An audit of hospital admissions for acute upper gastrointestinal haemorrhage

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SUMMARY
A retrospective survey was made of all 189 patients admitted with acute upper gastrointestinal haemorrhage to the Belfast City Hospital in one year. The commonest single reason for admission was peptic ulcer disease, but this was lower than in other published series from the United Kingdom. Overall mortality was 4.8%. The majority of patients did not require either blood transfusion or surgery. There may be potential benefits of endoscopic haemostatic techniques to deal with this condition.

INTRODUCTION
Acute upper gastrointestinal haemorrhage is a potentially serious condition and is a common cause of hospital admission. Surveys from Oxford,1 Birmingham2 and Nottingham3 indicate an annual admission rate of around 50 per 100,000 population — a figure which has changed very little in the past 30 years. Overall mortality rates in most large series have remained fairly constant at around 10% although the proportion of elderly patients has greatly increased.2 Deaths are virtually confined to this elderly group. Mortality figures vary: in a recent study from a district general hospital the mortality was 15%4 whereas various studies from teaching hospitals have reported rates of less than 10%.2 Differences in the underlying causes of bleeding or in management strategies may contribute to this variability.

Surgery has been the main intervention used to secure haemostasis. The role of early operation is debatable — in one study this reduced mortality5 but in another seemed to increase mortality.6 The deployment of resources may also influence outcome — an Australian hospital has claimed that management in a special multi-disciplinary unit can reduce the mortality from acute upper gastrointestinal bleeding.7 We have undertaken an audit of adult admissions to the Belfast City Hospital with acute upper gastrointestinal haemorrhage. We were interested to assess our performance and to consider ways in which management might be improved, particularly with regard to the potential role of endoscopic haemostatic techniques.

METHODS
A retrospective survey was made of the records of all patients admitted with acute gastrointestinal haemorrhage to the Belfast City Hospital during the year 1983.
Upper gastrointestinal bleeding

Cases were identified from the medical endoscopy unit records, theatre records of endoscopies and operations, Hospital Activity Analysis coded discharge summaries and the hospital mortality records for the year. Details of diagnosis, management and outcome were identified.

RESULTS

During the year there were 189 admissions for upper gastrointestinal haemorrhage. Of these 114 had presented with haematemesis, 53 with melaena and 22 with both these features. There were 128 males and 61 females; the age range was 15–98 years, mean 51·5 years. One male patient was admitted twice during the year. The cause of bleeding is shown in Table I. This was determined by endoscopy shortly after admission in 161 cases. The overall mean interval between admission and endoscopy was 2·8 days. Barium meal was the method of investigation in 8 patients. No cause was found for bleeding in 17 patients and a further 20 were not investigated.

| Number | Percentage |
|--------|------------|
| Oesophagitis/ulcer | 21 | 11·0% |
| Oesophageal varices | 3 | 1·5% |
| Mallory-Weiss tear | 7 | 3·5% |
| Gastritis/gastric erosions | 26 | 14·0% |
| Simple gastric ulcer | 13 | 7·0% |
| Gastric tumours | 3 | 1·5% |
| Duodenal ulcer | 48 | 25·5% |
| Duodenitis | 8 | 4·0% |
| More than one possible site | 23 | 12·0% |
| Undetermined site(s) | 17 | 9·0% |
| Not investigated | 20 | 10·5% |

In 24 cases (12·5%) there was more than one possible site of blood loss, but no evidence of bleeding to indicate the likely source at the time of endoscopy. This group included 10 patients who had duodenal ulceration but also other possible causes of bleeding. In 32·5% of cases the bleeding was unequivocally due to peptic ulceration.

The overall mortality was 4·8%. Details of the nine patients who died are shown in Table II. Two died from uncontrolled haemorrhage, a 54-year-old male with an oesophageal carcinoma and an 83-year-old female who refused investigation or active management. The others died as a result of associated complicating factors including hepatic encephalopathy, bronchopneumonia and heart failure.

Twenty-two patients underwent surgical operation. Thirteen were necessary to achieve haemostasis (six patients with duodenal ulcer, two with gastric ulcer, three with gastric erosions and one each with oesophageal varices and a Mallory-Weiss tear). Three operations were for perforated duodenal ulcer and six were
elective procedures (three for duodenal ulcer, one for gastric ulcer with gastrocolic fistula, one for carcinoma of the stomach and one for leiomyoma of the stomach). There were three deaths in these patients.

