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HAEMATEMESIS AND MELAENA*

WITH SPECIAL REFERENCE TO BLEEDING PEPTIC ULCER

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Haematemesis and melaena have been the cause of much controversy throughout this century. The dangers and advantages of morphine, blood transfusion, early feeding, and surgery have stirred the leaders of the medical and surgical professions to vigorous debate. Diametrically opposed views have continued to flourish and have been nurtured by inadequate and misleading statistics. In the past twelve years two major changes have occurred in the management of haematemesis and melaena. Marriott and Kekwick (1935) introduced the technique of drip blood transfusion, and Meulengracht (1935) focused the attention of the medical world on the value of early and liberal feeding.

Errors in Statistics

A knowledge of the fallacies of the statistics of haematemesis and melaena may help to explain the controversies of recent years concerning mortality and treatment. Errors may arise from the method of collection of the figures, from their mode of presentation, or from intrinsic variation between different series. The literature on haematemesis contains three types of series: mass hospital statistics, individual or personal series, and collected series. Mass hospital figures form the numerical bulk of mortality statistics. Their significance must depend on the accuracy of the final diagnosis recorded on the notes and on the method of compiling the annual statistical tables. Physicians vary greatly in the attention they give to the accuracy of the final diagnosis. A common error is the omission of the complication, and "gastric" or "duodenal" ulcer is recorded without reference to the haemorrhage which led to the admission of the patient.

Individual series are the only reliable sources for information. The personal attention to detail by an interested physician and the careful recording of the diagnosis probably explain the lower mortality in such series compared with contemporary mass statistics. One fallacy does exist: there is a tendency to publish a small series of 20 to 30 personal cases, illustrating a particular form of treatment. There is certainly a bias towards publishing successful rather than unsuccessful results, and the chance of halving a mortality in a short sequence of cases is appreciable.

Collected series of statistics are highly fallacious. There is a tendency to include groups of cases with different criteria of diagnosis. Many series exclude cases of melaena; others exclude cases with no radiological proof of ulcer. Rasberry and Miller (1943) include 24 papers concerning fewer than 40 patients out of a collected series of 33 papers. To bring together this number of short series is to accentuate greatly the possibly misleading influence of successful short sequences. The mode of presentation of figures is the source of much confusion in the literature on haematemesis and melaena. A great difficulty in the evaluation of many papers is the failure to state the criteria of diagnosis of bleeding, the criteria for diagnosing peptic ulcer, and the basis for excluding cases diagnosed as carcinoma.

It is particularly unfortunate that the majority of writers exclude so many of their fatal cases, and their figures are quoted and requoted without any reference to the major amputation which occurred in the original paper. It has been the rule to exclude deaths with associated heart failure, pyelonephritis, acute perforation, cellulitis of the arm, parotitis, etc. Such complications may be iatrogenic and should not be excluded. Only two exclusions would seem justifiable: first, when the patient dies from a clearly unrelated disease (for example, tuberculosis); secondly, if an individual, having recovered from his haemorrhage, undergoes an elective operation and dies, his death should not be attributed to the initial bleeding.

Another source of error is the exclusion of surgical cases. It is customary for cases of haematemesis and melaena to be admitted to medical wards, and in few, if any, hospitals can admissions be made at random to both medical and surgical wards. When operation has been advised the patients have been transferred to the surgical wards. Nevertheless, many writers have excluded their surgical transfers and the surgical deaths. Thus, Crohn (1927) excluded seven surgical cases (with five fatalities) in a series of 101 haemorrhages, and his published mortality is 4%. This gives an entirely erroneous impression of the prognosis of bleeding when quoted by later writers. A fallacy common in the early part of this century was to relate the number of deaths from haemorrhage to the total number of patients admitted with peptic ulcer. Hawkins's (1907) mortality of 0.7% was calculated on a total of 556 patients admitted with peptic ulcer, but the number with frank bleeding is not stated.

The third great source of error in the literature of gastro-duodenal bleeding arises from the intrinsic variation of different series. There are four major variables to be considered—age, sex, type and position of lesion, and social status. The mortality of haematemesis and melaena from peptic ulcer rises after the age of 45 and is negligible before that age (Goldman, 1936). The mortality may be lower in women than in men. Acute gastric ulcers are much less serious than indurated penetrating lesions. Chronic duo-

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denal ulcers may be less serious than chronic gastric ulcers. The nutritional status may be better in private patients than in the labouring classes.

