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A 10-year review of sudden death during sporting activities

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BACKGROUND Sudden death during sport is a rare but devastating event. Previous research has mostly focused on sudden deaths in young competitive athletes.

OBJECTIVE The purpose of this study was to characterize the demographics and etiologies of sudden cardiac death during sport in Australia.

METHODS All autopsies conducted at our forensic medicine facility between 2006 and 2015 inclusive were reviewed. Sporting-related deaths among those 7–65 years of age were identified. Data collected included subject height, weight, gender, circumstances of death, and pathologic findings at autopsy.

RESULTS A total of 19,740 autopsies were completed in the study period: 12,395 in subjects age 18–65 years (adults) and 385 in subjects age 7–17 years (children). There were 201 sports-related adult deaths at an incidence rate of 0.76–1.49 per 100,000 participant-years. Of the deaths, 74% were witnessed. Of the adult cases, 68% (n = 136) were due to cardiac causes, with coronary artery

disease the most frequent cause (n = 90 [45%]). Structural abnormalities were common in adult cardiac deaths; 51 (38%) had cardiac weight ≥ 500 g, and 75 (55%) had left ventricular wall thickness >15 mm. Of the 15 child deaths, 5 (33%) were arrhythmogenic or presumed arrhythmic, and 5 (33%) were inherited cardiomyopathies (2 hypertrophic cardiomyopathy, 3 arrhythmogenic right ventricular cardiomyopathy).

CONCLUSION Sudden cardiac death during sport is rare. Deaths are mostly due to coronary artery disease in adults and cardiomyopathy or arrhythmia in children. Because the majority of sports deaths are witnessed, they present an opportunity to enhance outcomes by cardiopulmonary resuscitation training and increased availability of automated external defibrillators at sports venues.

KEYWORDS Athletes; Cardiac arrest; Sports; Sudden cardiac death; Sudden death

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Introduction

Sudden death during sport is a rare but devastating event that generates significant social and media attention.¹ Sudden cardiac death in the young² and in young athletes^{3,4} has been extensively investigated, with screening in this population being intensely debated.⁵

Many developed countries have aging populations, with increasing numbers of “older” athletes. Furthermore, most

sports-related deaths occur in those older than 35 years.^{6,7} Research into sport-related deaths in adults is required to better inform sports organizations, the public, and sportspeople, and to guide prevention strategies including screening and use of automated external defibrillators.⁸ We performed a retrospective study to identify the incidence, circumstances, and pathology of deaths related to sporting activity in children and adults between 2006 and 2015 in New South Wales (NSW), Australia.

Methods

All autopsies conducted at the Department of Forensic Medicine (DOFM), Glebe, NSW, Australia, between 2006 and 2015 inclusive were reviewed. The DOFM, Glebe, performs $>60\%$ of autopsies in NSW (population 7.5 million). All sudden unexpected deaths without a known cause are referred for investigation by the DOFM.

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Case identification

All autopsies completed by the DOFM are maintained in a coronial database. This includes all files relating to the coronial case, including, but not limited to, police reports, witness statements, family interviews, medical records, and autopsy and coronial reports. The database was searched using Perceptive Search Software (Perceptive Software, Shawnee, KS). This search engine flags the search term and derivations of the search term in any document in the database. For example, the word “Football” will also identify cases that include “Football socks” and “Footballer,” as well as “the deceased was playing football.” Two of the authors (MD, AE) reviewed the flagged files to ensure the cases met age criteria. If the age criteria were met, the reports were reviewed in detail to ensure the deaths met inclusion criteria and did not meet exclusion criteria. All cases were cross-referenced to ensure no duplication.

