Circulation

ORIGINAL RESEARCH ARTICLE

Sudden Cardiac Death in National Collegiate Athletic Association Athletes: A 20-Year Study

Bradley J. Petek, MD; Timothy W. Churchill, MD; Nathaniel Moulson, MD; Stephanie A. Kliethermes, PhD; Aaron L. Baggish, MD; Jonathan A. Drezner, MD; Manesh R. Patel, MD; Michael J. Ackerman, MD, PhD; Kristen L. Kucera, PhD, MSPH, LAT, AT; David M. Siebert, MD; Lauren Salerno, BA; Monica Zigman Suchsland, MPH; Irfan M. Asif, MD; Joseph J. Maleszewski, MD; Kimberly G. Harmon, MD

BACKGROUND: Understanding the incidence, causes, and trends of sudden cardiac death (SCD) among young competitive athletes is critical to inform preventive policies.

METHODS: This study included National Collegiate Athletic Association athlete deaths during a 20-year time frame (July 1, 2002, through June 30, 2022). Athlete deaths were identified through 4 separate independent databases and search strategies (National Collegiate Athletic Association resolutions list, Parent Heart Watch database and media reports, National Center for Catastrophic Sports Injury Research database, and insurance claims). Autopsy reports and medical history were reviewed by an expert panel to adjudicate causes of SCD.

RESULTS: A total of 143 SCD cases in National Collegiate Athletic Association athletes were identified from 1102 total deaths. The National Collegiate Athletic Association resolutions list identified 117 of 143 (82%), the Parent Heart Watch database or media reports identified 89 of 143 (62%), the National Center for Catastrophic Sports Injury Research database identified 63 of 143 (44%), and insurance claims identified 27 of 143 (19%) SCD cases. The overall incidence of SCD was 1:63 682 athlete-years (95% CI, 1:54 065–1:75 010). Incidence was higher in male athletes than in female athletes (1:43 348 [95% CI, 1:36 228–1:51 867] versus 1:164 504 [95% CI, 1:110 552–1:244 787] athlete-years, respectively) and Black athletes compared with White athletes (1:26 704 [1:20 417–1:34 925] versus 1:74 581 [1:60 247–1:92 326] athlete-years, respectively). The highest incidence of SCD was among Division I male basketball players (1:8188 [White, 1:5848; Black, 1:7696 athlete-years]). The incidence rate for SCD decreased over the study period (5-year incidence rate ratio, 0.71 [95% CI, 0.61–0.82]), whereas the rate of noncardiovascular deaths remained stable (5-year incidence rate ratio, 0.98 [95% CI, 0.94–1.04]). Autopsy-negative sudden unexplained death (19.5%) was the most common postmortem examination finding, followed by idiopathic left ventricular hypertrophy or possible cardiomyopathy (16.9%) and hypertrophic cardiomyopathy (12.7%), in cases with enough information for adjudication (118 of 143). Eight cases of death were attributable to myocarditis over the study period (1 case from January 1, 2020, through June 30, 2022), with none attributed to COVID-19 infection. SCD events were exertional in 50% of cases. Exertional SCD was more common among those with coronary artery anomalies (100%) and arrhythmogenic cardiomyopathy (83%).

CONCLUSIONS: The incidence of SCD in college athletes has decreased. Male sex, Black race, and basketball are associated with a higher incidence of SCD.

Key Words: athletes ■ death, sudden, cardiac ■ exercise ■ sports

Editorial, see p XXX

Correspondence to: Kimberly G. Harmon, MD, University of Washington, 3800 Montlake Blvd, Seattle, WA 98125. Email kharmon@uw.edu

Supplemental Material is available at https://www.ahajournals.org/doi/suppl/10.1161/CIRCULATIONAHA.123.065908.

For Sources of Funding and Disclosures, see page XXX.

Circulation is available at www.ahajournals.org/journal/circ

ORIGINAL RESEARCH ARTICLE

Clinical Perspective

What Is New?

- The overall incidence of sudden cardiac death (SCD) among National Collegiate Athletic Association athletes has decreased over the past 20 years.
- Despite the reduction in SCD incidence, the SCD rate remains highest among specific cohorts, including Division I Black and White male basketball players (>1:2000 over a 4-year career).
- There were no cases of death attributable to COVID-19 myocarditis.

What Are the Clinical Implications?

- SCD remains higher in certain subgroups of athletes, including men's basketball and football. Continued efforts are needed to improve cardiovascular screening, risk stratification, emergency action plans, availability of automated external defibrillators, and training in cardiopulmonary resuscitation.
- Autopsy-negative sudden explained death and idiopathic left ventricular hypertrophy/possible cardiomyopathy were the leading postmortem findings after SCD in college athletes. Broader use of postmortem genetic testing is imperative to better determine causes of death.

Nonstandard Abbreviations and Acronyms

AN-SUD	autopsy-negative sudden unexplained death
AY	athlete-year
IRR	incidence rate ratio
NCAA	National Collegiate Athletic Association
PPCS	preparticipation cardiovascular screening
SCA	sudden cardiac arrest
SCD	sudden cardiac death

Sudden cardiac death (SCD) is the leading medical cause of death among young competitive athletes.[1,2] These events affect both the local community in which they occur and the broader national consciousness, because of their emotional impact and widespread media attention. As a consequence, SCD among young competitive athletes has been the focus of important scholarly work over the past several decades.[1–14] This focus has accommpanied corresponding efforts at improving emergency action plans,[15–17] automated external defibrillator accessibility,[18] and preparticipation cardiovascular screening (PPCS) for conditions leading to increased risk for SCD.[19–21] However, accurate data defining contemporary SCD incidence rates and how SCD incidence has evolved over time among young competitive athletes are limited.

The incidence and pathogeneses of SCD in athletes have important real-world implications, particularly in the context of efforts to mitigate risk. Estimates of SCD incidence in young athletes have been highly variable in previous studies, largely because of methodologic challenges. Some studies have lacked reliable methods for case identification (numerator) or a clearly defined population at risk (denominator). Many studies include populations with large age ranges (eg, 12 to 40 years) with widely varying risks for SCD, and many studies do not report incidence independently by population characteristics (eg, sex, race, sport). Despite these limitations, studies have consistently reported that male athletes have approximately 4 times the risk of female athletes,[1,2,4,5,10] and other factors such as race and sport are associated with differential risk.[1,2,22] Many studies focus only on sports-related SCD (ie, occurring during or within an hour of activity), which is a subset of all SCD in athletes occurring at any time and with any activity.[1,13,23]

This study examined SCD in a clearly defined and diverse population of college student athletes competing in the National Collegiate Athletic Association (NCAA) in the United States from 2002 through 2022 and builds on previous reports among this population.[1,24] We assessed the incidence of SCD over the course of the study period, underlying causes of death, and circumstances in which the SCD occurred among NCAA athletes.

METHODS

The data that support the findings of this study are available from the corresponding authors upon reasonable request. This study included NCAA athlete deaths from July 1, 2002, to June 30, 2022. Data were collected retrospectively for 2002 through 2004 and prospectively from 2005 through 2022. Each individual year was defined as the time period of July 1 through June 30 the following year to reflect the collegiate academic schedule. Athletes were defined as those competing in at least 1 varsity sport at Division I, II, or III institutions. Athletes who were not enrolled as a student at 1 of these institutions at the time of death were not included. Athletes from other athletic associations were not included. Athlete deaths were identified through 4 separate independent data sources—the NCAA resolutions list, the Parent Heart Watch database or prospective media report searches, NCAA insurance claims, and the National Center for Catastrophic Sports Injury Research database—which have been described previously.[1,2,13] All athlete deaths were compiled into 1 database, and duplicates were removed. For multisport athletes, their primary sport was classified as the sporting discipline with competitive season closest in time to their time of death. Because long-distance runners often compete in cross-country and track and field, they were included within both the track-and-field and cross-country sporting groups.

