Pathogeneses of Sudden Cardiac Death in National Collegiate Athletic Association Athletes

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Background—The pathogenesis of sudden cardiac death in college athletes has not been defined by systematic case identification.

Methods and Results—A total of 45 cases of sudden cardiac death were identified in National Collegiate Athletic Association (NCAA) athletes from 2004 to 2008 based on an internal reporting system and review of media reports. Autopsy reports were reviewed and adjudicated by a multidisciplinary panel. Cause of death could be reasonably determined in 36 cases; 3 athletes had no autopsy, 5 autopsy reports could not be obtained, and 1 autopsy had insufficient information to determine cause of death. The most common finding at death was a structurally normal heart or autopsy-negative sudden unexplained death (11, 31%), followed by coronary artery abnormalities (5, 14%), dilated cardiomyopathy (3, 8%), myocarditis related (3, 8%), aortic dissection (3, 8%), and idiopathic left ventricular hypertrophy/possible hypertrophic cardiomyopathy (HCM; 3, 8%). There was 1 case each (3%) of hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, long QT syndrome, commotio cordis, and Kawasaki disease. There was 1 case of death in a sickle cell positive athlete who also had left ventricular hypertrophy. The adjudicated diagnosis agreed with the official pathology report in only 59% of cases.

Conclusions—Unexplained death with a structurally normal heart is the most common finding after suspected sudden cardiac death in NCAA athletes. Hypertrophic cardiomyopathy is infrequently seen, and conclusions in autopsy reports may not accurately reflect the pathological findings. Standardized protocols for cardiovascular autopsies in athletes are needed, including postmortem genetic testing, particularly in autopsy-negative cases. (Circ Arrhythm Electrophysiol. 2014;7:198-204.)

Key Words: arrhythmias, cardiac ■ athletes ■ cardiomyopathy, hypertrophic ■ death, sudden ■ death, sudden, cardiac ■ etiology

Sudden cardiac death (SCD) is the leading cause of nontraumatic death in National Collegiate Athletic Association (NCAA) athletes.¹ The most common cause of SCD in athletes in the United States is thought to be hypertrophic cardiomyopathy (HCM).²⁻¹¹ In a renowned 1996 study,¹¹ with subsequent analysis of additional cases in 2009,⁶ 36% of deaths were attributed to HCM, whereas only 3% of cases were attributed to autopsy-negative sudden unexplained death (AN-SUD).¹⁰ Studies in athletes from other countries and in other age-matched noncompetitive athlete populations, such as in the US military and in general populations of young people both in the United States and abroad, have differed with AN-SUD representing a much larger proportion and HCM a relatively smaller proportion of deaths.¹²⁻²²

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For instance, in an often cited Italian study, there was only 1 death from HCM (2%).²¹ The differences between the pathogenesis of SCD in US and Italian athletes have been recognized and attributed to the heterogeneous racial diversity of the US population compared with the more homogenous genetic makeup of the Veneto region of Italy²⁴⁻²⁶ or to the effectiveness of screening programs in Italy.²⁶,²⁷ However, the discrepancies may also be related to differences in study methodology, case identification, and autopsy protocols. Understanding the pathogenesis of SCD in young athletes is critical because we develop and refine targeted and cost-effective prevention strategies.

The NCAA comprised >400000 student-athletes, all who are required to pass a preparticipation screening examination to compete. Participation numbers are tracked in each sport including statistics on sex, race, and division of play. This
study sought to leverage the unique aspects of this large, contained, and well-described population to better understand the causes of SCD in college athletes.

