Seasonal variations in out of hospital cardiopulmonary arrest

J P Pell, J Sirel, A K Marsden, S M Cobbe

Abstract

***Objective*—To determine whether there are seasonal variations in survival following out of hospital cardiopulmonary arrest.**

***Design*—Prospective cohort study using the Heartstart (Scotland) database.**

***Setting*—All of Scotland.**

***Patients*—10 890 people who suffered out of hospital cardiopulmonary arrest in the summer or winter between December 1988 and August 1997 inclusive.**

***Intervention*—Univariate comparisons of 5406 arrests occurring in summer with 5484 in winter, in terms of patient characteristics, management, and survival using χ^2 and Mann-Whitney U tests. Multivariate analysis of the association between season and survival following adjustment for case mix.**

***Main outcomes measures*—Survival to discharge from hospital, survival pre-admission, in-hospital survival.**

***Results*—Only 6% of people who arrested in winter survived to discharge, compared to 8% of those who arrested in summer (odds ratio 0.77, $p < 0.001$). People who arrested in winter had a poorer risk profile in that they were older, more likely to arrest at home, less likely to have a witness, and less likely to receive defibrillation. However, after adjustment for case mix, people who arrested in winter were still 19% less likely to survive compared to those who arrested in summer. Deaths pre-admission were significantly higher in winter (odds ratio 1.18, $p < 0.05$) but in-hospital deaths were not.**

***Conclusions*—People who suffer cardiopulmonary arrest in winter have a significantly lower likelihood of surviving. This is, in part, caused by the higher frequency of a number of recognised risk factors. However, their prognosis remains poorer even after adjustment for these factors.**

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Several studies have shown a winter peak in total coronary heart disease (CHD) mortality or fatal myocardial infarctions.[1–6] This may be because of seasonal variations in incidence,[7 8] or survival.[2 4] In contrast, studies on sudden cardiac death have shown no significant seasonal trend.[9 10]

Since 1988, data have been collected on all cardiopulmonary arrests attended by the Scottish ambulance service as part of the Heartstart (Scotland) initiative. Our study compares the survival of patients who arrested in winter with those who arrested in summer.

Methods

As part of the Heartstart (Scotland) initiative, front line ambulance crew members have collected data on every out of hospital cardiopulmonary arrest in Scotland which they have attended since October 1988. The information obtained includes demographic characteristics, arrest location and date, time delays, use of defibrillation and cardiopulmonary resuscitation (CPR), transfer to hospital, and admission. Additional information on the outcome of those admitted to hospital is collected using a second form which is completed by the medical records staff of the relevant hospital. The forms are collated and analysed by the department of medical cardiology at the Glasgow Royal Infirmary.

For the purposes of this study, summer was defined as June to August inclusive and winter as December to February inclusive. The study included all arrests which occurred in the summer or winter between December 1988 and August 1997 inclusive. The χ^2 and Mann-Whitney U tests were used to compare arrests which occurred in summer with those which occurred in winter in terms of patient and management characteristics, and crude rates of survival. Multivariate logistic regression analysis was used to determine whether the association between season and survival was significant after adjustment for differences in case mix.

Results

Over the period studied, forms were returned on 11 194 cardiopulmonary arrests which occurred in either summer or winter. Outcome data were missing for 304 (3%) of these patients. Therefore, the analyses were undertaken on the remaining 10 890 arrests. Of these, 5484 (50%) occurred in winter and 5406 (50%) in summer.

Overall, patients who suffered cardiopulmonary arrest in winter had a poorer crude survival rate with only 6% surviving to discharge from hospital, compared to 8% of those who arrested in summer (odds ratio 0.77, 95% confidence interval (CI) 0.67 to 0.90, $p < 0.001$) (table 1). Crude case fatality rates were significantly higher in winter for men $\geq$ 65 years old ($p < 0.01$), women $\geq$ 65 years old ($p < 0.001$), and women < 65 years old ($p < 0.05$). The crude case fatality rate for men < 65 years old was the same in summer as in winter.

