



Official reprint from UpToDate®

www.uptodate.com © 2023 UpToDate, Inc. and/or its affiliates. All Rights Reserved.



Wolters Kluwer

Methods to achieve hemostasis in patients with acute variceal hemorrhage

AUTHORS: [Jasmohan S Bajaj, MD](#), [Arun J Sanyal, MD](#)**SECTION EDITOR:** [John R Saltzman, MD, FACP, FACG, FASGE, AGAF](#)**DEPUTY EDITOR:** [Anne C Travis, MD, MSc, FACG, AGAF](#)

All topics are updated as new evidence becomes available and our [peer review process](#) is complete.

Literature review current through: **Sep 2023**.

This topic last updated: **Nov 03, 2022**.

INTRODUCTION

Among patients with cirrhosis, varices form at a rate of 5 to 15 percent per year, and one-third of patients with varices will develop variceal hemorrhage [1]. The current treatment options for acute variceal hemorrhage include medications ([vasopressin](#), somatostatin, and their analogs), endoscopy, transjugular intrahepatic portosystemic shunt placement, and surgery.

This topic will review the pharmacologic, endoscopic, radiologic, and surgical methods used to achieve hemostasis in patients with acute variceal hemorrhage. The general management of patients with variceal hemorrhage, primary and secondary prophylaxis against variceal hemorrhage, and a detailed discussion of endoscopic variceal ligation are discussed separately. (See "[Overview of the management of patients with variceal bleeding](#)" and "[Primary prevention of bleeding from esophageal varices in patients with cirrhosis](#)" and "[Prevention of recurrent bleeding from esophageal varices in patients with cirrhosis](#)" and "[Endoscopic variceal ligation](#)".)

The management of variceal hemorrhage is also discussed in a [2017 guideline](#) from the American Association for the Study of Liver Diseases [2], a [2014 guideline](#) from the [American Society of Gastrointestinal Endoscopy](#) [3], a [2015 international consensus statement](#) (Baveno VI) [4], and a 2015 guideline from the [British Society of Gastroenterology](#) [5]. The discussion that follows is consistent with those guidelines [6].

DEFINITIONS

The existing literature is confounded by the variable use of terminology across studies. As a result, several definitions were agreed upon during a consensus conference, which simplify evaluation of published studies and render new studies more comparable [7]:

- **Time zero** – The time of admission to a medical care facility.
- **Clinically significant bleeding** – Defined by a transfusion requirement of two units of blood or more within 24 hours of time zero together with a systolic blood pressure below 100 mmHg, a postural systolic change of more than 20 mmHg, and/or a resting pulse rate above 100 beats/min at time zero.
- **Acute bleeding episode** – Events in the interval of 120 hours (five days) from time zero.
- **Treatment failure** – Failure of therapy is defined by any of the following criteria if they occur within 120 hours of time zero:
 - Fresh hematemesis or >100 mL of blood in the nasogastric aspirate >2 hours after the start of a specific drug or endoscopic treatment
 - Development of hypovolemic shock
 - Drop in hemoglobin of ≥ 3 g within a 24-hour period
- **Early rebleeding** – Bleeding that occurs >120 hours but <6 weeks from time zero, provided initial hemostasis was achieved and maintained for at least 24 hours.
- **Late rebleeding** – Bleeding that occurs ≥ 6 weeks from time zero.

NATURAL HISTORY AND PROGNOSIS WITH TREATMENT

Older studies suggest that variceal hemorrhage will stop spontaneously in approximately half of patients, though rebleeding is common [8]. Bleeding is less likely to stop spontaneously in patients with Child-Pugh class C cirrhosis, active variceal bleeding at the time of endoscopy, or if the hepatic venous pressure gradient (HVPG) is greater than 20 mmHg [1]. Treatment with either endoscopic variceal ligation or endoscopic sclerotherapy is associated with decreases in both rebleeding rates and mortality. Even with current treatment, mortality at 6 weeks following variceal bleeding is 10 to 20 percent.

If the bleeding stops spontaneously, it is estimated that rebleeding will occur in approximately one-third of patients within six weeks (early rebleeding) and in 70 percent of patients over the long-term [9-11]. Approximately half of the patients with early rebleeding will rebleed within three to four days of the index bleed.