The age distribution may vary enormously (Table VI) and render useless the direct comparison of final mortality rates. Intrinsic variation in series is particularly apt to occur between private series, voluntary hospital series, and municipal hospital figures. There is an increase in the gastric/duodenal ulcer ratio with descent in the social scale (Morris and Titmuss, 1944), and there is no doubt that the age distribution is appreciably higher in municipal than in voluntary hospitals. There is a strong bias on the part of the general practitioner to send the old, decrepit, ill-cared-for, ill-nourished man to the local municipal hospital, where admission is a statutory obligation and therefore certain, rather than to a voluntary hospital, where a strong bias may be exercised by the junior staff against the admission of old and possibly chronic cases.

Comparison of figures from different countries introduces a possible error. Geographical variations in the ratio of acute and chronic gastric ulcer and in the gastric/duodenal ratio probably exist. The ease of admission to hospital under different medical services may influence the proportion of mild cases treated at home and therefore excluded from hospital statistics.

British Figures

In spite of the many fallacies of medical statistics and the possible inaccuracy of individual series, a trend in mortality is clearly seen when reviewing the major British papers. In the early part of the century the mortality of haematemesis and melaena from ulcer was considered to be under 5% (Paterson, 1924). The frequently quoted figure of 2.5% from Guy's Hospital (1911–20) given by Hurst (1924) refers to an analysis of the post-mortem figures, excluding surgical fatalities, corrected for necropsies not performed and related to an approximate number of admissions. The corrected figure of 4% does indicate the low mortality at this time.

An increase began probably about 1917 (Bulmer, 1932), reached a maximum of about 20% between 1925 and 1935, and during the past decade has declined to between 5 and 10% (Table I and Fig. 1). The increase in mortality was undoubtedly associated with two changes in the character of peptic ulcer. In the early part of this century acute ulcer causing bleeding was prevalent among women but carried a very low mortality (Bolton, 1913). The Registrar-General's mortality figures have demonstrated the virtual disappearance of this group of peptic ulcer in young women (Jennings, 1940; Tidy, 1944).

TABLE I.—*Mortality from Haematemesis due to Peptic Ulcer*

Key for Fig. 1	Name	Period	Cases of P.U.	Mortality (Corrected to conform with same criteria as present series)
C	Conybeare, quoted by Hurst (1924)	1911–20	Approx. 600	4%
B	Bulmer (1927)	1902–9	136	7%
B_2	,, ...	1910–17	179	7%
B_3	,, ...	1918–25	145	15%
B_4	Bulmer (1932)	1926–31	111	16%
Ch	Chiesman (1932) ..	1925–31	191	25%
C & P	Cullinan and Price (1932)	1925–9	105	18%
B & H	Burger and Hartfall (1934)	1921–30	137	21%
H	Hellier (1934)	1926–32	284	14%
A	Aitkin (1934)	1929–33	262	13%
H & B	Babey and Hurst (1936)	1919–35	82	7%
S	Smith (1945)	1934–45	180	6%
A J	Present series	1940–7	615	8%

Although the loss of these cases of acute ulcer would increase the absolute mortality rate, this was not the complete story, and from 1920 to 1930 a further change occurred in peptic ulcer. A twofold to threefold increase became apparent in the Registrar-General's figures, the increase being due to ulcers, particularly duodenal, in men over 40 (Tidy, 1944). That this increase is real and not due to improved diagnosis or increased surgical intervention is revealed by the statistics on acute perforation from Glasgow,

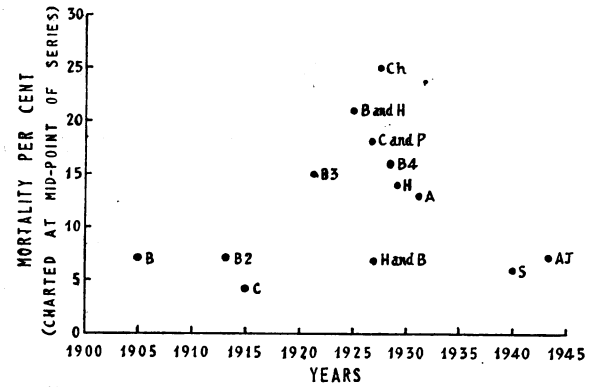

FIG. 1.—Mortality from haematemesis due to peptic ulcer. (For key, see Table I.)

which show a corresponding increase (Illingworth, Scott, and Jamieson, 1944). These changes are well reflected in the papers on haematemesis and melaena. The important mortality is in men over 40, and the lowest in women under 40. An analysis of papers from this country has shown that a remarkable increase occurred about 1920 onwards in the proportion of men over 40 and also in a marked decline in the younger women (Table II and Fig. 2).