Methods
Deaths were identified between January 1, 2004, and December 31, 2008, using 2 databases; the NCAA database and the Parent Heart Watch (PHW) database. The NCAA database was created from the NCAA Memorial Resolutions List (a list of voluntarily school-reported athlete deaths from any cause) and deaths reported directly to the NCAA. Names may be submitted to the NCAA at any time, and each November a solicitation for names of student-athletes who have died is sent to every NCAA member institution (all Divisions) to create the Memorial Resolutions List. All NCAA athletes are covered by the NCAA Catastrophic Injury Insurance Plan, and insurance claims were also reviewed. PHW is a nonprofit organization dedicated to the prevention and awareness of sudden cardiac arrest in young people. They have maintained an ongoing database created from systematic searches of media reports since 2000. Deaths among athletes 17 to 24 years of age were queried, and each case was reviewed to determine whether the athlete was a member of an NCAA team at the time of death. The NCAA and PHW databases were combined into a single database. A capture-recapture analysis is a statistical technique that can be used when data are collected using 2 distinct data sets to estimate completeness of a sample and was performed on the combined database.26–29

Missing information regarding deaths was acquired through Internet searches and media reports, e-mails, and telephone calls to sports information directors, head athletic trainers, athletic directors, and coroners. When possible, the athlete’s parents or guardians were interviewed. Deaths were classified as cardiovascular related based on review of all available resources and information. Autopsy reports for all deceased athletes were sought.

Definitions for pathological diagnosis were agreed on taking into account accepted definitions in the literature30–32 and definitions used in other studies (Table I in the Data Supplement).33–35 Autopsy reports were reviewed independently by a panel of experts consisting of 3 sports medicine physicians, a cardiac pathologist, a cardiomyopathy specialist, a genetic cardiologist, and an electrophysiologist, all with expertise in SCD in athletes. Differences of opinion were resolved through panel discussion to reach the adjudicated diagnosis. The adjudicated diagnoses were compared with the diagnoses published by the media and listed on the official pathology report. This study was approved by the Division of Human Subjects, University of Washington.

Results
There were 45 cardiovascular-related sudden deaths during the years 2004 through 2008 for an annual incidence of 1:43,770, the details of which have been reported previously.1 Eighty-seven percent of cases were registered in the NCAA database, whereas surveillance of media reports collected in the PHW database identified only 56% of cases. Insurance claims identified only 20% of cases. Capture-recapture analysis estimated that the number of deaths for that period was 49.1 (confidence interval [45.4–50.5]) or that the combined database had identified 90% to 100% of athlete deaths during that time frame.

Cause of death could be reasonably determined in 36 cases (80%). Of the 9 cases which could not be determined, 3 athletes had no autopsy, 5 autopsy reports could not be obtained, and 1 autopsy had insufficient information to determine a cause of death. Thirty-two autopsy reports were available for review. One autopsy report had insufficient information to determine cause. A probable cause of death was determined in 5 cases without obtaining the written autopsy report. In 3 cases, the autopsy report was read over the phone; 1 congenital coronary artery abnormality, 1 case of Kawasaki disease, and 1 case of a grossly and microscopically normal heart (ie, AN-SUD) in which additional history was also provided. In another case attributed to long QT syndrome (LQTS), it was determined that 2 of the decedent’s siblings also had LQTS. There was 1 case of commotio cordis.

Among the 36 cases (80%) where the cause of death could be reasonably determined (Figure 1), the most common finding was AN-SUD (11, 31%) followed by coronary artery abnormalities (5, 14%), dilated cardiomyopathy (3, 8%), myocarditis related (3, 8%), aortic dissection (3, 8%), and idiopathic left ventricular hypertrophy (LVH)/possible HCM (3, 8%). There was 1 case each (3%) of HCM, arrhythmogenic cardiomyopathy (ARVC), commotio cordis, and Kawasaki disease. There was 1 case of death in a sickle cell positive athlete who also had LVH. Toxicology screens were available for all autopsies and were negative or noncontributory.

Twenty-two percentage of media reports did not mention a cause of death, and 36% reported a nonspecific diagnosis such as natural causes or heart trouble. Media reports were accurate when reporting congenital coronary artery abnormalities or aortic dissections. The adjudicated diagnosis was in agreement with the official pathology report in only 59% of cases (Table II in the Data Supplement).

