Lung Transplantation

SIR,—The problems of lung transplantation have been reviewed in your leading article (12 September, p. 600). The combination of infection and rejection are a particular hazard in an organ that is exposed to the atmosphere, and it must be expected that prospects of success cannot improve until there are advances in medical management. However, not all the current evidence is unfavourable and some points are worth noting.

Mucus transport may be severely curtailed in animals after lung reimplantation, but recovery probably occurs after 90 to 120 days.[1]

Lymphatics are rapidly reconstituted, and loss of bronchial circulation does not appear to cause problems.

Denervation is likely to become an important problem only if the cough reflex from both lungs is lost or if the upper airway reflexes are interrupted.

A lung homograft provides a large vascular bed as well as a ventilation organ, and pulmonary hypertension may be improved as in F. Derom's patient.

The use of enhancing serum has been shown in animals[2] to be capable of preventing rejection without increasing the subject's susceptibility to infection.

The chance of a successful lung transplant may be considerably higher now than in the recent past. Like many of our colleagues, we believe that the most favourable cases to consider for this operation are cases of advanced pulmonary fibrosis or of primary pulmonary hypertension.—We are, etc.,

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Salicylates and Papillary Necrosis

SIR,—While I have the greatest admiration for the work of Dr. R. S. Nanra and Dr. P. Kincaid-Smith, in their recent article on experimentally-induced renal disease (5 September, p. 559) they have drawn a number of conclusions which do not seem justified. The gross effect of seasonal variation on their results in the A.P.C.-treated animals and our ignorance of the season in which the trial of diuretic substances was made makes comparison between trials difficult. However, analysis of their data (A.P.C.-treated animals excluded) fails to show any significant reduction in the incidence of renal papillary necrosis in rats given diuretic substances, and makes the subsequent discussion of their effect in mitigating the nephrotoxicity of salicylates seem irrelevant.

While it is obvious that patients with rheumatoid arthritis are at risk because of their high mixed analgesic intake, perusal of the references to the incidence of renal papillary necrosis and interstitial nephritis in these paients does nothing to clarify the aetiology. Thus Clausen and Pedersen[1] suggested that phenacetin might be the cause; Brun *et al.*[2] were unable to correlate the degree of interstitial nephritis with analgesic consumption; Lawson and Maclean[3] found that the consumption of phenacetin with salicylates was associated with more severe renal damage than salicylates alone or in combination with drugs other than phenacetin; Bulger *et al.*[4] made the pathological diagnosis of interstitial nephritis on the basis of very sketchy renal function tests without histological confirmation; Nanra *et al.*[5] felt it unnecessary to inquire into the type of analgesics taken by their eight patients with rheumatoid arthritis. The implication that salicylates are responsible for the renal papillary necrosis in these patients is not supported by the absence of this lesion in those abusing salicylates alone.[6]

While salicylates may indeed be responsible for renal damage in man, surely a more objective approach to the problem is indicated.—I am, etc.,

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Myocardial Infarction and the G.P.

SIR,—Dr. L. C. Bousfield and his colleagues (3 October, p. 54) give a timely reminder of the need for practitioners in domiciliary practice to take a more active part in preventing primary arrhythmic deaths in patients with acute myocardial infarction. We would endorse their view that, as well as administering atropine in cases with bradycardia, lignocaine should be administered intravenously, with or without an intramuscular injection, in appropriate doses to suitable cases. We would go further than the authors of the letter, however, and recommend that, assuming bradycardia can be corrected with atropine, each case of suspected or established acute coronary heart disease in domiciliary practice should be promptly treated with 80 mg. of intravenous lignocaine.

Our own experience of routine lignocaine administration in a coronary care unit would support the view that this drug should be given routinely in acute myocardial infarction. Fifty-five successive cases of acute myocardial infarction received 80 mg. of intravenous lignocaine immediately after admission to and monitoring in the coronary care unit. The bolus was followed in each case by an infusion of 1-2 mg. of lignocaine per minute. Only five of these patients showed frequent ventricular ectopic beats (five or more per minute, or multifocal complexes) during the subsequent 24 hours. None developed ventricular tachycardia or primary ventricular fibrillation. In a further 55 successive cases of acute myocardial infarction not given routine lignocaine, 34 showed frequent or multifocal ventricular ectopic beats during the first 24 hours. These patients were, of course, promptly treated with lignocaine and none developed primary ventricular fibrillation.

In our view routine and sustained lignocaine therapy, with or without atropine as indicated, is justified in domiciliary practice where the supervision of patients and the prevention and management of serious primary arrhythmias pose much greater problems than in a coronary care unit.—We are, etc.,

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Larva Migrans

SIR,—Dr. C. B. Vaughan's letter (17 October, p. 179) revived a personal memory. In the second world war I was stationed for 16 months at a military hospital in Lagos. One of my leisure pursuits was to tend the garden adjoining the officers' quarters, without wearing gloves.

There came a day when one of my fingers started itching. After several days of frenzied scratching I observed a palpable serpiginous track about an inch (2·5 cm.) or so in length along the inner border of the finger. The "thing" travelled backwards and forwards up and down the finger causing me much irritation and my brother officers mild amusement.

I was eventually persuaded to consult a civilian doctor practising in Lagos. I cannot now recall whether he called it *Ancylostoma braziliense* or *caninum*. He advised me to freeze the head with ethyl chloride for two minutes. I would like to challenge anyone to endure the freezing of a thin finger with ethyl chloride for two minutes. However, after a few freezings of shorter duration a pustule developed which was incized under pentothal. The "thing" was never seen again and there was never any intimation to visceral migration.—I am, etc.,

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Medical Students and Smoking

SIR,—Dr. J. P. Anderson's suggestion (10 October, p. 120), that a copy of the Royal College of Physicians' forthcoming second report "be placed in the hands of every medical student in the country" is very worthy of consideration, not only in Great Britain but even elsewhere. In a recent survey carried out in university students in Uganda, Arya and Bennett[1] reported that the medical faculty had the highest percentage of smokers compared with students of other faculties. Some of the medical students claimed that they limited their smoking to the point where the risk was less. Although more medical students on the whole knew about harmful effects than their non-medical counterparts few mentioned effects on systems other than respiratory. More medical students (18·3%) queried lung cancer as a result of smoking than non-medical students (6·7%), and more medical students started to smoke after

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