of hemorrhage with the use of balloon tamponade was achieved in 525 instances or 78% of the time. Major complications occurred in 14% of the cases and lethal complications occurred in 3%

More recent studies from Panés,[33] Haddock,[16] and Hunt[22] have shown that in 380 instances of variceal bleeding in which balloon tamponade was employed, control of hemorrhage was achieved in 357 (94%). Rebleeding, however, occurred in 137 cases (38%). Major complications were 0%, 10%, and 15% for Hunt's,[22] Panés,[33] and Haddock's[16] series respectively. Haddock[16] noted a 6.5% mortality rate secondary to tube placement, whereas Panés[33] and Hunt[22] had no mortalities attributable to use of balloon tamponade.

In a study by Teres et al,[46] esophageal tamponade was compared with intravenous vasopressin and nitroglycerin in the treatment of acute variceal bleeding. Balloon tamponade was significantly more effective than the drug therapy in controlling hemorrhage—86.5% versus 66%—but no other significant differences were found with respect to rebleeding, complications, or mortality. In two previous studies,[21,35] there were no significant differences noted between the two modalities.

Sclerotherapy, on the other hand, has been shown to be superior to balloon tamponade in the emergency treatment of bleeding esophageal varices.[31,34] The authors concur with Terblanche[44,45] on the management policies for the acute variceal bleeder. This involves pharmacologic therapy in the form of vasopressin and nitroglycerin infusions to lower portal vein pressures, followed by emergent endoscopy to confirm the diagnosis.[44,45] If possible, sclerosis should be undertaken at this time. When pharmacologic and endoscopic modalities are unsuccessful in controlling bleeding, balloon tamponade should be employed.[44,45] Hopefully, this will control the acute bleed, allow for stabilization of the patient, and provide time to plan for definitive treatment.

COMPLICATIONS OF BALLOON TAMPONADE

Complication rates range from 0% to 41% with the use of balloon tamponade and lethal complications have been reported to range anywhere from 0% to 20%.[10,15,22] In general, however, major complications occur in approximately 10% of patients whereas, in more recent series, lethal complications are reported at 0%.[3,4,16,22]

The most commonly reported major complication is pulmonary aspiration.[16,22,39] Less commonly, esophageal erosion or rupture, respiratory obstruction, alar necrosis, and balloon migration have been reported.[16,22,39] Isolated cases of hemoptysis and tracheoesophageal fistula formation, jejunal rupture, and thoracic duct lymph obstruction have been noted in the literature.[1,4,37] Minor complications include nasopharyngeal bleeding, chest pain, and balloon impaction.[5,15]

Pulmonary Aspiration

Pulmonary aspiration may be reduced by completely emptying the stomach prior to passing the balloon as well as providing continuous monitoring and removal of esophageal secretions. Control of the airway via an endotracheal tube with a cuffed balloon also is helpful, particularly if the patient has an altered mental status.

Esophageal Erosion or Rupture

Esophageal erosion and rupture generally can be avoided as long as the gastric balloon is not inflated in the esophagus and tamponade is not maintained for excessive periods of time. Excessive inflation of the esophageal balloon is also to be avoided and continuous monitoring by sphygmomanometer is warranted.

Balloon Migration

Balloon migration may also lead to esophageal rupture but, more commonly, one sees airway obstruction when the balloon migrates upward. A pair of bandage scissors should be at the bedside and if the patient develops respiratory distress, the gastric and esophageal balloon ports should be cut immediately. It is also important to note that the most common error limiting the efficacy of tamponade is improper positioning of the gastric balloon.[48]

Alar Necrosis

Alar necrosis is most easily prevented by avoiding pressure by the tube or tension device on the nares.

SUMMARY

The management of acute variceal bleeding continues to challenge those who care for patients with portal hypertension. Survival depends on rapid institution of an established protocol for resuscitation, diagnosis, and management of the patient.

Balloon tamponade plays an important part in the management of this problem along with pharmacologic and endoscopic modalities. It is important in closing, however, to note that guidelines for use cannot compensate for lack of experience and the authors agree with Vlavianos and colleagues[48] in stating that without experience in its use, balloon tamponade is of limited value.

