rather than vascular, is yet to be discovered, and these discoveries will lie in the hands of neurosurgeons who have learnt the appropriate microvascular skills.

CARYS M BANNISTER

Department of Neurological Surgery,
North Manchester General Hospital,
Manchester M8 6RB

On-demand analgesia equipment

SIR,—The recent paper by Dr W D White and others (21 July, p 166) concerning on-demand analgesia using a Janssen (JS10299) demand analgesia computer prompts us to comment on other equipment available for this purpose. Following an earlier paper describing a patient-controlled system for pain-relief during labour¹ we purchased the commercially available equipment—a Cardiff Palliator manufactured by Pye Dynamics Ltd. After extensive experience in the labour ward we concluded that there were three major design deficiencies with regard to patient management and safety.

Firstly, the dose of analgesic administered to the patient on demand is controlled by a three-place thumbwheel digital switch variable from 0 to 999 mg. This is accessible to anyone who can touch the machine. Since "if there is a button to be pressed, someone will press it" a safeguard was needed. Our choice of analgesic agent was diamorphine, as plasma concentrations achieved could be measured by immunoassay; the dose was 3 mg; and a change of only one space on the control—that is, from 003 to 013 mg—could have had serious consequences. We have placed a simple Perspex shield with three vertical slits over the whole switch. The settings can now be altered only by inserting a narrow metal peg kept with the machine. An equally important point is that the total dose indicator is reset by a simple push button below the dials. As this gives the only indication of how much drug the patient has received in the course of labour, any accidental resetting would wipe out the only record available. This button has been cut flush to the surface and resetting can be achieved only by using the metal peg. From the photograph available it appears that similar thumbwheel settings are used on the Janssen machine. What are the safeguards?

Secondly, there is no way of programming our machine so that a maximum total dose is not exceeded. The amount the patient is given per demand is dialled in, as is the time interval between demands. However, in a prolonged labour the patient could go on making repeated demands until the syringe is empty. If the syringe is refilled, the patient can make further demands. An added safeguard whereby a maximum permitted dose could not be exceeded was, in our opinion, vital. A small unit was designed by IRC; this has a two-place thumbwheel switch into which the maximum dose desired is dialled. Again the safety shield necessitates the use of the metal peg for setting. Once this preset total dose has been reached the syringe is halted and will continue to be used only if a larger dose is then dialled in. While the Janssen machine can apparently be set to a maximum dose delivered in any hour, can a maximum total dose also be set?

Thirdly, a successful demand for analgesia by the patient is indicated by a demand light coming on. This will continue to show until the minimum time interval dialled into the

machine is reached. For example, if the machine is set so that the patient can obtain only one dose of analgesic agent in 20 minutes then the light will show for this interval. However, an observer entering the labour room and seeing the light on does not know if it has been on for one minute or 19 minutes. Similarly, when the light is off it is not possible to know, other than by the patient's memory, whether it has been off for a minute or an hour. Such clinically useful information should be available—for example, a patient approaching full dilatation or requiring a general anaesthetic. We have designed an electronic timer unit with three digital displays. The first indicates total elapsed time from the first demand for analgesia, the second displays the time interval since the last demand, and the third gives the time interval of the demand before that. For women in labour these more detailed data are necessary for patient management. The Janssen machine apparently provides a printer, and this is a reasonable alternative. However, printers are prone to mechanical failure and usually more expensive to buy. How is information obtained if the printer fails?

When new equipment is described for patient care it would be useful, especially if it is to be patient controlled, if the authors would give comprehensive details of the equipment and their opinion on its suitability for the task it is claimed to do. In our opinion the commercial equipment we purchased had some deficiencies which we have corrected. However, the time and effort needed were considerable and we would hope to pass on these benefits to other users. The Janssen equipment may have got over some of these difficulties but it would be nice to be certain. And at what cost?

TOM LIND
R G WEATHERSTONE
I R CHAMBERS

MRC Human Reproduction Group,
Princess Mary Maternity Hospital,
Newcastle upon Tyne NE2 3BD

¹ Evans, J M, *et al*, *Lancet*, 1976, **1**, 17.