TABLE II

Author	Males over 40		Females under 40	
	Incidence	Mortality	Incidence	Mortality
Bulmer (1902–25)	17%	24%	46%	6%
Bulmer (1926–31) and Cullinan (1925–9)	50%	20%	11%	11%
Smith (1934–45)	40%	16%	11%	5%
Avery Jones (1940–7)	51%	10%	5%	4%

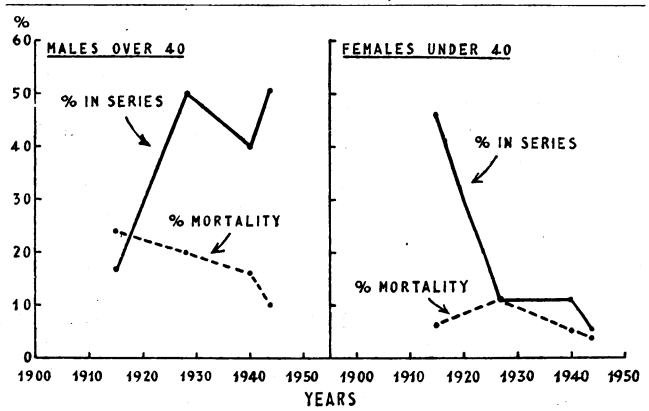

FIG. 2.—Change in age distribution of admissions for haematemesis from peptic ulcer. (See also Table II.)

The actual mortality rate for men over 40 has declined, but nevertheless the proportional increase was so great that the total mortality rose and reached very high levels in 1930. The fall in mortality in the past decade is undoubtedly due to the introduction of liberal feeding (therefore adequate fluids) and to the use of the drip blood transfusion of Marriott and Kekwick.

The controversy on haematemesis in past years was accentuated by Hurst and Ryle's (1937) contention during the 1930 period that the mortality was extremely low. Between 1919 and 1935 Hurst treated 82 cases of bleeding peptic ulcer in his wards at Guy's Hospital, and seven died. He excluded three cases—two perforated after admission

and one had pyelonephritis. The latter complication developed a month after the haemorrhage and may legitimately be excluded. His corrected mortality of 7% represents a result that could be obtained by an interested physician who avoided the extreme dehydration which contributed to the high level of the mass hospital statistics at that time. Ryle (1937) reported a mortality of 1.6% in 258 patients seen in consulting practice, excluding six complicated fatal cases. It would seem that many of the patients were first seen in his consulting-room after the bleeding episode had resolved, and his cases are therefore entirely different from those seen when admitted to hospital for acute haemorrhage.

No attempt will be made to present a corresponding analysis of the American and Scandinavian series, as there are too many imponderables to allow a critical comparison with the British papers. Figures for the early years of this century do not appear to be accessible in the American and Scandinavian literature, but the recent fall in mortality can readily be discerned. The Scandinavian figures during the 1930 period were never as high as the British and American series, and the fall to 1% (Meulengracht, 1939) has not been reproduced in this country or in America in any series of over 100 cases. It seems probable that admission to hospital is easier in Scandinavian countries, and many more mild cases are therefore included in their series. Only 30% of Meulengracht's cases showed radiological evidence of peptic ulcer, against 60% in this series, suggesting a lower proportion of chronic ulcers than in this country.

Besides the many statistical pitfalls there are semantic difficulties which encumber the literature on haematemesis and melaena. Recurrent bleeding is used by some writers when referring to recurrent episodes of bleeding over the period of years, and by others to mean recurrent bleeding after admission with haemorrhage. Massive bleeding is used by some to mean exceptionally severe haemorrhage and by others to distinguish cases from those with occult blood in the stools. Some writers use "haematemesis" generally to comprise all cases of haemorrhage, including melaena only, while others exclude such cases of melaena. In earlier papers bleeding gastric ulcer often included cases of duodenal or jejunal ulcer. Finally, post-operative cases and melaena may refer to patients who had a gastric operation in the past or to patients immediately after operation.

It is insufficiently realized that a single mortality figure is an exceedingly dangerous symbol. The surgeon may reassure himself and the patient that the mortality of a partial gastrectomy is low, but the extremely small risk in men aged 30–40 is offset by much increased hazard in older age groups. To appreciate the prognosis of bleeding peptic ulcer it is essential to know the risk with different types of lesions and in different age groups. The present series is large enough to enable this information to be readily available.

