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ORIGINAL ARTICLE

Seasonal Variation in Sudden Cardiac Death: Insights from a Large United Kingdom Registry

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ABSTRACT

BACKGROUND Sudden cardiac death (SCD) is relatively common and may occur in apparently healthy individuals. The role of seasonal variation as a risk factor for SCD is poorly understood. The aim of this study was to investigate whether SCD exhibits a predilection for specific seasons.

METHODS We reviewed a database of 4751 cases of SCD (mean age 38 ± 17 years) referred to our Center for Cardiac Pathology at St George's University of London between 2000 and 2018. Clinical information was obtained from referring coroners who were asked to complete a detailed questionnaire. All cases underwent macroscopic and histological evaluation of the heart, by expert cardiac pathologists.

RESULTS SCD was more common during winter (26%) and rarer during summer (24%), p = 0.161. Significant seasonal variation was not observed among cases of sudden arrhythmic death syndrome (SADS, 2910 cases) in which the heart is structurally normal. In contrast, a significant difference in seasonal distribution among decedents exhibiting cardiac structural abnormalities at the post-mortem examination (n = 1841) was observed. In this subgroup, SCDs occurred more frequently during winter (27 %) compared to summer (22%) (p = 0.007). In cases diagnosed with a myocardial disease (n = 1399), SCD was most common during the winter (27%) and least common during the summer (22%) (p = 0.027).

CONCLUSIONS While SADS occurs throughout the year with no seasonal variation, SCD due to structural heart disease appears to be more common during the winter. Bio-meteorological factors may be potential triggers of SCD in individuals with an underlying structural cardiac abnormality. (Hellenic Journal of Cardiology 2025;83:3-9) © 2024 Hellenic Society of Cardiology. Publishing services by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

INTRODUCTION

Sudden cardiac death (SCD) is a tragic event that occasionally affects apparently healthy individuals. While cardiomyopathies and primary arrhythmic syndromes are considered the most common causes of SCD in young individuals, coronary artery disease (CAD) predominates in older (> 35 years) individuals.[1,2]

The elucidation of the causes of SCD relies heavily on the post-mortem examination. This should be performed utilizing a systematic methodology, which

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includes macroscopic evaluation and histological analysis of the heart.

It is poorly understood whether seasonal variation and concomitant climatological fluctuation may be environmental triggers of SCD.[3] While a correlation between cold weather and myocardial infarction has been shown in previous studies, a possible association between seasonal variation and SCD has not been explored yet.[4,5] Limited data on arrhythmogenic cardiomyopathy (AC) show an increased incidence of ventricular tachyarrhythmias and SCD during the summer, and particularly at higher temperatures.[6] There is no evidence to support any seasonal effect of SCD in individuals with primary arrhythmic syndromes.[7-9]

The acknowledgment of a possible seasonal variation of SCD may be useful for formulating preventive lifestyle recommendations. Counseling on the safety of exposure to various climatological environments may be crucial in individuals with specific cardiac conditions.

The aim of this study was to investigate seasonal variation in SCD, in cases where the post-mortem diagnosis was ascertained by expert cardiac pathologists following a standardized protocol.

METHODS

SETTING. The Cardiac Risk in the Young (CRY) Center for Cardiac Pathology is established at St. George's University of London. The center receives over 400 whole hearts of cases of SCD across the United Kingdom each year. General pathologists are likely to refer when the clinical history is suggestive of a cardiac cause that could be inherited, especially when the death affects a young or athletic individual or when the cause of death is uncertain after the initial autopsy.

STUDY POPULATION. We reviewed a database of 4751 cases of SCD which were referred to the CRY center for cardiac pathology between January 2000 and December 2018, alongside their available demographic characteristics, clinical information, and date of the events. SCD was defined as death occurring within 12 hours of apparent well-being.

AUTOPSY EXAMINATION. All SCD cases underwent a full autopsy evaluation by the local pathologist who excluded extra-cardiac and toxicological causes. Comprehensive macroscopic examination of the whole heart and histological analysis were performed by expert cardiac pathologists (MNS, JW) in accordance with the guidelines on "Autopsy practice for sudden death with likely cardiac pathology" of the Royal College of Pathologists and the Association for European Cardiovascular Pathology.[10] All cardiac structures were systematically examined. The heart weight was recorded in grams and ventricular wall thickness excluding the papillary muscles and fat and internal cavity dimensions including the trabeculae were measured at a mid-ventricular level. A minimum of 10 blocks of tissue were taken for histological analysis. Sections of myocardium were fixed in formalin, embedded in paraffin, and stained with hematoxylin and eosin. If required a picrosirius red was used to highlight collagen.

