the prison medical service. Critics have recognised that many of its problems arise from the appalling state of the prisons themselves, and reform of the health services alone may thus achieve little. Problems have also arisen in prisons from the rundown of the NHS mental hospitals and the unwillingness of some psychiatric teams to accept mentally disordered offenders into their facilities. Although many have criticised the prison doctors, they have also acknowledged that some do an excellent job that other doctors are unwilling to do and that special skills are needed for practising medicine in prisons. Then, there have been doubts about the ability of the NHS to provide for the needs of prisoners.⁹ Would a cash limited district health authority give much priority to the needs of prisoners? It seems unlikely.

For all these reasons—combined with inertia—the policy has been to soldier on with attempts at piecemeal internal reform. In particular, a committee of the Royal College of Physicians recommended improving the training of prison doctors rather along the lines that have been successful in raising the quality of emergency medicine.^{10 11}

But internal reform is clearly not enough. There is little or no evidence of improvement and much of continuing problems. The European committee found many defects with the medical services and recommended that “all prison medical officers should belong to the wider health community.”¹¹ This message is supported by the chief inspector of prisons, who in his annual report produced yet again a catalogue of criticisms of the medical services full of phrases like “We can only reiterate that . . .” and “We continue to note the apparent lack of . . .”¹² Little or nothing improves, and his final conclusion was: “The standard of medical care afforded to prisoners falls far short of that provided in the community.” The nearest that we have to an “outcome” measure for the prison medical service is the suicide rate, and this, sadly, has continued to rise.^{13 14}

It was presumably considerations like these—combined with the present government’s enthusiasm for the market—that led the efficiency scrutiny to propose radical reform of the prison medical service. It suggested that the service should stop providing health care directly and instead purchase it from the NHS. The prison medical service would thus become a purchasing authority. The efficiency scrutiny was published in the autumn of 1990, and the Home Office is making heavy weather of implementation. It seems to be trying to turn an apple into an orange by stealth, a policy doomed to failure.

Why not go further? Why should the purchasing not also be done by the NHS? Firstly, combinations of district health

authorities and family health service authorities are busy developing purchasing skills in a way that the prison medical service would be hard put to match. Secondly, the purchasers have access to the public health skills that have been lacking in prisons. Thirdly, the purchasing authorities should be able to purchase for prisoners at the same time as purchasing for the rest of the community and so get a broader range of services at better prices. What can be achieved by NHS staff working in the prisons is illustrated in the paper on p 152: diabetologists from Liverpool, who in an earlier paper identified serious problems in delivering diabetic care in prison, were able to abolish serious diabetic instabilities and improve glycaemic control in diabetic prisoners by running a fortnightly clinic in the prison.

The funds for prison health care should probably continue to come through the Home Office rather than the Department of Health so that prisoners do not have to compete directly with other groups in the community for restricted resources. But purchasing might produce savings because the evidence is that prison medical services have been inefficient.¹² It would be necessary to develop special purchasing skills for prisons, and purchasing authorities would need to study prison health care needs and ensure that some staff devote time especially to prisons. Similarly, the purchasing authorities would need to write requirements for special training into provider contracts. But in the end prisoners have advantages over the general public in that the chief inspector, whose staff include people with medical skills, and Sir Donald’s committee will continue to watch the quality of the health services available to prisoners. Such wholesale reform would be unlikely to reduce quality and might improve it dramatically.

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Drug treatment for acute upper gastrointestinal bleeding

Works in selected subgroups of patients

Acute gastrointestinal bleeding from the oesophagus, stomach, or proximal duodenum results in about 25 000 admissions to British hospitals each year, with one in five patients requiring emergency surgery and one in 10 dying from the effects of severe blood loss.¹ Morbidity and mortality are particularly high in elderly patients, those who require large transfusions, and those with continuing or recurrent bleeding. Despite advances in diagnostic endoscopy and treatment of peptic ulcer disease, mortality from acute upper gastrointestinal bleeding has remained stubbornly high.

Admission to highly specialised units, close collaboration between gastrointestinal physicians and surgeons, and early surgical intervention for high risk patients can appreciably reduce mortality to 2-4%. The quest for therapeutic agents that reduce transfusion requirements, operation rates, and overall mortality has continued unabated over the past two decades.

An acidic environment impairs platelet function and haemostasis, and therefore reducing the secretion of gastric acid should reduce bleeding as well as encouraging healing.