The Series

The 687 patients are a consecutive series admitted between June, 1940, and January, 1947, inclusive, mainly to the dietetic wards under my care. Patients who had a brisk haemorrhage in hospital while under treatment for some other condition have been included. Patients with dark blood-stained vomit, associated with intestinal obstruction, peritonitis, diabetic coma, blood dyscrasias, etc., were not included. Every patient had had evidence of frank recent haematemesis and/or melaena with good evidence of blood loss. The hospital, being municipal, serves a defined area in N.W. London only, and the admissions are therefore representative of the medical problems of that district. The statistics have not been influenced by problem cases from other parts of London.

The series has been divided into two groups to allow separate consideration of 14 patients who came into hospital with acute perforation of a peptic ulcer and who then bled during the first two weeks after operation. It was felt that the additional hazard of the prior perforation rendered comparison with other groups unreal. These cases of haemorrhage after acute perforation were all treated in the same wards, which are for surgical as well as medical cases of peptic ulcer. The five patients who perforated after admission for bleeding are regarded as having complications of the haemorrhage and are included in the main series.

The patients were treated on the general lines described by Witts (1937). They received an initial purée diet and drip blood transfusions. The aim of transfusion was to try to maintain the haemoglobin at such a level that further bleeding, if it occurred, would not cause severe anoxaemia. Approximately one-third of the patients were transfused; the amounts used are shown in Table III. The lowest haemoglobin readings were below 40% in 28% of the peptic ulcer group.

TABLE III.—*Blood Transfusion*

No. of Bottles (540 ml.)	No. of Cases	No. of Bottles (540 ml.)	No. of Cases
1	47	11	4
2	72	12	1
3	38	13	3
4	22	14	1
5	15	15	1
6	11	16	2
7	3	17	1
8	5	18	2
9	5	20	1
10	2	21	1

Diagnosis.—A detailed history and an examination were recorded in all cases, and a barium meal was given as soon as the patient could get up, usually during the third or fourth week, but sometimes sooner. Gastroscopy was carried out in selected cases between three and ten days after the bleeding had ceased. This examination was made as a clinical investigation into the cause of bleeding. Great care and discretion were exercised and no serious complications occurred. Gastroscopy was performed in 116 out of 217 cases which clinical and radiological investigations failed to diagnose. In 65 cases a rapidly healing gastric ulcer was found. The final diagnoses are listed in Table IV.

TABLE IV.—*Final Diagnosis*

Peptic Ulcer Group	Admissions	Deaths
Chronic G.U. group:		
Chronic G.U. diagnosis established by radiograph, operation, or necropsy	110	24
Chronic G.U. and D.U.	12	—
Chronic D.U. group:		
Duodenal ulcer	190	16
Pyloric ulcer	17	3
Previous G.-E. or P.G. for D.U.	34	1
Acute lesion group:		
Acute gastritis	5	—
"Gastrostaxis" (no lesion discovered at necropsy)	2	2
Acute G.U.	67	2
Other "*x*-ray"-negative cases	143	—
Miscellaneous group:		
Oesophageal ulcer	2	—
Partial thoracic stomach	6	—
Not radiographed	27	—
	615	48
Gastric tumours:		
Simple	4	—
Malignant	18	6
Portal hypertension:		
Banti's syndrome	12	—
Cirrhosis hepatis	11	5
Portal thrombosis	2	2
Other causes	11	1
First series. Total	673	62
Second series	14	6
Haematemesis and melaena after admission for acute perforation of peptic ulcer		
	687	68

The diagnosis of carcinoma of the stomach was confirmed by histology or by the presence of metastases except in two cases where necropsy was performed at the coroner's office and the pathologists reported neoplastic gastric ulcer. A number of causes of massive haemorrhage have not yet been specifically diagnosed in these wards—for example, haemorrhage from localized arteriosclerosis of gastric vessels (Frank, 1946), which may cause repeated episodes of bleeding; hereditary telangiectasia, in which epistaxis is usually a feature of the previous history (Griggs and Baker, 1941); malignant or benign tumours of the oesophagus; multiple polyps of the oesophagus (Dickes *et al.*, 1942); small-intestine innocent tumours; leiomyoma; neurofibroma (Baker and Halley, 1946; Hanno and Mensh, 1944; Dudley, 1934; Klingenstein, 1938), haemangiomatosis, or carcinoma of the small intestine (Segal *et al.*, 1945); rupture of aneurysm of aorta, hepatic artery (Gordon-Taylor, 1943), or splenic artery (Murphy, 1942). The 12 admissions for Banti's syndrome were due to only two patients. The "other causes" included Meckel's diverticulum (2), renal failure (4), spurious haematemesis from pulmonary disease (2), and carcinoma of the pancreas (1).