Discussion
AN-SUD is the most common finding in NCAA athletes with SCD; whereas HCM was a much less common cause of death than previously reported in US athletes. This is similar to findings in other countries,15–18,35,36 in the US military,12,20 and in US nonathlete populations14; however, it differs from the US National Registry of Sudden Death in Athletes (Figure 2).6

Several reasons may explain this difference. Our study involved a narrow age range of highly trained athletes. In contrast, the US Registry includes a wider age range. It is also possible that this discrepancy stems from differences in methodology and case identification. This NCAA study was performed on a well-defined population and aided by an internal reporting system of student-athlete fatalities. Although it is possible that some cases of SCD were missed, the capture-recapture analysis indicated that 90% to 100% of deaths were identified in this cohort. The US National Registry of Sudden Death in Athletes has collected cases from media reports and other sources for >30 years. Registries, by their nature, risk ascertainment bias. In addition, autopsy or media-reported diagnoses do not always correlate with adjudicated diagnoses by experts.

There are few studies that examine the pathogenesis of SCD in athletes. In comparing the studies, the classification of athlete is inconsistently defined, age ranges are wide, and other than the US National Registry of Sudden Death in Athletes, the number of cases is small. Corrado et al21 prospectively followed 12- to 35-year-old Italian competitive athletes where there is mandatory reporting of deaths and a known denominator during 20 years. During that time there were 55 deaths with a 2% incidence of HCM. Suárez-Mier et al22 retrospectively reviewed 81 exertional deaths in any type of exerciser in Spain during a 15-year period and found a 10% incidence of HCM and a 23% incidence of AN-SUD. De Noronha et al23 reported on 89 athlete deaths of those who were <35 years old and who were referred for specialized autopsy with similar results of 12% HCM and

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AN-SUD indicates autopsy-negative sudden unexplained death; ARVC, arrhythmogenic cardiomyopathy; CAD, coronary artery disease; DCM, dilated cardiomyopathy; and HCM hypertrophic cardiomyopathy.
29% AN-SUD. Finally, Holst et al.\textsuperscript{18} reviewed 15 exertional deaths in competitive athletes in Denmark during a 7-year period and found no cases of HCM and 27% AN-SUD. Thus, consistent with the results of this NCAA cohort, other studies focusing solely on SCD in athletes demonstrate a lower incidence of HCM and a higher incidence of AN-SUD than what was reported in the US National Registry of Sudden Death in Athletes (Table).

The US military tracks all deaths through a mandatory reporting system. In a 25-year study of 18- to 35-year-old military recruits, there was a 6% incidence of HCM and a 30% incidence of AN-SUD\textsuperscript{12} and, in another study of active 18- to 35-year-old military personnel, there were comparable findings with HCM accounting for 13% of deaths and AN-SUD 41%.\textsuperscript{20} Similar distributions are seen in other studies based on the general population including those not exercising where death certificates, autopsies, and population statistics are used for analysis. In the Veneto region of Italy, all deaths in young people from 1979 to 1998 were recorded. There were 273 deaths, and standardized postmortem cardiac evaluations were performed in a prospective manner with medical histories and the circumstances surrounding death recorded.\textsuperscript{15} This study demonstrated a 7% incidence of HCM, whereas apparently normal hearts initially represented 28% (76) of cases.\textsuperscript{15} Findings of a relatively low incidence of HCM and a higher incidence of AN-SUD occurred in the general population in Australia (HCM 6%; AN-SUD 29%),\textsuperscript{16} England (HCM 5%; AN-SUD 14%),\textsuperscript{35} and Ireland (HCM 15%; AN-SUD 27%).\textsuperscript{19} In a US report from King County, Washington, during a 30-year period, HCM represented <3% of cases in a general population of 14- to 35-year-old individuals, and only 5% of cases related to exercise.\textsuperscript{14}

The pathogeneses behind SCD are highly dependent on the age range examined with myocardial infarction and coronary artery disease deaths representing incrementally higher proportions in age groups >30 years.\textsuperscript{16,17} When considering pathogeneses of SCD in young people, the upper age limit should be examined carefully to avoid bias of results. Likewise, as the age range is lowered to preadolescent ages, pathogeneses may be skewed toward congenital anomalies. The age range of epidemiological studies must be taken into consideration when interpreting results. This NCAA study involves a narrow age range of athletes 17 to 24 years old, which also may account for some differences with the US National Registry of Sudden Death in Athletes (8 to 39 years old).