The cause of each athlete death was determined through a combination of different methods, including review of autopsy and other official documents reporting cause of death, Internet

searches for online media reports and obituaries, and e-mails or telephone calls to the next of kin, coaches, athletic trainers, coroners, medical examiners, scholarship foundations, or physicians involved in the case. Demographic characteristics including age, race, sporting discipline, and exertional status at the time of death (ie, exertional, nonexertional, or unknown) also were determined using these methods. If no medical examiner report or cardiac autopsy was available, race was determined by media reports or athlete photographs. A death was considered exertional if SCD occurred during exercise or within 1 hour of exercise cessation. SCD was defined as a sudden unexpected death attributable to a cardiac cause, or a sudden death in a structurally normal heart with no other explanation for death and a history consistent with cardiac-related death that occurred within 1 hour of symptom onset or an unwitnessed death occurring within 24 hours of the person having been alive. Unwitnessed deaths were not categorized as cardiac unless additional information such as autopsy, negative toxicology screen, or other information was available that could verify the death was cardiac.

Athlete deaths were categorized into different groups, including accident, cardiac, cancer, other medical, suicide, homicide, sickle cell trait, sports-related head injury, heatstroke, or unknown. If the cause of death could not be determined accurately, it was designated as unknown. The total number of NCAA athletes competing during the study period and total athlete time (athlete-years) was calculated using the NCAA Sports Sponsorship and Participation Rates Report, NCAA Student-Athlete Ethnicity Report, and the NCAA Sports Sponsorship and Participation Rates Database.[25–27]

The cause of SCD or postmortem findings at cardiac autopsy were determined upon review of available autopsy reports using previously published definitions (Table S1).[1] When >1 pathologic abnormality was present, the abnormality most likely related to the athlete's death was considered the primary cause of SCD. Autopsy reports were reviewed by an independent panel of physicians with experience in sports cardiology, sports medicine, and forensic and cardiovascular pathology. Differences of opinion were adjudicated by panel discussion. If the adjudicated diagnosis was different from the cause of death presented on the autopsy report, the adjudicated cause of death was presented in the current analysis. This study was approved by the Division of Human Subjects at the University of Washington.

Statistical Analysis

Standard descriptive statistics were used to describe the demographic data. Continuous variables are presented as means and standard deviations, and categorical variables are presented as frequencies and percentages. The incidence of SCD was calculated as the total adjudicated SCD cases/total number of athletes participating in sport. Given the low incidence rate, the incidence rate was multiplied by 100 000 to calculate the incidence per 100 000 athlete-years. Incidence rates for individual sports were calculated if there were ≥5 SCD events within a given sporting discipline. Incidence rates over a typical 4-year collegiate career were calculated by multiplying incidence by 4 and reported throughout the article as 4 athlete-years. Given the non-normal distribution of incidence rates of non–cardiovascular death and SCD over the time period, Poisson regression with robust standard errors was used to assess changes in incidence rates over time. Incidence rate ratios (IRRs) and their corresponding 95% confidence intervals are presented. IRRs were scaled from 1-year to 5-year intervals so that the exponentiated measures represent the change in the incidence rate of SCD and non–cardiovascular death per 5 units (years). To assess the trend in SCD and non–cardiovascular death incidence rates over the 20-year time period visually, cubic smoothing spline curves were created and are presented in conjunction with the observed yearly incidence rates. Because previous studies have demonstrated that Black male basketball players may have the highest incidence of SCD,[2,3] a multivariable logistic regression model was built to assess whether the sport of basketball was associated with SCD after controlling for race and sex. Statistical analyses were performed using R: A Language and Environment for Statistical Computing (https://www.R-project.org; R Core Team) and SAS (version 9.4; SAS Institutes).

RESULTS

Study Population

A total of 1102 deaths over 9 106 516 athlete-years were identified throughout the 20-year study period. The most common cause of death was by accident (eg, motor vehicle accidents, falls; Figure 1). SCD was the most common medical cause of death, consisting of 143 of 1102 cases (13%). Baseline characteristics among the total death and SCD groups are presented in Table 1.

Case Identification

Of 143 total cases of SCD identified, the NCAA resolutions list identified 117 (82%), the Parent Heart Watch database and media reports identified 89 (62%), the National Center for Catastrophic Sports Injury Research database identified 63 (44%), and insurance claims identified 27 (19%). The NCAA resolutions list identified a similar percentage of cases across all divisions (Division I, 80%; Division II, 83%; Division III, 84%), whereas the Parent Heart Watch database and media reports identified progressively fewer cases across divisions (Division I, 72%; Division II, 69%; Division III, 38%). The National Center for Catastrophic Sports Injury Research database primarily included only "sport-related" deaths until 2014, thus would not be expected to identify all deaths.

Incidence and Trends of SCD Over Time

The overall incidence of SCD among NCAA athletes for the study period was 1:63 682 athlete-years [95% CI, 1:54 065–1:75 010]. The incidence rate stratified by baseline demographics, NCAA division, and sporting discipline are presented in Table 2. Male athletes had a higher incidence of SCD than female athletes (1:43 348 [95% CI, 1:36 228–1:51 867] versus 1:164 504 athlete-years [95% CI, 1:110 552–1:244 787]), and Black athletes had a higher incidence of SCD than White athletes (1:26 704 [1:20 417–1:34 925] versus 1:74 581 athlete-years [1:60 247–1:92 326]). The IRR for male athletes versus

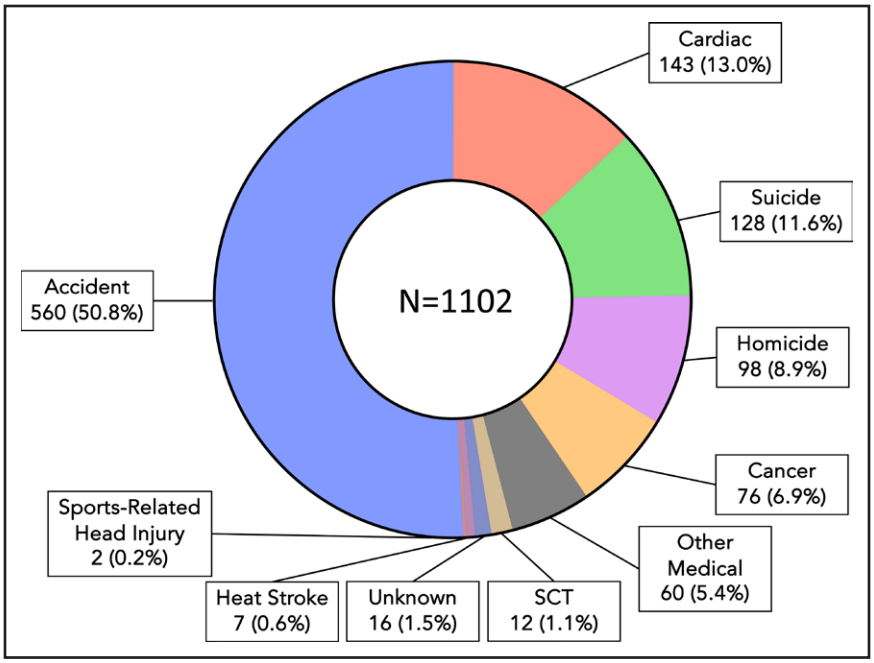

Figure 1. Causes of death among National Collegiate Athletic Association athletes (n=1102).
SCT indicates sickle cell trait.

