the large number of studies which have shown that there is a greatly increased chance of subsequent suicide after any overdose.

In general, the patient brought into hospital is resuscitated in the casualty department and subsequently admitted to whichever ward is receiving patients on that particular day. From that point onwards he becomes a nuisance, occupying a bed which may be required for a medical or surgical admission. In the light of current thinking, a psychiatric assessment now becomes an urgent necessity.

It is true that, in most cases, the routine psychological data can be ascertained by any well-trained doctor. But this is not enough. It is not merely psychosocial maladjustment that has precipitated the overdose. It is not merely depression or the inability to cope which has led to the deliberate self-inflicted trauma. Frequently it is the result of a mental torment, the interpretation of which cannot be found by reference to any textbook of psychiatry. Has the enormity of the individual problem really been considered? When the necessary evaluation has been made, how can the social and/or psychiatric problems which led to the drastic action in the first place be remedied and how can a subsequent suicide attempt be prevented?

It is essential that after recovery from the poisoning a full psychiatric and psychosocial assessment is made of each patient—a procedure which, if carried out properly, must take up a considerable amount of time and energy. It is essential that, where applicable, a trained social worker investigates the social background and environment of each patient and institutes whatever assistance and continued support can be given in this area. It is essential that each patient be given the opportunity of a follow-up psychiatric appointment, that he be supported, counselled, and guided through the difficult weeks, months, or even years of an unhappy mental state in a traumatic life situation. The use of psychopharmacological drugs, not always a necessity, must be considered, prescribed, and monitored where required; and thus, hopefully, with all the forces of psychotherapeutic help brought to bear, a further suicide attempt is avoided.

If the vigilance or chance discovery by a friend or spouse were not to take place, if the vigorous action of trained casualty staff were not implemented in time, and moreover if the full implications of an attempted suicide were not seriously considered, the completed suicide rate would increase by several thousands each year. The subsequent management of patients after a suicide attempt is a full-time specialty, and until this is recognised as such the death rate from repeated attempts will continue to remain high.

In the hospital in which I work I may see from eight to 12 attempted suicides each week. This means, for inpatients alone, a very high potential work load, in addition to an extremely busy outpatient follow-up service. Very few hospitals without such a specialist service can spare a member of the medical staff for such duties or for such a length of time. The proper assessment and the subsequent treatment of a patient who has attempted suicide are not the spare time job of a junior house officer. Together with the support and follow-up that are required, it can be the full-time work of at least one specialist consultant psychiatrist in any busy urban hospital.

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SIR,—The authoritative letter from the presidents of the Colleges of Physicians and Psychiatrists (25 October, p 1141) is premature. Its proposals are based largely on one study—that of Gardner *et al*¹ in Cambridge. Gardner and his colleagues did show that in the circumstances of their study there was no difference in the rate of subsequent repetition of self-poisoning in patients initially assessed either by senior psychiatrists on a rota or by specially trained medical teams. The study referred only to self-poisoning patients already admitted to medical wards. Readers and the two college presidents may not be aware that, subsequent to the introduction of Gardner's experiment in Cambridge several years ago, a smaller and smaller proportion of self-poisoning patients arriving at casualty departments are actually being admitted. According to Gardner (address to clinical meeting of Royal College of Psychiatrists, November 1979), the figure now stands at about 50%. We have no evidence yet about how the results of the sort of assessment which can be performed in the casualty department compare with those of a more careful assessment in the ward. It is also possible that hospitalisation in itself reduces the risk of repetition.²

Criticisms have been made elsewhere^{3,4} of the suggestion that there may be a widespread change to medical teams taking over initial psychiatric assessment of self-poisoning patients. The principal arguments are that this does not necessarily lead to the freeing of psychiatric resources, because of the high psychiatric referral rates associated with such a practice; and that it has not been satisfactorily demonstrated that junior physicians can adequately identify the minority of patients who might benefit most from psychiatric treatment. In the study by Blake and Bramble,⁵ also on medically admitted patients, it was found that junior doctors failed to identify 19% of those patients who would have been recommended for inpatient treatment by psychiatrists.

