapamil, which has been administered to a continuous vein, has been associated with hemodynamic collapse, especially in patients with a history of MI. S5. 1. S5.1.5.2-35-S5.1.5.2-39 Oral and intravenous verapamil is effective in the treatment of idiopathic interfascicular reentrant LVT. S5. 1. 5. Calcium channel bloickers should not be used in patients with VT HFrEF. Antiarrhythmia drugs and treatments5.1.5.1. Electrolytes The increase in the efficacy of potassium and magnesium has been proposed as useful additional funds for va prevention. S5.1.5.2-40,S5.1.5.2-41 Hypocaemia and hypomagnesemia are common consequences for diuretic therapy in HF, both have been associated with VA during acute myoester, S5.1.5.2-41, S5.1.5.2-42 and may increase the risk of vibration in a patient prolonging the QT period. S5.1.5.2-43 In fact, in patients with rsades de pointes, intravenous magnesium is a first-line treatment. S5.1.5.2-44 In patients with both magnesium and potassium deficiency, magnesium should be reconstituted to facilitate potassium substitution. For S5.1.5.2-45 Potassium, some recommend keeping potassium content between 4.5 mmol/l and 5 mmol/l to prevent VA and SCD. S5.1.5.2-46,S5.1.5.2-47 In a large observational study of patients with acute ERAs, the lowest in patients with serum potassium levels between 3,5 mmol/l and <4,5 mmol/L.S5.1.5.2-48 It is interesting that the amount of intravenous product did not increase unless potassium was <3 mmol/l or ≥5 mmol/l. Similarly, a large randomised double-blind study with intravenum magnesium after eye resistance time was of no use in 30 days of mortality. S5.1.5.2-40 Monitoring potassium and magnesium during aggressive diuresis and post-error period.5.1.5.2. n-3 Fatty acids and lipidsBoth n-3 polyustatic fatty acids and statin treatments may affect the prevention of SCD, which is thought to be due to stabilisation of the bilipide myosyte membrane involved in the maintenance of electrolyte gradients. S5.1.5.2- 49Data was promising on the effects of n-3 polyunsaturated fatty acids on the reduction of cardiovascular events and SCD. In 2006, an extensive meta-analysis of 19 observational and RCTs showed a significant association between the consumption of n-3 polyunsaturated fatty acids and scd prevention. The S5.1.5.2-50 Randomised GISSI study (Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto)-Prevenzione found that fish oil 1 g/d reduced mortality due to a lower scd. However, the following RCTs have not reiterated these benefits and have shown that n-3 polyunsaturated fatty acids are ineffective. S5.1.5.2-52-S5.1.5.2-56 Since studies showed that n-3 polyunsaturated fatty acids do not harm, patients can be sure of their safety. Longer-term data will hopefully clarify the contradictory results. Instead, statin drugs clearly reduce mortality and appear to reduce the risk of the heart disease associated with heart disease. S5.1.5.2-57 The prevailing mechanism remains uncertain. Prevention of coronary plaque tear or direct cardioprotective effect has been proposed. Experimental ischemical/reperfusion models demonstrate the cardioprotective effect of statins, and extensive observational analysis found this effect in humans. S5.1.5.2-42,S5.1.5.2-56-S5.1.5.2-58 This was further investigated in HF in several secondary analyses of statin patients in ICD prevention studies, including MADIT-CRT (Multicenter Automatic Defibrillator Implantation Trial-Heart Resynchronization Therapy), SCD-HeFT, AVID (with antiarrhythmics and implantable defibrillators), S5. 1. 5. 2-59 and DEFINITE (DEFibrillators In Non-Ischemic Cardiomyopathy Treatment Evaluation) studies with lower risk of SCD in statin patients. S5.1.5.2-58,S5.1.5.2-60-S5.1.5.2-62 However, this overall effect on HF was not established in the potential RCT of 2 rosuvastatins in HF; CORONA (controlled Rosuvastatin Multinational study on heart failure) and GISSI-HF (Gruppo Italiano per lo della Sopravvivenza nell'Insufficienza Cardiaca-Heart Failure). S5.1.5.2-63,S5.1.5.2-64 It appears that: statin effects are limited to a population at or at risk of developing atherosclerotic cardiovascular disease and/or ischemistry, and HF in general.5.2. Blocking SCD with HF drugsSext support text1. For patients with HF and depressed LV function, proper medical treatment is important to reduce SCD. These treatments have different beneficial effects on arrhythmia mechanisms. Beta blockers reduce the oxygen demand and electrical arousal of the heart muscle and combat the arrhythmias of sympathetic stimulation. Angiotensin-converting enzyme inhibitors and angiotensin receptor inhibitors reduce pre-loading and after, reduce the demand for oxygen in the heart muscle, prevent the formation of angiotensin II and slow down the progression of ventricular renovation and fibrosis. Mineralocorticoid receptor antagonists limit potassium loss, reduce fibrosis and increase myocardium intake of norepinephrine. S5.2-7RCTs in HFrEF patients have consistently shown that chronic beta-catcher therapy reduces overall mortality, VA syndrome and sen death therapy. S5.2-2, S5.2-4, S5.2-5,S5.2-9 Three beta-predators (i.e. bisoprolol, vetilol, long-term metoprolol succinate) have been shown to reduce mortality in patients with current or past symptoms of HFrEF without beta-sazole contraindications. Blocking an angiotensin-converting enzyme also reduces mortality and SCD. Angiotensin receptor blockers added to an angiotensin-converting enzyme inhibitor showed additional benefits in angiotensin-converting enzyme inhibitors someS5. spironolclone and eplerenone have also shown both overall mortality and reduced SCD. S5. 2-6, S5. 2-12, S5. Recent studies of angiotensin receptor neprilycin inhibitor (sacubitril/ valsartan) compared to an angiotensin-converting enzyme inhibitor showed a reduction in SCD and heart mortality. S5.2-145.3. Defibrillators for va and SCDSee treatment 7, 10.2, 10.3, 10.8 and 10.9. Defibrillation is very effective in stopping life-threatening VA. This treatment can be given by transvensitive ICD, subcutaneous implantable cardiooverter defibrillator, wearable cardiooverter defibrillator or external defibrillator. These devices continuously monitor the heart rhythm and provide treatment in response to tachycardia, which meets pre-programmed detection rates and the duration of arrhythmias. Most transvensive ICDs are implanted in the subclave area under clavicle control. subcutaneous implantable cardiooverter defibrillators are implanted in the left side of the chest between the left midaxilary and the left anterior extension track. Some patients with epicardial identification and pace lines are still implanted especially those with certain Heart disease. Transvensive ICD has been in clinical use for >3 decades, and robust data from high-quality RCTs support its use in different patient populations, including cardiac arrest survivors, VT and structural heart disease, and patients with significant LV dysfunction.5. Catheter ablation5.4.1. General considerationsCatheter ablation is an important treatment option for patients with VA when antiarrhythmia drugs are ineffective, un tolerated or the patient does not want to. Monomorphic VA usually has an origin or substrate that can be targeted at ablation. Ablation is an option for selected patients with polymorphic VT/ VF only if a recruit PVC focus or substrate can be identified. The ablation strategy, risks and results are related to the mechanism and location of the VA mechanism. Most VA's originate close to subendocardidiomia and are approached through transvensitis (right ventricular) or transaortic/ transseptic (left ventricular) catheterization. Some diseases give birth to VA from subepardium, which can be approached by epicardial mapping and ablation. Pericardial access is usually achieved with percutaneous subxymphoid injection. Catheter ablation usually involves attempts to induce VT with programmed electronic stimulation to confirm diagnosis and control ablation. Success-limiting problems include the inaccessibility of arrhythmia to mapping (common with idiopathic VA) or the origin of arrhythmias from an inaccessible site of cardiac cardiocardium (common in some cardiomyopathies).5.4.2. Va patients without obvious structural heart diseaseSee-8.VA not associated with underlying structural heart disease or genetic arrhythmia syndrome are commonly referred to as idiopathic. Most idiopathic VA's are monomorphic and are based on triggering an incinerator mechanism of activity or abnormal automaticity; A few are scheduled to try again. For patients who are asymptomatic and have ineffective antiarrhythmia medications, who are intolerant or unwilling to be tolerated by the patient, catheter ablation is a treatment option. The ablation strategy is to identify the place of origin that appears from the earliest place of electrical activation or, if this is not practical, from the pace mapping. The ablation of the idiopathic VA catheter usually occurs with an endocardial catheter, although an epicardial approach through coronary left-blood circulation or pericardial injection of subxymphoids may sometimes be necessary. Idiopathic VA ablation disorder is often caused by an inability to provoke arrhythmias in order to map electrophysiological laboratory or origin from a difficult area.5.4.3. Scar-related VTSee Section 8.In most patients with structural heart disease, monomorphic VT is to be re-attempted in areas of surviving myocardosis fiber associated with fiber scar regions. Nniiden Nniiden The strategy of these re-development circuits is to identify and remove the survival channels inside the scar, which are often associated with slow management that facilitates rebirth. In most VTs associated with a previous ER, the substrate is on the subendocard surface of the left ventricle. In NICM, reentrant circuits are more varied in location, are often accompanied by the epicardial surface of either chamber and often extend to the surface of midmyocardium, where ablation can be difficult to achieve on either surface. Fallot's tetralogy defines specific re-enterprise paths. S5.4.3-1 Electroatomic mapping, which helps clarify the relationship of electrophysiological abnormalities to cardiac anatomy, is commonly used. Scar ranges can be considered areas of relatively low electrogram voltage. The hemodynamic intolerance of scar-based VTs often limits mapping during VT. Ablation is often controlled by substrate mapping, where scar ranges and the potential reentry circuit substrate are limited to electroanatomical maps based on a cardiac electric curve and paceing properties that are hemodynamically assessed during stable sinus or paced rhythm. The catheterisation of scar-related VT requires advanced experience from the user, electrophysiological laboratory staff and anesthesiologist, as well as the availability of surgical spare and special mapming, imaging and ablation equipment. S5.4.3-2,S5.4.3-35.5. Surgery and revascularization procedures in patients with ischemical heart diseaseRecommendation-specific support text1. Myocardosis ischemia is the cause of persistent polymorphic VT/ VF, and revascularization is an effective treatment to prevent myocardosis ischemist. In patients with life-threatening rheumatoid arthritis, observational studies show that patients undergoing coronary artery bypass transplantation (CABG) had significantly better survival after consideration by other predictors. S5.5-1, S5.5-5-5 The risk of coronal resuscitation appears to be comparable in patients with complex sechemical heart disease randomised to PCI compared to CABG. S5.5-6 In patients with low LVEF and cabg-friendly coronary heart disease, the risk of coronary disease is lower with CABG than with medical treatment. S5.5-2,S5.5-7 Observational studies show that survivors of SCA and CABGS5.5-3 or PCI have a lower probability of death from revascularization. Mere S5.5-4 Revascularization is usually not enough to prevent a recurrence of continuous monomorphic VT; further evaluation of inductive VT is generally considered in patients undergoing impaired ventricular function and/ or scarring.2.Abnormal aorta of coronary arteries is observed in approximately 1% of patients undergoing routine and <0.2% of children and adolescents receiving echocardiography. S5.5-8 Although sechemical heart disease is observed in up to 24-55% of SCD cases in young patients, < 35 years abnormal aortic origin of coronary arteries is an important cause of coronary artery disease in adolescents and has been reported in 10-17% of patients included in postmortem studies. S5.5-10,S5.5-11 The abnormal origin of the coronary arteries can be identified by echocardiography, invasive coronary angiography, CT angiography or cardiac MRI. In patients with SCA or life-threatening joint pain expected to be associated with ischemistry due to abnormal origin of the coronary artery, repair or revascularisation is performed to relieve ischemistry and reduce recurrent rheumatoid feed. S5.5-6,S5.5-7,S5.5-12–S5.5-145.5.1. Arrhythmia management surgeryRecommendation specific support text1. Heart surgery as a separate procedure for VT is rarely performed, but plays a role in some highly symptomatic patients when antiarrhythmia drugs and catheter ablation fail or are not possible, especially if ablation is caused by an arrhythmia caused by an area not caused by catheter ablation, such as deep in the heart muscle, under or near epiardial fat or near coronary arteries. Surgical ablation of takycardia can also be performed during other cardiac surgical procedures, such as during surgical resection of major aneurysm due to an indicator of previous error activity, where the boundary zone is often a VT substrate, or during the placement of an LV auxiliary device (LVAD). S5.5.1-5–S5.5.1-7 The procedure requires detailed imaging of arrhythmias, usually by pre-co-ordination imaging and mapping, so surgical ablation is best performed in higher referral centres and in collaboration between experienced surgeons and electrophysiists.5.6. Autonomic modulationSynopsisSympatetic activation is proarrhytic and parasympathetic activation is usually an antiarrhythmic medicine in VT/ VF. Modulation of the autonomic nervous system to prevent arrhythmias is a new therapeutic modality. To prevent VA, autonomous modulation can be done either by interrupting a sympathetic outflow to the heart, pharmacological beta-blocking or stimulating the parasympathetic path (e.g. vagal nerve stimulators, spinal stimulators). Although autonomous modulation has been shown to be effective under certain conditions, such as long QT syndrome and caecolamine-related polymorphic polymorphic ventricular tacardia (see section 7.9), there is little evidence of its applicability to the broader VA, but studies are ongoing. Currently, there is little information about the role of vagal nerve stimulators and spinal stimulators in the prevention of VA/ SCD in humans, so the official recommendation could not be supported. S5.6-5Spekumis-specific support text1. Many patients with non-life-threatening VA only require reassuring, but others have symptoms that require treatment. Small RCT in patients with symptomatic significant reduction in the burden of arrhythmia the storm causes significant mortility and is associated with increased mortality. For the refractory treatment of VT/ VF storm (medicines, catheter ablation), cardiac sympathetic denervation has been demonstrated in several small, observational studiesS5.6-3, S5.6-6 and 1 RCTS5.6-4 to reduce the burden of arrhythmia. This has been demonstrated by the sympathetic denervation of the left or bilateral heart, and it has been suggested that sympathetic denervation of the bilateral heart may be better. S5. 6-3 Although data are limited, significant morbidity and limited options in these patients make cardiac sympathetic denervation a reasonable option in selected patients. Acute management of a specific VAFigure 2. Treatment of persistent monomorphic VT. The colors correspond to the recommended category in Table 1. See sections 7, 8.1.3, 8.2.3 and 10. *Known history verapamil sensitive or classic electrocardiogram presentation. ACLS refers to advanced cardiovascular support for vital signs; ECG, electrocardiogram; VA, ventricular arrhythmias; and VT, chamber tachycardia. Support text per recommendation1. The most common electrical mechanisms for cardiac arrest are VF and pulseless VT, but a significant number of cardiac arrests begin with severe bradyarrhythmia or asystolen. Survival is better in patients with VT or VF than in patients with bradyarrhytism or asystotic mechanism. S6-32 The rapid arrival of medical personnel is the main determinant of survival. Several strategies to respond to unexpected cardiac arrest, including rapid defibrillation and resuscitation in case of witnessed cardiac arrest, have improved the odds of survival for cardiac arrest victims. However, the absolute number and proportion of survivors is still low, except for unique conditions where victims have an exceptionally rapid response time in VF or VT, such as supervised intensive care units with survival >90%. S6-33–S6-36 Survival decreases rapidly after the first 2 minutes after the onset of cardiac arrest, so that after 4-5 minutes survival can be ≤25%, and for 10 minutes it is 0%. S6-33,S6-35,S6-36 Advanced life support functions not directly related to cardioversion and defibrillation in takyarrhymia management have led to the emergence of comprehensive protocols to guide respondents. These AHA documents cover aspects of the widespread expansion of clinical conditions and mechanisms. S6-1, S6-372. Administration of Amiodaron emergency care after at least three failed shocks and administration of epinephrine improved hospital intake compared to placebo S6-6 or 1, 5 mg/ kg lidococinS6-1.S6-4 RCT in adults with out-of-hospital cardiac arrest due to refractory VF or polymorphic VT, although survival in hospitalization and survival with favorable result did not improve the However, in the subset of patients with initial cardiac arrest caused by VF or pulseless VT, survival from hospital after driving amiodaron was higher than in patients with placebo. Administration of S6-5 Prokainamide outside the hospital in cardiac arrest caused by VF or pulse-free VT has been associated with more shocks, more pharmacological procedures, longer resuscitation times and lower survival. S6-383. Hemodynamic instability, including VF and pulseless monomorphic or polymorphic VT, causes loss of consciousness and leads to death untreated. A short period of time to control the current cardio version is the main determinant of survival, and defibrillation must be done as quickly as possible. Resuscitation is used until the perfusion rhythm is restored. If defibrillation is not successful during spontaneous circulating recovery, the respondents follow an advanced cardiovascular vital function. S6-1–S6-34. The identification and treatment of patients with cardiac arrest outside the hospital associated with acute coronary artery blockage is associated with better survival and better functional recovery. S6-37 Coronary artery blockage as a cause of cardiac arrest is not reliably predicted by clinical and electrocardiogram findings, S6-7 and coronary artery angiography should be considered (and not later in hospital or not at all) in stale patients with suspected cardiac etiology, regardless of whether the patient is in a coma or awake. In patients resuscited from SCA S6-9,S6-39 without ST elevation with angiography, one third were found to have a cuddly lesion and coronary intervention appeared to be associated with a higher likelihood of favorable neurological outcome. S6-105. Preliminary management of takycardia should be proceeded in accordance with published AHA Advanced Cardiovascular Vital Signs Management Guidelines. S6-40 The instant cardio version must be made due to hemodynamic instability during the presentation or if it develops later. The ECG should be put in a stable rhythm. Broad complex tachycardias ≥0.12 p., S6-37, defined by QRS duration, can be caused by VT, SVT abnormality, already tachycardia or pacemaker rhythm, such as pacemaker interval from tachycardia. Irregular wide-ranging tachycardia may be AF with an abnormality, a previously assumed AF (i.e. AF using an accessory pathway for anterograde conductivity), attenuation or VT. S6-37 Diagnosis must be confirmed and consultation with an arrhythmia specialist should be considered. S6-376. In one study, amiodarone more effectively stopped persistent VT than lidocane and improved its survival at the 24-hour mark. S6-26 In patients with repeated stable VT not at the time of AMI insert, intravenous has been shown to be better than for stopping lidococin arrhythmia. S6-11 One randomised study in 62 patients with better prokainamide than amiodarone stable VT. S6-13 Adverse reactions, including hypotension, were more common with amiodaron, but the difference was not statistically significant. Procainamide and its metabolites n-acetyl procainamide have potassium channel blocking properties that may prolong QT time. In patients who already have an increase in QT time, administration of procainamide may further prolong QT time and lead to point warping. S6-11,S6-12,S6-267. Intravenous lidococin is an alternative antiarrhythmic medicine that is long-term and widespread familiar. Without antiarrhythmics, lidococin consistently did not increase spontaneous circulatory recovery after defibrillation and did not involve an improvement in survival from hospital discharge. S6-4,S6-14,S6-41 In prospective, blinded, RCT lidococin was less effective than amiodarone after cardiac arrest outside the hospital shock due to refractory VF or polymorphic VT; But there were no differences between the two medications in surviving to discharge from the hospital. However, S6-4, S6-5 Subgroup analysis showed that li lidocant survival in hospital was better than placebo. S6-5, S6-428. Extensive meta-analysis of antiarrhythmic drugs in the insertion of AMI into beta blockers was associated with a significant reduction in mortality. S6-16 Beta-blockers can effectively dampen recurrent VF in patients with recent health infent and associated survival improvement. S6-179. In patients with recurrent VT/ VF (VT/ VF) amiodarone and/ or lidocane and repeated cardioversion at the time of the recent insertion of the refractory cardiac infractor, beta-saleen administration has been shown to improve survival at 1 week. For those who did not survive, mortality was mainly due to repeated VF. Also in a 1-year evening, survival was better in beta-spas workshops. S6-17,S6-18 Other measures to reduce sympathetic tone, including sedation and general anaesthesia, are also often used.10.Epinephrine produces beneficial effects for patients during cardiac arrest, mainly due to its alpha-adrenergic (i.e. vasoconstric) effects. S6-1 These alpha-adrenergic effects may increase coronary and brain perfusion pressure during resuscitation. The value and safety of the beta-adrenergic effects of epinephin are controversial because they can increase myocardial work and reduce subendocardial perfusion. S6-1 One study evaluated short-term and long-term results when compared with a standard dose of epinephrine placebo. S6-23 The standard dose of epinephrine was defined as 1 mg vein or vein every 3 to 5 minutes. Both survival to repatriation and survival with a good neurological outcome were of no use to the usual dose of epinephrine; however, was for analysis of either result. However, survival in hospital admissions improved and spontaneous blood circulation recovery improved with the help of standard dose epinephin. Several studies have compared the results of the standard dose of epinephrine with those of high dose epinephrine. These studies revealed no benefit from high doses of epinephrine compared to the usual dose of epinephrine with good neurological recovery, discharge survival or hospital admission survival. S6-1,S6-19,S6-21,S6-2211. Amiodarone stopped lidococin more effectively with persistent VT and improved its survival at the 24-hour mark. S6-26 In patients with repeated stable VT not at the time of AMI insertion, intravenous procainamide has been shown to be better than when lidococin arrhythmia is continued. S6-11 In one RCT in 62 patients, procainamide was better than amiodarone to stop stable VT. S6-13 Adverse reactions, including hypotension, were more common with amiodaron, but the difference was not statistically significant. Procainamide and its metabolites n-acetyl procainamide have potassium channel blocking properties that may prolong QT time. In patients who already have an increase in QT time, administration of procainamide may further prolong QT time and lead to point warping. S6-11 One of RCT 33 patients comparing sroomal to lidocane in the treatment of patients with hemodynamically stable VT showed that VT was discontinued in 69% of patients taking sotalol and 18% of patients taking lidocan. S6-25 Intravenous sotalol is approved for use in the United States. Sotalol has potassium channel blocking properties that may extend the QT time. In patients who already have an increase in QT time, administration of sotalol may further prolong QT time and lead to point warping. S6-2512. Epinephrine can increase coronary artery and brain perfusion pressure during resuscitation due to its vasoconstric effects. High doses of epinephrine (0.1 to 0.2 mg/ kg intravenously compared to the standard 1 mg dose) have been studied with RCT. Outside the hospital in cardiac arrest, which was unresponsive to defibrillation, administration of high-dose epinephrine improved survival to hospital admissions, but there was no difference in hospital survival compared to the normal dose of epinephrine. S6-19 Long-term survival did not improve either. The use of S6-21 Vasopressin is no longer recommended for the latest advanced cardiovascular life management algorithms. S6-113. Magnesium may dampen autoactivity, dampen early and late depolarisations and prevent calcium flow to cardiomyocytes. It is effective in suppressing VA associated with acquired long QT syndrome. However, 2 RCTs that investigated the use of intravenous magnesium in patients with and refractory VF, found no In an out-of-hospital cardiac arrest study, intravenous administration of 2 to 4 g of magnesium did not improve survival to hospital admission. S6-27 In a similar study involving cardiac inpatient arrest, magnesium did not improve spontaneous circulation recovery, survival to 24 hours, or survival from hospital discharge. S6-28 There are exceptions, such as marked hypocaemia or drug-induced upper rsades de points where intravenous magnesium administration is warranted.14.Several studies have tested the hypothesis that prophylactic administration of antiarrhythmic drugs could reduce the inadformation of post-MI VA and lead to better results. One meta-analysis assessed studies in which beta-anta-alpaers, Class I arrhythmias such as lidocane and prokainamide, and amiodarone were administered when AMI was set. Routine use of lidocane and prokainamide was associated with increased mortality, while beta-predators were associated with significantly lower mortality rates. S6-16 Limited data on amiodarone appeared to be promising, but a subsequent dose of amiodarone involving 1,073 patients found that high dose amiodarone administration resulted in higher mortality, although a moderate dose of amiodarone was not better than placebo. S6-2915. With stable, extensive QRS-complex tachycardia, it is often possible to distinguish between SVT and VT by looking at the patient's history and 12-lead ECG during tachycardia. Patients with extensive QRS complex tachycardia and known structural heart disease should be assumed to have VT until proven otherwise. Administration of a calcium channel inhibitor such as verapamil to a patient with VT may lead to severe hypotension or fainting. S6-31 The exception is verapamil sensitivity VT (interfascicular reentry), which occurs structurally in a normal heart; But this is often difficult to identify in the original presentation. S6-307. Continuous management of VA and SCD risk associated with certain disease states7.1. Sechemical heart disease7.1.1. Secondary secondary secondary prevention in patients with ischemical heart diseaseFigure 3. Secondary contraceptive patient with sechemical heart disease. The colors correspond to the recommended category in Table 1. See sections 4.3.1 and 7.1.1. *Exclude irreversible reasons. † history consistent with arrhythmia theology. ・ICD candidacy according to functional status, life expectancy or patient preference. EP stands for electrophysiological; GDMT, instructional-controlled management and care; ICD, implantable cardiovascular defibrillator; IHD, sechemical heart disease; LVEF, left ventricular ejection part; pts, patients; SCA, sudden cardiac arrest; Heart attack, sudden cardiac death; and VT, chamber tachycardia. Recommendation-specific In the AVID study, S7.1.1-1 ICD improved overall life compared to antiarrhythmia drugs (mainly patients who survived senous treatment or hemodynamically unstable VT, and mortality decreased by 2% proportionately and 7% 7% either. The Canadian Implantable Defibrillator Study (CIDS), S7.1.1-2, which was stopped early after the results of the AVID study were announced, showed a similar but not statistically significant benefit of ICD compared to the treatment of antiarrhythmic drugs. In a subsequent meta-analysis of data from 3 RCTs, both arrhythmia and overall mortality were statistically significantly reduced through secondary contraceptives and ICD drugs. Survivors of S7.1.1-3I life-threatening spies that may be caused by transient or reversible factors such as AMI, proarrhythmic medication effects or electrolyte disorders, ICD will not be implanted if the cause may be reversible. This patient population still requires in-depth assessment, treatment and close monitoring, and as in the AVID register, mortality was still high in a population that may have had a reversible cause of arrest. S7.1.1-8 A small increase in troponin is a challenge in selecting patients for ICD, since it is often not possible to determine whether the increase in troponin is due to the ischemistry and resuscitation of VT/ VF, in which case the ICD is likely justified, or an indication that ischemia caused arrhythmias, making ischemistry prevention a therapeutic focus. ICD treatments can improve the outcome of patients with hemodynamically tolerated long-term VT and structural heart disease S7. 