# Upper gastrointestinal bleeding in an African setting

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Patients with acute upper gastrointestinal bleeding present complex problems in medical management. In developed countries, mortality has not necessarily been helped by access to high technology or improvements in surgical technique [1,2]. While endoscopic information exists as to causes of gastrointestinal haemorrhage in several African countries [3,4] few studies have systematically examined treatment and outcome in this context. This prospective study sought to assess causes, treatment and outcome of patients presenting with acute upper gastrointestinal haemorrhage in Harare, Zimbabwe. We also tried to evaluate predictors of outcome in the light of recent improvements of definitions in this field [5].

## Methods and materials

Successive adult patients presenting with acute upper gastrointestinal bleeding to the two teaching hospitals in Harare, Zimbabwe, between September 1984 and July 1985 were studied. Entry criteria, definitions and the data collection form conformed to were those set out by the World Organisation of Gastroenterology survey of upper gastrointestinal bleeding [5,6]. To enter the study, patients must have suffered haematemesis and/or melaena evident to the medical or nursing staff or which had taken place within 10 days of admission. Patients who presented primarily with anaemia due to repeated minor bleeding were excluded from the study.

**Table 1.** Outcome.

| Diagnosis: principal site of bleeding | Admissions (%) | Settled No. | Percent of diagnostic category (%) | Ongoing bleeding No. | Rebleeding No. | Surgery No. | Death No. |
|---|---|---|---|---|---|---|---|
| *Underwent endoscopy* | | | | | | | |
| Duodenal ulcer | 46 (35) | 32 | (70) | 5 | 7 | 13 | 2 |
| Oesophageal varices | 36 (27) | 21 | (58) | 9 | 6 | 1 | 5 |
| Gastritis | 16 (12) | 15 | (94) | 1 | — | — | 1 |
| Gastric ulcer | 6 (5) | 2 | (33) | 1 | — | 2 | 1 |
| Mallory Weiss tears | 4 (3) | 4 | (100) | — | — | — | — |
| Gastric polyps | 5 (4) | 4 | (80) | 1 | — | 2 | — |
| CA stomach | 3 (2) | 2 | (67) | — | — | 1 | 2 |
| Pyloric canal ulcer | 2 (2) | 2 | (100) | — | — | — | — |
| No cause found | 4 (3) | 4 | (100) | — | — | — | — |
| *No endoscopy* | | | | | | | |
| Death before investigation | 7 (6) | — | (0) | 6 | — | — | 7 |
| Discharged | 2 (2) | 2 | (100) | — | — | — | — |
| TOTALS | 131 | 88 | | 22 | 13 | 19 | 18 |
| (% Overall outcome) | | | (67%) | (17%) | (10%) | (15%) | (13%) |

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**Table 2.** Multiple lesions at endoscopy.

|  | No. |
|---|---|
| Duodenal ulcer and prepyloric ulcer | 7 |
| Duodenal ulcer and gastric ulcer | 2 |
| Oesophageal varices and gastritis | 2 |
| Oesophageal varices and gastric polyps | 2 |
| Total | 13 |

Data were collected using a form based on that used in the World Organisation of Gastroenterology survey [6]. Initial recordings of data were made by the admitting doctor and substantiated by one of the investigating team as soon as possible after admission. Statistical analysis was by the Chi squared test of association and Students' $t$ test.

## Results

During the study period, 126 patients accounted for 131 separate admissions for upper gastrointestinal bleeding. Ninety-eight patients (88 per cent) were male and 28 patients (12 per cent) female, the male to female ratio being 3.5 to 1. The mean age of the whole group was 40 years. Only 15 patients (12 per cent) were 60 years or older, and 62 patients (49 per cent) were younger than 40.

Endoscopic findings and outcome are shown in Tables 1 and 2. Where multiple sites of bleeding were found, an attempt was made to identify the principal site of bleeding. In 88 (67 per cent) of the 131 admissions, bleeding settled on simple conservative management. There was evidence of ongoing bleeding in 22 instances (17 per cent) and of rebleeding after haemorrhage had initially settled in a further 13 instances (10 per cent). Seven patients died before endoscopy and 19 patients (15 per cent) underwent surgery.