### Table II

*Details of patients who died following admission for acute upper gastrointestinal haemorrhage*

| Sex | Age | Cause of bleeding                          | Cause of death                          |
|-----|-----|-------------------------------------------|-----------------------------------------|
| M   | 51  | Oesophageal varices                       | Post-operative encephalopathy          |
| M   | 54  | Oesophageal carcinoma                     | Uncontrolled bleeding                   |
| M   | 55  | Gastro-colic fistula from gastric ulcer   | Post-operative bronchopneumonia         |
| F   | 60  | Oesophageal varices                       | Encephalopathy                          |
| M   | 69  | Duodenal ulcer                            | Post-operative bronchopneumonia         |
| M   | 77  | Oesophageal carcinoma                     | Bronchopneumonia                        |
| M   | 77  | Duodenal ulcer                            | Congestive heart failure                |
| F   | 83  | Gastric carcinoma                         | Following transfusion cardiac arrest    |
| F   | 83  | Acute haematemesis. Refused investigation and treatment | Uncontrolled bleeding                   |

There were 61 patients in whom peptic ulcer disease alone was identified as the cause of haemorrhage. One patient with severe haemorrhage from a gastric ulcer and two patients with haemorrhage and perforation underwent surgery without initial endoscopy. Of the remaining 58 the number with endoscopically determined active bleeding and those with and without stigmata of recent haemorrhage are shown in Table III. Surgery was used to achieve haemostasis in seven of these patients. One patient with a vigorously bleeding ulcer underwent early surgery. In one with an actively oozing ulcer and history of chronic peptic ulcer disease, early surgery was performed because the patient refused to accept

### Table III

*Initial endoscopic appearance of peptic ulcers and their outcomes*

|                  | Further bleeding | Surgery |
|------------------|------------------|---------|
| Not bleeding     |                  |         |
| No stigmata of recent haemorrhage | 38  3  1 |
| Bleeding         |                  |         |
| With stigmata of recent haemorrhage (Ooze, visible vessel, blood clot, blood ‘spot’) | 19*  6  5* |

*Includes one patient who underwent early surgery to avoid the need for blood transfusion.*
blood transfusion on religious grounds. More ulcers with stigmata of recent haemorrhage re-bled compared to ulcers with no stigmata (6 out of 18 compared to 3 out of 38: p < 0.02, Chi squared analysis). The overall mortality from bleeding peptic ulcer was three out of 61 (5%). Surgery was performed in 15 (25%) and death occurred in two of these, a mortality rate of 14%.

**DISCUSSION**

The aims of this study were to assess our performance and consider ways in which we might improve. In terms of the overall mortality this series compares favourably with that in any other published study. In part at least this is likely to be due to the relatively low proportion of serious cases. About two-thirds of patients did not require blood transfusion and the majority overall (88%) required only conservative management. Many of the cases in this study were due to mucosal inflammation and/or erosion in the oesophagus, stomach or duodenum. The proportion of those admitted with haemorrhage from peptic ulcer disease (32.5%) was lower than in other published UK series, in which figures range from 44% to 58%. Nevertheless the mortality figures specifically for peptic ulcer cases were similar to the overall figure. This has been achieved without the setting up of a single specialist unit for gastrointestinal bleeding. Patients are admitted to both medical and surgical wards which liaise and co-operate as necessary. The operation rate of 25% for peptic ulcers is lower than in some hospitals which have a more aggressive policy but our more conservative approach, borne out by the results, is more in keeping with recent recommendations.

In seeking ways to improve our management one aspect to consider is the rather excessive proportion of minor cases which might not be the most efficient use of resources. We could try to be more selective about admissions, but the diagnosis and prognosis of patients at initial assessment without investigation is fraught with difficulties. A policy of early endoscopy in all cases would enable accurate diagnosis and possibly the early discharge of patients with minor problems.

An area of potential improvement is the use of non-surgical (endoscopic) methods aimed at securing haemostasis. These include laser photoagulation, various diathermic devices, heater probes and most recently injection of adrenaline. These techniques have mostly been used in an effort to control active bleeding from ulcers and to prevent re-bleeding from ulcers which are not actively bleeding. In the latter group various signs of recent haemorrhage have been shown to be associated with an increased risk of re-bleeding: a visible vessel, blood spots, adherent blood clot or active oozing from an ulcer crater. Our experience has confirmed that ulcers with these features tend to re-bleed more frequently than ulcers which do not have such stigmata. Eight patients admitted with bleeding from peptic ulcer required surgery to achieve haemostasis, and one died. If effective haemostasis had been achieved by an endoscopic method the need for further transfusion and operation might have been avoided. One patient with an actively bleeding single gastric erosion required surgery, and might also have been managed endoscopically. If such a device was used at initial endoscopy in those ulcer patients who were considered at risk of further bleeding, 20 patients in our series would have been treated. This might have avoided the further bleeding which did occur in seven and led to surgery in five. It might also have avoided surgery in the patient with an oozing ulcer who would not accept blood transfusion. The potential use of this technique at initial endoscopy is therefore 20 out of 58 ulcers (34%), and 11% of admissions for upper gastrointestinal bleeding.
It seems unlikely that any endoscopic technique would be effective or applicable in all cases. It would only be justifiable if the overall benefits outweighed any risk. The number of potential cases in one year is small, but important. If we are to improve our management, endoscopic haemostasis does offer that possibility and it is our intention actively to explore this area. It may at least be possible to reduce blood transfusion requirements and the need for surgery.

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