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Prevalence and Spectrum of Sudden Cardiac Death Predisposing Diseases: Are They the Same for the Athlete as the Non-Athlete?

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Introduction

Sudden cardiac death (SCD) is a rare but devastating problem in young people. The sudden unexpected death of a young athlete is a highly visible event that stimulates considerable concern. It has now become common for the media to report SCDs especially when it occurs in athletic teenagers while playing sports. Such reports create understandable anxiety and elicit clamor to do something to prevent SCD in the young. These uncommon but devastating catastrophes are usually proven to be the consequence of a variety of unsuspected congenital or acquired cardiovascular diseases. It is important to understand the incidence of SCD and the causes of SCD in young people in order to design rational approaches to the prevention and management of this devastating problem. This manuscript will highlight the prevalence and spectrum of SCD predisposing diseases and how they are affected by athletic participation. Current guidelines on how to limit athletic participation in patients with these diseases will also be addressed.

Epidemiology of SCD

So far there have been limited population based studies on SCD in children in the United States and Europe estimating the incidence between 1 and 3 per 100,000 person-years (table 1). Note that this is per population and not per children in the population. The precise frequency with which SCD occurs in young athletes is not known. There are a number of practical obstacles to the collection of such data. Estimates that rely on reporting from individual schools and institutions, as well as on media accounts, probably underestimate the occurrence of these events [22]. Most studies on the prevalence of SCD are retrospective and based upon death certificates. Such studies carry intrinsic bias and can be highly inaccurate. Also, the incidence of sudden cardiac arrest is clearly higher than that of SCD especially with the recent emphasis on CPR education and widespread availability of automatic external defibrillators.

In 1985, a report [11] from the Mayo Clinic of Olmsted County, Minnesota reported the incidence of sudden death based on review of death certificates in that county in all people aged between 1 and 22 years between January 1950 and October 1982. Based on the fact that only 12 out of 515 deaths in this age group (2.3%) were thought to be sudden and unexpected deaths, the incidence was reported to be 1.3 cases per 100,000-person years. Of these 12 deaths, 7 were thought to be due to cardiac causes.

Chugh et al. [5] from Oregon reported the incidence of SCD in children based on a three year county-wide study. This population based study found that in the three-year period, 33 children met criteria for SCD. The burden of pediatric sudden death was low (3% of all sudden deaths), but 90% occurred before the age of 1 year, and the majority were diagnosed as Sudden Infant Death Syndrome (SIDS) (70% of overall sudden deaths in children). The pediatric annual incidence rate per 100,000 was 1.7, compared with 60/100,000 for all ages. The pediatric annual incidence rate per 100,000 children was 7.5. In contrast to an adult survival rate of 8%, none of the children survived to be discharged from hospital.

To estimate the absolute number of sudden deaths in US competitive athletes, Maron et al. [23] assembled a large registry over a 27-year period using systematic identification and tracking strategies. They identified a total of 1866 athletes (19±6 years of age) who died suddenly or survived cardiac arrest throughout the United States from 1980 to 2006 in 38 diverse sports. Sudden deaths were predominantly due to cardiovascular disease (56%), but causes also included blunt trauma that caused structural damage (22%), commotio cordis (3%), and heat stroke (2%). Among the cardiovascular deaths, the highest number of events in a single year was 76, with an average of 66 deaths per year over 6 years; 29% occurred in blacks, 54% in high school students, and 82% with physical exertion during competition/training, whereas only 11% occurred in females. The two most common cardiovascular causes were hypertrophic cardiomyopathy (36%) and congenital coronary artery anomalies (17%). This national registry estimated the frequency of SCD at 0.6 deaths per 100 000 person-years. This rate is similar to that reported in competitive athletes over a recent 11-year period from the Veneto region of northeastern Italy (0.87 deaths/100 000 person-years), in which screening routinely included a 12-lead ECG, [6] as well as from the state of Minnesota (0.93 deaths/100 000 person-years), in which only history and physical examination were used [28].

In order to access the risk of SCD in adolescents and young adults engaged in sports Corrado et al. [7] did a 21-year prospective cohort study of all young people of the Veneto Region of Italy from 1979 to 1999, There were 300 cases of SCD, producing an overall cohort incidence rate of 1 in 100,000 persons per year. Fifty-five SDs occurred among athletes (2.3 in 100,000 per year) and 245 among non-athletes (0.9 in 100,000 per year). The relative risk of SCD among athletes versus non-athletes was 1.95 for males and 2.00 for females. The higher risk of SCD in athletes was strongly related to underlying cardiovascular diseases such as congenital coronary artery anomaly, arrhythmogenic right ventricular dysplasia, and premature coronary artery disease.

