Dear Editor,

ACUTE RHABDOMYOLYSIS IN A MARATHON RUNNER

Acute muscle breakdown is known to occur after severe exercise ("exertion rhabdomyolysis"). Animal experiments have shown various minor morphological changes after acute exercise. In humans, raised plasma enzymes (CPK, LDH, GOT, GPT), haemoglobinuria and haematuria have been noted after unaccustomed exercise. Electron microscopy on needle biopsies of the gastrocnemius muscle in volunteers just before a marathon and at various times afterwards have shown ultrastructural evidence of muscle cell necrosis. To our knowledge the muscle pathology has not been previously described at autopsy in a person whose death directly followed running a marathon, and we wish to report the unusually severe muscle changes found in such a case.

The patient, a man aged 47, was running a marathon, when he collapsed about 30 yards from the finishing line. (The case history has previously been documented, Lancet 1982, 2: 1096-7.) Briefly, he was resuscitated by external cardiac massage, defibrillation and ventilation and was transferred to hospital, where his rectal temperature was found to be 34.2°C and serum potassium 7.7 mmol/l (Normal 3.5-5.00). Plasma enzyme levels were as shown in Table 1.

Plasma enzyme levels in a 47-year-old man after running the marathon.

Days after marathon	Creatine kinase (Units/l) (Normal <150)	LDH (Units/l) (Normal <525)	AsT (Units/l) (Normal <48)	AlT (Units/l) (Normal <55)
0	42,300	1,460	1,010	290
1	223,000	5,250	5,180	880
2	148,900	4,700	4,050	790
3	70,000	4,600	2,790	800
5	5,400	3,400	670	430

Twelve days after admission to hospital the patient died. Apparently he had previously been a fit and healthy adult. Indeed, 6 weeks before he had successfully completed another marathon. At autopsy 13 hours after death the heart was enlarged (640 g). Post mortem angiography showed complete occlusion of the right coronary artery near its ostium, and marked narrowing of the descending branch of the left coronary artery, due in both cases to atheroma. Microscopically there was evidence of myocardial fibrosis.

Dissection of the fixed brain revealed hypoxic changes in the hippocampus and cerebral cortex, with diffuse multiple microinfarcts in the brain stem and basal ganglia. Death was certified as due to ischaemic heart disease.

A 2 × 1 × 1 cm piece of skeletal muscle was taken from the right quadriceps group (rectus femoris) along with a portion of pectoral muscle. Both pieces of tissue were fixed in formalin, paraffin-embedded and stained with H & E, PAS and MSB. Quadriceps was selected because several competitors stated after the race that they suffered considerable pain in the front of the thigh.

In the right quadriceps there was marked variation in muscle fibre size (Fig. 1) with a tendency towards rounding of fibres, allowance being made for a uniform degree of sarcoplasmic retraction, which is a normal post-mortem phenomenon in wax-processed muscle. Many muscle fibres were undergoing degeneration (Fig. 2), with focal myophagia. Vacuolated fibres were seen scattered throughout the fascicles, and there were small vacuoles in intrafusal fibres. Occasional regenerating (basophilic) fibres

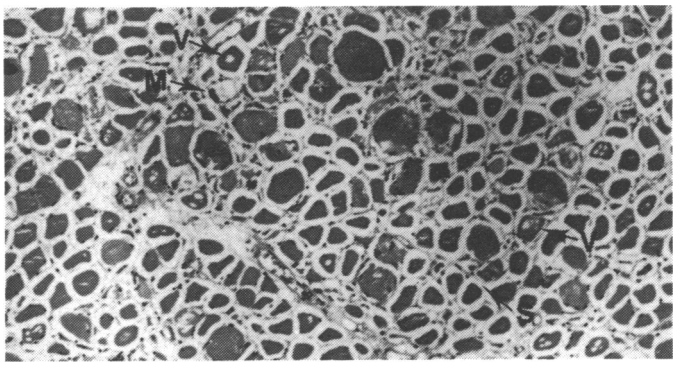

Fig. 1: Marked variation in muscle fibre diameter, with cytoplasmic vacuoles (V), myophagia (M) and only a sparse inflammatory infiltrate. Sarcoplasmic retraction (S) is a post mortem artefact.

Cross section of rectus femoris H & E × 160.

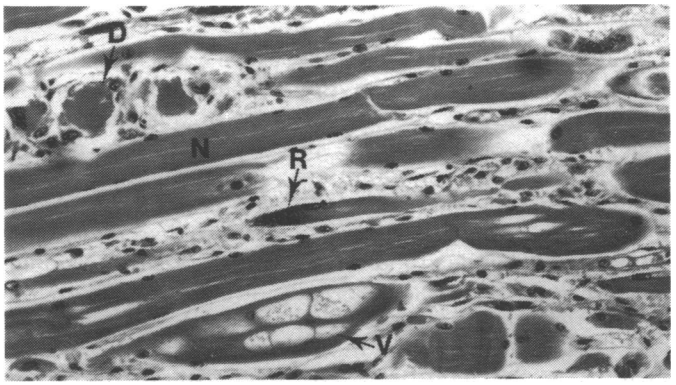

Fig. 2: Vacuoles (V) in muscle fibre, with degeneration (D) and regeneration (R) of fibres. Note normal fibres (N). Longitudinal section of rectus femoris. H & E × 240.

were seen, and throughout the endomysium small blood vessels were uniformly dilated. Small focal lymphocytic aggregates were also present within the endomysium, but there were no perivascular infiltrates. By contrast pectoralis major showed no fibre necrosis but there was occasional fibre atrophy, nuclear clumping, and, here and there, vacuolated fibres. The blood vessels appeared normal.

Skeletal muscle damage of the severity we have described has not, to our knowledge, previously been reported in a marathon runner. The pathogenesis of the changes is not clear. The morphology does not suggest ischaemia as a mechanism for the muscle damage. The fact that the patient had completed a marathon some weeks previously raises the possibility that this and/or the intervening training induced a degree of muscle damage which in some way predisposed him to the severe muscle fibre breakdown in the second race. An allergic reaction might be a mechanism for this but the relatively minor lymphocytic reaction does not support such a view. There is nothing in the patient's history to suggest a subclinical myopathy; no genetic disorder was demonstrated and there was no family history of overt muscle disease.

Rhabdomyolysis has been noted after Echovirus 9 infection but viral studies were not performed to exclude this, as the cause of death was considered at autopsy to be ischaemic heart disease and the unusual muscle changes were not anticipated.

Yours faithfully,

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