female athletes was 3.79 (95% CI, 2.45–5.88) and 2.79 (95% CI, 1.98–3.94) for Black race versus White race. Among the sporting disciplines with ≥5 cases of SCD, basketball and football had the highest incidence of SCD (1:19 164 and 1:31 743 athlete-years, respectively). When assessing incidence rates stratified by sex, race (Black versus White), NCAA division, and sporting discipline, Division I White male basketball (1:5848 athlete-years [95% CI, 1:2498–1:13 691]) and Division II Black female track and field athletes (1:24 942 athlete-years [95% CI, 1:4404–1:141 294]) had the highest incidence rates of SCD between the sexes (Table 3). The overall incidence of SCD over a typical athlete's career was 1:15 921 per 4 athlete-years. When considering a 4-year career, Divison I White male basketball players have an incidence rate of 1:1462 per 4 athlete-years and Divison I Black male basketball players have an incidence rate of 1:1924 per 4 athlete-years. After controlling for sex and race in multivariable logistic regression analysis, athletes participating in basketball were still at increased risk of SCD compared with other sports (odds ratio, 2.75 [95% CI, 1.73–4.34]; Table S2).

The yearly incidence rates for both total non–cardiovascular deaths and SCDs is presented in Figure 2. The incidence rate for non–cardiovascular deaths showed no significant change over time (5-year IRR, 0.98 [95% CI, 0.94–1.04]), whereas the incidence rate for SCD decreased significantly over the study period (5-year IRR, 0.71 [95% CI, 0.61–0.82]).

Causes of SCD

A total of 117 autopsies, 10 death certificates, and 3 genetic testing reports were collected among 127 athletes with a clinical history suggestive of SCD or among those with unknown cause of death. Twenty-seven cases (21%) were adjudicated as noncardiac after thorough evaluation of their clinical history and postmortem documentation. The cause of death among these noncardiac cases included other medical causes (9 of 27), suicide (8 of 27), accidents (5 of 27), drug overdose (4 of 27), and homicide (1 of 27). There were no athlete deaths secondary to lightning strikes.

A total of 143 of 1102 cases (13%) were adjudicated to have a definitive or likely cardiac cause of SCD. Among these, 93 (65%) were identified by review of the autopsy, and the other 50 (35%) were identified by other methods, presented in Table 4. A cause of SCD was adjudicated in 118 of 143 cases (83%) reported from any source. Twenty-five of 143 cases (17%) were considered "cardiac unknown," either in athletes with an exertional death with sudden collapse requiring cardiopulmonary resuscitation and no other clear cause (7 of 25) or among athletes with documentation of a likely SCD but with no autopsy or the autopsy performed did not have enough information to adjudicate a most likely cause of SCD (18 of 25).

The most common postmortem finding was autopsy-negative sudden unexplained death (AN-SUD; 23 of 118 [19%]), followed by idiopathic left ventricular hypertrophy/possible cardiomyopathy (20 of 118 [17%]) and hypertrophic cardiomyopathy (15 of 118 [13%]; Figure 3). Causes of SCD among only athletes with an autopsy available for review and adjudication (n=93) are presented in Figure S1. Two of 118 athletes (2%) were adjudicated to have an SCD secondary to hypokalemia, including 1 athlete with hypokalemia related to known bulimia nervosa and 1 athlete with known Gitelman syndrome. Commotio cordis accounted for 2 deaths over the 20-year time period, both occurring in the first 5 years of the study period.

Table 1. Demographic Characteristics, Total Deaths, and Sudden Cardiac Deaths Among Athletes in the National Collegiate Athletic Association

Characteristics	Total deaths (n=1102)	SCDs (n=143)
Age, n (%)	20 (2)	20 (2)
Sex*		
Male	861 (78)	119 (83)
Female	241 (22)	24 (17)
Race*		
White	702 (64)	84 (59)
Black	330 (30)	53 (37)
Hispanic	26 (2)	4 (3)
Asian	21 (2)	1 (1)
Native Hawaiian or Pacific Islander	6 (1)	1 (1)
Other or mixed race	6 (1)	0
Unknown	11 (1)	0
National Collegiate Athletic Association division*		
I	466 (42)	64 (45)
II	314 (29)	42 (29)
III	322 (29)	37 (26)
Primary sport*		
Football	334 (30)	43 (30)
Basketball	132 (12)	35 (25)
Track and field	100 (9)	19 (13)
Soccer	105 (10)	12 (8)
Baseball	84 (7)	8 (6)
Swimming	43 (4)	8 (6)
Other	304 (28)	18 (13)

*Presented as average (SD).

Eight athletes were adjudicated to have myocarditis as a cause of SCD. Of these, only 1 case occurred after the first reported COVID-19 infection in the United States, and this case was giant-cell myocarditis per the local medical team and coroners, a pattern not associated with COVID-19. There were 10 cases of coronary artery abnormalities adjudicated as the cause of SCD (9 of 10 coronary artery anomalies, 1 of 10 myocardial bridge). Of the athletes with coronary anomalies and known anatomy, 6 of 8 (75%) had an anomalous left coronary artery originating from the right aortic sinus. The remaining 2 of 8 (25%) had an anomalous right coronary artery originating from the left aortic sinus, with associated scar of the inferior left ventricular wall in both cases suggestive of ischemia in the distribution of the right coronary artery. Among the coronary anomaly cases with an in-depth description provided by cardiac autopsy (n=4), all were described as having an acute takeoff angle and an interarterial course (eg, coronary artery coursing between the aorta and pulmonary artery).

SCD events were deemed exertional in 72 of 143 athletes (50%), nonexertional in 59 of 143 athletes (41%), and unknown in 12 of 143 athletes (8%). Exertional SCD was most commonly found in athletes with coronary artery anomalies (100%), arrhythmogenic cardiomyopathy (83%), or coronary artery disease (71%) (Figure 4).

DISCUSSION

This study examined the incidence and causes of SCD among NCAA athletes over a 20-year time period, with several key findings. First, the incidence of SCD among

Table 2. Incidence of Sudden Cardiac Death, by Demographic Characteristics

Characteristics	Sudden cardiac death cases, n (%)	Incidence rate, athlete-years	95% CI
Total	143 (100)	1:63 682	1:54 065–1:75 010
Sex			
Male	119 (83)	1:43 348	1:36 228–1:51 867
Female	24 (17)	1:164 504	1:110 552–1:244 787
Race			
White	84 (59)	1:74 581	1:60 247–1:92 326
Black	53 (37)	1:26 704	1:20 417–1:34 925
National Collegiate Athletic Association division			
I	64 (45)	1:54 173	1:42 428–1:69 170
II	42 (29)	1:50 303	1:37 218–1:67 990
III	37 (26)	1:95 313	1:69 154–1:131 368
Primary sport			
Football	43 (30)	1:31 743	1:23 568–1:42 754
Basketball	35 (25)	1:19 164	1:13 781–1:26 651
Male	30 (21)	1:11 799	1:8265–1:16 843
Female	5 (3)	1:63 357	1:27 063–1:148 329
Track and field*	19 (13)	1:54 703	1:35 022–1:85 444
Male	13 (9)	1:40 074	1:23 420–1:68 568
Female	6 (4)	1:86 401	1:39 598–1:188 520
Soccer	12 (8)	1:79 954	1:45 739–1:139 764
Male	10 (7)	1:45 714	1:24 832–1:84 158
Female	2 (1)	1:251 153	1:68 876–1:915 826
Cross-country	9 (6)	1:62 380	1:32 820–1:118 566
Male	7 (5)	1:38 334	1:18 569–1:79 134
Female	2 (1)	1:146 542	1:40 188–1:534 362
Baseball	8 (6)	1:81 078	1:41 085–1:160 004
Swimming	8 (6)	1:52 638	1:26 673–1:103 878
Male	5 (3)	1:35 995	1:15 375–1:84 269
Female	3 (2)	1:80 375	1:27 335–1:236 335

Values are n (%). Sports with ≥5 sudden cardiac death cases throughout the study period were included.

