

SUDDEN CARDIAC DEATH

Relevant Guidelines

AHA/ACC/HRS 2017 Guideline on ventricular arrhythmias and prevention of sudden cardiac death

2017 AHA/ACC/HRS Guideline for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death. *JACC*. 2017

ESC 2022 Guidelines on ventricular arrhythmias and prevention of sudden cardiac death

2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. *Eur Heart J*. 2022.

Relevant Scientific Statements

AHA 2020 Scientific Statement on Sudden Cardiac Arrest.

Sawyer KN, et al. Sudden Cardiac Arrest Survivorship: A Scientific Statement From the American Heart Association. *Circulation*. 2020:CIR0000000000000747.

2020 APHRS/HRS expert consensus statement on sudden unexplained death.

2020 APHRS/HRS expert consensus statement on the investigation of decedents with sudden unexplained death and patients with sudden cardiac arrest, and of their families. *Heart Rhythm* 2021;18:e1–50.

2023 AHA Scientific Statement on Cardiac Catheterization Laboratory Management of Out-of-Hospital Cardiac Arrest

Cardiac Catheterization Laboratory Management of the Comatose Adult Patient With an Out-of-Hospital Cardiac Arrest: A Scientific Statement From the American Heart Association. *Circulation*. 2023

EHRA/HRS/APHRS/LAHS 2022 Expert Consensus Statement on Genetic Testing

Expert Consensus Statement on the State of Genetic Testing for Cardiac Diseases. *Europace* 2022

AHA 2024 Scientific Statement on Care after Cardiac Arrest

Critical Care Management of Patients After Cardiac Arrest: A Scientific Statement From the American Heart Association and Neurocritical Care Society. *Circulation*. 2024;149:e168-e200.

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Definition

Sudden cardiac death (SCD) is defined as death of cardiac origin occurring within 1 h from onset of symptoms. Sudden cardiac arrest (SCA) is sudden cessation of cardiac activity with hemodynamic collapse, typically due to sustained ventricular arrhythmia. Other definitions are provided in Table 1.¹

Epidemiology

Data on SCD may rely on inaccurate sources. Observations from a prospective autopsy study indicated that only half (55.8%) of sudden cardiac deaths defined by conventional criteria are autopsy-defined sudden arrhythmic deaths as defined by WHO criteria.² Approximately 50% of all cardiac deaths are sudden and this proportion remains the same despite the overall decrease in cardiovascular mortality the last decades. The proportion of all natural deaths due to SCD is 13%, whereas if a 24 h from onset of symptoms definition is used, it becomes 18.5%. In 2004, the Centers for Disease Control and Prevention in the US established the Cardiac Arrest Registry to Enhance Survival (CARES) to better monitor and study out-of-hospital cardiac arrests.

Out-of-hospital cardiac arrest is a leading cause of death, accounting for $\approx 50\%$ of all cardiovascular deaths.³ The estimated annual incidence in the United States for out-of-hospital cardiac arrest treated by emergency medical services is 184383 encounters, and survival to hospital discharge is estimated at approximately 11%.⁴ A similar incidence occurs probably in Europe, with reports of out of hospital cardiac arrest ranging from 0.04 to 0.2% of the population.⁵⁻⁶ The annual incidence of SCD increases as a function of advancing age, being considerably smaller in adolescents and adults younger than 30 y than it is in adults older than 35 y.⁷ Analysis of data from the Department of Defense Cardiovascular Death Registry in the USA reveals that the incidence of sudden unexplained death is 1.2 per 100,000 person-years for persons 18-35 years of age, and 2.0 per 100,000 person-years for those >35 years of age.⁸ Higher numbers were provided by the King County (Washington) Cardiac Arrest Database: 2.1 (0-2 years of age), 0.61 (3-13 years of age), 1.44 (14-24 years of age), and 4.4 (25-35 years of age) per 100,000 person-years, respectively,⁹ and in Denmark, with an annual incidence of SCD 2.3 (1-35 years of age), and 21.7 (36-49 years of age) per 100,000 persons.⁵ The annual incidence of sudden cardiac arrest among blacks was >2 -fold higher than in whites in both men and women, in the Oregon Sudden Unexpected Death Study.¹⁰ Population movement, and especially major train stations are associated with a higher risk of SCD.¹¹ Sudden cardiac death in women is less common than in men. It is more likely to be

