THE TREATMENT OF HAEMATEMESIS

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Haematemesis is one of the commonest medical emergencies that may threaten life. It is the most frequently encountered complication of peptic ulcer, occurring at some time in about 25% of cases,⁸ and peptic ulcer is easily the most important cause of the admission of patients with severe bleeding from the upper alimentary tract. In Avery Jones' series¹² of 2,526 admissions for this emergency, 2,298 were due to proved or probable peptic ulcers. Table 1 shows the distribution of the various causes of bleeding in admissions to the Central Middlesex Hospital for haematemesis and/or melaena between 1940 and 1957.

TABLE 1

Peptic ulcer group:				
Chronic gastric ulcer	..	..	..	423
Duodenal ulcer	..	..	..	844
Post-operative group	previous	or	partial	
gastrectomy	..	..	..	168
'Acute lesion' group	..	..	..	720
Hiatus hernia	..	..	..	47
Unclassified (incompletely investigated)	..	..	..	96
				2,298
Causes other than peptic ulceration:				
Carcinoma ventriculi	..	..	..	57
Portal hypertension	..	..	..	75
Other causes	..	..	..	96
				2,526

Diagnosis of the Cause of Bleeding

Though the probability is that the patient admitted after an attack of haematemesis and melaena is bleeding from a benign ulcerative process in the upper alimentary tract, there remains an important group of less common causes including portal hypertension, gastric carcinoma and other tumours of the gastro-intestinal tract, blood dyscrasias, and bleeding associated with the strain of vomiting (Mallory-Weiss syndrome) and general medical diseases such as periarteritis nodosa, uraemia and malignant hypertension. Their recognition is not

only an interesting diagnostic challenge but also an important factor in subsequent management.

The difficulty of treating bleeding peptic ulcer largely concerns the detection of those patients in whom the prognosis with medical measures must be considered poor and who will bleed to death unless surgery is employed. The problem is not always easily solved. Acute ulceration or erosion of the stomach or duodenum is a common lesion which usually responds well to medical treatment, but this diagnosis is not always obvious, being largely based on negative evidence and frequently made only in retrospect after a negative X-ray. The vessels by which an acute ulcer bleeds are mostly small and submucosal and operation should be undertaken only if there has been severe or current bleeding on two or three occasions accompanied by shock.

Extra-gastric sources of bleeding must also be excluded before patients are submitted to surgery. The history is one of the most valuable aids in achieving a correct diagnosis; thus where chronic peptic ulceration is responsible for the bleeding it will be unusual to find such a patient denying previous periodic dyspepsia with food and alkaline relief of his pain. The shorter the history of preceding dyspepsia, the more likely is the lesion to be acute and, therefore, best treated medically. Enquiry as to recent salicylate consumption may give a clue to correct diagnosis, but unfortunately this is not always obvious and the history may be misleading.

There is thus much need for a method of investigation that will yield diagnostic information in the acute stage of illness, for if chronic peptic ulceration can be diagnosed with confidence, surgical treatment is to be recommended in patients over the age of 50 who bleed again after admission to hospital. At the Central Middlesex Hospital the following procedure of early combined investigation has been adopted. As soon as the patient has been made comfortable after admission, a Ryle's tube is passed via the nose and about 5 ml. of gastric contents are aspirated hourly

by syringe suction and pH estimated electrometrically at the end of the intubation period, which finishes at 9 a.m. on the day after admission, when gastroscopy is performed. Later that morning or in the early afternoon of the same day, barium meal examination is done on the ward using a portable X-ray set and without manipulation or the need to move the patient from his bed. By combining the information derived from study of intragastric acidity,^{3, 4, 5} from gastroscopy, and from early X-ray examination, it has proved possible to achieve a correct diagnosis of the cause of bleeding in 80% of patients admitted with haemorrhage from peptic ulcer, usually within 24 or 36 hours from admission.^{6, 7} The reliability of the early diagnostic procedures in forecasting correct diagnosis was significantly greater than the clinical impression of the cause of bleeding in each of the three groups of chronic gastric ulcer, chronic duodenal ulcer and acute ulcer.