Table 1 Comparison of cardiopulmonary arrests occurring in summer and winter in terms of patient characteristics, management, and overall survival

	Summer (n = 5406)	*Winter (n = 5484)*	*Significance**
Age			
Median (IQR)	65 (55–73)	67 (57–74)	p < 0.01
Missing	77	92	
Sex			
Male	3799 (70%)	3817 (70%)	NS
Female	1596 (30%)	1660 (30%)	
Missing	11	7	
Arrest location			
Home	2800 (58%)	2956 (61%)	p < 0.01
Other location	2025 (42%)	1906 (39%)	
Missing	581	622	
Defibrillation			
Yes	3079 (58%)	2949 (54%)	p < 0.001
No	2258 (42%)	2471 (46%)	
Missing	69	64	
CPR ⩽ 4 minutes			
Yes	1718 (53%)	1667 (51%)	NS
No	1527 (47%)	1593 (49%)	
No CPR/missing	2161	2224	
Presence of witness			
No	1154 (24%)	1248 (26%)	p < 0.01
Yes	3578 (76%)	3505 (74%)	
Missing	674	731	
Discharged alive			
Yes	431 (8%)	345 (6%)	p < 0.001
No	4975 (92%)	5139 (94%)	

*On χ^2 or Mann-Whitney U test.
IQR, interquartile range.

Table 2 Multivariate logistic regression analysis of factors associated with survival to discharge from hospital

	Odds ratio	*95% CI*	*Significance*
Age	1.00	(0.99 to 1.00)	NS
Sex			
Male*	1.00	–	
Female	1.15	(0.90 to 1.47)	NS
Location			
Not home*	1.00	–	
Home	0.51	(0.41 to 0.64)	< 0.001
Defibrillation			
No*	1.00	–	
Yes	8.52	(5.68 to 12.79)	< 0.001
CPR ⩽ 4 minutes			
No*	1.00	–	
Yes	4.35	(3.31 to 5.72)	< 0.001
Presence of witness			
No*	1.00	–	
Yes	3.50	(1.10 to 11.14)	< 0.05
Season			
Summer*	1.00	–	
Winter	0.81	(0.65 to 1.00)	< 0.05

*Reference categories.

Data were missing on the presence of a witness and arrest location for 13% and 11% of patients, respectively (table 1). Based on those patients in whom data were available, patients who arrested in winter possessed a worse risk profile for survival. They were significantly older (p < 0.01), were more likely to arrest at home (p < 0.01), were less likely to have their arrest witnessed by either a bystander or ambulance crew member (p < 0.01), and were less likely to receive defibrillation (p < 0.001) (table 1).

On multiple logistic regression analysis, cardiac arrest in winter was still associated with a significantly lower likelihood of survival to discharge after adjustment for case mix (table 2). Patients who arrested in winter were 19% less likely to survive to discharge from hospital.

Multiple logistic regression analysis showed that patients who arrested in winter were more likely to die before admission to hospital (odds ratio 1.18, 95% CI 1.01 to 1.38, p < 0.05). However, survival to discharge in those who were admitted did not differ significantly by season. There was no clear time trend in the winter:summer ratio of case fatality rates over the period studied.

Discussion

Our study showed that the percentage of patients surviving to discharge following cardiopulmonary arrest was higher in summer than winter. Although this difference was significant, the absolute difference in percentages was small. As a result prognosis remained poor even in summer.

A winter peak in CHD mortality has been shown in both northern and southern hemisphere countries.[1–6] In England, this accounts for an additional 20 000 deaths per annum.[11]

Variations in myocardial infarction admissions and trial recruitment have been cited as evidence that this reflects a seasonal variation in CHD incidence.[1 7 8] However, seasonal variations in mortality are greater than those in admission,[2] suggesting that case fatality rates may also vary throughout the year. Enquselassie *et al* showed that CHD events were more likely to be fatal when the temperature was low.[4] These findings are supported by the results of our study. The numbers of resuscitation attempts were comparable in summer and winter, but overall survival was significantly lower in winter even after adjustment for the poorer case mix of patients.

The seasonal variations reported in CHD are likely to reflect seasonal variations in one or more risk factors. The risk factors proposed have included environmental factors, lifestyle and physiological risk factors, and respiratory infections.