Splenectomy for massive splenomegaly

SIR,—We would like to comment and expand on some of the statements in your leading article (4 August, p 293). Massive splenomegaly is encountered as part of the clinical picture in a number of diseases as indicated, but it may in addition be the only presenting manifestation without the usual accompanying signs of the underlying disease. Recently we have seen two patients who complained only of abdominal pain and who had splenomegaly at least 16 cm below the costal margin. Neither had lymphadenopathy or other abnormalities on physical examination; chest radiographs were normal. Bone marrow biopsy revealed the microscopical appearances of Hodgkin's disease in one case and of non-Hodgkin's lymphoma in the other.

Plain abdominal radiographs, radionuclide scans, and ultrasound can confirm the diagnosis of splenomegaly but occasionally may give misleading results. In a recent case which we have seen, the spleen was thought to extend 17 cm below the costal margin; the appearances of massive splenomegaly were shown by each of these investigations. However, laparoscopy, an investigation you do not mention, revealed the organ to be the enlarged

left lobe of liver; subsequent histology showed cirrhosis. The spleen did not extend below the costal margin.

No mention is made of the use of radioisotope studies to determine the sites of red cell destruction and of extramedullary erythropoiesis in attempting to predict the value of splenectomy in patients with myelofibrosis. In a few such patients radiotherapy to the spleen can be beneficial, particularly when the patient is not fit for or refuses splenectomy; it does not preclude splenectomy at a later date.

In the final paragraph some complications of splenectomy are mentioned, but it is not clear that some of the comments refer to splenectomy in general while others refer to the complications of removing a massive spleen. Splenectomy for staging in Hodgkin's disease carries a mortality of less than 0.5%, rather than 15%.¹

Postsplenectomy thrombocytosis may be marked in degree and persistent for months or years; but the potential thrombotic complications depend on a number of factors, and few of these are defined at present. We have observed the changes in the platelet count after staging splenectomy in 25 patients with Hodgkin's disease. Thrombocytosis reached a maximum in the second week after splenectomy and subsided in one to two months, and in no case did the count exceed $1000 \times 10^9/l$. Thrombotic complications were not seen and anticoagulants were not given. In one series of 318 patients undergoing splenectomy for conditions other than myeloproliferative disease, postoperative thrombocytosis was not associated with a significantly increased risk of thromboembolism.² Hirsh and Dacie³ showed that the occurrence of severe postsplenectomy thrombocytosis depended on whether or not the patient remained anaemic after operation. It is clear that the complications and risks of splenectomy vary with the underlying disorder as well as with the size of the spleen.

DOUGLAS SHAW
G RANKEN TUDHOPE

Department of Pharmacology and Therapeutics,
Ninewells Hospital,
Dundee DD1 9SY

¹ Miller, J B, and Ultmann, J E, in *Hodgkin's Disease*, ed M J Lacher, p 44. New York, Wiley, 1976.

² Boxer, M A, Braun, J, and Ellman, L, *Archives of Surgery*, 1978, **113**, 808.

³ Hirsh, J, and Dacie, J V, *British Journal of Haematology*, 1966, **12**, 44.

SIR,—Your leading article (4 August, p 293) on splenectomy for massive splenomegaly presents a somewhat superficial view of this not uncommon clinical problem. In recent years many of the effects of the "big spleen" syndrome have been well described and it is now necessary to define more specifically those haematological problems which provide convincing evidence for splenectomy. For example, the anaemia associated with splenomegaly may be due to a combination of splenic red cell pooling, splenic haemolysis, and plasma volume expansion, mechanisms that can be assessed by isotopic techniques in most large hospitals. Surely the concept of hypersplenism, useful as it was 30 years ago, has now been replaced by a better understanding of many of the physiological and pathological results of splenic enlargement.