Recently, Harmon et al. [16] attempted to come up with an estimate of the incidence of SCD in National Collegiate Athletic Association (NCAA) student-athletes. From January 2004 through December 2008, all cases of sudden death in NCAA student-athletes were identified by use of an NCAA database, weekly systematic search of public media reports, and catastrophic insurance claims. During the 5-year period, there were 273 deaths and a total of 1,969,663 athlete participant-years. The incidence of SCD was 1:43,770 participants per year. Among NCAA Division I male basketball players, the rate of SCD was 1:3100 per year. Therefore from population based studies it appears that the incidence of SCD in both athletes and non-athletes is extremely low.

How are athletes different?

A competitive athlete has been defined as one who participates in an organized team or individual sport that requires regular competition against others as a central component, places a high premium on excellence and achievement, and requires vigorous and intense training in a systematic fashion [29]. This definition is arbitrary, and it should be emphasized that many individuals participate in "recreational" sports in a truly competitive fashion.

Studies encompassing diverse populations of athletes have demonstrated that long-term training often leads to greater cardiac mass caused by an increase in left ventricular diastolic cavity dimension, wall thickness, or both, as well as right ventricular enlargement [36]. This remodeling is relatively mild in absolute terms, and the differences between athletic and nonathletic populations are generally small (although statistically significant). In particular, alterations in left ventricular wall thickness or cavity dimension may be more striking in sports such as rowing/canoeing, cycling, and cross-country skiing [30]. Consequently, the need to differentiate between athlete's heart and cardiac disease is more likely to arise in athletes training in such disciplines.

It is possible to distinguish the athlete's heart from hypertrophic cardiomyopathy **(**HCM) solely on the basis of left ventricular end-diastolic cavity dimension. A cavity >55 mm in an athlete with borderline wall thickness would constitute strong evidence against the presence of HCM; conversely, a cavity dimension <45 mm would probably be inconsistent with the athlete's heart [30]. Elite female athletes rarely show left ventricular wall thicknesses >12 mm and consequently do not appear to fall within the "gray zone" between athlete's heart and HCM [35]. Therefore, female athletes with borderline left ventricular wall thickness (in the presence of normal cavity size) are most likely to have pathologic hypertrophy.

Diseases Predisposing to SCD

The various causes of SCD in children can be broadly divided into arrhythmic and non-arrhythmic causes (Table 2). As can be seen, by and large the causes of SCD are inborn cardiac abnormalities with structural defects such as an abnormal coronary artery or an arrhythmogenic problem such as prolonged QT syndrome [24]. However, in many instances it can be hard to separate one form the other. An example of this would be HCM, where the heart is myopathic as well as being arrhythmogenic. In some conditions, such as SIDS, the cause of the SCD is unknown. While channelopathies have been shown to be the cause or associated with SCD in about 10% of SIDS, SIDS by and large remains a poorly understood and mysterious condition [1].

1. Hypertrophic Cardiomyopathy

HCM is a primary and familial cardiac malformation with heterogeneous expression and diverse clinical course for which several disease-causing mutations in genes encoding proteins of the sarcomere have been reported. HCM is a relatively common disorder, occurring in up to 0.17% (about 1 in 500) of the general population [25]. Of note, in the 2 highly visible tragedies concerning competitive athletes with cardiovascular disease-- Hank Gathers and Reggie Lewis (elite college and professional basketball players)--the clinical and pathologic findings were most consistent with HCM [26].

SCD in HCM has shown a predilection for young and asymptomatic individuals, frequently occurring during moderate or severe exertion similar to its demographic profile in athletic populations. Disease variables that appear to identify patients at greatly increased risk include prior aborted cardiac arrest or sustained ventricular tachycardia, family history of sudden death, or of other premature HCM related death, identification of a high-risk genotype, multiple-repetitive nonsustained ventricular tachycardia on ambulatory Holter ECG recording, recurrent syncope, and possibly massive left ventricular hypertrophy [41]. The magnitude of the left ventricular outflow gradient has not been associated with an increased risk for SCD.