associated with a nonischemic cause, and primary myocardial fibrosis, in particular, is more common among women with SCD.¹² The incidence of *sports-related sudden death of any cause in the general population* is estimated 0.5 to 2.1/100,000 persons per year.^{13 6, 14 15} The vast majority of sports-associated sudden cardiac arrest cases occur during middle age, but they represent a relatively small proportion (5%) of overall sudden cardiac arrest cases.¹⁶ However, although SCD is the leading medical cause of death in athletes, its precise incidence is unknown.¹⁷ It is higher in *professional athletes*, with a reported incidence of SCD as 1:8253 participants per year in the National Collegiate Athletic Association (NCAA) in the USA. Among NCAA Division I male basketball players, the incidence was 1:5200 per year.¹⁸ In other studies, the reported incidence of SCD ranges from 0.24 to 3.8:100,000 per year with the higher rates seen in African/Afro-Caribbean (black) athletes (see also Chapter 83).¹⁹⁻²¹ In a more recent study on NCAA athletes, the incidence of SCD decreased throughout a 20-year study period, but remained high among specific cohorts, including Division I Black and White male basketball players (>1:2000 over a 4-year career).²² Sports-related SCD has been found to occur 5–33-fold less frequently in *women* than in men, and the sex difference persists despite a rapid increase in female participation in sports.²³

The 10-year risk of SCD is predicted by the 2013 ACC/AHA CVD Pooled Cohort risk equations.²⁴ A newer SCD prediction model considering age, male sex, black race, current smoking, systolic blood pressure, use of antihypertensive medication, diabetes mellitus, serum potassium, serum albumin, high-density lipoprotein, estimated glomerular filtration rate, and QTc interval (but not low LVEF) has produced better results.²⁵

The changing epidemiology of cardiac arrest is demonstrated in Figure 1.²⁶

Aetiology

General population

In young persons aged less than 35 years, most SCDs are caused by potentially *inherited heart diseases*, including primary arrhythmogenic disorders (genetic channelopathies and arrhythmogenic cardiomyopathies), but coronary artery disease is also an important potential cause.¹ In adolescents, between childhood and adulthood, from ages 10 to 19 years, sudden arrhythmic death syndrome and myocardial diseases are the most common causes.²⁷ Arrhythmogenic cardiomyopathy, hypertrophic cardiomyopathy, and coronary anomalies are associated with sudden death during exercise

From the age of 35 years, *coronary artery disease* becomes the most common cause of SCD, although potentially inherited heart diseases remain a common cause of SCD at least until the age of 50 years (Figure 2).¹ In an autopsy study, leading causes of presumed sudden cardiac deaths were coronary disease (32%), occult overdose (13.5%), cardiomyopathy

(10%), cardiac hypertrophy (8%), and neurological (5.5%).² In the large Swedish Cardiopulmonary Resuscitation Registry, myocardial ischaemia or infarction as aetiology decreased during 2004–20 from 67.4% to 28.3%, although together with heart failure were the main causes of cardiac arrest.²⁸ In the POST SCD study, in a 12-year countywide postmortem histopathological investigation, MI accounted for 41% of community autopsy-confirmed SCD, with MINOCA comprising approximately 10% of acute MI cases. RCA disease was as common as LAD disease among SCDs due to MI, and one-third of SCDs due to acute MINOCA had nonarrhythmic causes (wall rupture or pump failure).²⁹ Two-thirds of community arrhythmic SCDs occurred in individuals without diagnosed disease despite substantial cardiac pathology; half of these “silent” arrhythmic deaths had occult MI or DCM.³⁰ In the *young* (<35 years), a significant cardiac abnormality is not found in up to 40% of the cases and the most common cause is presumed to be arrhythmias, mostly in the context of an apparently normal heart.^{9 31–33} **Females** survivors of cardiac arrest are less likely to have underlying coronary artery disease (45%), whereas valve disease and dilated or arrhythmogenic cardiomyopathy are more common compared to men.³⁴ Biomarkers such as total cholesterol to high-density lipoprotein cholesterol ratio, cardiac troponin I, NT-proBNP, and high-sensitivity CRP, are all independently and additively associated with sudden death.³⁵ In individuals aged <50 years, sudden death may also be caused by noncardiac diseases, such as pulmonary embolism, meningitis and cerebrovascular bleeding, in 28% of cases.³⁶ One or more established cardiovascular risk factors (obesity, diabetes mellitus, hypertension, hyperlipidemia, smoking) may be found in 58% of SCD cases in the young.³³