1. However, this has not been proven anywhere in RCT. VT ablation has been used as an alternative in selected patients with well-tolerated VT who appear to reduce relapse but the effect on long-term mortality is unknown; There is not yet enough evidence to recommend this approach as an alternative to ICD implantation. S7.1.1-9, The financial results of the S7.1.1-102.ScD secondary contraceptive ICD implantation were evaluated in the AVID and CIDS studies,S7.1.1-11,S7.1.1-12 and simulation designs 7.1.1-13 and observational study of Medicare beneficiaries. S7.1.1-14 All studies compared ICD recipients with non-ICD recipients, all of which found that ICD recipients had longer overall lifespan and higher medical lifecycle costs. All studies reported that the added value of ICD(S7.1.1-11-S7.1.1.14) adds between $64,000 and $100,000 per lifecycle year. S7. 1. 1- 153. In a study of 70 patients with unexplained fainting who underwent an electrophysiological examination, positive findings were observed in 37 patients; 31 and VT. During the three-year follow-up year, patients with positive electrophysiological research had (61% vs. 15%), than those with negative electrophysiological research. S7.1.1-7 ICD is justified for patients with fainting and uninsinsible continuous monomorphic VT, even if they do not otherwise meet the criteria for primary prevention (Figure 4).7.1.1.1. Coronary spasm Recommendation-specific support text1. Coronary artery convulsion is caused by vasomotor dysfunction and may occur in the presence or absence of athemosclerotic iscino heart disease. Vasospasm episodes can lead to VA, syncope and SCD. Treatment includes eliminating the risk factor, including quitting smoking, and vasodilator therapy, including dihydropyridine calcium channel inhibitors with or without nitrates. A more detailed summary of treatments for coronary artery convulsions can be found in other instructional documents. S7.1.1.1-7,S7.1.1.1-82.Patients who survive coronary artery spasms are at risk. S7.1.1.1-5 Recurrent, even life-threatening, can be prevented if coronary artery spasm can be effectively addressed through risk factor conversions, smoking cessation and continuous treatment of nitrates and dihydropyridine carlestium channel inhibitors. However, S7.1.1.1-9 SCA or VA may resent despite medical treatment or if compliance is poor. Whether a wearable cardiooverter defibrillator can provide protection during medical treatment has not been assessed, but it is interesting. S7.1.1.1-10 ICD may stop convulsions, patients with coronary artery disease who have coronary artery disease and survive treatment with SCA are at risk, and some support the use of ICD based on reported event figures from observational studies in these patientsS7.1.1.1-5 even before determining the patient' s response or following medical treatment. Recurrent SCA may occur despite medical treatment. Regardless of the approach, changing the risk factor (e.g. illicit use of drugs), quitting smoking and continuous treatment with dihydropyridine calcium channel inhibitors with or without nitrates are essential treatments. S7.1.1.1-97.1.1.2. After CABG VT/VF, the early prevalence of long-term VT or VF after CABG is low, but these TA's are associated with high hospital mortality rates. S7.1.1.2-1 VF at a very early stage (intraoperatively or within 24 hours of inoflation disorders) may be due to transient effects of reperfusion, electrolyte and acidic disturbances, and the use of inotropics. Patients with vf or polymorphic VT more frequently after the period of action often have ischemia, while patients with monomorphic VT usually have an old infarction and ventricular scar. S7.1.1.2-2 Polymorphic VT/VF after CABG requires a therapeutic approach to treatment, including any need to assess the patensity of the graft and to identify and complications and acute electrolyte or acid-related disturbances. Risk factors for the presence of monomorphic VT after cabg include prior myocardial infarction, ventricular scar, LV dysfunction and the placement of bypass graft over non-collateral clogged coronary artery astima in the chronic infarction zone. S7.1.1.2-3 Unlike polymorphic VT and VF, continuous monomorphic VT is usually not caused by acute ischemistry. Many of these patients have uninsinsible long-resistant VT in electrophysiological research. Treatment of symptomatic vasat after CABG follows the recommendations for acute and continuous management of VT described elsewhere in this document. In patients without persistent VT or VF but had LV dysfunction prior to going through CABG, implantation of ICD did not improve survival. S7.1.1.2-4 In patients with LV dysfunction who are in revascularisation, it is possible that the LV function may improve, so many support the reassessment of the LV function 3 months after revascularisation prior to ICD implantation. S7.1.1.2-5 In patients with high NSVT loads and reduced LVEF, electrophysiological examination may be useful for risk sharing. Those with permanent VT may benefit from ICD.S7.1.1.2-6 Wearable cardiooverter defibrillator may play a role in patients at risk of interference at an early stage after revascular contamination to allow for recovery of ventricular function. S7.1.1.2-77.1.2. Primary prevention of sepaksta disease in patients with ischemical heart diseaseFigure 4. Primary prevention of mixed departmental diseases in patients with sechemical heart disease. The colors correspond to the recommended category in Table 1. See section 7.1.2. *Scenarios for early ICD placement under certain conditions, such as in patients with a syncer motif or fainting †Vaded HF therapy includes CRT, heart transplantation and LVAD thought due to VT. These are detailed elsewhere in the expert report of the High Representative/ACC/AHA. S7.1.2-24 CRT refers to cardiac resynchronisation therapy; EP, electrophysiological; GDMT, instructional-controlled management and care; HF, heart failure; ICD, implantable cardiovascular defibrillator; IHD, sechemical heart disease; LVEF, left ventricular ejection part; Myocardial infarction; NSVT, unsustainable chamber tachycardia; NYHA, New York Heart Association; pts, patients; Heart attack, sudden cardiac death; VT, chamber tachycardia; and WCD, wearable cardiovascular defibrillator. Support text per recommendation1. The justifications for providing ICD to patients with NYHA Grade II or III HF in addition to LVEF ≤35% are based on the survival benefit observed in SCD-HeFT and MADIT-II (where the cut-off of LVEF was less than 35% and 30%). Choice n planting must be separated. Patients with serious involvements related to < 1 year survival tend not usually ICD candidates. The recommendation to wait at least 40 days after the MI before installing primary prevention ICD is based on the that such patients were excluded from MADIT-II and SCD-HeFT, and 2 other RCTs had no survival benefit from ICD implanted early in acute MI.S7.1.2 to 10,S7.1.2-112.MADIT-II study S7. 1 ≤. The subgroup analysis supported the benefit of the ICD from the survival of this subgroup. The financial results of the primary prevention ICD implantation of S7.1.2-23.ScD were assessed in 3 RCTs [MADIT-I, S7.1.2-12 MADIT-II, S7.1.2-13 and SCD-HeFTS7.1.2-14], 1 observational study,S7.1.2-15 and 4 simulation models,S7.1.2-16-S7.1.2-19, all of which had generally consistent results. All studies reported an increase in survival and life expectancy and higher lifecycle costs for medical care with ICD than without ICD. Additional cost-efficiency ratios were generally < $50,000 per year increased by ICD, providing high value for current guidelines. S7.1.2-20 The value given by the ICD was consistently high as life expectancy was projected to increase by >1.4 years. S7.1.2-18 On the other hand, when ICD implantation did not increase survival, because in cabg-patch study S7.1.2-18 ICD did not provide a value, since higher costs were not included in the increase in life expectancy.4.MUSTT (Multicenter Unsustained Tachycardia Trial) showed that patients with prior cardiac support, NSVT and decreased LVEF and inductive VT in electrophysiological research had higher overall mortality rates than similar patients without inductive long-term VT. S7. 1.2-21 In patients who received ICD after antiarrhythmia medications did not inhibit uninsinsible VT, mortality was lower than in those who did not receive ICD. Although the criteria for admission to MUSTT required LVEF to be ≤40%, the mean LVEF was 30% in the patients selected for the study, and the ICD was not randomized, but was selected by the treating physician for patients with VT who could not be suppressed with antiarrhythmia drugs. MUSTT enabled the registration of patients who ≥4 days after acute ERM detector or revascular access. ICD was not beneficial in 2 other RCTs that looked at the efficacy of ICD in the acute phase of MI.S7.1.2-10,S7.1.2-11 In one observational study of the center, an electrophysiological study was conducted after a 9-day median acute myoemic injury in 115 patients, LVEF <40% and ICDs recommended for patients with inductive VT. The median follow-up was 12 months. Long-lasting vt was induced in 27% of patients, and in 22% of patients receiving ICD, ICD discontinued spontaneous VT in monitoring. No patient without inductive VT had Retrospective analysis of the United Network for Organ Sharing (UNOS) register, which stretched from 1999 to 2014, data on 32,599 patients showed that a median of 154 days for follow-up 3638 patients (11%) died on the waiting list for heart transplantation (9% in the ICD group and 15% in the non-ICD group; P<0.0001). The presence of ICD in the IPO was associated with a relative decrease in mortality of 13%. In the subgroup of LVAD patients (n=9478), ICD was associated with a 19% relative reduction in mortality risk. S7. 1. 2-9 In the second study, in which 380 patients were listed for heart transplantation between 2005 and 2009 in 1 tertiary cardiac transplantation centre, 122 patients received ICD before or within three months of being listed for a heart transplant. Non-ICD patients were more likely to die on the transplant list. In the multi-variable model, ICD was not associated with an improvement in survival; However, this analysis was limited by a small sample size. S7. 1.2-8 Another small study (n=79) conducted in one facility showed that ICDs reduce the risk of mixing in patients with LVEF ≤30% waiting for a heart transplant; However, this study was limited by the low number of patients. S7.1.2-6 In a retrospective multicenter study of 1089 heart transplant patients, 550 patients (51%) was an ICD. In 216 patients, ICD was for primary SCD prevention and in 334 patients ICD was for secondary contraception. The remaining 539 patients did not receive ICD. On the waiting list over a median period of 8 months, ICD was associated with a reduction in overall mortality for primary and secondary contraceptive cohorts (estimated at 1 year: 88±3% compared to 77±3% compared to 67±3%; P =0,0001). This relationship between ICD and improved survival was maintained even after adjustments by potential mixers. S7.1.2-76.RCT does not provide sufficient information on the value of ICD in patients with NYHA class IV HF. Ambulatory Class IV patients with HF were included in the COMPANION study (Medical Treatment Comparison, Pace and Defibrillation of Heart Failure), which showed a general improvement in functional status and survival with a CRT defibrillator. S7.1.2-23 Unless such a patient is candidates for CRT or advanced HF therapy such as heart transplantation or LVAD, ICD is not expected to significantly prolong survival. S7.1.2-237.1.3. Treatment and prevention of recurrent May Day in patients with ischemical heart diseaseFigure 5. Treatment of recurrent VA in patients with sechemical heart disease or NICM. The colors correspond to the recommended category in Table 1. See sections 5.6, 6, 7.1.3 and 7.2. *The cord should start by ensuring that the ICD is properly programmed and that any pre-prey causes, including worsening heart failure, are addressed. 1000 for optimal ICD programming, see hrs/EHRA/APHRS/SOLAECE expert report 2015. S7.1.3-26 APHRS shows the Asia-Pacific Heart Rhythm Society; EHRA, European Heart Rhythm Society; HRS, Heart Rhythm Society; IHD, sechemical heart disease; ICD, implantable cardiovascular defibrillator; PVC, premature chamber complex; NICM, non-hitting cardiomyopathy; SOLAECE, Sociedad Latinoamericana de Estimulación Cardíaca y Electrofisiología; VF, ventricular fibrillating; and VT, chamber tachycardia. Support text per recommendation1. The most common antiarrhythmic drugs used to suppress VA are amiodarone and sotalol, while mexyletin, kinidine and ranolatsin are used occasionally. S7.1.3-17,S7.1.3-18 Amiodarone appears to be more effective than sotalol and has low levels of ventricular proarrhym, but has an increased risk of medication-related adverse reactions leading to discontinuation in many patients within 18 to 24 months of initiation of treatment. S7.1.3-1, S7.1.3-19 Data on the effectiveness of sotalol to suppress VA are contradictory, but given its more favorable adverse reaction profile than amiodarone, it may be a better first-line arrhythmia drug in appropriate patients. However, S7.1.3-1-S7.1.3-3 Sotalol is generally avoided in patients with significantly reduced LVEF <20% due to negative inotropic effects and the risk of inotropic effects. In a double-blind placebo-specific study in which 674 patients with HF and ≥10 PVC/ h and LVEF ≤40% randomly indicated to receive amiodarone (336 patients) or placebo (338 patients), there was no significant difference in overall mortality or SCD between the two patients. The total mortality rate of amiodarone-treated NICM patients decreased (P =0.07). S7.1.3-202. Va storms are associated with increased mortality. S7.1.3-12 The arrhythmia substrate is usually in subendocardium. Randomised VANISH (Ventricular Tachycardia Ablation vs. Escalated antiarrhythmic therapy in ischemical heart disease) studyS7. Primary outcome, combination of death, VT storm or ICD shocks occurred in 59.1% of the ablation group and 68.5% in the escalated treatment group. There was no difference in mortality between the groups. Repeated ICD attacks, VT storm and treatment-related adverse reactions were lower in the ablation group. Subgroup analysis in patients with Amiodarone-treated VT had better results with ablation than amiodarone or the addition of mexiletine to amiodarone. In patients taking medicines other than amiodarone, catheter ablation did not reduce the risk of ICD shocks or VT storms compared to switching to amiodarone. Although repeated VT catheter ablation after ablation is associated with increased mortality,S7.1.3-9 whether mortality has decreased with catheter ablation has not been demonstrated. Processed complications occur in approximately 6% of patients, most of whom are related to access to blood vessels, but stroke, tamponade and atrioventricular lobes may occur. Action mortality is <1% in experienced centers. S7.1.3-4, S7.1.3-9Valysed monomorphic VT often occurs in random individual episodes in patients with a previous ER. Several non-randomized studies have shown that catheter ablation reduces repetitive VT or ICD shocks. S7.1.3-5, S7.1.3-7,S7.1.3-8 Meta-analysis of 5 VT ablation studiesS7.1.3-5 reported that VT was repeated in 35% of patients after catheter ablation compared to 55% of arrhythmia drugs. In catheter ablation study S7. 1.3-7 patients with ≥3 long-term VT episodes in the previous 6 months, 53% did not have recurrent VT at six-month follow-up; The median of VT periods decreased from 11.5 to 0. Ablation is better than increased medication therapy, which was reflected in a combined endpoint of death, VT storm or ICD shocks in the VANISH study. S7. 1. 3- 43. Patients with a previous patient who develop permanent monomorphic VT have frequent episodes. VTACH (Ventricular tachycardia ablation coronary artery disease implantable in addition to defibrillators) studyS7. After two years, VT had been renewed in 53% of the ablation group and 71% of the control group. Ablation extended the duration of recurrent VT from a median of 5.9 months to 18.6 months. S7.1.3-11 Several non-randomised studies have shown that that catheter ablation reduces the risk of recurrent VT or ICD shocks in patients with previous MI.S7.1.3-5,S7.1.3-7,S7.1.3-8 Catheter ablates multicenter study S7.1.3-7 in patients, who had ≥3 long-term VT periods in the previous 6 months, 53% did not have recurrent VT for six-month follow-up; The median of VT periods decreased from 11.5 to 0. Meta-analysis of 5 VT ablation studiesS7. Another study of 63 patients with recurrent VT after the conceding disorder showed acute success with catheter ablation in 83% of mappable VT and 40% of non-small VTs. S7.1.3-8 Ablation superiority compared to escalating therapy in patients with repeated VT despite antiarrhythmia medications. Vanish trial. S7.1.3-4 See section 5.6.4.CASTS7.1.3-21 showed higher mortality or non-oral cardiac arrest in post-MI patients treated with encainide or flecainide when used to suppress PVC and NSVT. S7.1.3-13 Propafenon is associated with increased mortality in SCA survivors compared to beta blockers, amiodaron and ICD. Implantation of S7.1.3-225.ICD before suppressing repeated or persistent VA puts the patient at high risk of repeated shocks, which can be psychologically harmful and has been associated with increased mortality. S7.1.3-23,S7.1.3-246.Sustained monomorphic VT in the setting of previous myoesthosis is typically due to scar-related re-development and not acute ischemia. Although it may be appropriate to recommend revascularisation when there is another revascularisation indication, revascularisation alone is unlikely to reduce the recurrence of monomorphic VT and specific treatments such as antiarrhythmia inhibitory medicine or ablation may be necessary to prevent recurrence. S7. 1.3-16 On the contrary, revascularisation may be beneficial in patients with sechemical heart disease and VF, polymorphic VT or ischemia-related exercise arrhythmias. S7.1.3-257.2. Nonischemic CardiomyopathyRecommendation-specific Supportive Text1.Heart MRI enables the assessment of structural heart disease and the assessment of the functioning of the LV and camper van, including the quantification of the mass and volume of LVEF, LV and the valvulaar structure. An MRI scan of the heart can help assess myocardicular infiltration processes and scars, suggesting delayed hyperenhans associated with va. S7.2-1–S7.2-4, S7.2-62.Delayed hyperenhancement has been associated with worse results, including SCD. S7.2-1-S7.2-33.It is important to take into account the genetic etiology of the NICM. The main objective of NICM genetic testing is to identify at-risk relatives with a disease-causing mutation and to help clarify the prognosis. Lam's A/C and NKX 2.5 mutationsS7.2-7-S7.2-12 are associated with a particularly high risk of early leadership disease, arrhythmias and SCD, and their identification often leads to consideration of early use of ICD. However, it is not known whether early pharmacological treatment of mutation-positive, asymptomatic subjects can prevent or delay the occurrability of the disease or whether genetic testing will eventually improve survival.7.2.1. Secondary secondary secondary prevention in patients with NICMFigure 6. Secondary and primary secondary secondary and primary secondary colors correspond to the recommended category in Table 1. See section 7.2. *ICD candidacy for functional status, life expectancy or according to preference. 2° means secondary; EP, electrophysiological; GDMT, instructional-controlled management and care; HF, heart failure; ICD, implantable cardiovascular defibrillator; LVEF, left ventricle chamber fraction; NICM, non-hitting cardiomyopathy; SCA, sudden cardiac arrest; Heart attack, sudden cardiac death; VA, ventricular arrhythmias; and WCD, wearable cardiovascular defibrillator. Support text per recommendation1. S7.2.1-1,S7.2.1-2,S7.2.1-4 The most commonly used antiarrhythmia drugs were amiodarone, beta blocker or both, although in the CASH (Heart Arrest Study Hamburg), S7.2.1-4 there was also a propafenone hand that was discontinued early due to increased mortality. A total of 1963 patients were admitted to the study, but only 292 patients (14.8%) was NICM. A meta-analysis of shared AVID and CIDS data found that the overall mortality rate of NICM patients decreased by 31% compared to medical treatment. S7.2.1-3 Although this analysis was outnumbered, the observed reduction in mortality was consistent with the benefit observed throughout the study population. AVID study S7. 2. Therefore, stable VT is likely a sign for a substrate capable of producing subsequent deadly arrhythmias. S7.2.1- 52. Small observational studies showed high mortality rates and frequent appropriate ICD attacks in patients with fainting and NICM. S7.2.1-7–S7.2.1-9 Recently, the assumption that malignant TIAs are the probable cause of the synkope and that the ICD is protective has been questioned. In a subgroup analysis of SCD-HeFT involving 472 patients, ICD did not reduce recurrent fainting or increased mortality risk associated with fainting. S7. A subgroup analysis of the Multicenter Automatic Defibrillator Trial-Reduce Inappropriate Therapy (MADIT-RIT) found that only 39% of patients had fainting arrhythmias. S7. 2.1-11 These studies suggest that the fainting of some HF patients may be an indicator of end-stage cardiomyopathy associated with poor prognosis. S7. 2.1-11 Long-lasting VT/ VF not induced by the DEFINITE sub-number was observed in a minority of patients but was associated with appropriate ICD therapy. S7. 2.1-14 Another study of electrophysiological testing of NICM found uninsinsible VT/ VF in 27.8% of patients associated with future ICD events. S7. In a study in 2.1-15 NICM patients, heart mortality correlated with LVEF, but not with the inductiveness of electrophysiological research. S7.2.1-16 This information makes it uncomfortable for many experts to conceal the ICD hold from patients with NICM who may have syncopoint due to a VA test, although electrophysiological research does not suggest an inducable sustainable VT.3.Access ICD may be limited by financial, medical or personal considerations. In addition, not all patients at high risk of SCD meet ICD indications, such as those with Class IV HF HF HF potential without CRT or life expectancy for <1 years. The RCT meta-analysis of mixed renal disease prevention mixed with Amiodaron included 15 studies in which 8522 patients were prescribed amiodaron or placebo/ control. S7.2.1-12 Amiodarone reduced scd risk by 29%; However, it did not reduce overall mortality and was associated with an increased risk of pulmonary and thyroid toxicity. In the subgroup analysis, the benefit of amiodarone appeared similar in patients with ischemia cardiomyopathy and in patients with NICM. S7.2.1-12 In separate meta-analysis S7.2.1-13, the evidence was sufficient to support the efficacy of amiodaron in reducing SCD and total mortality in or on cardiac arrest survivors, with cardiac arrest or palpitations due to VA. Subgroup analysis of the VALIANT study (Valsartan in acute myocardial infarction) found that amiodaron was associated with increased mortality in NYHA Class III HF patients. S7.2.1- 17 This information requires a careful and nuanced approach to the use of amiodaron to prevent secondary secondary contraception in patients with NICM. 7. Primary prevention of SCD in patients with NICMRecommendation-specific support text1. Four prospective RCTsS7.2.2-1,S7.2.2-2,S7.2.2-5,S7.2.2-6 initially evaluated ICDs for primary prevention of mixed diseases in patients with NICM. TwoS7.2.2-2,S7.2.2-6 were small studies that were closed early at a low event rate. DEFINITE,S7.2.2-5 ICD reduced the risk of SCD, and the trend towards decreased overall mortality. ScD-HeFT included 792 NICM patients. S7.2.2-1 Total mortality at age 5 was 27% in the placebo group and 21% in the ICD group (P =0.06). A combined analysis of these studies showed a significant 31% decrease in overall mortality of ICD compared to medical treatment. S7. 2.2-4 Danish study for the evaluation of the efficacy of ICD in patients with non-ischemical systolic heart failure in mortality) StudyS7. In this study, 1,116 patients with NICM LVEF <35% and Grade II, III or IV (if CRT was designed) were randomized to HF for ICD or not for ICD. 58% of patients with ICD and medical treatment had CRT (either ICD or pacemaker). Therefore, DANISH results should not be generally generaled in patients with NICM who are not eligible for CRT. With a median of 5.6 years of follow-up, ICD reduced scd from 8.4% to 4.3%, but there was no difference in overall mortality. S7.2.2-11 Several meta-analyses have been published. One submitted information on an ICD with or without CRT and survival benefit ICD.S7.2.2-13 Data on the second patient level used in 2 studies and adopted a more robust approach to reduce heterogeneity by excluding CRT patients and patients randomised to antiarrhythmia drugs; A 25% relative reduction in risk of mortality with ICD was demonstrated. S7.2.2-122.Laminopathies are mainly diseases caused by mutations in Lamin's A/C gene, which produce various hereditary diseases, such as subtypes of muscular dystrophy and progerian. Heart isolation is also at stake, and it is an important cause of family cardiomyopathy. S7.2.2-9 The disease is highly penetrant that all sufferers have evidence of illness at the age of 60. Cardiac manifestations may include atrial fibrillament, conductive disorders, VA and NICM. Several observational studies reported a high risk of seconds when the heart is involved. S7.2.2-7–S7.2.2-10 One study reported the most common form of death in SCD (46%) both isolated cardiovascular and neuromuscular phenotype Lamin diseases. S7.2.2-9 269 LMNA mutation positive specimen cohort S7.2.2-10 NSVT during open heart cardiographic monitoring LVEF <45% in the first assessment, male sexual and non-missensitation mutations were independent risk factors for VA. Malignant tumours were observed only in ≥2 of these risk factors. S7.2.2- 10 Nothing in the study has tested the effect of ICD on long-term survival.3. S7.2.2-1,S7.2.2-11 Although such patients were included in the DEFINITE study, only 99 (21.6%) Of the 458 patients in the DEFINITE study, class I HF. S7.2.2-5 It is therefore uncertain whether primary contraceptive ICD will improve survival in such patients.4. Patients in ambulatory class IV HF were included in the COMPANION study, whose functional status and survival had generally improved with CRT defibrillator. S7.2.2-3 Unless such a patient is candidates for CRT or advanced HF therapy such as heart transplantation or LVAD, ICD is not expected to significantly prolong survival. S7.2.2- 37.2.3. Treatment of repeated renal impairment in patients with NICMRecommendation specific support text1. Although ICDs are highly programmable and provide antitachycardia sync therapy,