The criteria for defining specific cardiac pathologies are summarized in **Table 1**. Sudden arrhythmic death syndrome (SADS) was a diagnosis of exclusion, defined as a structurally normal heart with no evident abnormality on macroscopic and histological evaluation, and a negative toxicology screen.[10]

CLINICAL INFORMATION. The referring coroner and pathologist were asked to complete a questionnaire enquiring about the demographics of the deceased, past medical history, family history, cardiac symptoms, the nature and level of physical activity, and exact circumstances of death. The data were derived from several sources including interviews with the family of the deceased, potential witnesses of the SCD, and reports from the deceased's family physician. Circumstances of death were subdivided broadly into death related to exercise, at rest, or during sleep. Data was collected prospectively and stored on our electronic database.

STATISTICAL ANALYSIS. The chi-squared test was used to examine whether there was an even seasonal distribution of SCD events (25% in each season). To assess seasonal variations, we considered all cases from March to May (spring), June to August (summer), September to November (autumn), and December to February (winter). Moreover, the monthly occurrence of SCD events was examined while adjusting for the different number of days in each month (28 to 31 days) by dividing the number of cases by the number of days in the related month and multiplying by 30. The average number of events per month has been used. The statistical test implemented is the Chi-Square test of homogeneity to investigate whether there is an equal distribution of the number of events across the 4 seasons. Finally, Poisson regression was used to quantify the effect of season on the number of SCD events via the Incident Rate Ratio (IRR). IRR quantifies the risk of the incident of a season relative to another season. The Poisson models accounted for the time variable and the number of days in the month. All models were compared to the NULL model (i.e., no effect of the

TABLE 1 Pathological Criteria for Defining Cardiac Pathology

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

season or month on the monthly or seasonal number of incidents) via the ANOVA procedure to assess whether the inclusion of a seasonal effect significantly improves the model's goodness of fit. The analysis was performed in R v.3.5.1 [R Core Team (2018). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL https://www.R-project.org/].

RESULTS

The database included 4751 deceased (65% males) with a recorded date of death between the 1st of January 2000 and the 31st of December 2018.

Most decedents were white (n = 3855, 81%), 171 (4%) were black and 240 (5%) were of other ethnic groups (predominantly Chinese, Indian and Pakistani). Table 2 shows the main demographic characteristics of the sample and the causes of SCD.

CAUSES OF DEATH. A structurally normal heart suggestive of SADS was found in 2910 (61%) of cases. The remaining 1841 (39%) cases were diagnosed with cardiomyopathies (n = 1399, 29%), CAD (n = 128, 2%), congenital heart disease (n = 118, 2%), valvular heart disease (n = 99, 2%), aortic dissection (n = 81, 2%) and cardiac tumor (n = 16, 0.5%). In the subgroup of individuals diagnosed with CAD, congenital coronary artery anomalies accounted for 72 cases (the remaining 56 cases were found to have obstructive atherosclerotic disease). A comparison between male and female decedents showed a similar distribution of underlying etiologies.