General Considerations in Treatment

Though, to a large extent, the correct treatment of haematemesis depends on the diagnostic probabilities established by careful examination and, where possible, confirmed by appropriate investigation, certain general measures apply to all cases of bleeding from the alimentary tract. There is no longer any controversy about the advisability of liberal feeding. Meulengrachts' original diet¹⁴ has been modified in this country by Witts²⁵ and it is this regime that is usually followed; most patients do well on a two-hourly soft diet with fluids as desired between, but if nausea is a prominent complaint then two-hourly milk feeds are sufficient. There is no evidence that such feeding increases the liability to further haemorrhage, the risk of which remains at about 25% of admissions for bleeding peptic ulcer.¹² Certainly it is unphysiological to deprive an exsanguinated patient of food and fluid at such a time of special need and there is no doubt that modern treatment has eliminated the deaths which formerly occurred from therapeutic dehydration.

Strict confinement of the patient to bed is essential in the presence of severe or continued bleeding, but there is no reason why the patient whose course is uncomplicated should not be allowed up by the second or third hospital day. There is no evidence to suggest that recovery after haematemesis is hastened by confinement to bed, the dangers of which are enhanced in older patients with associated degenerative diseases. There is little doubt that, in the presence of satisfactory clinical progress, the physician can best serve the interests of his patients by allowing early ambulation, and Pollard and Summerskill¹⁸ have shown that such a policy confers important benefits.

An injection of sodium phenobarbitone, 200 mg., may be required on admission to secure the patients' mental relaxation; occasionally, morphine, 10 to 15 mg., may be needed for the anxiety and restlessness of shock but medication other than vitamin concentrates and antacids is best avoided, though iron may be given by mouth from the start. Constipation is the rule after haematemesis and is usually well tolerated by the patient. Purgatives should not be administered but there is no objection to the use of a glycerine or 'Dulcolax' suppository. Aspirin should always be avoided and, instead, Panadol given for incidental pains or discomfort.

It is unusual for ulcer pain to persist after haemorrhage has occurred and continued severe pain suggests the possibility of concomitant perforation (a particularly lethal combination) or of an independent cause such as cardiac infarction. A continuous intra-gastric milk drip, giving 6 pints in 24 hours through a small nasal tube, is an extremely valuable treatment if the symptoms of active peptic ulceration recur.

Though brisk continued bleeding from peptic ulcer is an indication for surgical treatment, there are a few patients in whom operation can reasonably be considered as out of the question because of severe associated disease or the features of overwhelming senility. Nevertheless, however unpromising the prognosis may appear, neither hope nor treatment should be abandoned. Distension of the stomach with blood clot may be an important cause preventing the atonic stomach of a shocked patient from arresting the bleeding by contraction. Emptying the stomach with a Senoran's evacuator followed by lavage with ice-cold water may help to stop bleeding and the use of a topical haemostatic such as thrombin after washing the stomach out with 1:1,000 adrenalin has been advocated in the treatment of bleeding acute peptic ulcer. In such cases the stomach should first be emptied through a large-bore tube and then lavaged with 1:1,000 adrenalin; finally, thrombin in a suitably viscous vehicle such as methyl cellulose is instilled into the stomach.

Blood Transfusion

An hourly record should be kept from admission of pulse and blood pressure, and blood immediately taken for haemoglobin or haematocrit and into a plain tube for grouping and cross-matching for transfusion. There can be no hard-and-fast rules as to when blood transfusion is required. Whilst blood volume determinations would enable the clinician accurately to assess the amount of blood lost, such techniques are, as yet, unsuitable for routine use and reasonable working rules for the employment of blood transfusion are a pulse rate

of 120 or more, a systolic blood pressure of 100 mm. Hg. or less, or if the haemoglobin is below 50%. Tibbs²⁴ found that the diastolic blood pressure reflected the diminution in blood volume more accurately than the systolic pressure, except in hypertensive patients who maintain their diastolic pressure better than normals in the presence of bleeding. A diastolic pressure of 60 to 65 mm. Hg. indicated moderate to severe blood loss. Transfused blood does not act as a haemostatic but there is no evidence to suggest that it provokes recurrence of bleeding. It is best regarded as a remedy for haemorrhagic shock and as giving the patient a further margin of safety should he bleed again. Prompt transfusion is particularly important in the elderly to prevent the irreversible cerebral damage that may complicate prolonged shock. If severe anaemia is allowed to develop a compensatory increase in venous pressure leads to greater cardiac filling and improved cardiac output. This hyperkinetic phase presents with a full bounding pulse, raised pulse pressure and jugular venous distension. The venous pressure also rises during the initial stages of transfusion, and though it falls again as the arterial pressure rises, overtransfusion in the hyperkinetic phase carries the risk of precipitating heart failure.