Generalised cooling of the body increases blood pressure and cardiac output.[12] In some patients with angina, localised cooling of the face can cause similar effects.[13 14] In Britain, winter blood pressures exceed summer blood pressures by around 5 mm Hg.[15] Temperature changes can account for practically all of the seasonal variations observed in CHD mortality.[7]

Seasonal variations in CHD mortality differ in magnitude across Europe. Variations are least in countries with strict building regulations and plentiful natural energy resources.[16] In a study comparing six European regions, CHD mortality was independently associated with living room temperature, bedroom heating, levels of inactivity, and use of protective clothing.[17]

Subjects with CHD have lower concentrations of vitamin D metabolites.[18 19] The association between vitamin D and CHD is independent of smoking, body mass index, treatment for hypertension, vigorous leisure activity, total serum cholesterol, and a previous history of angina or myocardial infarction.[18] Higher exposure to ultraviolet radiation in summer may, therefore, protect against CHD events.

Body weight increases in winter because of both increased fat intake and less frequent physical activity.[20–22] While regular exercise is

beneficial, unaccustomed exertion, such as shovelling snow, can increase the risk of sudden cardiac death.[23] The association between high levels of physical activity and sudden death is greater in cold temperatures than warm temperatures.[24]

Cholesterol concentrations are higher in winter than summer.[20 25 26] Cholesterol and vitamin D have a common precursor.[27] The relative amounts produced may be influenced by exposure to sunlight.[26] There is a strong positive association between latitude and mean blood cholesterol, and a strong negative association between hours of sunshine and CHD mortality.[26]

Lower temperatures result in increases in fibrinogen, platelets, red cells, and plasma viscosity, and a reduction in plasma volume.[28] As a result, haematological parameters vary throughout the year.[29]

A number of microbial agents have been implicated in the pathogenesis of CHD, including *Chlamydia pneumoniae*, *Helicobacter pylori*, and cytomegalovirus.[30–33] A number of investigators have postulated that seasonal variations in CHD may be attributed, in part, to the fact that respiratory infections are more common in winter months.[34] Respiratory infections may increase the risk of developing or dying from CHD through an increase in plasma fibrinogen[35] and endotoxin inhibition of fibrinolysis.[36]

H pylori infection is associated with peptic ulcers[37 38] which, as with CHD, are characterised by winter peaks.[37–39] Moshkowitz and colleagues showed seasonal variations in *H pylori* infection which were highly correlated with the seasonal variations observed in peptic ulcers.[38] However, this finding has been refuted by other investigators.[37 39] Some investigators have suggested an association between *H pylori* and CHD,[32 33] but others have shown no significant association after adjustment for other risk factors.[40 41]

Seasonal variations are not consistent across age and sex groups. Douglas *et al* showed greater winter peaks in CHD mortality in older age groups.[2] This is likely to reflect a combination of factors including poorer autonomic control, poorer heating and insulation in the home environment, more time spent at home, less use of protective clothing, and lower levels of physical activity. In our study, winter peaks were apparent in men of 65 years or older, but not in younger men.

Seretakis *et al* showed a time trend in the seasonality of disease in the USA.[42] The winter:summer total death ratio declined by about 2% per annum between 1938 and 1970 and reached a plateau thereafter. The decline was less in the more southern states where winters were milder. The decline was attributed to improvements in indoor and vehicular heating and air conditioning. In our study, there was no obvious time trend in the winter:summer death rate ratio. This may reflect the fact that no significant changes in microclimate control have taken place in more recent decades.

In a study by Enquselassie *et al*, temperature did not influence whether CHD deaths occurred suddenly or more than one day following the onset of symptoms.[4] In contrast, Douglas *et al* demonstrated greater seasonal variations in out of hospital mortality than in-hospital mortality.[2] Our study results were similar to those of Douglas *et al*,[2] in that deaths before admission exhibited a significant increase in winter whereas deaths in hospital did not.

Clearly, emigration is not an option for most of those who are at risk of dying from cardiopulmonary arrest or any other manifestation of CHD. However, the evidence suggests that a number of simple precautions could be taken in winter. These include adequate indoor heating, wearing protective clothing, especially outdoor protection of the face, and avoiding unaccustomed strenuous exercise. Attempts should also be made to ensure that our lifestyles, in relation to diet, regular exercise, and smoking, are at least as healthy in winter as they are in summer.

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