as a 95% confidence interval so that the reader can deduce for himself how much reliance to put on what was found. In the absence of one or both of these approaches the value of their negative finding cannot be estimated by the reader.

The use of bias controlling methods may not be necessary in a preliminary assessment of the effects of a new agent as long as any positive finding is not regarded as showing a causal relation between the treatment and the "response." The value of a negative finding is not enhanced by the presence of a control group; it is still necessary to quote power or confidence intervals, or both.

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Infectious diseases physicians and microbiologists

SIR,—We were grateful that the article by Dr M E Ellis (15 November, p 1303) appeared as a Personal View, since we hope his view is not widely shared.

We would not disagree that misuse of antimicrobials occurs in many UK hospitals. Dr Ellis's suggestion of antibiotic "cover" for the child with watery diarrhoea is surely one situation in which this can occur.

Nowadays serious infection in hospitals in the UK occurs as an aspect of illness in patients suffering from other conditions. Diseases due entirely to infection still occur but are not the main problem with which we have to contend. As clinical microbiologists we spend much of every normal day on the wards seeing patients. For instance, in this regional cardiothoracic centre we treat 25-30 cases of infective endocarditis each year. We have been known to auscultate the occasional heart, but in general we find echocardiograms and the expert opinion of our cardiologists colleagues to be more use. Apart from looking at the urine for evidence of nephritis we too are at a loss to find much use for a microscope in this condition. In any case the microscope is in the laboratory and the patient is not.

The implication that all our clinical skills have atrophied because we do not perform operations or conduct an outpatient clinic is one which we find insulting. In many hospitals in the UK good diagnostic medicine is practised by doctors who may have their offices near a laboratory but who also attend and treat patients. Clinical haematologists seem to have combined the two elements which Dr Ellis finds so disparate. We have yet to meet the two masters referred to by Dr Ellis.

Dr Ellis does have the grace to suggest that there may be an element of paranoia about his view. May we tactfully suggest that the self confessed isolation which he endures is delaying the cure? If he emerges into the rest of the hospital service we clinical microbiologists would be pleased to see him. We would probably learn something from him. He would certainly learn something from us.

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Paradoxical gas embolism in a scuba diver with an atrial septal defect

SIR,—One aspect of the management of the scuba diver who developed paradoxical gas embolism (15 November, p 1277) was puzzling. Dr P T Wilms-hurst and others inform us that the diver was advised not to dive again after surgical correction of his atrial septal defect. Why was this? Was

the surgical repair inadequate? Was the patient permanently disabled neurologically by the gas embolism? Or were the diver's medical advisers just being overcautious?

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AUTHOR'S REPLY—I am grateful to Dr Konarzewski for the opportunity to clarify why the patient was advised not to dive again. There was no suggestion that his surgical repair was inadequate.

The patient described had a paraparesis which resolved only slowly during the year after therapeutic recompression. Even in those individuals who make a rapid and good functional recovery from spinal decompression sickness, there is evidence that large areas of spinal damage can be shown histologically.¹ There is also concern that those who recover from neurological decompression sickness after prolonged treatment might be predisposed to future decompression sickness.² Certainly, some caisson workers appear to have a physical predisposition to decompression sickness.³ The data available to the Medical Research Council decompression sickness panel suggest that among compressed air workers 5% of those who have decompression sickness are responsible for 40% of the bends (V Hempleman, personal communication). Among amateur divers a similar trend is observed in the incident statistics of the British SubAqua Club, with repeated serious bends occurring in a few individuals who are responsible for a significant proportion of the therapeutic recompressions performed (S Shaw, personal communication).

Given that successive bends generally prove more difficult to treat and leave greater residual neurological damage, I would advise an individual against resuming diving after neurological decompression sickness unless rapid and complete recovery had followed recompression. I believe that similar advice not to dive would have been given to this diver by most authorities concerned with safe diving practices.

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Carcinoma in situ of the contralateral testis

SIR,—Both Dr von der Maase and his colleagues (29 November, p 1398) and Mr T B Hargreave (p 1389) suggest that the efficacy of radiation in controlling carcinoma in situ of the testis is unknown and that "low dose radiation . . . is an exciting study which needs further confirmation."

However, it has been known for some time that radiation does prevent the development of second tumours. At the Christie Hospital the field used for prophylactic irradiation in early seminoma (and previously teratoma) always included the contralateral testis.¹ Although the need for its inclusion may be questioned, its effectiveness in preventing second tumours has never been in doubt. In over 1000 patients irradiated from 1960 to 1980 and followed to 15 years no case of a second testicular tumour was observed when one might have expected 50 cases if the incidence of carcinoma in situ is 5%. The dose of radiation to the testis in this

series was 30 Gy in 20 fractions over 28 days from a 4 or 8 MV linear accelerator, which is probably not dissimilar in biological effect from the quoted dose of 20 Gy delivered over 10 days.

Further assessment of Leydig cell function is important. In a Manchester study of 30 men treated by orchidectomy and radiotherapy for seminoma the mean basal follicle stimulating hormone and luteinising hormone values three years after irradiation were significantly greater and the mean basal testosterone concentration significantly lower than those in a group of 14 men treated by orchidectomy only for teratoma.²

It is also interesting to note that of 130 patients with stage 1 teratoma of the testis under surveillance from 1979³ only one patient has so far developed a contralateral tumour. He had previously required chemotherapy for para-aortic relapse (cisplatin, vinblastine, etoposide, and bleomycin) and subsequently died of metastases from his second tumour.

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Acquired cystic disease of the kidney

SIR,—We read with interest Mr C J Rudge's leading article (8 November, p 1186) and wish to add some comments illustrated by the following case report.

A 51 year old man who had been receiving continuous ambulatory peritoneal dialysis for six months was admitted in severe congestive cardiac failure and died shortly afterwards. He had originally been found to have renal impairment (serum creatinine concentration 250 $\mu\text{mol/l}$ (2.8 mg/100 ml)) in 1971 during investigation of hypertension and angina. Renal biopsy showed hypertensive nephrosclerosis. Despite poor compliance with various antihypertensive regimens, his serum creatinine concentration remained below 500 $\mu\text{mol/l}$ (5.7 mg/100 ml) until 1985, and he did not require continuous ambulatory peritoneal dialysis until April 1986. In June 1986 he reported an episode of frank, painless haematuria. Subsequent cystoscopy and retrograde ureterograms were negative and a computed tomogram was considered to show only the changes of acquired cystic disease of the kidney.

At necropsy the kidneys, each 80 g, were shrunken to 8 cm long and contained numerous cortical cysts, up to 0.4 cm in diameter, containing clear fluid. A single solid dark brown tumour, 1.5 cm in diameter, projected from the upper surface of one kidney, which on histological examination proved to be an invasive adenocarcinoma, of mixed papillary and clear cell type. No other tumours or metastases were found.

Although acquired cystic disease of the kidney is usually considered in relation to patients on dialysis, it also occurs in patients with end stage renal disease who have not received dialysis.¹ Since our patient had been on continuous ambulatory peritoneal dialysis for only six months, the acquired cystic disease and renal adenocarcinoma probably predated this treatment; another case report supports this proposal.² We therefore suggest that screening for renal cysts should not be delayed in patients with chronic renal failure until they reach end stage renal disease. Secondly, while we agree with the screening strategy outlined by Mr Rudge, particularly as 16-20% of patients with acquired cystic disease may develop tumours,^{3,5} it is noteworthy that the tumour was not diagnosed