The risk of SCD associated with vigorous athletic competition in individuals with HCM is well documented, and is the basis for recommended exclusion from competitive sports independent of other risk factors [9]. Although SCD risk with recreational exercise is less well defined, published guidelines for participation in recreational sports offer a valuable framework for counseling patients [33]. Exercise stress testing is an important diagnostic and prognostic tool in the evaluation of HCM patients, providing an objective measure of functional capacity, physiologic

hemodynamic responses to stress, presence of ischemia, and left ventricular outflow tract obstruction and arrhythmias. Athletes with a diagnosis of HCM should be excluded from most competitive sports, with the only possible exception being those of low intensity in individual cases based on the results of expert cardiovascular evaluation [33].

2. Congenital Coronary Artery Abnormalities

Congenital coronary artery abnormalities (CCAA) occur in up to 1% of the population [13]. After HCM, CCAA with origin from the wrong sinus of valsalva are the second most common cause of SCD on the athletic field in the USA [23]. CCAA do not always cause symptoms and sudden death is often the first presentation. Although an anomalous right coronary artery arising from the left coronary sinus is four times as common as an anomalous left coronary artery arising from the anterior sinus, it is the latter that is by far the more common cause of SCD with vigorous physical activity. Of the four types of anomalous left coronary arteries, the interarterial type, where the left coronary artery passes anteriorly between the aorta and the right ventricular outflow tract, places the patient at greatest risk of SCD.

A feature of CCAA is the fact that SCD occurs associated with vigorous exercise [4]. The pathophysiology of cardiac arrest in athletes with anomalous coronary arteries has been related to abrupt ventricular fibrillation precipitated by exercise-related myocardial ischemia which, in turn, is the result of the aortic expansion which compresses the anomalous vessel against the pulmonary trunk, increases the acute angulation of the coronary takeoff, and aggravates the “slit-like” shape of the lumen [2].

The association between CCAA and sudden death has been investigated by autopsy studies of individuals who have undertaken high levels of exertion. A retrospective cohort study of 6.3 million US military recruits over a 25-year period identified 126 cases of non-traumatic death [12]. A coronary artery abnormality was identified as the cause of death in 21 cases, accounting for 17% of deaths. A left anomalous coronary artery was found to be responsible at postmortem in all of those cases. Basso *et al* [2] investigated 27 athletes found to have died of CCAA, all of whom died during or immediately following exertion. Of the 27 deaths, 23 were attributed to left anomalous coronary artery and four to right anomalous coronary artery.

In patients with high risk CCAA, competitive sports are generally restricted prior to surgical intervention [31]. The risks postoperatively are not clearly defined at this time and should be made on an individual basis after appropriate testing, including evaluation during exercise.

3. Myocarditis

Myocarditis has traditionally been considered an important cause of SCD in young individuals. Although estimates have suggested that the incidence of myocarditis is approximately 1 per 100 000, the true incidence remains largely unknown due to the significant number of cases that go undiagnosed [22].

The majority of cases of myocarditis in children in the United States are secondary to a viral infection with Coxsackie B being the most well-known cardiotoxic virus [21]. The clinical presentation of children with myocarditis varies from asymptomatic cases to those with complete cardiovascular collapse. They can also have acute AV node damage with heart block and/or ventricular tachycardia from the cardiac inflammation.

The risk of cardiovascular collapse and fatal arrhythmias is potentially exacerbated with exercise making athletes with myocarditis at a greater risk of SCD. It is important to take subtle discomforts suggesting a diagnosis of myocarditis seriously especially in athletes and initiate further evaluation. Athletes with clinical diagnosis of myocarditis should be temporarily excluded from competitive and amateur leisure-time sports activity. This recommendation is independent of age, gender and does not differ for athletes with only mild symptoms, or those under treatment

with drugs. After resolution of the clinical picture (at least 6 months after the onset of the disease), clinical reassessment is indicated prior to resuming competitive sports [33].

4. Marfan's Syndrome

Although the incidence of Marfan's syndrome worldwide is relatively rare (about 1 in 5,000 live births), it is speculated that the prevalence may be much higher among individuals participating in sports in which tall structure and long limbs are a beneficial asset such as volleyball, basketball, and high jump [42]. Cardiovascular complications have been found to occur in 30–60% of patients with Marfan's syndrome and additionally, 70% of patients with Marfan's syndrome will die of acute cardiovascular complications.