which can end most episodes of VT without shock, preventing both appropriate and inappropriate shocks remains an important concern. The Optimal Pharmacological Therapy in Cardioverter Defibrillator Patients (OPTIC) study randomised 412 patients with documented VT and VF who received ICD within 21 days of a documented arrhythmiaS7.2.3-1 and a beta-blocker, just a sotalol or just a beta-blocker. In one year, shocks occurred in 38.5% of the beta-log inhibitor alone, 24.3% of the solo list and 10.3% amiodarone plus beta-log. At 1 year, the disseal rate of study medication was 18.2% for amiodaron, 23.5% for wartalol and 5.3% for a beta workshop alone. Adverse pulmonary and thyroid events and symptomatic brady cardia were more common in patients randomised to amiodaron. Therefore, amiodarone and beta-catcher prevented ICD shocks more effectively than sotalol, but as the risk of medication-related adverse reactions increased. S7. 2.3-1 Sotalol should not be used in patients with LVEF <20% due to its negative inotropic effects.2. Mri scans of the heart often refer to the location of the scars, which are usually at the heart of the miterial annulus or partition. The S7.2.3-4,S7.2.3-5 VT substrate may be subendocardial, subepicardial or intramyocardial, and all locations may affect and require endocardial and epicardial ablation. In the Heart Center of Leipzig VT (HELP- VT) study, successful ablation of all VT morphologies S7.2.3-2 was achieved in 66.7% of NICM patients compared to a 77.4% success rate in sechemistry cardiomyopathy. An epicardial approach to ablation was necessary in 30.2% of NICM patients compared to 1.2% with ischemical cardiomyopathy. Epicardial ablation was an independent predictor of successful ablation. Acute and long-term ablation success is lower in NICM compared to post-MI patients. Long-term survival-free VT relapse after catheter ablaation appears to be better in ischemica patients than NICM (57% vs. 40.5% after 1 year). S7.2.3-2 Risks are similar to those observed in post-myocardial infarction VT ablation and, if necessary, there are additional risks of availability and ablation of epi-crises. Although any NICM may cause scar-related VT, cardiac sarcoidosis (see section 7.6) and Lamin mutations are specifically associated with long-lasting monomorphic VT. S7.2.3-67.3. Arrhythmiagenic right ventricular cardiomyopathySynopsisArrhythmogenic correct ventricular cardiomyopathy is hereditary cardiomyopathy, which mainly affects the right ventricle but can affect the left ventricle, causing the myocardium to be replaced by fibrosis and fatty tissue, which often cause VA and SCD. Support text per recommendation1. Selected primary relatives refer to relatives who are willing to further test and who could benefit from further screening and testing (and not terminally ill patients or those who do not wish to be screened and tested). Arrhythmiagenic correct ventricular cardiomyopathy is often caused by a mutation involving a desmosomal protein, and is usually an autosomal dominant heritage with a variable agglomeration. SCD may be original arrhythmia gene correct ventricular cardiomyopathy. Clinical screening of ECG, cardiac imaging and ambulatory rhythm monitoring and/or exercise testing can identify family members at risk of arrhythmia-genic right ventricular cardiomyopathy. Arrhythmiagenic correct ventricular cardiomyopathy is clinically observed in approximately 35-40% of first-degree relatives, S7.3-3,S7.3-4 most commonly in siblings or symptomatic first-degree relatives. S7.3-4 When the proband is identified by the mutation causing the disease, targeted genotypic screening can identify mutation positive relatives,S7.3-1, and about 35% of positive individuals eventually develop a progressive manifestation of the disease. S7.3-1,S7.3-4 Arrhythmiagenic right ventricular cardiomyopathy positive individuals who do not initially manifest the disease, between 8 % and 16 % have a significant arrhythmia over the next 7-39 years. S7.3-1, S7.3-4, S7.3-26 Early identification of affected or potentially ill family members may allow lifestyle changes in sports participation and serial monitoring for the development of electrocardiogram abnormalities, symptoms, ventricular dysfunction or arrhythmias. In view of the subtle complexities of genetic testing of cardiomyopathy in the arrhythmia gene right ventricle, the decision to continue family screening is facilitated by informed genetic advice to discuss the cost of testing, the possible absence of an individual gene as a determinant of the manifestation of the disease, the psychological effects of the progression of an uncertain disease and the effects on lifestyle change, for screening and possible treatment.2.Cardiac MRI provides a high-quality assessment of the function, size, abnormality of regional wall movement and the extent of scar and fibrosis (late gadolinium enhancement), which account for 30-95% of patients with clinical diagnosis of arrhythmia-genic correct ventricular cardiomyopathy. S7.3-5, S7.3-6, S7.3-37,S7.3-38 Cardiac MRI detects bipolar participation in 34-56% of patients, and isolated LV participation was observed in 4-9% of patients. S7.3–37–S7.3–40 Cardiac MRI should include the assessment of late gadolinium enhancement by quantification of fibrosis. Applying the criteria of the 2010 working group to the cardiac MRI criteria for the diagnosis of arrhythmia-genic right ventricular cardiomyopathy has improved the specificity of this test. S7.3-5,S7.3-8 Electrocardiogram and Holter findings precede observable cardiac MRI abnormalities in arrhythmiagenic right ventricular cardiomyopathy mutation positives, and only 4% of patients with normal electrocardiogram and Holter score have cardiac MRI abnormalities, suggesting that the heart rate and assessment of function by MRI of the heart may be unnecessary in mutation-positive individuals without electrical abnormalities. S7.3-7 Presence of both abnormalities and abnormal cardiac MRI findings may identify patients at increased risk of long-lasting spies. S7.3-7,S7.3-38 The scar ranges observed in the cardiac MRI area are correlated with the location of the VT substrate identified by endocardial and epicordial mapping. S7. In the early stages of the disease, basic cardiac examination may provide useful information and electrocardicular and rhythmic ailments to monitor the progression of the disease over time. Experience and expertise in the interpretation of cardiac MRI are important. S7.3-5, S7.3-83.Arrhythmiagenic right ventricle cardiomyopathy is characterized by progressive ventricular myocytes loss to replace fatty or fibrous tissue, and is associated with progressive ventricular dysfunction that can be associated with both chambers. VA, fainting and SCD can occur relatively young, especially during the second and third decades and often during physical activity. S7.3-1, S7.3-16, S7.3-22, S7.3-41 Sustained VT is an important predictor of SCA and SCD or appropriate ICD shocks in patients with arrhythmia-genic correct ventricular cardiomyopathy. S7,3-10, S7, 3-13 In patients receiving ICD for primary prevention, appropriate ICD shocks are reported in 24-48% of patients. S7.3-9,S7.3-10,S7.3-12,S7.3-13 Long-term VT for cardiomyopathy patients with arrhythmiagenic right ventricle is monomorphic at 55-90% of IC-based cycles VT.4.Frequent PVC, >760-1000/24 hours during therapy rhythm monitoring, using S7.3-12,S7.3-36 antitachycardia paceing algorithms, correlates with the risk of arrhythmia. The presence of S7.3-9, S7.3-23 NSVT or persistent VT is an important predictor of heart disease. S7.3-9,S7.3-12,S7.3-13,S7.3-42,S7.3-43 The increased risk of arrhythmia caused by intense exercise is consistent with the beta-adrenergic modulation of disease manifestation. S7.3-17,S7.3-20,S7.3-21 The observation register reported that atenolol or amiodarone therapy was associated with less clinically relevant spying, while sotalol was not associated with an effect or increased arrhythmia. In general, S7.3-15 Ambulatory monitoring is used to assess the burden and adequacy of beta-sagging treatment. S7.3-9, S7.3-14, S7.3-23, S7.3-425.Patients with arrhythmia correct ventricular cardiomyopathy have a significantly increased risk of mixer during exertion. S7. 3-16, S7. 3-17, S7. 3-20, S7. Strong exercise in patients with arrhythmiagenic correct ventricular cardiomyopathy has been shown to impair myocardia function through echocardiography and cardiac MRI. S7.3-19 Participation in high-intensive/endurance exercise accelerates the progression and risk of communicable disease/disease in patients with arrhythmia and mutation positives, as well as the mutation-positive family Patients with arrhythmia correct ventricular cardiomyopathy who participate in competitive sports have a higher risk of VT or SCD than those who participate in recreational sports or are inactive. S7.3-17–S7.3-19,S7.3-21 Exercise affects the progression of the disease linearly; family members who limited activity below the minimum level of activity guidelines recommended by the AHA (<650 metabolic equivalent hours per year [MET-Hr/year]) are unlikely to develop VA or disease progression. S7. In a study of arrhythmiagenic right ventricular cardiomyopathy probands and physical activity, athletes (defined as ≥4 hours of vigorous physical activity/ week) were found to have reduced bipolar function compared to non-athletes in arrhythmia-genic right ventricular cardiomyopathy patients and mutation-positive family members. S7.3-19 Many recommend limiting the intensity and duration of exercise <650 MET-Hr/year or 12.5 MET-Hr/week. S7.3-216.The proband of cardiomyopathy in the arrhythmia-genetic right ventricle is usually diagnosed based on the occurrence of clinical symptoms and the criteria of the arrhythmia-gene right ventricular cardiomyopathy working group, including: abnormalities in ECG, structural and functional changes in first-degree relatives of ventricles, arrhythmias and arrhythmia-genetic right ventricular cardiomyopathy. S7.3-6 The pathogenic genetic mutation was added to the task force's high criteria in 2010.S7.3-44 The proceeds of genetic testing with probands suspected to be arrhythmiagenic right ventricular cardiomyopathy are usually between 30% and 54%, and are up to 58% in patients with a strong family history of SCD in several members. S7.3-3,S7.3-25,S7.3-45 Negative genetic test for arrhythmia-genic right ventricular cardiomyopathy does not exclude disease, and a positive genetic test does not currently guide treatment. S7. In 3-22 Proband with a clinical diagnosis of arrhythmiagenic right ventricular cardiomyopathy, the identification of pathogenic mutations provides limited prognosis VT/ VFS7. S7. In a large multicenter study, the presence of positive mutations in probands was not associated with differences in mortality or heart transplantation. However, identifying the pathogenic mutation facilitates targeted genetic screening of this mutation in first-degree relatives, who can identify about 60-70 percent as gene-friendly, S7.3-1 as the highest with siblings and symptoms. S7.3-4 Screening for a specific mutation can identify some genetically positive family members before the disease occurs, while at the same time freeing others from the need for lifestyle changes and long-term monitoring. S7.3-2,S7.3-37.Syncope is reported in 16-39% of patients with arrhythmiagenic right-hand cardiomyopathy is often associated with physical activity, and some studies have been associated with a high risk of arrhythmia. S7. 3-10, S7. In patients with arrhythmiagenic correct ventricular cardiomyopathy and implanted ICDs, fainting was an important predictor of appropriate shocks in one study, S7.3-10, but not in other studies. S7.3-9,S7.3-12,S7.3-13,S7.3-43 Studies have not provided information on ventricular function or ECG abnormalities in patients with fainting, limiting its assessment as an independent risk factor. Fainting can be a sign of progression of a basic disease and must be included in the decision-making process of ICD implantation with the patient.8. Asymptomatic patients with arrhythmiagenic correct ventricular cardiomyopathy and no va or ventricular dysfunction is usually observed without any arrhythmia treatment other than beta-blocker therapy, a continuous temporary reassessment of the development of arrhythmias or ventricular dysfunction. S7. 3-46, S7. Atenolol was shown to reduce VA in one study. S7.3-15 Ambulatory monitoring and/or exercise testing may be performed to assess beta-generalisation >adequacy.9. VTs of arrhythmia-genic right ventricular cardiomyopathy are monomorphic, S7.3-12, while long-term monomorphic VT is uninsinsible in electrophysiological study in 55% of patients. S7.3-36 VT is usually associated with scarred, and the scar of subepardium is usually wider than that of endocardium. S7.3-27 In experienced centers, the use of epiardial mapping and ablations is associated with better results. S7.3-27,S7.3-28,S7.3-30,S7.3-31,S7.3-33 Important complications, including pericardial tamponadi, Myocardial infarction and death occur in 2.3 to 3.3 percent of ablation cases, S7.3-27-S7.3-29, highlighting the need for performance in centers with specialized expertise in epithalatic procedures. Ablation reduces the frequency of recurrent VT, although in 27-55% of patientsS7. Ablation of VT in patients with arrhythmiagenic right ventricular cardiomyopathy does not eliminate the need for ICD in appropriate candidates. The risk of vt relapse due to disease progression should be checked with patients when considering ablation. There are no randomised comparisons of antiarrhythmics to suppress recurrent VT. Beta-blockers, sotalol and amiodarone have been used. S7.3-15 In the observative series, sotalol muted inducing VT in 58% of patients with <10% of patients with relapse of arrhythmias in monitoring. S7.3-48 The effectiveness of different drugs seems to vary, so more studies needed.10.In arrhythmiagenic right ventricle cardiomyopathy, areas fibrofatty scar RV free wall create areas delayed chamber activation cause fractional tendencies after QRS, known as epsilon waves ekg(merkittävä kriteeri) ja signaalin keskimääräisen EKG:n myöhäiset mahdollisuudet (vähäinen kriteeri) rytmihäiriögeenisen oikean kammion kardiomyopatian diagnosoinnissa vuonna 2010. S7.3-6 Kun EKG QRS:n vakiokesto on ≤110 ms, epänormaalin signaalikeskiarvoisen EKG:n kriteereihin sisältyy mikä tahansa seuraavista: suodatettu QRS-kesto ≥114 ms, terminaalin kesto QRS <40 uv= exceeding= 37= ms,= or= a= root= mean= square= voltage= in= the= terminal= 40= ms= of= ≤20= uv.s7.3-6= abnormal= findings= on= signal= averaged= ecg= correlated= with= disease= severity= on= cardiac= mri,s7.3-35= and= increased= adverse= events= in= males.s7.3-34= in= an= assessment= of= the= diagnostic= use= of= testing= for= arrhythmogenic= right= ventricular= cardiomyopathy,= signal= averaged= ecg= was= of= greater= value= than= cardiac= mri= or= biopsy.s7.3-1411.the= value= of= an= electrophysiological= study= is= uncertain= in= asymptomatic= arrhythmogenic= right= ventricular= cardiomyopathy= patients= with= preserved= ventricular= function= in= predicting= subsequent= risk= for= scd.= studies= of= programmed= ventricular= stimulation= in= patients= with= definite= or= probable= arrhythmogenic= right= ventricular= cardiomyopathy= include= most= symptomatic= patients ,= making= recommendations= on= asymptomatic= patients= difficult.= electrophysiological= studies= induce= sustained= vt= in= approximately= 60%= of= patientss7.3-10,s7.3-36;= many= of= whom= have= had= prior= spontaneous= episodes= of= sustained= vt.= in= patients= with= primary= prevention= icds,= inducible= sustained= vt= did= not= predict= subsequent= appropriate= icd= shocks.s7.3-13= in= 1= study= including= symptomatic= patients,= patients= without= inducible= vt= were= less= likely= to= receive= appropriate= icd= shocks.s7.3-9= in= asymptomatic= patients= without= evidence= of= va= on= ambulatory= monitoring,= a= negative= electrophysiological= study= may= have= limited= value= in= decision-making= for= an= icd.7.4.= hypertrophic= cardiomyopathyfigure= 7.= prevention= of= scd= in= patients= with= hcm.colors= correspond= to= class= of= recommendation= in= table= 1.= see= section= 7.4= for= discussion.= *icd= candidacy= as= determined= by= functional= status,= life= expectancy,= or= patient= preference.= †risk= modifiers := age=></40> <30 y,= late= gadolinium= enhancement= on= cardiac= mri,= lvot= obstruction,= lv= aneurysm,= syncope=>5 y. VERENPAINE osoittaa verenpainetta; HCM, hypertrofinen kardiomyopatia; Hx, historia; ICD, implantoitava kardiovaskulaarinen defibrillaattori; LVOT, vasen kammion ulosvirtauskanava; LVWT, vasemman kammion seinämän paksuus; Magneettikuvaus, magneettikuvaus; NSVT, kestämätön kammiotakykardia; Sydänkohtaus, sydänperäinen äkkikuolema; ja VT, kammiotakykardia. Katso HCM:n määritelmä ACCF/AHA HCM-ohjeista. S7. 4- 36Sysifinen tukiteksti1. S7.4-1,S7.4-6 Niiden selection that are suitable candidates for implantation of ICD can be a difficult clinical decision due to the individuality of each patient and family,</30> due to,</30> definitions of risk factors and risk variables, sparse clinical data, relatively rare amount of both HCM and SCD in most clinical practices and possible complications to live with ICD. Table 8 lists the risk factors and risk converters associated with mixing in patients with HCM. The ICD risk deposit should be performed every 1 to 3 years in patients with HCM. There is increasing evidence that late gadolinium intensification with cardiac MRI is linked to the risk of sudden death and is included as a risk converter. S7.4-37–S7.4-39 LV aneurysm may be associated with a risk of persistent monomorphic VT. S7.4-40 Age is also an important consideration, since the risk of sudden death is higher <30 years old and low in patients with the original presentation after the age of 60, S7.4-5,S7.4-26.S7.4-412.HCM is the most common cause of SCD in individuals <40 years of age. S7.4-26 Persons who have survived a SCD, VF or permanent VT period leading to fainting or hemodynamic compromise require ICD implantation. S7.4-1, S7.4-6, S7.4-9, S7.4-10 Although the use of ICD is not evaluated in HCM patients who have survived SCD, 1 study reported that 54% of patients with secondary contraceptive ICD received appropriate ICD treatment at an average follow-up of 4.6 years. S7. Certain patients with HCM may be candidates for subcutaneous implantation with an implantable cardiovascular defibrillatorS7. However, more data are needed on this group, especially given their higher risk of overcooking the T-wave, which can increase the risk of inappropriate ICD shocks.3. Genetic advice should precede genetic testing of family members to better understand the usefulness and cost of testing. S7.4-18,S7.4-20,S7.4-43 Based on family history, clinical screening and pedigree analyses, the model of inheritance has been ensured to identify and manage relatives at risk. S7.4-13,S7.4-14,S7.4-18,S7.4-19,S7.4-43-S7.4-45 Since the family HCM is the prevailing disorder, the risk of the affected patient passing the disease on to each offspring is 50%. When a pathogenic mutation is identified in an index patient, the genetic condition of each family member can be easily verified. Relatives with sudden HCM have the same pathogenic HCM mutation as an index patient. Pathogenic mutations can also be identified in other relatives with unknown clinical status. These mutation-positive individuals shall be evaluated by physical examination, electrocardiogram, S7.4-11,S7.4-17 and echocardiographyS7.4-12,S7.4-16,S7.4-17, and if HCM is identified, these persons shall be assigned risk stratation. Genetic-positive subjects without evidence of HCM may have HCM's future development and benefit from the ongoing clinical assessment. S7.4-15,S7.4-46,S7.4-47 If the mutation that the proband brings is a bona fide mutation causing the disease, mutation-negative family members and their descendants are not at increased risk of developing HCM and do not need further assessment. However, such mutation-negative family members should have a sonar to ensure genotype and phenol type concordance.4.In in a study of 1,053 patients with clinically expressed HCM, 359 patients (34%) genotype positive for HCM-related mutation ≥1 HCM-related genes. S7.4-22 Whether probandi genetic testing results improve outcomes is uncertain, but identifying the mutation can help inform relatives about screening.5.Genetic counseling is important in HCM patients, and genetic screening of relatives is also important unless there are living first- or secondary relatives. Most HCM is caused by an autosomal dominant mutation in genes that encode sarkomerek proteins or sarcomer-related proteins. Pathogenic sarkomiprotein gene mutation patients with HCM recognise the risk of LV dysfunction and adverse reactions, regardless of the myofilament involved. S7.4-13-S7.4-15,S7.4-18,S7.4-19,S7.4-22 A single mutation in one of the alleles (or copies) of gene 2 is sufficient to cause HCM; However, 5% of HCM patients ≥2 mutations in the same gene or genes, which may indicate poor results. S7.4-13,S7.4-34,S7.4-48 When genetic testing reveals a mutation in an index patient, detecting the genetic condition from first- and secondary relatives can predict the risk of developing HCM. S7.4-14,S7.4-49 Relatives with exaggerated HCM have the same pathogenic HCM mutation as an index patient.6.Several studies have described an independent relationship between hypertrophy and SCD, when hypertrophy is ≥30 mm.S7.4-2,S 7.4-3,S7.4-23,S7.4-24 The risk does not suddenly increase in patients with wall thickness of ≥30 mm, but rather it increases linearlyS7.4-24 and appears to be more predictable in younger patients. A young adult with a hypertrophy approaching 30 mm may have a similar or higher risk of SCD than an older patient with a wall thickness of up to ≥30 mm.S7.4-23,S7.4-50 Patients with HCM have an increased risk of SCD if they have a first-degree relative who has experienced SCD presumably caused by HCM. Family history seems to predict SCD independently, although supportive studies are small and observational. S7.4-25,S7.4-26 Syncope can be neurally or medication-related, as well as due to VA, and requires careful evaluation before being considered as a risk factor for SCD. S7.4-8, S7.4-26 or a syncope thought to be non-neural was associated with SCD scd only when it occurred in the last 6 months, but not if the largest >5 years earlier. S7.4-87.Although durable VT is clearly associated with SCD, NSVT data is less durable. Most studies do not support NSVT as an independent mixed seglostatic risk factor in patients with HCM, S7. 4-26, S7. 