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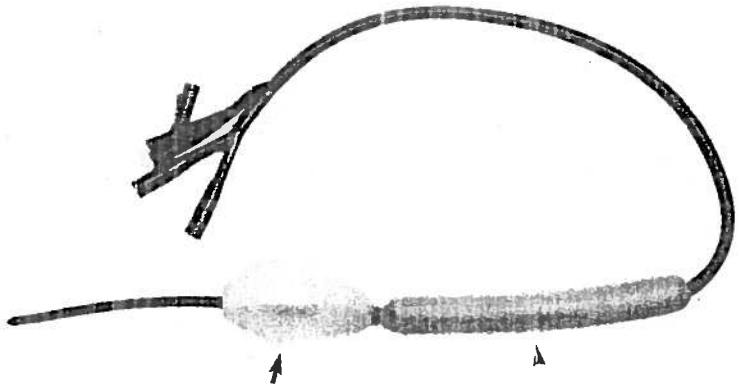

Figure 1. Minnesota tube. Arrows point to the gastric and esophageal balloons. The inflation ports are capped while the aspiration ports are open.

Technique of Insertion of Gastroesophageal Balloon* (Fig. 2)

- Test all balloons and tube channels.
- Attempt to empty the stomach by saline lavage using either a nasogastric or Ewald tube.
- Airway control should be considered. Although not mandatory, it may be advisable to endotracheally intubate the patient prior to passage of the balloon. If the patient has an altered mental status, then endotracheal intubation should be strongly entertained as part of the insertion technique. Even though passage of the balloon may be more difficult, there is less chance of airway compromise and aspiration.
- Refrigerating the tube may stiffen it and make it easier to pass. Other techniques, such as longitudinally incising an Ewald tube and placing the Minnesota tube inside, or guiding the balloon into the stomach via a silk suture passed through a nasogastric tube, have also been described as making passage less difficult.
- Lubricate the tube with lidocaine jelly. A nasal or oral route may be used.
- The patient should be semi-erect during passage of the tube but if this is not possible, the patient should be on his or her left side with his or her head slightly down.
- The tube should be passed into the stomach. The gastric port is flushed with air while auscultating over the epigastrium to ensure that the tube is in the appropriate position. The gastric balloon is then inflated with 50 to 100 mL of air and an abdominal radiograph is obtained to assure that the gastric balloon is below the diaphragm. If the film reveals that the balloon is in the stomach, it should be inflated slowly to a total of 250 to 300 mL of air.
- The tube is then pulled up so that the gastric balloon impacts on the gastroesophageal junction. Two to three pounds of tension should be applied and maintained. How this tension is maintained varies from simply placing a piece of foam rubber around the tube and wedging it into the nose to a helmet-mounted constant traction spring. The authors favor the use of a well-padded, single-bar football helmet because it avoids undue pressure on the nasal cartilage and is relatively easy to use.
- The gastric and esophageal lumens should next be placed on low intermittent suction. Remember, these are not sump-type lumens and therefore are not as effective if placed on continuous suction.
- If, after a period of 4 hours, the variceal hemorrhage does not appear to be controlled, the esophageal balloon should be inflated to 25 to 45 mm Hg. This pressure should be monitored continuously by a bedside sphygmomanometer. The esophageal balloon generally should not remain inflated for more than 24 hours because of the risk of mucosal necrosis.
- Subsequent radiographs to check the position of the balloons should be made at 24-hour intervals or sooner should the patient's status change because balloon displacement has been described.
- The patient should be monitored continuously for signs of respiratory distress, aspiration, and chest pain. Suction equipment as well as a pair of scissors to cut the balloon ports, if necessary, should be at the bedside.
- If variceal hemorrhage ceases, the tube is left in place with the gastric or gastric and esophageal balloons inflated for 24 hours. Hunt,[22] recently has recommended 48 hours. If no rebleeding occurs during this time, the esophageal balloon is deflated if it had been inflated. The gastric balloon is left inflated for an additional 24 hours and, again, if there is no rebleeding, it is deflated. After both balloons are deflated the tube is left in place an additional 24 hours and then removed if no further bleeding occurs.
- If bleeding recurs, the appropriate balloons should be reinflated while alternatives to control hemorrhage are considered.

*References 2,3,11,16–18,20,22,23,29,36.