I would suggest that a summing up of the available evidence should lead to the Scottish verdict of "Not proven" and a call for more evidence to be produced.

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¹ Gardner R, *et al*. *Br Med J* 1978;iii:1392-4.

² Kennedy P. *Br Med J* 1972;iv:255-7.

³ Dyer JAT. *Br Med J* 1979;iii:205.

⁴ Kennedy P, Oswald I. *Br J Psychiat* 1979;135:92.

⁵ Blake DR, Bramble MG. *Br Med J* 1979;ii:1763.

Alcohol and the emergency medical service

SIR,—The recent paper by Dr S Holt and others (6 September, p 638) has again highlighted the impact of alcohol abuse on the medical service. Resulting injuries have certain unique features: (a) they are eminently preventable; (b) most often innocent individuals bear the brunt of the consequent morbidity and mortality; (c) as a number of drunk drivers are unlicensed and uninsured, their victims have no redress; (d) the drunks cause disruption in hospitals, upsetting other patients and being non-cooperative with hospital staff; (e) offenders are often habitual.

Certain measures may help to reduce the incidence of this epidemic and the cost of

treatment. Offenders should be made to pay for the full cost of treatment of themselves and their victims. There is already a precedent, as drivers pay for the emergency treatment of victims of road traffic accidents. Inebriated pedestrians should be held responsible for accidents occurring in their drunken states. Finally, excessive indulgence leading to accidents occurs most often in public houses. Since drivers must be insured to drive it will be a mere extension of the statute to require those who patronise public houses or who run them to purchase some form of third party cover against the possible consequences of their pastime.

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Prevention or cure for stress-induced gastrointestinal bleeding?

SIR,—The author of your leading article (6 September, p 631) seems unaware of most of the clinical and experimental data on H₂-receptor antagonists and stress ulceration and has drawn conclusions that are seriously misleading. Studies in animals demonstrate that metiamide or cimetidine *does* prevent gastric mucosal injury induced by cold, restraint, or haemorrhage shock,¹⁻³ although the mechanism may be independent of acid inhibition.⁴ The relevance of animal models to human stress ulcer is, of course, questionable and evidence from clinical trials is more important.

The report you quote by McDougall *et al* on patients with fulminant hepatic failure is not "the only controlled study." In patients with severe head injury a placebo-controlled trial convincingly indicated that cimetidine prevented acute gastrointestinal bleeding.⁵ In this study five of 26 patients receiving cimetidine (300 mg every four hours) suffered gastrointestinal bleeding and required a total of 11 units of blood. In contrast 18 of 24 control patients bled and required 41 units of blood. Three patients in the control group required operation, two for bleeding and one for a perforated gastric ulcer. Thus in these two categories of high-risk patients cimetidine has at least reduced the need for blood transfusions—a by no means limitless resource. Although the decrease in operation rate or mortality does not reach statistical significance in either study the trends are favourable and should not be ignored.

Comparisons between the prophylactic effectiveness of cimetidine with antacids are important but the study of Priebe *et al*, the cornerstone of your leading article, has limitations and has been severely criticised.⁶ Haemorrhage was defined mainly by occult blood testing of nasogastric aspirates. Although seven cimetidine-treated patients showed "evidence of bleeding" frank blood was found in only two patients and transfusion requirements were trivial: one patient received 1000 ml and 2000 ml was divided among three other patients. The need for transfusion in these three patients and hence both the statistical and the clinical significance of the authors' conclusions seem questionable. No attempt was made to determine the site of bleeding to support their argument. The problem of "acute gastrointestinal bleeding" in their patients seems very small—perhaps because both antacid and cimetidine were effective. This is the conclusion drawn by the authors of a further randomised double-blind comparison of antacid and cimetidine prophylaxis in patients with burns to more than 30% of their body surface.⁷ Neither treatment group suffered major haemorrhage or perforation, but endoscopy was carried out in all patients and showed significantly fewer gastric erosions in patients receiving cimetidine.