All would agree that while the haematological changes provide the strongest indications for splenectomy there are also powerful reasons for removing the very large spleen when it has become an intolerable and cumber-

some burden and the site of recurrent infarctions. Apart from the few cases of myelosclerosis with splenic haemopoiesis most big spleens are associated with many harmful effects of the "big spleen" syndrome notwithstanding the basic disease state. Provided that splenectomy is not delayed until it becomes a last-ditch procedure it is likely to improve the quality if not the quantity of life in a chronically ill patient.

There is also a very real place for splenectomy in the management of the patient with undiagnosed splenomegaly. Most large series of splenectomies contain a group of patients in whom the cause for the splenic enlargement was uncertain before operation. Usually histological examination of the excised spleen will reveal the nature of the basic disease but in some patients meticulous histological examination will still fail to provide a diagnosis, and long-term follow-up over a period of years may be necessary to detect the frequent development of haematological disease, usually lymphoma. With gross splenomegaly and an uncertain diagnosis many advantages may accrue from splenectomy, even considering the appreciable risks of the operation itself.

P J TOGHILL

Department of Medicine,
University Hospital,
Nottingham NG7 2UH

Suicide and attempted suicide

SIR,—The excellent article on "Suicide and attempted suicide" by Professor W H Trethowan (4 August, p 319) reminds us of the very great changes which have occurred not only in the law and the public's attitude to self-destruction but in the agents used in these forms of "communication between a person in distress and his environment," to use Professor Trethowan's own words. Few of these gestures, as he points out, prove fatal; but as the pharmaceutical scene has changed over the last 20 years so have the agents used in these gestures of despair.

We¹ reported in 1964 from St Stephen's Hospital, Fulham, on 126 patients (100 female, 26 male) admitted under our care into our general medical beds with suicidal gestures between 1955 and 1960. None died. One-third were considered straightforward and not suffering from psychiatric abnormalities; the other two-thirds were seen by the psychiatrist but only a small minority were transferred for psychiatric treatment and care. The agents most commonly used were barbiturates, as these were then the drugs most often present in the home drug cupboards; but now the more popular agents are the non-barbiturate sedatives and anxiolytics and the more popular analgesics. At the time of our study physical measures were very rarely used and gas fires in only 15%, mostly in the older age groups. Where such suicidal gestures were repeated the same agents, usually barbiturates, were often used again. M W Johns² has noted that hospital admissions due to acute barbiturate poisoning have decreased in England and Wales since 1965, roughly in parallel with barbiturate prescribing in the NHS, their place being taken by the less dangerous benzodiazepines and other agents.

It would seem that the most popular therapeutic agents of the day—that is, the ones in the family drug cupboard—will be the ones most likely to be used in these gestures

in the future as they have been in the past; but the fact that an agent, effective and non-toxic when used therapeutically in the correct dosage, can prove lethal when used for suicidal purposes in excessively high dosage, often with large amounts of alcohol, can hardly be set against it in terms of everyday therapeutics. What might be called pharmacological parasuicides, emotional gestures using whatever is in the home drug cupboard, occasionally prove fatal because of unanticipated side effects—for example, liver damage after ingestion of excessive amounts of paracetamol—but this risk will continue so long as drugs have side-effects and so long as the sad and distressed make these gestures of despair.

F DUDLEY HART

London W1

¹ Taylor, D J E, *et al*, *Practitioner*, 1964, **192**, 251.

² Johns, M W, *British Medical Journal*, 1977, **1**, 1128.

Wernicke's encephalopathy

SIR,—Your leading article (4 August, p 291) suggests that Wernicke's encephalopathy should be both treated early and ideally prevented. It is a rare complication of chronic alcoholism, only occurring in those patients who have a genetic abnormality of transketolase, which binds thiamine pyrophosphate less avidly than normal.¹ If all alcoholics were recognised and tested for this abnormal enzyme the development of the condition could be prevented in those vulnerable to it by regular supplements of thiamine, if necessary by the parenteral route, in much the same way as many schizophrenics receive depot neuroleptics.