We used accepted pathological definitions in the literature\textsuperscript{23,30–34}; however, we considered the possibility that differences in HCM definition may account for some of the variability in results between studies. Therefore, the autopsies were re-examined with definitions used to evaluate athlete deaths in the US National Registry of Sudden Death in Athletes.\textsuperscript{9} This did not significantly change results with only 1 case being recategorized as HCM. There was 1 death in an athlete with sickle cell trait (SCT), for which it was difficult to definitively adjudicate the cause of death. Athletes with SCT have an increased risk of exertional death.\textsuperscript{37} After death, cells will sickle in athletes with SCT and, therefore, the presence of sickled cells at autopsy does not indicate that sickling was related to the mechanism of death. Unlike individuals with sickle cell disease who often have cardiac abnormalities, most individuals with SCT have morphologically normal hearts. The athlete in the NCAA cohort with SCT who died met criteria for LVH but did not have any myocardial disarray or any other pathological finding noted on histopathology to be classified as HCM.\textsuperscript{38,39} The only history available for this athlete was a media report noting that he was known to have previous cardiac disease. A history of sudden collapse would indicate likely SCD, whereas a conscious collapse with a gradual decline is
more consistent with an exertional sickling death. As there was no history obtainable, causality was assigned to both, and the case was listed as idiopathic LVH/possible HCM/SCT.

In this study, the quality of the autopsies varied considerably from athlete to athlete. This is the most significant limitation of this investigation and is a limitation of any study based on a retrospective review of autopsy reports. The importance of this study compared with others is the defined population minimizing the possibility of ascertainment bias. In addition, the methodology to arrive at autopsy diagnosis is more clearly described in this article than in others, and the limitations in the autopsies performed are somewhat mitigated by careful review by an expert panel. Although there are still clear limitations to this methodology, other articles that are commonly cited state “we were largely dependent on primary data.” This is a limitation of all current US athlete studies and will only be overcome by more standardized data collection.

The training and expertise of local coroners and medical examiners as well as the protocol used to perform the autopsy vary by jurisdiction. Autopsy reports typically consist of a gross description of the heart, measurements, and histopathologic description. Measurements of various cardiac features, including the ventricular free walls and interventricular septum, were reported inconsistently in this study. It is possible a more specific cause of death could have been found in some of our cases classified as AN-SUD if a more detailed and comprehensive autopsy was performed. In an Italian study, 28% of deaths were initially categorized as pathologically normal hearts; however, with more specialized autopsy examination, 79% of these (60 of 76) were later determined to have a specific cause. This suggests that, in this population, evaluation by a cardiac pathologist may ultimately result in a more refined diagnosis. We attempted to overcome this limitation by gathering as much contextual information about the case as possible and by assembling a multidisciplinary expert panel to review and adjudicate each case. We were also unable to obtain autopsy reports or additional information for 8 cases (18%), 3 of which were black, male basketball players. It is conceivable that if these cases were available for review, more cases of HCM would have been discovered. Diagnostic criteria for HCM and other cardiomyopathies after death need to be adjusted from those used in living patients.