The hemodynamic stress placed on the aorta by increased blood pressure and stroke volume during intense activity could increase the rate of aortic enlargement thereby increasing the risk of aortic rupture. Aortic dilatation also leads to aortic regurgitation and more seriously left ventricular failure and/or aortic dissection.

Aortic dilatation (which is related to risk of acute aortic dissection) is the primary determinant of whether individuals with Marfan's syndrome should be judged medically eligible for athletic competition. According to the Bethesda Guidelines for athletic participation in patients with Marfan's syndrome, if aortic root diameter is less than 40 mm, and there is no mitral regurgitation or family history of SCD, then low to moderate intensity competitive sports are permitted [37].

5. Congenital Heart Disease

In order to determine the incidence of SCD in patients with congenital heart disease, Silka et al. [40] evaluated all patients < 19 years old undergoing surgical treatment of common forms of congenital heart disease in the state of Oregon between 1958 and 1996. The risk of late sudden death for patients surviving operation for common congenital heart defects was 25 to 100 times greater than an age-matched control population. This increased risk was primarily represented by patients with cyanotic or left heart obstructive lesions. The risk of sudden death appeared to be time dependent, increasing primarily after the second postoperative decade. Exercise increased the risk of SCD for certain cardiac lesions and was less clear for others. Of the seven patients with transposition of the great arteries that died suddenly, five were participating in active physical exertion at the time of death.

The recommendations from the consensus document of an international expert panel appointed by the European Society of Cardiology are that regular exercise at recommended levels can be performed in patients with simple congenital heart lesions with successful repair or no need for therapy [19]. Most can attend sports with no restrictions. Special concern should however be given to those patients with a significant ventricular dysfunction or recent history or risk of arrhythmia. Most patients with complex lesions have some degree of residual disease, making them less suitable for participation in competitive sport. Patients with tetralogy of Fallot who have undergone surgery are the best-studied subgroup within those with congenital heart disease. These patients are advised to limit their activities to low to moderate dynamic and static sports [19].

6. Arrhythmogenic Right Ventricular Dysplasia

Arrhythmogenic right ventricular dysplasia **(**ARVD) is the most common cause of SCD in young people and athletes within the Veneto region in northeastern Italy [8]. Based on the Italian studies, most deaths in ARVD patients occur during exercise. Although this disease is also a feature of athletic field deaths in the United States, its frequency is clearly in the range of <5% in reports from outside Italy [28].

Various mechanisms have been suggested to explain the propensity for ARVD to precipitate effort-dependent sudden cardiac arrest. Physical exercise may acutely lead to an increase in the

RV afterload and an enlargement of the cavity, which in turn may elicit ventricular arrhythmias by stretching the diseased RV myocardium [10]. Alternatively, a “denervation supersensitivity” of the RV to catecholamines has been suggested [45]. Sympathetic nerve trunks may be damaged and/or interrupted by the RV fibrofatty replacement, which distinctively progresses from the epicardium to the endocardium, resulting in a denervation supersensitivity to catecholamines. Arrhythmogenic mechanisms in the denervated supersensitive myofibers include dispersion of refractoriness and reentry, triggered activity, or both. In a subgroup of patients with familial ARVD, a cardiac ryanodine receptor (RYR2) missense mutation leading to abnormal calcium release from the sarcoplasmic reticulum has been identified [44].

Athletes with clinical diagnosis of ARVD should be excluded from most competitive sports, with the possible exception of those of low intensity, proven absence of arrhythmias and no incidence of exercise-related symptoms [33]. This recommendation is also independent of age, gender and phenotypic appearance and does not differ for athletes without symptoms.

7. Long QT Syndrome

Congenital long QT syndrome **(**LQTS) comprises a distinct group of cardiac channelopathies characterized by delayed repolarization of the myocardium, QT prolongation, and increased risk for SCD in the setting of a structurally normal heart. Aborted cardiac arrest or SCD is the sentinel event in 5% to 10% of LQTS cases [39]. It is estimated that identifiable and treatable cardiac channelopathies account for approximately one-third of autopsy-negative sudden deaths in the young [1].