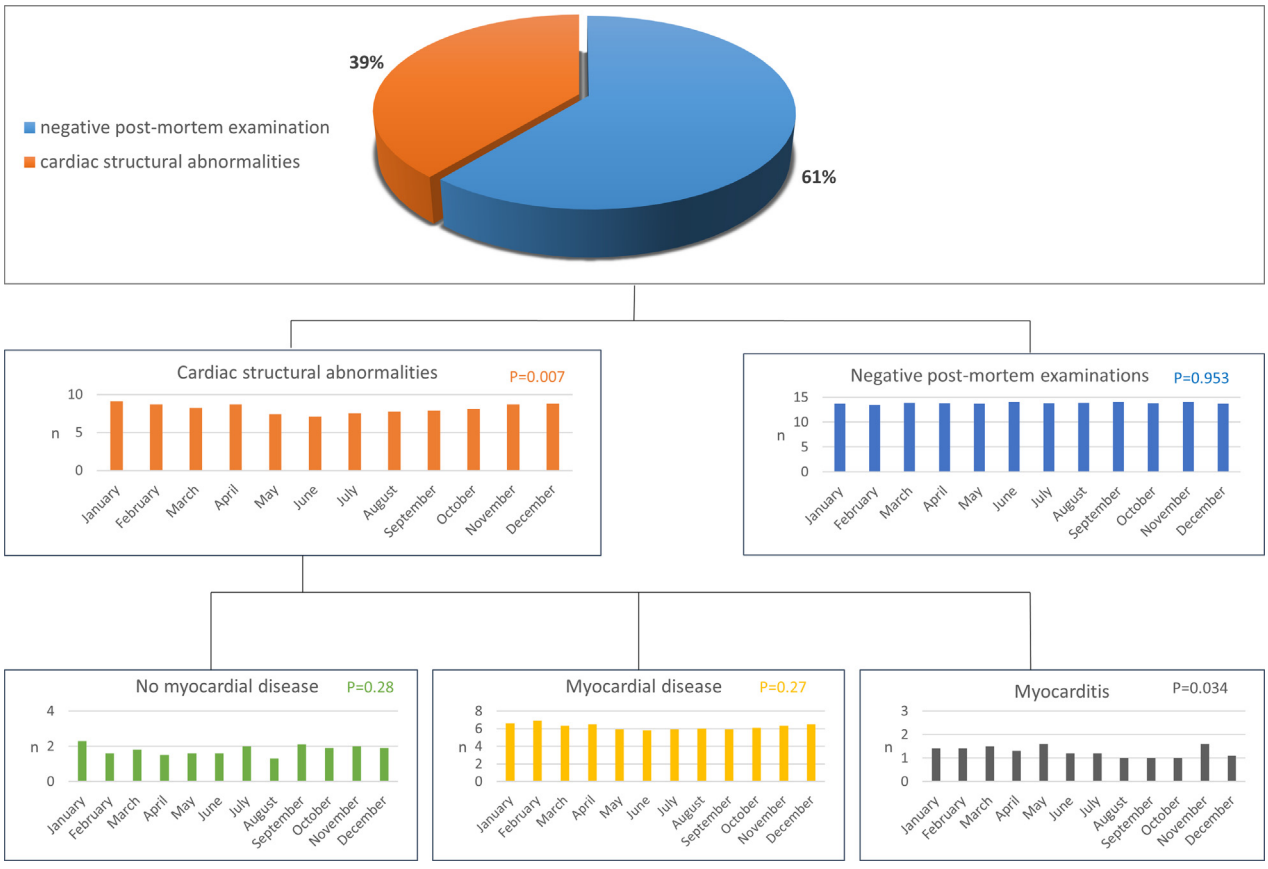
SEASONAL VARIATION. Sudden cardiac events occurred more commonly during the winter (26%) and less frequently during the summer (24%), however, this difference in the distribution was not statistically significant (p = 0.161). A similar

TABLE 2 Seasonal distribution of SCD

	N	WINTER	SPRING	SUMMER	AUTUMN	p-value
OVERALL DEATHS	4751	1244 (26)	1174 (25)	1136 (24)	1197 (25)	0.161
Autopsy Negative	2910	738 (25)	719 (25)	732 (25)	721 (25)	0.953
Autopsy Positive	1841	506 (27)	455 (25)	404 (22)	476 (26)	**0.007** *
Coronary artery disease	128	34 (27)	23 (18)	27 (21)	44 (34)	0.05
Cardiomyopathies	1399	381 (27)	360 (26)	304 (22)	354 (25)	0.03
Valvular heart disease	99	29 (29.3)	23 (23)	19 (19)	28 (28)	0.45
Aortic dissection	81	24 (30)	17 (21)	21 (26)	19 (24)	0.72
Congenital heart disease	118	36 (31)	27 (23)	29 (25)	26 (22)	0.56
Cardiac Tumor	16	4 (25)	4 (25)	4 (25)	4 (25)	0.99
Age						
Under 18	485	140 (29)	116 (24)	121 (25)	108 (22)	0.21
18-35	2073	522 (25)	501 (24)	506 (24)	544 (26)	0.54
36-45	1065	271 (25)	266 (25)	257 (23)	271 (26)	0.92
46-55	610	155 (25)	161 (26)	137 (22.5)	157 (26)	0.53
Over 55	518	156 (30)	130 (25)	115 (22)	117 (23)	0.04
Gender						
Female	1659	436 (26)	418 (25)	375 (23)	430 (26)	0.79
Male	3092	809 (26)	756 (24)	760 (25)	767 (25)	0.87

*p value: Chi-square test of homogeneity for the frequency of SCDs across the seasons, demonstrating a predilection of events in winter in decedents with a cardiac structural abnormality (p = 0.007).