4-27, but the risk increases if risk converters occur, especially among patients <30 years of age. S7. Up to a third of HCM patients have abnormal blood pressure response during exercise tests (defined to varying degrees of either a 20 mmHg decrease in blood pressure or an increase in systolic blood pressure of at least 20 mmHg during the effort). S7.4-28,S7.4-29 This finding is assumed to be a risk factor for SCD; However, it is unclear how this relates to the blockage of the dynamic LV outflow channel, which occurs during exertion, a hemodynamic state that can be easily modified by medication or mechanical procedures. The importance of abnormal blood pressure response in exercise predicting SCD increases in the presence of risk converters (Table 8).8.Most studies have found that NSVT alone has a low positive predictive value for SCDS7.4-2,S7.4-26,S7.4-27; Therefore, the use of ICD is more appropriate if risk transformers are also present. Abnormal blood pressure response to exercise has also been associated with the risk of sudden death, S7.4-5, S7.4-28,S7.4-29, but it is unclear how this is related to the blockage of the dynamic LV outflow channel, which occurs with effort that is often treatable. The importance of abnormal blood pressure response in exercise to predict SCD increases when risk converters occur (Table 8).9. S7. Amiodaron has been associated with better survival time observational studies and is an option for patients for whom ICD is not possible due to limited survival expectations or patient preferences. S7.4-30, S7.4-3110.Approximately one third of successive HCM patients undergoing electrophysiological examination have polymorphic VT or VF due to programmed ventricular stimulation, but the results of programmed stimulation do not predict scd risk. Programmed ventricular stimulation in patients with HCM has low prognosis and a non-ne ne necumstatic risk of complications. S7.4-32, S7.4-33, S7.4-51 Electrophysiological studies may help clarify the diagnosis of extensive complex takycardia or supraventricular takycardia or bundle crotch reincarnation guide therapy.11.SCD may group into certain HCM families, and the possibility that certain sarcopmere mutations may pose an SCD risk is hypothetical. However, in selected patients with HCMS7.4-34,S7.4-35, it is not possible to kliinisesti hyödyllinen suhde genotyypin ja scd-riskin välillä. Joissakin tapauksissa haittatapahtumien (ja niihin liittyvien sekundääristen sekundääristen riskitekijöiden esiintyvyys) esiintyvyys oli pienempi potilailla, joilla oli alun perin pahanlaatuisia mutaatioita, kuin potilailla, joilla oli hyvänlaatuisia mutaatioita. S7.4-34,S7.4-35 Valitsemattomien peräkkäisten avohoitopotilaiden sarjojen tiedot viittaavat siihen, että useimmat mutaatiot ovat uusia ja rajoittuvat tiettyihin perheisiin. S7.4-34,S7.4-35 Näin ollen rutiinimutaatioseulonnalla ei näyttäisi olevan juurikaan ennustearvoa HCM:ssä. S7. 4- 52 Lyhytaikainen äkkikuoleman riski potilailla, jotka ovat genotyyppipositiivinen mutta joilla ei ole muita taudin ilmenemismuotoja, näyttää olevan pieni. S7.4-53 Siksi ICD: tä ei ole osoitettu näille henkilöille. Taulukko 8. Tärkeimmät kliiniset ominaisuudet, jotka liittyvät lisääntyneeseen sekumpleellisen sekoityksen riskiin potilailla, joilla on HCMStablished-riskitekijöitä* Selviytyminen sydämenpysähdyksestä VT: n tai VF S7. 4- 1: n, S7. 4- 5, S7. 4- 6: n spontaaneilla jatkuvilla VT: llä, joka aiheuttaa pyörtymistä tai hemodynaamista kompromissia S7.4-1, S7.4-4 5,S7.4-6 HCM S7.4-25,S7.4-26 LV-seinämän paksuuteen liittyvä scd:n perhehistoria ≥30 mm S7.4-2,S7.4-3,S7.4-23,S7.4-24 Tutkimaton 6 mo S7.4-8,S7.4-26 NSVT ≥3 lyö S7.4-2,S7.4-26,S7.4-27 Epänormaali verenpainevaste harjoituksen aikana†S7.4-5,S7.4-4-2728,S7.4-29Potential risk modifierit‡ <30 y= s7.4-5,s7.4-26 delayed= hyperenhancement= on= cardiac= mri= s7.4-37–s7.4-39,s7.4-54 lvot= obstruction= s7.4-2,s7.4-4 syncope=>5 y ago S7.4-8,S7.4-26High-risk osasarjat§ LV <50% S7.4-527.5. MyocarditisRecommendation-Specific Supportive Text1.Myocarditis is an inflammatory process often related to infection. S7.5-1,S7.5-5–S7.5-9 When patients are treated in centers with the availability of mechanical hemodynamic support procedures, cardiac catheterization, endomyocardial biopsy, advanced cardiac imaging procedures, and arrhythmia management including ICD implantation, outcomes appear improved. S7.5-1 The acute course of myocarditis varies ranging from an asymptomatic finding of transient ST-T changes noted on ECG to cardiogenic shock and recurrent VA. S7.5-10–S7.5-12 Acute management is largely supportive and can rapidly advance to requiring mechanical support. S7.5-13,S7.5-14 Cardiac arrhythmias range from conduction abnormalities to life-threatening VT and VF. S7.5-15–S7.5-17 Arrhythmias may require antiarrhythmic medications and/or device therapy. S7.5-18 Giant cell myocarditis is fairly uncommon, but it is of particular importance because it typically affects young individuals and is usually fatal if untreated. S7.5-2–S7.5-4,S7.5-19 VT may require antiarrhythmic medications such as amiodarone and/or an ICD that in some instances can be used as a bridge to more advanced HF therapies such as LVAD transplant. Myocarditis and SCD have been reported with HIV infection. S7.5-20,S7.5-21 Systemic s7.4-527.5.= myocarditisrecommendation-specific= supportive= text1.myocarditis= is= an= inflammatory= process= often= related= to= infection.s7.5-1,s7.5-5–s7.5-9= when= patients= are= treated= in= centers= with= the= availability= of= mechanical= hemodynamic= support= procedures,= cardiac= catheterization,= endomyocardial= biopsy,= advanced= cardiac= imaging= procedures,= and= arrhythmia= management= including= icd= implantation,= outcomes= appear= improved.s7.5-1= the= acute= course= of= myocarditis= varies= ranging= from= an= asymptomatic= finding= of= transient= st-t= changes= noted= on= ecg= to= cardiogenic= shock= and= recurrent= va.s7.5-10–s7.5-12= acute= management= is= largely= supportive= and= can= rapidly= advance= to= requiring= mechanical= support.s7.5-13,s7.5-14= cardiac= arrhythmias= range= from= conduction= abnormalities= to= life-threatening= vt= and= vf.s7.5-15–s7.5-17= arrhythmias= may= require= antiarrhythmic= medications= and/or= device= therapy.s7.5-18= giant= cell= myocarditis= is= fairly= uncommon,= but= it= is= of= particular= importance= because= it= typically= affects= young= individuals= and= is= usually= fatal= if= untreated.s7.5-2–s7.5-4 ,s7.5-19= vt= may= require= antiarrhythmic= medications= such= as= amiodarone= and/or= an= icd= that= in= some= instances= can= be= used= as= a= bridge= to= more= advanced= hf= therapies= such= as= lvad= or= transplant.= myocarditis= and= scd= have= been= reported= with= hiv= infection.s7.5-20,s7.5-21= systemic=></50% S7.4-527.5. MyocarditisRecommendation-Specific Supportive Text1.Myocarditis is an inflammatory process often related to infection. S7.5-1,S7.5-5–S7.5-9 When patients are treated in centers with the availability of mechanical hemodynamic support procedures, cardiac catheterization, endomyocardial biopsy, advanced cardiac imaging procedures, and arrhythmia management including ICD implantation, outcomes appear improved. S7.5-1 The acute course of myocarditis varies ranging from an asymptomatic finding of transient ST-T changes noted on ECG to cardiogenic shock and recurrent VA. S7.5-10–S7.5-12 Acute management is largely supportive and can rapidly advance to requiring mechanical support. S7.5-13,S7.5-14 Cardiac arrhythmias range from conduction abnormalities to life-threatening VT and VF. S7.5-15–S7.5-17 Arrhythmias may require antiarrhythmic medications and/or device therapy. S7.5-18 Giant cell myocarditis is fairly uncommon, but it is of particular importance because it typically affects young individuals and is usually fatal if untreated. S7.5-2–S7.5-4,S7.5-19 VT may require antiarrhythmic medications such as amiodarone and/or an ICD that in some instances can be used as a bridge to more advanced HF therapies such as LVAD or transplant. Myocarditis and SCD have been reported with infection. S7.5-20,S7.5-21 Systemic > aneurysm S7.4-40,S7.4-55,S7.4-56 LVEF</30> LVEF</30> erythematous can cause cardiac and cordiorditis, but only rarely VT or VF. S7. 5-8, S7. In patients with Chagas disease, acute myocarditis is rare, but more than a third of patients with the disease develop late myocarditis with progressive HF. Amiodarone appears to be effective in treating VA. S7. Icd, S7.5-24 and radio frequency catheter ablation have been successfully used to control repetitive VA on some papatietts in the late stages of 5-23 Myocardies. S7.5-252.Giant cell cardiac myocardia is quite rare, but it is especially important because it typically affects young individuals and is usually fatal if untreated. The diagnosis is confirmed by an endomyocardial biopsy. Patients may develop a heart block that requires temporary or permanent pacemakers. ICD and antiarrhythmia drugs such as amiodarone are often used in the acute phase to treat VT or VF and reduce the risk of SCD. S7.5-2–S7.5-4,S7.5-19,S7.5-26–S7.5-287.6. Heart SarcoidosisFigure 8. cardiac sarkoidosis patients. The colors correspond to the recommended category in Table 1. See section 7.6. *ICD nomination according to functional status, life expectancy or patient preference. † repeated monomorphic VT is shown in Figure 2. CEP stands for electrophysiological; GDMT, instructional-controlled management and care; ICD, implantable cardiovascular defibrillator; LVEF, left ventricular ejection part; MRI, MRI; PET, positron emission tomography; SCA, sudden cardiac arrest; Heart attack, sudden cardiac death; VA, ventricular arrhythmias; and VT, chamber tachycardia. Support text per recommendation1. Sarcoidosis is a systemic granulomatic disease whose cause is unknown. Lung participation is most common, but any organ can make a difference. Cardiac inclusion, diagnosed with cardiac MRI or positronic emission tomography (PET), has been reported in up to 55% of patients with extra cardiac disease, while most patients diagnosed with cardiac sarcoidosis were diagnosed with one report. S7. Cardiac expressions include lead disorders, VA and depressed ventricular function with or without HF, and these contribute significantly to higher mortality rates in cardiac sarcoidosis than sarcoidosis without cardiac participation. S7. In a 6-2 25-year-old study in which 110 patients in Finland with heart sarcoidosis with HF during the presentation had a significant LV dysfunction in diagnosis (LVEF <35%), and isolated cardiac sarcoidosis predicted adverse reactions. S7. Va 6-1 VA may also occur in patients with relatively normal LV function, some of whom have RV involvement, which may arrhythmia gene correct ventricular cardiomyopathy. Multiple reports of patients with cardiac sarcoidosis and ICDs implanted with either primary or secondary secondary scd indicates a high number of appropriate ICD treatments, S7.6-3-S7.6-5, which support the use of ICD for primary and secondary prevention of mixed diseases, according to indications used for other cardiomyopathy. The frequency of presiding deviations often also requires a brady-carding device.2.Patients with cardiac sarcoidosis may have VA and SCD therapy, even if LVEF is normal, and there is no well defined identification of patients at risk for treatment, despite the survival of LV. Several studies have assessed the role of cardiac MRI in VA and SCD projections. Meta-analysis S7.6-6 involving 760 patients in 10 studies found that late gadolinium intension was associated with increased overall mortality and more VA compared to those without late gadolinium improvement. Applicability is limited by the precise quantification of the late gadolinium-increasing burden, which may allow for a more nuanced risk-spread. According to some studies, there is a threshold effect, and extensive participation of LV and motorhome is a particularly risky feature. S7. 6-7, S7. However, a late gadolinium enhancement may occur even if LVEF is >50% and is associated with a 4.9% or VT risk per year compared to 0.24% per year when there was no improvement in late gadolinium in one observational study. S7.6-7 PET is also increasingly used to assess inflammation and scar, but data are limited. In one report, the occurrence of inflammation and the involvement of a campervan in PET scanning were associated with an increased risk of death or, for example, an increased risk of death. S7.6-10 Electrophysiological studies in 76 patients with signs of cardiac sarcoids found that 11% had inductive VT. The median 5-year follow-up in 75% of patients with inductive VT had spontaneous VT or death compared to 1.5% of patients without inductive VT. S7.6-183.Electrophysiological research has been proposed as a potential tool for the stratification of VA and SCD risks in patients who had demonstrable evidence of cardiac sarcoidosis based on imaging studies or biopsies, but do not have documented arrhythmias or arrhythmia symptoms and do not meet the primary prevention requirements for ICD implantation. One study evaluated 76 patients with MRI scans of PET or heart, documented cardiac sarcoidosis and underwent an electrophysiological examination. S7.6-12 Eight (11%) were uninsinsible with durable ICAIs and received ICDs, while the rest did not receive ICD because they were not induced. LVEF was lower in patients with inductive renal impairment (36.4+ 4.2% vs. 55.8 + 1.5%). Five-year median for follow-up of the eight patients in the group with inductive renal impairment, were in VA treatment or died when the corresponding figure in the negative group was one. S7.6-12 An important caveat is that it is unclear whether electrophysiological research is more predictable than just LVEF, since inductive inductive with LVEF. In addition, the mean LVEF of inducing patients in this study continued to decrease during the follow-up period. S7.6-124.In and LV dysfunction, lead disorders, including cardiac blockage, may also be a common manifestation of cardiac sarcoidosis. Patients with documented VA and LV dysfunction are at increased risk of cardiac events such as cardiac death. One study compared the results in 22 patients with high atrioventricular lobes as the original expression of cardiac sarcoidosis, 31 patients who originally had VT and/ or HF. after a median score of 34 months in patients with a heart lobe had less HF hospitalisation, but fatal cardiac events, including persistent TAs, were similar to VT and/ or HF, suggesting that the risk of fatal cardiac events is high regardless of the initial clinical presentation. S7. 6-13 In the same study, steroid administration led to clinical healing, and some patients recovered from conductivity, but steroid effectiveness was not common and did not appear to protect against harmful cardiac events. S7.6-135.Several studies have attempted to assess the role of immunosuppression in reducing VA in patients with cardiac sarcoidosis, but the results have been inconsistent. In addition, immunosuppressive therapy (usually glucocorticoids) in several patients has worsened, including an electrical storm that develops in some patients within 12 months of initiation of treatment. S7.6-15 One study reported a reduction in the burden of arrhythmia with steroid therapy, but only when administered in the early stages of the disease; those with advanced LV dysfunction experienced no benefit. S7. 6-16 Systematic combined therapy was successful in 63% of patients in a series in which medical treatment included both steroids and arrhythmias, after which radio frequency catheter ablation if necessary. S7. Immunosuppressive therapy may serve a two-way purpose in addition to arrhythmias, as it may help stabilize disease progression and prevent further deterioration of LV function, although this has not yet been demonstrated in the RCT. S7.6-1 PET scanning to assess inflammation and scar is also increasingly used in sarcoidosis, but data supporting its use to control arrhythmias are limited.7.6.1. Other infiltrative cardiomyopathiesFiltive cardiomyopathies are a heterogeneous group of uncommon systemic diseases associated with heart participation. In some infiltral cardiomyopathy, such as Fabry's disease, TTas are rare. Some, such as hemochromatosis, are highly treatable, especially when diagnosed early. In all cases: cardiac arrhythmias. Most studies of infiltroving cardiomyopathy and arrhythmias are small and observational,S7. See sarcoidosis section 7. Until recently, cardiac amyloidosis was associated with a very poor prognosis in patients who eventually bowed to progressive HF. S7.6.1-2 This perception changes due to advances in medical treatment of amyloidosis in the light chain, which has led to better results. However, S7.6.1-3 Decisions need to be identified because the data are still too limited to allow for formal recommendations, as published reports on the effectiveness of ICD in amyloidosis are small, observational and monitored to a limited extent. S7. 6.1-4 Is the placement of ICD in the light chain more beneficial than transtereitin-related amyloidosis, as most studies involved patients with predominantly amyloid-using photo-chain amyloid, whose exposure value may be higher and the prognosis generally worse. It is uncertain whether ICDs are effective in primary prevention of mixing treatments, but many deaths in cardiac amyloidosis patients do not appear to be preventable ICD. Heart failure7.7.1. HF with reduced Ejection fractionSynopsis Patients with HFrEF have an increased risk of VA and SCD. The risk increases regardless of HFrEF etiology. S7. 7.1-6 SCD accounts for a higher proportion of deaths in patients with milder HF symptoms and a lower proportion in patients with moderate/ severe HF symptoms. The reported prevalence of S7.7.1-7 ScD varies depending on the definition used and the population studied. While many deaths classified as sudden are indeed caused by deadly VA, others may be due to bradyarrhymia, pulse-free electrical activity and sudden hemodynamic deterioration. S7.7.1-7-S7.7.1-9 Medical treatment with neurohormonal agents reduces the risk of SCD by reducing both the prevalence of VA and disease progression. S7.7.1-7,S7.7.1-10-S7.7.1-12 Despite HFrEF GDMT, some patients are still at risk of scd, and ICD can be beneficial. See ICD indications 7. CrT has also been shown to reduce the inaducence of sepacstatic sinuses in appropriate patients. S7. The pathophysiology of scd 7.1-13SF is complex due to the interactions of both functional and structural changes in HFrEF patients, increasing susceptibility to sepelt. S7.7.1-14 Although many risk factors are common among HFrEF patients, the reason scd strikes a particular person is generally unknown; however, some individuals may have a genetic predisedness. S7.7.1-15 Varying degrees of myocardial fibrosis, neurohormonal activation and increased wall strain alter electrophysiological connection, ion flow (electrical renovation) and calcium processing, which are likely to contribute to the development of deadly VA. S7.7.1-16 Factors affecting the heart include electrolyte deviations, sympathetic activation, hemodynamic stress and hypoxia associated with volume transfer and diuretic use. Support text per recommendation1. Many patients with advanced HF marked for heart transplantation would otherwise not be eligible for ICD due to the severity of the disease, including NYHA Class IV status and/ or the use of an inotropic infusion. Although there are no randomized data on the use of ICD in this patient population, observations and extensive registry studies in patients waiting for a heart transplant suggest better survival in patients with ICD.S7.7.1-1, S7. 7.1-4, S7. 7.1-5 One alternative to ICD in this patient population is a wearable cardiooverter defibrillator. S7.7.1-2, S7.7.1-3 The recommendation in this section is relevant for patients without ICD who have a plan to discharge the patient to wait for a heart transplant, and not, for example, for patients who remain hospitalized without the intention of repatriating a discharge pre-transplant. In patients with LVAD, the decision to give ICD is generally independent, regardless of whether they expect a heart transplant, but rather the indication in these patients is usually based on the need to treat renal impairment. S7. 7. 1- 177. 7. 2. HF With a preserved ejection fraction approximately half of HF patients have survived LVEF. S7. 7.2-1 These patients tend to be older and have more additional receives than HFrEF patients. Although the proportion of secondary mixing levels is lower in HF patients with maintained ejection fraction (HFpEF) than in patients with HFrEF, S7, 7.7.2-2, almost a quarter of all deaths in HFpEF patients are sudden. The challenge of scd prevention in HFPEF patients is to identify which patients are at sufficient risk of benefiting from preventive treatments. Studies studying non-invasive second-sectic risk factors in HFpEF patients do not identify consistent factors, with the exception of sechemical heart disease. S7.7.2-2,S7.7.2-6 Therefore, there is no approved non-invasive test to identify high-risk patients with HFpEF. Invasive risk of layering with electrophysiological research shows promise in this population. S7.7.2-7,S7.7.2-8 This topic is currently being studied in the PRESERVE-EF (Risk Stratification in Patients With Preserved Ejection Fraction) study (NCT02124018). The Writing Committee carefully considered whether to include a recommendation related to an electrophysiological study in patients with HFpEF and ischemical heart disease. However, the evidence was considered insufficient to Support. However, the benefits and disadvantages of electrophysiological research may reasonably be considered in certain patients with HFpEF and ischemical heart disease who: symptoms indicating VA.7.7.3. Left Chamber Assist Device Recommendation-specific support text1. Patients with LVAD are at high risk of VA assistance, especially in patients with a previous arrhythmias. S7.7.3-2-S7.7.3-4 The increased VA risk may be due to myocardial infarction irritation due to LVAD inflow cannula insertion, LVAD suction effect LV compression, inotropic support often needed by some patients, and re-polarization changes that may occur after LVAD placement. While some patients with LVAD tolerate VT/VF, others experience a decrease in flow because the campervan is not supported; fainting and hypoperfusion can lead to. ICD can allow the va to be quickly discontinued before significant hemodynamic consequences. Data on ICDs in patients with LVAD are from a series of observations. A systematic review of six observational trials found that within 7 months, 26% of LVAD patients had died. S7. Mortality was lower in patients with a previous ICD (16% vs. 32%), suggesting a 39% reduction in relative risk in the overall mortality analysis. S7. 7.3-1 Patients with a previous pre-LVAD ≥ 10 times the risk of post-LVAD stabilization. S7.7.3-2-S7.7.3-4 Many preliminary studies that demonstrated ICD benefits used older pulsatil LVAD devices. S7.7.3-2,S7.7.3-5 Studies on the use of ICD with new, continuous flow LVAD connections have shown inconsistent benefits. S7.7.3-1,S7.7.3-4,S7.7.3-6,S7.7.3-7 It should be noted that approximately 2 out of 10 LVAD infections develop LVAD infection in the first year. S7.7.3-8,S7.7.3-97.7.4. Use of ICD after heart transplantationSitomission-specific support text1. The development of the disease in the transferred heart puts some patients at increased risk of mixing, which is 10% to 35% in observational studies. S7.7.4-4,S7.7.4-5 Both rejection and reduced LVEF predict SCD. The mechanisms behind SCD in cardiac transplant patients include damage to the system of leadership and VA infection itself during coronary vascular dislopathy or acute rejection. Several small series of cases that would monitor a suitable end to ICD suggest that ICD may be beneficial for selected patients, especially those with severe allograft vasculopathy, unexplained fainting, previously SCA and severe LV dysfunction. S7.7.4-1–S7.7.4-3 In addition, a patient with severe seograft vasculopathy who is being considered for re-transplantation may be suitable for ICD as a bridge device. The secondary contraceptive subjects of heart transplant patients are identical to those of other patients.7. Neuromuscular disordersSynopsis Muscle dystrophies are a group of hereditary diseases that the muscles of the skeleton and heart. Some mainly act as NICM (e.g. NICM). Duchenne, Becker and limb waist types 2C, 2F and 2I), 2I), others occur primarily as degeneration of the system of management, with a variable link to cardiomyopathy (e.g. myotrene dystrophy types 1 and 2, Emery-Dreifuss, waist type 1B of the limbs; summarised in Table 9). S7.8-15 Since SCD can be caused by either VA or bradyarrhythmia due to the rapid and unpredictable progression of the management system disease, the challenge for the clinician is to identify patients who would benefit from preventive pacemaker or ICD implantation. Patients with muscular dystrophy with symptoms of arrhythmia should be extremely concerned. S7.8-15 The current guide focuses on VA guidelines and implantation of ICD. The permanent pacemaker is discussed in another ACC/AHA/HRS guide. S7.8-16Table 9. Neuromuscular disorders associated with heart diseaseMuscular dystrophyInheritanceGene/protein-influencedPrimary Cardiac Pathology Disorders Involved Deaths Related to sudden death? DuchenneX-linkitetty ressiiviNenDystrophinNICM>90%Respiratory, HFYes, epävarma etiologiaBeckerX-linkitetty ressiiviNenDystrophinNICM60%–75%HF, hengitystieysit, epävarma etiologiaLimb-girdle tyyppi 1BAutosomaalinen hallitsevaLamin A/CConduction system disease ja NICM>90%Äkillinen, HFYesLimb-girdle tyyppi 2C-2FAutosomaalinen resessiiviSarcoglycanNICM<25%Respiratory, hfuncertainlimb-girdle= type= 2iautosomal= recessivefukutin-related= proteinnicm20%–80%respiratory,= hfuncertainmyotonic= type= 1autosomal= dominantctg= repeat= expansionconduction= system= disease= and= nicm60%–80%respiratory,= sudden,= hf30%= of= deaths,= uncertain= bradycardia= versus= tachycardiamyotonic= type= 2autosomal= dominantcctg= repeat= expansionconduction= system= disease10%–25%normal= causesreportedemery-dreifussx-linked= and= autosomal= dominant= or= recessiveemerin,= lamin= a/cconduction= system= disease= and= nicm=>90%Äkillinen, HFYesFacioscapulohumeralAutosomal dominantD4Z4 toistuva supistusPohjasti johtavuussairaus5%–15%Normaalit syyt, hengityselimet harvoin Ei raportoituSuosituskohtainen tuki Text1.In yleinen, ICD: n käyttöaiheiden lihasdystrofiaa sairastavilla potilailla tulee noudattaa tavallisia ICD- suosituksia NICM- potilaille (ks. kohta 7. 2.1 secondary contraception and section 7. 2.2 scd primary prevention with NICM). A high suspicion of bundle crotch reentrant tachycardia is justified in patients with myoton dystrophy with extensive QRS-complex tachycardia or tachycardia-related symptoms. S7.8-22.In muscular dystrophies of the waist of emery-Dreifuss and type 1B limbs associated with Lamin A/ C mutations, SCD is approximately one third of all deaths. S7. Observational studies show a significant amount of appropriate ICD therapy in patients with cardiac conductivity disorders who are gene positive for lamin's A/ C mutation, even if LV remains functioning. Havainnoivassa tutkimuksessa </25%Respiratory,> </25%Respiratory,> 38% had isolated skeletal muscle-acting effects, but included patients with cordivity defects and other risk factors (including PR time >240 ms, left bundle arm block, NSVT or bradykardia requiring permanent pacemaker) life-threatening TA's were relatively common; In 52% of patients receiving appropriate ICD therapy, including approximately 40% of patients with LVEF≥45%. S7. The study involving lamin's A/ C mutation, in which approximately 21% had skeletal muscular dystrophyphenotype, SCD and appropriate ICD therapy, was associated with NSVT, LVEF and <45%, male sexual and Lamin A/ C nonmissense mutations. S7. 