*Of the track and field athletes, 9 were distance runners competing in track and field and cross-country, and therefore are also counted in the cross-country section.

Table 3. Cohorts With Highest Sudden Cardiac Death Incidence Rates Within Black or White Race

Cohort	Sudden cardiac death cases	Incidence rate (athlete-years)	95% CI
Male			
Divison I, White, basketball	5	1:5848	1:2498–1:13 691
Divison I, Black, basketball	8	1:7696	1:3900–1:15 188
Divison II, Black, soccer	1	1:8140	1:1438–1:46 112
Divison II, Black, basketball	6	1:8542	1:3915–1:18 638
Divison II, Black, cross-country	1	1:8568	1:1513–1:48 536
Divison III, Black, basketball	5	1:8698	1:3716–1:20 363
Divison I, Black, cross-country	1	1:9026	1:1594–1:51 131
Divison I, Black, track and field	5	1:11 406	1: 4872–1:26 702
Divison II, White, basketball	3	1:11 882	1:4041–1:34 937
Divison I, White, swimming	4	1:14 575	1:5668–1:37 479
Divison I, Black, football	15	1:16 932	1:10 262–1:27 939
Divison II, White, football	9	1:17 938	1:9348–1:34 095
Female			
Divison II, Black, track and field	1	1:24 942	1:4404–1:141 294
Divison II, Black, basketball	1	1:30 880	1:5452–1:174 932
Divison I, Black, track and field	1	1:66 748	1:11 783–1:378 122
Divison I, White, swimming	2	1:43 296	1:11 874–1:157 878
Divison I, Black, basketball	1	1:46 566	1:8221–1:263 793
Divison II, White, basketball	1	1:46 725	1:8249–1:264 693
Divison III, White, basketball	2	1:47 336	1:12 982–1:172 608
Divison II, White, cross-country	1	1:48 754	1:8607–1:276 187
Divison II, White, track and field	1	1:72 858	1:12 862–1:412 735
Divison III, White, cross-country	1	1:92 041	1:15 815–1:507 504
Divison II, White, soccer	1	1:97 361	1:17 187–1:551 543
Divison III, White, track and field	1	1:61 182	1:16 779–1:223 099

Sports with ≥5 sudden cardiac death cases throughout the study period were included in the analysis (baseball, basketball, cross-country, football, soccer, swimming, and track and field). Of the track and field athletes, 9 were distance runners competing in track and field and cross-country, and therefore are also counted in the cross-country section.

NCAA athletes decreased throughout the 20-year study period, whereas noncardiovascular deaths remained stable. Second, the incidence of SCD among certain subgroups remains high (eg, Division I White and Black male basketball players have an incidence of SCD of >1:2000 over a typical 4-year collegiate career). Third, AN-SUD remains the most common postmortem finding among NCAA athletes (19%). Fourth, SCD occurred with exertion in only 50% of cases but this varied significantly by pathogenesis. Exertional death was most common among athletes with coronary artery anomalies and arrhythmogenic cardiomyopathy. Fifth, there were no cases of death attributable to COVID-19–related myocarditis in the 2.5 years after the beginning of the SARS-CoV-2 pandemic.

The true incidence of SCD among young competitive athletes has been a widely debated topic for decades, with incidence estimates varying widely as a function of study methodology.[1–3,7,11,12,14,22–24,28–31] This study included multiple methods for comprehensive case identification. No one method identified all cases of SCD, demonstrating the need for multiple methods of case identification when mandatory reporting is not required. This study also demonstrates the limits of reliance on media reports, particularly in lower-profile levels of sport. Catastrophic insurance claim data identified only 27 of 143 SCD cases (19%), indicating it is a poor method in isolation to identify the total number of SCD cases. In addition to multiple methods of case identification, this study used an accurate population at risk with demographic information available, including sex, race, and sport.

Efforts to improve primary (PPCS) and secondary (emergency action plans) prevention have been implemented over the past 20 years. In the present study, we found that the incidence of SCD among NCAA athletes

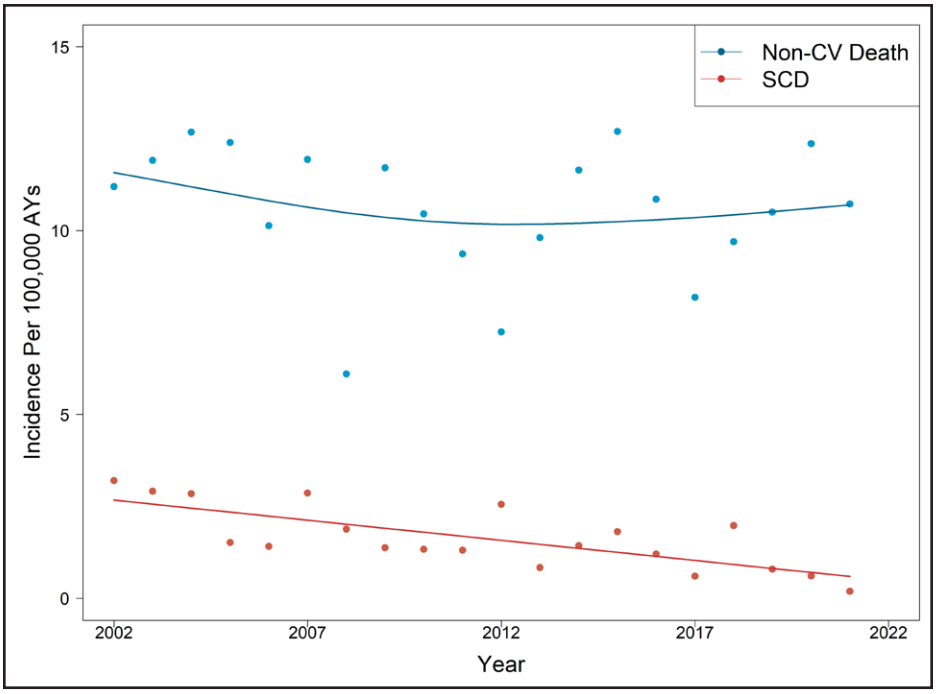

Figure 2. Yearly non–cardiovascular death and sudden cardiac death incidence among National Collegiate Athletic Association athletes.

