EMERGENCIES IN GENERAL PRACTICE**HAEMATEMESIS AND MELAENA**

BY

THOMAS HUNT, D.M., F.R.C.P.*Senior Physician, St. Mary's Hospital, London, W.2*

It is estimated that between 20,000 and 30,000 patients are admitted to hospital annually in England and Wales for serious gastro-duodenal bleeding. In most cases this is sudden, dramatic, and urgent, and the haemorrhage is obvious, either as a frank haematemesis or as a sudden and unmistakable tarry stool, often with diarrhoea. The vomit may be dark, like coffee-grounds, owing to the conversion of haemoglobin into acid haematin in the stomach, or it may be fresh and red just as it comes from an arterial haemorrhage. In either case it usually differs from a severe haemoptysis by being acid in reaction and non-frothy, unlike blood brought up from the lungs. The patient may be severely shocked and actually faint, or he may only become cold, weak, and restless. The pulse is quick, the blood pressure low, a cold sweat develops, and the patient, especially if elderly, often becomes confused and delirious. Pallor may be intense, and a peculiar pinched appearance of the face and nose is sometimes very striking.

In some cases, however, bleeding is insidious and difficult to recognize, so that it may continue unsuspected for long periods. It is important to remember, not only that blood oozing from the upper part of the alimentary tract may be insufficient in amount to turn the stools black, but also that patients do not in all cases take notice of dark stools, even if they are passed. In any cases of unexplained pallor, fainting, weakness, or symptoms of anaemia such as dyspnoea on exertion or oedema of the ankles the patient should be questioned about the colour of his motions and asked to notice them, particularly if he has not previously done so; it must not be forgotten, however, that many "tonics" and dyspeptic mixtures contain iron or bismuth, which darken the stools, and that much meat or green vegetables such as spinach have the same effect. A history that the patient has passed dark stools must not, therefore, at once be accepted as evidence of bleeding, and whenever possible this must be checked by a chemical test for occult blood. These tests are simple to perform, require no elaborate technique or materials, and can be carried out easily by the practitioner himself; the finding of such melaena demands, of course, a full history, examination, and investigation.

The simplest test is the Gregerson,* and for this it is not necessary to ban chlorophyll-containing foods, meat, or iron mixtures from the diet, as an immediately positive result definitely indicates blood, whilst a faintly positive one may be ignored. The possibility that the blood has come from the mouth, or from piles, or from swallowing sputum must, of course, be remembered in assessing the significance of a positive test.

**Gregerson's Test.* A powder containing 0.29 g. of barium peroxide, with 0.025 g. of benzidine, is dissolved in 5 ml. of glacial acetic acid and a few drops of the solution are run on to a thin smear of faeces on a filter paper. A blue colour appearing at once or within a few seconds indicates the presence of blood, but appearing only after 20 to 30 seconds is of no significance.

Causes of Bleeding and Differential Diagnosis

Many large series of cases of haematemesis have been published, and from these and from my own experience the main causes, with their percentage incidence in Britain, may be approximately classified as follows:

Chronic duodenal ulcer	..	..	..	40	} 70
Chronic gastric ulcer	..	..	..	30	
Acute ulcer (gastro-duodenal erosions)	..	..	..	20	
Cirrhosis of the liver	..	..	..	5	
Carcinoma of the stomach	..	..	..	3	
Other causes	..	..	..	2	

In practice, the distinction between a chronic duodenal ulcer and a chronic gastric ulcer may be impossible to make and is, in fact, of relatively little importance compared with the vital distinction between an acute erosion and a chronic ulcer. This is a matter of extreme importance, since in the former case any form of surgical treatment is contraindicated, whilst in the latter it may be urgent and life-saving. In some cases the diagnosis is clear, since previous radiological examination may have shown an ulcer, and the history may be typical. In others it may be highly suggestive from the characteristic periodicity and the pain-food relationship.

It is rare, but by no means unknown, for a patient who has a chronic recurrent ulcer to deny any previous indigestion, but as a rule sudden haematemesis, with no history of any preceding pain or dyspepsia, is likely to be due to an acute erosion. This cause of bleeding is not well understood. It is certainly not infrequent, and, if *all* those cases in which no exact cause can be found and barium-meal examination is negative are grouped under this heading, it probably accounts for some 20 to 30% of all gastro-duodenal haemorrhages.

In some cases there is a strong presumption that an irritant drug may have caused the haemorrhage, and it is always important to inquire whether the patient had a cold, toothache, or a headache before the bleeding, as he may very well have forgotten that he had taken aspirin, "veganin," or some similar tablet, which might have caused the erosion. Drugs in tablet form are particularly concerned, and in a few cases the patient may know that he is sensitive to aspirin. In others expectorant mixtures containing quinine or ipecacuanha, or drugs such as phenylbutazone or *para*-aminosalicylic acid, precipitate bleeding, as may some sedatives, such as "sedormid."