Search terms

The terms “sudden death,” “sudden cardiac,” and “cardiac arrest” were searched to increase search sensitivity. Additional searches were made for specific sports, for example, “Basketball.” (For a full list of search terms, see [Supplementary Table S1](#)). “Walking” and “Yoga” were not included. Individual sport searches were chosen to cover common sports participated in Australia according to the Australian Bureau of Statistics and Australian Sports Commission data.^{9,10} They include the top 10 sports and >70% of total sports participated in Australia.¹⁰

Inclusion criteria

Deaths included subjects age 18–65 years inclusive (adult cohort) and age 7–17 years inclusive (child cohort). The same search strategy and terms were applied to the DOFM database for each cohort. Deaths were classified as “sudden” if the death occurred within 1 hour from onset of symptoms in witnessed cases and within 24 hours of the individual last being seen alive in unwitnessed cases. “Cardiac” deaths were defined as those that, based on available evidence, were caused by abnormalities directly related to the cardiovascular system. Noncardiac deaths were those that did not meet the “cardiac” criteria. Sporting deaths were further classified as “organized sport”—sporting activity requiring registration to an organization; “regular nonorganized sport”—sporting activity participated on at least a weekly basis for fitness or recreation that did not require registration with a sporting or organizational body; or “leisure activity”—*ad hoc* sport without specific training or organization. Deaths by suicide, homicide, or drug overdose were excluded.

Postmortem examination was completed by experienced forensic pathologists in accordance with Royal College of Pathologists Australasia guidelines as previously described.² Patient data collected included subject age, gender, height, weight, background medical history, and immediate circumstances at the time of death. Pathologic findings of all included patients were recorded, including macroscopic

and histologic examination of internal organs. Multiple myocardial blocks were reviewed. Toxicologic analysis was performed in all cases, except for those for which the mechanism of death was deemed clear, such as a witnessed cycling accident with fatal head trauma for which an abbreviated autopsy may be completed. In cases having a cause of death that was difficult to ascertain based on the initial pathologist report, 2 authors (IB, RP) reviewed the available data and with 2 other authors (MD, AE) reached a consensus on the final cause of death.

Definitions

Presumed arrhythmic deaths were defined using the classification of Hinkle and Thaler¹¹ in the absence of significant coronary artery disease or cardiomyopathy defined as follows. Significant coronary artery disease was defined as a lesion >70 of coronary artery lumen diameter. Coronary artery disease related death was defined when significant coronary disease was present or when there was evidence of plaque rupture and/or myocardial ischemia/necrosis. Left ventricular hypertrophy (LVH) was defined as >15 mm thickness at the left ventricular (LV) free wall. Diagnosis of specific cardiomyopathies was based on international guidelines.^{12,13}

Statistical analysis

Continuous variables are summarized using mean \pm SD. Percentages are used for categorical variables. The Student *t* test was used to detect a difference in the means for continuous variables. Comparison of categorical variables was completed using the χ^2 or Fisher exact test where appropriate. *P* < .05 was considered significant. Incidence rates per 100,000 participant-years were calculated by dividing the number of events in 1 year (total deaths or sudden cardiac deaths) by the total number of participants of sporting activity in NSW identified in government-collected data,^{14,15} adjusted for the proportion of autopsies in NSW served by the DOFM, Glebe. Statistical analysis was performed using IBM SPSS Statistics for Macintosh, version 24.0 (IBM Corp, Armonk, NY).

Ethics approval

The study was conducted with permission from the Office of the NSW State Coroner and performed in accordance with the Sydney Local Health District ethics guidelines, Sydney Local Health District (SLHD) Ethics Review Committee, Protocol Number X15-0171. All autopsies were performed in accordance with the New South Wales Human Tissue Act 1983.

Results

From 2006 to 2015 inclusive, 19,740 total deaths underwent coronial autopsy: 12,395 in subjects age 18–65 years (adult cohort) and 385 in subjects age 7–17 years (child cohort). There were 216 sudden sporting deaths in total; of these, 201 met inclusion criteria in the adult cohort and 15 in the child cohort ([Figure 1](#)).