Meta-analysis of the use of H₂ receptor antagonists in patients with bleeding suggests that the drugs marginally reduce surgical referral and mortality, maximum benefit being shown in patients with bleeding gastric ulcers.³ Meta-analysis, however, is subject to publication bias and may not take into account studies with negative results that have not been published. In a recent large trial of over 1000 patients with upper gastrointestinal bleeding intravenous famotidine failed to influence the transfusion requirements, operative rate, or mortality.⁴

Further evidence that the suppression of acid fails to influence the immediate outcome of acute upper gastrointestinal bleeding has been provided by the study of Daneshmend and coworkers published in this issue (p 143).⁵ In this large double blind, placebo controlled trial of 1147 patients admitted with haematemesis with or without melaena the authors concluded that 24 hours of intravenous omeprazole followed by three days of oral treatment did not influence rates of bleeding or operation or mortality. As the doses of omeprazole that were used have been shown virtually to abolish acid secretion the study provides compelling evidence that luminal acid has little influence on the course of acute bleeding from the upper gastrointestinal tract. The overall mortality in this trial was low at 6% but may have been influenced by the exclusion criteria and close supervision by a highly qualified and motivated team.

Another approach to managing patients with acute bleeding has been to reduce fibrinolysis at the ulcer site. Plasminogen activators are present in gastroduodenal mucosa, with high concentrations of plasmin in the gastric venous effluent of patients with peptic ulcer. Tranexamic acid is a plasminogen inhibitor that has the added advantage of reducing the fibrinolytic action of luminal pepsin. A study of 256 patients showed that tranexamic acid more than halved the overall mortality from acute upper gastrointestinal bleeding.² Unfortunately, the two groups were not well matched, with more older patients, anaemia, and hypotension and a higher transfusion requirement in the placebo group. A subsequent meta-analysis of six randomised, double blind, placebo controlled trials of 1267 patients showed a significant reduction in operation rate (30%) and mortality (40%) in those treated with tranexamic acid.⁶ The outcome of this meta-analysis was, however, biased by the results of the study quoted above,² which was by far the largest. The value of tranexamic acid therefore remains uncertain.

Somatostatin and its analogue, octreotide, have many actions on gut function, but inhibition of acid secretion and reduction of splanchnic blood flow are particularly pertinent to the treatment of upper gastrointestinal bleeding. In unselected patients admitted with acute bleeding, however, neither agent has been conclusively shown to influence rebleeding or operative or overall mortality.^{7,8}

Although these studies provide little optimism for using drugs in treating acute bleeding, they may have expected the impossible. Acute upper gastrointestinal haemorrhage results from various pathological conditions such as peptic ulcer, gastroduodenitis, oesophagitis, varices, and cancer. That a single therapeutic agent should not consistently reduce bleeding from such an array of disorders should not surprise. More encouraging results have been obtained from studies of selected subgroups. For more than 30 years vasopressin has been successfully used to stop variceal bleeding, with a

success rate of between 35% and 60%. Comparative trials have shown that somatostatin is as good as, if not better than, vasopressin and has fewer side effects.⁹ Two recent placebo controlled trials found that intravenous somatostatin was better than placebo in controlling variceal haemorrhage, although it did not influence the overall mortality.^{10,11}

β Blockers such as propranolol have no role in managing acute gastrointestinal bleeding but do reduce subsequent recurrence of bleeding from varices and gastropathy caused by portal hypertension.^{12,13} A recent meta-analysis of 26 clinical trials of β blockers in preventing variceal bleeding concluded that these agents significantly reduced bleeding and overall mortality related to bleeding.¹² This effect, however, applied only to the secondary prevention of haemorrhage from varices, with borderline benefit being shown for primary prevention in patients with large varices. Acid suppression has also been found to be valuable in the subgroup with bleeding from "stress ulcer disease." Recent evidence has created some concern that reducing gastric acidity in these patients may increase the risk of nosocomial pneumonia.¹⁴ The cytoprotective agent sucralfate seems as effective as acid inhibition in reducing bleeding and mortality in such patients and does not increase the incidence of pneumonia. Such observations are, however, based on few patients, and further studies are awaited with interest.

On current evidence "blanket" treatment with a single drug therefore seems to have little role in managing acute upper gastrointestinal bleeding. In selected subgroups such as patients with varices, portal hypertensive gastropathy, and stress ulcer disease, however, drug treatment may significantly reduce morbidity and mortality.

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