animals revealed renal changes similar to those already described for other acute infective diseases.⁴

Human subjects and higher mammals vary more in their response to bacterial toxins than smaller experimental animals. Hence gross renal changes do not always occur in acute infections or severe endotoxaemia, although albuminuria with a few red cells is surely not uncommon.

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¹ Hopkin, D A B, and Laplane, R, *Annals of the Royal College of Surgeons of England*, 1978, **60**, 108.

² Reilly, J, et al, *Le Rôle du Système Nerveux en Pathologie Rénale*. Paris, Masson, 1942.

³ Marquiez, R A, and Ladet, M, *Le syndrome malin au cours des toxi-infections. Le rôle du système neuro-vegetatif*. Report to the Congress of Paediatrics, Paris, 1938.

⁴ Reilly, J, et al, *Annales de Médecine*, 1950, **51**, 597.

Isolation to control infection

SIR,—In the admirable paper by Professor K D Bagshawe and his colleagues (2 September, p 684) there are included in the table two diseases concerning which the recommendations are misleading.

For *Taenia solium* infection it is stated that no isolation is required but precautions are necessary when dealing with dressings and discharges in the ward. The ova of *T solium* are dangerous and if swallowed cause cysticercosis, with epilepsy and other serious complications. As these ova are passed with the patient's stool and may contaminate fingers and clothing, isolation and adequate measures to dispose of infective faeces are essential.

Secondly, yellow fever is stated to require no isolation. There is evidence that it has been transmitted by blood in areas in which it is not endemic. Precautions similar to those used for serum hepatitis should therefore be taken in dealing with such patients' blood and tissues. Even in Britain it would also be advisable to ensure that the patient's room is kept free from mosquitoes and other biting insects, for mechanical transmission by these is theoretically possible. In endemic areas strict isolation of the patient from mosquitoes and other insects is an established necessity.

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Stroke after myocardial infarction

SIR,—We were interested to learn (12 August, p 457) that the experience of Drs P L Thompson and J S Robinson with strokes after myocardial infarction led them, as it did us,¹ to adopt a selective anticoagulation regimen. Our regimen, however, differs from theirs in two ways.

Firstly, we use a plasma fibrinogen level of 7.5 g/l (750 mg/dl) or more as an indicator of risk. The estimation, using nephelometry, is not difficult: we found it more predictive and it obviates the possibility of missing the creatine kinase (CK) peak. Moreover, there are patients whose fibrinogen levels, for one reason or another, are consistently higher than normal. In such patients a relatively small infarct could push the fibrinogen up to dangerous levels.

Secondly, but perhaps more important, is the timing of anticoagulation. We too found that the majority of strokes occurred in the first week after infarction and not uncommonly by the fourth day. To wait even 48 h for the CK to peak may add to the risk of thromboembolism. We therefore anticoagulate all our patients from admission and stop on the sixth day if they are not at risk.

We use subcutaneous heparin (10 000 U twice daily) for six days and give a loading dose of warfarin on the fourth day. If, on the sixth day, the plasma fibrinogen has reached 7.5 g/l we continue warfarin for six weeks. Otherwise we stop all anticoagulation.

In two groups, each of 120 patients, the first unanticoagulated and the second anticoagulated in this way, the incidence of all thromboemboli was 10 and one respectively ($P < 0.01$) and the incidence of strokes was five and one. We have continued to use this regimen for a further 18 months and have not encountered a stroke in patients so treated.

The ideal way of dealing with the problem may well lie somewhere between our two methods, but there seems little doubt that some form of selective anticoagulation after myocardial infarction can safely prevent a measurable amount of tragic morbidity.

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¹ Fulton, R M, and Duckett, K, *Lancet*, 1976, **2**, 1161.

SIR,—I read with interest the paper by Drs P L Thompson and J S Robinson (12 August, p 457) on the relation of stroke to size of myocardial infarct.