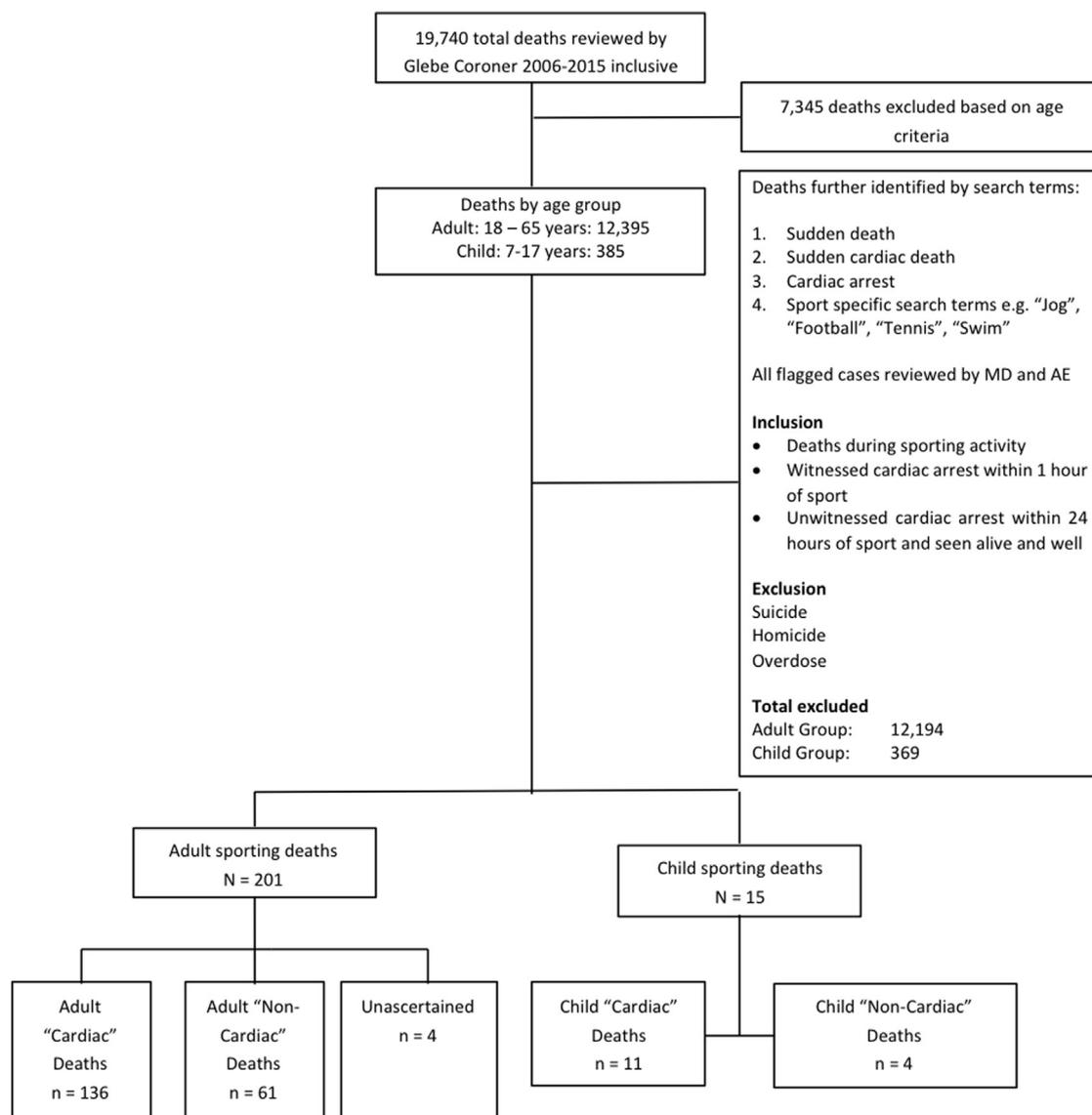


Figure 1 Identification and classification of sports-related deaths.

Adult cohort

Details of the adult cohort are listed in [Table 1](#) and deaths by sport in [Table 2](#). Cardiac causes accounted for 136 of total adult deaths (68%); 61 (30%) were noncardiac, and 4 (2%) were undetermined.

Mean age at death was 44 ± 13.5 years; 90% (180) were male. One hundred forty deaths (71%) occurred at ≥ 35 years of age. One hundred ninety-two deaths (96%) occurred at the time of the sporting event; 157 deaths (78%) were witnessed. Coronary artery disease ($n = 90$ [45%]) was the most common cause of death overall, and drowning ($n = 39$ [19%]) was the most common cause of noncardiac deaths.