You also suggest fortifying foodstuffs with thiamine. Vitamin depletion in alcoholics has been studied by Thomson,² who found many contributory mechanisms, including malnutrition, malabsorption, increased metabolic demands, and impaired utilisation. The only logical way round this would be to incorporate high doses of thiamine in alcoholic beverages before consumption, say 5 mg in a pint of beer, and 50 mg in a bottle of spirits. This could be done during manufacture or during inspection for duty purposes. After all, we have long accepted vitamin fortification of our breakfast cereal and margarine.

GARETH JONES

Whitchurch Hospital,
Cardiff CF4 7XB

¹ Blass, J P, and Gibson, G E, *New England Journal of Medicine*, 1977, **297**, 1367.

² Thomson, A D, *Clinics in Endocrinology and Metabolism*, 1978, **7**, 405.

SIR,—Your leading article (4 August, p 291) rightly draws attention to the frequency with which this eminently treatable but potentially fatal condition is missed. We are preparing a report of the results of a recent 24-month study of all patients referred to a single medical service screened for the presence of an "alcohol problem." In this period 73 such patients were identified, an incidence of 3%. Of these no fewer than nine had the Wernicke-Korsakoff syndrome, and none had been diagnosed before admission. Polyneuropathy was present in 10, tremor in 14, and cerebellar degeneration in three, with response to vitamin B complex in many instances.

A full clinical nutritional examination revealed virtually no other evidence of

deficiency. Liver involvement was frequent. A seven-day dietary recall showed a deficient thiamine intake in about 90% of these patients, a higher incidence of deficiency than for any other nutrient.

The close association with alcoholism, with its rising rate and especially high incidence in Scotland, gives cause for concern. Recognition by general practitioners of early thiamine deficiency in alcoholics might be life saving. You call for preventive measures—control of alcoholism and fortification of foodstuffs. The former is difficult to achieve but the latter in the form of increased fortification of bread, from which nearly one-third of our thiamine comes, would be simple, cheap, and safe.

D S McLAREN

Department of Physiology,
University of Edinburgh EH8 9AG

DANIEL H A BOYD

Leith Hospital,
Edinburgh EH6 6TH

Herpetic proctitis and sacral radiculomyelopathy in homosexual men

SIR,—Mr P L Samarasinghe and others (11 August, p 365) have drawn attention to the importance of herpetic proctitis in homosexual men. Severe anorectal herpes infections may be the commonest cause of acute urinary retention in young men. We in fact described two such cases in 1975.¹

The appearance of the rectal mucosa in the cases described by Mr Samarasinghe ranged from "hyperaemia and oedema to frank ulceration." *Chlamydia trachomatis* can cause exactly this picture of inflammatory rectal change² but, of course, not (to my knowledge) the associated neurological syndrome. In my series of homosexual men with herpetic proctitis, of which I hope to publish fuller details soon, ulceration was present on the anal mucosa up to the anorectal junction. Above this point—that is, on the rectal mucosa itself—no ulceration was present, but rather a hyperaemia that ranged from mild to severe, with areas of haemorrhage. Two asymptomatic excretors of virus (Mr Samarasinghe's "silent shedders") both had a normal anorectal mucosa as seen by the naked eye, but showed obvious hyperaemic change when the rectum was viewed and magnified through an operating microscope. The absence of ulceration on non-squamous-celled epithelium, however, is not a new observation: Coutts³ observed hyperaemia and haemorrhage only on the urethral mucosa in patients with herpetic urethritis as long ago as 1948.

DAVID GOLDMEIER

Department of Genitourinary Medicine,
University College Hospital,
London WC1E 6AU

¹ Goldmeier, D, Bateman, J R M, and Rodin, P, *British Medical Journal*, 1975, **1**, 425.

² Goldmeier, D, *A Study of Non-specific Proctitis in Homosexual Men*. MD thesis, University of London.

³ Coutts, W E, *British Journal of Venereal Diseases*, 1948, **28**, 205.

Relation between herpes simplex virus and carcinoma

SIR,—The paper on herpetic proctitis by Mr P L Samarasinghe and others (11 August, p 365), reporting urinary dysfunction as a complication, was of considerable interest and served also as a reminder of interrelation