"gamble with the nation's health." Should this not be a time when the medical profession should clearly abandon its traditional role of keeping aloof from discussions of the "drink problem" and take a clear stand?

M M GLATT

St Bernard's Hospital,
Southall, Middx.

[1] Glatt, M M, *British Journal of Addiction*, 1958, **55**, 51.
[2] Wilson, G B, *British Journal of Inebriety*, 1942, **39**, 76.
[3] Glatt, M M, *Alcoholism a Social Disease*, London, English University Press, 1975.
[4] De Lint, J, *British Journal of Addiction*, 1975, **70**, 3.
[5] *Daily Telegraph*, 28 February, 1976, p 8.

SIR,—I am a medical student, but at present I am a patient in hospital. I have just been offered an alcoholic beverage as a non-medicinal nightcap supplied, presumably, on the NHS (I declined the offer.)

I would like to question whether this is in accordance with your recent leading article (14 February, p 359) which protested against the increasing availability of alcohol to the public and in which you suggested that "there should be a moratorium on all measures which make drink more available." Is a hospital ward now a place where alcohol consumption is to be encouraged?

B H WILKINS

Orpington, Kent

Beta-blockers in anxiety and stress

SIR,—Your recent leading article on beta-blockers in anxiety and stress (21 February, p 415) suggests there is little to choose between the various available compounds in this respect.

With regard to selectivity, it is known that the various beta-blockers differ in their pharmacology. The action of beta-blockers in relieving the somatic manifestations of anxiety may result from peripheral blockade of sympathetically mediated symptoms. The widespread distribution of such symptoms would suggest that the most appropriate and effective agent for this purpose would be the most non-selective compound available and that propranolol may then be the drug of choice.

In this context there may be a rational basis on which to choose between the available beta-blockers—a minor point of disagreement with your article.

KENNETH G F BENTON

Wessex Renal Unit,
St Mary's Hospital,
Portsmouth

Diagnosis of intracranial haemorrhage

SIR,—Your leading article on this subject (28 February, p 483) touches on the management of head injuries, pointing out that "in those fortunate hospitals with a [computerised axial-tomography (CAT)] scanner the precise diagnosis may now be made on admission, and if necessary a patient may be in the operating theatre in about an hour." This is true, but what is of at least equal importance is that the patient with a closed head injury with no sign of intracranial haemorrhage could be put under curare-like relaxants and hyperventilation on admission and therefore given the best chance of survival with minimal morbidity, with no need to monitor him on clinical neurological findings, since repeated CAT scanning will reveal the development of acute extradural haematoma or other surgically remediable lesion.

An open head injury can be safely treated by hyperventilation, as found with missile injuries in Belfast, but as the method of hyperventilation masks the clinical signs of progressive intracranial haemorrhage in a closed head injury, such as dilating pupil, development of hemiparesis, etc, the method is unsafe and the advantages are outweighed by the risks. We consider that the immediate accessibility of CAT is essential for any neurosurgical unit and that for a busy head injury unit not to have immediate and continuous access to a scanner demonstrates failure to provide optimum treatment for patients.

COLIN GLEADHILL

Department of Neurological Surgery,
Royal Victoria Hospital,
Belfast

Pseudo-obstruction due to clonidine

SIR,—We read with interest the case report by Drs R Bear and K Steer (24 January, p 197) and would like to record a similar experience relating to a patient who was subjected to two laparotomies.

A 52-year old woman presented in September 1970 with visual failure due to malignant hypertension. Blood-pressure control with standard antihypertensive medication remained suboptimal until the addition in November 1971 of clonidine in a dose varying between 450 and 900 μg daily. Serum creatinine at that time was 203 μmol/l (2·3 mg/100 ml). She was readmitted in November 1972 complaining of abdominal pain and severe constipation of recent onset. Barium enema examination revealed a tender, spastic caecum but no other abnormality. Treatment for her abdominal symptoms was symptomatic. In October 1973 a laparotomy was performed with a provisional diagnosis of intestinal obstruction due to a polyp-induced intussusception. At operation a few adhesions and a dilated large bowel full of faeces were found but no evidence of organic obstruction.