When compared to NSW matched sporting participation, the annual death incidence rate was 0.76–1.49 per 100,000 participant-years for total deaths and 0.50–0.98 for cardiac deaths over the study period.

Adult cardiac deaths

Causes of cardiac death are listed in [Table 3](#). The mean age of cardiac death patients was 47 ± 12 years; 126 (93%) were male. One hundred twenty-nine of the 136 cardiac deaths (95%) occurred during sport activity, and 101 (74%) of cardiac deaths had witnessed arrests. Among cases for which information on cardiac risk factors was available; 3% had a history of ischemic heart disease, 3% had a confirmed history of diabetes mellitus, 21% were current or ex-smokers, 18% had a history of hypercholesterolemia, and 24% a history of hypertension.

Cardiac decedents had a higher mean body mass index (BMI; 30.3 vs 26.7 kg/m^2 ; $P < .001$), cardiac weight (489 ± 112 g vs 370 ± 75.2 g; $P < .001$), and LV wall thickness (17 ± 3.5 mm vs 14 ± 2.4 mm; $P < .001$) than those who died of noncardiac causes.

Table 1 Characteristics of adult sudden deaths age 18–65 years

	All deaths	Cardiac deaths	Noncardiac death	P value
No. of subjects	201	136	61	
Age (years)	44 ± 13.5	47 ± 12.0	36 ± 13.9	<.001
Organized sport	46 ± 12.6	47 ± 12.2	37 ± 12.5	
Regular sport	46 ± 12.7	48 ± 12.4	39 ± 12.7	
Leisure sport	39 ± 14.4	48 ± 11.0	36 ± 14.6	<.001
Male	180 (90)	126 (93)	52 (85)	.07
Age >35 years	140 (71)	109 (78)	28 (45)	<.001
Body mass index	29 ± 5.9	30 ± 6.3	27 ± 4.3	<.001
Documented risk factors				
At least 1	55 (27)	50 (37)	4 (7)	<.001
≥2	23 (11)	23 (17)	0	<.001
Sporting activity				
Organized sport	95 (47)	82 (60)	11 (18)	<.001
Regular sport	40 (20)	33 (24)	7 (11)	<.03
Leisure Sport	66 (33)	20 (15)	43 (71)	<.001
Structural changes				
Mean cardiac weight (g)	456 ± 116	489 ± 112	370 ± 75.2	<.001
Cardiac weight >500 g	52 (26)	51 (38)	1 (2)	<.001
Mean LV wall thickness (mm)	16 ± 3.5	17 ± 3.3	14 ± 2.4	<.001
LV wall thickness >15 mm	83 (43)	75 (55)	8 (13)	<.001
Mean septal thickness	16 ± 3.3	17 ± 3.3	14 ± 2.3	<.3
Evidence of fibrosis	115 (57)	106 (78)	8 (13)	<.001

Values are given as n, mean ± SD, or n (%) unless otherwise indicated.
LV = left ventricle.

Coronary Artery Disease (n = 90)

The mean age of coronary artery disease death patients was 49 ± 10.1 years; BMI was 31 ± 5.5 kg/m². Twenty-five (28%) had 3-vessel disease, 23 (26%) had 2-vessel disease, 37 (41%) had single-vessel disease, and 8 (9%) had significant left main disease.

Presumed arrhythmic deaths

Presumed arrhythmic deaths occurred in 19% (26) of cases; mean age was 47 ± 14.0 years. Only 5 patients had a completely structurally normal heart at autopsy, and no cases

at the time of death had a confirmed past history of a channelopathy. Sixteen patients (62%) had evidence of fibrosis, and 17 (65%) had LV wall thickness >15 mm. Eleven of the patients with presumed arrhythmic death patients had cardiac mass >500 g.

Myocarditis and cardiomyopathies

Histologically proven myocarditis occurred in 2 patients. Only 6 (4%) had a documented primary diagnosis of dilated cardiomyopathy. Five patients were diagnosed with hypertrophic cardiomyopathy (HCM), 3 arrhythmogenic right ventricular cardiomyopathy, 2 cardiac sarcoidosis, and 2 aortic dissection.

