coronary care. The patient with an apparently mild myocardial infarction is at as great a risk of developing fatal ventricular dysrhythmia as the patient with a clinically severe infarct.⁴

In Newcastle the interval between the receipt of the call from the practitioner and the arrival of the coronary care team with the patient (mean 21 minutes) contrasts with the Belfast time (median 10 minutes). The inordinate delay may contribute to the lack of interest of the Newcastle practitioners in the scheme. Dr. Dewar and his colleagues indicate that only 42% of the Newcastle practitioners use the mobile unit and that the number is not increasing. A questionnaire conducted by one of us (Dr. Zaidi) in March 1969 indicated that 89% of the Belfast practitioners use the mobile scheme.

Another important difference between the two schemes is that in Belfast only personnel who have gained experience in the hospital coronary care unit are allowed to go on the mobile team. We agree with Dr. Dewar and his colleagues that it is most important to have adequate inpatient coronary care facilities for reception of patients managed by a mobile unit. It should be stressed, however, that all the available data indicate that at least two-thirds of the deaths from myocardial infarction occur outside hospital and that therefore when problems of staffing exist priority should be given to staffing of the mobile unit.—We are, etc.,

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Car Driving and the Heart

SIR,—The effects of motor-car driving on the normal and abnormal heart, reported by Dr. P. Taggart and others (18 October, p. 130) are of considerable interest. I wonder, however, if these workers were to use as their control study the same subjects attempting to cross busy city roads, whether the final verdict would be reversed—that is, that a person in whom angina is easily provoked should be advised to be a driver rather than a pedestrian?—I am, etc.,

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SIR,—I trust the paper by Dr. P. Taggart and others (18 October, p. 130) does not induce readers to pursue their advice against the issue of driving licences to persons with angina and "borderline" failure. While the authors admit the existing evidence that syncope from previously known coronary disease is rarely the cause of fatal traffic accidents, they seem quite prepared to deprive a very large number of people of their only means of getting about and continuing their employment on the basis of their very equivocal clinical findings in the cases they investigated.

We all know that these patients can continue to lead independent and useful lives for many years under favourable conditions. Hurrying about, carrying parcels, dodging traffic, and standing at bus stops are not less harassing to them than driving a light car with a good heater, and can lead to just as many accidents, involving others as well as themselves.

Car accidents are caused by errors of judgement more often due to latent illness, fatigue, haste, obstinacy, aggressiveness, or emotional instability than to patent heart disease. The heart patient knows he cannot afford to take chances, and will limit, by necessity if not by choice, the number, length, and speed of his car journeys and the size of his car. If his symptoms still cause distress or impairment of judgement when driving, he will pull into the side of the road, and if this happens too often he will be obliged to give up driving altogether for a time or permanently if need be. But this may not happen for five—or even 25—years after the first appearance of angina and symptoms of "borderline" failure.—I am, etc.,

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Treating Preinfected Wounds

SIR,—Mr. J. W. S. Rickett and Mr. B. T. Jackson (25 October, p. 206) report a reduction in wound infection after appendicectomy in patients who received topical ampicillin during closure of the muscle layers of the wound. Before the significance of this finding can be assessed, one must surely know what organisms were cultured from the infected wounds, and what methods of isolation were employed. The authors state that all wound infections grew "large bowel organisms." Were these *Escherichia coli*, *Proteus* sp., *Pseudomonas pyocyanea*, or what? Surely it is important to distinguish colonization of the wound with bacteria, an innocuous situation in most patients, from superinfection, a dangerous precursor of systemic infection.

A final point about the study which disturbs me is the comparison of an antibiotic with a carbohydrate powder. In the laboratory in our department we have found that standard laboratory organisms grow rather well in solutions containing 1% lactose and some protein. Might not the powder have, in fact, encouraged additional infection?—I am, etc.,

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SIR,—A query from a medically naïve zoologist. Mr. J. W. S. Rickett and Mr. B. T. Jackson (25 October, p. 206) conclude that their double-blind trial on the use of topical ampicillin in appendicectomy wounds "indicates that topical application of this drug reduces postoperative wound infection."

The placebo used in their trial (lactose) is a nutrient substrate eminently utilizable by large-bowel organisms. Thus, might one not expect a significant increase in the incidence of wound infections in the placebo group compared with any experimental group,

whether it were treated with ampicillin or sterile talcum powder?

Perhaps someone can convince me that treating preinfected wounds with bacterial substrates does not increase the incidence of overt infections.—I am, etc.,

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SIR,—I wonder whether Mr. J. W. S. Rickett and Mr. B. T. Jackson (25 October, p. 206) realize that, by using lactose powder as a placebo in their double-blind trial of topical ampicillin in appendicectomy wounds, they may have invalidated their findings.

The vital requirement of the control placebo in a double-blind trial is that it must be inactive in the specific context of the trial. While lactose is an excellent placebo in the controlled trial of almost any orally administered drug, in the specific context of the trial of a topical antibiotic it is far from inactive. The local high concentration of lactose which is produced by sprinkling it into the wound provides an excellent medium for the growth and multiplication of pathogenic bacteria. It is not, therefore, surprising that the control group in this trial had such a high infection rate.—I am, etc.,

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Renal Transplantation and Leucocyte Migration

SIR,—The diagnosis of rejection of an allotransplanted kidney can be difficult, as pointed out by Dr. M. G. M. Smith and others (1 November, p. 275), but I feel that they have overplayed the limitations of conventional renal function tests.

If for a brief moment we set aside lymphocytes and antibodies and concentrate on the mechanics of rejection it will become apparent that some of us now agree that acute rejection equals an acute haemodynamic upset.¹ This upset varies from frank exclusion of flow from the outer cortex to vascular mural oedema decreasing perfusion pressure, and the net result is impaired renal perfusion. The 15-minute phenol red test² can detect this defect within 30 minutes of deciding to measure the effective renal plasma flow. If a functional pattern of impaired renal perfusion is detected by a low phenol red excretion, then a differential diagnosis from renal artery stenosis (by an arteriogram) and ureteric obstruction (by an I.V.P.) can be made long before the results of a leucocyte migrating test (20 hours) become available. Total renal blood flow is not reliable—until rather late in rejection. What is important in the early stage is the distribution of the blood, and the effective renal plasma flow reflects this.—I am, etc.,

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