Age Distribution.—Gastro-duodenal haemorrhage is more often fatal in the elderly, and the age distribution (Table V) must be taken into consideration in comparing

TABLE V.—*Peptic Ulcer Group: Age Distribution*

Age	Males		Females		Total Deaths	Mortality
	No.	Deaths	No.	Deaths		
10–19 ..	3	—	2	—	—	—
20–29 ..	24	1	11	—	1	3%
30–39 ..	63	—	18	1	1	1%
40–49 ..	109	5	37	1	6	4%
50–59 ..	90	6	55	2	8	6%
60–69 ..	81	11	47	5	16	12%
70–79 ..	34	10	28	3	13	21%
80+ ..	7	1	6	2	3	23%
	411	34 (8%)	204	14 (7%)	48 (7·8%)	

these results with other series. The proportion of patients over 60 is considerably higher than in previous series in this country or elsewhere (Table VI). The high proportion

TABLE VI.—*Cases over 60 Years of Age*

Name	Region	Period	No.	No. over 60
Bulmer (1927)	Birmingham	1902–26	460	9 (2%)
Burger and Hartfall (1934)	London	1921–30	137	13 (10%)
Cullinan and Price (1932) ..	,,	1926–30	105	11 (10%)
Kirsner and Palmer (1939)	America	1929–39	230	30 (13%)
Meulengracht (1936) ..	Denmark	1932–6	206	36 (17%)
Clemmesen and Lund (1939)	Norway	1934–8	263	40 (15%)
Scott (1940)	Glasgow	1936–40	110	18 (16%)
Schiff (1944)	America	1937–44	160	34 (21%)
Present series	London	1940–7	615	203 (33%)

(33%) of elderly people in this series has been maintained since the end of the war and cannot be explained by the loss of younger patients into the Forces. Comparison with previous figures is difficult, as there are no pre-war series from municipal hospitals, and the bias towards sending the older and more decrepit patients to these hospitals is probably the cause of the high proportion. The figures nevertheless suggest that the age of the ulcer population may be increasing.

Mortality.—The total mortality of the peptic ulcer group is 7.8%. Under 45 the mortality remains at 2%, then rises to 6% between the ages of 45 and 59, 12% between 60 and 69, and finally 21% over 70.

Fatal Cases

There were 48 fatal cases among 615 patients in the peptic ulcer group, and an analysis of these cases is presented with reference to nature of lesion, presence of complications, recurrent bleeding, and mode of death. Necropsy was carried out in 45 out of the 48 cases.

Bleeding (Table VII) accounted for 34 out of the 48 fatalities, the others being due to acute perforation (5), pulmonary embolus (1), coronary thrombosis (2), and exhaustion (6).

At necropsy it was usual to find one large open vessel in the floor of the ulcer, and it was remarkable that death had occurred usually not quickly but after several recurrent bleedings in the course of as many days. Bleeding with acute collapse must have occurred from the large exposed vessel, not once but perhaps six times. It would seem probable that in most cases bleeding from such a large vessel could have occurred for only a short period, perhaps 10 to 15 minutes, and then ceased. At operation it was more usual to find the vessel beginning to spurt when manipulated than when first seen.

TABLE VII.—*Time of Death from Bleeding*

	No.	Suddenly while in Hospital	Days							
			1	2	3	4	5	6	7	7+
Acute lesion group ..	3	—	1		1		1			
Chronic gastric ulcer group	14	1	2	2	2	2		2	1	2
Duodenal ulcer group ..	17	1	4	3	1	3	1	1		3

The distribution of the ulcer fatalities by diagnosis, age, and sex is set out in Tables VIII and IX, which particularly indicate the prognosis in different age groups. It is apparent that the mortality of chronic gastric ulcer is twice as high as duodenal ulcer. There is, however, one fallacy; the duodenal ulcer group undoubtedly contains many more acute ulcers, whose presence is shown more readily in the constricted space of the duodenal cap than they would be radiologically in the body of the stomach. The mortality of the acute lesions is extremely low.

