in other organs in man, it provides some evidence of liver function. Other liver-dependent coagulation factors, as measured by the prothrombin time (one-stage and two-stage), were well maintained. There was also evidence of the development of a hypercoagulable phase as suggested by the slow fall in fibrinogen, increasing factor VIII levels, and thrombocytopenia. Antilymphocyte globulin, however, was also being administered and fibrin degradation products were not measured.

Although the histological appearances are difficult to evaluate in terms of function, it was considered that in the absence of arterial thrombosis the liver would have been expected to survive. There was only moderate evidence of rejection as indicated by mononuclear cell infiltration.

In conclusion, satisfactory function has been demonstrated in a human liver allograft after prolonged ischaemia, protected by the simple method of core-cooling in situ and infusion of a preservative solution. Whereas experimental evaluation of this technique in the dog provided only three and a half hours of safe ischaemia (Schalm, 1968), this interval may apparently be extended in man.

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Sudden Death after Exertion in Apparently Healthy Boy

British Medical Journal, 1969, 4, 30-31

It was taught, and perhaps still is, that the normal heart cannot be overtaxed. That this idea may need revision is suggested by the following case.

CASE REPORT

A 15-year-old Glasgow boy scout had been perfectly well during the few weeks before his death on 8 May 1965. In particular, there was nothing to suggest a recent infection. On the morning of his death he had taken part in an athletic meeting at his school, but apparently had not unduly tired himself. He had had a light lunch before going to Aberfoyle to start a 12-mile (19-km.) walk to Rowardennan in the company of other boy scouts. He was one of a group of three and had forged well ahead of his companions by running. He had left the course, but it was impossible to attract his attention, and he ran into some woods which were crossed by deep drainage trenches. The effort required to make progress across such terrain must have been considerable. About eight miles (13 km.) from the start the other runners caught up with the boy, who complained of cramp in his legs and was unable to run any further. He soon collapsed, and his companions, realizing that he was unwell, tried to get help. When a farmer arrived the boy was dead.

Necropsy.—The body was that of a well-built adolescent male. There was intense cyanosis of the fingers and ears. The skull was normal, the dura was not tight, and the venous sinuses were clear. The brain weighed 1,415 g. and was completely normal when sectioned serially after fixation. Apart from an area of melanosis of

the leptomeninges at the second and third cervical segments, the spinal cord was normal. There was a small quantity of clear yellow fluid in the pericardial sac. The heart, which weighed 290 g., showed dilatation of both ventricles, the right more than the left, but there was no structural abnormality. The myocardium, endocardium, and valves were normal. The coronary arteries were normal. The aorta was not hypoplastic and the carotid arteries were normal. The pleural cavities were clear, but the main air passages contained a considerable quantity of frothy blood-stained fluid. The mucosa of the trachea and main bronchi was very congested. The lungs together weighed 750 g. and were slightly oedematous. The alimentary tract was normal, apart from a haemorrhagic area in the gastric mucosa. The liver and pancreas

FIG. 1.—Many fibres were bright red—evidence of damage—and appear dark. There was no inflammatory exudate. (Cresyl violet and acid fuchsin. $\times 320$. Green filter.)

were extremely congested. The spleen was slightly enlarged, weighing 150 g.; there were a few soft slightly enlarged lymph nodes in the mesentery; and the thymus was normal. No abnormality was found in the genitourinary or the endocrine system.

Histology.—The liver and adrenals were extremely congested. The kidney, thymus, pancreas, and mesenteric lymph nodes were normal. Sections of the heart stained with haemalum and eosin showed no evidence of myocarditis, but there was a patchy eosinophilia. Further sections of myocardium stained with cresyl violet and acid fuchsin showed pronounced degeneration of many muscle fibres (Figs. 1 and 2) (Bajusz, 1963). This degeneration was also seen in sections stained with phloxine-tartrazine (Lendrum, 1947; Lendrum *et al.*, 1962) (Fig. 3).

FIG. 2.—In a longitudinal section the transverse striations were bright red in the damaged fibres. (Cresyl violet and acid fuchsin. $\times 400$. Green filter.)

FIG. 3.—Damaged fibres show again as darker fibres due to the increased uptake of phloxine. (Phloxine-tartrazine. $\times 400$. Green filter.)

COMMENT

This boy was in perfect health until he complained of cramp in his legs and rapidly lost consciousness and died. There was no family history of sudden unexplained death.

No structural abnormality was found in his heart, though the cyanosis, congested viscera, and pulmonary oedema indicate that he died of heart failure. There is no reason to suppose that he had taken any vasodilator drugs, nor was there any evidence of myocarditis. It seems, therefore, that his heart had sustained too severe a strain.

Sections of his heart stained with acid fuchsin showed marked acute degenerative changes such as would be expected if the blood supply were insufficient to meet the metabolic demands of the heart muscle. That this state of affairs is not infrequently found in a less severe form is suggested by the fact that some athletes recognize the existence of a "pain barrier," which they are able to "run through." This pain barrier could be anginal in nature.

Reindell *et al.* (1960), however, were unable to find evidence of coronary insufficiency in the electrocardiograms of athletes during training or in competitions. Nor could they find any pathological changes in the heart muscle. In the present case sections stained with haemalum and eosin also looked normal, and it was only when special stains were used that the evidence of severe but recent damage was discovered.

Jokl and Melzer (1940), in a study of fatal collapse during work and sport, did not find any evidence that a previously healthy person could die from exertion. Nevertheless, Case 41 in their first table had no convincing evidence of disease, necropsy merely showing a dilated heart. He had died while engaged in athletics of some kind, and the cause of death might have been similar to that in the present case.

A trained athlete is almost certainly in a much better condition to attain the necessary increase in coronary flow to maintain the blood supply to the heart than a 15-year-old boy. The only explanation that can be put forward for this boy's death is that by taking part in a cross-country run he overexerted himself and his heart, which was unable to increase its blood supply sufficiently.

Like Pheidippides, 2,000 years earlier, taking the news of victory at Marathon to Athens, he killed himself by athletic effort.

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