Fifty-one patients (38%) with cardiac deaths had cardiac weight >500 g; 42 (82%) of these displayed fibrosis on histology. Forty-three hearts (84%) with cardiac weight >500 g had LV wall thickness >15 mm. Forty (78%) of those with cardiac weight >500 g had defined structural heart abnormalities; the majority were coronary artery disease (n = 31). Other defined abnormalities in patients with cardiac weight

Table 2 Adult deaths by sport

Sport	Total deaths		Cardiac deaths	
	n (%)	Age (years)	n (%)	Age (years)
Soccer	21 (10)	41.8 ± 9.46	21 (15)	41.8 ± 9.46
Football	2 (1)	23.5 ± 3.54	0 (0)	—
Gym	29 (14)	45.4 ± 11.10	27 (20)	46.3 ± 11.10
Touch/Oztag	7 (4)	39.4 ± 9.96	7 (5)	39.4 ± 9.96
Swimming	59 (29)	39.7 ± 14.56	17 (13)	51.2 ± 12.6
League/union	3 (2)	34.0 ± 6.25	2 (1)	36.5 ± 6.25
Tennis	6 (3)	56.7 ± 8.82	6 (4)	56.7 ± 8.82
Golf	12 (6)	58.1 ± 8.18	12 (9)	58.1 ± 8.18
Netball	2 (1)	26.5 ± 0.71	2 (1)	26.5 ± 0.71
Rowing	1 (1)	63.0 (mean)	1 (1)	63.0 (mean)
Basketball	3 (2)	51.7 ± 5.77	3 (2)	51.7 ± 5.78
Cycling	22 (11)	50.9 ± 12.57	9 (7)	54.3 ± 6.25
Running	28 (14)	41.9 ± 11.86	24 (18)	44.0 ± 11.24
Cricket	5 (3)	33.4 ± 12.90	4 (3)	35.5 ± 13.87
Squash	1 (1)	64.0 (mean)	1 (1)	64 (mean)
Total	201		136	

Values are given as n (%) or mean ± SD unless otherwise indicated.

Table 3 Cardiac causes of deaths

Coronary artery disease	90 (66.2)
Presumed arrhythmia	26 (19.1)
Dilated cardiomyopathy	6 (4.4)
Hypertrophic cardiomyopathy	5 (3.7)
Arrhythmogenic right ventricular cardiomyopathy	3 (2.2)
Sarcoidosis	2 (1.5)
Myocarditis	2 (1.5)
Aortic dissection	2 (1.5)
Total	136 (100)

Values are given as n (%).

>500 g included HCM in 3, dilated cardiomyopathy in 3, sarcoidosis in 1, aortic dissection in 1, and myocarditis in 1 (Table 4). The remaining 11 deaths were presumed to be arrhythmic deaths, of which 10 (91%) had LVH >15 mm; 5 (45%) had fibrosis.

Cardiac decedents with cardiac weight >500 g were older (51.2 ± 10.7 years vs 41.8 ± 12.9 years; $P < .001$) and had a higher BMI (31.2 ± 6 kg/m² vs 28.33 ± 5.5 kg/m²; $P < .001$) than those with cardiac weight <500 g.

Adult noncardiac deaths

There were 61 noncardiac deaths during sport; mean age was 36 ± 13.9 years. Of these, 39 (64%) were drowning deaths, and 18 (30%) were traumatic (due to a high rate of cycling or road accidents). Four deaths were classified as “other”: 1 from snake envenomation while jogging, 1 accidental death by smoke inhalation after sport, 1 from an obstructed 3rd ventricle, and 1 secondary to a spontaneous subarachnoid hemorrhage. There were no sporting deaths due to epilepsy.

Undetermined deaths

In 4 patients (2%) the cause of death could not be reliably determined. All but 1 of these deaths occurred when the patient was either swimming or scuba diving and had circumstances and/or pathology that made the final diagnosis not possible by either the initial forensic pathologist or the reviewing study authors.