TABLE VIII.—*Male Fatal Cases*

Age	Chronic G.U. Group		D.U. Group		Acute Group		Misc. Group	
	No.	Deaths	No.	Deaths	No.	Deaths	No.	Deaths
10–19 ..	—	—	2	—	1	—	—	—
20–29 ..	2	1	15	—	4	—	3	—
30–39 ..	12	—	25	—	20	—	6	—
40–49 ..	20	4	57	1	28	—	4	—
50–59 ..	24	4	41	2	24	—	1	—
60–69 ..	21	5	37	6	23	—	—	—
70–79 ..	6	3	14	7	9	—	5	—
80+ ..	—	—	2	1	5	—	—	—
All ages ..	85	17	193	17	114	—	19	—
Mortality	20%		9%		—		—	

TABLE IX.—*Female Fatal Cases*

Age	Chronic G.U. Group		D.U. Group		Acute Group		Misc. Group	
	No.	Deaths	No.	Deaths	No.	Deaths	No.	Deaths
10–19 ..	—	—	—	—	2	—	—	—
20–29 ..	3	—	4	—	3	—	1	—
30–39 ..	2	—	5	1	10	—	1	—
40–49 ..	4	1	11	—	21	—	1	—
50–59 ..	9	1	14	—	28	1	4	—
60–69 ..	10	3	8	1	23	1	6	—
70–79 ..	8	2	5	—	13	1	2	—
80+ ..	1	—	1	1	3	1	1	—
All ages ..	37	7	48	3	103	4	16	—
Mortality	19%		6%		4%		—	

The relation of death to brisk recurrent bleeding shows that there is a correlation between death and recurrent bleeding from chronic ulcer but not from acute lesions in which recurrent bleeding is almost as frequent and often as severe (Table X).

TABLE X.—*Brisk Recurrent Bleeding After Admission*

Age	Chr. G.U. Group			D.U. Group			Acute Group			Misc. Group		
	A	B	C	A	B	C	A	B	C	A	B	C
10–19				1								
20–29	1	1		1								
30–39	4			3	1		5			2		
40–49	7	3		6	1		12			1		
50–59	9	4	2	5	2		9			1		
60–69	7	4		8	3	2	11	1				
70–79	3	3		6	4	1	5	1		1		
80+				1			1	1				
	31	15	2	31	11	3	43	3	—	5	—	—
Total No. ..	122			241			217			35		
Proportion with A	26%			13%			20%			14%		
Mortality with A	48%			35%			7%			—		

A = Brisk recurrent bleeding. B = Died. C = Successful operations.

Left ventricular enlargement was found in 10 out of the 45 necropsies and severe arteriosclerosis in 22. Oedema of the lungs was uncommon; massive oedema was present in only one case and terminal oedema of the bases in four. One chronic gastric ulcer was accompanied by an acute ulcer which had developed after laparotomy and simple suture of the bleeding vessel. An exposed vessel was seen in 32 out of the 45 cases and an aneurysmal swelling was noticed four times. The large size of the chronic ulcers was very striking; 75% were over 3 cm. in diameter, the largest being 13 cm.; but in spite of the large size there was no histological evidence of malignancy.

By the bedside the fatal cases could be clearly distinguished as those with a grave clinical complication which would essentially rule out the thought of surgical treatment. Thirty-five of the fatal cases had such complications as being over 75 years (8), severe emaciation (5), generalized oedema (4), cerebral thrombosis, heart failure, pneumonia on admission, gross bronchiectasis, etc. The general condition of the remaining 13 cases was sufficiently reasonable for surgical treatment to be contemplated.

The Role of Surgery

During the first 400 admissions surgery was undertaken as a last desperate measure in three cases, all fatal. It was considered a legitimate clinical observation to try to determine which cases would still have died under modern medical treatment. During this time it became apparent that there was a small group of nine uncomplicated cases in which life might have been saved by timely surgery (Table XI).

TABLE XI.—*Uncomplicated Cases (Medical)*

Sex	Age	Ulcer	Surgical Risk
M.	25	Chronic G.U. (3 × 2 cm.)	Fair
F.	30	,, D.U. (8 × 5 cm.)	,,
M.	47	,, J.U. (2 cm.)	,,
M.	56	,, G.U. (2 cm.)	Good
M.	60	,, G.U.	Fair
M.	61	,, G.U. (6 cm.)	Very poor
M.	70	,, G.U. (1·5 cm.)	Fair
M.	70	,, D.U. (1 cm.)	Poor
M.	72	,, (5 × 3 cm.)	Fair

As a result of the analysis it was decided to undertake an emergency partial gastrectomy on those patients, particularly if over 50, with good clinical evidence of a chronic ulcer who were free from medical complications and who had a brisk recurrent haemorrhage after admission to hospital. Persistence of pain after admission and evidence of arteriosclerosis would be further points in favour of operation. Evidence of chronic ulcer has meant a history of known previous chronic ulceration or recent pain, particularly if severe, lasting more than three weeks. The temptation to lay down a hard-and-fast dividing line should be resisted, as there is so much individual variation, and every case must be considered on its own merits.