8-4 These observed studies support the use of ICD when a pacesing topic occurs and probably also when there is evidence of cardiac progressive participation such as cardiac conduction disorders, NSVT or decreased LVEF. S7. There is little information on the rare form of 8-8 X-linked resessive Emery-Dreifuss muscular dystrophy (associated with the Emerin gene mutation), but arrhythmias may occur less frequently than lamin's A/ C mutations. S7.8-153.Cardiac inclusion may occur in several neuromuscular dystrophy (Table 9). For the determination of cardiac inclusion, 12-lead ECG and sonar are important in the preliminary clinical assessment independent of the state of symptoms. In general, the greater the cardiac participation, including evidence of disease, ventricular dysfunction and atrial rhythm of its distal conductivity, the more likely the VA is to occur. The preliminary evaluation of myoton dystrophy patients includes ambulatory monitoring. In asymptomatic patients, some experts advocate annual monitoring during the hidden phase of the disease, and the annual 12-lead ECG sifts through derivative exemptions. However, the optimal frequency of electrocardiogram screening is unknown. S7. 8-18 When cardiac participation is present for either leadership, atrial rhythm or ventricular dysfunction, low threshold to study symptoms or electrocardiogram findings, the clinician is determined by the need for pacemaker implantation, invasive electrophysiological studies or ICD implantation is optimal.4.Up to a third of deaths in myoton dystrophy patients are sudden. S7. Although it is commonly caused by the conduction block and asystole, the interference phase due to VT/ VF has been identified in patients with effective permanent pacemaker treatments, and spontaneous vad therapy has been documented in some patients. S7.8-13,S7.8-19 The risk of a blender in pacemaker patients suggests that the pacemaker may be favored. However, these patients also have a high risk of respiratory failure as a competing cause of death. Therefore, patients with severe skeletal muscle, pacemaker or ICD may not improve outcomes. S7.8-15 Common ICD:n tai tahtiterapian valitsemiseen on on Compared to type 1 myotonia patients, patients with type 2 myoton dystrophy are not well studied, but they may also benefit from the same approach.7. Planting cardiac chickenpathySynopsis ICD in asymptomatic low-risk patients with cardiac channelopathy with a positive family history of second-by-second treatment because the published data do not support the only indication. S7.9-13-S7.9-18Salute-specific support text1. Clinical screening of first-degree relatives of patients with hereditary arrhythmia symptoms is crucial for identifying affected family members. Targeted screening of genotypical positive patients with long QT syndrome, caecolamine-related polymorphic polymorphic tacycardia and Bruganda syndrome may identify individuals at risk of these adverse reactions. S7.9-2 to S7.9-5 Screening ECGs may not be sufficient for diagnosis because patients with caemomorphic polymorphic polymorphic tachycardia have normal rest ECG and up to 25% of genotypical positive patients with long QT syndrome, QTc intervals ≤440 ms. S7.9-2 In young patients with long QT symptoms and caecolamine polymorphic chamber tachycardia, S7.9-2,S7.9-19-S7.9-22 screening, it is especially important to guide treatment and introduce preventive measures, including avoidance of possible provocative drugs (www.crediblemeds.org). S7. Since up to 15% of mutations associated with previously caecolamine-related polymorphic polymorphic ventricular tachycardia do not appear to cause disease, S7. Caution is recommended to avoid unnecessary treatment or restriction of sports in phenotype negative caesophamine polymorphic chamber tachycardiation positive individuals. In particular, some patients may wish to be unused in genetic testing, so genetic advice should focus on this issue.2.Patients with cardiac channelopathy (i.e. long QT syndrome, caecolamine-related polymorphic polymorphic tachycardia, Brugida syndrome, early reolarization syndrome and short QT syndrome) and previous SCA have a significantly higher risk for later SCA or SCD. Implantation of S7.9-7-S7.9-13,S7.9-25-S7.9-28 ICD reduces the risk of death in at-risk patients. S7, 9-9, S7, 9-29-S7.9-31 Suitable ICD treatment VF/ rapid VT is reported in 8-33% of channelopathy patients, while inappropriate shocks and device complications are reported in 8-35%. S7.9-10,S7.9-29,S7.9-30,S7.9-32-S7.9-36 Simultaneous beta-catches in long QT syndrome and caecolamine-rgic polymorphic ventricular tachycardia patients are required to minimise inappropriate shocks, equipment programming and appropriate lead selection. Ventricular anxiety without ICD implantation was associated with significant SCA or SCD in long QT syndrome patients. S7. 9-37– S7. In selected LQT1 patients with SCA without beta-general therapy, beta-general therapy is offered as an alternative to ICD implantation in patients who refuse ICD. S7. 9- 407. Specific cardiac tract syndromes7.9.1.1. Congenital long QT syndromeFigure 9. Prevention of SCD in patients with long QT syndrome. The colors correspond to the recommended category in Table 1. See section 7.9.1.1. *ICD nomination according to functional status, life expectancy or patient preference. †Real patients with LQTS include QTc >500 ms, genotypes LQT2 and LQT3, women with the genotype LQT2, <40 years= of= age,= onset= of= symptoms= at=></40> <10 years= of= age,= and= patients= with= recurrent= syncope.= icd= indicates= implantable= cardioverter-defibrillator;= l qts,= long= qt= syndrome;= vt,= ventricular= tachycardia.figure= 10.= long= qt= syndrome= type= 1.figure= 11.= long= qt= syndrome= type= 2.figure= 12.= long= qt= syndrome= type= 3.recommendation-specific= supportive= text1.beta= blockers= reduce= adverse= cardiac= events= for= long= qt= syndrome= type= 1= (figure = 10)= (=>95%), long QT syndrome type 2 (Figure 11) (>75%), and women with long QT syndrome type 3 (Figure 12) >60%. S7.9.1.1-1–S7.9.1.1-5 Beta predators are effective in men, with long QT syndrome type 3S7.9.1.1-3,S7.9.1.1-35,S7.9.1.1-36, but in selected patients beta workshops can protect against SCA. S7.9.1.1-36,S7.9.1.1-37 Several observational studies have reported effectiveness in reducing the risks of long QT syndrome with propranolol, atenolol and nadolol with appropriate teaching, S7.9.1.1-26,S7.9.1.1-28,S7.9.1.1-38–S7.9.1.1-40, while metoprolol appears ineffective. S7.9.1.1-41 RCTs to estimate the relative efficacy of certain beta-catchers are not available, although many centres prefer the use of nadolol. In a type 1 long study with QT syndrome 1, the risk of atenolol for VA was reduced, while nadolol was not associated with a risk reduction. S7. 9. 1. The efficacy of long QT syndrome type 2 nadolol was reported to be better. Patients receiving S7.9.1.1-1,S7.9.1.1-2 Beta-2 Patients receiving beta sacr/2 should receive continuous monitoring to assess changes in QTc over time and to assess the adequacy of the beta blockade. S7.9.1.1-26,S7.9.1.1-282.High-risk patients with long QT syndrome include QTc >500 ms, genotypes long QT syndrome type 2 and long QT syndrome type 3, women with genotype long QT syndrome type <40 years= of= age,= onset= of= symptoms= at=></40> <10 years of age, and patients with prior cardiac arrest or recurrent syncope. S7.9.1.1-3,S7.9.1.1-8,S7.9.1.1-11,S7.9.1.1-30,S7.9.1.1-38 Women with long QT syndrome type 2 are at a higher risk of postpartum arrest/SCDS7.9.1.1-42,S7.9.1.1-43 and should receive prepregnancy counseling. Patients with long QT syndrome and recurrent syncope while receiving a years= of= age,= and= patients= with= prior= cardiac= arrest= or= recurrent= syncope.s7.9.1.1-3,s7.9.1.1-8,s7.9.1.1-11,s7.9.1.1-30,s7.9.1.1-38= women= with= long= qt= syndrome= type= 2= are= at= a= higher= risk= of= postpartum= cardiac= arrest/scds7.9.1.1-42,s7.9.1.1-43= and= should= receive= prepregnancy= counseling.= patients= with= long= qt= syndrome= and= recurrent= syncope= while= receiving= a=></10 years of age, and patients with prior cardiac arrest or recurrent syncope. S7.9.1.1-3,S7.9.1.1-8,S7.9.1.1-11,S7.9.1.1-30,S7.9.1.1-38 Women with long QT syndrome type 2 are at a higher risk of postpartum cardiac arrest/SCDS7.9.1.1-42,S7.9.1.1-43 and should receive prepregnancy counseling. Patients with long QT syndrome and recurrent syncope while receiving a > 2,</10> 2,</10> Sabbath has an increased risk of SCA or appropriate ICD shocksS7.9.1.1-9, and escalation of treatment is warranted to prevent SCD. Previous studies reported the benefit of antibradycardia anxiety, and recurrent fainting or cardiac arrest was reported in 7 to 24% of patients. S7.9.1.1-44-S7.9.1.1-47 In high-risk patients, observational studies support the effectiveness of ICD in preventing mixed diseases, taking into account the left heart sympathetic denervaion to reduce the frequency of ICD shocks. S7.9.1.1-16,S7.9.1.1-48,S7.9.1.1-49 Sympathetic denervation of the left heart can reduce VA load, but up to 27% of high-risk patients experience at least one relapse. S7.9.1.1-16,S7.9.1.1-48,S7.9.1.1-50 Left heart sympathetic denervation may be more effective in patients with long type 1 QT syndrome and long QT syndrome type 3. S7.9.1.1-48,S7.9.1.1-51 Long patients with QT syndrome may experience fainting due to vasovagal fainting, non-compliance with medicines or proarrythmia of concomitant medications. S7.9.1.1-5 Clinical assessment, including the need to change or escalate genotype, QTc interval, drug compliance and joint decision-making treatment, is important. The use of additional medicines is

controlled by a long type of QT syndrome. In long QT syndrome, type 3 ranolatsin, mexlentine and flecainide reduce QTc and have been used to reduce recurrent arrhythmias. S7.9.1.1-6,S7.9.1.1-7,S7.9.1.1-103.Mexlentine is an additional medication that can be used in patients with long QT syndrome and recurrent ICD shock. Sympathetic denervation of the left heart is associated with a reduction in appropriate ICD shocks and VA strain. S7.9.1.1-13–S7.9.1.1-16 Reduction of QTc <500 ms left heart sympathetic dener the frequency of repeated risk and symptoms of ICD shocks has been correlated with the frequency of repeated ICD shocksS7.9.1.1-16,S7.9.1.1.1-52; However, SCD or SCA is reported in 3-10% of patients. S7.9.1.1-15,S7.9.1.1-16,S7.9.1.1-48,S7.9.1.1-50 Although the burden of arrhythmia often decreases, up to 27% of high-risk patients experience at least one relapse. S7.9.1.1-13,S7.9.1.1-14,S7.9.1.1-48 Patient outcomes improve if the sympathetic denervasion of the left heart is performed in centers with surgical expertise in this procedure. The use of additional medicines is controlled by a long type of QT syndrome. In long type 3 QT syndrome, ranolatsin, mexlentine and flecainide reduce QTc and have been used to reduce recurrent arrhythmias. S7.9.1.1-6, S7.9.1.1-7,S7.9.1.1-104. Proceeds of genetic testing with long QT phenotype positive patients range from 50% to 86%, with higher range in patients with significant QT extension or positive family history as a mixture. S7.9.1.1-17,S7.9.1.1-21,S7.9.1.1-53 Negative genetic test does not exclude diagnosis of long QT syndrome based on clinical evaluation. In asymptomatic patients with otherwise unexplained prolonged QTc ≥480 ms serial ECGs, genetic testing may help confirm diagnosis and supplement prognosis data in addition to clinical signs and QTc duration. S7.9.1.1-5,S7.9.1.1-18–S7.9.1.1-20,S7.9.1.1-30,S7.9.1.1-35,S7.9.1.1-54-S7.9.1.1-565.Prospective, observational study of patients, suspected of long QT syndrome, patients with a previous case of fainting or cardiac arrest, and either the affected first-degree relative or the limit or extended QTc interval were subjected to a treadmill test and bicycle exercise, ecgs have been recorded earlier, during and after the exercise, as well as in different positions. S7.9.1.1-27 long QT syndrome was confirmed by genetic testing in all affected persons. In patients with near-normal rest intervals at QTc intervals, QTc's 4-minute recovery extensions ≥445 ms had a high sensitivity to correctly identify patients with long QT syndrome. S7. 9. 1. In a study in younger patients, an increase in QTc time >460 ms after 7 minutes of recovery predicted long QT syndrome of type 1 or long type 2 QT syndrome compared to control. S7. 9. 1. In a study using punctured bicycle exercise, QTc syndrome increased significantly more in patients with latent long QT syndrome than in either control or patients with baseline QTc extensions. S7.9.1.1-24 These findings may be useful in determining whether there is a long QT syndrome. Monitoring the adequacy of beta-sagging treatment through physical activity testing may be useful, especially in school-age patients. S7.9.1.1-26,S7.9.1.1-28 Beta-saccotic therapy may be associated with a reduction in supin and peak exercise QTc, except for long QT type 1 patients with C loop mutations. S7.9.1.1-256.Approximately 10-36% of genotyping positive patients with long QT syndrome have QTc-≤440 ms, most commonly in patients with long QT syndrome type 1. , general patients suitable for age and gender. S7.9.1.1-31 Beta workshops significantly reduce cardiac infeers. S7.9.1.1-1-S7.9.1.1-5,S7.9.1.1-30,S7.9.1.1-36,S7.9.1.1-38,S7.9.9 1.1-41,S7.9.1.1-57 In the first three decades of living life, the highest risk during S7.9.1.1-11,S7.9.1.1.1-18 beta-saper therapy may reduce the risk of SCA. S7.9.1.1-26,S7.9.1.1-28,S7.9.1.1-36,S7.9.1.1-38 QTc changes in particular during puberty and during and after pregnancy, indicating that ECG QTCs should be evaluated annually or with medication changes, and it is possible to assess the effectiveness of the medication by exercise testing. An asymptomatic adult (male) long patient with QTT syndrome with a normal QTc interval may decide to refuse beta-sascoder therapy. The risk of adverse events of S7.9.1.1-11,S7.9.1.1-347.Va. is influenced by the patient's resting QTc interval, age, gender and long QT syndrome genotype/mutation. In asymptomatic men with long QT syndrome, the risk of cardiac events is highest in childhood, S7.9.1.1-2,S7.9.1.1-8,S7.9.1.1-11,S7.9.1.1-30 at a time when drug ad observance is challenging. In young women with LQT2 and QTc >500 ms, has increased risk SCAS7.9.1.1-2,S7.9.1.1-11,S7.9.1.1-18-S7.9.1.1-20,S7.9.1.1-30,S7.9.1.1-35 especially during the nine months after childbirth, and may be candidates for primary contraception for ICD placement or wear a wearable cardiooverter defibrillator. S7.9.1.1- 308. The risk of side effects increases in patients with long QT syndrome and an increase in QTc syndrome >500 ms. S7.9.1.1-2,S7.9.1.1-12,S7.9.1.1-26,S7.9.1.1-35,S7.7 9.1.1 1-41,S7.9.1.1-58 QT-extended medicinal products (www.crediblemeds.org)S7.9.1.1-59 should not be used in patients; have long QT syndrome, unless there is a suitable alternative; Careful monitoring of QTc during treatment is recommended for consideration, taking into account discontinuation of treatment at significant QTc extensions. Concomitant use of stimulants or non-stimulating attention deficit/ hyperactivity medications was associated with an increased risk of fainting/ cardiac arrest with long QT syndrome, especially in men, in one study, S7. 9. S7.9.1.1-60 Episodes of targeting shocks may be saffed by exposure to QT prolonged medication or hypocalemia caused by diuretics or gastrointestinal illencism. Attention to maintaining normal potassium and magnesium balance when medications or depletion-promoting situations meet is an important part of treatment. In rare cases, there is a fever that prolongs the QT period in patients with long type 2 QT syndrome; fever should be reduced with antipyreticsS7.9.1.1-61 (Table 10). Table 10. Commonly used QT prolonged drugsS7.9.1.1-59,S7.9.1.1-62 Individuals of QT prolongation drugs*Antiarrhythmic medicinesPsychotropic MedicinesAntibioticsOthersDisopyramideHaloperidolErythromycinMetadoniProkainamide (N-acetylprokainamide)PhenothiazinesPentami ThedinProbucolQuinidineCitalopramAzithromycinDroperidolDofetilideTricical
chloroquineOndsetronDronedaroneantidepressantitCiprofloxacinIbutilideFlukonazoleSotalolLevofloxacinAmiodaroni†MoksifloxacinCartromycinItrakonazoleKetoconazole Calycoliamine-rged polymorphic chamber tachycardiaFigure 13. polymorphic VT caecoliamine during polymorphic ventricular tacardia. Figure 14. Prevention of SCD in patients with Brugida syndrome. The colors correspond to the recommended category in Table 1. See section 7.9.1.3. *ICD nomination according to functional status, life expectancy or patient preference. 1° means primary; ECG, electrocardiogram; EP, electrophysiological; ICD implantable cardiooverter defibrillator; Heart attack, sudden cardiac death; VT, chamber tachycardia; and VF, chamber color. Support text per recommendation1. Catecholaminergic polymorphic polymorphic ventricular tachycardia is characterized by a stressor-related polymorphic or bipolar VT (Figure 13) associated with fainting and SCA. SCA/ SCD is reported in 3-13% of patients. S7.9.1.2-1,S7.9.1.2-2,S7.9.1.2-8 Beta-blocker treatment is associated with a decrease in myoescist blockers. S7.9.1.2-1,S7.9.1.2-2 Some experts prefer the use of nadolol over other beta-sasmails; direct comparison data for beta-alloy inhibitors are not available. It is important to use the maximum tolerated dose of beta-catcher. Small observational studies suggest the potential benefit of calcium channel inhibitors of nondihydropyridine in the treatment of caecolamine-rgic polymorphic ventricular tacaride. S7.9.1.2-9, S7.9.1.2-102.Flecainide, in combination with a beta-predator, can dampen ventricular ectopia by up to 76% of caecolamine-rg polymorphic polymorphic ventricular tachycardia during exercise testing or clinical monitoring. S7.9.1.2-2,S7.9.1.2-6,S7.9.1.2-11 The power of refractory VA, verapamel or propafenone can also be effective. S7.9.1.2-9, S7.9.1.2-10,S7.9.1.2-12 ICD implantation for patients with caecolic carg polymorphic ventricular tachycardia should be reserved for patients with a history of SCA or patients with reframeal resistance stalls receiving combination therapy. Inappropriate shocks have been reported in 20-30% of patients with calycolamine-rged polymorphic ventricular tacardia with ICD.S7.9.1.2-2,S7.9.1.2-1 3-1 S7.9.1.2-16-ICD programming of caecoolamine-rg polymorphic polymorphic ventricular tachycardia shall be optimised to provide VF therapy and minimise the risk of inappropriate shocks and potentially fatal electrical storms. S7.9.1.2-13,S7.9.1.2-15 Left heart sympathetic denervation due to caecolamine ergic ventricular tacardia can reduce the frequency of repeated ICD attacks by 32% 75 %S7.9.1.2-3-S7.9.1.2-5,S7.9.1.2-17,S7.9.1.2-18 even if repeated fainting, SCA, or 9-32% of patients have reported 9-32% of patients other minor complications in 20-70% of patients. It is best if the sympathetic denervation of the left heart is carried out in centers with expertise in this procedure. Improving the effectiveness of medical treatment or sympathetic denervation of the heart is important patients with recurrent appropriate ICD ICD testing may be useful in the diagnosis of caecolamine-rgic polymorphic polymorphic takycardia, which is indicated by the development of bipolar VT with stress or stress. The recognition of calycolamine-rgic polymorphic ventricular takycardia as a cause of stress symptoms should lead to aggressive treatment to prevent a significant risk of SCD. Treatment of calycolia-rg polymorphic ventricular tacardia is not controlled by the condition of the genotype, but screening of first-degree relatives can be facilitated by genetic testing. S7.9, 1.2-20 Ryanodine receptor mutations have been reported in 47% of probands that were de novo mutations >70%. S7.9.1.2-7 The condition of the Ryanodin genotype has not correlated with the severity of the disease or the response to the drugs. S7. 9. 1. In very young patients with idiopathic VF, mutations in calmodulin have been identified and are associated with high letia. S7.9.1.2 to S7.9.1.2-24 Studies of mutations proposed for pathogenic testing of caecolamine polymorphic polymorphic tachycardiagens show that up to 15 % of variants were present in exopomic databases of the entire population, raising questions about the monogenic cause of calycolimic polymorphic ventricular caracardia. S7.9.1.2-20,S7.9.1.2-257.9.1.3. Brugada SyndromeSynopsisRefer for the 2017 ACC/AHA/HRS Guidance on the Management of Ventricular Rhythm Patients and The Prevention of Sudden Cardiac Death for full systematic evidence review for further information and analysis. S7.9.1.3-15 Question for Brugada syndrome patients for asymptomatic patients, what is the link between abnormal EP study and scd and other endpoints of arrhythmias? (Part 1) and the Writing Committee's review of the literature as a whole were used to frame decision-making. Recommendations based on a systematic review of the ERC are evidence-based, are superscripted in SR (e.g. SR). Factors identified as potential triggers for VF and SCA for Brugada syndrome include some psychotropic drugs and anaesthetics, cocaine, excessive alcohol intake and fever (www.brugadadrugs.org). S7.9.1.3-21,S7.9.1.3-22 These substances should be avoided and fever requires early and aggressive measures under the temperature. S7.9.1.3-23Sal context1. The positive family history of S7.9.1.3-1–S7.9.9.1.3-5 The positive family history of Brugida syndrome or SCA is not a significant predictor of the adverse effects of Brugida syndrome. S7.9.1.3-1,S7.9.1.3-2,S7.9.1.3-4,S7.9.1.3-5 ICD implantation in asymptomatic patients with no spontaneous type 1 Brugada has not been shown to be beneficial.2.Brugada syndrome is characterized by the rise of the coveted ST in the second leadership V1 or V2, thirdly or fourthly, as a result of the administration of a medicine that inhibits space either spontaneously or as a result of the administration of a sodium channel inhibitor, if there are no other causes of st elevationS7.9.1.3-24 and negative T-waves in the right prechloral cord, and is associated with VF-related fainting or SCA, mainly in young men, although reported in all age groups. Type 1 Brugada ECG with desired ST height in the right prechloral cord may occur spontaneously, during fever or vagotone concentrations or after a medication challenge with sodium channel inhibitors. The complex fractioning of QRS is visible in a minority of patients. Patients with spontaneous craving st height with a history of fainting or previous SCA have the highest risk of potentially deadly spies. ICD implantation has been shown to reduce mortality in symptomatic patients with Brugada syndrome. S7.9.1.3-25,S7.9.1.3-263.The ablation of areas of late activation of abnormal epi-crises in the campervan can suppress repeated va-active driving, as demonstrated by a small number of patients. S7.9.1.3-8,S7.9.1.3-9,S7.9.1.3-11,S7.9.1.3-27 In these reports, the spontaneous type 1 Brugada model of the ECG can be eliminated >75% of patients, and the relapse of VT/ VF is greatly reduced. S7.9.1.3-9-S7.9.1.3-11 After Ablation, experience and follow-up is limited and ICD treatment is recommended for patients with a recent blackout or SCA. Several patients with Brugida syndrome treated with kinidine had no deaths over 9 years, although kinidine adverse reactions were reported in 38% of patients, these authors agreed that kinidine could be used as an alternative in patients selected for ICD. S7.9.1.3-74.Observational studies indicate that kinidine may suppress VF storm Brugida syndrome and a low risk of arrhythmias was observed in the long-term study.7.9.1.3-7 There was no low risk of arrhythmia in patients treated with kinidine. Side effects of kinidine occur in up to 37% of patients. Ablation of the catheter targeting areas of epicardial right ventricular abnormalities has also been shown to reduce recurrent VF cycles and normalise ECG.7.9.1.3-9 to 7.9.1.3-115.Promaconide administration, flecainide or ajmaline may be useful to cause elevation of type 1 ST in patients suspected of having Brugada syndrome as a cause of symptoms but without a type 1 electrocardiogram at baseline. The medication challenge should be stopped with the development of VA, tagged QRS widening or type 1 Brugada electrocardiogram. S7.9.1.3-14,S7.9.1.3-28 Use of high electrocardiography electrode in the second and in intermediate mode to store the electrocardiogram improves the detection of type 1 Brugada ECG. S7.9.1.3-29 Asymptomatic patient with Previously, Brugida syndrome may be offered a sodium channel inhibitor challenge for diagnostic evaluation, although a positive test does not require chronic treatment due to the low risk in this environment. S7. 9. 1. With asymptomatic type 1 Brugada electrocardiogram finding, the medication challenge does not provide an extra diagnostic value.6. S7.9.1.3-13 The specificity of programmed stimulation in the risk assessment is reduced by including a triple extra stimuli. S7.9.1.3-6,S7.9.1.3-13 The value of programmed stimulation in an asymptomatic patient with spontaneous type 1 Brugada ECG has been subjected to several studies. S7.9.1.3-1,S7.9.1.3-2,S7.9.1.3-4,S7.9.1.3-5 The report concluded that the prognosis value has decreased over time, possibly in patients with less serious phenovatypes, has been identified and studied. S7.9.1.3-1 Some experts use the results of programmed chamber stimulation to inform joint decision-making taking into account the ICD. Symptomatic ventricular stimulation programmed with brugida syndrome does not add anything to patient evaluation because ICD is justified. S7.9.1.3-2, S7.9.1.3-4, S7.9.1.3-67.The yield of genetic testing in phenovaty positive patients is approximately 20-30% in brugada syndrome. S7.9.1.3-4,S7.9.1.3-16,S7.9.1.3-18,S7.9.1.3-19,S7.9.1.3-30,S7.9.1.3-31SCN5A variants are the majority of this subset of genotypical positive Brugada syndrome. However, 2-10% of otherwise healthy individuals host a rare SCN5A variant. S7.9.1.3-20,S7.9.1.3-31 Negative genetic test does not exclude the diagnosis of Brugida syndrome, which is usually based on electrocardiogram and clinical characteristics. Risk stratation is based on symptoms and clinical findingsS7.9.1.3-32; the state of the genotype is not correlated with the risk of adverse reactions. S7.9.1.3-5,S7.9.1.3-18,S7.9.1.3-19,S7.9.1.3-33 Identification of pathogenic mutation may facilitate the identification of the carrier station by family members; enabling lifestyle change and possible treatment.8.Factors identified as possible triggers for Brugida syndrome VF and SCA include some psychotropic drugs and anesthetics, cocaine, excessive alcohol intake and fever (www.brugadadrugs.org). S7.9.1.3-21,S7.9.1.3-22 These substances should be avoided and fever requires early and aggressive measures to reduce temperature. S7.9.1.3-23Figure 15. Brugida syndrome.7.9.1.4. Early repolarization J-wave syndromeRecommendation-specific support text1. Early repolarization pattern prevalence at ECG, where the increase in the J-point in lower or lateral wires is at least 0.1 mV, is reported to be up to 5.8% in adultsS7.9.1.4-1 and is more Men. The early repolarization model was lost in 10 years of follow-up > 60% of young men. S7. 9. 