Child cohort

Fifteen deaths occurred in the child cohort (Table 5). Thirteen deaths (87%) occurred during sporting activity. The remaining 2 deaths (13%) occurred within 24 hours after sporting activity. Sixty-seven percent were witnessed; 11 (73%) were attributable to cardiac causes. Only 1 patient had a positive family history (long QT syndrome); no patients died of coronary artery disease, anomalous coronary artery abnormality, sarcoidosis, or myocarditis. One patient with Glanzmann thrombasthenia died of a neurologic bleed after a head strike, and 1 child died of peritonitis after a football tackle.

Discussion

In this retrospective autopsy study spanning more than 10 years and more than 12,000 cases, sports-related cardiac deaths were rare. Deaths were largely due to coronary artery disease in adults and cardiomyopathy or arrhythmia in younger individuals. Because the majority of sports deaths

Table 4 Cardiac deaths with cardiac weight >500 g

Coronary artery disease	31
Presumed arrhythmia	11
Hypertrophic cardiomyopathy	3
Dilated cardiomyopathy	3
Sarcoidosis	1
Aortic dissection	1
Myocarditis	1
Total	51

Values are given as n.

Table 5 Characteristics of the 15 sudden deaths in the child cohort

No. of subjects	15
Age (years)	13 ± 3.6
Male	14 (93%)
Body mass index (kg/m ²)	22 ± 3.2
Cause of death	
Arrhythmic	5 (33%)
Hypertrophic cardiomyopathy	2 (17%)
Arrhythmogenic right ventricular cardiomyopathy	3 (17%)
Cardiomyopathy	1 (6%)
Drowning	2 (11%)
Peritonitis	1 (6%)
Neurologic (intracerebral hemorrhage)	1 (6%)
Sporting activity	
Organized sport	8 (53)
Regular sport	2 (13)
Sport	
Soccer	3 (20)
Swimming	3 (20)
Gym	1 (7)
Touch/Oztag	1 (7)
League/union	3 (20)
Basketball	1 (7)
Cycling	1 (7)
Running/jogging	2 (13)

Values are given as n, mean \pm SD, or n (%).

were witnessed, they present an opportunity to enhance outcomes via community and club-based cardiopulmonary resuscitation (CPR) training and increased availability of defibrillators at venues.

Sports-related deaths evoke significant emotional and media responses and can lead to concerns over the benefit of physical activity. Event organizers face medicolegal issues regarding the need for preparticipation screening, even though sudden death during sport is rare. Among competitive young athletes, the incidence rate is reported to be 6–9 per million competitors per year.³ The general population has a higher incidence,⁶ as does the middle-age population (35–65 years), at 21.7 deaths per million per year.¹⁶ Our data support the infrequent nature of these events, with an adjusted annual incidence rate of 0.50–0.98 for sudden cardiac deaths.

Importantly, 94% of cardiac deaths occurred during or immediately after sporting activity; 60% occurred during an organized sporting activity. This finding presents an important opportunity to improve outcomes via provision of CPR training and defibrillators at sporting venues. Outcomes after cardiac arrest during sports are reported to be significantly better than nonsports cardiac arrests, partially explained by higher rates of witnessed events, bystander CPR, and shockable rhythms.⁷ Access to defibrillators in public access places and sporting venues has shown promising results.¹⁷ Training of team officials in bystander CPR and rapid resuscitation protocols should be encouraged by all sporting associations.

A variety of sports have experienced sudden deaths, most of which had moderate to high dynamic and low static components according to Bethesda criteria.¹⁸ This is likely related to the higher participation rates in those sports rather than

characteristics intrinsic to the sport itself. Of the cardiac deaths, no single sport was overrepresented. Soccer, running, swimming, and gym activities contributed the majority of deaths, differing from previous studies in which soccer¹⁹ and cycling⁷ predominated. This effect is likely driven by geographical variation in the amount and types of sports participated in.

The majority of deaths were in males, a finding consistent with recent studies¹⁶ in which the rates of sudden cardiac death in males were 2–25 times greater than in females.²⁰ Although some of this discrepancy is likely due to men having higher participation rates, the selection of sports used for the study; sport duration and intensity; and potential intrinsic gender differences in triggers and vulnerability warrant further investigation.