In the past the mortality of delayed surgery has been high—for example, Finsterer (1939), 26.9%; Gordon-Taylor (1937), 36%—but a high mortality for delayed operation is not necessarily an argument against it if by waiting one can distinguish a group of patients who carry an exceptionally high mortality with medical treatment. It will be seen from Table X that such a group will carry a mortality of some 50% or more under medical treatment. Surgery, even at a risk of 20 to 30%, may still effect a saving in life. Among the past 267 admissions seven such patients have had an emergency partial gastrectomy performed by Mr. J. D. Fergusson, with one death (Table XII).

TABLE XII

Sex	Age	Diagnosis	Brisk Recurrent Bleeding in Hospital	Day of Operation after Onset of First Haemorrhage	Operation	Result
M.	51	Chr. G.U.	2	2nd	Part. gast.	Recovery
M.	57	Chr. G.U.	2	5th	,,	,,
M.	65	Chr. pyloric ulcer	4	8th	,,	,,
M.*	58	Double chr. G.U.	1	1st	,,	,,
M.	66	Chr. D.U.	3	2nd	,,	,,
M.	70	Chr. G.U.	3	6th	,,	,,
M.	75	Chr. D.U.	1	1st	,,	Died

* This patient belonged to the second series—that is, he bled after admission for acute perforation.

There is therefore a small group of patients with chronic ulcer and brisk recurrent bleeding after admission in whom delayed emergency partial gastrectomy can be successful. Finsterer (1939) achieved a 5% mortality by operating on all cases within 48 hours. With the high age distribution and the frequency of associated medical complications in municipal hospital practice, it is extremely improbable that routine emergency surgery immediately on all admissions would result in a lower total mortality than from medical treatment together with limited surgery as proposed.

The Role of Blood Transfusion

Although most clinicians use drip blood transfusions in the management of gastro-duodenal haemorrhage, there are still some who remain unconvinced of its value. Smith (1945) in a critical survey of 200 cases does not advise transfusion. The arguments for and against will be briefly considered.

The risk of reactions to blood is a real one, and mild rigors, fever, or slight jaundice may follow in a small proportion (under 5%) even when cross-grouped. Again, there is a risk of inducing Rh antibodies in the Rh-negative individual. This may lead to reactions during subsequent transfusions, and women who become sensitized may thereafter be incapable of bearing a live child. Normal pregnancy results in sensitizing about 4% of Rh-negative mothers with Rh-positive husbands. Therapeutic blood transfusions, however, sensitized about half those at risk (Wiener and Gordon, 1947). As 15% of women are Rh-negative, this aspect must be seriously considered when transfusion is contemplated for women during the reproductive period of life.

A blood transfusion may overload the circulation, increase the right auricular pressure, raise the cardiac output, and possibly lead to acute left ventricular failure and oedema of the lungs. Sharpey-Schafer (1945) has demonstrated graphically the embarrassment which may occur in the circulation after transfusion in severely anaemic patients, and has further demonstrated the risks of transfusion in relation to the changes in haemodynamics which follow an acute haemorrhage.

It has been thought that blood transfusion may perpetuate or restart the bleeding, but the incidence of recurrent bleeding has actually fallen since the introduction of drip transfusions and it has not been the experience in this series that they do restart the bleeding. It has been argued that blood transfusion may raise the blood pressure. Drip blood transfusions do not increase the blood pressure more than would occur without transfusion (Avery Jones, 1939a). There does not appear to be any reliable evidence that subsequent haemopoiesis is depressed by transfusion, and the risk of transmitting infective hepatitis, syphilis, or malaria is very small.

Against these arguments are cogent reasons for giving drip blood transfusions. Death from bleeding does not occur quickly in most cases, but follows two, three, four, or more brisk recurrent bleedings in as many days. Therefore there is time to replace lost blood by drip transfusion even if given at the rate of 540 ml. in four hours. If the blood lost is replaced, and if the haemoglobin level is maintained above 40–50%, the patient is in a much better position to withstand further bleeding, should it occur.