1. Patients have been identified with early repolaration syndrome when, in addition to the early repolarisation model of ECG, they have either symptoms such as fainting or arrhythmia. When a patient has an early repolarisation pattern in the ECG, it is important to exclude reversive causes such as ischemia. Patients with early re-polarisation are more susceptible to the development of VF during acute cardiac chemistry and/ or QRS abnormalities due to LV hypertrophy or a bundle arm block. S7.9.1.4-6-S7.9.1.4-82.Patients with cardiac arrest or VF in the determination of the electrocardiogram at early re-polarisation are at increased risk of re-recurrent VF episodes in at least 40% of patients. S7.9.1.4-3,S7.9.1.4-4,S7.9.1.4-9 Antiarrhythmia drugs, excluding kindin/ hydrokinidine, are low in effectiveness in the prevention of repeated VA medications. S7.9.1.4-3, S7.9.1.4-43.To date, genetic testing has not reliably identified mutations prone to early re-polarization. S7.9.1.4-57.9.1.5. Short QT syndrome Recommendation-specific support text1. The intestion of ≤340 ms is estimated to be 5/ 10,000 in people <21 years of age and more common in men. S7.9.1.5-1,S7.9.1.5-4,S7.9.1.5-8,S7.9.1.5-9 Random observations of A QTc ≤320 ms observation in asymptomatic patients require follow-up and follow-up without prophylactic drug therapy. S7.9.1.5-1, S7.9.1.5-22.Patients with cardiac arrest with short QT syndrome are known to be at increased risk of recurrent cardiac arrest. S7.9.1.5-3-S7.9.1.5-5 Approximately 18% of small patients with short-term QT and implanted ICDs have received appropriate ICD treatments during short-term follow-up. S7.9.1.5-3,S7.9.1.5-5,S7.9.1.5-6 Kinidine therapy can reduce the number of ICD shocks. S7.9.1.5-3,S7.9.1.5-5,S7.9.1.5-63.Significantly reduced QTc values ≤300 ms are associated with an increased scd risk, especially during sleep or rest in adolescents, with a median QTc of 285 ms. S7.9.1.5-5,S7.9.1.5-9 Clinical score that includes QTc duration, documented polymorphic VT or VF clinical history, unexplained fainting, family history autopsy negative SCD or sudden death syndrome, and positive genotyping results have been suggested to identify patients at increased risk of freaking out. S7.9.1.5-4,S7.9.1.5-10 Kinidine therapy leads to an increase in QTc and may be an alternative to ICD implantation in selected patients. S7.9.1.5-3,S7.9.1.5-5,S7.9.1.5-64.Setting an electrical storm with refractory VF and short QT syndrome isoproterenol infusion can be effective in restoring/maintaining the sinus rhythm. S7.9.1.5-75.Pathogenic mutations approximately 10-20% of patients with short QT syndrome, including KCNH2 (SQT1), KCNQ1 (SQT2) and KCNJ2 (SQT3). S7.9.1.5-4 Due to the rarity of the disease, genotytype/phenoptype correlations are not available, which limits knowledge of the state of the genotype.8. Va structurally normal heartSynopsisMost idiopathic VA is caused by a focus mechanism triggered by triggered activity or abnormal automaticity, some, especially interfascicular reentrant LV tachycardias, due again. The clinical manifestations of idiopathic VA are very varied and range from benign, asymptomatic PVC to persistent VT or even VF. In the context of the preliminary finding, the assessment of structural heart disease is justified by physical examination, ECG and imaging, usually echocardiography. In the case of abnormalities or family history of SCD, the symptoms will guide further evaluation and treatment. If the patient is asymptomatic and shows no signs of cardiac channelopathy, adequate assurance of benignity is sufficient. If arrhythmias are suspected to be sufficiently common to cause ventricular dysfunction over time, it is justified to regularly monitor the reassessment of the chamber function (see section 10.8). For mild symptoms, avoiding aggravating factors such as excessive caffeine consumption or sympathitometics may suffice. Treatment of a beta-blocker or nondihydropyradine calcium channel inhibitor reduces the symptoms in some patients. Class I antiarrhythmia drugs can be effective, but they are usually avoided due to side effects. In patients who need suppression of arrhythmias and for whom antiarrhythmia medications are ineffective, intolerable or undesirable, catheter ablation can be a very effective treatment (see section 9). The ablation strategy is to identify the place of origin that appears from the earliest place of electrical activation or, if this is not possible, from the pace mapping. The most common place of origin of idiopathic VA is from the correct ventricular outflow channel (RVOT) or LV ostium, which consists of the oval opening of its LV, to which the aorta is attached in the front and the left atrium is attached to the back. The likely origin can reasonably be predicted from the VA's QRS morphology, which gives a good idea of the approach required and the likelihood of success and risk. Ablation disorder is often associated with the fact that there is no procedure or va origin for VA determination in the hard-to-use area of the heart. These phos sometimes produce continuous monomorphic VT. S8-5-S8-7Scommendation specific support Text1.In randomised, double-blind, placebo-contrast study in 52 patients with symptomatic VA and PVC levels of 21,407±,740 beats in 24 hours, atenolol significantly reduced the number of symptoms (P =0.03) and PVC (P =0.001), while placebo did not affect PVC (P =0.78) or mean heart rate (P =0.44). S8-8 Randomised prospective comparison of antiarrhythmics with catheter ablation, metoprolol or propafenon was a modest efficacy to suppress RVOT VA, although its rate of recurrence was much higher than catheter ablation. S8-92. With RCT in 233 patients with ≥30 PVC per hour, d-sotalol was shown to frequently reduce PVC, but only racemic dl-sotalol is currently available. S8-10 Randomised randomised comparison of antiarrhythmics with catheter ablation showed a modest efficacy of treatment with metoprolol or propafenone when used to suppress RVOT-PVC, although its rate of recurrence was much higher than catheter ablation. S8-9 Calcium channel inhibitors of nondihydropyridine reduce arrhythmias. S8-1,S8-2,S8-11,S8-128.1. Outflow tract and atrioventricular annular VARecommendation Specific support Text1.In 1 RCT, catheter ablation was better than antiarrhythmia drugs, which often suppressed PVC due to RVOT. S8. Observational studies have shown that radio frequency catheter ablation is effective in the treatment of RVOT and idiopathic VA from the LV outflow channel. S8.1-2,S8.1-5-S8.1-16 The ablation point may be below or above the RVOT pulmonic valve. S8.1-9,S8.1-13 Although most RVOT VA can be crushed in a camper van, 10% can require ablation inside pulmonic sinuscusps. S8.1-9 Serious complications are rare. LV outflow tract VA, The ablation point may be inside aortta cusp sinuses, S8.1-11,S8.1-14,S8.1-16 alla aor S8.1-2,S8.1-6 in aorto-mitral continuityS8.1-1-S8.1-3 or on the epic surface of the LV summit. S8.1-3,S8.1-17,S8.1-18 Mitral and tricuspid annulae are less common sites of idiopathic VA, but these VA can also be effectively treated with catheter ablation. S8.1-1,S8.1-19,S8.1-20 About 10% of the idiopathic VA can emerge from the LV summit. Some can be ablosed from a large heart or epicardial surface, but others arise from an inaducable area close to the left coronary artery, which closes effective ablation. S8.1-14 Internal locations in the country of origin are rare, but they may require ablation on both the endocardial and epicardial surfaces of LV ostium. S8.1-3 Complications related to the ablation of outflows are rare, but bleeding complications associated with the use of arteries and veins, perigardial tamponade and coronary artery damage may occur.2.In the prospectic randomised comparison of antiarrhythmic drugs with catheter ablation, metoprolol or propafenone shown to have modest efficacy when used with RVOT suppression of n, albeit at a much higher rate of recurrence than catheter ablation. S8.1-4 Calcium channel inhibitors of non-dihydropyradine dampen arrhythmias some patients. S8.1-48.2. Papillaari muscle VARecommendation-specific papillary muscles of tukiteksti1.LV or camper van may be the va's place of origin in the presence or absence of structural heart disease. S8.2-1-S8.2-5 Idiopathic left and right ventricular papillary muscle VA are most commonly PVC and NSVT and are usually associated with exercise and can induce intravenous administration of epinephin or isoproterenol. S8.2-3 These arrhythmias have a centerpiece, non-reentrant mechanism. Any of the papillary muscles in 3 camper vans can be the starting point and catheter ablation is usually effective. S8. 2-2 In one study, successful ablation was achieved in all eight patients with a 17±20% reduction in PVC load to 0.6±0.8%. S8.2-2 In the left chamber, the starting point can be either posteromediaal or anterolateral papillary muscles. S8. 2-1, S8. 2-4, S8. 2-5 Multiple VA QRS morphologies were observed in 47% of patients, and some patients require ablation on both sides of the papillary muscle. S8.2-4 Sufficient catheter stability can be challenging. Acute ablation success is high, but recurrences are more common than in idiopathic outflow tract VA. Serious complications, including valve injury, appear to be rare. The risk of catheter ablation is bleeding associated with access to arterial and venin treatment and a low risk of pericardial spontaneous treatment.8.3. Interfascicular Reentrant VT (Belhassen Tachycardia)Recommended support text1. The idiopathic LVT is to re-attempt the part of the LV Purkinje system, usually left rear fascicle as a circuit-backward degradable limb and an incompletely defined segment of LV tissue as an anterofgradi limb, part of which is verapamil sensitive. S8.3-1–S8.3-3 These VTs typically can withstand QRS with the right bundle arm block assembly with a superior shaft. Less often, the lower-axis VT or relatively narrow QRS VT occurs as a result of alternative re-enterprise routes, which also involve part of the Purkinje system. Beta-blockers or verapamil usually stop these arrhythmias, but they do not prevent relapse in some patients. S8.3-1–S8.3-3 The most common form catheter ablation target is usually the haggling of the disterograded limb of the anterofraation limb of the Purkinje system along the lower part of the LV partition near its intersection with the fascicle at the left rear. Catheter ablation is acutely successful > in 90% of patients at approximately 10% risk of relapse. This VT can resemble a fascicular VA due to the combustion mechanism of the left front or left rear fascicles of the LV His-Purkinje system. These fasccular arrhythmias usually have a focus mechanism aimed at catheter ablation, the earliest electronic activation recorded in presystolised with potential. Catheter ablation is very effective and fascicular VA. Serious complications are rare and include bleeding at an arterial or vein site and a low risk of a bundle branch block or atrioventricular lobe.2.Idiopathic LVT is based on a retraining mechanism involving tissue with slow leadership properties along the LV partition as an anterergrade limb and the normal left rear fascicle retrograde limb of the His-Sailkin system. The slow leader zone is verapamil sensitivity. S8.3-3–S8.3-6 These arrhythmias typically have the right bundle crotch block morphology with a superior axis, although turning the circuit can produce a relatively narrow QRS during VT. Verapamil typically stops these arrhythmias in the anterograde slow management zone. S8. 3-3 to S8. 3- 63. S8.3-5,S8.3-8–S8.3-108.4. Idiopathic polymorphic VT/VFRecommendation specific support text1. genetic testing can provide diagnosis in up to 13% or 60% of younger (<40 years old) SCA, S8.4-3 survivors with the most common genotypes associated with long-time QT syndrome, caecolamine-related polymorphic polymorphic ventricular tachycardia and Brugada syndrome. S8.4-8 Drowning/near drowning events are specifically related to LQT1 and caecolamine-related polymorphic chamber tachycardia; genetic mutations in long QT syndrome and caecolamine-rgic polymorphic ventricular tacardia have been observed in 23% of patients with unexplained near drowning episodes. S8. 4-15 In one study S8, 4-6 stress-related cardiac arrest, especially in children, may be associated with long QT syndrome, caecoliminergical polymorphic ventricular tachycardia or calmodulin/triadine-mediated long QT syndrome/caecholam polymorphic polymorphic ventricular tachycardiautations that may require further specialized genetic tests. S8.4-1,S8.4-2,S8.4-4,S8.4-16–S8.4-18 Single driver car accidents should lead to the taking into account of arrhythmias. The yield of genetic testing is higher if the family history is scd at a young age. Referral to a specialised genetic testing centre is important if local expertise is not available.2.VF without identifiable structural heart disease or known genetic arrhythmia syndromes, such as calycolia-rg polymorphic polymorphic ventricular tachycardia, long QT syndrome, short QT syndrome, Brugida syndrome or J-wave syndromes are usually caused by short PVC caused by the jarje system, either in the right or left ventricle, or less frequently in the ventricular heart cardioxies. S8.4-9–S8.4-13 The risk of recurrence after revitalization of idiopathic VF is very high. S8.4-12 Among 38 consecutive patients from 6 different centers who ablation of 87% of primary idiopathic VF initiated by short PVC had had ≥2 VF episodes in the previous year. S8. Since idiopathic VF carries a very high risk of recurrent VF, ICD is indicated to prevent scd. Ablation of the triggering centre of gravity catheter has proven to be very effective in eliminating recurrent PVC's that cause VF in these patients. S8. During 4-11 63 months of postprosedural monitoring 7 (18%) In 38 patients for whom idiopathic VF catheter ablation was caused by short-typing PVC, vf relapse occurred with a 4-month median follow-up. Five of these 7 patients underwent repeated ablation without vf relapse. Although catheter ablation is highly effective with idiopathic VF, the risk of relapse is still considerable after an apparent successful procedure and the patient should be protected by ICD. Subcutaneous ICD may not be a good treatment for these patients as the risk of T-wave oversaturation is higher in this patient population; However, the information is limited. S8.4-103.The Idiopathic VF can be started by PVC's, which are generated by outflows or his-Purkinje system inside either the right or left ventricles. S8.4-11,S8.4-14,S8.4-19-S8.4-21 Some patients have VF episodes (electrical storm), which typically occur polymorphic VT/VF compounds at the beginning. PVC usually has consistent QRS morphology and a short switching interval and can be targeted at controlling arrhythmias. S8.4-11 For the PVC of the Canne system, the ablation target is the high-frequency Purkinje potential before PVC. When cycles induce short-bound VCCs caused by outflows, the ablation target is the site of the earliest chamber activation. Patients with idiopathic VF have frequent VT/ VF episodes with relative tipping. S8.4-11,S8.4-14 To maximize successful ablation probability, the procedure is best performed during frequent PVC. When VCCs can be identified, ablation is very successful, but late recurrences are observed in approximately 10% of patients in such a way that implantation of ICD makes sense, even if ablation is acute. The risk of catheter ablation is bleeding at the site of use of artery or venin and a low risk of pericardial tamponade. Treatment with kinidine acutely and chronically may suppress recurrent VF episodes in some patients. S8.4-229. Cardiomyopathy caused by PVC Text supporting homes1. Frequent VCCs (usually >15% of total strokes) can cause a reversing LV malfunction. S9-5-S9-18 However, sometimes it is difficult to verify whether PVC's caused the LV malfunction or whether progressive LV often caused PVC. LV dysfunction has been associated with a higher PVC load (>10% and generally >20%), NSVT, PVC-retrograde P-wave and interpolated PVC. In a prospective study of catheter ablation of cardiomyopathy caused by S9-6, S9-15 PVC, ablation was fully successful in 80% of patients. S9-19 LV was normalised within 6 months in 82% of the 22 patients with baseline depressed ventricular dysfunction. Therefore, frequent VCCs can be a reversible cause of LV dysfunction that can be effectively treated with catheter ablation. It is often difficult to determine whether apparent LV dysfunction or inability to accurately assess LV function often reflects uterus activity. In patients with established.2.In high PVC density with normal ventricular function, optimal treatment and monitoring to prevent and detect impaired ventricular function has not been performed in double-blind parallel studies in 30 patients with or without ischemical heart disease with or without cardiac disease with >30 PVC/ h compared to sotalol propranolol, had proarrthmic effects in 1 patient receiving sotalol. There was no significant difference in PVC suppression (sotalol 65%, propranolol 44%), and a decrease in chamber pairs was 99% for sotalol and 49% for propranolol. QTc was significantly increased in patients treated with sotalol. S9-20 double blindness, randomized placebo contrast study, in which 674 patients with HF and LVEF <0.40 who were attributed to ischemistry or NICM and ≥10 PVC per hour, amiodarone significantly reduced VA, slowed heart rate and was associated with a 42% increase in LVEF at the 2-year mark, which was not a significant trend for reduced mortality. S9-4 It is not known whether treatment affected ventricular dysfunction in these patients. VA and SCD Relate to specific populations10.1. Athletes Athletes, TAs range from isolated PVC, couples and NSVT to continuous VT and SCA leading to SCD. S10.1-1 Rare PVC's and short repetitive NSVTs, especially without structural heart disease, are more common in non-athletes, but they tend to be benign, require only limited work and rarely lead to the abandonment of sports. By contrast, longer runs of the NSVT, especially those caused by physical activity, and continuous VT and SCA/SCD are rare, but their prevalence in athletes is higher than in similar age groups. Reported SCD estimates range from 1 per 53,703 athlete years in the National Collegiate Athletic Association databaseS10.1-4- <1/200,000 minnesota high school students. S10.1-5 Of the studies for which epidemiological research protocols are assessed better, estimates were between 1/40,000 and 1 per 80,000. S10.1-6 These figures compare with the general population risk between 1.0 and 1.9/100 000 000 and young adults. In addition, S10.1-7,S10.1-8 Risk differences appear to be both in sports and gender, and men have a higher risk than women in most sports, S10.1-7,S10.1-9 blacks have a higher risk than whites, and male basketball players are the single highest risk group in the United States, 1/5200 athlete years. The S10.1-4A study, which included both competitive and recreational athletes, showed that both groups have a higher risk of SCD than the general population, recreational athletes have a higher cumulative number, S10.1-7 SCD occurs at an older age, and disease distribution is different. Postmortem data from SCD in athletes reveal that 25-40% are autopsy negative, suggesting the role of genetic molecular disorders in these victims S10.1-4, S10.1-10,S10.1-11 and family members. The second limitation of S10.1-12 ScD data analysis in athletes focuses on non-hearted causes, some of which mimic cardiac events. Non-cardiovascular causes include acute neurological disorders, drug abuse, heat stroke, rhabdomyolysis, sickle cell disorders, suicides and accidents. However, S10.1-13,S10.1-14 Athlete arrhythmias are still the most common medical cause of death and many occur as the first cardiac event. The most common structural cause of SCAs and SCD in athletes in the United States is HCM, followed by an abnormal origin of coronary arteries, heart and corditis affecting a smaller but significant proportion. S10.1-15 In addition to these, other hereditary disorders contribute to the distribution of the causes of SCD in athletes, many of whom can be suspected or identified in careful family history and preparation ECGs.In common, arrhythmia management in athletes follows that in non-athletes. As regards interventions, it is generally recommended that AEDs be available in the training and facilities of competitive athletes, S10.1-16, where there are fewer specific statements on the availability of AED at venues (e.g. tennis courts) or in conditions (e.g. jogging or small group running) where recreational sports occur. Many athletes who have had remedial measures (correction of birth defects or developmental defects, such as abnormal origin of coronary arteries)S10.1-17,S10.1-18 are hereditary care disordersS10.1-19 or have ICD implantsS10.1-1 and can participate in athletics depending on the nature and severity of the disease and with appropriate precautions and advice on possible residual risks. S10.1-19,S10.1-20 For example, athletes with acquired disorders such as myoestivitis are advised not to exercise for at least 3-6 months after the disease has been resolved.10.2. Pregnancy Recommendation-specific support text1. Women with long QT syndrome should be advised of maternal and foetal risks prior to pregnancy to ensure continuous beta-blocker therapy. SCA The risk of SCD is significantly higher 9 months after giving birth, especially in women with LQT2. S10.2-1,S10.2-6,S10.2-7 Extensive retrospective analysis of the Long QT Syndrome Registry showed that the odds ratio for the synopsis, SCA or SCD was 40.8 for women with long QT syndrome after 9 months of childbirth; beta-blockers were independently at reduced risk during pregnancy. S10.2-7 Common arrhythmias during pregnancy do not increase among women receiving beta-blocker therapy. S10.2-1,S10.2-6,S10.2-7 In the case cotrtrate study, women treated with LQT1 who did not receive beta-blockers during pregnancy, especially those with a history of fainting, had a significant risk of SCA or fainting. S10.2-8 The frequency of events returned to prepregncy level after 9 months. S10. The use of beta-blockers during pregnancy is associated with a decrease in the birth weight and hypoglycaemia of the newborn, S10,2-9, but does not involve an increased risk of miscarriage. S10.2-8, S10.2-10 Fetal bradycardia is associated with fetal long QT syndrome and must not independently cause discontinuation of beta-blocker therapyS10.2-11-S10.2-14; these babies are at increased risk of death and need careful neonatal monitoring and care. S10,2-13 Since 50% of offspring may have long QT syndrome, which has the highest risk of side effects in infancy and childhood, screening of the newborn at birth and in infancy due to long QT syndrome is important. S10.2-82.Available information on electric fields for properly installed AED patches suggests that the foetus is safe; observation is not available to the contrary. Anterolateral defibrillator pad placement is recommended with a lateral pillow/paedo placed under breast tissue, which is an important aspect in a pregnant patient.3.ICD in pregnant women is safe and effective. S10. The risk of exposure to foetuses is minimal in rare cases where pregnant women have an immediate indication for ICD or less common signs of VT ablation during pregnancy. S10.2-5,S10.2-15 The procedure is usually carried out after the first trimester, unless there are circumstances that require previous procedures. Wearable cardiooverter defibrillator has been used in peripartum cardiomyopathy pending repeated evaluation of ventricular recovery. S10. A subcutaneously implanted cardiooverter defibrillator is a possible alternative to traditional ICDs, although no data are available to support the recommendation.10.3. Elderly patients with komorbiditiesSynopsisRefer, Systematic review of the 2017 ACC/ AHA/HRS guidelines on the management of ventricular rhythm patients and prevention of sudden cardiac death to review further data and analyses Perfectly. S10.3-1 Results from the question What is the effect of ICD implantation older patients and patients with significant involvements? (Part 2) and the Writing Committee's review of the literature as a whole were used to frame decision-making. The recommendations are based on evidence that includes a systematic review of the ERC and is marked with the superscript SR (e.g. SR). Co-diseases included various combinations of kidney disease, chronic obstructive pulmonary disease, atrial fibrillation and heart disease. Support text per recommendation1. The older age is defined ≥75 years. ERC analyses are useful when it is clearly demonstrated that age or subdiments alone should not be exclusions of ICD. However, the information contained in the analysis is limited. Firstly, most of the data comes from non-government studies, and both selection and unrecognizable confounding biases can never be fully adapted... It is likely that more fragile patients are not already properly provided with ICD, so they are not included. Secondly, since most of the studies are randomized, these findings only imply a link and not a causal link. Older adults are also prone to higher complications, shorter life expectancies (and therefore fewer years during which they could benefit from ICD) and varying preferences. S10.3-2 For these reasons, it is important to adopt a particularly nuanced and patient-centred approach to the treatment of these patients.10.4. Chronic kidney disease Patients with chronic kidney disease have an increased risk of SCD compared to the general population, but the risk vs. benefit of primary prevention ICD has been unclear; observational data have been contradictory and patients with moderate to severe RCD, in particular dialysis patients with end-stage renal disease (ESRD), were not included in the main RCT-et al of ICD. S10.4- 1- S10. there were significant limitations in previous data because comparing patients treated with ICD has conflicted with a control group without primary prevention ICD, and renal failure is likely to affect survival benefit. S10.4-6 Patients with CKD, especially ESRD receiving dialysis, appear to be at increased risk of ICD-related complications. A significant proportion of sudden deaths are not linked to the veteran population in this population. Therefore, the ERC was asked to address the effect of ICD on mortality in patients with CKD. The ERC conducted a specific analysis in 5 studies of renal dysfunction. Meta-analysis of these studies showed a link between ICD implantation and improved survival. S10, 4-8 An important limitation is that only two studies specifically examining patients with ESRD and most of the data analysed came from: Studies. S10.4-8,S10.4-9 Due to these limitations, the Writing Committee data were not sufficient to report the recommendation for ICD implantation in patients on dialysis. Decisions on ICD in patients with CKD, especially in patients with ESRD, shall be identified and shall take into account, inter alia, the functional status of the patient, the number of infectious diseases and preferences.10. Valvular Heart Disease Patients with valvularine heart disease should be evaluated and treated in accordance with GDMT when controlled heart disease and LVEF become depressed GDMT applied to NICM to reduce scd risk. S10. In patients with control-to-23 Va patients with controlled heart disease, any mechanism responsible for VA in other heart conditions, including sechemical heart disease, myoesthesis, severe LV hypertrophy, adrenergic arrhythmias or hereditary molecular averse. Patients with valvulaar heart disease and VA are generally evaluated and treated using the current recommendations for each disorder. S10.6-1 The VA mark alone is not a sign for repairing or replacing the valve. Generally speaking, there is more information on mixing risk in patients with aortic hta than in other controlled lesion at a risk of 1-1.5% per year. S10.6-2 Most patients who die suddenly have symptoms of their valve disease. S10.6-3,S10.6-4 Although repeated NSVT may cause severe aort htauma to faint, the treatment of such a patient is guided by the severity of the controlled lesion. The prolapse of the miteral valve has been involved in the cause of SCD, although a study of 18,786 patients showed no increased risk of SCA in patients with a prolapse of the sappiaflet miteral valve compared to a single prolapse of the mitral valve in the prospector or no mitral valve prolapse. S10.6-5 LV fibrosis in papillary muscles is described in some mitral valve prolapse patients with joint pain or coronary artery impairment. S10.6-6 In addition, possible SCD syndrome, which includes the miteral valve prolapse of the bile band, female sex, T-wave abnormality and complex ventricular ectopia, has been described. S10. 