A comparison of the combination of antacids and cimetidine with antacids alone (suggested by your leading article) has already been reported in the treatment of patients with established acute mucosal bleeding.⁸ Haemostasis was achieved earlier with cimetidine but it is noteworthy that bleeding stopped within 48 hours in only 33% of either treatment group. This fact—that established acute gastrointestinal haemorrhage may be difficult to control—and the disastrous results of surgical intervention in the few patients in whom medical treatment is unsuccessful contradict your final sanguine conclusion that “treatment may reasonably be reserved for those in whom bleeding occurs.”

It is true that prophylaxis should be reserved for high-risk patients—those with severe burns, head injuries or multiple injuries, and major operations, especially when these conditions are complicated by hypotension, jaundice, sepsis, renal failure, or severe respiratory insufficiency. Cimetidine infused slowly intravenously has fewer side effects than antacids, may be used even when the gut is not functioning, and is more convenient. For the most effective prophylaxis intragastric pH should be maintained above 4 and antacids may occasionally need to be added to cimetidine to achieve this.

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¹ Safaie Shirazi S, Foster LD, Hardy BM. *Gastroenterology* 1976;**71**:421-5.

² Strauss RJ, Stein TA, Wise L. *Am J Surg* 1978;**135**:120.

³ Levine BA, Sirinek KR, McLeod CG, Teegarden DK, Pruitt BA. *Surg Gynecol Obstet* 1979;**148**:399-402.

⁴ Owen DAA, Parsons ME, Farrington HE, Blakemore R. *Gastroenterology* 1979;**77**:979-85.

⁵ Halloran LG, Zfass AM, Gayle WE, Wheeler CB, Miller JD. *Am J Surg* 1980;**139**:44-8.

⁶ Spenney JG, Hirschowitz BI. *N Engl J Med* 1980;**303**:108.

⁷ McElwee HP, Sirinek KR, Levine BA. *Surgery* 1979;**86**:620-4.

⁸ Teres J, Bordas JM, Rimola A, Bru C, Rodes J. *Dig Dis Sci* 1980;**25**:92-6.

* *We are aware of the experimental evidence in animals suggesting that H₂-receptor antagonists will prevent gastric mucosal injury in certain circumstances, but other studies quoted in our leading article indicate that adequate antacid titration is likely to be more effective. In any case, as Dr Hetzel would agree, experimental work is of doubtful relevance to human stress-induced gastrointestinal bleeding.

The study of Halloran *et al* that he quotes does show convincing evidence that stress-induced gastrointestinal bleeding is reduced by cimetidine. Whether this is relevant to other patients at risk of stress-induced bleeding is uncertain; for particular mechanisms, notably gastric acid hypersecretion, are probably important in patients with severe head injury. Furthermore, bleeding was not prevented in a fifth of those patients with head injury treated with cimetidine, and vigorous antacid therapy might well be more effective. We did not cite the other two controlled studies mentioned by Dr Hetzel because they were inconclusive. The first (by McElwee *et al*) was conducted by the United States Army Institute of Surgical Research, a group that had used antacid prophylaxis routinely in over 750 patients with burns, of whom only one had had major gastrointestinal bleeding. Their controlled trial contained just 27 patients, only 13 of whom received cimetidine—hardly sufficient for a confident conclusion. In the second study (by Teres *et*

al) only 15 ml of antacid was given two hourly—a quarter or less of the dose used by Priebe *et al*—and intragastric pH levels were not reported. This low dose of antacid will not maintain a gastric pH above 4 and would not be expected to influence acute mucosal lesions. The study of Priebe *et al* has indeed been criticised but the criticisms have been answered by the authors.¹ The fact that they have found the problem of stress-induced gastrointestinal bleeding to be small, even in previous studies when some patients received no prophylaxis, supports our view that routine prophylactic treatment is unnecessary.

Possibly, then, cimetidine will reduce the incidence of stress-induced gastrointestinal bleeding in certain specific circumstances, notably in acute hepatic failure and severe head injury; but vigorous antacid therapy may well be more effective. We have no evidence that the eventual outcome will be influenced by either form of treatment. In other conditions in which stress-induced gastrointestinal bleeding occurs the mechanisms may differ, and current evidence indicates that vigorous antacid treatment prevents bleeding whereas the efficacy of cimetidine is uncertain. Cimetidine may be convenient to administer, but its intravenous use may be hazardous, particularly in patients with hepatic and renal failure,² and we need larger studies to establish its safety in such patients. As we said in our leading article, present evidence is not sufficient to justify the widespread use of prophylactic cimetidine in intensive care units, though clearly further trials are needed.—Ed, *BMJ*.