Bleeding from an acute gastric erosion is often very severe, and often occurs in young people in apparently perfect health, and for no apparent reason that they can give. There is no tenderness or evidence of ulcer, and no history pointing to liver disease or primary blood dyscrasia. In a few cases the haemorrhage has recurred on several occasions, as neither the patient nor his doctor has been able to discover any cause.

Recurring haematemeses, however, for which no ulcer or other lesion has been found, may have some other less frequent, but important, causes, one of which is particularly characteristic—namely, hereditary telangiectasia. In this condition, multiple angiomas occur and as a rule can be found on examination, especially on the face, lips, or mouth, and there is a family history of gastric bleeding. The patient has almost always suffered from severe epistaxis during infancy and childhood. In one patient, aged 71, there had been over a dozen severe gastric haemorrhages since the

age of 30, on almost every occasion needing transfusion, and he had been once explored and eight times submitted to x-ray examination for ulcer, with negative results. There had never been any pain or indigestion, but, on questioning, he admitted to dangerous bouts of nose-bleeding when he was a small boy. On examination a few naevi were found on the skin, and at a second operation, which was undertaken after a radiologist had demonstrated a supposed gastric ulcer niche after a barium meal, no sign at all of any ulcer was found and the diagnosis of hereditary telangiectasia was confirmed.

In cases of portal hypertension with cirrhosis of the liver the haematemesis is also painless, but there is often a history of dyspepsia and the patient may have been a heavy drinker or have had recurrent jaundice (hepatitis); spider naevi may be found on the skin, with sometimes a characteristic facies. The liver is often palpable and in some cases the spleen also; but absence of these findings does not by any means exclude cirrhosis. The distinction from chronic ulcer, whilst often very difficult, is an extremely important one, since morphine, anaesthetics, and operation all carry serious risks in cases of cirrhosis.

Other causes of bleeding are much less frequent but may sometimes present difficulties in diagnosis. Blood diseases, such as leukaemia or purpura, heart failure, and hypertension may occasionally lead to haematemesis, and the rather more frequent hiatus hernia, with oesophagitis or ulcer, and carcinoma of the oesophagus or stomach must also be thought of. In rare cases a gastric polyp may be a cause of recurrent severe haemorrhage without pain.

Treatment of Severe Cases

The atmosphere of panic and alarm which a severe haematemesis produces in the patient and in those around him may easily spread to the doctor, and it is vital to keep calm and to give an air of reassurance and confidence as much as possible. In actual fact it is rare in these cases for a few minutes to become a matter of life and death, and it is a good thing to stress the favourable aspects of the emergency, the high recovery rate, and the well-understood lines of treatment. It may, with advantage, be pointed out that a loss of 10 to 12 pints (6 litres) of blood is not more than the body can tolerate without very serious danger, and a pint (about half a litre) is a usual amount for a blood donor to give. The degree of haemorrhage can be roughly estimated by the pallor, coldness, sweating, and collapse of the patient, the rate of the pulse, and the level of the blood pressure.

The first essential is to keep the patient warm by blankets or hot-water bottles, but this does not mean overheating. It is indeed important not to overdo it, for though the skin temperature may be low, this is partly nature's protective means of conserving internal heat, and it is unwise to promote undue dilatation of skin vessels and increase perspiration, which of course favours dehydration. Provided, however, that fluids are allowed—as they should be—some external warmth is advisable and is certainly of much comfort to the patient.

If there is much dyspnoea oxygen should be given, but this is of relatively little value if the haemoglobin to carry the oxygen is very low, and in any case is almost useless if given by the old method of holding a funnel in front of the patient's face. Some means, such as a plastic bag, a mouth-piece fitting the patient, or a nasal catheter must be adopted, but even if this is done it is not likely to be of much help if there is only pallor without cyanosis. If the haemorrhage is severe, the home conditions are bad, the patient is alone or in a hotel, or has little help available, admission to hospital is essential. The dangers of an ambulance journey are far less than those of remaining at home—but it is usually quite safe to wait a short time before having the patient moved. During this time, if the blood pressure is very low, it is worth raising the foot of the bed, but drugs to raise the pressure, such as noradrenaline, are not advisable unless a blood trans-

fusion can be given simultaneously to replace the blood lost. When telephoning the hospital it is helpful to give details of age, pulse rate, and blood pressure rather than the bare statement of a haematemesis. The use of morphine, $\frac{1}{4}$ gr. (16 mg.) hypodermically, to calm the patient and relieve shock is at this stage usually advisable, but the exact dose and the time it is given must be told to whoever takes the patient to hospital. The dangers of morphine in haematemesis are of causing undue depression of the medullary centres, especially in elderly patients with emphysema, or of the case proving to be one of cirrhosis of the liver, in which even the normal dosage may cause deep coma. It must not be forgotten also that in a very appreciable number of patients morphine itself causes vomiting, so that a dangerous vicious circle may be set up. These dangers do not, however, loom so large in the case of the initial dose given at the time of the bleeding, which greatly helps the patient to withstand the ambulance journey to hospital, as in subsequent doses.