6-7 The current guideline provides guidance for the treatment of NICM patients, whether they are valvularic or otherwise derived (see primary contraception and secondary contraception). Gender-related differences in the risk of SCD Data on the confluence between sex and VA and SCD are largely limited to epidemiological, cohort and observational studies. In a number of population studies, which focused mainly on mixed departments mixed mixed mixed with ischemica heart disease, it has been shown that male and female age gradients have been at mixed age. S10.6–8–S10.6–10 These include a 10-year delay in the prevalence of SCD among women compared to men. however, the risk factor charge has the same relative effect as men, and the risk increases 17-fold from the lowest to the highest tithing. S10, 6-9 Importantly, 69% of women's SCD was one of the first cardiac events. S10.6-8 Study The risk of SCD stratation among 45, 55, 65 and 75-year-olds was consistently found to be lower and similar to the lifetime scd risk rate in women as in men in each stratage. S10.6-10 The difference between women and men is somewhat smaller under the age of 75 and before, largely due to a reduced risk for men. The total lifetime risk of SCD was one in nine for men and one in 30 for women. In studies conducted after cardiac arrest outside the S10.6-10 Hospital, women were older, had more mixable codes and fewer shock rhythms (VT/ VFs) than men. S10.6-11,S10.6-12 This overall involved a somewhat lower probability of survival; However, women with VT/VF and those with pulseless electrical activity had better results than men. A retrospective analysis of cardiac arrests outside the S10.6-12 Hospital showed that survival improved over a 10-year period, with more favorable outcomes for men and younger women. S10.6-13 In two studies, the results were better in women with VT/ VF, despite adverse risk factor profiles in women. S10.6-14,S10.6-15 Another large study showed that despite a similar return to spontaneous blood circulation and survival before hospital, younger women had lower 1 month of neurologically intact survival than the 50-60 age group. The S10.6-16 17-year retrospective analysis showed no difference between men and women, although overall results improved. S10.6-17 The proportion of heart disease-related interference disorders related to chemistry among women who survived cardiac inseminations outside the hospital was significantly lower than in men, but sechemical heart disease remained the strongest predictor etiologically,S10.6-18 and women were also significantly less likely to have severe LV dysfunction (LVEF ≤35%) or previously recognised sechemistry heart disease. S10.6-19 Women seem less likely to benefit from therapeutic hypothermia in postcard arrest; However, in the younger age group, neurological recovery in women was better than in older women. S10.6-20 Women are less likely to have an SCA at competitive sporting events. A large study of both recreational and competitive athletes in the broad age group found that SCA women at sporting events had one in 20 men. The S10.6-21A extensive literature review for 1980-1992 showed that the proportion of women recorded cardiovascular arrhythmias was 70%. S10.6-22 This corresponds to the QT intervals for men and women. Retrospective analysis of discontinuation of kinidine treatment showed a significant difference between men and women (66% vs. 84%) largely prolonged QT S10. An ablation study of a 6-23 VT catheter found that the overall result was similar between men and women. S10.6-24 The only gender difference was a higher probability that women have RVOT VT, and a higher probability that men have LV outflow LV outflow VT.10.7. Arrhythmias caused by medication Recommendation-specific support text1. Typical arrhythmias associated with digoxin toxicity are enhanced atrial, intersectional or ventricular automaticity (with ectopic beats or tachycardia), which are often associated with an irioventricular block. Fascicular or bidirectional S10.7-11 VT refers to digoxin tooxy. S10.7-12 Severe digoxin overdose causes hypercaemia and cardiac stop. The diagnosis is based on a combination of typical arrhythmias, associated symptoms (visual disturbances, nausea, mentation changes) and elevated serum levels. Thyroid insufficiency, hypocaemia or renal dysfunction may be aggravating factors. S10.7-12 The treatment of digoxin poison is based on the degree of difficulty. In mild cases, distopping medication, monitoring of rhythm and maintaining normal serum potassium levels may be sufficient. S10. 7-11 Intravenous magnesium is often administered if it contains venous thrombes. S10.7-12 Sometimes temporary paceing of an atrioventricular block or asystole may be necessary. S10.7-13 For more severe poisoning (serum digoxin levels above 4 ng/ ml and severe arrhythmias such as VT), the choice is a digoxin-sexin-high Fab antibody. S10.7-1 In one series of 150 severely intoxicated patients, the response was rapid (from 30 minutes to 4 hours) and 54% of patients with cardiac arrest survived hospitalization. S10.7-1 Adverse reactions include exacerbation of the underlying disease (increased ventricular velocity during af, exacerbation of HF) and hypocaemia. Doses lower (and cheaper) than the total neutralising dose are sufficient as long as cardiac arrest is not immediate. S10.7-2 Monitoring of digoxin levels is unreliable after antidigoxin antibody is given.2. Monitoring high-risk patients during the initiation of antiarrhythmic drugs prolonging QT and identifying the syndrome when it occurs are the first steps. Temporary paceing is very effective in controlling torsades de pointes, which is repeated after potassium and magnesium supplementation. S10.7-3 Isoproterenol can also be used to increase heart rate and remove postectoppi breaks. S10.7-33.Intravenous magnesium can suppress flips de points without necessarily shortening QT, although serum magnesium is normal. S10.7-4,S10.7-5 Repeated doses may be necessary, titrationed to suppress ectopa and unsustainable VT cycles when harvesting the application factors. S10.7-4 Magnesium toxy (reflexion progressing to respiratory depression) may occur at high serum concentrations, but this risk is very low at doses usually used to treat the upper edges, 1-2 g intravenously. Variants of clinical long QT genes S10.7-14Allelic have been identified in patients with medication-inded torsades de points. Further, the entire exome sequence exome sequence increased loads of rare potassium channel variants at risk of osciliting de-points caused by medicines. S10.7-17,S10.7-19 These findings do not yet support general genetic screening for drug-inded torsades de points. Long QT syndrome may be genetically modified in an index case of drug-inded torque de points, and if he did not survive this event, electrocardiogram screening of first-degree relatives may be performed.4.Maintaining serum potassium levels between 4,5 mEq/L and 5 mEq/L reduces QT and may reduce the possibility of repeated torsades de pointes. S10.7-6, S10.7-75.In large clinical studies, sodium channel inhibitors increased mortality in patients convalescing from study S10.7 to 20, but similar trends were also seen in previous mexlevinS10, 7-21 studies and disopyramide. Flecainide based on S10.7-22 CAST is contingent in ischemian patients with a history of cardiac incidence and is avoided in patients with other structural heart diseases. S10.7-20Natrium channel inhibitors increase defibrillation energy demand and synchronizationsS10.7-8,S10.7-9; As a result, patients may require reprogramming or checking or modification of paces or ICD systems in their medication treatments (although modern paces systems that provide automatic pace-trigger testing and adjustment of syncing output have mitigated the risk of recovery). Sodium channel inhibitors can convert AF into a slow atrial cheat that can show 1:1 atrioventricular conduction with wide QRS mixes that can be confused with VT. S10.7-23 Sabbaths of the Sodium Channel, such as prokainamide and flecainide, may periodically cause typical Brugida syndrome ecg. S10.7-24,S10.7-25 This has been reported not only with antiarrhythmics, but also with tricyclic antidepressantsS10.7-26 and cocaineS10.7-27 (www.brugadadrugs.org). S10. It is not known whether this represents the disclosure of people with clinically unapparentical Brugada syndrome (see section 7.7). 9. 1. 3) or the other end of a wide range of responses from sodium channel inhibitors. When sodium channel inhibition is set, limited animal data suggest that the administration of sodium as sodium chloride or sodium bicarbonate may slow down or dampen frequent or cardioversion-resistant VT. S10. 7-29 Successful treatment with beta blockersS10. S10.7-316.QT prolongation drugs (www.crediblemeds.org)S10.7-32 are not used in patients with congenital or acquired long QT syndrome unless there is a suitable alternative or benefit far exceeds the risk. Vsärsades de pointes episodes may prey on a drug that prolongs QT, and QT (for genetic and clinical risk factors) increases this risk. S10.7-10 Medicines inrsades de pointes löytyy useista lääkitysluokista, mukaan lukien rytmihäiriölääkkeet, antihistamiinit, antibiootit, sienilääkkeet, masennuslääkkeet, psykoosilääkkeet, opiaatit ja syöpälääkkeetS10.7-10 (taulukko 10).10.8. Aikuisten synnynnäinen sydänsairausTable 11. Congenital Heart Disease: Risk Factors for VA/SCDCongenital Heart DiseaseIncidence of VAIncidence of SCDHigher Risk CharacteristicsSimple complexity ASDS10.8-44,S10.8-47,S10.8-57–S10.8-622%–6% <1.5%Ventricular pacingrv= dilatationpulmonary= hypertensionnkx2.5=></1.5%Ventricular> <3%Moderate complexity tetralogy= of= fallots10.8-1,s10.8-2,s10.8-5,s10.8-6,s10.8-28,s10.8-34,s10.8-36,s10.8-44,s10.8-46,s10.8-47,s10.8-54–s10.8-56,s10.8-62–s10.8-6514%–31%1.4%–8.3%unexplained= syncopefrequent= or= complex= vasustained= vtqrs= duration= ≥180= msinducible= sustained= vtatrial= tachycardiadecreased= lvefdilated= right= ventriclesevere= prsevere= ps aortic= stenosis= s10.8-27,s10.8-44,s10.8-5610%–34%3%–20%unexplained= syncopesevere= lv= hypertrophyaortic= stenosis= mean= pressure= gradient=>40 mm HgVentricular dysfunction Coarctation of aortaS10.8-28,S10.8-29,S10.8-44,S10.8-46,S10.8-56,S10.8-622%2%Aneurysm at repair siteAortic stenosisSystemic hypertensionPremature coronary artery disease Ebstein's anomalyS10.8-45,S10.8-47,S10.8-552%3%–6%CardiomegalyAtrial fibrillationWide complex tachycardiaMitral regurgitationDilated RVOTSevere complexity Transposition of the great arteriesS10.8-27 ,S10.8-44–S10.8-48,S10.8-51,S10.8-55,S10.8-56,S10.8-62Atrial switch Atrial switch2%3%–9.5%Mustard repair Arterial switch2%1%Prior VSD closure cc-TGA10%17%–25%Unexplained syncopeAtrial tachycardiaCoronary orifice stenosisSystemic ventricular dysfunctionSevere tricuspid regurgitation Truncus arteriosusS10.8-66,S10.8-6710%4%Multiple surgical repairsCoronary anomaliesVentricular dysfunction and/or hypertrophy Fontan repair for univentricular physiology*S10.8-27,S10.8-37,S10.8-44,S10.8-45,S10.8-47,S10.8-55,S10.8-685%–17%2.8%–5.4%Atrial tachycardiaLonger duration of follow-upAscitesProtein-losing enteropathyFigure 16. Sepato-intraatiota sairastavilla potilailla, joilla on aikuisten synnynnäinen sydänsairaus. Värit vastaavat taulukon 1 suositusluokkaa. Katso kohta 10.8. *Korkean riskin piirteet: aiempi palliatiivinen systeeminen keuhkoshunteille, selittämätön pyörtyminen, usein PVC, eteistakykardia, QRS- kesto ≥180 ms, alentunut LVEF tai diastolinen toimintahäiriö, laajentunut oikea kammio, vaikea keuhkojen regurgitaatio tai ahtauma tai kohonneet BNP- tasot. †Vastilla VT:llä tarkoitetaan usein toistuvia PVC:tä ja/tai kestämätöntä VT:tä. ACHD viittaa aikuisten synnynnäiseen sydänsairauteen; BNP, B-tyypin natriureettinen peptidi; EP, sähköfysiologinen; ICD, implantoitava kardiovaskulaarinen defibrillaattori; LVEF, vasemman kammion ulosheitto-osa; PVC, ennenaikaiset kammiokompleksit; SCD,</3%Moderate> SCD,</3%Moderate> heart death; TOF, Fallot Tetralogy; VA, ventricular arrhythmias; and VT, chamber tachycardia. Synopsis Fallot 's (TOF) synopsis Tetralogy is defined as congenital heart disease with RVOT blockage and ventricular septic failure, which often requires the right ventricle to position the pulmonary artery canal or to replace the pulmonary valve; contains the tof and the right ventricle of the double socket. Moderate complexity congenital heart disease is defined as congenital heart disease, which requires intractal surgical repair, other than individual atrial and ventricular septal defects; includes deviation of TOF, aortic htauma, aortic coaarchation and tricuspid valve Ebstein. Severe complexity congenital heart disease is defined as cyanotic congenital heart disease, which requires intractable repair in infancy, often with stoppage procedures; includes the transposition of major arteries, truncus arteriosus and single-chamber anatomy (Figure 16). Support text per recommendation1. VT's link to RV hemodynamic abnormalities was first determined in patients with corrected tof. S10.8-33 Several studies since then have shown a correlation between hemodynamic residue and ventricular dysfunction with VT or SCD risk in patients with congenital heart disease. S10.8-1,S10.8-3–S10.8-6,S10.8-18,S10.8-34–S10.8-36 Presentation frequently or complexly VA may indicate worsening hemodynamic function, coronary artery endangerment or reduced perfusion in the insertion of ventricular hypertrophy. The evaluation may also include exercise testing to assess functional capacity. S10.8-35 Careful assessment of hemodynamic state to optimise leadership is important. S10. During hemodynamic evaluation, potentially treatable hemodynamic residual problems such as outflow channel queuing or significant regurgitation that may benefit from either a catheter or surgical procedure may be identified. S10.8-3,S10.8-7,S10.8-10,S10.8-12,S10.8-37 Patients with clearly impaired ventricular function, increased end-

diabetes pressures or pulmonary hypertension should be treated for underlying haemodynamic problems as part of the management of arrhythmias.2. The correlation of residues of hemodynamic abnormalities with VA has been studied most extensively in patients with corrected TOF, where RV hypertension, residual lung outflow blockage or regurgitation and RV expansion are risk factors for VT/ SCD. S10.8-1,S10.8-2,S10.8-4,S10.8-8,S10.8-33,S10.8-34,S10.8-36 In these studies, frequent PVC's correlate with clinical or inductive sustainable VT risk. The combined approach of structural abnormality surgery with map-controlled arrhythmia surgery has been used with success, S10.8-3,S10.8-8,S10.8-10,S10.8-12, but removal may be limited by VT's deep endocardial or LV origin and operational mapping limitations; empirical approach to VT surgery is usually not because its effectiveness is limited and carries a risk of ventricular arrhythmias. S10. Replacement of the pulmonary valve in tof patients may lead to an improvement in haemodyanmics and functional state, but may not eliminate the risk of VTS10.8-3, S10.8-12; Postoperation reassessment for the need for ICD will be carried out after an early recovery period.Correcting hemodynamic/structural abnormalities affecting VT may improve ventricular function and reduce symptoms, but may insufficiently prevent the risk of subsequent VT or SCA. The use of ICD in adult patients with congenital heart disease in secondary contraception is approximately 50% of implantations currently, in middle age 36-41 years. S10.8-13-S10.8-17 In patients with congenital heart disease, shock rates are between 3% and 6% per year, and the number of shocks suitable for secondary contraceptive topics has increased similarly or slightly. S10.8-14,S10.8-15,S10.8-17 Patients with adult congenital heart disease have more complications and inappropriate shocks than other adult populations. S10.8-13-S10.8-17,S10.8-394.ICD implantation challenges in patients with adult congenital heart disease may include anatomical complexity, intra-carniac shunts and limited vascular access to the ventricle. In patients with adult congenital heart disease receiving ICD, the number of complications has increased by 26-45%, as has the number of inappropriate shocks in 15-25% of patients. S10.8-13-S10.8-16,S10.8-40 Limited studies on the use of subcutaneous implantable cardiovascular defibrillator implantation, especially in patients with single-chamber anatomy,S10.8-41 reported improvement by using the right parasternal lead positioning for screening. S10. Patients with one chamber or systemic right ventricle chamber may not tolerate defibrillation threshold testing, resulting in a failure of the multiorganic system. Patients with complex anatomy, such as older patients with monotonous physiology, or patients with significantly impaired ventricular function with significant hypertrophy or multiple prior surgeries may benefit from prior consideration of a heart transplant prior to progression of renal or hepatic dysfunction.5. unexplained fainting, frequent PPCs, etching cardia, QRS duration ≥180 ms, deterioration of LVEF or diastolic dysfunction, enlarged right ventricle, severe pulmonary regurgitation or htauma or elevated BNP levels. Patients with TOF physiology and suboptimal hemodynamic state are more likely to have permanent VT, S10, 8-18, S10, 8-19, S10, 8-33, S10, 8-35 and induced long-lasting VT correlates with an increased risk of SCA in a multicentre cohort study. S10.8-19 if residual abnormalities are important, catheter or surgical treatment of important lesion prior to the consideration of ICD implantation.6.In multicentre cohort, unindested long-lasting VT in tof patients was an independent risk factor for subsequent clinical VT or SCDS10,8-19; the early study had cardiomegaly and previous palliative shunts. Patients with corrected TOF account for approximately 50% of ICD implantations of congenital heart disease in adults. S10.8-13-S10.8-16,S10.8-40 Appropriate ICD attacks occur in up to 7.7% of patients with TOF receiving ICD for primary contraception compared to 9.8% per year in patients with secondary prevention ICD. S10.8-14 Since there are a lot of inappropriate shocks in 20-30% and complications in at least 30% of patients with congenital heart disease, S10,8-14-S10,8-17,S10.8-39,S10.8-40,S10.8-43 In addition to the financial and psychological burden, shared decision-making essential.7.In primary prevention ICD in patients with recurrent continuous monomorphic VT, VT catheter ablation can be effective. S10.8-21–S10.8-25 During surgical abloiting of arrhythmia, hemodynamic correction should be considered. Patients with complex adult congenital heart disease should be cared for in experienced centers. After successful catheter ablation of VT, implantation of ICD for those without ICD is an individual decision based on the general functional and physiological state and joint decision-making. Careful monitoring of the monitoring of recurrent arrhythmia is essential.8. The greatest risk of SCD associated with reconstinated congenital heart disease reported from large concurrent cohorts is in patients with the incorporation of major arteries into at-risk repair, ebstein tricuspid valve abnormality, aorticstenosis and unconventional physiology. S10.8-44-S10.8-47 Patients with senning or mustard atrial fibrillan correction should increase the risk of SCA, especially during exertion. S10. Atrial fibrillament is incompatible, limiting the ability to increase volume, and may be associated with pulmonary blood vessels htauma and increased final diastolic pressures. Rv ischemistry and infarction occur and perfusion defects have been observed in myocardial infarction perfusion studies >40% of patients in this population. S10.8-49,S10.8-50 Cardiac arrest risk factors in patients with blood transfusion and atrial fibrillal correction, include previous closure of ventricular septic error, symptoms of HF, atrial arrhythmias, RVEF <30% to 35% and QRS duration ≥140 ms. S10.8-48,S10.8-51 In a single multicentre study assessing the results of ICD implantation in patients with previous atrial fibrillation lack of beta-predator was associated with a high risk of appropriate ICD treatment. S10. 8-26 Eteishythmias often precede VT in transfusion, and treatments for etheriac caracardia, including catheter ablation, tachycardia-inhibiting syncing algorithms and beta blockers, are important to reduce ICD shocks. S10.8-26,S10.8-52,S10.8-539.The risk of sepatic disease increases in patients with adult congenital heart disease compared to the general population, and the median age of death varies between the ages of 30 and 49. S10.8-27,S10.8-44,S10.8-47,S10.8-54,S10.8-55 The risk of SEPA diseases is highest in patients with moderate to severe complexity congenital heart disease, accounting for approximately 25% of the cause of death in the heart. S10.8-5,S10.8-27,S10.8-28,S10.8-44–S10.8-46,S10.8-55,S10.8-56 Patients, with septic defects and a positive family history of septic defects, cardiomyopathy or bundle/lead defects may have a gene mutation of NKX2.5, increasing the risk of early SCD; genetic testing and early consideration of ICD implantation, if justified. S10.8-57-S10.8-59 Patients with corrected complex forms of congenital heart disease have undergone multiple intracardiac intervention surgeries in the first few decades, resulting from hypertrophy and the risk of subendocardial ischemia, as well as scar formation affecting VT/VF. Risk factors for SCD include increased complexity of heart disease, VA, SVT, gradual increase in QRS duration, systemic ventricular dysfunction and subpulmonary ventricular dysfunction. S10.8-1,S10.8-5,S10.8-6,S10.8-14,S10.8-28,S10.8-29,S10.8-2 It is unrealistic to extrapolate data on specific measures for the functioning of the chambers of NICM adult patients, which justify implantation of primary prevention ICD. The development of unexplained fainting in patients with moderate to severe complexity in adult congenital heart disease may be a mixing risk; Electrophysiological research, taking into account ICD as primary contraception, may be useful.10.ICDs implanted in adult patients with congenital heart disease aged 40 and 50 to primary contraceptives are now >40-67% of implanted devices in patients with adult congenital heart disease. S10.8-13,S10.8-15,S10.8-16,S10.8-41 In these patients, appropriate shocks are administered in 14-22% of patients during the first 3-5 follow-up years. S10.8-13,S10.8-15,S10.8-16 In patients with congenital heart disease and severely depressed ventricular function or one ventricular anatomy, defibrillation threshold testing may pose too high a risk. In patients without blood vessels or previous fontan repairs, there is a risk that hemortism will be re-operation epicardial ICD implantation may outweigh the potential benefits, and assessment of transplants may be better. Subcutaneous implantable cardiooverter defibrillator implantation may be an appropriate option for some patients. S10.8-42,S10.8-5311.Adult patients with complex adult congenital heart disease typically have varying degrees of hypertrophy and ventricular dysfunction, increasing their risk of worsening ventricular function with antiarrhythmia drugs. In the only large study of antiarrhythmics for congenital heart disease, phlecainide was associated with proarrhym in 5.8% of patients and SCA in 3.9% of patients. S10. The use of amiodaron is usually reserved for refractory symptomatic VA or asymptomatic VA, which can exacerbate ventricular dysfunction due to the high risk of adverse reactions such as thyroid dysfunction, especially in women and patients with monotonous physiology. S10.8-31,S10.8-3211. Defibrillators non-transvent ICDs1.1. Subcutaneously implanted cardiovascular defibrillatorSynopsis Patients being considered for subcutaneous implantable cardiooverter defibrillator, pre-implanted ECG to determine QRS-T wave morphology are necessary to reduce the risk of aliasing VT/ VF and risk of inappropriate shocks. S11.1-9–S11.1-11 Subcutaneous implantable cardiooverter defibrillator is implanted using mainly anatomical landmarks, minimizing the need for x-through. The subcutaneously implanted cardiooverter defibrillator consists of a pulse generator placed on the midax cellar line between the fifth and sixth intermediates and lead with 2 sensing electrodes and a shocking coil placed under the skin next to the sternum. Like transvent ICD, the pulse generator housing acts as an electrode for defibrillation, but it can also serve as an accessory to an electrode sensor. A subcutaneously implanted cardiooverter defibrillator is not able to achieve adequate identification of arrhythmias in all patients and electrocardiogram screening is necessary to evaluate identification prior to implantation. S11.1-10,S11.1-11 Some are in favour of exercise testing after implantation of the device to ensure proper identification with the exercise. Both transvensive and subcutaneous implantable cardiovascular defibrillators are SVT-VT discriminatory, which can be programmed to facilitate discrimination against SVT against VT; However, these discriminators do not always work. If long-term treatment with VT is confirmed, disssation of arrhythmias is given. All ICDs provide shocks to stop VT or VF, but the shocks of the awake patient are painful and are associated with a reduction in QoL. Transvenual ICD is capable of bradycardia pace as well as antitachycardia pace, which can end many VTs painlessly. Subcutaneously cardiooverter defibrillators offer limited postshock bradycardia synchronization, but do not provide bradycardia or antitachycardia. Nniiden Nniiden implantable cardiooverter defibrillator recommendations supersede, but do not invalidate, the fulfilment of waiting times and other requirements for ICD/CRT implantation as defined in other parts of this Document. Support text per recommendation1. The subcutaneous implantable cardiooverter defibrillator was designed to avoid the need for intravenous access and some complications associated with the insertion of transvensive lead S11.1-1-S11.1-4, which include pneumothorax, hemothorax and heart tamponadi. S11.1-12 Difficulties in intravenous access can prolong implantation and sometimes lead to failed ICD implantation. These difficulties are more likely to occur in patients with limited access to the kidneys, such as patients with ESRD. In a study of 27 ESRD patients, the subcutaneously implanted cardiooverter defibrillator was not associated with an increased risk of processed complications or inappropriate shocks. S11. The risk of infection appears to be lower with a subcutaneous implantable cardiooverter defibrillator than with transvensive ICD. S11.1-1-S11.1-4 Therefore, subcutaneous implantable cardiooverter defibrillator can be preferred in patients, with a high risk of infection, such as those with a history of device infection, ESRD, diabetes mellitus, or who are chronically immunosuppressed.2.Non-randomized studies show that a subcutaneous implantable cardiovascular defibrillator reliably detects and converts VF during defibrillation threshold testing and successfully stops spontaneous continuous VT, which occurs during monitoring. S11.1-1,S11.1-13 In one study of 314 patients, the 180-day complication-free rate was 99%, and stopping VF with first shock was >90%. S11. All spontaneous VT/ VF episodes recorded in 21 patients (6.7%) was not associated with lead defects, endocarditis or bacthermia, tamponade, cardiac perforation, pneumothorax or hemotoracae in connection with a subcutaneous implantable cardiooverter defibrillator. S11. In 1 to 2,472 patients enrolled in the EFFORTLESS (S-ICD Clinical Outcome and Cost-Effectiveness Factors Assessment) Registry, S11.1-3, the number of complications-free was 94%, 360 days. The effectiveness of the first shock conversion was 88% and 100% successful clinical conversion after up to 5 shocks. In 882 patients included in the study of the release of the research equipment and the EFFORTLESS register, S11. 