Sudden death during marathon racing: hyperpyrexia versus myocardial ischaemia

Sir,

The report from South Africa\(^1\) asserts that an experienced marathon runner with hypertrophic cardiomyopathy died from myocardial "ischaemia", collapsing near the 20 mile mark in a race. The "naked eye" necropsy showed no cause of death. The coronary arteries were normal. They support their diagnosis of "ischaemia" with a low potassium/sodium ratio in the myocardium. However, this low ion ratio only indicates injury, probably caused by ventricular fibrillation.\(^2\) The specific term "ischaemia" should be limited to those cases with significant coronary artery disease.

We maintain worldwide, forensic surveillance over marathon runners,\(^3\) and have encountered similar cases. Fatal and nonfatal arrhythmias have occurred during racing in association with exertion-induced heat illness, dehydration, pyrexial illness, and the consumption of drugs. It is unfortunate that Noakes \textit{et al.} were limited to the examination of a single, autolysed organ, the heart. The Figure showed complete loss of myofibril nuclear detail, suggesting that death occurred during hyperpyrexia, and refrigeration was not available during the 48-hour delay before necropsy. Timely microscopical studies could have ruled out viral infection. Chemical tests for alcohol, caffeine, and other drugs should have been performed in view of the negative "naked eye" necropsy. Knowledge of racing conditions helps evaluate the possibility of heat stress and dehydration. These basic forensic steps would have added greatly to the value of their report.

The use of the term "ischaemia" instead of "heat injury" in these cases suggests an increased hazard of road racing. Appropriate safety precautions cannot be taken until the true dangers are known. In addition, the treatment for collapse resulting from heat stroke is different from the treatment for ischaemic heart disease. To delay cooling measures can be fatal.

Other reports from South Africa have confused the effects of racing hyperpyrexia with "ischaemic heart disease".\(^4\) Our own surveillance has uncovered similar irregularities among the reports of Noakes \textit{et al.}\(^5\) When a 19-year-old died during a marathon, Opie\(^6\) reported that coronary "atheroma" was the cause of death; however, the necropsy showed "normal" coronary arteries.\(^7\) When a 35-year-old marathon runner died after taking long training runs, Opie\(^8\) reported that the necropsy showed "ischaemic heart disease with a number of small infarcts". However, there was no necropsy,\(^7\) and the electrocardiogram was consistent with heat stroke.\(^8\) We believe that this confusion delays any efforts at improving racing conditions.

Noakes \textit{et al.} chastise the stricken runner because he "chose to ignore his symptoms and to conceal these from his doctors". They caution that "symptoms suggestive of myocardial ischaemia must be investigated". Their previous report suggests otherwise. When six runners developed exertional symptoms,\(^9\) all four who refused to remain in hospital survived. The single death occurred in hospital where no treatment for heat stroke was given. This runner had been taking long training runs up to 40 miles during the hot "surfing season" when symptoms appeared. His electrocardiogram suggested heat illness,\(^8\) but he was treated for "ischaemic" heart disease. We suggest that inappropriate medical care will not improve the survival of these runners, and we excuse any South African marathon runner who elects to avoid doctors under these conditions.

We disagree with the authors' two conclusions: that marathon running "precipitated ischaemia" and "contributed to the cardiomyopathy". There is nothing in their report to support their views. By emphasising the cardiomyopathy they have missed the very real hazard of marathon running itself, namely exertion-induced heat illness.

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References

\(^1\)Noakes TD, Rose AG, Opie LH. Hypertrophic cardiomyopathy associated with sudden death during marathon racing. \textit{Br Heart J} 1979; 41: 624–7.
This letter was shown to Drs Noakes and Opie who reply as follows:

Dr Bassler has done much to promote the cause of marathon running, and we thank him for the opportunity to respond to the views in his letter.