Factors associated with early rebleeding include [1]:

- Age greater than 60 years
- Cirrhosis due to alcoholic liver disease
- Severe bleeding initially (hemoglobin less than 8 g/dL)
- Thrombocytopenia
- Encephalopathy
- Ascites
- Bleeding seen at the time of endoscopy
- Bleeding from gastric varices
- Large varices
- Red color signs or platelet clot on the varices
- High HVP
- Renal failure

Factors associated with late rebleeding include:

- Severe liver failure
- Ascites
- Hepatoma
- Active alcoholism
- Red color signs on the varices

Treatment with endoscopic variceal ligation decreases the risk of rebleeding to approximately 30 percent, and the risk of death to approximately 25 percent. Endoscopic sclerotherapy is associated with a decrease in the risk of rebleeding to 40 to 50 percent, and a decrease in the risk of death to 30 to 60 percent. (See "[Prevention of recurrent bleeding from esophageal varices in patients with cirrhosis](#)" and "[Endoscopic variceal ligation](#)".)

GENERAL APPROACH TO MANAGEMENT

The initial management of patients with acute variceal hemorrhage includes hemodynamic resuscitation (while avoiding over transfusion) and ensuring the patient's airway is protected. Patients with cirrhosis should also receive prophylactic antibiotics, such as a fluoroquinolone or

third-generation cephalosporin. These issues are discussed in detail elsewhere. (See "[Overview of the management of patients with variceal bleeding](#)".)

The first step in stopping variceal bleeding is the initiation of pharmacologic therapy ([algorithm 1](#)). Pharmacologic therapy should be started in all patients with upper gastrointestinal bleeding who have varices or who are at risk for having varices (eg, patients with cirrhosis). Pharmacologic therapy should **not** be delayed pending confirmation that the source of bleeding is indeed from varices. In the United States, pharmacologic therapy typically consists of an [octreotide](#) bolus (50 mcg intravenous [IV]) followed by a continuous infusion (50 mcg IV per hour). Where available, [terlipressin](#) is often used. Terlipressin is administered at an initial dose of 2 mg IV every four hours and can be titrated down to 1 mg IV every four hours once hemorrhage is controlled. Pharmacologic therapy is typically continued for three to five days following cessation of bleeding. (See '[Pharmacologic therapy](#)' below.)

For patients with esophageal varices, acute bleeding is typically managed with endoscopic variceal ligation, though occasionally endoscopic sclerotherapy is used. The goal should be to perform an upper endoscopy after fluid resuscitation and within 12 hours of presentation. If the bleeding cannot be controlled endoscopically, treatment options include transjugular intrahepatic portosystemic shunt (TIPS) placement or surgical shunting. For bleeding gastric varices, treatment is with cyanoacrylate injection where available. However, this agent is not approved for the treatment of bleeding gastric varices and its use reflects off-label use of the agent in the United States. If cyanoacrylate injection is not an option, TIPS placement is typically used. Balloon tamponade is an option for temporarily stopping bleeding from esophageal or gastric varices while definitive treatment is being arranged, but it is associated with serious complications, including esophageal rupture. Bleeding ectopic varices may be managed with TIPS placement or surgery. (See '[Management of esophageal varices](#)' below and '[Management of gastric varices](#)' below and '[Management of ectopic varices](#)' below.)

PHARMACOLOGIC THERAPY

Vasoactive medications decrease portal blood flow and are used for the treatment of acute variceal hemorrhage. They include [vasopressin](#), somatostatin, and their analogs ([terlipressin](#) and [octreotide](#), respectively). As a group, vasoactive medications have been shown to decrease mortality and improve hemostasis in patients with acute variceal bleeding [[12](#)]. However, terlipressin is the only agent individually shown to reduce mortality [[13](#)]. Pharmacologic therapy should be started at the time of presentation in a patient who has known varices or is at risk for varices [[4,14](#)]. It should **not** be held pending confirmation of the diagnosis. Terlipressin is the preferred agent in many countries outside of the United States, whereas octreotide is the agent

available in the United States. Pharmacologic therapy should be continued for three to five days [4,14].

In a meta-analysis of 30 randomized trials with 3111 patients with acute variceal bleeding, compared with placebo, the use of vasoactive medications was associated with improved hemostasis (relative risk [RR] 1.2, 95% CI 1.1-1.3) and decreases in seven-day mortality (RR 0.74, 95% CI 0.57-0.95), transfusion requirement (pooled mean difference -0.70 units, 95% CI -1.0 to -0.38), and duration of hospitalization (pooled mean difference -0.71 days, 95% CI -1.2 to -0.19) [12].

