We undertook in March 1992 an audit of lithium use during inpatient psychiatric treatment at Porirua Psychiatric Hospital, New Zealand. Clinical files were examined for 30 patients randomly selected from 62 patients prescribed lithium. Although lithium concentrations were monitored satisfactorily, pretreatment tests were inadequate in four patients whose lithium treatment was initiated during the past year, and monitoring tests were satisfactorily completed in only 16 patients. A patient with an ejection systolic murmur noted on physical examination had no electrocardiographic recording before starting lithium. Four patients had no physical examination, assessment of heart rate and rhythm, or measurement of serum creatinine concentration for four years during continuous lithium treatment; two of them had a raised serum creatinine concentration at the last measurement but creatinine clearance was not measured. No reason for use of lithium could be found in six patients' files. In only one of the 30 files was there documentation that the patient had been informed of the reason for the use of lithium and the need for regular salt and water intake. None contained documentation that the patient had been informed of toxic effects and what to do should these occur.

Another concern in the use of lithium is its potential to induce anomalies of the cardiac valves and major vessels if taken during the first trimester of pregnancy. Of the 10 female patients whose files were examined, four were premenopausal and had not undergone a sterilisation procedure. A negative pregnancy test had been obtained before starting lithium for three of the four, but in no file was there documentation that the possible effects of lithium during pregnancy or the need for adequate contraception and planning of future pregnancies had been discussed with the patient.

We encourage the use of published recommendations for monitoring lithium treatment¹ and endorse the need for documentation in the patient's file that important information about lithium has been given to and understood by the patient.

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Treating small cell lung cancer

EDITOR,—I disagree with Robert Souhami and believe that, though small cell lung cancer may often be a disseminated disease, an above average survival in patients who have undergone surgery of the primary tumour should be considered to be the result of case selection only if a large scale randomised trial has ruled out an effect from surgery.¹ Such a trial should be considered to be a priority in the search for progress in the treatment of small cell lung cancer.

When I worked in Malmö in the beginning of the 1980s three patients—more than 10% of an unselected group—had undergone resection of the primary tumour before they were given combination chemotherapy (F E von Eyben *et al*, third world conference on lung cancer, Tokyo, 17-20 May 1982).² They remained alive for a substantial period after the chemotherapy was stopped. A few years later I gave the same chemotherapy regimen to another series of patients not treated surgically, and despite an impressive objective response none of these patients was cured.

Long term follow up of patients treated only

with surgical resection of the primary tumour shows that a small proportion may be cured by this modality alone. Other studies of small series of patients who underwent surgery before chemotherapy show above average survival—that is, three times the survival associated with chemotherapy alone. Accordingly, a randomised trial should study the gain achieved by adding surgery to treatment with chemotherapy. As the few data available consistently suggest an improved survival from this combined approach, I await the results with great interest. Control of the primary tumour, which is often bulky, is an important aspect of treatment. The optimum role of surgical resection of the primary tumour among the many options for treating small cell lung cancer should be determined.

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Monitoring ambulatory blood pressure in general practice

EDITOR,—David J Webb and colleagues rightly warn against indiscriminate and uninformed use of ambulatory blood pressure monitoring in general practice and using instruments of doubtful accuracy.¹ Though true, this should not obscure the need for a better method of making important clinical decisions, often entailing lifelong treatment, than the present system of random clinic readings with a mercury sphygmomanometer.

The recommendations of the British Hypertension Society that patients with a diastolic pressure of 96-110 mm Hg should be asked to attend at least six times over three months and that those with an average pressure of ≥ 100 mm Hg should be treated² is not only expensive in terms of clinic time and patients' time but may still not exclude the fifth of patients whose blood pressure outside the surgery is normal but who react specifically to measurement in the clinic.³ Some patients fail to attend for follow up and have to be reminded. Others, whose blood pressure is just under the level for treatment, are recalled in a year's time; if their blood pressure is then found to be back in the problem range they start on another group of assessment visits. It is not surprising that many patients are found to be receiving treatment unnecessarily.⁴

Webb and colleagues point out that normal values for ambulatory monitoring have yet to be established. Much work needs to be done, but an informed guess on the basis of the largest population study so far reported is that a reasonable cut off for treatment would be a mean 24 hour diastolic blood pressure of 90 mm Hg.⁵ The assumptions made in such an estimate are not greater than those necessary in applying the results of clinical trials to current practice. Blood pressures obtained during ambulatory monitoring will eliminate "white coat hypertension" and are better predictors of target organ damage than casual measurements.⁶ They are also valuable in assessing the adequacy of control during treatment.⁷ Finally, they allow the identification of patients whose blood pressure does not drop during sleep, who are at increased cardiovascular risk.⁸

