Etiology of Sudden Death in Sports
Insights From a United Kingdom Regional Registry

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ABSTRACT

BACKGROUND Accurate knowledge of causes of sudden cardiac death (SCD) in athletes and its precipitating factors is necessary to establish preventative strategies.

OBJECTIVES This study investigated causes of SCD and their association with intensive physical activity in a large cohort of athletes.

METHODS Between 1994 and 2014, 357 consecutive cases of athletes who died suddenly (mean 29 ± 11 years of age, 92% males, 76% Caucasian, 69% competitive) were referred to our cardiac pathology center. All subjects underwent detailed post-mortem evaluation, including histological analysis by an expert cardiac pathologist. Clinical information was obtained from referring coroners.

RESULTS Sudden arrhythmic death syndrome (SADS) was the most prevalent cause of death (n = 149 [42%]). Myocardial disease was detected in 40% of cases, including idiopathic left ventricular hypertrophy (LVH) and/or fibrosis (n = 59, 16%); arrhythmogenic right ventricular cardiomyopathy (ARVC) (13%); and hypertrophic cardiomyopathy (HCM) (6%). Coronary artery anomalies occurred in 5% of cases. SADS and coronary artery anomalies affected predominantly young athletes (<35 years of age), whereas myocardial disease was more common in older individuals. SCD during intense exertion occurred in 61% of cases; ARVC and left ventricular fibrosis most strongly predicted SCD during exertion.

CONCLUSIONS Conditions predisposing to SCD in sports demonstrate a significant age predilection. The strong association of ARVC and left ventricular fibrosis with exercise-induced SCD reinforces the need for early detection and abstinence from intense exercise. However, almost 40% of athletes die at rest, highlighting the need for complementary preventive strategies. (J Am Coll Cardiol 2016;67:2108–15) © 2016 by the American College of Cardiology Foundation.

Sudden cardiac death (SCD) is a tragic event that occasionally affects apparently healthy individuals (1), including young (<35 years of age) athletes (2-5). A spectrum of cardiac diseases is implicated in SCD, with variable prevalence depending on the age and other demographics of the cohort (6). Many reports regarding the causes of SCD in athletes are limited by the lack of a detailed post-mortem examination performed by an experienced cardiac pathologist. A recent study comparing the interpretation of autopsy findings between a referring pathologist and a specialist cardiac pathologist demonstrated a 40% disparity with respect to actual cause of death (7).

Knowledge of precise causes and precipitating factors of SCD may influence national strategies to
The objective of this study was to investigate the causes and circumstances of SCD in a large cohort of athletes, as determined by post-mortem examination performed by an expert cardiac pathologist.

METHODS

The Cardiac Risk in the Young (CRY) center for cardiac pathology is located at St. George’s University of London. The center is led by an expert cardiac pathologist (M.N.S.) and receives over 400 whole hearts from cases of SCD across the United Kingdom annually. General pathologists are likely to refer to the CRY center when the clinical history is suggestive of inherited cardiac disease, especially when the death affects a young or athletic individual or when the cause of death is uncertain after the initial autopsy.

STUDY POPULATION. We retrospectively reviewed cases from a database of 3,684 cases of SCD referred to the CRY center for cardiac pathology between 1994 and 2014. SCD was defined as death occurring within 12 h of apparent wellbeing. We retrieved a subgroup of 357 cases (9.7%) of individuals who had engaged in regular sport activities, defined as >3 h of organized physical training per week. The majority (84%) of referrals occurred between 2004 and 2014. Competitive athletes were defined as those who were involved in organized sports requiring participation in regular, formal competition. Circumstances of death were subdivided broadly into death occurring during exercise and death during rest or sleep.

POST-MORTEM EXAMINATION. All SCD cases underwent a full post-mortem evaluation by the local pathologist. Following the exclusion of extra-cardiac causes, the heart was sent to our center after written consent of the coroner and the family of the deceased. The local pathologist also performed an initial cardiac autopsy before referring the heart in 58% of cases. A thorough toxicology screen was conducted in all cases in accordance with the usual investigation of sudden and unexpected deaths in the United Kingdom. Comprehensive macroscopic examination of the whole heart and histological analysis were performed in accordance with guidelines on “Autopsy practice for sudden death with likely cardiac pathology” of the Royal College of Pathologists (10) and the Association for European Cardiovascular Pathology (11). All cardiac structures were systematically examined. Heart weight was recorded in grams, and ventricular wall thickness and internal cavity dimensions were measured at mid-ventricular level, excluding the papillary muscles and fat. A minimum of 10 blocks of tissue were taken for histological analysis as reported previously (6,7). Sections of myocardium were fixed in formalin, embedded in paraffin, and stained with hematoxylin and eosin and with elastic Van Gieson stain to highlight myocardial fibrosis.