90.1% were discontinued with 1 shock and 98.2% were discontinued during the five blows available. The estimated 3-year inappropriate shock rate was 13.1% most due to over-insension of heart signals and mortality was 4.7%. Device-related complications occurred in 11.1% of patients. The ongoing study compares subcutaneous implantation impact on the effect transvenation with ICD for the consequences of inappropriate shocks, complications, shock efficacy and mortality. S11.1-133.The subcutaneous implantable cardiooverter defibrillator is not capable of bradycardiatating, bipolar paces or antitachycardia. Therefore, patients who need any of these types of syncing of ICD should not be offered a subcutaneous implantable cardiooverter defibrillator. S11.1-6 Some clinical scenarios may include a transverse pacemaker for bradycardia when the patient has the subcutaneous implantable cardiooverter defibrillator required; this may be carried out as long as the pace is not unipolar. Unleaded pacemakers for bradycardiatata-pace patients will be evaluated by a subcutaneous implantable cardiooverter defibrillator in the near future.11.2. Wearable Cardioverter-DefibrillatorSynopsisThe inflatable cardiooverter defibrillator is a vest-like device worn under clothes that continuously monitors the heart rhythm and automatically produces an electric shock when VF or VT is detected. This appliance is intended to be used continuously, 24 hours a day, unless the user is swimming or showering. The U.S. Food and Drug Administration has approved a wearable cardiooverter defibrillator in the U.S. for patients at risk of SCA who are not candidates for or opting out of an implantable defibrillator. S11.2-6 AHA Science Advice summarizes the information and recommendations on the use of wearable cardiovascular defibrillator. S11. The efficacy of wearable cardiooverter defibrillator in VF identification and defibrillation has been demonstrated in several studies, although rcts do not support the use of wearable cardiooverter defibrillator. Of the 3,569 patients who received the device for different reasons, there were 80 VT/ VF events in 59 patients in the U.S. manufacturer's register for at least one day with a frequency of 1.7% per patient year. First shock power was 99%, and the aftershock was 90%. Overall, 2% of patients experienced inappropriate shock. S11.2-1Sysific support text1. The removal of ICD for a certain period of time, most commonly due to infection, exposes the patient to the risk of untreated VT/SCD unless monitoring and access to external defibrillation is maintained. In one series of 354 patients who received wearable cardiooverter defibrillator, the indication was infection in 10%. S11.2-3 A wearable cardiooverter defibrillator may allow patients to leave the hospital protected from VT/SCA until a clinical situation allows the re-placement of ICD.2.The patients listed in this Recommendation are represented in clinical series and registers demonstrating the safety and efficacy of wearable ICD. Patients newly diagnosed with cardiac repair, newly diagnosed NICM, recent revascularization, cardiac inflammation and secondary cardiomyopathy are at increased risk of VT/ SCA. However, a wearable cardiooverter defibrillator is of unprotected benefit under these conditions, partly because the clinical situation may improve over treatment and time. In patients waiting for a transplant, even if life expectancy <1 year without transplantation, and depending on clinical factors such as intravennicular inotropic use and ambient VA response, a wearable cardiovascular defibrillator may be an alternative to ICD.11. Automatic external defibrillatorExternal defibrillation can save lives when used within minutes of the onset of VF. AED is an effective way to deliver defibrillation to people with out-of-hospital cardiac arrest, and its use in first response patients is safe and effective. S11.3-1-S11.3-3 Federal efforts have been effective in placing AEDs in airports/aircraft and federal buildings, while varying efforts at state and community level have been effective in placing AEDs in many. but not all, schools, sporting events, dense residential areas and airports, as well as police and fire department vehicles. S11.3-4-S11.3-7 Most states require recovery protocols with or without an AED ranking, although alternative indoor sports facilities may have higher retention rates and increase survival from other public interiors. In the S11.3-8 21 million research population, survival in hospitalization was almost double that when cardiac arrest outside the hospital was sought compared to AED. The expanded and coordinated placement of S11.3-9 AEDs in the community, including in high-risk geographic locations such as schools and organized sports arenas, can significantly increase the proportion of patients with cardiac arrest who receive AED treatment. S11.3-10 The U.S. Food and Drug Administration has approved the over-the-counter sale of AED. About 70% of SMA occurs at home, and the survival rate for hospital discharge after an AED placement in emergency care services is significantly lower for arrest at home (12%) compared to public regulations (34%). S11. However, in RCT 3-11 AEDS, home AED placement did not improve the survival of patients recovering from the appropriate location of the anus MI.S11.3-12 to reduce time delay after the onset of SCA. In addition to prevention, critical components of surviving SCA include the immediate identification and activation of the emergency response system, early high-quality resuscitation and rapid defibrillation for shock rhythms. S11.3-1312. Catheter retraining The specific aspects of VT are due to the rebirth of bundle branches. Catheter ablation is the recommended treatment for this VT that < 10% of patients with monomorphic VT and structural heart disease (see section 7.2.3). Support text per recommendation1. Bundle-branch reentrant VT can occur in any heart disease associated with slow infra-hisian conductivity. The most common mechanism is antegrade led over the right bundle branch and backwards the led over the left bundle branch, which produces the QRS morphology of the left bundle branch block during VT, which is often quick and poorly tolerated. Ablation of the catheter of the right or left bundle branch interrupts the circuit and is usually healing. After S12-1-S12-3 Ablation, severely impaired atrioventricular conduction may occur, which requires permanent paces that can have hemodynamic consequences. S12-4,S12-6 Many patients have other scar-related VTs or meet eligibility criteria due to the severity of ICD-related heart disease.2. And this is more likely in patients with non-ischemic rather than ischemical cardiomyopathy, and in arrhythmiagenic right ventricular cardiomyopathy. In the S12-7-S12-9 HELP-VT study, S12-4 epicardial ablation was needed in 30% of NICM-related VT patients and 1.2% of patients with ischemic cardiomyopathy. However, extensive QRS with the marked courting of the original QRS component and the QS complex between lateral or inferior wires during VT refers to an epicardial circuit in NICM, however, the ECG does not reliably predict epicardial VT locations in patients with a history of myocardial infarction. S12-8, S12-10 Perkardian contagions prevent percutaneous access for some patients, especially many who have had heart surgery in the past. Perkutan percardial access to mapping and ablation is associated with a severe number of complications of approximately 5% and tamponate from a RV sting or wound that may require sudden surgery or be fatal, coronary artery damage and frenin nerve damage may occur. S12-11,S12-12 Reported experience comes from tertiary referral centres.13. Postmortem evaluation SCDRecommendation specific support text1. A comprehensive postmortem protocol has been recommended for routine evaluation of subjects (typically <40 years of age) who die suddenly without prior diagnosis of the state of death and circumstances that may reasonably be part of the cause of unexpected SCD. S13-1 One study documents the added value of postmortem examination at a specialized center for cardiac pathology, S13-2, which has special value to clarify the apparent overdiagnosis of the heart muscle in non-specialised centers. Pathological findings limited to a specialized management system were demonstrated in 22% of cases. S13-9 Misdiagnosis of cardiomyopathy was reported in 37% of cases that were eventually found to be structurally normal. Etiological data from the special heart assessment are not generalizable to the general population as the age was skewed at the time of scd. In another study in severgical infection patients <1 year= to=>was 80 years of age (average, 38.2 years; median, 38 years), peak SCD incidence ranged from 31 to 60 years of age, and 5 to 7 times the number of men/women in this age group. S13-10 In the whole group, 42% of SCD was due to sechemistry heart disease, 12% viral cardiac physicism and 5% cardiomyopathy, and 15% were unexplained by autopsy. In subgroup <35 years= of= age,= 13.5%= were=d= to= to= attributeic= heart= disease= and= 24.9%= were= unexplained.= in= the= subgroup=>55 years only 0.8% were unexplained. In patients who die suddenly despite ICD, Interrogation of the ICD is important to ensure the proper functioning of the device, and can provide information about the mechanism of death.2.Comprehensive heart screening, including a 12-lead ECG, possible signal average ECG, echocardiogram and polits rhythm monitoring, or physical activity testing of first-degree relatives caused by sudden death, can identify the probable cause of cardiac death to be aroused in up to 30% of cases. S13-11–S13-13 Genetic testing should be carried out on the basis of the results of the initial evaluation. S13-3 Genetic testing in selected first-order relatives may lead to the identification of hereditary diseases, including long QT syndrome, cayelolamine-related polymorphic polymorphic tachycardia, Brugada syndrome, arrhythmiagenic right ventricular cardiomyopathy and HCM in 4-30% of families. S13-11,S13-12,S13-143. For family risk profiling, it is important to use a disease-specific genetic test panel corresponding to autopsy findings. It is important to profile family members of the SCD victim suspected of having hereditary cardiomyopathy in the autopsy. While phenotyping of phenotyping of surviving family members is crucial, scd-proband genotyping provides a mechanism for effective monitoring of relatives with the disease-causing mutation found in proband. In order to collect high-quality DNA for such testing, coroners, hospital pathologists and private pathologists need standards to collect and store samples for subsequent genetic testing. Family members of the suddenly dead SCD probands (first cardiac event, of natural causes, most recently alive and well within 12 hours, and in autopsy findings with uncertain structural abnormalities (e.g. ventricular hypertrophy, myocardial fibrosis or minor ischemic heart disease [n=41]), there were 51% of the genetic variants associated with sudden arrhythmia deaths while 47% in the comparison group with proband autopsies</35> </1> </1> completely negative. S13-154. Identifying the genotype can make family screening easier. S13-1614. Terminal CareSynopsisA particularly challenging area of medicine recognizes when life-prolonging treatments can become burdensome or even harmful. This is especially true at the end of life in patients with ICD syndrome, where once life-prolonging disorders can only cause unnecessary mortility and anxiety for both patients and loved ones. Support text per recommendation1. Current evidence suggests that many patients are unaware of the possibility that their ICD can be deactivated without surgery. S14-1-S14-3 During decision-making, physicians do not routinely inform patients of the deactivation of ICD. S14-4 doctors even disagree on whether discussions about deactivation should take place when patients make a decision on an ICD-related procedure. S14-5 As a result, patients often do not include hopes of deactivation in pre-treatment planning documents. S14-6 Thus, surrogates usually make decisions about deactivating ICD without prior discussions with the patient. S14-6 Hypothetical scenarios allowed ICD patients to identify scenarios where they could deactivate their ICDs if they wanted to. S14-1,S14-7 This conversation may take place at any time, but it is especially important that it is disabled during the first ICD implantation, during re-implantation and during the preparation of pre-treatment plans.2.When ICDs are not phased out at the end of life, patients and families suffer unnecessarily. Families have had unpleasant experiences watching their loved one die while being repeatedly shocked by the ICD. S14-8 In one survey of hospice staff, half of those surveyed found that the deceased patient was shocked by ICD in the year before the study. S14-9 This is unnecessary and easily preventable by having caring, patient-centered conversations with patients and their loved ones. In general, patients want their doctors to start these conversations, S14-2, S14-10, so this recommendation is carefully worded to make the responsibility for starting a conversation about the clinician. Ethically, patients and surrogates are free to deactivate tachycardia. S14-11-S14-13 Most patients opt only for deactivation of antitachycardia functions, leaving the pace function on. Even end-of-life paceing (either for bradycardia or resynchronization therapy) can be an important part of a patient's QoL and can facilitate more alert and meaningful personal interaction. These differences are easily misunderstood, so they need a careful explanation.15. Shared decision-makingSynopsis Most of their lives people prefer to do everything they can prevention and prolongation of life. However, many people may get to a point in their lives where SCD is not the worst result. Patients can report desire To die in his sleep. Decisions related to S15-6 SCD can be quite emotional; in accordance with the patient's wishes, a shared decision to carry out end-of-life therapy may involve carers, such as family members or friends. Support text per recommendation1. Taking patient preferences into account is important for VA diagnosis and management decisions. Patients' preferences for invasive treatments and acceptance of SCD risk vary and may develop throughout the disease. The Writing Committee supports a common decision-making approach as part of the overall treatment of va and SCD-at-risk patients. A generally accepted definition of joint decision-makingS15-7 contains 4 elements: 1) includes at least two participants, a clinician and a patient; (2) both parties share information; (3) Whereas both parties take steps to build consensus on their preference; and (4) an agreement is reached on the implementation. Sharing a decision doesn't mean giving a patient a list of risks and benefits and telling them to make a decision – a practice some writers have called rejection. S15-8 In particular, a recommendation based on evidence or guidelines is not a shared decision-making process. Instead, a recommendation based on both evidence and an understanding of patients' health goals, preferences and values is essential to achieving real joint decision-making. The possibility of deactivation of existing ICD also needs to be discussed with patients with terminal illnesses.2. However, a patient with HF or advanced nonkardiac disease may decide to waive reimbursement of ICD if the state of health and functional status is constantly deteriorating, either from progressive HF or other competing disease. Unfortunately, research suggests that patients are poorly informed when they understand the risks, benefits and downstream burdens of their ICDs. Patients with ICD generally overestimate the benefit of this treatment and underestimate its risks. S15-1-S15-3 Similarly, patients who refuse ICD often also underestimate their personal risk of VA and SCD. S15-4,S15-5 Physician decision-making studies show that doctors often overestimate the benefits and downplay potential harm. Replacing S15-3Insimän ICD is also an important time when patients and physicians should discuss whether replacing ICD is still consistent with patients' goals. What made sense at 70 may not make sense in your 80s. Patients may have had progressive illness or poor QoL. These factors may all change the risk-benefit ratio of ICD and patient preferences.16. Cost and value considerations The key principles of value assessment as part of clinical practice guidelines have been Detail. S16-1 The financial results of clinical treatment strategies can be documented equestrically using the same study models as for the determination of clinical outcomes, including RCTs and observational comparisons. In addition, simulation models are often used to assess the value of management strategies, since cost-effectiveness studies are standard for comparing life-time outcomes, and clinical trials usually have up to a few years of follow-up. The expert group has published standards for financial modelling in health care. S16-2Cathing assessments of alternative treatment strategies for VA and prevention of freaking out have primarily assessed ICD, including several RCTS16-3-S16-7 and observational studies,S16-8,S16-9 and simulation models. S16-10-S16-14 Patients treated with ICD in all studies received higher long-term costs. The high initial cost of an ICD device and implantation procedure leads to higher long-term costs, as there are few, if any, cost savings from implanting ICD. ICDs without a resynchronization feature do not reduce hospital readmission and can increase lateness costs due to device monitoring, complications and replacement. However, the cost of the device and procedure can change significantly over time. The cost-effectiveness assessments of the ICD based on the experiment are based on three to six years of monitoring, which is significantly shorter than the standard lifetime perspective for cost-effectiveness designs. Since most of the additional costs of ICD arise immediately, while most potential effectiveness (years of survival added by ICD) accumulates over many years, estimates of the cost-effectiveness of ICD based on limited research monitoring are systematically biased to show lower value. Research-based economic studies projecting long-term ICD results have consistently found more favourable cost-effectiveness ratios than estimates limited to study monitoring. S16-4-S16-7 The lifetime simulation model used in each primary prevention ICD study also continuously reports more favourable estimates than those based on limited study monitoring. S16-11 Since the ACC/AHA Clinical Practices Guidelines for Value Assessment use lifetime estimates benchmarks in the proposed framework,S16-1 we have generally relied on model-based estimates of ICD cost-effectiveness in applying value ratings to the recommendations of this Guide. The original cost of an ICD device is similar regardless of clinical indications, so the cost-effectiveness variations of the ICD are mainly due to possible differences in clinical survival in different patient populations. The effect of the useful life added by the ICD on its cost-effectiveness increase is shown in Figure 17: 17: rapidly unfavourable when the survival period is less than 1 year, especially less than 0.5 years. This inverse relationship strongly suggests that the value given by ICD is highest when the risk of arrhythmia death due to VT/VF is relatively high and the risk of non-arrhythmia death (either heart or nonkardiac) is relatively low, so survival can be expected to increase significantly from ICD. Therefore, proper patient selection is essential to use ICD to prevent SCD. It should also be recognised that cost-effectiveness is also affected by the costs of the ICD and implantation procedure, which are likely to change significantly over time. Figure 17. Years of life* (example)*Image based on formula: Additional cost-efficiency ratio = $50,000/QALYs are added to the additional cost efficiency of the ICD. CE showed cost-effectiveness, ICD, implantable cardiooverter defibrillator; LYA, added year of life; and QALYs, quality-adjusted life cycles. Empirical evidence suggests that ICDs are not effective for primary prevention of SCD when implanted early after CABGS16-15 or acute myocardial infarction. S16-16, S16-17 Analysis of individual patient-level data from the three secondary prevention study showed significant variability (P =0, (011) Clinical efficacy of ICD in patients with LVEF ≤35% (risk ratio: 0.66) and LVEF >35% (risk ratio: 1.2). S16-19 By contrast, there is little evidence of the effectiveness or cost-effectiveness of ICD based on age or gender, among other things. S16-20 Most studies on the efficacy and value of ICD have been conducted in patients with impaired HVAC function due to a previous MI or NICM. There is little information on the efficacy or value of ICD in other potential clinical indications, such as cardiac channelopathy or HCM, although studies have shown that their potential cost-effectiveness in such patients depends on the underlying risk of secoside, and there is little evidence of its value in low-risk patients. S16-1417. The quality of lifeICD implantation has not had a significant impact on QoL in the general population of patients enrolled in RCT. However, S17-1-S17-3 However, several studies have shown that patients with inappropriate ICD shocks have a worse QoL than patients with ICD who have not had inappropriate shocks. S17-2 As ICD is designed to prevent SCD instead of reducing symptoms, it is not expected to improve QoL or functional status directly, but may have indirect negative effects on some equipment complications or indirect positive effects in some patients because of the In place.18. Evidence gaps and future research needs Show that there are still numerous advances in the prevention and management of SCD, SCD and VA in risk-spreading. These deficiencies include:Identifying the patients most likely to benefit from ICD among all ICD-eligible patients. Combinations of new tracers (including genetic and imaging marks) and tracers shall be investigated. Characterizing the role of ICD in subgroups of patients who are not well represented in key ICD studies. These subgroups include ≥ 80 years of age and patients with kidney disease, in particular dialysis patients with ESRD therapy or multiple sub-diseases. Methods for identifying and treating patients at high individual risk of second cellularisation who are not identified by the current ICD eligibility criteria, including patients within 40 days of cardiac infennopathy. Determination of the role of ICD in patients with HCM, arrhythmiagenic correct ventricular cardiomyopathy, cardiac sarcoidosis and hereditary cardiac channelopathies in prospective studies (preferably RCT). Determine the best approach for patients who are caused by replacing an optional ICD generator due to battery depletion, but who may now have a low risk for SCA, such as if a significant improvement in LVEF has occurred. More information is available on the effectiveness and effectiveness of the subcutaneous implantable cardiooverter defibrillator compared to transvent ICD techniques and the scope of the necessary tests, as well as its use with other new technologies, including unleaded pacemakers. to perform RCT scores for vt catheter ablation in ischemic heart disease and cardiomyopathies evaluating processed endpoints, mortality, suppression of arrhythmias, QoL and costs. Improving the identification of persons who do not have significant ventricular dysfunction and are at risk of SCD. Identification of SCD mechanisms and risk factors in patients with HFpEF. Develop better methods for identifying and abloiting arrhythmia substrate in structural heart disease. A better risk of deposition of sudden-death-related diseases and syndromes such as sechemistry heart disease, NICM, congenital heart disease in adults and Brugida syndrome develops. Recognizing what causes different types of long QT syndrome, calycoliamine-rged polymorphic polymorphic ventricular thycardia, Brugida syndrome, HCM and arrhythmiagenic right ventricular cardiomyopathy, and promote the relationship between genotype and phenotype, genotypical risk and genotypical tailoring of treatments from hereditary cardiomyopathies and hereditary channelpathies Patients. Definition of the most suitable and useful use of wearable cardiovascular defibrillators. Develop methods for identifying and treating patients at high personal risk for the treatment of seconds who are not identified by the current ICD eligibility criteria. Defining cmr's role in order to increase risk On behalf of the SCD. Increasing research funding in this area through existing and new mechanisms is crucial. Some have proposed research funding strategies that would provide incentives for companies in the insurance sector while supporting unresolved research objectives. Such approaches should be tested.

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