In considering factors that predicted outcome, all the patients were divided into two groups: those in whom bleeding settled uneventfully (the 'good outcome' group) and those who experienced continued bleeding, rebleeding, underwent emergency surgery or who died during the admission (the 'poor outcome' group). Good outcome patients had on admission significantly higher blood pressures and haemoglobin values, lower initial heart rates and were significantly younger than those in the poor outcome group (Table 3).

**Table 3.** Initial blood pressures, heart rates, haemoglobin values, and ages in poor and good outcome patients.

|  | Poor outcome | | Good outcome | | | |
|---|---|---|---|---|---|---|
|  | $\overline{x}$ | s.d. | $\overline{x}$ | s.d. | $t$ statistic | $p$ |
| Systolic BP | 106 | ± 19 | 120 | ± 24 | 3.24 | 0.001 |
| Heart rate | 101 | ± 19 | 91 | ± 16 | 1.96 | 0.01 |
| Haemoglobin | 7.4 | ± 3.6 | 9.2 | ± 4.2 | 2.34 | 0.02 |
| Age (years) | 44 | ± 14 | 37 | ± 15 | 2.64 | 0.01 |

When individual factors were considered the correlation with poor outcome was greatest for initial heart rates higher than 109 per minute, followed in descending order of risk by haemoglobin values of less than 5g/dl, systolic blood pressures less than 100 mmHg and age of 50 years or more (Table 4). Nonetheless, 26 (60 per cent) of the poor outcome group had either none or only one of the above risk factors.

**Table 4.** Risk factors for poor outcome: relative risks and 95 per cent confidence intervals for 4 risk factors.

| Factor | Relative risk (95% limits) | $\overline{x}^2$ | $p$ |
|---|---|---|---|
| Heart rate $\geq$ 110/min | 2.39 (1.51–3.77) | 13.96 | <0.001 |
| Haemoglobin <5 g/dl | 2.1 (1.22–3.6) | 7.32 | <0.001 |
| Systolic BP <100 mmHg | 1.99 (1.19–3.35) | 6.82 | <0.01 |
| Age $\geq$ 50 years | 1.93 (1.21–3.09) | 7.52 | <0.01 |

## Presentation and outcome by disease category: patients undergoing endoscopy

### Duodenal ulceration

Duodenal ulceration accounted for 35 per cent of admissions (46) and for 39 per cent of patients who underwent endoscopy; 35 patients were male and 11 female, with a male to female ratio of 3.1 to 1. The mean age of this group (39 years) was not significantly different from those with other sites of bleeding (40.5 years). Thirteen patients (28 per cent of the group) underwent surgery. Indications for operation were rebleeding (seven), ongoing bleeding (five) and concurrent perforation (one). Truncal vagotomy with pyloroplasty was performed in eight patients, and partial gastrectomy in five; bleeding vessels were ligated in five. Two patients with bleeding duodenal ulcers died, both postoperatively; one patient died shortly after a second operation was performed because of continued bleeding despite vagotomy and pyloroplasty; in the other, death was attributed to postoperative pulmonary embolism.

In eight of the 14 patients with a 'poor outcome' a clot or vessel was visible in the ulcer base, whereas this was found in only two patients in whom bleeding settled on conservative management ($\overline{x}^2 = 14.83$ $p<0.001$). The initial systolic blood pressures of patients with duodenal ulceration and a poor outcome ($\overline{x} = 107$ mmHg) were significantly lower than those of good outcome patients ($\overline{x} = 121$ mmHg) ($t$ statistic = 2.41 $p<0.05$) but there were no significant differences in initial heart rate or haemoglobin values between good and poor outcome patients with duodenal ulceration.

### Oesophageal varices

Oesophageal varices accounted for 36 episodes of bleeding in 32 patients, (25 males and seven females) during the study period. In addition, four patients with varices were found to have other types of bleeding: gastritis in two cases and polyps in two. No patient had concurrent varices and peptic ulceration. The mean age of patients with bleeding oesophageal varices was 42 years.

In 21 admissions for varices (58 per cent), bleeding settled with conservative management. Nine patients suffered continuous bleeding and six had rebleeding. Only one patient underwent surgery, a stapling procedure.