Criteria for defining specific cardiac pathologies have been previously described (6,12) and are summarized in Table 1. Sudden arrhythmic death syndrome (SADS) was a diagnosis of exclusion, defined as a structurally normal heart with no evident abnormality on macroscopic and histological evaluation and a negative result for toxicology screening (13-15).

CLINICAL INFORMATION. The referring coroner and pathologist were asked to complete a questionnaire regarding the demographics of the deceased, medical history, family history, cardiac symptoms, nature and level of physical activity, and exact circumstances of death. Data were derived from a number of sources including: interviews with the family of the deceased, potential witnesses to the SCD, and reports from the deceased’s family physician. Data were collected prospectively and stored in an electronic database.

STATISTICAL ANALYSIS. Statistical analysis was performed using PASW version 18.0 software (PASW, Inc., Chicago, Illinois). Results are mean ± SD for continuous variables or numbers of cases and percentages for categorical variables. Comparison of groups was performed using Student t test for continuous variables with correction for unequal variance when necessary and chi-square test or Fisher exact test, as appropriate for categorical variables. Univariate and multivariate logistic regression analyses were used to determine the factors associated with death during exertion. Age, sex, and variables that were univariately correlated with the dependent variable were selected and entered into the forward stepwise multiple regression model.
TABLE 1 Pathological Macroscopic and Microscopic Criteria Defining Main Underlying Diseases

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Macroscopic Criteria</th>
<th>Microscopic Criteria</th>
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<tbody>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>Left ventricular wall thickness &gt;15 mm circumferentially or focally and/or heart weight &gt;500 g*</td>
<td>Myocyte hypertrophy, myocyte disarray (&lt;20% of myocardial disarray in at least 2 tissue blocks of 4 cm²), and interstitial fibrosis</td>
</tr>
<tr>
<td>Idiopathic left ventricular hypertrophy</td>
<td>Left ventricular wall thickness &gt;15 mm and heart weight &gt;500 g*</td>
<td>Myocyte hypertrophy with or without fibrosis in the absence of myocyte disarray</td>
</tr>
<tr>
<td>Idiopathic left ventricular fibrosis</td>
<td>Normal heart weight and wall thickness with/without scarring macroscopically</td>
<td>Fibrosis (&lt;20% in at least 2 tissue blocks of 4 cm²) with no myocyte disarray</td>
</tr>
<tr>
<td>Arrhythmogenic right ventricular cardiomyopathy</td>
<td>Right or left ventricular thinning, fatty replacement, fibrosis on the epicardial surface or outer wall</td>
<td>Fat and fibrosis (&lt;20% in at least 2 tissue blocks of 4 cm²) in the wall of the right and/or left ventricle, particularly in outer wall, with degenerative changes in the myocytes</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>Normal or dilated ventricles</td>
<td>Inflammation (&lt;20% in at least 2 tissue blocks of 4 cm²) with myocyte necrosis</td>
</tr>
<tr>
<td>Anomalous coronary artery</td>
<td>Anomalous origin of the coronary artery, coronary artery atresia, stenosis</td>
<td>Fibrosis/acute/chronic infarction in the left ventricle</td>
</tr>
<tr>
<td>Coronary atherosclerosis</td>
<td>Atherosclerosis with estimated luminal narrowing &gt;75%</td>
<td>Acute or chronic infarction in the left ventricle</td>
</tr>
<tr>
<td>Dilated cardiomyopathy</td>
<td>Increase in heart weight (&lt;500 g in males, &gt;400 g in females) with dilated left ventricle (&gt;4 cm) and thin wall (&lt;10 mm). Absence of coronary artery disease.</td>
<td>Diffuse interstitial and replacement fibrosis (&lt;20% in at least 2 tissue blocks of 4 cm²) in the left ventricle with degenerative changes in the myocytes</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
<td>Prolapse of mitral valve above the atrioventricular junction with ballooning between chordae in one or both leaflets</td>
<td>Myxoid degeneration with expansion in spongiosa of leaflets and destruction of fibrosa layer</td>
</tr>
<tr>
<td>Bicuspid aortic valve</td>
<td>Fusion of 2 aortic cusps, with or without cusps of a raphe often with significant valve stenosis</td>
<td></td>
</tr>
<tr>
<td>Morphologically normal heart</td>
<td>Normal</td>
<td>Normal</td>
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</table>