Blue dots indicate non–cardiovascular death yearly incidence rate; blue line, cubic smoothing spline curve of non–cardiovascular (CV) death incidence over the study period; red dots, sudden cardiac death (SCD) yearly incidence rate; and red line, cubic smoothing spline curve of SCD incidence over the study period. AY indicates athlete-year.

has decreased over time (5-year IRR, 0.71 [95% CI, 0.61–0.82]). While the exact mechanism for this finding is unknown (because concurrent data on early detection and management of cardiac diseases identified by PPCS and the number of cases of resuscitated sudden cardiac arrest (SCA) were not available for the time period studied), our data suggest that strategies to reduce SCD among competing athletes may be having a positive effect. PPCS is required by the NCAA; however, an ECG is not required. A 2018 study reported that 56% of Power 5 schools used ECG in addition to history and physical examination for preparticipation screening.[32] It is unknown how often ECG is used in less-resourced institutions. PPCS with ECG and other modalities became common during the COVID-19 pandemic and some schools may have continued this pratice.[33] Cardiovascular screening following COVID-19 infection in young athletes often led to the diagnosis of underlying inherited cardiovascular conditions unassociated with COVID-19.[34] Likewise, improved survival after SCA could account for lower SCD rates.[35,36] Emergency action plans are required by the NCAA and should be reviewed and rehearsed annually.[17] Survival from SCA is increased when an athletic trainer is present, and has increased steadily over time in high school athletes with exercise-associated SCA.[36] A study in 2005 reported that 0 of 5 college athletes with SCD survived but later studies have shown progressively improved survival rates in young competitive athletes between 68% and 89%.[4,35–38]

In the current study, the most common pathogenesis of SCD or postmortem finding on cardiac autopsy was AN-SUD, which is similar to some[1,11,12] but not all[2,3,7,22,31] previous reports. The rate of AN-SUD was 19% when including multiple methods of case adjudication (Figure 3) and 23% when only including cases adjudicated by review of clinical history and cardiac autopsy (Figure S1). These data add to the growing literature suggesting that AN-SUD has a higher incidence than specific cardiomyopathic diagnoses such as hypertrophic cardiomyopathy. Because the addition of postmortem genetic testing to a cardiac autopsy can increase the diagnostic yield after an SCD event among those with AN-SUD, this remains an important area for future work.[29,39–43] Future research should analyze the frequency of recommendations from local coroners and diagnostic yield for postmortem genetic testing in AN-SUD cases. Investigation of the quality of life and diagnostic yield among families who undergo cascade screening to better understand the cause of their loved one's death or to prevent adverse outcomes among other family members should also be considered.

Despite overall decreasing rates of SCD, populations of NCAA athletes still have a significantly increased risk. Male athletes had almost 4 times the risk of SCD compared with female athletes and Black athletes had nearly 3 times the risk of SCD compared with White athletes. Male basketball and football athletes once again demonstrated an increased risk of SCD compared with

Table 4. Diagnoses for Non-Autopsy Adjudication of Sudden Cardiac Death (n=50)

Method of identification	No.
Talked with coroner, medical examiner, or medical team*	15
Anomalous coronary artery	2
Arrhythmogenic cardiomyopathy	1
Cardiac unknown	6
HCM	3
Hypokalemia	1
Kawasaki disease	1
Myocarditis	1
Media report	12
Anomalous coronary artery	2
Cardiac unknown	6
Commotio cordis	1
HCM	1
Myocarditis	1
Other (after cardiac transplant)	1
Exertional collapse without other explanation	7
Cardiac unknown	7
Death certificate	7
Cardiac unknown	3
HCM	2
Myocarditis	1
WPW	1
Personal or family history and history consistent with SCD	4
Cardiac unknown	1
Commotio cordis	1
Long QT syndrome	1
WPW†	1
Discussion with next of kin	4
AN-SUD	2
Cardiac unknown	2
Medical or legal report of autopsy	1
HCM	1

AN-SUD indicates autopsy-negative sudden unexplained death; HCM, hypertrophic cardiomyopathy; SCD, sudden cardiac death; and WPW, Wolff-Parkinson-White syndrome.

*All cases were adjudicated by discussion with the coroner, other than 1 case of HCM, which was discussed with the team athletic trainer, and 1 case of myocarditis by the treating physician.

†Death certificate was also obtained, which stated WPW as the cause of SCD.

other sports. When controlling for sex and race in multivariable logistic regression analysis, basketball remained independently associated with a higher risk of SCD. It is unclear why this association exists, and this requires further scientific inquiry. Given the high incidence rates in certain populations, more intensive screening strategies among these high-risk groups may be warranted.

There were no cases of SCD attributed to COVID-19 infection. There was one adjudicated case of myocarditis after the first reported US case of COVID-19 infection, but this was determined to be secondary to giant-cell myocarditis by local medical teams and coroners. This finding adds to previous studies among young competitive athletes that found no significant increase in adverse cardiovascular events after the diagnosis of COVID-19 infection with >1 year follow-up.[33,44] Recent current events have also renewed interest in commotio cordis.[45] There were only 2 cases of commotio cordis over the 20-year time period, although cases are thought to occur more frequently in pediatric athletes given a more compliant chest wall and participation in sports with a firm projectile.[46]

Limitations

This study has several limitations that warrant discussion. First, there is no mandatory reporting system throughout the United States, so it is possible that cases of SCD may have been missed, which would make incidence rates higher than reported. However, we used 4 independent search strategies to increase the accuracy of SCD estimates. Second, there is significant variability in the approach to cardiac autopsy as well as reporting, which can limit population-based studies.[47] Widespread adoption of practice recommendations and leveraging of cardiovascular pathology consultations and postmortem genetic studies may improve the diagnostic yield after an SCD event among those with AN-SUD.[29,39–43] Third, the cause of death was unknown in 16 cases, which could affect overall results. Fourth, postmortem genetic testing was only available among 3 of 93 athletes (3%). Fifth, we do not have data on the incidence of resuscitated SCA or PPCS practices and findings over the same time period to draw definitive conclusions regarding causal factors underlying the decreased incidence of SCD. The lack of data on resuscitated SCA may lead to bias in the current study, because some athlete subgroups may be more likely to be resuscitated than others, and also rates of resuscitated SCA in conjunction with SCD are important for a complete understanding of risk of conditions leading to SCA or SCD in young competitive athletes. Sixth, rates of SCD in nonathletic college students were not available to make a comparison between the rates of SCD among collegiate athletes and nonathletes. Seventh, race was determined by media reports or athlete photographs in cases without cardiac autopsy or medical examiner reports, which could have affected the results.

CONCLUSION

The incidence of SCD among NCAA athletes has decreased over 20 years. However, the incidence of SCD remains relatively high among male athletes, Black

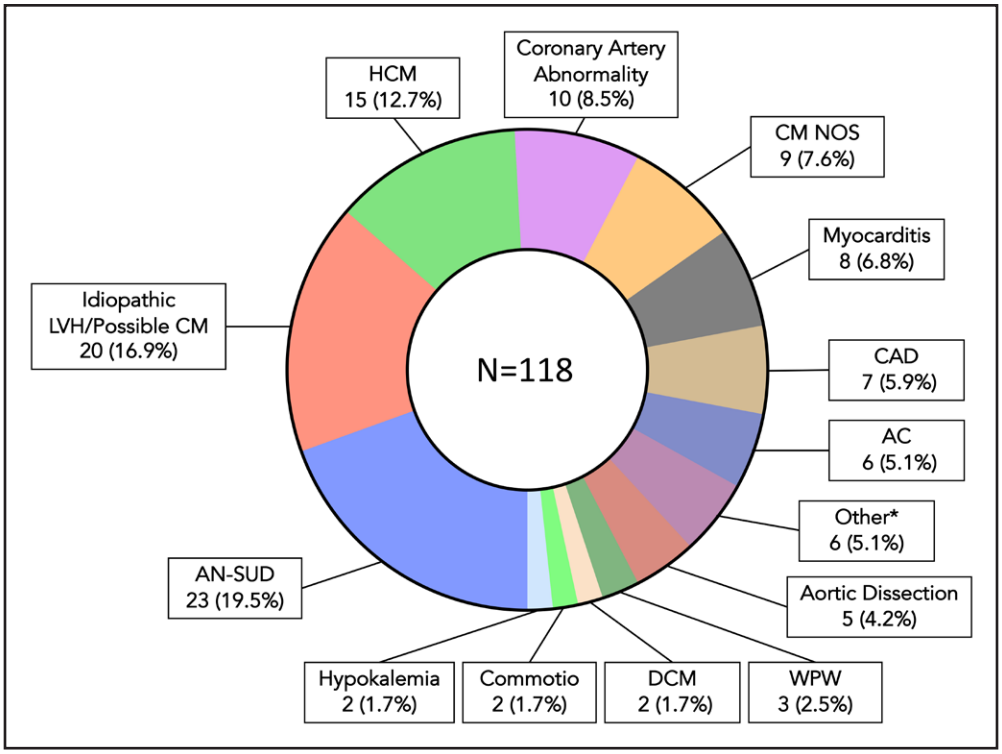

Figure 3. Causes of sudden cardiac death or findings on cardiac autopsy among National Collegiate Athletic Association athletes (n=118).