Figure 2. Comparison of pathogeneses of sudden cardiac death. ARVC indicates arrhythmogenic cardiomyopathy; CM, cardiomyopathy; HCM, hypertrophic cardiomyopathy; LVH, left ventricular hypertrophy; MI, myocardial infarction; NCAA, National Collegiate Athletic Association; SCT, sickle cell trait; and SUD, sudden unexplained death.
wall thickness and left ventricular cavity dimensions for the diagnosis of HCM are based on diastolic measurements; however, the heart muscle is often in rigor (simulated systole) at the time of autopsy. Measurements will vary based on the state of decomposition of the body and degree of agonal dilatation and wall thinning. Thus, any interpretation of measurements should take into account the postmortem interval and decompositional state of the specimen. Heart weight was reported consistently in all autopsies; however, inconsistency among pathologists in how much of the great vessels are trimmed, whether or not postmortem clot is sufficiently removed before weighing, and whether or not the heart is weighed pre- or postfixation, can add additional variability to these studies. The histopathology reported in the autopsies varied significantly as well. In many jurisdictions, microscopy is limited by cost considerations, and a case had no microscopic analysis at all. In this case, the information in the autopsy was judged to be insufficient to determine cause of death. Terminology also differed between autopsy reports, and it was not clear whether enough sections were obtained or whether they were taken from areas of the heart needed to evaluate for specific conditions.

This study is possibly limited by the number of cases examined. There were 45 cardiac deaths identified; however, for 20% (9 cases), the cause could not be determined either because an autopsy was not performed, could not be obtained, or had insufficient information to make a diagnosis. It is possible that the proportion of specific causes of SCD in NCAA athletes could change with a larger study size. However, the systematic collection method of this data makes it particularly valuable.

Finally, it is possible that these results may have been skewed because athletes with structural abnormalities such as HCM were detected by the required preparticipation screening and restricted from play. However, this possibility would apply to any of the structural or electric cardiac disorders that could be discovered by history and physical examination, and not just HCM. It is unclear whether the current screening model adequately identifies athletes with at-risk conditions or could have affected the natural distribution of SCD causes. Most studies investigating screening protocols demonstrate a low sensitivity to detect conditions associated with SCD by history and physical examination alone. In fact, a 1996 report showed that only 20% of athletes may have prodromal symptoms of cardiovascular disease before SCD, and only 1% was properly detected on preparticipation screening.

The increasing use of postmortem molecular studies will likely provide additional clarity to questions of diagnosis. Postmortem genetic testing currently identifies a known gene mutation for an ion channelopathy in 25% to 35% of cases of AN-SUD. In addition, genetic testing may elucidate the diagnosis in cases of idiopathic LVH/possible HCM and other cases of cardiomyopathy. Standardized autopsy protocols are needed to more accurately determine the causes of SCD in athletes.

Conclusions

AN-SUD is the most common finding on autopsy at death, whereas HCM is less common than previously thought in NCAA athletes. This is analogous to findings in other countries, in the US military, and in US nonathlete populations, although it differs from data reported in the US National Registry of Sudden Death in Athletes. Methodological differences may account in part for this discrepancy, and additional investigation with higher quality autopsies is needed. It is crucial that standardized autopsy protocols be followed in cases of SCD in young people including postmortem genetic testing. Autopsies should be performed either locally, when available, or at specialized centers where the knowledge and expertise to carefully evaluate the causality of SCD in the young exists. Centralized data collection with mandatory reporting of deaths and standardized protocols would significantly improve the quality of information and our understanding of the causes of death in young athletes. The diagnoses from media descriptions are often inaccurate, and even formal autopsy diagnosis may not correctly reflect pathological findings in the report. A meaningful discussion regarding screening and prevention cannot be engaged without an accurate understanding of the incidence and pathogeneses of SCD in athletes.

Acknowledgments

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Disclosures

None.

References

The most common pathogenesis of sudden cardiac death in US athletes has traditionally been thought to be hypertrophic cardiomyopathy. However, this study reveals that autopsies in deceased National Collegiate Athletic Association (NCAA) athletes most often reveal a structurally normal heart (31%), characterizing the victim as having autopsy-negative sudden unexpected death. Identified causes include coronary artery anomalies (14%), dilated cardiomyopathy (8%), myocarditis (8%), and aortic dissection (8%), with hypertrophic cardiomyopathy demonstrated in only 3% of student-athlete deaths. These findings are similar to studies of athletes in other countries and in the US military. The reasons for discrepancies between this and earlier US studies are unclear, but possibly related to the more thorough case identification in the setting of a predefined cohort (NCAA athletes) in this study. This study also validates an earlier study on the same cohort of athletes that found that the incidence of sudden cardiac death was much higher than previously thought. In 43,000 overall with some high-risk groups such as Division I men’s basketball having risks as high as 1 in 3000, by addressing criticisms that these were not cardiac-related deaths. A more precise understanding of the pathogeneses of cardiomyopathies in US athletes is important implications because we consider more effective ways to screen for and prevent sudden cardiac death, the leading cause of nontraumatic death in young athletes.