Cardiac causes, particularly coronary artery disease, was the most common cause of death, consistent with reports that included older cohorts (age >35 years).^{21,22} This is in contrast to younger cohorts, for which arrhythmias and inherited cardiomyopathies were more prevalent.^{2,23,24} Structural abnormalities were common in our cardiac deaths, with only 4 % having completely structurally normal hearts, a finding consistent with previous age-matched studies.^{25,26} In our study, presumed arrhythmic deaths (n = 26) are likely to be underestimated in favor of reporting defined structural abnormalities. Of note, 38% of our cardiac death patients had cardiac weight >500 g, 84% had LVH, and 82% had evidence of myocardial fibrosis. Cardiac weight and ventricular wall thickness increase with age, BMI,²⁷ and training effect,²⁸ which may contribute to these findings. Importantly, both LVH and fibrosis can independently contribute to arrhythmogenesis, unmask arrhythmogenic syndromes,²⁹ and increase the risk of sudden cardiac death.³⁰ In a significantly younger cohort, Finocchiaro et al³¹ reported 20% of athletic death patients had cardiac weight >500 g; 42% of these deaths were classified as idiopathic LVH ± fibrosis, and only 6% as HCM. It is likely that a proportion of deaths in our cohort were precipitated by LVH and/or fibrosis rather than other concomitant pathologies. With clinical surveillance in relatives and/or genetic testing, pathologies may be revealed over time that were not captured at the time of initial autopsy. The presence of LVH ± fibrosis is increasingly recognized in sudden cardiac death, and its presence as an innocent bystander or arrhythmic precipitant is unclear and is the subject of further research by our and other groups.²⁹

The combination of a high prevalence of coronary artery disease-related deaths, cardiovascular risk factors, and the relatively high frequency of structural abnormalities raises questions regarding the value preparticipation screening. Current preparticipation screening guidelines for sportspeople focus on young athletes^{32,33} and are directed at detection of inherited cardiomyopathies and channelopathies, which are less applicable to older cohorts.

Sudden cardiac death from coronary disease increases with the intensity of physical exertion and age.¹⁶ The risk is also related to the baseline level of habitual activity, with those having a higher level of baseline exercise (ie, the

more fit regular athlete) having substantially lower relative risk than those who usually are sedentary (the occasional athlete).³⁴ Therefore, older sports participants (>40 years) who are exercise naïve or have a low level of baseline activity and elevated cardiovascular risk profile may represent a group that would be most appropriate for further research on the benefit of preparticipation screening. Currently, the evidence for benefit of existing preparticipation screening programs based on questionnaire and standard ECGs is limited.^{26,35} The use of echocardiography may be appropriate in select populations but carries a significant increase in cost and the possibility of false-positive results.

Study limitations

Our study is retrospective in nature, and police reports and interview data were not standardized, so premorbid details were limited. Past medical history and risk factors relating to the decedent were variably reported, which may lead to underreporting of risk factors. Because we did not review every case file, it is possible that there were cases of sporting deaths not identified by the search strategy; however, the search strategy is designed to be sensitive based on the best available data.

We recognize that arrhythmias are the “final pathway” leading to death in some patients with structural and coronary artery heart disease, thus making the precise cause of death difficult to define. This is an inherent limitation of all retrospective postmortem analyses when there are coexisting pathologies. We believe it is important to identify the underlying cardiac abnormalities that precipitate or predispose such events, which also have the potential to be identified by screening mechanisms. We did not prospectively define idiopathic LVH ± fibrosis at autopsy; therefore, we were unable to classify deaths into this group.

Conclusion

Sport-related cardiac deaths are rare. Deaths were largely due to coronary artery disease in adults and cardiomyopathy or arrhythmia in younger individuals. Most deaths were witnessed, so they present an important opportunity to enhance outcomes via encouragement of community and club-based CPR training and increased availability of automated external defibrillators at venues.

Dedication

The work presented in this manuscript is in tribute to Mr Ashokkumar Ariyaratnam, who sadly died while competing in a soccer match. Our sincerest condolences to his family and friends.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrthm.2018.04.019>.

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