*Heart weight >400 g in women. In this cohort, heart weight was normal in 39% of HCM cases. In a small proportion of cases, HCM was diagnosed on the basis of the presence of myocyte hypertrophy, myocardial disarray, and fibrosis on microscopy, despite normal heart weight and wall thickness on macroscopic evaluation.

RESULTS

CLINICAL CHARACTERISTICS. The mean age at death of the 357 athletes was 29 ± 11 years (range: 7 to 67 years; median: 27 years), with a large male predominance (n = 330, 92%). The average body mass index and body surface area were 25 ± 5 kg/m² and 2.2 ± 0.4 m², respectively. A significant proportion of individuals were competitive athletes (n = 245 [69%]), participating in regular training and competition in team (n = 155 [43%]) or individual (n = 90 [26%]) sports. The rest of the cohort (n = 112 [31%]) consisted of recreational athletes. Sporting disciplines included running (n = 92 [25%]; 40 participating in half-marathons and marathons), football (n = 91 [25%]), cycling (n = 30 [8%]), gymnastics (n = 30 [8%]), swimming (n = 22 [6%]), weightlifting (n = 20 [6%]), rugby (n = 19 [5%]), tennis (n = 6 [2%]), golf (n = 6 [2%]), boxing (n = 5 [1%]), and other sports (n = 36 [10%]).

The majority of the athletes were asymptomatic (n = 288 [81%]). Of the 69 symptomatic athletes (29%), 27 (8%) had palpitations, 20 (6%) had chest pain, 18 (5%) had syncope, and 4 (1%) reported decreased exercise tolerance. Five patients experienced palpitations due to paroxysmal atrial fibrillation, including 1 in the context of Wolff-Parkinson-White syndrome. One athlete received a diagnosis of myocarditis 4 years before death, and 1 athlete was hospitalized 4 months prior to death for suspected myocarditis. There was a family history of premature sudden death (defined as death of a first-degree relative <50 years of age) in 28 cases (8%). The main comorbidities were asthma (n = 28%, 8%), epilepsy (n = 5, 1%), and treated arterial hypertension (n = 4, 1%).

CAUSES. The main causes of death are shown in the Central Illustration. A normal post-mortem indicative of SADS was the most common finding and accounted for 149 deaths (42%). Myocardial disease was present in 130 cases (35%). Among these cases, idiopathic left ventricular hypertrophy (LVH) and/or fibrosis accounted for 59 deaths (16%), followed by arrhythmogenic right ventricular cardiomyopathy (ARVC) (n = 48 [13%]), and hypertrophic cardiomyopathy (HCM) (n = 23 [6%]). The majority of individuals with ARVC also demonstrated LV involvement, with 17 cases (35%) exhibiting fibrofatty infiltration of the LV and 41 cases (85%) showing evidence of left ventricular (LV) fibrosis. Coronary artery pathology occurred in 7% of cases, with coronary artery anomalies accounting for the majority of the cases.

CAUSES OF DEATH BY AGE AND SEX. The prevalence of specific cardiac pathologies varied with age (Central Illustration). SADS was most common in younger cases and showed a reduced trend with increasing age (Figure 1). A structurally normal heart was reported in 56% of children and adolescents (<18 years), 44% of young adults (18 to 35 years of age), and 26% of older individuals (>35 years of age; p < 0.001 comparing <18 and >35 years of age; p = 0.004 comparing 18 to 35 years of age and those >35 years of age). Coronary artery anomalies were also more prevalent in younger individuals, accounting for 11% of deaths in children...
and adolescents compared to only 2% in adults >35 years of age. In contrast, diseases of the myocardium were more common in older athletes (Central Illustration). Idiopathic LVH and/or fibrosis was present in 10% of individuals <18 years of age and in 26% of those >35 years of age (p = 0.01). ARVC was detected in only 4% of individuals <18 years of age and in 18% of those >35 years of age (p = 0.009).