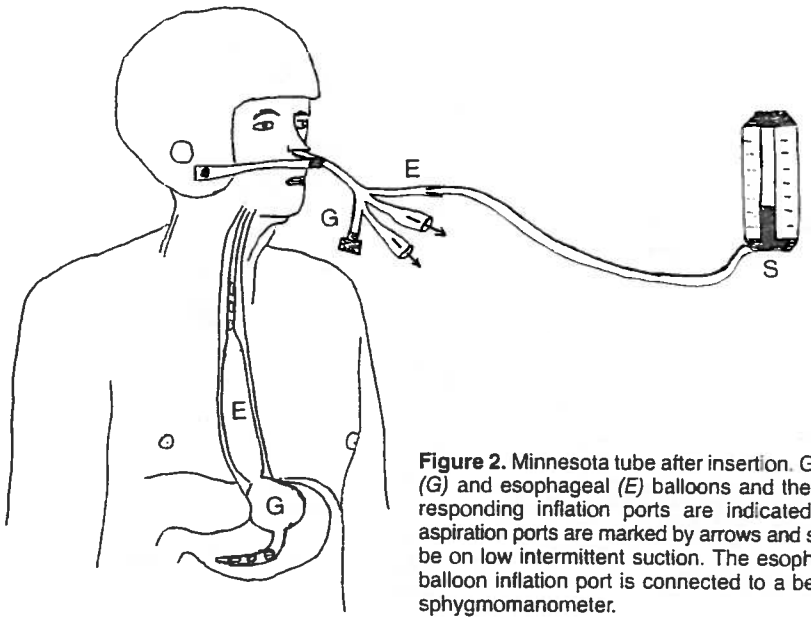

Figure 2. Minnesota tube after insertion. Gastric *(G)* and esophageal *(E)* balloons and their corresponding inflation ports are indicated. The aspiration ports are marked by arrows and should be on low intermittent suction. The esophageal balloon inflation port is connected to a bedside sphygmomanometer.

EFFICACY OF BALLOON TAMPONADE

Chojkier and Conn[7] reviewed their experience along with the literature regarding the efficacy of balloon tamponade from 1969 to 1979. They found that in 494 patients, there were 672 episodes of esophageal variceal bleeding. Control

eeding while other modalities of definitive treatment are being arranged. Aspects balloon tamponade are discussed in further detail later in this article.

ranshepatic Embolization

Transhepatic embolization is used to obliterate the variceal inflow tract, most otably the coronary vein, as well as to thrombose esophageal varices. A variety f agents have been employed, with initial success in controlling bleeding rang- ng from 45% to 90%.[13] Rebleeding has been reported in up to 86% of patients vithin days to months, however.[13] The incidence of intra-abdominal bleeding is pproximately 20% and mortality from the procedure is 5% to 10%.[13] Material nay embolize into the portal vein and aggravate the portal hypertension. Trans- iepatic embolization therefore is used only as a temporizing procedure in very poor risk patients who continue to bleed despite other measures.

Devascularization

Devascularization procedures are those that ligate the individual varices. Suguira and colleagues[42] described a procedure of transthoracic esophageal tran- section and devascularization combined with transabdominal esophagogastric devascularization, splenectomy, preservation of the vagi, sparing of the coro- nary-azygous system, and finally fundoplication and pyloroplasty. The goal of the procedure is to reduce the gastroesophageal venous pathway of portal hyper- tension while at the same time preserving hepatic function. Although their results with this procedure have been excellent (overall mortality of 3% to 5.2%), no U.S. investigators have been able to reproduce these results.

Surgery

Surgical shunt procedures are ideally undertaken in Child's A or B patients who continue to bleed despite vasopressin, sclerotherapy, or balloon tamponade and before excess transfusion.[27,43,47] These procedures probably are no longer generally indicated in Child's C patients who continue to bleed from varices. To accurately delineate the anatomy as well as assess portal hemodynamics, selective celiac or superior mesenteric arteriography with venous phase studies should be performed.

Nonselective shunting will control bleeding and prevent ascites formation, but there is a significant risk of developing early or late hepatic failure with subsequent encephalopathy.[30,43] This hepatic failure appears to result from the loss of portal perfusion. The goal of selective shunting is to stop hemorrhage while at the same time maintaining portal perfusion, thus preventing the devel- opment of encephalopathy.[30,43]

In randomized trials comparing distal splenorenal shunts (DSRS) with por- tosystemic shunt, encephalopathy was significantly decreased in the DSRS group. There was no significant difference in survival at 12 years, however.[9,30] Zemell et al[51] recently reported their experience in eight patients who underwent trans- jugular intrahepatic portosystemic shunt placement (TIPS). There were minimal morbidity and no deaths secondary to the procedure. One patient had rebleeding

19 days postprocedure and this was controlled by percutaneously enlarging the intrahepatic stent. All shunts were patent at 4 months and no encephalopathy developed.