There were five deaths, all from haemorrhagic shock. Four deaths occurred within the first 24 hours of admission. Vasopressin infusions had been used in four of the patients with the addition of oesophageal tamponade in two.

### Gastritis

Gastritis accounted for 16 admissions. A history of recent ingestion of salicylates was obtained in 10 instances and of alcohol in eight instances. In one case erosions were associated with a gastric bezoar. Two patients had a poor outcome: one was found to be pancytopenic and subsequently died and another continued bleeding for 60 hours, but then settled.

### Other causes of bleeding

Bleeding gastric ulcers accounted for six per cent of admissions. One of the patients in this group had an associated cardiomyopathy and died from heart failure, and a further two patients underwent surgery. Gastric polyps were found in two admissions and Mallory–Weiss tears in four cases. There were three cases of gastric carcinoma, two dying in hospital. Pyloric canal ulcers without associated duodenal ulceration were found in two patients.

In five cases endoscopy did not reveal cause of bleeding; in four of these the bleeding settled and did not recur but one patient was subsequently found to have a gastric leiomyoma at surgery.

### Mortality

There were 18 deaths in this study, giving an overall mortality rate of 13 per cent.

Seven patients died before endoscopy could be performed. Consent for postmortem was only obtained in one instance, a patient with varices and micronodular cirrhosis. Two patients died with clinical evidence of portal hypertension and were thought to have suffered variceal bleeding. Two patients had previously documented peptic ulcer disease. The remaining two patients died of haemorrhagic shock from an undiagnosed source.

In considering the 18 deaths, the causes of death were classified as follows.

1. *Haemorrhagic shock (56 per cent)* accounted for 10 deaths, six occurring after variceal bleeds, and a further two in patients who died before endoscopy but had evidence of portal hypertension.
2. *Post operative (11 per cent)*. Two patients died after operations for duodenal ulcer disease.
3. *Inevitable deaths (17 per cent)*. Included here are two deaths from gastric carcinoma and one in a patient with marrow aplasia who was found to have gastric erosions.

4. *Associated disease (11 per cent)*. Two patients died of heart failure. In one this was probably related to over-vigorous transfusion.
5. *Others (5 per cent)*. One patient died of aspiration pneumonia after an episode of haematemesis while he was obtunded.

### Discussion

In this study we sought to evaluate whether initial clinical data could predict which patients with upper gastrointestinal bleeding were at risk for continued bleeding, rebleeding or death. Selection of such a subgroup is particularly important in a developing country where health resources may not permit specialised or intensive care for all patients with gastrointestinal haemorrhage.

We found that tachycardia, hypotension a low initial haemoglobin and an age greater than 50 all correlated with a poor outcome. These results are similar to those found in other settings [1,7] and do suggest criteria for special care. Nonetheless, it should be pointed out that more than half the patients with a poor outcome in this series had none or only one of the above risk factors, implying no clear subgroup could be defined that could be safely left without intensive monitoring.

Endoscopic data also had a bearing on outcome in this series. Varices carried by far the worst prognosis, and, in common with other series [8], a visible vessel or clot at the base of a duodenal ulcer predicted continued bleeding or rebleeding.

The mortality rate of 12 per cent in our patients is slightly higher than has been recorded in Western series of upper gastrointestinal bleeding [2,9]. In comparing our figures with those from developed countries differences in both age structure and in causes of bleeding need to be considered.

The mean age of patients in this series was 40: only 12 per cent were 60 years or older. This age structure parallels that found in other studies of gastrointestinal bleeding in Africa [3,4] but is strikingly different from recent experiences in Europe where almost half the patients are over 60 years old [2]. Mortality in upper gastrointestinal bleeding is known to be related to increasing age [2,10]. Thus, if age alone is considered, mortality from upper gastrointestinal bleeding in Zimbabwe should be much lower than in the West.

Endoscopy revealed that duodenal ulceration was the commonest cause of haemorrhage in this series: this accords with previous endoscopic information from Zimbabwe [4] and helps refute the notion that duodenal ulceration is an uncommon cause of haematemesis here [11].