Mean heart weight was 421 ± 110 g. Seventy hearts (20%) exhibited an absolute value of >500 g. Of these, the majority were diagnosed with idiopathic LVH with or without fibrosis (n = 30 [42%]), followed by HCM (n = 13 [19%]), and ARVC (n = 12 [17%]) (Figure 1).

There were only 27 females in our cohort. The majority (55%) showed a normal heart at post-mortem

Sudden death is shown in the overall population (A), in subjects <18 years of age (B), subjects 18 to 35 years of age (C), and subjects >35 years of age (D). In the overall population, the subgroup classified as “Other” (n = 43) comprised: mitral valve abnormalities/prolapse; n = 7, myocardial infarction with normal coronaries; n = 4, bicuspid aortic valve; n = 3, aortic dissection; n = 3, cocaine/steroid use; n = 2, cardiac sarcoidosis; n = 1, atrium septal defect (ASD). In the remaining 23 cases, the cause of death could not be attributed to a single disease entity or condition and the post-mortem findings were considered of uncertain significance. LVH = left ventricle hypertrophy; RV = right ventricle.
examination. Idiopathic fibrosis accounted for 11% of deaths, ARVC for 7%, and HCM for 4% of the other deaths. None of the female athletes showed idiopathic LVH.

CIRCUMSTANCES OF DEATH. The majority of athletes died during exertion \((n = 219 \ [61\%])\), including a small proportion of individuals \((n = 14 \ [4\%])\) who died during altercation. Of the 138 subjects who died at rest, 47 \((34\%)\) died during sleep. The age and sex distributions of athletes who died during exertion and those of athletes who died at rest were similar, and only a minority of subjects \((8\%)\) had a family history of sudden death. Patients who died at rest were more likely to demonstrate a normal heart at post-mortem examination (Table 2). Conversely, athletes who died during exertion were more likely to have ARVC \((20\% \text{ vs. } 3\%, \text{ respectively}; p < 0.001)\), LV fibrosis \((39\% \text{ vs. } 22\%, \text{ respectively}; p < 0.001)\), and coronary artery anomalies \((7\% \text{ vs. } 1\%, \text{ respectively}; p = 0.01)\).

Multivariate analysis identified ARVC (hazard ratio \([HR]\): 6.01; 95% confidence interval \([CI]\): 1.97 to 18.32; \(p = 0.001\)) as the strongest independent predictor of SCD during exercise, followed by LV fibrosis \([HR]: 2.11; 95\% CI: 1.15 \text{ to } 3.88; p = 0.01\) (Table 3).

DISCUSSION

This study reports on a large number of young athletes dying suddenly in the United Kingdom for whom all post-mortem examinations were conducted by a cardiac pathologist with expertise in conditions predisposing to SCD. In comparison to a smaller study published by our group \((6)\), the large sample size of almost 70% competitive athletes allowed for a greater degree of certainty relating to the impact of specific pathologies associated with SCD during intensive exercise.

CAUSES OF SCD IN ATHLETES. In agreement with our earlier study \((6)\) and with studies in U.S.
collegiate athletes (16) and young military personnel (17), a normal heart indicative of a diagnosis of SADS was present in a significant proportion of athletes. A structurally normal heart accounted for 42% of the overall cohort in this study, compared to the 23% we reported previously (6). Although the high prevalence of SADS in our cohort may be partly explained by a referral bias, its high prevalence in U.S. cohorts (31% in collegiate athletes and 41% in young military personnel) underscores the importance of inherited primary arrhythmia syndromes as a major cause of SCD in athletes (16,17).