Liver Transplant

Hepatic transplantation is the only procedure that can cure both the portal hypertension and the underlying liver disease and is the treatment of choice for patients with end-stage liver disease who are otherwise fit.[43]

DEVELOPMENT OF BALLOON TAMPONADE

Esophageal tamponade was first described by Westphal in 1930,[50] when an esophageal sound was used to control a variceal hemorrhage. In 1947, Rowntree et al[38] described the attachment of an inflatable latex bag to the end of a Miller-Abbott tube to successfully control variceal bleeding.

Sengstaken and Blakemore[40] first reported the technique of double balloon esophageal and gastric tamponade to control bleeding varices in 1950. The tube that they developed had three channels—one for gastric balloon inflation, one for esophageal balloon inflation, and one for gastric aspiration.

Linton[26] introduced a single balloon tube in 1953. This tube consisted of a gastric balloon and a channel for gastric aspiration. The Linton tube was designed to determine whether upper gastrointestinal bleeding was arising from above or below the cardia, but by compression of the coronary vein, it was effective in controlling variceal hemorrhage.[26] The Nachlas modification of the Linton tube was introduced in 1955 and consisted of the addition of a third channel to aspirate secretions above the gastric balloon.[32]

In 1968, Edlich and associates, at the University of Minnesota,[12] introduced a modification of the Sengstaken-Blakemore tube. The Minnesota tube had an additional channel that was used for aspiration of esophageal secretions above the balloons, thus decreasing the risk of aspiration pneumonitis (Fig. 1). The Minnesota tube is preferable to the Linton-Nachlas tube because it provides the advantage of inflating the esophageal balloon should hemostasis not be achieved by the gastric balloon alone.[12]

INSERTION OF A GASTROESOPHAGEAL BALLOON

It is generally agreed that active variceal hemorrhage unresponsive to pharmacologic or endoscopic control should be temporarily controlled by balloon tamponade. Relative contraindications to insertion of a gastroesophageal balloon are the presence of an esophageal stricture or recent esophageal surgery.[29]

Table 1. CLASSIFICATION OF PORTAL HYPERTENSION

Presinusoidal
Increased hepatopetal flow
Hepatic arterial-portal venous fistula
Splenic arteriovenous fistula
Massive splenomegaly
Extra-hepatic portal venous obstruction
Development defects of portal vein
Portal vein thrombosis
Omphalitis
Sepsis
Trauma
Enterocolitis
Extracellular fluid depletion (severe)
Blood dyscrasias
Neoplasm
Idiopathic
Intra-hepatic portal venous obstruction
Schistosomiasis
Myeloproliferative diseases
Sarcoidosis
Congenital hepatic fibrosis
Arsenic toxicity
Primary biliary cirrhosis (not secondary to biliary tract disease in gallbladder or large bile ducts)
Neoplasm
Primary portal hypertension
Sinusoidal (occurs with elements of intrahepatic presinusoidal or postsinusoidal portal hypertension)
Alcoholic and nutritional cirrhosis
Post-necrotic cirrhosis
Biliary cirrhosis (secondary to disease of gallbladder and large bile ducts)
Hemochromatosis
Wilson's disease
Postsinusoidal (Budd-Chiarl Syndrome)
Intrahepatic venous obstruction
Alcoholic hepatitis
Oral contraceptives
Extrahepatic venous obstruction
Constrictive pericarditis
Right sided heart failure
Congenital obstruction of suprahepatic inferior vena cava
Trauma, sepsis, myeloproliferative disorders, neoplasm pregnancy, contraceptives

inciting event, however, is not known. Of the initial upper gastrointestinal bleeds in cirrhotics, 60% are due to esophageal varices, 20% are due to gastritis, and 20% are due to Mallory-Weiss tears.[6] If untreated, the mortality rate from these bleeds is approximately 50% to 70%. In treated patients, 60% go on to have a second major hemorrhage within 1 year.[6] Hence, the goal in these patients is to identify and control the hemorrhage promptly and efficiently.