CLINICAL PERSPECTIVE

The most common pathogenesis of sudden cardiac death in US athletes has traditionally been thought to be hypertrophic cardiomyopathy. However, this study reveals that autopsies in deceased National Collegiate Athletic Association (NCAA) athletes most often reveal a structurally normal heart (31%), characterizing the victim as having autopsy-negative sudden unexpected death. Identified causes include coronary artery anomalies (14%), dilated cardiomyopathy (8%), myocarditis (8%), and aortic dissection (8%), with hypertrophic cardiomyopathy demonstrated in only 3% of student-athlete deaths. These findings are similar to studies of athletes in other countries and in the US military. The reasons for discrepancies between this and earlier US studies are unclear, but possibly related to the more thorough case identification in the setting of a predefined cohort (NCAA athletes) in this study. This study also validates an earlier study on the same cohort of athletes that found that the incidence of sudden cardiac death was much higher than previously thought. In 43,000 overall with some high-risk groups such as Division I men’s basketball having risks as high as 1 in 3000, by addressing criticisms that these were not cardiac-related deaths. A more precise understanding of the pathogeneses of cardiovascular deaths in athletes has important implications because we consider more effective ways to screen for and prevent sudden cardiac death, the leading cause of nontraumatic death in young athletes.
Pathogenesis of Sudden Cardiac Death in National Collegiate Athletic Association Athletes
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**Supplemental Table 1: Definitions for Pathologic Diagnosis of Sudden Cardiac Death in Athletes**

**Autopsy negative sudden unexplained death**
- Normal heart pathologically
- No obvious explanation for death
- Presumed arrhythmia

**Hypertrophic cardiomyopathy**
- Heart weight ≥ 50% of the expected mean weight based on gender, age, and body size for weight or height (using the Mayo nomograms) plus at least one of the following:
  - Histologic myocyte disarray
  - Septal mitral valve contact lesion (implying systolic anterior motion of the anterior mitral valve leaflet)
  - Asymmetric LVH, particularly ventricular septal – left ventricular free wall ratio ≥ 1.3 (note: symmetric and apical variants will typically have normal ratios)
- Significant (>75% of the area of a section) myocyte disarray in a basal or mid-ventricular section but not meeting weight criteria

**Idiopathic LVH/ possible HCM**
- Heart weight ≥ 50% of the expected mean weight based on gender, age, and body size for weight or height (using the Mayo nomograms) without myocyte disarray and
  - Non-dilated LV chamber
- Heart weight does not meet weight criteria for HCM or idiopathic LVH/ possible HCM using Mayo nomograms but
  - Pathologic features are suggestive of HCM (i.e. outflow tract obstruction, LVH > 16 mm)
  - No/minimal myocyte disarray
  - Non-dilated LV chamber

**Arrhythmogenic right ventricular cardiomyopathy**
- Gross fibrofatty replacement of right ventricular free wall (excluding anterior RV in older individuals)
- The fatty change appears “infiltrative” with a perpendicular pattern with respect to the epicardial surface
- Variable degrees of fibrosis, vacuolization, and/or lymphocytic myocarditis