¹ Priebe HJ, Skillman JJ, Bushnell LS, Silen W. *N Engl J Med* 1980;**303**:109.

² Blain PG. *Adverse Drug Reaction Bulletin* 1980; No 83:300-4.

Passing a nasogastric tube

SIR,—I have read your valuable article on passing a nasogastric tube (25 October, p 1128) and should like to make two suggestions.

It is certainly helpful and permissible in most circumstances for the patient to drink while the tube is being passed—any fluid so taken can be aspirated once the tube enters the stomach. It is not possible, however, for a patient to drink continually from a glass and for the tube to be passed at the same time. The two processes get in the way of each other. The problem, however, is solved if a feeder with a spout is used.

There is no doubt also that securing the tube to the nose is more comfortable for the patient than sticking it to the cheek; but, alas, many patients have greasy noses and some of the newer adhesive plasters (Micropore, Dermicel) float off during the night. Old-fashioned zinc oxide strapping is much safer and stays put much longer with the greasiest of noses.

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SIR,—We were pleased to see the article by Mr A Tucker and Mrs Jennifer Lewis on passing a nasogastric tube (25 October, p 1128). Many doctors have little practice, as often the intubation is done by the nursing staff. However, we disagree with the general use of large-bore nasogastric tubes for feeding. There are situations where a large nasogastric tube is necessary—for example, to establish that the

stomach is emptying properly in acutely ill patients—but under normal circumstances it is preferable to pass a fine tube for the comfort of the patient.

Some of the fine-bore tubes can remain in situ for a number of weeks without deterioration, whereas the Ryle's tubes should be changed after four to five days as there is a greater risk of nasopharyngeal ulceration and gastric erosions. Many fine-bore tubes come with an introducing wire or a mercury bolus for ease in passing; they are also radio-opaque, unlike Ryle's tubes, so the position can be checked by x-ray examination.

The majority of modern tube feeds, whether prepared or made up in a diet kitchen, will go down the finest tube (1 mm) without any risk of blockage. It is not always necessary to use sterile water as the first feed. A patient is also able to begin to eat or drink with a fine tube in place without any undue discomfort.

Nasogastric feeding is a traumatic experience for the patient. Care and consideration should be given in selecting and introducing any tube.

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Parity and breast cancer

SIR,—Following our report (9 August, p 419) of an association between parity and age at presentation of breast cancer, Dr Edward Trapido has described an analysis of 611 breast cancer patients (18 October, p 1072) in whom mean age at presentation of parous women (57.26 years) was not significantly less than that of nulliparous women (58.86 years; $p=0.15$). He also noted that among parous women mean age at presentation did not decrease with increasing parity, and concluded that his data did not support our study. In contrast, our attention has been drawn by Dr P Juret to his study¹ of 790 patients, in which it was found that parous women did present at significantly younger ages than nulliparous women, as in our sample. We believe that these seemingly conflicting results can be reconciled on the basis of our proposal that (a) breast cancer is usually initiated in early adult life, and (b) in a proportion of parous women tumour growth is accelerated by pregnancy.

Dr Trapido infers from our hypothesis that there should be a linear inverse relationship between parity and age at presentation. This is not the case, nor was it found in our study. Consider a large random sample of women with breast carcinomas. By our hypothesis they can be divided into nulliparous women (group A), parous women whose tumours are accelerated by pregnancy (group B), and parous women whose tumours are not influenced by pregnancy (group C). We would predict that group C would present at the same mean age as group A. Group B would present at a younger mean age; they would also be less likely to attain high parity before manifesting the disease. Among women of high parity, therefore, group C would be overrepresented and group B underrepresented. Mean age at presentation would thus be lowest in women of intermediate parity; in higher-parity groups the mean age would rise towards that for nulliparous women but should not exceed it. These trends are