We have used an ambulatory monitor (Spacelabs 90207) in our hypertension clinic over the past year as an adjunct to traditional methods of assessment. It has given us a much clearer understanding of what it is we are treating and confidence that we are giving effective treatment to the right patients. It is

the most important advance since Riva Rocci introduced the mercury sphygmomanometer nearly 100 years ago.⁹

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Bovine spongiform encephalopathy and risk to health

EDITOR,—In response to Paul J Harrison and Gareth W Roberts's editorial on spongiform encephalopathies¹ E Coyle and Ian Harvey rightly suggest that systematic collection of data on Creutzfeldt-Jakob disease is essential to assess any potential risk from bovine spongiform encephalopathy.² This recommendation was originally made by the Southwood committee, and since May 1990 the Department of Health and the Scottish Home and Health Department have funded the systematic surveillance of Creutzfeldt-Jakob disease throughout the United Kingdom at the Western General Hospital in Edinburgh. As part of this inquiry a case-control study is being carried out on all cases with particular reference to dietary history, occupational history, and other putative risk factors.

There has been no evidence of any change in the epidemiological characteristics of Creutzfeldt-Jakob disease since the advent of bovine spongiform encephalopathy, and a paper on this will shortly be submitted for publication. Although the scientific evidence suggests that any risk to the human population from bovine spongiform encephalopathy is probably remote, the prolonged incubation period for the spongiform encephalopathies indicates that it will be many years before a risk to public health from bovine spongiform encephalopathy can be finally excluded.

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Fulminant hepatitis B in infants

EDITOR,—S V Beath and colleagues report on three infants with fulminant hepatitis B born to hepatitis B virus carrier mothers positive for antibody to hepatitis B e antigen (anti-HBe).¹ In our clinic recently two patients—one 3 months old

and the other 3½ months old—with fulminant hepatitis whose mothers had similar hepatitis B virus markers to those of the mothers in Beath and colleagues' report died within 36 hours despite intensive medical support.

Both of the patients were positive for hepatitis B surface antigen (HBsAg), antibody to hepatitis B core antigen (anti-HBc; IgM), and hepatitis B e antigen (HBeAg) and negative for antibody to HBsAg (anti-HBs), anti-HBc (IgG), and anti-HBe. The mothers were positive for HBsAg, anti-HBc (IgG), and anti-HBe and negative for anti-HBs, anti-HBc (IgM), and HBeAg.

Our cases together with those reported by Beath and colleagues support the possibility of fulminant hepatitis occurring in infants born to hepatitis B carrier mothers positive for anti-HBe.^{2,4} These infants' susceptibility to fulminant hepatitis is not clear, although negativity for HBeAg is associated with low infectivity. Recently, two reports have shown a point mutation on the pre-core region of hepatitis B virus and have suggested that, in the absence of HBeAg, cytotoxic T cells attack HBcAg on hepatocytes, causing fulminant hepatitis.^{5,6}

We agree with Beath and colleagues that all babies born to mothers carrying HBsAg should be vaccinated, and we believe that further research on these patients will help to explain the pathogenesis of fulminant hepatitis.

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Hypercalcaemia in patients receiving dialysis

EDITOR,—I Greaves and colleagues draw attention to the high incidence of hypercalcaemia in patients with chronic renal failure.¹ A colleague and I recently completed a prospective survey comparing the incidence of ionised hypercalcaemia (>1.35 mmol/l) in patients receiving haemodialysis and continuous ambulatory peritoneal dialysis.² When measured monthly the frequency of hypercalcaemia was 2.14 episodes per patient year for patients receiving continuous ambulatory peritoneal dialysis and 0.45 episodes per patient year for patients receiving haemodialysis. As Greaves and colleagues point out, the hypercalcaemia is usually asymptomatic. In our study of 66 patients only three of 69 detected episodes of hypercalcaemia were symptomatic.