Myocardial disease accounted for 40% of cases. Idiopathic LVH and/or fibrosis and ARVC were the predominant diagnoses. The significance of idiopathic LVH is uncertain. The entity may be an innocent bystander, but the possibility of pathological LVH as a variant of HCM, cannot be excluded, particularly in the presence of LV fibrosis (12). Idiopathic LVH may also be a trigger for arrhythmia in individuals with underlying primary arrhythmia syndrome. In a recent study by our group, familial evaluation of victims of SCD with autopsy findings consistent with idiopathic LVH, identified primary arrhythmia syndromes in 6 of 13 (46%) families and probable HCM in only 1 family (12). In such circumstances, a false diagnosis of HCM has potentially significant implications for surviving relatives who may be subjected to targeted screening for cardiomyopathy rather than the extensive evaluation, including pharmacological provocation tests, to detect primary arrhythmia syndromes (18).

In this study, HCM contributed to only 6% of deaths. That is in contrast to the established perception that HCM is the commonest cause of SCD in athletes (4,19). This may partly reflect the stringent diagnostic criteria applied by our group, which requires the presence of >20% of myocardial disarray in at least 2 tissue blocks of 4 cm² (14). In contrast, nonspecialist pathologists may attribute exercise-induced adaptations such as LVH to HCM (7) without conducting a detailed histological analysis of the heart. This probability highlights the importance that post-mortem examinations in athletes should be performed by pathologists with a high level of experience in conditions predisposing to SCD. We cannot disregard the possibility of selection bias, as general pathologists may be less inclined to refer diseases such as HCM to our center if they are confident about the diagnosis.

An important minority (8%) showed idiopathic fibrosis. Possible explanations include healed myocarditis or incomplete expression of cardiomyopathy. However, it is also possible that long-standing intense exercise may be a causal factor. Studies have previously reported raised serum concentration of biomarkers of myocyte injury following endurance events, an increased prevalence of myocardial fibrosis on cardiac magnetic resonance and a higher burden of atrial and ventricular arrhythmias in veteran endurance athletes (20–22). More than 80% of athletes died suddenly without warning symptoms, underscoring the possible limitations of cardiac screening based only on medical history and physical examination (23).

**EFFECT OF AGE.** In agreement with previous studies (6,24), SADS exhibited a significant age predilection. SADS accounted for more than one-half of all deaths in children and adolescents, but for only 26% of deaths in individuals older than 35 years of age. In contrast, myocardial disease was more prevalent with advancing age. ARVC, a condition commonly associated with SCD in young athletes, was rarely detected in children and adolescents (4%) but accounted 18% of deaths in individuals >35 years. Our results are consistent with a large autopsy study in 200 cases of ARVC (25), where the average age of death was at 33 years and almost 40% of deaths occurred after age 35. Interestingly, ARVC was associated with an increased heart weight in 25% of cases.

**RELATIONSHIP BETWEEN SUDDEN CARDIAC DEATH AND EXERCISE.** As reported previously (4,26), we demonstrated that SCD in athletes occurs more frequently during exercise. The strongest predictor for SCD during exertion was ARVC. Athletes with ARVC were 6 times more likely to die on exertion compared to those with other cardiac pathologies.

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**TABLE 3 Multivariate Analysis of Death During Exercise**

<table>
<thead>
<tr>
<th>Univariate Analysis</th>
<th>Multivariate Analysis</th>
</tr>
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<tbody>
<tr>
<td><strong>Hazard Ratio</strong></td>
<td><strong>p Value</strong></td>
</tr>
<tr>
<td><strong>(95% CI)</strong></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1.29 (0.58–2.81)</td>
</tr>
<tr>
<td>Age at death</td>
<td>0.99 (0.98–1.12)</td>
</tr>
<tr>
<td>Caucasian ethnicity</td>
<td>1.09 (0.43–2.72)</td>
</tr>
<tr>
<td>Family history of SD</td>
<td>0.96 (0.43–2.12)</td>
</tr>
<tr>
<td>Competitive athlete</td>
<td>1.13 (0.71–1.8)</td>
</tr>
<tr>
<td>LV fibrosis</td>
<td>2.43 (1.48–4.01)</td>
</tr>
<tr>
<td>Heart weight*</td>
<td>0.96 (0.95–0.97)</td>
</tr>
<tr>
<td>ARVC</td>
<td>8.36 (2.93–23.84)</td>
</tr>
<tr>
<td>Coronary artery anomaly</td>
<td>5.32 (1.20–23.51)</td>
</tr>
<tr>
<td>SADS</td>
<td>0.44 (0.29–0.68)</td>
</tr>
<tr>
<td>HCM</td>
<td>0.80 (0.34–1.88)</td>
</tr>
</tbody>
</table>

