superseded by Einstein's theories, so alternative medical paradigms may also still be useful in appropriate situations.

S R BUCKNALL

Glasgow G62 8NL

1 Kuhn T. *The structure of scientific revolutions*. Chicago: University of Chicago Press, 1962.

Bacterial contamination of home nebulisers

SIR,—Local experience may be of some relevance to the correspondence about bacterial contamination of home nebulisers (30 October, p 812; 14 November, p 1281).

Atomiser units of five "cool mist tents" on a paediatric ward were contaminated with an oxidase positive, Gram negative organism producing yellow colonies on the blood agar after 48 hours' incubation. Decontamination of the machines with an autoclave with a pasteurisation cycle was soon followed by recontamination by the organism, which was identified as *Pseudomonas paucimobilis*. Staff had been instructed to use only sterile water when filling the atomiser reservoirs and to store the machines in a dry state. Questioning revealed the practice of "washing out" the units with tap water before dry storage. Bacterial examination of the ward taps revealed heavy colonisation by *P. paucimobilis*. The persistence of contamination despite prolonged dry storage was explained by laboratory studies in which dried smears made from thick suspensions of *P. paucimobilis* showed viability for up to 50 days at room temperature. During the period of study there were no clinical isolates of *P. paucimobilis* from patients, though evidence of pathogenic potential exists.^{2,3}

This experience prompts me to caution against advising patients to use unboiled tap water when "washing" nebulisers and to note that although desirable, dry storage of such equipment is not always an infallible means of preventing contamination.

B A S DALE

Dumfries and Galloway Royal Infirmary,
Dumfries DG1 4AP

- 1 Dale BAS, Williams J. *Pseudomonas paucimobilis* contamination of cool mist tents on a paediatric ward. *J Hosp Infect* 1986;7:189-92.
- 2 Southern PM, Kutscher AE. *Pseudomonas paucimobilis* bacteraemia. *J Clin Microbiol* 1981;13:1070-3.
- 3 Hajiroussou V, Holmes B, Bullas J, Pinning CA. Meningitis caused by *Pseudomonas paucimobilis*. *J Clin Pathol* 1979;32:953-5.

The blood transfusion service and zidovudine treatment for AIDS

SIR,—There has been recent correspondence about a shortage of blood for transfusion in the South West Thames region (Drs J E W Van de Pette and Janet A Shirley, and Dr K L Rogers (24 October, p 1061, 1062)). We should like to draw attention to an increasingly large group of patients in this region with a transfusion requirement which might be difficult to meet given the decline in blood donations.

St Stephen's Hospital cares for many patients with human immunodeficiency virus (HIV) infection, many of whom need blood transfusion. We have noticed an increase during 1987 in the number of HIV positive patients transfused, this being due partly to the ever expanding numbers of patients ill with the acquired immune deficiency syndrome (AIDS) but also to the introduction of the antiviral agent zidovudine into the management of HIV disease. This agent induces severe anaemia in about a quarter of recipients.¹

During the 10 months January to October 1987

53 HIV positive patients were transfused. The number of transfusions was 94, and 314 units of blood were given. Of the 53 patients transfused, 31 were receiving zidovudine. These 31 patients had 68 transfusions altogether to a total of 213 units of blood; 23 required transfusion more than once (up to 6 times).

In comparison, during January to October 1986, when there were fewer patients with AIDS and no zidovudine, 46 transfusions were given to 25 patients (8 had more than one transfusion); the total number of blood units used was 158. These figures are distorted by the fact that one patient, who had acute leukaemia, required 13 transfusions (total 43 units). Excluding this patient would have made the increase in blood use during 1987 more apparent. Even including this patient it is obvious that we have at least doubled our consumption of blood from 1986 to 1987.

The heavy financial costs of managing patients with AIDS and of using zidovudine are well known. Our blood transfusion figures indicate that money is not the only resource of importance in the management of HIV infected individuals. The South London Transfusion Centre is already badly stretched to provide blood for all patients within the region. Escalating use of zidovudine and increasing numbers of sick patients with AIDS can only increase the demand for a commodity which is perilously scarce. Unless the number of blood donors increases to meet current demands it might even be necessary to restrict the use of zidovudine.

CHRISTINE COSTELLO
NAHEED MIR
JOHN LUCKIT

Department of Haematology,
St Stephen's Hospital,
Chelsea, London SW10 9TH

- 1 Richman DD. The toxicity of azidothymidine in the treatment of patients with AIDS and AIDS-related complex. *N Engl J Med* 1987;317:192-7.

