

THE MEDICAL CARE OF THE PATIENT WITH HAEMATEMESIS AND LIVER FAILURE*

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Since the beginning of medical history the liver has been the subject of much speculation, study and experimentation. The ancients accorded it the position of first importance in bodily function. Galen regarded the liver as the focus of animal heat, as an organ intended for the formation of blood, and for the origin of the veins of the body. Vesalius corrected many of these incorrect speculations, and the dynamics of portal hypertension have only recently been appreciated.¹ The neurological manifestations of hepatic disease were written about by Hippocrates in 400 B.C.²

In 1900 Preble³ discussed 60 cases of fatal gastro-intestinal haemorrhage, due to cirrhosis of the liver.

Therapeutic Management

Patients with portal hypertension of differing aetiologies may bleed for various reasons. They may bleed from oesophagogastric varices, or from gastric or duodenal ulcers, to which patients with portal hypertension appear to be more prone. They may bleed after an acute alcoholic bout, which may result in an acute gastritis. They may, rarely, bleed from other sites due to varices of the small bowel, or from an increased bleeding tendency due to deficient clotting factors. The patient we have in mind for this discussion is one who has had a haemorrhage and is in liver failure—a common combination.

The combination of liver decompensation and haemor-

rhage in portal hypertension is indeed a grave one, and it is essential that the medical team in charge of the case should have a clear and detailed programme of action. The aspects of haemorrhage and liver decompensation will be dealt with under separate headings, but this protocol will be understood to form a whole in the therapeutic approach towards such a patient.

DIAGNOSIS AND TREATMENT OF BLEEDING OESOPHAGEAL VARICES

Diagnosis

These patients will often present with a picture of gastro-intestinal bleeding, portal hypertension, and the tell-tale signs of liver disease which are seen in the hands, skin and other organs. They usually present with a slow ooze and melaena rather than with frank haematemesis. The intestines may be filled with blood before haemorrhage is clinically recognized.

Gastro-intestinal bleeding in a patient with compromised liver function, especially liver cirrhosis, has injurious effects on the liver cells due to anaemia caused by lowered oxygenation of the blood. Increased metabolic demands are made on the liver cell during active protein catabolism following intestinal haemorrhage. Also blood pressure may fall and reduce hepatic arterial perfusion with resultant cellular necrosis of regenerating liver nodules.

At the outset it is important to arrive at a correct diagnosis as to the anatomical site of the bleeding. Non-alcoholic patients usually bleed from varices and it is

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here where a careful history, clinical examination and special investigations prove invaluable.

Oesophagoscopy, a positive BSP retention and raised blood ammonia levels may be helpful. The Sengstaken oesophageal tamponade technique may aid diagnosis. Water-soluble Gastrographin examination may be done on the bleeding patient. Splenic venography is useful in good hands, where the diagnosis of varices is doubted. Radioactive iodine injected into the spleen with counters over the liver and lower end of the sternum, may detect a shortened circulation time to the oesophagus in the patient with varices.

Treatment

General Measures for Haemorrhage

Morphine is avoided as this may aggravate the coma in the patient. If any sedation is required in a restless patient, barbiturates such as phenobarbitone or amylobarbitone in low dosage (30 mg. *t.d.s.*) may be tried. Sparine has also been used. The drugs that are not detoxified in the liver, but are excreted in the urine, are preferable.

Blood transfusion is required to maintain the haemoglobin above 10 G/100 ml. Fresh blood is used as it is still rich in the clotting factors which are defective in these patients. At least 3 or 4 pints must be kept in reserve. Veins are precious and should be preserved by using skin venepuncture, rather than the cut-down technique.

Oesophageal Tamponade

The tube of Sengstaken and Blakemore is used. It is a 3-lumened tube with a gastric and oesophageal balloon, and a gastric tube. The lumen of the gastric tube must be wide enough to allow the aspiration of blood clots.

Method of insertion. The pharynx is well sprayed with 4% lignocaine, and the well-lubricated tube is passed either through the mouth, or nose if there is no severe septal deformity. A mixture of 20 ml. of 70% Diodone in 100 ml. water is used to fill the gastric balloon which is placed *in situ* under the X-ray screen, snugly fitting into the fundic portion of the stomach.