In an effort to reduce hypercalcaemia in selected patients with continuous ambulatory peritoneal dialysis without resorting to use of aluminium containing phosphate binders we have used peritoneal dialysis solution containing a lower calcium concentration (1.25 mmol/l *v* the standard 1.62 mmol/l). In eight patients treated in this way for three months the serum calcium concentrations tended to fall (mean (SD) 1.43 (0.1) *v* 1.39 (0.04) mmol/l), although this did not achieve significance

(*p*=0.2; figure). Calcium containing phosphate binders (calcium carbonate) had to be reduced or stopped in five patients. It is to be hoped that the imminent introduction of calcium acetate may help alleviate the problem of hypercalcaemia related to phosphate control as this agent is less well absorbed³ and has greater phosphate binding ability than calcium carbonate.

Effect of changing calcium concentration in dialysis solution on serum ionised calcium concentration after three months' treatment. Vertical lines indicate means and SD

I agree entirely that serum calcium concentrations should be monitored closely in renal failure but was surprised to note that corrected calcium rather than ionised calcium concentration was being used. In our study as many as 5% of cases of hypercalcaemia would have been missed if we had relied on the formula used by Greaves and colleagues. I suggest that the ionised calcium concentration should be used to monitor patients with renal failure, particularly when intervention with calcium raising agents (such as 1,25-dihydroxycholecalciferol) is being considered.

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EDITOR,—I Greaves and colleagues' paper¹ highlights the dramatic increase in the incidence of hypercalcaemia among patients with renal disease (to 63% of all cases of hypercalcaemia) in one hospital over the past 10 years.² We believe, however, that this increase principally reflects the specialist nature of the hospital surveyed. Queen Elizabeth Hospital, Birmingham, is a major tertiary referral centre; it has large renal dialysis and transplantation units and also specialist radiotherapy and liver transplantation units but lacks an accident and emergency unit or outpatient clinics in general medicine and surgery. This point is made in the original report by Fiskin *et al* in 1980³ but omitted by Greaves and colleagues.

Our recently published survey of the causes of hypercalcaemia was based on Selly Oak Hospital, a typical district general hospital less than 3.2 km from the Queen Elizabeth Hospital.³ Of 121 consecutive patients with hypercalcaemia studied, six had a serum creatinine concentration greater

than 250 µmol/l; two had haematological malignancies and three had multiorgan failure. Primary hyperparathyroidism was the cause of hypercalcaemia in 63 (52%) patients; in 38 cases this diagnosis was made during our study. In comparison, Greaves and colleagues report only nine (5.5%) patients with hyperparathyroidism, all of whom had been admitted for elective parathyroidectomy. At Selly Oak Hospital the serum calcium concentration is measured routinely as part of a biochemical profile; the practice of discretionary testing at Queen Elizabeth Hospital may lead to failure to identify patients with asymptomatic hypercalcaemia.

Despite the difference in hospital setting between the two studies we have found it useful to compare the data obtained. The combined incidence of malignancy associated hypercalcaemia and hyperparathyroidism in the series of Greaves and colleagues was 3.4 patients/week, similar to our finding of 3.5 patients/week. The overall incidence of hypercalcaemia found by Greaves and colleagues was roughly three times that found by us (12.5 *v* 3.8 patients/week), the difference being entirely due to hypercalcaemia in patients with renal disease. This gives additional support to Greaves and colleagues' conclusion that hypercalcaemia has become an important complication of renal failure and transplantation and their management.

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Breast feeding in developing countries

EDITOR,—Jose C Martines and colleagues give some of the reasons why many breast feeding mothers in developing countries supplement their milk with water or teas from the first week of life.¹ Another important reason is the belief that such fluids have "inner cleansing" effects on their babies.

In some societies and cultures the baby's first drink is plain water. This may be a few teaspoonfuls or even finger tip quantities in drops. This initial drink is a symbol of welcoming the baby. Subsequently plain water or sugar and water are fed to the baby while the colostrum is discarded as unclean; breast feeding is started when the milk becomes clearer after a few days.^{2,3}

Water and teas are not regarded as supplements to breast milk in the real sense, being given at the beginning of the feed, between feeds, or at the end of the feed. This practice has stood the test of time, and it will not be easy for health professionals to persuade mothers against it. As one of the main reasons for discouraging the practice is diarrhoea in the babies due to dirty water, perhaps the emphasis should be on the importance of giving clean water to infants. Surely all pregnant women should be informed how and why they should breast feed. They should be taught that the inside of the baby is not dirty and that the passage of meconium is normal. If health professionals persuade rather than effectively educate these mothers not to give water to their babies the mothers might resort to other methods of cleaning the baby's inside with enemas, suppositories, and purges, which could be more dangerous.⁴

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