FIGURE 1 Seasonal variation of SCDs according to the underlying autopsy diagnosis



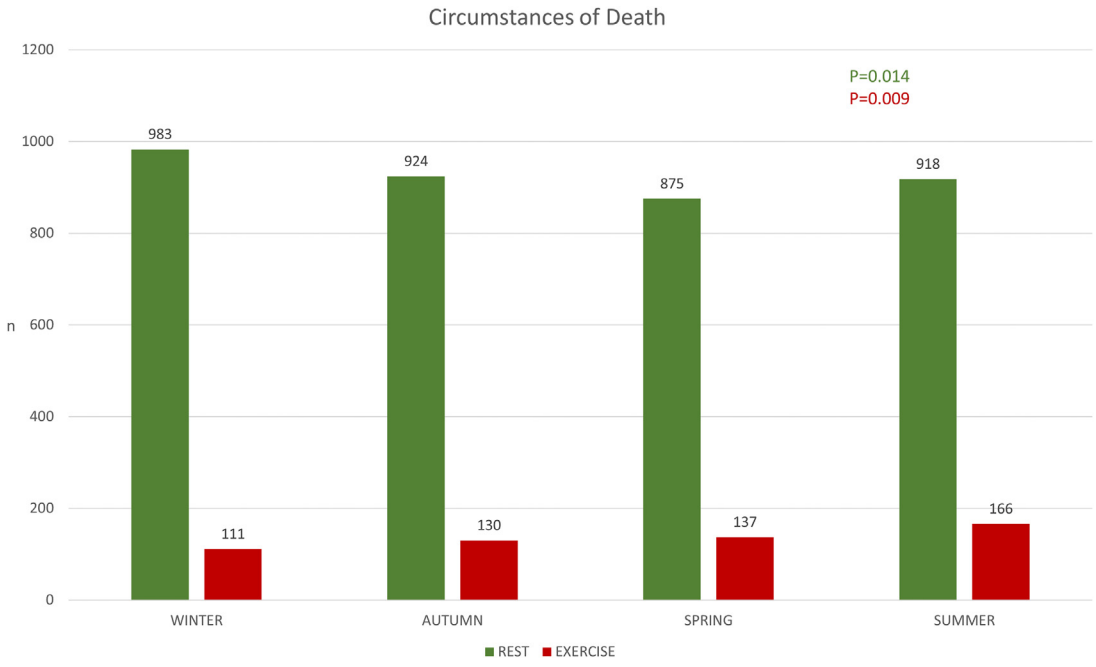
n: average number of events per year distributed in months. p value: Chi-square test of homogeneity for the frequency of SCDs across the seasons, which investigates whether the frequency of events is statistically similar in every season. There is an unequal frequency of SCDs with cardiac structural abnormalities (p = 0.007) across the four seasons, indicating a predilection for winter.

non-seasonal distribution was demonstrated when male and female decedents were analyzed separately. **Figure 1** and **Table 2** demonstrate the distribution of analyzed events during the year.

No differences in terms of seasonal distribution were found in decedents with a structurally normal heart (p = 0.953). In contrast, a seasonal variability with events occurring more frequently during the winter (27%) and less frequently during the summer (22%) (p = 0.007) was observed in cases with cardiac structural abnormalities at the post-mortem examination (n = 1841). In individuals with structural cardiac disease, SCDs were 18% less likely to occur during the summer, compared to winter (IRR = 0.819, p = 0.003) and the seasonal effect was statistically significant (p = 0.005). In cases where the post-mortem examination revealed cardiomyopathy (n = 1399), SCD was most common during the winter (27%) and least common during the summer (22%) (p = 0.027). In cases where the cause of death was coronary artery disease, SCDs were most frequent during the autumn (34%) and the least during the summer (21%) (p = 0.047). Cases of myocarditis (n = 102) exhibited a seasonal preference, being more frequent in spring (36%) and least frequent in autumn (18%) (p = 0.0034).

CIRCUMSTANCES OF DEATH. SCDs occurred at rest in 3700 cases (78%) and during exercise in 544 (12%). A seasonal effect was noted in fatal events occurring at rest, with a predominance in winter (n = 983, 27%) (P = 0.014). A statistically significant seasonal predilection was also noted in SCDs related to exercise with a predominance of summer (n = 166, 31%) (p = 0.09) (**Figure 2**, **Table 3**).

FIGURE 2 **Seasonal distribution of SCDs according to circumstances of death**



p value: Chi-square test of homogeneity for the frequency of SCDs at rest across the seasons demonstrates a predominance of events in winter (p = 0.014), while SCDs during exercise demonstrate a predominance of events in summer (p = 0.009).