*Other: 1 each of long QT syndrome, complications of congenital heart disease, idiopathic left ventricular hypertrophy (LVH)/possible sickle cell trait, Kawasaki disease, complications after heart transplant, and sudden cardiac death in individual with pacemaker for idiopathic atrioventricular block. AC indicates arrhythmogenic cardiomyopathy; AN-SUD, autopsy-negative sudden unexplained death; CAD, coronary artery disease; CM, cardiomyopathy; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; NOS, not otherwise specified; and WPW, Wolff-Parkinson-White syndrome.

athletes, and male basketball athletes, with an incidence over a typical 4-year collegiate career among White and Black Division I male basketball athletes of >1:2000 in 4 athlete-years. Efforts to improve PPCS and timely recognition and care of an athlete after SCA represent opportunities to improve outcomes.

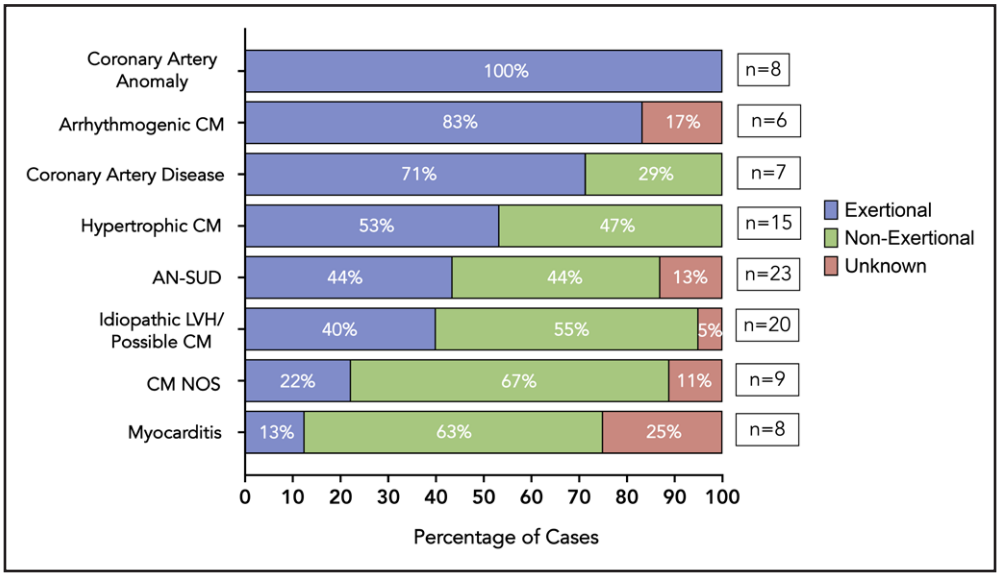

Figure 4. Exertional status at time of death by common causes of sudden cardiac death.

AN-SUD indicates autopsy-negative sudden unexplained death; CM, cardiomyopathy; LVH, left ventricular hypertrophy; NOS, not otherwise specified; and SCD, sudden cardiac death.

ARTICLE INFORMATION

Received July 2, 2023; accepted August 24, 2023.

Affiliations

Sports Cardiology Program, Knight Cardiovascular Institute, Oregon Health and Science University, Portland (B.J.P.). Division of Cardiology (B.J.P., T.W.C., A.L.B.), Cardiovascular Performance Program (B.J.P., T.W.C., A.L.B.), Massachusetts General Hospital, Boston. Division of Cardiology and Sports Cardiology BC, University of British Columbia, Vancouver, Canada (N.M.). Department of Orthopedics and Rehabilitation, University of Wisconsin Madison (S.A.K.). Swiss Olympic Medical Center, Lausanne University Hospital (CHUV), Switzerland (A.L.B.). Institute for Sport Science, University of Lausanne (ISSUL), Switzerland (A.L.B.). Department of Family Medicine and Center for Sports Cardiology, University of Washington, Seattle (J.A.D., D.M.S., L.S., M.Z.S., K.G.H.). Division of Cardiology, Duke Heart Center, and Duke Clinical Research Institute, Duke University School of Medicine, Durham, NC (M.R.P.). Department of Molecular Pharmacology & Experimental Therapeutics, Windland Smith Rice Sudden Death Genomics Laboratory (M.J.A.), Department of Cardiovascular Medicine, Division of Heart Rhythm Services, Windland Smith Rice Genetic Heart Rhythm Clinic (M.J.A.), Department of Pediatric and Adolescent Medicine, Division of Pediatric Cardiology (M.J.A.), and Department of Laboratory Medicine & Pathology (J.J.M.), Mayo Clinic, Rochester, MN. Department of Exercise and Sport Science, University of North Carolina at Chapel Hill (K.L.K.). Family and Community Medicine, The University of Alabama at Birmingham Heersink School of Medicine (I.M.A.).

Acknowledgments

This research is supported, in part, by the National Center for Catastrophic Sports Injury Research at the University of North Carolina at Chapel Hill. National Center for Catastrophic Sports Injury Research is supported by the National Collegiate Athletic Association, the National Federation of State High School Associations, the American Football Coaches Association, the National Athletic Trainers' Association, the National Operating Committee on Standards for Athletic Equipment, and the American Medical Society for Sports Medicine. Conclusions drawn from or recommendations based on the data provided by the National Center for Catastrophic Sports Injury Research are those of the authors and do not necessarily represent the official views of the National Center for Catastrophic Sports Injury Research or any of the supporters. The authors thank the Parent Heart Watch and National Collegiate Athletic Association for data collection and Kyle Conley for work on this study.

Sources of Funding

None.

Disclosures

Dr Ackerman is a consultant for Abbott, Boston Scientific, Bristol Myers Squibb, Daiichi Sankyo, Invitae, Medtronic, Tenaya Therapeutics, Thryv Therapeutics, and UpToDate. Dr Ackerman and Mayo Clinic are involved in an equity/royalty relationship with AliveCor, Anumana, ARMGO Pharma, and Pfizer. None of these entities was involved in this study. Dr Baggish has received funding from the National Institute of Health/National Heart, Lung, and Blood Institute, the National Football Players Association, the American Heart Association, and the American Medical Society for Sports Medicine to study cardiovascular outcomes among elite athletes and receives compensation for his role as team cardiologist from the US Olympic Committee/US Olympic Training Centers, US Soccer, and US Rowing. Dr Drezner has received funding from the American Medical Society for Sports Medicine, the American Heart Association, and the National Center for Catastrophic Sports Injury Research. Dr Harmon has received funding from the American Medical Society for Sports Medicine, Football Research, Inc, the Pac-12, and the American Heart Association. Dr Kucera is supported by funds from the National Center for Catastrophic Sports Injury Research.