Terlipressin — **Terlipressin** (triglycyl lysine **vasopressin**) is a synthetic analog of vasopressin that is released in a slow and sustained manner, permitting its administration via intermittent injections. Terlipressin is not available in the United States but is used in several other countries. Terlipressin is administered at an initial dose of 2 mg IV every four hours and can be titrated down to 1 mg IV every four hours once hemorrhage is controlled. At least 20 randomized trials have evaluated its efficacy [13]. A meta-analysis found a reduction in all-cause mortality with terlipressin compared with placebo (RR 0.66, 95% CI 0.49-0.88) [13]. Because terlipressin has been associated with hyponatremia, sodium levels should be monitored daily when it is being used [4]. In addition, terlipressin can cause ischemic injury, including myocardial infarction, skin necrosis, and bowel ischemia [15].

Only a few studies directly compared **terlipressin** with somatostatin, **octreotide**, or endoscopic treatment [12]. Those studies suggest that terlipressin has similar efficacy for the control of acute bleeding. Compared with octreotide, terlipressin may have more sustained hemodynamic effects in patients with bleeding varices. A study comparing the acute hemodynamic effects of terlipressin to octreotide in stable patients with cirrhosis found a sustained effect of terlipressin on portal pressure and blood flow compared with only a transient effect from octreotide [16]. A randomized trial comparing variceal ligation in combination with terlipressin or octreotide found that terlipressin was not inferior to octreotide for control of esophageal variceal bleeding or in-hospital survival [17].

Somatostatin and its analogs — Somatostatin inhibits the release of vasodilator hormones such as glucagon [18], indirectly causing splanchnic vasoconstriction and decreased portal inflow. It has a short half-life and disappears within minutes of a bolus infusion. It is given as a 250 mcg bolus followed by a continuous infusion of 250 mcg per hour. It is continued for three to five days. **Octreotide** is a long-acting analog of somatostatin. Somatostatin is not available in the United States, but octreotide is. Octreotide is given as a 50 mcg bolus followed by a continuous infusion of 50 mcg per hour and is also continued for three to five days.

Pharmacodynamics — Following a bolus injection of somatostatin or [octreotide](#), portal venous inflow, portal pressures, azygos flow, and intravariceal pressures decrease within seconds. Of these effects, the most consistently observed is the decrease in collateral flow (azygos flow), whereas the changes in portal pressures, as measured by wedged hepatic pressures, are the most variable.

One of the most detailed studies to evaluate the pharmacodynamics of [octreotide](#) included 68 patients with cirrhosis who were given octreotide by four different intravenous methods [19]:

- A 50 mcg bolus, a 500 mcg bolus, or a placebo bolus
- A 50 mcg bolus followed by a continuous infusion of 50 or 250 mcg per hour or a placebo infusion
- An initial 50 mcg bolus followed by repeated 50 mcg boluses or placebo boluses
- A placebo bolus followed by continuous infusion of [octreotide](#) 50 mcg per hour

[Octreotide](#) caused a marked but transient decrease in portal pressure and azygos blood flow, and an increase in mean arterial pressure. These effects lasted only five minutes, even with the addition of continuous infusions. Repeated bolus injections had shorter, less marked effects, while continuous infusion did not decrease portal pressure, suggesting that there was rapid desensitization.

These findings make it unclear why [octreotide](#) should be of any benefit in portal hypertension since its effect on portal pressure is short-lived. However, the potential benefits of octreotide (and somatostatin) in portal hypertension may extend beyond the effects noted above. Both drugs inhibit the release of glucagon and other hormones that have important roles in mediating the normal increase in mesenteric blood flow that occurs postprandially [20-22]. Variceal hemorrhage is associated with an increase in intestinal blood flow, presumably mediated by pathways that are activated by the presence of blood, a high-protein substance, in the gut [23]. Octreotide can blunt this response for at least 48 hours [24]. In addition, activation of somatostatin receptors may decrease the rebound increase in portal venous pressure that occurs when blood enters the gastrointestinal tract and during correction of hypovolemia.

Efficacy — A number of clinical trials have compared somatostatin or [octreotide](#) with other treatments or placebo for the management of active bleeding. While somatostatin and octreotide help achieve hemostasis and prevent rebleeding, neither has a clearly established benefit on mortality [25,26]. Somatostatin and octreotide are associated with initial hemostasis rates of 63 to 100 percent when used alone or in combination with endoscopic therapy [27-32]. Early rebleeding is seen in 9 to 31 percent of patients [33-36]. Treatment with somatostatin or octreotide infusions when used in addition to sclerotherapy is superior to sclerotherapy alone

or somatostatin alone for the prevention of early rebleeding and possibly survival [34,37,38]. A systematic review found that combination therapy with somatostatin or octreotide and endoscopic variceal ligation improved the five-day success rate compared with endoscopic variceal ligation alone [25,39]. However, no mortality benefit could be demonstrated.