Much has been learned about the genetic basis of long QT syndrome. Many genetic sub types affecting various cardiac ion channels have been described. Of the three major subtypes, long QT type 1 is predominantly associated with arrhythmia during exercise. Beta-blockers provide significant reduction in SCD in these patients. For diagnosis of LQTS in athletes the QTc interval should exceed 0.47 s in male subjects and 0.48 s in female subjects [37]. When this diagnosis is made, the recommendation is for restriction of athletes from competitive sport, except those with low intensity [37]. A unique recommendation applies to individuals with LQT1 mutation, who should refrain specifically from competitive swimming, because of the strong association between this sport and cardiac events [32].

8. Catecholaminergic Polymorphic Ventricular Tachycardia

Catecholaminergic polymorphic ventricular tachycardia **(**CPVT) is characterized by adrenergically induced ventricular tachycardia associated with syncope and sudden death. The true prevalence of CPVT is unknown. CPVT can mimic concealed LQTS and may be under recognized among physicians referring patients because of a suspected channelopathy.

CPVT is characterized by arrhythmias during exercise. Patients often present with symptoms of dizziness or syncope during exercise and induction of arrhythmias (primarily VT) during exercise testing is the best way to diagnose such patients. In patients with CPVT, polymorphic or bidirectional VT can be provoked with exercise in 63% and adrenaline in 82% [43]. With regard to individuals with definite diagnosis of CPVT, both the Bethesda conference and European Society of Cardiology documents restrict participation from sports [37].

9. Short QT Syndrome

The hereditary short QT syndrome has emerged as yet another rare channelopathy. This autosomal dominant syndrome can afflict infants, children, or young adults; often a remarkable family background of SCD is elucidated. The electrocardiogram is characterized by a strikingly short QT interval (typically <320 milliseconds); virtual absence of the ST segment; and tall, peaked, narrow-based T waves [15]. There is a marked propensity for paroxysmal atrial fibrillation, and increased risk for SCD from ventricular tachyarrhythmias.

Because of the limited number of patients reported with a short QT interval, it has not yet been possible to come up with clear guidelines to risk stratify such individuals. The consensus paper on behalf of the Study Group on Sports Cardiology of the European Society of Cardiology Competitive, however, recommends that in patients with short QT syndrome, sports are not allowed except for those with low static or dynamic demand. Until more clinical data become available, restraint should be used to even allow moderate leisure-time activity, certainly avoiding sudden bursts of activity [17].

10. Brugada Syndrome

Brugada syndrome (BrS) is characterized by coved ST elevation in the right precordial leads and lethal ventricular arrhythmia in an apparently structurally normal heart. Symptomatic BrS is rare in children. In contrast to adults where over1500 adults with BrS have been reported, Probst et al [38] described only 30 children and adolescents (≤16 years of age) with BrS through the collaboration of investigators from 13 different referral centers in 3 European countries. This means that during the 15 years that passed since the original description of the BrS, <3 children (on average) were identified at each of the tertiary centers joining forces for this report. The majority of children (60%) were asymptomatic, and their BrS -type ECG was noted during family screening. Although more than 90% of adults with BrS symptoms are male, no obvious male predominance existed among symptomatic or asymptomatic children with BrS.

Patients with BrS seem primarily at risk for arrhythmias during febrile illnesses or during sleep. Although a clear association between exercise and sudden death in the BrS has not been established, disqualification from all competitive sports is nevertheless recommended by the European Society of Cardiology document, with a potential exception in Bethesda Conference only for low-dynamic and low-static sport [32, 34].

11. Wolff-Parkinson-White syndrome

The Wolff-Parkinson-White **(**WPW) pattern is relatively common and found in the range of 2 to 4 individuals per 1000 [20]. Sudden death can be the first manifestation of the WPW syndrome. The lifetime risk of mortality related to this in asymptomatic individuals has been estimated to be in the range of 1 per 1000 (0.1% annual risk). The mechanism of sudden death is probably the occurrence of atrial fibrillation with a very rapid ventricular rate that leads to ventricular fibrillation.

Supraventricular tachyarrhythmias, particularly if related to WPW, may in some cases endanger an athlete's professional career due to hemodynamic consequences during athletic activity, which in some instances may be life-threatening. Current guidelines do not always recommend a routine electrophysiological study in patients with an asymptomatic WPW ECG pattern, especially in children younger than 12 years. Individuals who engage in highly competitive sports activities may however be exceptions and warrant an electrophysiological study [14].