Following *acute MI* there is increased risk during the first months either due to tachyarrhythmias or other complications such as re-infarction or myocardial rupture,³⁷ and myocardial scar predisposes to monomorphic VT. Acute coronary occlusions are present in 20% patients with out of hospital cardiac arrest without ST-segment elevation, irrespective of hemodynamic status. Acute coronary occlusions are typically associated with chest pain before cardiac arrest and an initial shockable rhythm.³⁸ Apart from patients with ST-segment elevation, patients resuscitated from a shockable rhythm should be subjected to coronary angiography.^{39–41} However, although most patients with a cardiac arrest have demonstrable coronary artery disease, less than half seem to have suffered an acute myocardial infarction.⁴² ⁴³ Only 38% of cardiac arrest survivors will develop evidence of myocardial infarction,⁴⁴ and the use of tenecteplase during advanced life support for out-of-hospital cardiac arrest did not improve outcome.⁴⁵ Sudden death characterizes the mode of death in 30–50% of patients with chronic heart failure and a reduced ejection fraction.⁴⁶ Cascading failure can become manifest electrically as ventricular tachyarrhythmias or as acute mechanical failure, which manifest as asystole, bradyarrhythmia, or electromechanical dissociation.⁴⁶ The strongest predictor of sudden cardiac death remains reduced LVEF, with a cutoff of $\leq 35\%$, as determined in

randomized, controlled studies, more individuals with LVEF >35% die suddenly than those with lower LVEF although the exact proportion of arrhythmic vs non-arrhythmic deaths is not known.⁴⁷ Cardiomyopathy related to obesity or alcoholism, fibrotic cardiomyopathy and cardiac sarcoidosis are the most common causes of **non-ischemic** sudden cardiac death.^{48, 49}

Idiopathic VF is defined as a resuscitated cardiac arrest victim, preferably with documentation of VF, in whom known cardiac, respiratory, metabolic and toxicological etiologies have been excluded through clinical evaluation.⁵⁰ Up to 30% of patients initially diagnosed with IVF qualify for a specific disease during follow-up.⁵¹ Several mutations of ion channels coding and other genes have been associated with idiopathic ventricular fibrillation.⁵² In apparently healthy patients with preserved ejection fraction (CASPER Registry), an aetiological diagnosis was possible in approximately half of cardiac arrest survivors, the rest being considered cases of idiopathic VF presumably due to intrinsic electric abnormalities, such as early repolarization.⁵³ Short-coupled ventricular fibrillation likely was found responsible for at least 6.6% of unexplained cardiac arrest in this registry.⁵⁴

Coronary spasm is also a cause of cardiac arrest, particularly in male smokers with minimal or no preexisting coronary artery disease.⁵⁵ Among out-of-hospital cardiac arrest survivors without structural heart disease, provokable coronary spasm and ventricular arrhythmias at electrophysiology testing are common and can be seen in Brugada syndrome.⁵⁶

Mitral valve prolapse in female patients with ECG repolarization abnormalities, and frequent complex ventricular ectopy, has also been associated with out of hospital cardiac arrest,^{57 58} with a prevalence of 6.6%.⁵⁹ Higher **FT4** levels are associated with an increased risk of SCD, even in euthyroid patients.⁶⁰ SCD is a frequent mode of death in **myotonic dystrophy 1 (Steinert's disease)**, especially in patients with a history of non-sustained VT.⁶¹ SCDs account for most cardiac and many non-AIDS natural deaths in **HIV**-infected patients.⁶² **Lower socioeconomic status, depression, anxiety, social isolation, and acute emotional stress** have all been linked to an increase risk of SCD.^{63, 64} An association of **air pollution** (fine particulate matter with an aerodynamic diameter <2.5 µm and ozone) with out of hospital cardiac arrest has also been demonstrated.⁶⁵ Visceral **obesity** increases the risk of sudden cardiac death.⁶⁶ A potential correlation between **high caffeine consumption** (>10 cups of coffee per day) as well as **high-energy drinks** that contain high doses of caffeine and stimulating ingredients such as taurine and guarana, and sudden cardiac arrest has been reported.⁶⁷ **Anticholinergic drugs** (such as atropine, diphenhydramine, chlorpromazine) use that results in high anticholinergic burden has been associated with SCD.⁶⁸ In families whose members have had SCD, genetic testing may diagnose an inherited arrhythmia (**genetic channelopathy**) in up to 29%.⁶⁹ Several studies have also demonstrated a **familial predisposition** to SCD that may or may not be related to genetic channelopathies.^{70, 71} A **circadian variation** is documented with the peak incidence of SCD occurs in the morning

hours from 6 am to noon (and is blunted by beta blockers) with a smaller peak in the late afternoon for out-of-hospital VF arrests, and is highest on Monday.⁷²