Usually gastro-duodenal bleeding stops within 12 or 24 hours of admission, most often permanently, and during this time 1,500 to 3,000 ml. of blood may be given by slow drip transfusion (40 drops per minute). Occasionally there is a continuous slow loss of blood requiring intermittent transfusion and this seems to happen most commonly in patients bleeding from acute lesions; the vessels by which an acute ulcer bleeds are mostly small and submucosal, and such bleeding lacks the dramatic quality of arterial haemorrhage from the base of a chronic ulcer. Exceptionally, chronic ulcers bleed so severely that the only hope for the patient lies in intra-arterial transfusion and immediate operation.

Transfusions of 500 ml. are unnecessary and wasteful. Large volumes of blood given rapidly carry risks of citrate intoxication and hyperkalaemia. The former may lead to defective clotting and myocardial failure and it is wise to give 10 ml. of 10% calcium gluconate intravenously after every fourth bottle of blood. Potassium intoxication carries the very real threat of cardiac arrest, particularly if bank blood near the limit of its expiry is used, in which serum potassium levels may reach 25 mEq/l.¹³ Warming the blood before use will encourage the return of potassium to the cells and the danger can be further diminished by the infusion of dextrose solutions. However, when such massive transfusions are in question, the need for surgical

arrest of haemorrhage should be urgently reviewed.

Surgery of Bleeding Peptic Ulcer

Though the majority of patients recover under medical management, there is undoubtedly a significant proportion who will bleed to death unless surgery is employed. If chronic peptic ulceration can be diagnosed with reasonable confidence, operative treatment is to be recommended for those older patients in whom bleeding is severe, or recurs after admission to hospital. As early as 1918, Finsterer⁹ proposed the application of radical surgical measures, the operation being performed within the first 24 to 48 hours from the onset of haemorrhage; his reported operative mortality for early cases was 5%. Gordon-Taylor commented: 'Finsterer's first 48 hours is still the optimum period for surgical attack in haematemesis and the golden age of gastric surgery will have been attained only when all cases of haemorrhage from chronic ulcer come to operation within that space of time'. And Tanner²³ found that the best results were obtained by early and frequent surgery. While the mortality with medical management can be as low as the 2.5% achieved by Meulengracht,¹⁵ it is unlikely that a universal surgical approach will provide either comparable success or achieve wide acceptance. In general, surgical intervention has been limited to selected patients^{2, 16, 17} the best results being achieved by prompt surgery in patients with proved or probable peptic ulcers. Avery Jones,¹¹ in a study of 400 consecutive admissions for haematemesis, found particular indications for emergency gastrectomy in patients over 50 years with good clinical evidence of chronic ulcer who had brisk recurrent haemorrhage after admission. Probably the most important factor influencing mortality in haematemesis is the age of the patient; above 60 years there is a steep increase in the proportion of deaths from bleeding peptic ulcer and it is in these cases that the best surgical contribution to the reduction of mortality can be made.

With awareness of the factors influencing mortality in bleeding peptic ulcer, a policy of selective surgical intervention was introduced at the Central Middlesex Hospital in 1946. Surgery was readily considered in patients known to have a chronic ulcer if they bled again after admission. No patient was denied operation in the face of severe recurrent bleeding, but a much stronger bias was exerted in favour of operation over the age of 40. Surgical treatment was undertaken in patients with a presumed or proven acute ulcer if there was severe recurrent bleeding on two or three occasions accompanied by shock. Although bleeding acute ulcer has a very low mortality in younger