The oesophageal balloon is then inflated to 30 mm.Hg, careful note being taken of any respiratory embarrassment that may be caused. The tube is fixed either by strapping it to the face, or over a pulley with weights, with not more than 600 G traction being used. The gastric and oesophageal balloon clamps should be clearly marked.

Complications of tamponade include upward displacement with asphyxia; ulceration of oesophagus and pharynx due to prolonged and repeated insertions; failure to deflate the gastric balloon; failure to pass the tube; the patient may bite through the tube; oesophageal rupture⁴ due to forcible extraction of tube with an inflated gastric balloon.

Length of use. The balloons must be deflated within 36 hours and removed after 24 hours. If bleeding resumes one may reinflate and transfer the patient to theatre for surgery.⁵ Hunt, at St. Bartholomews, prefers to use this method to resuscitate his patients in preparation for surgery. Balloons should be fully tested for leaks before use and should be discarded after use.

Intravenous Vasopressin

Vasopressin (Pitressin) is used because it specifically lowers portal venous pressure, by constricting the splanchnic arteriolar bed, causing an increased resistance to inflow. This is thought to be the reason for its effect in controlling the bleeding from oesophageal varices.

Twenty units of vasopressin are given in 100-200 ml. 5% dextrose and water over 10-20 minutes. There is a transient rise in systemic arterial pressure and the portal pressure falls for about three-quarters of an hour. Side-effects such as pallor, abdominal colic and evacuation of the bowel are often seen. If these reactions are absent the vasopressin has lost its biological activity. It should be used with caution in patients with coronary artery disease. Rebleeding is common after an hour, but this treatment may be repeated as desired. No alternative drug has been found to be of use. Merigan and other workers in Boston⁶ concluded after a controlled study of 30 patients that the bleeding was controlled for 24 hours in 55% of patients, and that the effect was transient in most cases. The mortality is not altered by the use of the drug, and its main value is in pre-operative preparation of cases with less active hepatocellular disease. Sherlock prefers this method of therapy to balloon tamponade.⁷

Intragastric Cooling

Wangenstein,⁸ and Walker *et al.*⁹ have recently presented arguments in favour of this method of control for haemorrhage. Sherlock and her co-workers insert a gastric balloon which is not put on traction. The fundus of the stomach was cooled and arrest of bleeding was complete in 8 cases. Rebleeding is common and this technique is recommended for use in cases being prepared for surgical correction. The complications of cooling are far less than those where a Sengstaken-Blakemore tube was used. The method is expensive and limited to large centres with the requisite equipment for intragastric freezing.

TREATMENT OF HEPATIC COMA AND PRECOMA

Prevention

1. The use of hypnotics must always be considered with care. Barbiturates are preferred, especially the long-acting, short-chain drugs excreted by the kidney (see above).
2. Acute infections are vigorously treated and with the correct antibiotic.
3. Due attention is given to the fact that ammonium salts and urea used for diuresis are detrimental to such patients.
4. Gastro-intestinal bleeding is treated with vigour.
5. Abdominal paracentesis may precipitate coma and should be avoided if possible.
6. Acetazolamide and chlorothiazide are discontinued.

General Therapeutic Measures

(i) *Exact nursing* is the *sine qua non* in these patients, especially if deep coma is present. The patient should be kept clean, and the intake and output of fluids should be measured (an indwelling catheter may be necessary). Vital signs are recorded daily, or more frequently, as may be required by the condition of the patient; a record of all clinical, laboratory and therapeutic details is desirable. Optimum treatment may be had in a metabolic unit if it is available. The treatment may be considered similar to that of a diabetic coma, except that no specific therapy like insulin is available in these cases.

(ii) *Daily measurement of:* (a) haematocrit readings to assess the hydration state of the patient; (b) blood urea and blood CO₂ content, to allow the therapeutic team to assess the

state of the kidneys; and (c) regular estimations of serum electrolytes, especially to assess the state of body sodium and potassium, are done to control replacement if required. Blood sugar estimations are required to detect the occasional case where coma may be due to a hypoglycaemic state. Stools are all inspected and tested for blood if there is no visible evidence of bleeding.

(iii) *Diet.* The minimal requirement for these patients is 1,600-2,000 calories, which may be given in the form of glucose drinks or highly concentrated (20% or 40%) intravenous glucose infusions. These solutions are given through an indwelling catheter placed in the superior vena cava or innominate vein. An alternative is 10% laevulose, which produces much less irritation of the vessel wall.