During the first hour or two after a severe haemorrhage it is often advised to give the patient nothing by mouth at all, but frequent small drinks of cold water or fruit juice, 2 to 3 fl. oz. (60 to 90 ml.) at a time, are both safe and necessary and give some slight feeling of confidence also.

No medicines, other than morphine, are indicated at this stage, either orally or by injection, but later a sedative is often advisable, and then paraldehyde, 3 or 5 ml. intramuscularly, is very useful, or soluble phenobarbitone, 3 gr. (0.2 g.) subcutaneously, may be used. Alkalis have little if any place in treatment. If the patient remains in his home a nurse should be in attendance, and blood should be taken as soon as possible for grouping and estimation of the haemoglobin. More elaborate analysis of the blood chemistry—non-protein-nitrogen, sodium, chloride, and potassium—requires a skilled laboratory and hospital facilities.

Intravenous Therapy

In cases of moderate severity the question of intravenous therapy may not arise, but in more serious cases, if vomiting continues at all, an intravenous drip must be set up at once. The main indications for giving intravenous fluids are thirst, a dry tongue, oliguria, and a low blood pressure. If a drip is needed, intravenous glucose-saline should be given at the rate of about 100 drops a minute for the first one or two litres, after which the rate must be slower and accurate control of the electrolyte balance of the body becomes essential.

Blood transfusion may be a life-saving measure, but should not be given as a routine. It is the only effective and really physiological means of raising blood pressure if the patient is intensely blanched, shocked, and dyspnoeic; in this case the use of packed cells may enable a large oxygen-carrying capacity to be given in a relatively small volume. Any risk that such transfusion may provoke further bleeding is very small indeed, and if the blood is given slowly is almost negligible, whereas the continuation of a seriously low blood pressure is an urgent danger to life and far outweighs any fears of fresh bleeding resulting from the rise of pressure after transfusion.

In less urgent cases blood need not necessarily be given unless the haemoglobin is below 50% and the systolic blood pressure falls below 80. The patient's pulse should be charted hourly, and if the rate remains at about 120 a minute this generally means that bleeding is continuing, and is one of the clinical indications for blood transfusion. It must be remembered that the haemoglobin estimation immediately after haemorrhage may be deceptively high owing to the concentration of the blood which follows the bleeding, whilst later it is proportionately reduced by dilution with tissue fluids drawn into the circulation to maintain the blood volume. In elderly patients with emphysema it must also be remembered that there may have been some pre-existing polycythaemia and perhaps hypertension which may give a false impression of the haemoglobin and blood pressure.

Less Severe Cases

In many cases, however, the condition is not serious enough for any more elaborate measures to be needed than rest, warmth, and careful feeding. There need be no fear of giving some food and drink at the earliest stage. If the patient is thirsty he must be encouraged to drink in frequent small amounts, and cold milk or fruit drinks may be left at the bedside. Light solids may also be given from the beginning, though here it may be left largely to the patient's feelings whether he takes them or not. In younger patients solids, such as bread-and-butter, apple jelly, cream cheese, boiled egg, custard, junkets, or stewed fruit are usually welcomed within an hour or two of the bleeding, but with older patients it is often better to offer them only purées, malted-milk drinks, or jellies, without pressing them to take more. Chicken, fish, and lightly cooked tender meat may be given on the second or third day if the patient desires. At all costs thirst and hunger must be prevented, and even if the patient is afraid to eat or drink he should be encouraged to do so.

Operation

The later treatment depends upon the cause of the haemorrhage. If the patient is known to have a chronic ulcer, the question of emergency surgery must be considered from the start. Immediate operation, carried out with the help of blood transfusion, offers the chance of stopping the bleeding and radically treating the ulcer by partial gastrectomy at one and the same time, and is often advisable if expert surgical and hospital facilities are at hand. Such treatment is urgently called for if the patient has a second haematemesis within 24 to 48 hours of the first, if his age is over 50, and if there has been a long ulcer history with previous episodes of bleeding. If operation can be safely done at once, the patient is saved both the risk of further bleeding and the time spent on medical treatment, which he may well grudge if after a few weeks he has then to give more time for the resection of his ulcer. The operation, however, tends to be rather easier for the surgeon and safer for the patient if it can be held over till the ulcer has begun to heal well and the bleeding has stopped, so that if the diagnosis of chronic ulcer is not certain then the advantages of immediate laparotomy are less clear and the indication for operation is more uncertain. As a rule, if the haemorrhage continues, the history is suggestive of ulcer, and if the patient is over 60 immediate surgery is usually advisable, but the risks are certainly considerable (see below). A good blood bank and expert nursing are essential requisites.