During the next two years she had several readmissions to hospital because of progressive deterioration of renal function, serum creatinine rising to 636 μmol/l (7·2 mg/100 ml) and severe painful constipation. Her blood pressure was reasonably well controlled with clonidine, beta-adrenergic blocking drugs, and loop diuretics. In November 1975 emergency laparotomy was undertaken for suspected intestinal obstruction when she presented with severe abdominal pain, distension, and radiological fluid levels, though bowel sounds never disappeared. The findings at operation were identical with those of October 1973.

In January 1976 clonidine therapy was gradually withdrawn; bowel function returned to normal within three to five days but blood-pressure control was difficult in spite of the addition of the maximum tolerated doses of vasodilator drugs.

Experience with clonidine at the cardiovascular clinic and in the wards of this hospital extends to over 800 patients treated during the past eight years.[1] We regard clonidine as a very effective drug for the treatment of all grades of hypertension, from mild to malignant, including hypertension of pregnancy and the control of hypertensive emergencies. Side effects are usually minimal and well-tolerated, the commonest being drowsiness, dryness of the mouth, and insomnia. Rebound hypertension following the sudden cessation of clonidine therapy occurs in some subjects and can be promptly controlled provided the mechanism is recognised.

In the previous communication from this hospital it was stated that "constipation occurred occasionally but ileus was not seen." Now that pseudo-obstruction due to clonidine has been recognised more attention will have to be given to this potentially lethal complication. The exact mechanism remains uncertain; alteration of central autonomic visceral control seems more likely than peripheral parasympathetic blockade similar to the now virtually forgotten mecamylamine ileus.

G E BAUER
K J HELLESTRAND

Sydney Hospital,
Sydney, New South Wales

[1] Raftos, J, *et al*, *Medical Journal of Australia*, 1973, **1**, 786.

Propranolol schedule before paradoxical hypertension

SIR,—It would be interesting to know the interval between the doses of propranolol and the increase in daily total dosage that preceded the unexpected rise in blood pressure observed by Dr I Blum and his colleagues (13 December, p 623) in eight psychotic patients who were given 600-5000 mg daily.

Transient hypertension occurred in our first case of schizophrenia treated with propranolol[1] when we followed the same regimen, giving propranolol every three hours around the clock[2] and increasing the dose by 400-800 mg daily. Toxic effects led us to modify this regimen. The drug was given twice daily because the biological effects of propranolol persist longer than the pharmacological half life of 2-3 hours.[3] Further, we raised the dose by some 40-80 mg/day—that is, at about one-tenth of the rate of increase described by Dr Blum and his colleagues. In 55 patients with schizophrenia no further case of hypertension was seen, and other toxic effects became rarer and milder with the modified regimen.[4]

It would thus be valuable to know if Dr Blum and his colleagues found that the conditions that preceded paradoxical hypertension with high doses of propranolol also included giving it every few hours and raising the dose steeply. If so this rare complication of propranolol treatment may be not only treatable (with alpha-blockade) but also preventable.

N J YORKSTON

Bethlem Royal Hospital,
Beckenham, Kent

C W H HAVARD

Royal Free Hospital,
London NW3

S A ZAKI
JUDITH THEMEN

Friern Hospital,
London N11

[1] Yorkston, N J, *et al*, *British Medical Journal*, 1974, **4**, 633.
[2] Steiner, M, *et al*, *Kupat Holim Yearbook*, 1972, **2**, 201.
[3] Carruthers, S G, *et al*, *British Medical Journal*, 1973, **2**, 177.
[4] Yorkston, N J, *et al*, *European Journal of Clinical Pharmacology*. In press.

Toxic effects of propranolol on the heart

SIR,—In relation to the extensive use of beta-adrenoceptor blocking agents the reported cases of suicide attempts with these drugs are relatively rare. With propranolol Drs W Wermut and M Wójcicki (15 September 1973,