Commotio cordis occurs across a spectrum of non-sport-related settings including assault, motor vehicle accidents, and daily activities.⁷³

Athletes

The overall incidence of *sudden cardiac arrest and death in athletes* varies with age. In the adolescent age group (13 to 17 years), it is reported to be 1 case in 66,000 to 88,000 athlete-years, with a higher incidence in specific populations, such as elite male soccer players (1 in 14,794 athlete-years) and male basketball players (1 in 21,000 athlete-years).⁷⁴ Population-based data on the incidence of sudden death from cardiac causes show a sharp increase beginning at the age of 25 years, which reflects a greater prevalence of coronary disease among adults than among younger persons.⁷⁴ The incidence of cardiac arrest during sports in men is 13 times higher than in women.⁷⁵

In *professional athletes* 11 to 40 years of age, sudden death from cardiac causes with a pathologically normal heart on autopsy — autopsy-negative, sudden, unexplained death — is the most common finding (20%), suggesting an arrhythmia due to a channelopathy as the cause of death.⁷⁴ Among unselected cases of sudden cardiac arrest with a negative autopsy in young people, a molecular autopsy reveals a relevant cardiac gene variant in 27% of cases.³² Coronary artery anomalies and coronary artery disease account for approximately 18%, LV fibrosis and hypertrophy for 11%, and HCM for 12% of cases.³² *Commotio cordis* caused by relatively innocent impact to the chest (overall incidence 2%), is one of the leading causes of SCD in sports such as baseball, lacrosse, and hockey.⁷⁶ Findings from the RACER initiative indicate that marathons and half-marathons are associated with a low overall risk of cardiac arrest or sudden death (1/100000), most commonly attributable to hypertrophic cardiomyopathy (26%) or atherosclerotic coronary disease (16%).⁷⁷ However, some of these cardiac arrests might have been provoked by heat stroke.⁷⁸ In the UK Registry on sudden death in sports, myocardial disease including LV hypertrophy and/or fibrosis was detected in 40% of cases, ARVC in 13% and HCM in 6%. Almost 40% of athletes died at rest.⁷⁹ In a following report on young athletes, arrhythmogenic cardiomyopathy, coronary anomalies and *commotio cordis* were the main causes of sudden death.²⁷ In the RACE Paris Registry life-threatening/fatal events during long distance races were rare, most often unpredictable and mainly due to acute myocardial ischaemia.⁸⁰ CAD is the leading cause of sudden cardiac death in athletes >35 years of age (i.e. ‘Masters athletes’).^{81, 82} Vigorous exertion can trigger cardiac arrest or SCD, especially in untrained persons, but habitual vigorous exercise diminishes the risk of sudden death during vigorous exertion.⁸³ Professional bodybuilders had a higher risk of SCD than amateurs.⁸⁴ Most studies have found inverse associations between regular physical activity and SCD.⁴⁴ In female athletes, a significant proportion of sports-

related SCD causes are non-structural or unexplained after autopsy, and channelopathies may play an important role.²³ For the interpretation of the ECG in athletes see the relevant Chapter.