*For every 10 g increase. CI = confidence interval; other abbreviations as in Table 3.
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(92% experienced SCD on the athletic field). LV fibrosis was also an independent predictor of exercise-induced SCD. Coronary artery anomalies were a rare cause of SCD, but the majority of deaths occurred during exertion. Although HCM is considered the leading cause of exercise induced SCD in athletes, deaths from HCM in our cohort did not show any predilection for exercise. Our results, however, should be interpreted with caution given the relatively low numbers of HCM-related deaths in this study. In agreement with current published reports, SADS accounted for the majority of deaths at rest (54%) compared to a third of deaths (34%) during exertion.

**CLINICAL IMPLICATIONS.** Given that almost 40% of athletes died outside the context of exercise, including 13% during sleep, it is highly unlikely that the provision of AEDs in public venues would have prevented these deaths. Many of the SCDs during rest were related to SADS and possibly to primary arrhythmia syndromes that are often detectable with an ECG in asymptomatic individuals. Pre-participation screening may therefore be useful in this scenario. With ARVC, ECG may be abnormal in 55% to 75% of cases (27-29), and ECG-based pre-participation screening might be effective at detecting athletes with the condition. Immediate availability of an AED could be potentially life-saving for cases that are not detected during pre-participation screening. Finally, LV fibrosis is increasingly recognized in athletes but is not considered in isolation as a reason for exercise restriction. This study suggests that LV fibrosis is a trigger for exercise induced fatal arrhythmias in some athletes and warrants longitudinal assessment of asymptomatic athletes with isolated LV fibrosis (30).

**STUDY LIMITATIONS.** The CRY Centre for Cardiac Pathology at St George's University of London is more likely to receive hearts from subjects where the clinical history is suggestive of an inherited cardiac disease. Local pathologists are more likely to refer challenging cases, such as athletes with ambiguous autopsy findings or athletes for whom an obvious cause of death cannot be established. These facts introduce a potential referral bias; pathologies such as coronary artery atherosclerosis and HCM may be under-represented in this cohort. Similarly, the prevalence of less well-defined entities such as idiopathic LVH and a morphologically normal heart may be overestimated. Nevertheless, we receive a high volume of unexpected SCD referrals (~400 per year); 58% are in individuals <35 years of age at death, including athletes. Considering that SCD in young athletes is a rare event, the large number of post-mortem examinations performed in our unit in this cohort suggests that the results are a genuine representation of the type and frequency of cardiac diseases implicated in SCD in young athletes.

It is possible that subtle or incomplete expressions of cardiomyopathy may have been misclassified as SADS; however, considering our thorough laboratory protocol, it is highly unlikely that such cases accounted for a significant proportion of deaths attributed to SADS.

Our study does not include data relating to survivors of sudden cardiac arrest. As such, it is possible that the results are biased toward lethal causes of sudden cardiac arrest such as cardiomyopathies and primary arrhythmia syndromes, whereas diseases more amenable to survival following cardiac arrest are under-represented (8,9).

**CONCLUSIONS**

Conditions predisposing to SCD in sport demonstrate significant age predilection. SADS accounts for most of the deaths in the very young, whereas cardiomyopathies predominate with increasing age. Although the majority of athletes die during exertion, almost 40% die at rest, highlighting the need for complementary preventative strategies in addition to provision of AED. The strong association of ARVC with exercise-induced SCD reinforces the need for competitive sport restriction in athletes with the condition. Finally, the high prevalence of idiopathic LVH or fibrosis underscores the need for further research in the field in order to delineate their significance.

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**PERSPECTIVES**

**COMPETENCY IN MEDICAL KNOWLEDGE:** Causes of sudden death in athletes vary with age. SADS is prevalent in children and adolescents, whereas cardiomyopathies are the most common cause in adults. ARVC is strongly associated with sudden death during exertion.

**TRANSLATIONAL OUTLOOK:** Further studies are needed to better understanding the causes and circumstances of sudden death to facilitate development of preventive strategies.
REFERENCES


KEY WORDS arrhythmogenic right ventricular cardiomyopathy, athletes, sudden death