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CORRESPONDENCE

Correspondents are asked to be brief

Cardiac Transplantation Today

SIR,—Your leading article on cardiac transplantation (13 November, p. 377) is most welcome. I would like to join you in paying tribute to Dr. N. Shumway and his colleagues for obtaining results which compare favourably with those reported for renal transplantation by the Kidney Transplant Registry as recently as 1965.

The fact that these results have been achieved by using methods of immunosuppression which are widely known and which have been, and are being, used in renal transplant recipients, disposes of the notion that the immunological barrier in heart transplantation is of a different order of magnitude from that which occurs with kidney transplants—a notion which became current as a result of the numerous dismal failures in various parts of the world which followed Professor C. Barnard's first successful clinical heart transplant some four years ago.

What then are the reasons for Dr. Shumway's success? I would suggest in particular the long period of patient experimental work which preceded the first clinical trial, and the readiness to make use of the lessons already learned by people engaged in clinical transplantation of the kidney. Is there then any reason why, given the same combination of competence in the fields of cardiac surgery, and immunosuppression, and the characteristics of common sense and humility which Dr. Shumway possesses so abundantly, comparable success to that achieved at Stanford could not be achieved elsewhere?

One factor which might affect the answer to this question concerns the criteria of death applicable to the donor, since this clearly affects the so-called "warm ischaemia time" of the transplant. It would be interesting to know whether the criterion of brain death is used at Stanford, since if this is the case, the question would arise as to whether the heart would tolerate the longer period of ischaemia which would result from using the criteria which are generally accepted in this country. My guess is that a human heart transplant would be no less tolerant of ischaemia than a human kidney transplant. In the last resort only clinical trial will show whether or not this is true, but the first step should be to investigate the question in experimental animals.—I am, etc.,

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Coronary Deaths—How Unexpected?

SIR,—There is a widespread conviction that fatal myocardial infarction strikes with little or no warning. Your nihilistic leading article on "Coronary Deaths" (9 October, p. 64) unhappily reinforces this conviction, because it draws its conclusions from studies which have shown that the conventionally accepted warning signs, such as chest pain and E.C.G. changes, may be delayed almost to the moment of death. It fails to admit the possibility of a wealth of earlier and therefore more valuable predictive evidence.

It is becoming more widely recognized that myocardial infarction is not a sudden event but the outcome of a long-standing coronary arterial disease process,[1] which may be well established even in the second and third decades of life.[2] Necropsies in "unexpected" coronary death show that the pathological changes of this long-standing disease process chiefly affect the blood supply of the left ventricle, and it is reasonable to explore the possibility that dysfunction of the left ventricle could give rise to symptoms and signs long before the stage of pain and muscle necrosis.

We have taken a close interest in the "coronary" personality and the pre-infarction phase of coronary heart disease, and have concluded that there are both recognizable behavioural characteristics and cardiac symptoms and signs of value in the diagnosis of this condition.[3] The symptoms suffered by the patient usually extend over many months, during which there is a deterioration of general health, increasing fatigue, and loss of effort capacity with uncomfortable exertional dyspnoea. Business efficiency deteriorates, tensions rise, and irritability becomes more marked, but the patient ignores these symptoms, which he commonly attributes to ageing. We have encountered patients who have lost more than 80% of their usual capacity for physical effort without ever having experienced angina pectoris.[4] In some cases a single severe attack of ischaemic heart pain on effort acts as a warning not to repeat the experience. It is a common finding that the patient will deny or rationalize a gross deterioration which is obvious to his wife and friends. The aggressive, type A behaviour pattern displayed by many of these individuals has been well described.[5]

The physical sign which indicates that the heart is the probable cause of this deterioration is the palpable and audible atrial gallop, which is most easily elicited by examining the patient in the left lateral position. We have found this sign in 65% of a series of 72