12. Congenital Heart Block

Congenital heart block occurs in approximately 1-5% of pregnancies in mothers with anti-Ro/La antibodies, independent of the mother's disease status, and in approximately 15-20% of pregnancies following the birth of a child with neonatal lupus [3]. It is a permanent condition that entails significant morbidity and mortality, with a majority of all affected infants requiring pacemakers and with an 80% cumulative probability of survival at 3 years of age.

In children and young adults with complete heart block, severe ventricular ectopy is common at exercise which may predispose them to in an increased risk for SCD [46]. As majority of the older children with isolated congenital heart block will have pacemakers, their activity limitations will be similar to patients with pacemakers in whom competitive or recreational sports participation is allowed in sports with minor to moderate cardiovascular demand [18].

13. Commotio Cordis

Blunt, nonpenetrating chest blows causing instantaneous SCD (*commotio cordis*) have been the subject of increasing attention. The blunt chest impact over the anatomic position of the heart is usually produced by a projectile or by bodily collision with another athlete. The chest blow is of low energy and not perceived as unusual for the sporting event nor apparently of sufficient magnitude to result in death due to trauma alone.

The US Commotio Cordis Registry, Minneapolis, Minnesota reported 128 cases as of September 1, 2001 [27]. Ages of the 128 cases ranged from 3 months to 45 years; 44% were 12 years or younger and 22% were 18 years or older; 95% were male.

At the time of their commotio cordis event, 62% were engaged in organized competitive sports at the youth, high school, college, and professional levels. Although 11 sports were represented, 58% of these commotio cordis events occurred during baseball or softball games and 16% at ice hockey games. Other commotio cordis events (38%) occurred during a variety of innocent-appearing and recreational activities in informal and largely unstructured settings, such as in the backyard, playground, or neighborhood school yard or while participating in a variety of daily activities and circumstances unrelated to sports. Of the individuals experiencing commotio cordis, 84% died as a consequence of their event and 16% survived. Even though the data from this registry did not allow determination of the incidence of commotio cordis, the cases included demonstrated that commotio cordis occurs most commonly in the setting of sporting events but can also occur during relatively benign circumstances that are part of daily living and unrelated to athletic activities.

Conclusion

The precise incidence of SCD and the prevalence of SCD predisposing diseases remain unclear. SCD in young competitive athletes usually is precipitated by physical activity and due to a heterogeneous spectrum of cardiovascular diseases, most commonly HCM. Risk stratification remains a challenge in the absence of adequate knowledge and understanding of the epidemiology of SCD. In order to better answer these questions, a large-scale, comprehensive registry with the potential to answer many questions that relate to SCD in children, adolescents, and young adults would be extremely useful.

Table 1: Epidemiology of SCD in the Young

Report	Publication date	Dates data Collected	Age range	Event Rates	Method of Estimation
Driscoll et al. [11]	1985	1950-1982	1 and 22 years	1.3 cases per 100,000 person years	Population based study by review of death certificates
Chugh et al. [5]	2009	2002-2005	< 18 years	1.7 cases per 100,000 person years (90% <1 year)	Population based study in Portland, Oregon by emergency room and medical examiner records
Maron et al. [27]	2009	1985-2007	High school and college athletes	0.93 deaths per 100,000 person-years	Registry and insurance records in Minnesota
Maron et al. [22]	2009	1980-2006	Athletes < 40 years	0.61/100 000 person-years	National registry for sudden deaths in US athletes
Corrado D et al. [6]	2006	1979-2004	12-35 years	Athletes: 1.9 deaths/100,000 person-years Non Athletes: 0.79 deaths/100,000 person-years	Prospective cohort study in Veneto, Italy
Harmon et al. [16]	2011	2004-2008	NCAA Athletes	2.28 deaths per 100,000 person-years	NCAA Database, media reports, insurance claims

NCAA: National Collegiate Athletic Association

Table 2: Etiology of SCA in children and adolescents

Structural and functional causes

- Hypertrophic cardiomyopathy
- Congenital coronary artery abnormalities
- Myocarditis
- Marfan's syndrome
- Congenital heart disease
- Arrhythmogenic right ventricular dysplasia
- Primary pulmonary hypertension
- Cardiomyopathy (restrictive, dilated)

Primary electrical abnormalities

- Long QT syndrome
- Catecholaminergic polymorphic VT
- Short QT syndrome
- Brugada syndrome
- Wolff Parkinson White syndrome
- Congenital heart block

Acquired

- Commotio cordis

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