Ventricular fibrillation in the Antarctic: an unexpected event

Sir,

Ventricular fibrillation is a common complication of acute myocardial infarction.¹ This arrhythmia is potentially reversible if direct current cardioversion is promptly administered.² We wish to highlight, with a case report, the benefit of carrying a defibrillator in the medical kit of groups of young, apparently low risk individuals visiting remote parts of the world.

A 42 year old apparently healthy male serving on a British Antarctic Survey ship in the Antarctic, developed severe central chest pain whilst supervising one of the regular life-boat drills. This came on after a brief period of vigorous activity (enthusiastic cranking of a stubborn engine). Half an hour later, on return to the mother ship, the pain worsened and he sought medical attention.

An initial limited electrocardiogram (ECG) demonstrated ST elevation in leads II and III, and ST depression in lead I. A subsequent 12 lead ECG confirmed the diagnosis of an inferior myocardial infarction.

Within the hour he had a cardiac arrest. This was secondary to ventricular tachycardia which degenerated into ventricular fibrillation. This was successfully terminated by a single 200 Joule direct current shock, and a lignocaine infusion was administered. The rest of his management was routine and uncomplicated, except that thrombolysis was not available.

Two months post-infarct he was admitted to the Cardiology Unit at Aberdeen Royal Infirmary where cardiac catheterization demonstrated an irregular right coronary artery with a severe distal stenosis, and inferior left ventricular akinesia. Due to the presence of persistent post-infarction angina he subsequently underwent angioplasty and is now symptom free.

The defibrillator was available during what was the first season of its inclusion in the medical indent. Without it, the sailor would certainly have died.

From the Framingham study, the incidence of acute myocardial infarction in men aged 35–44 years is 21 per 10,000.³ The overall mortality of acute myocardial infarction is 50%, and half of these deaths occur in the first 2 hours, mostly from ventricular fibrillation.¹

In the absence of left ventricular failure or cardiogenic shock, ventricular fibrillation related to acute myocardial infarction is nearly always correctable.¹ We therefore recommend the routine package of a defibrillator in the medical kit of those serving in remote areas.

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Fluconazole-associated acute adrenal insufficiency

Sir,

Adrenal insufficiency is a recognized complication of the acquired immunodeficiency syndrome (AIDS). We wish to report a case of acute adrenal insufficiency that occurred in a patient with AIDS due to the oral antifungal agent fluconazole.

A 34 year old Hispanic male was admitted in August 1989 with complaints of progressive weight loss, fevers and odynophagia. He had a prior history of AIDS with *Pneumocystis carinii* pneumonia and cytomegalovirus (CMV) retinitis. Physical examination revealed generalized wasting, a temperature of 39°C and oral candidiasis. His serum electrolytes were entirely normal. He was given zidovudine 100 mg by mouth every 4 h and was started on oral ketoconazole but did not demonstrate any clinical response. On 1 October 1989 he was started on amphotericin B with partial resolution of the oral candida and odynophagia. Fluconazole was approved for the patient on 17 October 1989 and he was started on 200 mg by mouth per day. Three weeks later he developed hyperkalaemia (6.1 mmol/l), hyponatraemia (134 mmol/l), and low serum bicarbonate (17 mmol/l). There was no elevation in creatinine or blood urea nitrogen levels. A synthetic ACTH stimulation test revealed a baseline cortisol level of less than 0.5 µg/dl that failed to rise 30 min post-stimulation. A diagnosis of acute adrenal insufficiency was made. The fluconazole was stopped and steroid replacement begun. The electrolyte abnormalities resolved. An abdominal computed tomographic scan failed to reveal any demonstrable gross adrenal pathology. The patient died 2 weeks later. Permission for a post-mortem examination was not obtained.

Ketoconazole is known to produce adrenal suppression.¹ The basis for adrenal suppression by the azole anti-fungal agents is by suppression of the cytochrome P-450 enzyme system in the adrenal cells.¹ In consequence, ketoconazole has been used to treat Cushing's syndrome utilizing this property.² Adrenal suppression in humans by fluconazole has not been previously reported.^{3,4} Studies in rats have however shown a suppressive effect *in vitro* of fluconazole on the adrenal P-450 system,⁵ but the concentration of fluconazole required to produce suppression of adrenal steroid production in this model was two orders of magnitude higher than the concentration of ketoconazole required to produce a similar effect.

In light of the clinical presentation in our case, we propose that the patient developed acute fluconazole-