hyponatraemia. It would have been useful to know how many of Dr Walters and Dr Hallam's patients had liver or renal disease.

There are other considerations: corticosteroids can alter the relation between sodium and glucose, and in hyperosmolar non-ketotic diabetic syndrome, both plasma sodium and glucose concentrations are usually raised.[1 2]

It is difficult, if not impossible, to establish a causal relation between plasma sodium and glucose concentrations on the basis of randomly collected data. We agree that measuring blood glucose concentration in hyponatraemic patients would be useful and may unearth some previously undetected hyperglycaemic patients. In most cases, however, hyponatraemia is probably due to causes other than hyperglycaemia.

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## Hospices

SIR,—I suspect that most general practitioners would agree with Dr D C Hogg's views about terminal care (12 May, p 1453). We have a deep seated fear that terminal care is being specialised and institutionalised. The following facts might give some comfort.

A retrospective study over 36 months in a singlehanded rural practice with a population of 1830 identified 27 terminally ill patients, 18 of whom were managed in the community and nine in hospital. This is an inception rate for the terminally ill of 4·9/1000 at risk/year, and compares with a suggested rate of 6·8/1000/year in Levy's Glasgow study.[1] The mean length of terminal care in this practice was 5·3 weeks compared with 3·3 weeks suggested by Levy and 7·1 weeks by Reilly and Patten.[2]

Projecting this inception rate to the average district of 250 000, and assuming a bed occupancy of 75% and mean stay of 5·3 weeks, 166 full time terminal care beds would be needed for all to be cared for in hospital. The DHSS norm would give 12·5 terminal care beds for the average district so I think that those of us who see domiciliary care as the preferable option have little to fear. An additional reassurance would be for hospices to allow general practitioners clinical control if desired, subject to a house committee's jurisdiction.

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## Treatment of theophylline poisoning

SIR,—Dr A N Laggner and others (19 May, p 1497) have revived the debate about proving the efficacy of active treatment in severe poisoning.[1] Their paper and previous reports show that charcoal haemoperfusion lowers serum theophylline concentration.[2 3] The question remains of how much of the ingested dose is actually removed and whether this alters the final outcome? Only one report estimated the amount of theophylline removed, which was 58% of the ingested 1·25 g dose after 170 minutes of haemoperfusion.[4] Patients have survived massive theophylline overdose without haemoperfusion and without serious sequelae.[5]

Increased clearance of absorbed theophylline and inhibition of further absorption can be achieved by using oral activated charcoal.[6 7] Haemoperfusion may be indicated in cases of severe toxicity but as it requires skilled staff and specialised apparatus that is available in relatively few hospitals the unproved value is outweighed by the risks of transferring severely ill patients from hospitals without facilities. Doctors with no access to haemoperfusion who are caring for patients poisoned by theophylline should consider the use of oral activated charcoal and intensive care, and not suppose that haemoperfusion is obligatory or even a proved effective treatment.

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## Idiopathic ulceration of the small bowel

SIR,—Dr M J Glynn and others describe two patients with severe cardiac disease who developed idiopathic ulceration of the small bowel which caused bleeding and ultimately death (31 March, p 975). Both patients had heart failure, one from mitral incompetence and the other from cardiomyopathy. Intestinal ischaemia may have caused these multiple ulcers of the jejunoileum.

We have seen six patients in the last 11 years with non-specific ulceration of the small intestine. These patients were generally younger (mean age 44 years) than those described by Glynn (aged 57 and 71 years). Neither had any serious cardiovascular disease, although one man had taken Navidrex-K tablets for several months. Five of the six patients presented with gastrointestinal bleeding, but additionally the ulcers tended to heal with fibrosis, leading to obstructive symptoms. Histological examination showed non-specific ulceration which was often multiple and accompanied by pyloric metaplasia. There was no evidence of ischaemia. Local resection has been curative, except for one patient who developed recurrent ulceration of the bowel. These ulcers do not seem to carry the poor prognosis associated with ischaemic ulcers therefore.

Non-specific enteric ulceration is probably commoner than published reports suggest, and the diagnosis should be considered in any case of unexplained bleeding from the gastrointestinal tract.

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## Memory loss

SIR,—Dr Robert A Wood (12 May, p 1443) focuses mainly on biological factors relevant to the aetiology, pathogenesis, and presentation of memory loss but is less clear about management and alarmingly dismissive when discussing psychological and social factors. He states: "Hysterical amnesia is rare. ... Feigned amnesia is common. ... Hysteria resolves with time." The same can be said about resolution in uncomplicated myocardial infarction, most infections, and most non-displaced non-compound fractures. This is not to dismiss the importance of good management in these situations.

A hysterical memory loss represents a crisis for the patient and his family. Proper resolution and normal function are best ensured by appropriate psychological help, to encourage healthier and more adaptive ways of coping. In psychiatry, as in all branches of medicine, diagnosis is important in so far as it suggests appropriate management. In the age of the microcomputer management will probably be the main function of the clinician and algorithms will best be performed by the "microchip."

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## Carbohydrate antigens in serum of patients with carcinoma

SIR,—Professor J Holmgren and others (19 May, p 1479) describe the detection by monoclonal antibody of carbohydrate antigen CA 50 and report that serum concentrations of CA 50 are increased in a high proportion of patients suffering from various proliferative diseases. The authors suggest that the probable sources of CA 50 are the cancer cells themselves, and this may be true of other carbohydrate antigens as well. We have examined the carbohydrates expressed in oligosaccharide moieties of glycoproteins in breast cancer tissues from 60 patients. Lectins (proteins or glycoproteins of non-immune origin with a high affinity for selected carbohydrates) were used for detection. Paraffin sections of surgical specimens were incubated with lectin which was then identified by immunoperoxidase techniques.[1]

With *Helix pomatia* lectin (specific for N-acetyl-galactosaminyl residues) the staining pattern was significantly stronger ($p < 0.01$) in sections of cancers which had subsequently metastasised than in sections from cancers which had not. This appeared to be more clearcut than the difference in serum CA 50 concentrations between early and advanced breast cancers reported by Professor Holmgren's team. An indicator based on a serum assay, however, would be far more convenient clinically.