In comparison with the previous endoscopic study in Zimbabwe [4], we found a higher proportion of oesophageal varices (27 vs 15 per cent) and a lower percentage of gastric ulcers (5 vs 17 per cent). The difference may be due to selection bias, as the criteria for entry into the previous study are not known. Nonetheless, the proportion of varices in our study is much higher than in a comparable study of sequential hospital admissions for

gastrointestinal haemorrhage in Birmingham (27 vs 6 per cent) [2]. This clearly has bearing on mortality which is known to be much higher for oesophageal varices than for other causes of bleeding [1]. The differences in ages and causes of bleeding between Zimbabwean and British patients are reflected in the different causes of death in the two groups. Most of our patients died of haemorrhagic shock: many of these deaths occurred within 24 hours of admission, and in more than half bleeding originated from oesophageal varices. By contrast, most deaths in the Birmingham study were due to thrombotic vascular disease, postoperative complications or the consequences of malignancy [2]. This implies that improved treatment of haemorrhagic shock, particularly with bleeding from oesophageal varices, is the most important measure required to reduce immediate mortality from gastrointestinal haemorrhage in our setting.

Fortunately, endoscopic injection sclerotherapy, which complements the use of vasopressin and balloon tamponade, has become available at the two teaching hospitals in Harare since September 1985 and this will go some way in arresting continuing variceal bleeding. In the long term, however, we have to concentrate on the prevention of variceal re-bleeding in our patients. A trial, which has demonstrated the efficacy of propranolol in the preven-tion of variceal re-bleeding in patients with non-cirrhotic portal fibrosis has just been completed by one of us [12]. Another trial, evaluating sclerotherapy in the prevention of variceal rebleeding in our patients, is in progress.

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# Book Review

*Philosophical Medical Ethics* by Raanon Gillon. John Wiley & Sons, 1986, 189 pages, Price £8.50.

This book brings together in a convenient form the series of articles which Raanon Gillon contributed to the *British Medical Journal* during 1985 and 1986. They were presumably commissioned, for who would write on medical ethics without being asked to do so? That is not a rhetorical question, since practising doctors have largely preferred doing their job to drawing out its ethical implications; and the consequent vacuum has been, perhaps too easily, filled by retired medical pundits like myself, or by philosophers, theologians, lawyers and lay ethicists whose contribution is perhaps best summarised as 'docta ignorantia'. Speaking personally, as I tend to do, I give a warm welcome to this book, at two levels. At the lowest pragmatic level, it makes an elegant substitute for a tattered collection of *BMJ* pages. More seriously, it brings to an area of great public and professional interest the considered judgements of someone with almost unique qualifications for the task. Gillon has formal academic qualifications both in philosophy and in medicine, so he has learnt the ground rules of both disciplines; he is engaged in the day to day practice of medicine; and as Editor of the *Journal of Medical Ethics* he is well placed to know where practical problems are arising, even in fields of medicine in which he is not himself engaged.

Gillon is well aware of the general aloofness of doctors to the formal treatment of ethical problems, and in his preface he is considerate enough to give guidance to 'pontificators', 'abstainers', and 'sceptics' on the different ways in which they might begin to tackle the subject matter of his book. He also apologises to doctors for its complexity, and to philosophers for its simplicity, which suggests to me that for any doctor with a degree of interest in moral philosophy the level may be about right. I can only say that the original serial publication and now the collected book complement one another for the reader, the one allowing small and reasonably addictive doses, the other making cross-reference easier, much assisted by a good index.

Normally, a review should summarise the content of a book. On this occasion, the book itself is a summary of a vast literature. Instead, may I try to say why I believe it is important that a doubly qualified doctor should be writing on this matter rather than a lawyer, theologian, or sociologist. Lawyers deal with *crimes,* often in a fashion which needs another lawyer to interpret it. Theologians deal with *sins,* and their claims to speak for higher authority are often tinged with subjectivism. Sociologists deal with *follies,* not always in a sensible way. Unethical medical behaviour is rarely a crime recognised by law; it may or may not be a sin; and it is often compounded by folly, even if that is not its root. At the risk of being taxed with medical chauvinism, I believe that experience of the problems in practice, especially by someone with a philsophical framework as well, is a more valuable base for writing on these matters, than the partial insights of those concerned professionally with crimes, sins and follies. Of course, lawyers, theologians and sociologists can make a legitimate contribution to the debate; but as a summary for the practising doctor I commend the present offering.

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