DISCUSSION

Our study investigates the seasonal variation of sudden cardiac death in a large cohort where the postmortem examination was performed by expert cardiac pathologists. While in individuals who had a structurally normal heart at autopsy, suggestive of SADS, fatal events were recorded to be approximately equal among seasons (~25%), significant seasonal variation was observed among decedents with structural cardiac abnormalities where events were more common during the winter (27%) and the least common during the summer (22%).

SEASONAL VARIATION IN SUDDEN CARDIAC DEATH. Previous studies focused on the epidemiology of myocardial infarction showed some degree of seasonal variation.[11] Acute events in the context of atherosclerotic coronary pathology are documented to have a predilection for cold weather conditions.[12,13] A higher incidence of mortality attributed to CAD, including SCD, is generally demonstrated in a low-temperature environment, according to most published registries.[14] In a slightly dissimilar pattern, our study showed that autumn was the season when most of SCDs due to CAD occurred.

According to our findings, SCDs in individuals with cardiac structural abnormalities were less likely to occur during the summer. Previous studies have also identified a predilection of winter and low temperature in SCD among individuals with known heart disease.[15] This is also in agreement with a study that investigated the annual periodic trend of sustained ventricular arrhythmias derived from intracardiac electrograms of patients with an implanted defibrillator.[16]

Seasonal distribution of SCD may be explained by how extrinsic meteorological factors interfere with various biological parameters, and certain behavioral elements, which may contribute to the development of fatal arrhythmias in individuals with a predisposing electrical or structural substrate. The meteorological conditions in

TABLE 3 **Sample characteristics of circumstances of death (N = 4751)**

Circumstances of Death	N	Winter	Spring	Autumn	Summer	p value
Rest	3700 (78)	983 (27)	924 (24)	875 (23)	918 (26)	0.014*
Exercise	544 (12)	111 (20)	130 (24)	137 (25)	166 (31)	**0.009**^

*p value: Chi-square test of homogeneity for the frequency of SCDs at rest across the seasons demonstrating a predominance of events in winter (p = 0.014). ^p value: Chi-square test of homogeneity for the frequency of SCDs during exercise across the seasons, demonstrating a predominance of events in summer (p = 0.009).

winter may result in an unfavorable hormonal equilibrium with negative repercussions on endothelial function and coagulation response.[17,18] Both fibrinogen and factor VII clotting activity (FVIIc) plasma values appear greater in winter-time, something that may potentially increase the susceptibility for thrombotic cardiovascular events. The activation of the sympathetic nervous system by cold weather has a heavy catecholamine-mediated impact on blood pressure and heart rate. Vitamin D levels are reduced due to less sunlight exposure.[19-21] These variables may result in a state of increased oxidative stress and endothelial dysfunction. Several behavioral components, such as nutritional habits and physical inactivity, tend to vary significantly during different seasons of the year and weather conditions, and this may have an impact on the arrhythmic risk.[22] Viral and bacterial infections are more common during the winter.[23,24] Our study showed also a seasonal predilection of fatal events at rest during the winter. In contrast, SCD during exercise was more common during the summer. This may be explained simply by the tendency to engage in outdoor sports activities during the summer, rather than during the winter. Perhaps future studies looking closely at the types of exercise along with the specific environment of athletic activities (indoor or outdoor) can shed light on this finding.

CLINICAL IMPLICATIONS. A full understanding of the seasonal distribution of SCDs may have clinical implications on lifestyle recommendations and preventive strategies. Although in most of our cases, SCD was the first manifestation of a silent cardiac disease, we speculate that individuals with an underlying structural substrate may be at risk especially when exposed to a cold environment. With genetic testing gradually evolving and becoming an integral component in the investigation and prevention of SCD, any such evidence regarding predisposing seasonal or climatological conditions may prove useful for additional research.

LIMITATIONS. General pathologists are likely to refer to the CRY Center of Cardiac Pathology when the clinical history is suggestive of an inherited cardiac condition, especially when the death affects a young or athletic individual or when the cause of death is uncertain after the initial autopsy. These facts introduce a potential referral bias: it is probable that pathologies such as coronary artery atherosclerosis, aortic dissection, and HCM may be under-represented in this cohort. Instead, normal heart suggestive of SADS may be over-represented in our cohort.

CONCLUSION

Sudden cardiac death in individuals with an underlying structural cardiac condition is more common during the winter. In contrast, no seasonal variation is observed in cases of SADS. Meteorological factors may act as potential triggers of SCD in individuals with an underlying structural or electrical substrate. The complex interaction of seasonal and climatological circumstances with the propensity for potentially fatal arrhythmias should be further investigated with the aim of preventing these tragic events.

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DECLARATION OF COMPETING INTEREST

None

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