**Dilated cardiomyopathy**
- Heart weight ≥ 50% of the expected mean weight based on gender, age, and body size for weight or height (using the Mayo nomograms) without myocyte disarray and
  - LV wall < 10 mm
  - LV chamber diameter (at the mid-ventricular level, excluding trabeculations) > 3.0 cm (note: agonal dilatation should be excluded by examining for cell separation and other post-mortem artifact histologically)
    - If absolute chamber diameter not measured, then comments about gross chamber dilatation (without agonal dilatation from autolysis)
  - Histologically, myocyte hypertrophy with variable interstitial fibrosis (usually pericellular-type)

**Myocarditis related**
- Active lymphocytic myocarditis
  - Inflammatory infiltrates of the myocardium with associated myocyte injury/necrosis
- Borderline myocarditis
  - Inflammatory infiltrates of the myocardium without associated myocyte injury/necrosis
- Healed myocarditis

**Coronary artery abnormalities**
- Coronary artery anomalies
- Myocardial bridging
- Tunneled coronary arteries
- Coronary artery dissections

**Coronary artery disease**
- Atherosclerotic coronary arteries with > 70% lumen occlusion and
  - More likely than not this was primary cause of death

**Commotio cordis**
- SCD after blunt trauma to the chest
- No other cardiac pathology
### Supplemental Table 2: Differences in Causes of Death

<table>
<thead>
<tr>
<th>SPORT</th>
<th>Cause stated in media report</th>
<th>Cause stated in official autopsy report</th>
<th>Adjudicated cause</th>
</tr>
</thead>
<tbody>
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<td>Commotio cordis</td>
<td>No autopsy</td>
<td>Commotio cordis</td>
</tr>
<tr>
<td>Baseball</td>
<td>No cause</td>
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<td>Idiopathic LVH/possible HCM</td>
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<tr>
<td>Basketball</td>
<td>Anomalous coronary</td>
<td>Anomalous coronary (left coronary)</td>
<td>Coronary artery abnormality</td>
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<tr>
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<td>No cause</td>
<td>Anomalous right coronary artery</td>
<td>Coronary artery abnormality</td>
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<tr>
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<td>No cause</td>
<td>Origin of the right and left coronary from the right sinus of valsalva</td>
<td>Coronary artery abnormality</td>
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<tr>
<td>Basketball</td>
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<td>Aortic dissection</td>
<td>Aortic dissection</td>
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<tr>
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<td>Aortic dissection</td>
<td>Aortic dissection</td>
<td>Aortic dissection</td>
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<tr>
<td>Basketball</td>
<td>HCM</td>
<td>DCM</td>
<td>DCM</td>
</tr>
<tr>
<td>Basketball</td>
<td>Cardiomyopathy</td>
<td>Cardiomyopathy</td>
<td>DCM</td>
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<tr>
<td>Basketball</td>
<td>Cardiac arrest</td>
<td>Cardiomyopathy with LVH</td>
<td>Idiopathic LVH/possible HCM</td>
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<tr>
<td>Basketball</td>
<td>Natural causes</td>
<td>Microscopic cardiomyopathy with cardiomegaly</td>
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<td>SCT/Idiopathic LVH/possible HCM</td>
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<tr>
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<tr>
<td>Basketball</td>
<td>HCM</td>
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</tr>
<tr>
<td>Basketball</td>
<td>HCM</td>
<td>Unable to contact next of kin</td>
<td></td>
</tr>
<tr>
<td>Basketball</td>
<td>No cause</td>
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<tr>
<td>Crew</td>
<td>Dehydration</td>
<td>Fluid and electrolyte imbalance</td>
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<tr>
<td>Crew</td>
<td>Heart trouble</td>
<td>No autopsy</td>
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<tr>
<td>X-country</td>
<td>Anomalous coronary</td>
<td>Anomalous coronary (left coronary)</td>
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<tr>
<td>X-country</td>
<td>SUD</td>
<td>No autopsy</td>
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<tr>
<td>X-country</td>
<td>Cardiac arrest</td>
<td>Global ischemic necrosis of the Myocardium without atherosclerosis</td>
<td>SUD</td>
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<tr>
<td>Football</td>
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<td>Myocardial bridging to the left anterior descending artery</td>
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<td>DCM</td>
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