Supplemental Material

Figure S1

Tables S1 and S2

REFERENCES

1. Harmon KG, Asif IM, Maleszewski JJ, Owens DS, Prutkin JM, Salerno JC, Zigman ML, Ellenbogen R, Rao AL, Ackerman MJ, et al. Incidence, cause, and comparative frequency of sudden cardiac death in national collegiate athletic association athletes: a decade in review. *Circulation*. 2015;132:10–19. doi: 10.1161/CIRCULATIONAHA.115.015431
2. Peterson DF, Kucera K, Thomas LC, Maleszewski J, Siebert D, Lopez-Anderson M, Zigman M, Schattenkerk J, Harmon KG, Drezner JA. Aetiology and incidence of sudden cardiac arrest and death in young competitive athletes in the USA: a 4-year prospective study. *Br J Sports Med*. 2021;55:1196–1203. doi: 10.1136/bjsports-2020-102666
3. Malhotra A, Dhutia H, Finocchiaro G, Gati S, Beasley I, Clift P, Cowie C, Kenny A, Mayet J, Oxborough D, et al. Outcomes of cardiac screening in adolescent soccer players. *N Engl J Med*. 2018;379:524–534. doi: 10.1056/NEJMoa1714719
4. Toresdahl BG, Rao AL, Harmon KG, Drezner JA. Incidence of sudden cardiac arrest in high school student athletes on school campus. *Heart Rhythm*. 2014;11:1190–1194. doi: 10.1016/j.hrthm.2014.04.017
5. Harmon KG, Asif IM, Maleszewski JJ, Owens DS, Prutkin JM, Salerno JC, Zigman ML, Ellenbogen R, Rao AL, Ackerman MJ, et al. Incidence and etiology of sudden cardiac arrest and death in high school athletes in the United States. *Mayo Clin Proc*. 2016;91:1493–1502. doi: 10.1016/j.mayocp.2016.07.021
6. Holst AG, Winkel BG, Theilade J, Kristensen IB, Thomsen JL, Ottesen GL, Svendsen JH, Haunso S, Prescott E, Tfelt-Hansen J. Incidence and etiology of sports-related sudden cardiac death in Denmark: implications for preparticipation screening. *Heart Rhythm*. 2010;7:1365–1371. doi: 10.1016/j.hrthm.2010.05.021
7. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes analysis of 1866 deaths in the United States, 1980-2006. *Circulation*. 2009;119:1085–1092. doi: 10.1161/CIRCULATIONAHA.108.804617
8. Marijon E, Tafflet M, Celermajer DS, Dumas F, Perier MC, Mustafic H, Toussaint JF, Desnos M, Rieu M, Benameur N, et al. Sports-related sudden death in the general population. *Circulation*. 2011;124:672–681. doi: 10.1161/CIRCULATIONAHA.110.008979
9. Risgaard B, Winkel BG, Jabbari R, Glinge C, Ingemann-Hansen O, Thomsen JL, Ottesen GL, Haunso S, Holst AG, Tfelt-Hansen J. Sports-related sudden cardiac death in a competitive and a noncompetitive athlete population aged 12 to 49 years: data from an unselected nationwide study in Denmark. *Heart Rhythm*. 2014;11:1673–1681. doi: 10.1016/j.hrthm.2014.05.026
10. Corrado D, Basso C, Rizzoli G, Schiavon M, Thiene G. Does sports activity enhance the risk of sudden death in adolescents and young adults? *J Am Coll Cardiol*. 2003;42:1959–1963. doi: 10.1016/j.jacc.2003.03.002
11. Finocchiaro G, Papadakis M, Robertus JL, Dhutia H, Steriotis AK, Tome M, Mellor G, Merghani A, Malhotra A, Behr E, et al. Etiology of sudden death in sports: insights from a United Kingdom regional registry. *J Am Coll Cardiol*. 2016;67:2108–2115. doi: 10.1016/j.jacc.2016.02.062
12. Finocchiaro G, Radaelli D, D'Errico S, Papadakis M, Behr ER, Sharma S, Westaby J, Sheppard MN. Sudden cardiac death among adolescents in the United Kingdom. *J Am Coll Cardiol*. 2023;81:1007–1017. doi: 10.1016/j.jacc.2023.01.041
13. Harmon KG, Asif IM, Klossner D, Drezner JA. Incidence of sudden cardiac death in National Collegiate Athletic Association athletes. *Circulation*. 2011;123:1594–1600. doi: 10.1161/CIRCULATIONAHA.110.004622
14. Maron BJ, Haas TS, Ahluwalia A, Rutten-Ramos SC. Incidence of cardiovascular sudden deaths in Minnesota high school athletes. *Heart Rhythm*. 2013;10:374–377. doi: 10.1016/j.hrthm.2012.11.024
15. Casa DJ, Almquist J, Anderson SA, Baker L, Bergeron MF, Biagioli B, Boden B, Brenner JS, Carroll M, Colgate B, et al. The inter-association task force for preventing sudden death in secondary school athletics programs: best-practices recommendations. *J Athl Train*. 2013;48:546–553. doi: 10.4085/1062-6050-48.4.12
16. Drezner JA, Courson RW, Roberts WO, Mosesso VN Jr, Link MS, Maron BJ; Inter-Association Task Force. Inter-association task force recommendations on emergency preparedness and management of sudden cardiac arrest in high school and college athletic programs: a consensus statement. *Heart Rhythm*. 2007;4:549–565. doi: 10.1016/j.hrthm.2007.02.019
17. Casa DJ, Guskiewicz KM, Anderson SA, Courson RW, Heck JF, Jimenez CC, McDermott BP, Miller MG, Stearns RL, Swartz EE, et al. National Athletic Trainers' Association position statement: preventing sudden death in sports. *J Athl Train*. 2012;47:96–118. doi: 10.4085/1062-6050-47.1.96
18. Mason Z, Watson AM, Drezner JA. Emergency preparedness for sudden cardiac arrest in amateur athletic union basketball teams: an opportunity to improve outcomes in higher risk athletes. *Clin J Sport Med*. 2022;32:617–619. doi: 10.1097/JSM.0000000000001062
19. Drezner JA, O'Connor FG, Harmon KG, Fields KB, Asplund CA, Asif IM, Price DE, Dimeff RJ, Bernhardt DT, Roberts WO. AMSSM position statement on cardiovascular preparticipation screening in athletes: current evidence, knowledge gaps, recommendations and future directions. *Br J Sports Med*. 2017;51:153–167. doi: 10.1136/bjsports-2016-096781