(1) Our case report

That our marathon runner with hypertrophic cardiomyopathy also had ischaemic chest pain seems very probable on the history. Therefore, it seems reasonable to suggest that the runner died from myocardial ischaemia. The term “myocardial ischaemia” can certainly be used in the absence of coronary artery disease, for example in patients with aortic stenosis and angina.1 In our patient the massive left ventricular hypertrophy and the anginal pain precipitated by effort and emotion indicate myocardial ischaemia, even without the tissue K+/Na+ values. There was no pathological evidence of autolysis as the body had been refrigerated shortly after death.

Exercise, especially if severe, is usually held to be contraindicated in patients with hypertrophic cardiomyopathy. It would be surprising if the severe exercise of marathon running could be held to have been beneficial for our patient.

(2) Hyperpyrexia versus ischaemic heart disease

Dr Bassler contends that South African doctors are unable to distinguish between hyperpyrexia and ischaemic heart disease and that he would “excuse any South African marathon runner who elects to avoid doctors under these conditions”. In support of this statement he supplies a series of half-truths. The facts of the matter are the following:

(i) that South African researchers were among the first to draw attention to the dangers of hyperpyrexia in marathon running2;

(ii) that one of the present authors (TDN) has written extensively in both the medical3 and lay journals4 regarding the dangers and preventable aspects of marathon and ultra-marathon running in a hot environment like that of South Africa;

(iii) that despite similar environmental conditions, hyperpyrexia and heatstroke in runners appear to be more of a problem in North America5 6 than in South Africa;

(iv) that the 35-year-old marathon runner who was admitted to hospital and subsequently died, did not die from heatstroke. Our preliminary erroneous letter that there was a necropsy has been corrected on numerous occasions.7–9 Heatstroke could not explain the abnormal electrocardiogram of this patient who had not run at all on the day of his death, but had been in the sea, surfing—a combination of circumstances totally excluding heat injury. The patient suffered from repetitive exertional chest pain; during one 4½ mile time trial his wife recalls that the patient was forced to stop running four to five times.7 8 Such anginal pain is a feature of ischaemic heart disease, not of heatstroke. By repeatedly and incorrectly presenting this case as an example of heatstroke on the basis of an erroneous interpretation of the electrocardiogram10 Dr Bassler destroys the force of his arguments. It is difficult to see how treating a fully lucid man (a finding which by itself excludes a diagnosis of heatstroke), who has just been surfing, for heatstroke, and ignoring his severe praecordial chest pain would be medically justifiable;

(v) that though the delayed cooling of a patient with heatstroke may indeed be fatal, the correct treatment for the apnoeic patient who dies suddenly during marathon running, as did our patient with cardiomyopathy, is cardiopulmonary resuscitation and not cooling; and

(vi) that there is as yet no published evidence showing that electrocardiographic abnormalities in marathon runners can be explained on the basis of hyperpyrexia nor that heatstroke can present as instantaneous exercise-related death.9 11

Dr Bassler’s contention that four of our runners with myocardial infarction refused to remain in hospital is an understandable misinterpretation of ambiguities in our 1977 paper.7 Though one patient refused admission to hospital, none of the other patients refused to stay in hospital, but were discharged normally by their physicians. In the days immediately after their discharge from hospital, however, three patients began walking and jogging again. It was this early return to an exercise training
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programme which they had been advised against by their physicians. We apologise for these ambiguities in our earlier paper.

We eagerly await Dr Bassler’s detailed publications refuting these statements and look forward to reading his other, as yet unpublished, studies showing that death in marathon racing is more likely to be a result of viral infection, alcohol, caffeine, and other drugs than to myocardial or coronary artery disease. We are grateful that he did not accuse us of failing to determine whether the runner’s diet was inadequate in yeast, yogurt, peanuts, beer, wheat germ, or vitamin C as a dietary deficiency of any one of these is also held by Dr Bassler to be a cause of sudden death in marathon runners.12 Athletes who die suddenly during marathon races are more likely to die from necropsy-proven diagnoses such as myocardial disease or coronary artery disease8 than from the other obscure reasons Dr Bassler gives.

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References