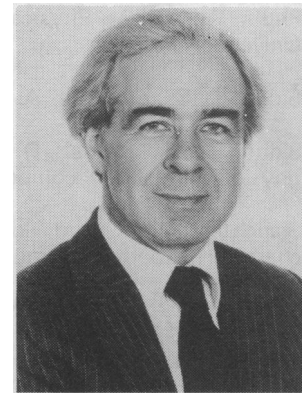




M. A. Parsons



P. B. Anderson



B. T. Williams

AN "UNAVOIDABLE" DEATH IN A PEOPLE'S MARATHON

M. A. PARSONS, MB, ChB, MRCPATH.,* P. B. ANDERSON, BM, BCh, MRCP** and B. T. WILLIAMS, MD, FFCM†

*Departments of *Pathology and †Community Medicine, University of Sheffield*

***The Chest Clinic, Northern General Hospital, Sheffield*

ABSTRACT

With the increase in jogging and distance running as a means of attaining and maintaining fitness, and in the light of the difficulty in advising certain groups of patients about their capacity for such exercise, we report on several potentially avoidable factors in the death of a 45 year old Sheffield Marathon runner in June 1983.

Key Words: Marathon death. Exercise counselling.

INTRODUCTION

Approximately 150 people's marathons are run in the UK each year, some with half-marathon options (Anonymous, 1982). Approximately one in five runners are injured during such races (Nicholl and Williams, 1983), and over half the intending runners are injured in training (Maughan and Miller, 1982). Reports of deaths are, however, rare. We report here a death in the June 1983 Sheffield marathon and estimate its avoidability.

CASE REPORT

A 45 year old white male runner, 192 cms tall and well-proportioned, collapsed at 10.30 hours, 3½ miles after the start of the race. The air temperature was 17°C and the relative humidity 57%. Ambulance and hospital

resuscitation attempts failed and he died 30 minutes later.

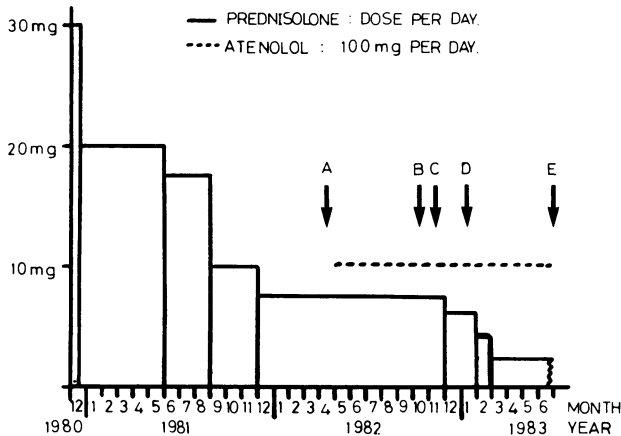
His occupation as an electrical engineer in the oil industry was physically demanding and involved climbing on shore marine installations in different climatic conditions. In October, 1980, though asymptomatic, an industrial medical examination discovered labile hypertension (BP 160/100-180/110 mg.Hg.). Chest X-rays showed bilateral fine parenchymal lung nodules, linear shadowing and hilar lymphadenopathy. Respiratory function tests were within normal ranges. ECG showed mild ischaemic changes. Despite a negative Kveim reaction sarcoidosis was diagnosed clinically and radiologically. Treatment with oral prednisolone was commenced and this continued with reducing dosage till his death (Fig. 1). Eight weeks later he could run 50 feet up ladders without stopping.

In 1981 he started gradual non-competitive running to aid fitness and as a hobby (he was, and remained, a keen yachtsman).

In April 1982 he commenced graduated training for a marathon six months later. In May 1982 his GP pres-

Address for correspondence:

Dr. M. A. Parsons,
Department of Pathology,
Medical School,
Beech Hill Road,
Sheffield S10 2RX



Medication and marathon runs.

Key

- A Commenced training for endurance running
- B Ran 26 miles
- C Ran 18½ miles
- D Ran 14½ miles
- E Collapsed after 3½ miles

cribed the beta-blocker atenolol for hypertension (see Figure), and discussed with him the implications for his clinical conditions and medication of the vigorous lifestyle, which he would not compromise. He was, however, always judicious about not exceeding his perceived capacity.

He completed a marathon (26 miles) in October 1982, and 18½ miles and 14½ miles respectively of marathons in November 1982 and January 1983, achievements which were made known only later to his clinical specialist. At clinic review two weeks before death nothing untoward was found. He completed a 10 mile hill run in the week before his intended half-marathon in Sheffield. Prednisolone (2.5 mg) was accidentally omitted two days before, taken normally the day before, the next intended dose being after the race.

At autopsy the heart was enlarged (510 g) with

symmetrical left ventricular hypertrophy. The two major branches of the left coronary artery were occluded (80% and 50% focally) by atheroma without thrombosis. There was mild subendocardial fibrosis. Active sarcoidosis was diffusely present throughout the lungs and pleurae, in the spleen, lymph nodes, liver and bone. The adrenal glands (total weight 4 g) showed cortical atrophy and lipid depletion.

We concluded that 3 years of prednisolone therapy for sarcoidosis led to adrenal atrophy and that the stress of running and absence of sufficient replacement prednisolone before the race precipitated acute adrenal insufficiency with hypotension. Myocardial ischaemia and cardiac arrest followed as a result of reduced blood flow through the partially stenotic atheromatous coronary arteries.

There was no Inquest, as H.M. Coroner considered this "a natural, albeit unusual, death".

DISCUSSION

Marathon organisers usually advise intending runners who are inactive or who are in any doubt about their health to seek prior medical advice. It would be impractical to require a certificate of fitness from every entrant. This runner took a calculated risk in the light of what he was told about the suppression of his stress-response systems and his proven capabilities at endurance running. In view of this, even if an ACTH challenge had been performed and showed reduced adrenal capacity to respond, it is doubtful whether he would have been deterred. Possibly the omission of prednisolone 48 hours earlier contributed. It is questionable to counsel enhanced steroid cover for the event itself and not for all the training runs. We conclude that in the circumstances reported the death was unavoidable, but it confirms the need to accommodate artificially to stressful situations when the relevant body systems have been suppressed.

ACKNOWLEDGEMENTS

We thank the family of the patient and Dr. H. H. Pilling (H.M. Coroner, South Yorkshire) for their assistance and encouragement in reporting this case, and Mrs. M. Hogg and Mrs. J. Saunders for their assistance in the preparation of this manuscript.

REFERENCES

- Anonymous, 1982 "Marathon update". Running Magazine, June 21.
- Maughan, R. J. and Miller, J. D. B., 1982. Popular marathons: forecasting the casualties (Correspondence). British Medical Journal 285: 1736.
- Nicholl, J. P. and Williams, B. T., 1983 "Injuries sustained by runners during a popular marathon". British Journal of Sports Medicine 17: 10-15.