OKT3 and cerebral oedema

SIR,—The recent paper by Dr P A Rowe and colleagues (31 October, p 1099) highlighted the problem of pulmonary oedema induced by monoclonal antibody against the CD3 receptor of T lymphocytes (OKT3) in patients with acute allograft rejection. Cerebral oedema has not been previously described. We report on a patient who developed this complication after receiving OKT3.

A 34 year old highly sensitised woman received her second cadaveric renal transplant through the United Kingdom Transplant SOS scheme. Initial immunosuppression was with prednisolone, cyclosporin, and azathioprine. At six days she had an episode of acute rejection proved by biopsy, which was treated with OKT3 although she was more than 3% above her dialysis weight. Despite intravenous frusemide, within 30 minutes she developed pulmonary oedema, which was relieved by ultrafiltration. Next morning she had headache, photophobia, nausea, and neck stiffness. She was febrile and again had pulmonary oedema.

Lumbar puncture produced cerebrospinal fluid at a pressure of 34.5 cm H₂O with 53 polymorphs/ μ l. No organisms were seen. Antibiotics were started and ultrafiltration resumed with immediate improvement: within 18 hours she was completely better. Because of the results of microscopy of the cerebrospinal fluid and negative cultures the antibiotics were stopped after five days.

The high cerebrospinal fluid pressure with rapid clinical improvement after fluid removal suggests that our patient had cerebral oedema and not bacterial meningitis. Headache is a common side

effect of OKT3 but cerebral oedema has not been described.

D M THOMAS
A J NICHOLLS
T G FEEST
H RIAD

Royal Devon and Exeter Hospital (Wonford),
Exeter EX2 5DW

Treatment of primary biliary cirrhosis

SIR,—Dr N D C Finlayson paints a gloomy picture for patients and physicians alike and in our opinion is too nihilistic (10 October, p 867).

Our unit's interest in specific treatments for primary biliary cirrhosis¹ began with the use of corticosteroids in 1958 and has continued with the first controlled trial of colchicine treatment, which started in 1979.² While we agree with Dr Finlayson that problems exist in the design, execution, and interpretation of clinical trials in primary biliary cirrhosis, some of these are potentially soluble. Our own approach has been to use a "pair matched" study design, which ensures excellent comparability of patients in treatment and placebo groups and avoids the need for statistical recalculation as used in the last azathioprine trial.³

While the possibility of a small beneficial effect of azathioprine cannot be discounted, to our knowledge all clinical trials of this drug in primary biliary cirrhosis have now been abandoned. The results of continuing trials of corticosteroids and cyclosporin are awaited and it is clear that controlled studies of bile acid treatment are indicated. Of the treatments currently available, however, colchicine, which Dr Finlayson dismisses in one sentence, appears the most promising. Three independent controlled trials have now shown significant biochemical improvements with this drug and one has shown improved survival.^{2,4,6} Nevertheless, larger longer term studies are required to establish the full potential of this drug in the treatment of primary biliary cirrhosis.

Future drug trials in primary biliary cirrhosis should be large enough to exclude type II error and patients will require follow up for many years, preferably using sequential dynamic tests of liver function and serum markers of fibrogenesis such as procollagen III peptide, to establish new therapeutic end points. Drug trials should include asymptomatic patients since many of these have portal hypertension,⁷ active fibrogenesis, established cirrhosis, and decreased survival.⁸

Progress has been made in both the symptomatic and specific treatment of primary biliary cirrhosis and in the assessment for and timing of liver transplantation.^{3,9} Further progress now depends on medical practitioners referring patients with primary biliary cirrhosis to centres willing to undertake well designed, large scale, and long term studies of drug treatments in this disease.

C BABBS
A SMITH
T W WARNES

Liver Clinic,
Manchester Royal Infirmary,
Manchester M13 9WL

- 1 Warnes TW. Treatment of primary biliary cirrhosis. *Seminars on Liver Disease* 1985;5:228-40.
- 2 Warnes TW, Smith A, Haboubi NY, Johnson PJ, Hunt I. A controlled trial of colchicine in primary biliary cirrhosis. *Hepatology* 1984;4:1022.
- 3 Christensen E, Neuberger J, Crowe J, et al. Beneficial effect of azathioprine and prediction of prognosis in primary biliary cirrhosis. *Gastroenterology* 1985;89:1084-91.
- 4 Warnes TW, Smith A, Lee FI, Haboubi NY, Johnson PJ, Hunt L. A controlled trial of colchicine in primary biliary cirrhosis. *J Hepatol* 1987;5:1-7.
- 5 Kaplan MM, Alling DW, Zimmerman HJ, et al. A prospective trial of colchicine for primary biliary cirrhosis. *N Engl J Med* 1986;315:1448-54.