If the blood loss is by slow oozing it is quite feasible to keep pace with the bleedings by drip transfusion. If surgery becomes necessary the patient can be maintained at a much safer level than if no transfusions were given. Once the haemoglobin is below 30% there is a risk of anoxaemic manifestations with disorientation, restlessness, particularly at night, and lack of co-operation. Such manifestations may well induce a vicious circle, as the patient becomes intolerant of further transfusion and nursing. Cerebral anoxaemia is particularly serious in elderly patients, and a short period may induce irreversible cellular changes which cause coma and death. Occasionally a severe initial collapse, with great reduction of the circulating blood, may lead directly to a state of profound and persistent oligaemic shock and death, which might have been averted if the restoration of the blood volume had been assisted.

Again, severe anaemia will cause a sharp increase in the output of the heart (Sharpey-Schafer, 1945), and the accompanying high venous pressure may induce cardiac failure and oedema, and in the presence of these phenomena the risk of transfusion will be considerably increased. If the patient becomes severely anaemic he may be considerably distressed from headache, and his intense pallor is an added anxiety for the medical staff and relatives. Lastly, there is the small risk of permanent amaurosis, which may follow recurrent bleeding in an anaemic patient.

The available evidence supports the use of blood transfusion, but a bias in favour of transfusion must be exerted with discretion and not enthusiasm. All possible measures must be taken to minimize the known risks. Women in the reproductive period of life should be Rh-tested; severely anaemic patients should be under constant supervision during the transfusion and a watch kept on the jugular veins (with the patient propped up) and on the pulse rate. Blood should be cross-grouped, and the bottle carefully inspected before use. The same blood group should be employed whenever possible. The experience of this series has shown that blood transfusion is a most valuable therapeutic aid in the management of gastro-duodenal haemorrhage. If it is given as a drip transfusion the patient can often be maintained at such a level of haemoglobin that he is able to withstand any further bleeding without developing anoxaemic manifestations. In view of the work of Sharpey-Schafer it is clearly desirable to maintain the haemoglobin above 40%, and secondary cardiac embarrassment may thereby be avoided. The patient is spared the headache of severe anaemia, and the risk of amaurosis is minimized. The risk of acute cardiac failure from careful transfusion is considerably less than that of leaving the patient's haemoglobin below 40% when bleeding may recur.

Transfusions have been given with the M.R.C. blood transfusion set, and in very few cases has it been necessary to cut down on a vein. Usually 540–1,080 ml. has been transfused in four to eight hours, but on occasion 1,620–3,240 ml. has been given continuously at the same rate. Except in a few collapsed patients, the blood has never been given quickly. One patient died, possibly as the result of the transfusion, but necropsy revealed the presence of coronary infarction. On the other hand, in several patients insufficient transfusion undoubtedly contributed to their death. Minor reactions, slight jaundice, and urticaria have occurred in a very small minority of cases.

[*The second lecture, with a list of references, will appear in our next issue.*]

GASTRO-ENTERITIS OF UNKNOWN AETIOLOGY

AN OUTBREAK IN A MATERNITY UNIT

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In recent years increasing attention has been paid to a form of gastro-enteritis for which, despite improved methods of laboratory diagnosis, no known cause has been discovered. A number of such outbreaks have been described in this country,[1–10 18 19] the U.S.A.,[11–16 20] and on the Continent.[17 21] The diagnosis depends on the recognition of a distinctive clinical picture differing from bacterial gastro-enteritis, on the presence of an epidemic, and on negative laboratory findings.

These outbreaks, excluding "epidemic diarrhoea of the newborn,"[23 24] can be divided into two categories with rather different symptoms. On the one hand there is the epidemic nausea and vomiting described by Rischel,[17] Miller and Raven,[18] and Bradley,[19] in which diarrhoea is not a predominant symptom; into this group falls, possibly, the less well defined "winter vomiting disease" of American observers.[15 16 20] On the other hand there is a variety in which diarrhoea is the most striking and constant symptom, as exemplified in outbreaks described by Smith and Davies,[1] Brown, Crawford, and Stent[2] in this country, and by Reimann *et al.*[13] and other observers[11 12 14] in the United States.

This paper is concerned with the second type of illness, characterized by diarrhoea of abrupt onset, with frequent watery stools of a yellowish-grey colour without macroscopic blood or pus. Fatal cases have not been reported among adults, but the fatality rate among infants is variable.

Epidemiologically, though most of the outbreaks reported have occurred in selected groups in institutions, both sexes and all age groups are susceptible, the incubation period is short, and the outbreak tends to be of the protracted rather than of the explosive "food-borne" type. The attack rate is often high in semi-closed communities and the disease is particularly troublesome in maternity wards. The outbreaks have a variable seasonal incidence. That described by Smith and Davies[1] was in the autumn