Later Progress and Management

The stools should be watched for the disappearance of melaena, but if, as is advisable, iron is given early their colour will remain dark, even though the bleeding has stopped, and chemical tests must be utilized to show the presence or absence of blood. The bowels do not usually require any special attention for the first few days, when there is often some diarrhoea with offensive tarry stools. Later, constipation is usual—as may be the case from the beginning—and a weak soap enema may be given on the fourth or fifth day. At the same time a mild laxative such as liquid paraffin and magnesium hydroxide emulsion (*N.F.*, 1952), $\frac{1}{2}$ fl. oz. (14 ml.) once or twice a day should be ordered. It is important to keep the bowels open at this stage, for the altered blood in the intestines is thought to cause pyrexia, which may be a source of anxiety and uncertainty, and, though it is possible that the temperature is, in fact, due to general metabolic changes in body proteins after haemorrhage, it nevertheless often settles after the bowels have been emptied. It is not always easy to decide when a barium-meal examination may safely be undertaken, but in most cases this can certainly be carried out within the first ten days. It is best to do the examination at the earliest moment possible, as the healing of an ulcer is often very rapid after

a haemorrhage, and radiological examination two to three weeks after the bleeding has stopped may give an indecisive result. The dangers of early x-ray examination—within a few days of the bleeding—are slight and consist more in the risk of syncope and exhaustion of the patient than in the barium meal itself causing further bleeding, even though the radiologist may have to palpate fairly deep to demonstrate an ulcer satisfactorily.

Oral iron may be started from the beginning of treatment, and certainly within a week of the bleeding. Organic iron is more readily absorbed than inorganic, and not more than about 25% of such a salt as ferrous sulphate is, in fact, utilized by the body. Some preparation of ferrous gluconate, saccharate, or succinate is the most useful and least irritant, and it is estimated that about 30 mg. of iron in this form will raise the haemoglobin by 1%. Intravenous iron may be necessary but is not always easy to give; it is advisable only when there is extreme intolerance to oral preparations or the blood fails to improve after adequate oral dosage. A suitable oral preparation is ferrous gluconate, 0.3 g. three times a day. The official iron and ammonium citrate mixture very often causes gastric disturbance, as do the pills of ferrous sulphate unless specially coated to reach the intestine undissolved. Ascorbic acid should be given with iron as an aid to healing and to quicken the rate of recovery of the anaemia; the dose should be 200 mg. daily, or double this amount if the patient has for any reason been on a deficient diet before the haemorrhage.

Prolonged bed rest is not advisable after haemorrhage, and the patient may be allowed to get up for the commode from the beginning unless the bleeding has been very severe. He may go to the bathroom and sit in a chair for short periods as soon as his blood pressure has risen to a satisfactory level and the haemoglobin is above 60%.

In most cases he may be allowed out after three to four weeks, but the future treatment must, of course, be guided by the causative condition. The risk of post-haemorrhagic thrombosis is increased by long immobility in bed and can probably be lessened by early ambulation and the free administration of fluids.

Treatment of Oesophageal Varices

Oesophagoscopy will usually show the presence of varices, as may in many cases a barium swallow. Numerous methods of local treatment have been devised, including injection of the veins with sclerosing fluids, but none has given much success. Various operations designed to reduce portal tension of the porto-caval shunt type have been shown to reduce the varices in some cases, and ligation of the splenic artery has also been advocated, but with little to support it. Spontaneous disappearance of the varicosities is not unknown.

Prognosis

The risk of immediate death from haemorrhage is highest in cases of ruptured oesophageal varices secondary to portal hypertension, and it is a frequent mode of death in Banti's syndrome; in cases of cirrhosis over half the patients die within a year of the bleeding. In cases of haemorrhage from chronic peptic ulcer occurring in patients under 40 years of age death is rare; the risk is less than 1 in 20 amongst patients under 50, but rises to at least 1 in 10 in those over 60 and to about 1 in 5 in those over 70. In bleeding due to acute erosions death is also rare, even though the bleeding may be very severe, and the mortality in younger subjects is certainly not more than 1%. Loss of blood by vomiting is in general associated with a worse prognosis than when there is only melaena, perhaps because the loss is more rapid in the former case.

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