In this scheme a "new" NHS built from the skill and experience of the old one for major conditions and an independent health service for routine conditions would be entirely complementary to each other. Margaret Thatcher has the opportunity, ability, and courage to implement this sort of radical surgery before health care in Britain becomes an incurable lost cause, both for those delivering it and, even worse, for those needing it.

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Easing pain or hastening death?

SIR,—Dr Wilson's enjoyable personal view (17 October, p 994) evoked some familiar memories, but his account of dying Tom was a little disturbing. Presumably Tom's pain had been well controlled on regular oral analgesia, quite probably morphine although not necessarily so. The return of his pain was thus due to his now being too ill to swallow his analgesics, as shown by the advent of the death rattle. This can be expected. Perhaps a supply of oxycodone suppositories in the home would have allowed the family to maintain his comfort, or they might have been asked to telephone when Tom failed to take his medication.

Anyway, Tom was in pain, distressed by retained secretions, and diamorphine and hyoscine by injection were the appropriate response. The role of these drugs, as understood by Tom's wife, should have been that of relieving these symptoms, which they clearly did. But the passage may be read to imply that they also precipitated his death.

Many people need at least one injection before they die and one of these injections will be the last, but if the dose is in proportion to that of the oral drug given previously it should not cause the death. Diamorphine and hyoscine are effective drugs for the control of the symptoms Dr Wilson mentions. They should not be left until the patient is in extremis and clearly should not be given in order to advance the patient's death.

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Haemofiltration as a cause of electrolyte imbalance

SIR,—The report by Dr N D Barber and colleagues of hyponatraemia in patients undergoing haemofiltration (24 October, p 1025) will come as no surprise to those who have used this technique for several years for the treatment of acute renal failure. The loss of sodium and bicarbonate through these filters is very large, and before specially manufactured replacement fluids were available intravenous sodium bicarbonate and twice physiological saline was often necessary. Dr Barber's paper clearly indicates that the use of haemofiltration replacement fluid has not overcome these electrolyte problems, which occur largely as a result of the considerable quantity of fluid removed during continuous haemofiltration. Moreover, the patient reported on was not in renal failure. If haemofiltration is performed because of renal impairment very much larger volumes of fluid (up to 30 litres daily) have to be removed to avoid haemodialysis. Fluid balance and electrolyte problems are therefore commonplace.

Recently, a modification of haemofiltration has been described (N Schneider, R Geronemus, tenth international congress of nephrology, 1987) which prevents these electrolyte fluctuations

Plasma concentrations on final day of treatment

Case No	Sodium (mmol/l)	Bicarbonate (mmol/l)	Creatinine (μmol/l)
1	140	25	452
2	139	28	702
3	138	22	511
4	142	25	469
5	137	26	314

and also provides a very simple method of dialysis. This consists simply of running peritoneal dialysis fluid through the haemofiltrate compartment of the haemofilter, allowing diffusion of small molecules from the plasma.

We have used this technique on five patients in acute renal failure in the intensive care units of two district general hospitals, neither of which has a renal unit on site. Four patients were in renal failure after major surgery and one had a combination of tubular necrosis with gross myocardial failure. Ages ranged from 65 to 82 and all patients were unstable with low cardiac outputs. A Gambro AV50 haemofilter was used and blood access was obtained by a Scribner shunt in three patients and femoral cannulas in two. Intermittent peritoneal dialysis fluid (Dianal 1.36%, Travenol Ltd; sodium concentration 140 mmol/l) was passed through the haemofiltrate compartment at 1 litre/h and the volume of haemofiltrate removed was adjusted in the usual way according to the patients' needs by subtracting 1 litre from the hourly output from the haemofilter. Haemofiltrated volumes varied from 2 to 6 litres/day. Patients received this treatment for two to 14 days (total 48 days) and the plasma sodium concentration and venous bicarbonate are shown in the table.

This adaptation of continuous arteriovenous haemofiltration has several advantages. Firstly, plasma electrolyte concentration is rapidly equilibrated with that of standard dialysis fluids that are manufactured under a product licence. Moreover, electrolyte and fluid balance fluctuations are much less likely to occur as the total volumes removed are small. Secondly, it provides an extremely simple method of haemodialysis that requires neither nurses trained in dialysis nor renal physicians and can be undertaken in any intensive care unit. Finally, patients can now undergo dialysis without the haemodynamic problems associated with conventional haemodialysis or peritoneal access, which is so often made impossible by recent abdominal surgery.

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The natural course of gold nephropathy

SIR,—In their study on the natural course of gold nephropathy (26 September, p 745) Dr C L Hall and colleagues provided information on duration of proteinuria. Our patients are frequently concerned about this side effect and its slow resolution.

Unfortunately the study did not tell us the criteria for referral to a nephrologist for the problem of proteinuria during gold therapy, and it is therefore difficult to be certain that the data in the study are representative. It is also disappointing that the frequency of alternative diagnoses other than gold nephropathy is not given. In the cases of amyloid, analgesic nephropathy, and systemic lupus erythematosus, these would probably have altered the treatment of the underlying rheumatic problem.

For the patient once the anxiety about a renal

abnormality is overcome, the main problem is usually the loss of therapeutic efficacy. If the histological examination suggests a non-progressive renal lesion it would seem reasonable to start alternative treatment, penicillamine for instance, with regular urinary protein measurements. Other renal disease, as noted above, may cause changes in urinary protein indistinguishable from those related to drug toxicity. We therefore feel that the approach towards biopsy one year after development of proteinuria while taking gold might not always be in the patient's best interests and are surprised that the leading article by Dr A J Collins (26 September, p 739) on the subject omits any mention of biopsy.

Finally we are concerned about the unreferenced support in this leading article for systemic steroid treatment for heavy proteinuria in gold nephropathy. While steroids are used in nephrotic syndrome from other causes, this is to the best of our knowledge not the practice of most rheumatologists. Indeed steroid treatment may, in itself, produce locomotor problems—for example, avascular necrosis or osteoporosis—and there would need to be good evidence to justify high dose corticosteroid prescription in a self limiting condition.

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Measuring performance or balancing the budget

SIR,—We fear that both Dr John Wattis and Dr Nigel Tyre (17 October, p 1000) understate the hazards that are contained in the performance indicators currently proposed for the NHS.¹ Psychogeriatrics will be lost without trace. The issues are given cursory attention in a single paragraph which is common to both consultation papers 7 and 8.

Firstly, the very existence of special services is denied: "Psychogeriatric does not describe a speciality recognised by the Royal College of Psychiatrists.... Without the boundary of a recognised speciality it is difficult to focus on this aspect of service." It may be difficult, but surely it is not impossible. Every week job descriptions are issued requiring psychiatrists to take responsibility for psychogeriatric services, and more than 150 now devote themselves to this work.²

Secondly, the major elements of our activity are ignored. Good and effective psychogeriatric practice is characterised by liaison with other hospital specialists and involvement with patients in their own homes, rest homes, and so forth, alongside primary health care workers, social services, voluntary organisations, and families.³ During 1986 we were responsible for 251 liaison consultations, 543 home assessments, and 2731 follow up visits (including regular follow up visits to part III homes).

This information is not required by the Körner data set and thus will not be used to assess or describe our performance. Yet these activities are better measures of our service to patients than is the rate of admission to our beds, which is what Körner will provide. The interpretation of admission rates is difficult; more so when there is no information about liaison and community activity. Does a high rate indicate industry or failure to provide alternative services? Does a low rate reflect slothful indifference or success in providing alternatives?

Thirdly, the age and diagnostic characteristics of patients reviewed by psychogeriatric services are restricted: most services accept patients over 65