Pathophysiology

Mechanisms

Ventricular tachycardia or fibrillation was thought to be the most common cause of out-of-hospital cardiac arrest, accounting for approximately three quarters of cases, the rest 25% caused by bradyarrhythmias or asystole.^{85, 86} More recent studies suggest that the incidence of ventricular fibrillation or pulseless ventricular tachycardia as the first recorded rhythm in out-of-hospital cardiac arrest has declined to less than 30% in the past several decades.⁸⁷⁻⁸⁹ From 1980 to 2000, an increasing proportion of SCA cases presented as PEA or asystole than VF. This may reflect increasing beta-blocker use or better treatment of coronary disease.²⁶ This has an impact on resuscitation, because survival is lower for non-VF compared with VF arrests, risk prediction, and understanding. Pulseless electrical activity (electromechanical dissociation) and asystole are proportionally more frequent mechanisms than VT/VF, with recent data demonstrating an incidence of 19-23%, with the remaining patients (approximately 50%) having asystole.⁹⁰ Non-shockable rhythms (brady-asystole and pulseless electric activity) now predominate and are deemed to be largely non-survivable. However, non-shockable rhythms may evolve to shockable ventricular fibrillation or pulseless VF/VT during the course of resuscitation in up to 25% of patients.⁹¹ The majority of survivors are in the subgroup of persons whose initial rhythm is ventricular fibrillation or pulseless ventricular tachycardia.⁸⁸ Ventricular fibrillation is a cause of cardiac arrest and if untreated the arrhythmia is usually fatal, but spontaneous reversions to sinus rhythm have been recorded. Non-arrhythmic mechanisms such as myocardial rupture or aortic aneurysm rupture may also result in SCD.

Survival after Cardiac Arrest

Specific recommendations for acute management of cardiac arrest are presented in Chapter 60. The aggregate overall survival after out of hospital cardiac arrest remains low approximately 7.6%, being better with VT/VF arrests than pulseless electrical activity.⁹² Survival with a favorable neurologic outcome is highest among individuals who present with a witnessed shockable rhythm, received bystander cardiopulmonary resuscitation, achieve return of spontaneous circulation within 15 minutes of arrest, and have evidence of ST-segment elevation on initial ECG after return of spontaneous circulation.³ A population-based cohort study on out of hospital cardiac arrest survivors in Ontario, Canada, detected an

improved 30-day survival from 9.4% in 2002 to 13.6% in 2011.⁹³ Survival is better in places with facilities for bystander resuscitation.¹⁴ Improved survival rates are also more prominent in patients 18-80 years of age.⁹⁴⁻⁹⁵ Recent data from the US Cardiac Arrest Registry to Enhance Survival (CARES), suggest that rates of survival from out-of-hospital cardiac arrest have improved among sites participating in a performance improvement registry.^{4,96} Delivery of automated external defibrillators by drones together with use instructions to bystanders is a new, exciting possibility.⁹⁷ Quality of life of survivors is good at least for the next 12 months,⁹⁸ and in the majority of survivors is similar to that of the general population.⁹⁹ Long-term survival among patients who have undergone rapid defibrillation after out-of-hospital cardiac arrest is similar to that among age-, sex-, and disease-matched patients who did not have out-of-hospital cardiac arrest.⁹⁹ The number of people surviving sudden cardiac arrest is increasing, but physical, cognitive, and emotional effects of surviving cardiac arrest may linger for months or years.⁴ Several noncardiac conditions can predispose to high mortality among initial survivors of out-of-hospital cardiac arrest, including stroke, sepsis, and pulmonary embolism.¹⁰⁰

Public access defibrillation (Table 2) may increase this survival to 40% or even more.¹⁰¹⁻¹⁰³ Defibrillators in public locations, such as train stations,¹¹ however, may reduce the incidence of sudden cardiac death only when they are accompanied by training programs of the local population.¹⁰⁴

Investigations in Survivors

A full cardiac assessment is needed in SCD survivors.⁵⁵ Patients presenting with ventricular fibrillation or sustained monomorphic ventricular tachycardia are at a considerable risk of recurrence, particularly in the presence of reduced left ventricular function. Studies of out-of-hospital cardiac arrest survivors as well as of patients with sustained ventricular tachycardia have shown that the actuarial incidence of sudden death at two years following the presenting arrhythmia varies from 15 to 30%. Up to 74% of patients with out of hospital cardiac arrest have VF recurrence during prehospital care and the time in VF is associated with worse outcome.¹⁰⁵ **Early coronary angiography** in patients resuscitated as a result of a shockable rhythm with immediate PCI improves survival.^{40,41} In the absence of ST elevation, the usefulness of this approach is not established (Figure 3).^{3,106,107} A diagnostic pathway for possible primary myocardial infarction following sudden death is presented in Figure 4.¹⁰⁸ However, early coronary angiography may be of benefit even in the absence of ST elevation on the by potentially salvaging myocardium and decreasing the incidence of systolic heart failure in survivors.¹⁰⁹ Transfer to a dedicated cardiac arrest center for expedited delivery to the cardiac catheter laboratory also did not reduce mortality.¹¹⁰ The **CREST score** (coronary