TABLE 2
HAEMATEMESIS AND MELÆNA
ANNUAL MORTALITY—PEPTIC ULCER GROUP

Year	No.	Deaths	Mortality	
1941	52	5	5.7%	Medical policy
1942	71	2		
1943	92	4		
1944	106	9	6.6%	
1945	124	8		
1946	118	11		
1947	91	9	7.4%	Selective surgical policy
1948	113	6		
1949	136	9		
1950	119	15	9.4%	
1951	203	13		
1952	188	16		
1953	179	16	7.9%	
1954	176	12		
1955	186	7		
1956	153	7	4.1%	
1957	195	8		
2,302		157		

people, the mortality is appreciable over the age of 60.¹²

As Tanner²² has stressed, the criterion for success or failure of a surgical policy is its influence on overall mortality and not the mortality for surgical intervention as such, which depends on the type of risk accepted. The result of the adoption of selective surgical treatment at the Central Middlesex Hospital is shown in Table 2 which indicates that the overall mortality from bleeding peptic ulcer has been reduced to 4%.

Further analysis of the figures showed that patients with recurrent bleeding at any age from gastric ulcers, and those bleeding from duodenal ulcers over the age of 60 have particularly benefited from a surgical policy. Chronic gastric ulcer is a particularly strong indication for surgery; bleeding from this source carries a high mortality under medical treatment and its operative arrest by gastrectomy is generally easier than is the surgery of bleeding chronic duodenal ulcer. Though these figures cover only those patients with proved or probable peptic ulcer, there are many other possible causes of severe gastro-intestinal haemorrhage which may require surgical intervention, such as tumours of the stomach, vascular lesions of the intestine, diverticular disease, and portal hypertension.

The success of any surgical approach to the treatment of haematemesis requires close co-operation between physicians and surgeons if, as seems proper, patients continue to be admitted to medical wards. In every case all evidence of chronic peptic ulcer from previous investigation must be considered in conjunction with the history

before admission in order to assist the final choice between operative and conservative treatment. The adoption of a policy whereby early diagnosis of the cause of bleeding can be achieved^{6, 7} greatly facilitates correct appraisal.

If operation be decided upon, a little time may be allowed to improve the patients' condition by blood transfusion unless bleeding be so profuse as to permit no delay. The surgical treatment of bleeding peptic ulcer requires an experienced gastric surgeon and preferably a senior anaesthetist. There is a risk of aspiration into the lungs and it is important that a cuffed endotracheal tube be inserted quickly. It is equally important for the surgeon to remove all blood clot from the stomach at operation to minimize the danger of post-operative vomiting and inhalation.

Partial gastrectomy is the operation of choice in all patients; local suture of bleeding points, or local excision of a gastric ulcer have little to recommend them. Any lesser operation than gastric resection is usually futile. There is a difficult problem facing the surgeon who is forced to operate in the face of massive recurrent haemorrhage when no lesion can be identified. The abdomen should never be closed if the surgeon is satisfied that the blood is coming from the upper reaches of the gut and not from lower down; either a wide gastrotomy for inspection and palpation of the gastric mucosa should be made—a prolonged and sometimes uncertain procedure—or a 'blind' gastrectomy performed. In the latter case the ulcer responsible for bleeding will be found in almost every patient on opening the resected specimen.

Tracheotomy and Gastrectomy

Severe chronic bronchitis and emphysema is a constantly recurring clinical accompaniment of haematemesis. Such cases can present formidable operative risks and so bias the physician against operation that medical measures are persisted with despite every evidence that only surgical treatment can stop the bleeding. It is in these cases of severely impaired respiratory function that tracheotomy, done at the time of gastrectomy, can transform the post-operative outlook by making efficient bronchial drainage possible with intermittent suction and by improving ventilation. Experience at the Central Middlesex Hospital in a small number of cases has been encouraging, and in most instances the tracheotomy tube can be removed in 7 to 10 days. An alternative approach, successfully applied by Schooling and Simon,²⁰ was by use of artificial respiration with a Beaver Mark 2 respirator connected to a short cuffed endotracheal tube inserted via a tracheotomy through which bronchial toilet was performed with

intermittent suction. This method has the advantage over tracheotomy alone of ensuring adequate respiration in patients whose ventilating function depends almost entirely on diaphragmatic movement, and who pass into severe respiratory distress after an upper abdominal operation.