TREATMENT OPTIONS FOR ACUTE VARICEAL BLEEDING

Although there are some areas of disagreement with regard to the treatment of acute variceal bleeding, all authors agree that the patient should be stabilized

and have his or her diagnosis confirmed. Resuscitation should begin with volume replacement in the form of blood and blood products. Overzealous use of normal saline should be avoided because it can worsen the ascites and the portal hypertension caused by the secondary hyperaldosteronism that frequently is present in patients with cirrhosis. Monitoring in an intensive care unit is necessary, frequently with invasive techniques. Gastritis and ulcer prophylaxis is warranted, particularly prior to diagnostic confirmation. Nasogastric lavage is beneficial in that it can help clear clots and make subsequent endoscopic visualization better, can decompress the stomach to permit gastric contraction, does not appear to cause variceal bleeding, and can decrease the risk of possible aspiration. Sedation should be used cautiously because of the impaired liver metabolism.

After resuscitation is initiated, the patient should undergo emergent endoscopy to confirm the diagnosis. At this time, classification using Child's scale is useful in management.[6]

Vasopressin

Vasopressin is employed to decrease portal pressure by decreasing splanchnic blood volume through vasoconstriction of the splanchnic arterioles.[8] The intravenous route is associated with fewer complications and is as effective as intra-arterial infusions.[8] Initially, 20 units are given over 20 minutes and then a continuous infusion of 0.2 U to 0.8 U/minute is started. This should be continued until the bleeding stops or for 3 days. The infusion should then be tapered by a rate of 0.1 U/minute every 6 to 8 hours.

Because of the associated coronary artery vasoconstriction and propensity toward hypertension and sinus bradycardia, nitroglycerin infusions should be started with the vasopressin.[9] Close attention to fluid balance is also necessary to detect excess fluid retention.

Sclerotherapy

Sclerotherapy controls 80% to 90% of initial variceal bleeds, but rebleeding has occurred in anywhere from 2.9% to 66% at 2 years postsclerosis.[49] In addition, the effect on survival is uncertain because the underlying disease process is not changed.[28] When performed, sclerotherapy should be continued at intervals until all varices are obliterated and patients should have routine follow-up. Sclerotherapy is useful in patients who cannot tolerate surgical intervention and as an adjunct to vasopressin in the acute situation. It has also found use in patients who rebleed after shunt surgery and as a temporizing measure to allow time for improvement and upgrading of the patient's Child's classification. Sclerotherapy is not as useful for gastric varices because these are technically difficult to inject.[25] Complications of sclerotherapy include perforation, dysphagia, ulceration, and chest pain.[25,28,49]

Balloon Tamponade

Balloon tamponade is most useful today if bleeding continues despite vasopressin or sclerotherapy. It is a temporizing measure to control acute variceal

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SENGSTAKEN-BLAKEMORE TUBE PLACEMENT

Use of Balloon Tamponade to Control Bleeding Varices

Michael D. Pasquale, MD, and Frank B. Cerra, MD

OVERVIEW OF PORTAL HYPERTENSION

Portal hypertension is defined as an increase in the portal vein pressure above the normal 5 to 10 mm Hg. This may result from either obstruction of antegrade portal flow or, less commonly, increased portal blood flow.[9,19] In the presence of portal hypertension, collateral venous circulation develops so as to decompress the high pressure portal system into the low pressure systemic circulation.[6,19] Enlargement of hemorrhoidal plexuses, caput medusa, and esophageal varices result from this collateralization.

Portal hypertension may be classified as being either presinusoidal, sinusoidal, or postsinusoidal as shown in Table 1.[41] It is important to note that there is hepatic parenchymal damage with sinusoidal and postsinusoidal portal hypertension, whereas the hepatic parenchyma is spared with presinusoidal portal hypertension.[6] The most common cause of portal hypertension worldwide is hepatosplenic schistosomiasis but in the United States, it is cirrhosis of various types.[19,41] The most important pathologic feature of cirrhotic portal hypertension, which involves intrahepatic presinusoidal, sinusoidal, and postsinusoidal features, appears in the microcirculation of the liver.[9] This microcirculation is obstructed by a reduction in volume and distortion of the hepatic sinusoids caused by hepatic fibrosis and by pressure of regenerating liver nodules.[9] This then leads to obstruction of antegrade flow with resultant portal hypertension.

Thirty percent of all patients with cirrhosis develop esophageal varices, and of these, 30% bleed.[9,19] Most of these patients bleed within 2 years of the diagnosis of their esophageal varices. Although the degree of portal hypertension has not been shown to correlate with bleeding, the size of the varix does.[24] The exact

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