It is clear from their study that strokes, in this setting, are associated with very high levels of creatine kinase (CK). However, this may not be due entirely to the presence of a large myocardial infarct per se. Thus Dimant and Grob¹ found that 25% of stroke patients without myocardial infarction have elevated CK levels with a median level $2\frac{1}{2}$ times the upper limit of normal. The CK in such cases almost certainly arises from cardiac muscle and is probably a result of the subendocardial haemorrhages and myocytolysis seen secondary to cerebral injury.²

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¹ Dimant, J, and Grob, D, *Stroke*, 1977, **8**, 448.

² Connor, R C R, *British Medical Journal*, 1968, **3**, 29.

Carcinoma of bronchus presenting with Henoch-Schönlein purpura

SIR,—Dr S A Cairns and others (12 August, p 474) describe two cases of squamous cell carcinoma of the bronchus presenting with Henoch-Schönlein purpura. It is suggested that tumour antigen may have precipitated the attack.

On 20 July 1956 a man aged 59 consulted his general practitioner on account of pain and swelling in both knees and a purpuric rash affecting both lower legs. No treatment was offered initially, but he returned one week later, when the rash was more marked and he mentioned that on one occasion only he had coughed up a little blood. He was thought to have Henoch-Schönlein purpura, started on

oral penicillin, and advised to submit weekly urine specimens. On 10 August the urine contained frank blood and he was admitted to hospital on 13 August. At that time he still had a rash typical of Henoch-Schönlein purpura over both lower legs. His blood pressure was 180/80 mm Hg. The urine contained albumin +++ and numerous red blood cells. His haemoglobin concentration was 9.6 g/dl, and leucocyte count $10.8 \times 10^9/l$ ($10\ 800/mm^3$) with 56% polymorphs. X-ray of the chest showed some collapse of the right lower lobe.

He was treated with combined penicillin and streptomycin and the rash gradually faded and the urine cleared except for some persistent red blood cells. However, the collapse became rather more complete, although the right hilar shadow was normal. He was transferred to Oxford on 29 September 1956. There a specimen of sputum was obtained containing typical squamous carcinoma cells and bronchoscopy showed "right stem bronchus blocked by cauliflower mass." On 22 October the middle and right lower lobes were resected. The pathologist reported: "This is one of the earliest carcinomata of the bronchus seen here." Lymph nodes were clear.

There has been no recurrence of rash, nephritis, or tumour. At the age of 81 he is in fair health and caring for his invalid wife.

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SIR,—I was interested to read the paper by Dr S A Cairns and others (12 August, p 474) concerning two patients with squamous cell carcinoma of the bronchus who presented with Henoch-Schönlein purpura.

Last year we showed at the clinical section of the Royal Society of Medicine a man who presented in October 1973 with an acute Henoch-Schönlein syndrome including haemoptysis.¹ In contrast with Dr Cairns's cases he subsequently had recurrent episodes of purpura which only cleared after pneumonectomy for squamous cell carcinoma of the bronchus in November 1975. He remains well; it is of particular interest that the patient gave a history of Henoch-Schönlein purpura as a child.

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¹ Mitchell, D M, and Hoffbrand, B I, *Journal of the Royal Society of Medicine*. In press.

Antifibrinolytic agents for ruptured intracranial aneurysms

SIR,—Might I be permitted to reply to the criticisms raised by Drs J van Rossum and A R Wintzen (19 August, p 568) of my study (15 April, p 945) of the effect of prolonged antifibrinolysis on ruptured intracranial aneurysms and at the same time comment on their trial,¹ which showed no benefit for antifibrinolytic treatment?

Although my study was not double-blind, it did possess randomly selected controls. As the criteria for failure of treatment (that is, death or recurrent haemorrhage confirmed by lumbar puncture or necropsy) were fairly objective occurrences, knowledge of which patients were receiving drug treatment could not be expected to bias the results in favour of the treatment group. However, the control