20. Hainline B, Drezner J, Baggish A, Harmon KG, Emery MS, Myerburg RJ, Sanchez E, Molossi S, Parsons JT, Thompson PD. Interassociation consensus statement on cardiovascular care of college student-athletes. *Br J Sports Med*. 2017;51:74–85. doi: 10.1136/bjsports-2016-096323
21. Maron BJ, Zipes DP, Kovacs RJ. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: preamble, principles, and general considerations: a scientific statement from the American Heart Association and American College of Cardiology. *J Am Coll Cardiol*. 2015;66:2343–2349. doi: 10.1016/j.jacc.2015.09.032
22. Maron BJ, Haas TS, Murphy CJ, Ahluwalia A, Rutten-Ramos S. Incidence and causes of sudden death in US college athletes. *J Am Coll Cardiol*. 2014;63:1636–1643. doi: 10.1016/j.jacc.2014.01.041
23. Jayaraman R, Reinier K, Nair S, Aro AL, Uy-Evanado A, Rusinaru C, Stecker EC, Gunson K, Jui J, Chugh SS. Risk factors of sudden cardiac death in the young: multiple-year community-wide assessment. *Circulation*. 2018;137:1561–1570. doi: 10.1161/CIRCULATIONAHA.117.031262
24. Harmon KG, Asif IM, Klossner D, Drezner JA. Incidence of sudden cardiac death in National Collegiate Athletic Association athletes. *Circulation*. 2011;123:1594–1600. doi: 10.1161/CIRCULATIONAHA.110.004622
25. Irick E. *NCAA Sports Sponsorship and Participation Rates Report (1981-82 through 2012-13)*. The National Collegiate Athletic Association; 2013.
26. Irick E. *Student Athlete Ethnicity Report*. The National Collegiate Athletic Association; 2013.
27. National Collegiate Athletic Association. NCAA Sports Sponsorship and Participation Rates Database. Published December 2022. https://www.ncaa.org/sports/2018/10/10/ncaa-sports-sponsorship-and-participation-rates-database.aspx. Accessed May 28, 2023.
28. Landry CH, Allan KS, Connelly KA, Cunningham K, Morrison LJ, Dorian P, Rescu I. Sudden cardiac arrest during participation in competitive sports. *N Engl J Med*. 2017;377:1943–1953. doi: 10.1056/NEJMoa1615710
29. Bagnall RD, Weintraub RG, Ingles J, Duflou J, Yeates L, Lam L, Davis AM, Thompson T, Connell V, Wallace J, et al. A prospective study of sudden cardiac death among children and young adults. *N Engl J Med*. 2016;374:2441–2452. doi: 10.1056/NEJMoa1510687
30. Karam N, Pechmajou L, Narayanan K, Bougouin W, Sharifzadehgan A, Anys S, Weizman O, Perrot D, Waldmann V, Beganton F, et al. Evolution of incidence, management, and outcomes over time in sports-related sudden cardiac arrest. *J Am Coll Cardiol*. 2022;79:238–246. doi: 10.1016/j.jacc.2021.11.011
31. Maron BJ, Haas TS, Ahluwalia A, Murphy CJ, Garberich RF. Demographics and epidemiology of sudden deaths in young competitive athletes: from the United States national registry. *Am J Med*. 2016;129:1170–1177. doi: 10.1016/j.amjmed.2016.02.031
32. Miars CW, Stamatis A, Morgan GB, Drezner JA. Cardiovascular screening practices and attitudes from the NCAA autonomous "Power" 5 conferences. *Sports Health*. 2018;10:547–551. doi: 10.1177/1941738118775039
33. Moulson N, Petek BJ, Drezner JA, Harmon KG, Kliethermes SA, Patel MR, Baggish AL, Asif IM, Borchers J, Edenfield KM, et al. SARS-CoV-2 cardiac involvement in young competitive athletes. *Circulation*. 2021;144:256–266. doi: 10.1161/CIRCULATIONAHA.121.054824
34. Klein CF, Petek BJ, Moulson N, Baggish AL, Churchill TW, Harmon KG, Kliethermes SA, Patel MR, Drezner JA. Non-COVID–19 cardiovascular pathology from return-to-play screening in college athletes after COVID-19 [published online July 17, 2023]. *Heart*. doi: 10.1136/heartjnl-2023-322645. https://heart.bmj.com/content/early/2023/07/17/heartjnl-2023-322645
35. Drezner JA, Peterson DF, Siebert DM, Thomas LC, Lopez-Anderson M, Suchsland MZ, Harmon KG, Kucera KL. Survival after exercise-related sudden cardiac arrest in young athletes: can we do better? *Sports Health*. 2019;11:91–98. doi: 10.1177/1941738118799084
36. Schattenkerk J, Kucera K, Peterson DF, Huggins RA, Drezner JA. Socioeconomic factors and outcomes from exercise-related sudden cardiac arrest in high school student-athletes in the USA. *Br J Sports Med*. 2022;56:138–143. doi: 10.1136/bjsports-2021-104486
37. Drezner JA, Rogers KJ, Zimmer RR, Sennett BJ. Use of automated external defibrillators at NCAA Division I universities. *Med Sci Sports Exerc*. 2005;37:1487–1492. doi: 10.1249/01.mss.0000177591.30968.d4
38. Drezner JA, Rao AL, Heistand J, Bloomingdale MK, Harmon KG. Effectiveness of emergency response planning for sudden cardiac arrest in united states high schools with automated external defibrillators. *Circulation*. 2009;120:518–525. doi: 10.1161/CIRCULATIONAHA.109.855890
39. Fellmann F, van El CG, Charron P, Michaud K, Howard HC, Boers SN, Clarke AJ, Duguet A-M, Forzano F, Kauferstein S, et al; on behalf of European Society of Human Genetics, European Council of Legal Medicine, European Society of Cardiology working group on myocardial and pericardial diseases, European Reference Network for rare, low prevalence and complex diseases of the heart (ERN GUARD-Heart), Association for European Cardiovascular Pathology. European recommendations integrating genetic testing into multidisciplinary management of sudden cardiac death. *Eur J Human Genet*. 2019;27:1763–1773. doi: 10.1038/s41431-019-0445-y
40. Lahrouchi N, Raju H, Lodder EM, Papatheodorou E, Ware JS, Papadakis M, Tadros R, Cole D, Skinner JR, Crawford J, et al. Utility of post-mortem genetic testing in cases of sudden arrhythmic death syndrome. *J Am Coll Cardiol*. 2017;69:2134–2145. doi: 10.1016/j.jacc.2017.02.046
41. Stiles MK, Wilde AAM, Abrams DJ, Ackerman MJ, Albert CM, Behr ER, Chugh SS, Cornel MC, Gardner K, Ingles J, et al. 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–e50. doi: 10.1016/j.hrthm.2020.10.010
42. Ackerman MJ. State of postmortem genetic testing known as the cardiac channel molecular autopsy in the forensic evaluation of unexplained sudden cardiac death in the young. *Pacing Clin Electrophysiol*. 2009;32:S86–S89. doi: 10.1111/j.1540-8159.2009.02393.x
43. Isbister JC, Nowak N, Yeates L, Singer ES, Sy RW, Ingles J, Raju H, Bagnall RD, Semsarian C. Concealed cardiomyopathy in autopsy-inconclusive cases of sudden cardiac death and implications for families. *J Am Coll Cardiol*. 2022;80:2057–2068. doi: 10.1016/j.jacc.2022.09.029
44. Petek BJ, Moulson N, Drezner JA, Harmon KG, Kliethermes SA, Churchill TW, Patel MR, Baggish AL, Asif IM, Baggish AL, et al. Cardiovascular outcomes in collegiate athletes following SARS-CoV-2 infection: 1-year follow-up from the outcomes registry for cardiac conditions in athletes. *Circulation*. 2022;145:1690–1692. doi: 10.1161/circulationaha.121.058272
45. Association of Black Cardiologists. ABC Issues Statement on Buffalo Bills Safety Damar Hamlin's Return to the NFL. Published June 20, 2023. https://www.prnewswire.com/news-releases/abc-issues-statement-on-buffalo-bills-safety-damar-hamlins-return-to-the-nfl-301855720.html. Accessed June 21, 2023.
46. Link MS. Commotio cordis. *Circ Arrhythm Electrophysiol*. 2012;5:425–432. doi: 10.1161/CIRCEP.111.962712
47. Kelly KL, Lin PT, Basso C, Bois M, Buja LM, Cohle SD, d'Amati G, Duncanson E, Fallon JT, Firchau D, et al. Sudden cardiac death in the young: a consensus statement on recommended practices for cardiac examination by pathologists from the Society for Cardiovascular Pathology. *Cardiovasc Pathol*. 2023;63:107497. doi: 10.1016/j.carpath.2022.107497