Treatment of Bleeding Oesophageal Varices

The development and subsequent rupture of oesophageal varices constitute the most important complication of portal hypertension. In Britain emergency admission to hospital because of bleeding from this cause is relatively infrequent (3% of all admissions to the Central Middlesex Hospital for haematemesis and melaena) compared with American experience where as many as 12.5% of admissions for upper gastro-intestinal haemorrhage have been ascribed to portal hypertension.¹⁹

Bleeding from oesophageal varices is usually severe and exsanguinating, most often originating in the region of the oesophago-gastric junction. In patients whose portal hypertension depends on extra-hepatic portal vein obstruction the prognosis is good, and once bleeding has stopped there is usually a rapid return of good health. In patients with cirrhosis, however, haemorrhage is notoriously difficult to control and is only too often followed by deepening jaundice, ascites and coma. There is, therefore, every reason to arrest haemorrhage in cirrhosis with the least delay.

Temporary control of haemorrhage from oesophageal varices can be obtained in most cases by means of compression with the triple-lumen Sengstaken-Blakemore tube. This is inserted until the gastric balloon is well below the cardia and inflated with 100 ml. of water. The inclusion of 20 ml. 'Hypaque' contrast medium enables its position in the stomach to be easily located by portable plain X-ray film of the abdomen and gentle traction on the tube is sufficient to anchor the balloon at the cardia, this position being maintained by strapping to the cheek with adhesive plaster. Traction on the tube using a pulley system and weights may be dangerous and is unnecessary. The oesophageal balloon is next inflated to a pressure of 20 to 30 mm. of mercury. The gastric balloon compresses the upper part of the stomach against the diaphragm and controls bleeding from the submucosal plexus of veins in the region of the cardia whilst the oesophageal balloon helps to anchor the tube in addition to exerting direct pressure on oesophageal varices. The third lumen of the tube should be used for feeding the patient with 20% glucose solution, for instilling neomycin, potassium supplements and purgatives and for diagnostic aspiration. If bleeding continues, as judged by blood staining of the aspirated gastric contents, it is reasonable to assume that the

haemorrhage is originating elsewhere, often from an associated gastric or duodenal ulcer. This may sometimes be confirmed by the introduction of a radio-opaque medium such as 'Gastrografin' into the stomach via the tube, using the technique described by Chandler *et al.*⁷

It is always difficult to decide on the appropriate time for removal of the tube. The procedure is not without risk, and the dangers of asphyxia from the inflated balloon slipping up into the pharynx and of gastric and oesophageal ulceration increase the longer the tube is left in position. Generally the oesophageal balloon is deflated after 24 hours and it is advisable that decompression be carried out in the morning so that if there is recurrent haemorrhage this can be dealt with as a day-time emergency and not in the early hours. Before withdrawal it is wise policy to leave the apparatus lying *in situ* before aspirating the gastric balloon and removing the tube, to ensure that bleeding has stopped.

There is perhaps no other emergency in which general medical treatment, in addition to the measures adopted to control bleeding, are of such importance. Adequate blood transfusion is essential and oxygen may be required to combat cerebral anoxia. Hypoprothrombinaemia may be a factor disturbing the clotting mechanism in these patients and vitamin K should be administered parenterally. The prevention of coma is of first importance and this risk can be minimized by reducing intestinal bacterial activity with neomycin 2 g. four times a day. The administration of an enema will help to remove as much as possible of blood from the bowel. Both diuretics¹ and pituitrin²¹ have been shown to be effective in lowering portal pressure and both are worthy of trial.

The patient who continues to bleed despite all medical measures presents a formidable problem, for there is no reliable emergency surgical measure for dealing with life-threatening haemorrhage. Indeed, in many patients the bleeding is only one of many abnormalities incidental to liver failure and a fatal termination. The construction of a porto-caval shunt is a long procedure which the severely-ill cirrhotic patient is poorly fitted to withstand. Probably some lesser procedure, such as ligation of the varices, or Tanner's operation²² in which the left gastric vessels are ligated and divided, the upper stomach transected and the ends anastomosed again, is the best method of achieving a more than temporary control of the situation, but the mortality is still high. Should the patient's condition improve sufficiently, consideration can then be given at a later date to a venous shunt operation but, despite early enthusiasm for these procedures, it seems likely that the prognosis of the bleeding cirrhotic patient is

determined by the disease in the liver rather than by the pressure in the portal vein.

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