artery disease, initial heart rhythm, LVEF<30%, shock at the time of admission, and ischemic time >25 minutes), based on data from the International Cardiac Arrest Registry, is also useful in determining which patients may benefit from urgent coronary angiography, percutaneous revascularization, or mechanical circulatory support after resuscitation.¹¹¹ An **ECG score** may also identify patients with coronary artery disease prone to sudden arrhythmic death.¹¹² The **CAHP** (Cardiac Arrest Hospital Prognosis) score represents a simple tool for early stratification of out of hospital cardiac arrest survivors. It considers the following variables that are independently associated with poor neurological outcome: age, non-shockable rhythm, time from collapse to basic life support, time from basic life support to return of spontaneous circulation, location of cardiac arrest, epinephrine dose, and arterial pH.¹¹³ The differentiation between disease-causing mutations and irrelevant genetic variants is not always possible. In the Cardiac Arrest Survivor with Preserved Ejection fraction Registry, **genetic testing** with whole-exome sequencing identified a pathogenic or likely pathogenic variant in arrhythmia or cardiomyopathy genes in 10% of cases.¹¹⁴ Recommendations for genetic testing in patients with ventricular arrhythmias and/or aborted sudden cardiac death are provided in Table 2.

Family members of young SCD victims are also at increased risk for ventricular arrhythmias and ischemic heart disease. Screening of first-degree relatives, especially those <35-year old, is important.¹¹⁵ When findings suggest cardiomyopathy or genetic channelopathy evaluation of family members is also necessary. Examination of relatives (cascade family screening) may have a significant diagnostic yield.¹¹⁶ Investigations of a genetic basis of SCD such as the candidate gene approach has explored the potential association between SCD in CAD and genes associated with genetic channelopathies, and genome-wide association studies are promising (Figures 5-9 and Table 2).¹¹⁷ However, where a cardiac arrest survivor has been comprehensively evaluated and idiopathic ventricular fibrillation is diagnosed, the yield of clinical screening of relatives is low.¹¹⁸

The following tests are useful for establishing a diagnosis in SCD survivors:

ECG (WPW syndrome, HCM, long QT).

Echocardiography (heart failure, cardiomyopathies, valve disease, congenital heart disease).

Coronary angiography (coronary artery disease, congenital coronary anomalies, coronary spasm).

Exercise test (ischemia, LQTS, CPVT).

Electrophysiology testing (induction of arrhythmia, pharmacologic provocation for Brugada, LQTS, CPVT). Procainamide testing may provoke a Brugada pattern irrespective of baseline ECG and should be considered in the workup of SCD.¹¹⁹

Cardiac MRI (ARVC, sarcoidosis, myocarditis, myocardial injury from coronary

spasm).

Cardiac implantable electronic devices systematic postmortem interrogation is useful to determine the cause and timing of death.¹²⁰

Genetic testing is indicated when an inherited phenotype is detected (ARVC, Brugada, CPVT, LQTS), and in sudden cardiac death in the young.

Cardiac biopsy may also be needed in elusive cases.

A **molecular autopsy** may also be considered as part of the community forensic investigation to enhance prevention for other family members (Table 3). Thus, in cases of documented sudden cardiac death without an obvious cause, collection of postmortem blood (in EDTA, ie the purple-top tube to enable DNA extraction), for subsequent DNA analysis may identify a cause of death in up to 30 of cases.¹²¹ Combining post-mortem genetic testing with clinical evaluation of relatives may increase the diagnostic yield to 40%, and the main causes of death are CPVT and LQTS.¹²² A forensic examination, including a toxicology screen, may establish the diagnosis in cases of traumatic, toxic, or cardiac causes, whereas a negative pathological examination, suggests a genetic channelopathy.¹²³

Postmortem CMR is also a valuable tool to non-invasively document pathological findings such as myocardial infarction or severe myocardial hypertrophy,¹²⁴ and postmortem computed tomography coronary angiography is now possible (Table 4).¹²⁵ However, despite every effort, up to half of the causes of cardiac arrest may remain unexplained. Recommendations for evaluation of relatives of sudden arrhythmic death syndrome decedents are provided in Table 5.

Athletes who survive sudden cardiac arrest can return to sports without excess risk after undergoing appropriate disease-specific treatment. For many — although not all — cardiac diseases, professional societies now view a return to play after sudden cardiac arrest as reasonable or something that may be considered, through shared decision making, with emergency action plans and follow-up in place.³²

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