All dietary protein is stopped in the acute phase, and protein may be added to the diet in increments of 20 G every second day in the recovery phase. This protein must be divided into 4 portions during the day and not given as an additive to 1 meal. Should the patient relapse, he should then be placed on a protein-free diet once more. Chronic patients may not be able to tolerate more than 40-60 G of protein per day.

The restriction of protein over a confined period is not harmful, but must not be extended unnecessarily. Supplements of vitamin B complex and vitamin K are given parenterally.

(iv) *Antibiotics.* Bacterial action on ingested protein and blood may be arrested by various broad-spectrum antibiotics. Many of the usual antibiotics carry a very real risk of the patient developing an acute staphylococcal enterocolitis.

The antibiotic most widely used is Neomycin which although rather expensive, is poorly absorbed. Only rarely are there any undesirable side-effects, such as ototoxicity or nephrotoxicity. Neomycin should be given in divided doses of 6 G/day. Patients will tolerate 4 G/day for prolonged periods with little ill-effect. The routine use of this antibiotic is essential in cases of acute coma of hepatic origin.

(v) *Purgation.* Free bowel action must be encouraged in all cases, and this is best done by water enemata at least once a day, and magnesium sulphate purgation.

(vi) *Potassium.* Special emphasis is placed on the correction of low potassium states. Intravenous infusion of potassium should be used with care and only given slowly in an emergency.

(vii) *Delirium tremens.* If the patient should develop delirium tremens, alcohol should be avoided in the therapeutic programme. Small doses of chlorpromazine are used in preference.

(viii) *Treatment of doubtful value.* Glutamic acid to inactivate ammonium ions to harmless glutamine, has not proved of value in the therapy of hepatic coma. Arginine and corticosteroids are of no value. Haemodialysis has been used in acute viral hepatitis with coma, but is not of any proven value; heparinization makes this treatment in bleeding varices impractical.

Oral cation exchange resins for the treatment of ammonia intoxication have been reviewed by Zuidema *et al.*¹⁰ By giving potassium and sodium cycle polystyrene resins they found that they could produce a lowering of the blood ammonia; 20 G of resin is dissolved in 30 ml. Sorbitol and given 6-hourly by gastric tube or enema. They recommended this treatment for bleeding varices in coma and felt that it was superior to other methods for lowering blood ammonia. Poor results were obtained with cases of long-standing liver disease with failure, and this treatment was recommended for use in patients with

bleeding varices in hepatic failure, or after surgery for portocaval anastomosis.

Protocol for Acute Bleeding from Oesophago-gastric Varices with Liver Failure

1. The patient is seen by a joint medical and surgical team.
2. Initial efforts are made to establish the site of haemorrhage.
3. Shock and hypovolaemia are treated.
4. Special nursing care is instituted.
5. Bleeding is controlled by methods described.
6. Coma is controlled as detailed.

CONCLUSIONS

Hunt⁵ feels that a realistic approach is required. Bleeding in these cases is usually of 2 types: the first kind stops with medical treatment, but in the second type the bleeding starts again or is continuous and is fatal. It is this latter group that is the immediate concern of the surgeon and the decision has to be made on what to do and when to do it.

As soon as it is known that bleeding is continuing despite medical treatment (usually 36-48 hours, or earlier in cases with a severe haemorrhage), surgery is indicated. Hunt operates in spite of coma, ascites and jaundice, which he says are the usual concomitants of the cirrhotic patient; he stresses that multiple mechanical methods of attempting to attain haemostasis may delay surgery until it is too late. Certain adjunct procedures are also mentioned such as oesophagoscopy with injection of 5% phenol and almond oil alongside a bleeding point.

Balloon tamponade is used as a first-aid measure to prepare the patient for surgery, and if the patient bleeds after 24-36 hours when the balloon is deflated, it is reinflated and the patient is taken to the theatre for a Boerema-Crile operation (undersewing of the varices).

SUMMARY

The medical management of the combination of haematemesis and liver failure is discussed. Emphasis is placed on the correct diagnosis of the bleeding site and the management of shock and hypovolaemia. Care should be taken when choosing hypnotics. Certain measures to lower blood ammonia are enumerated. A plea is made for a positive approach to this problem, close liaison between surgeon and physician being essential.

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