Somatostatin and octreotide have also been compared with terlipressin. A trial with 780 patients randomly assigned patients with variceal bleeding to receive somatostatin, octreotide, or terlipressin [40]. All patients also underwent endoscopy. The somatostatin, octreotide, or terlipressin was given for a total of five days. There were no differences among the groups in the rates of initial hemostasis, rebleeding, or mortality.

Somatostatin and its analogs have few side effects and are preferred over vasopressin and balloon tamponade. A meta-analysis of trials comparing somatostatin with vasopressin found two benefits with somatostatin [41]:

- A higher relative risk (1.62) of achieving initial control of the bleeding. The absolute benefit was such that only three or four patients had to be treated with somatostatin for one to derive benefit over therapy with vasopressin.
- A lower risk of adverse effects (0 versus 10 percent with vasopressin).

Vasopressin — Vasopressin directly constricts mesenteric arterioles and decreases portal venous inflow [42]. Vasopressin was previously used for the treatment of variceal bleeding, but it has been replaced by other medications such as octreotide (in the United States) and terlipressin (outside of the United States). It is rarely used because the benefit of bleeding cessation appears to be counterbalanced by enhanced mortality due to extrasplanchnic vasoconstrictive properties and resultant myocardial, cerebral, bowel, and limb ischemia [43].

BALLOON TAMPONADE

Balloon tamponade is an effective way to achieve short-term hemostasis in patients with bleeding esophagogastric varices, but due to complications and rebleeding upon balloon deflation, its use is reserved for temporary stabilization of patients until more definitive treatment can be instituted [4]. Esophageal stent placement has been used as an alternative to balloon tamponade in patients with uncontrollable acute variceal bleeding [44]. (See 'Esophageal stents' below.)

Three balloons have been used: the Sengstaken-Blakemore tube (which has a 250 cc gastric balloon, an esophageal balloon, and a single gastric suction port), the Minnesota tube (a

modified Sengstaken-Blakemore tube with an esophageal suction port above the esophageal balloon), and the Linton-Nachlas tube (which has a single 600 cc gastric balloon) [45].

Initial control of variceal bleeding with balloon tamponade has been observed in 30 to 90 percent of patients in a number of reports [46-50]. The variability in success rates is probably due to patient selection, the concomitant use of other types of therapy, and experience of the staff in using these tubes. Balloon tamponade appears to be less successful in patients who have failed pharmacologic therapy and in patients with early rebleeding.

Balloon placement — Before balloon tamponade is attempted, the patient should be intubated to prevent aspiration. Since this is a temporizing measure in most cases, arrangements for definitive treatment (endoscopic therapy, transjugular intrahepatic portosystemic shunt [TIPS] placement, or surgery) should be made. (See '[Management of esophageal varices](#)' below.)

Equipment that is required includes:

- A tamponade tube kit (with the tube and clamps)
- A manometer (not needed for Linton tubes)
- Large-volume syringes
- A traction/pulley system to maintain constant tension on the tube
- Adequate suction

Before tube placement, all equipment should be readily at hand. The balloon(s) should be inflated with air and held underwater to assess for leakage and then deflated. With the patient in the supine or left-lateral position, the tube is lubricated and carefully inserted through the mouth (preferred) or nostril until at least 50 cm of the tube has been introduced.

Once the tube is placed, the ports are suctioned to remove all air. The gastric balloon is then inflated with 100 mL of air. A radiograph should then be obtained to confirm placement of the gastric balloon below the diaphragm (accidental inflation of the balloon in the esophagus or a hiatal hernia could lead to rupture). Once confirmed, the balloon is filled with an additional 350 to 400 mL of air (for a total of 450 to 500 mL of air). Once inflated, the air inlet for the gastric balloon should be clamped.

After the gastric balloon is inflated, the tube is pulled until resistance is felt, at which point the balloon is tamponading the gastroesophageal junction. The tube is then securely fastened to either a pulley device or taped to a football helmet to maintain tension on the tube (and thus continued tamponade at the gastroesophageal junction). A one to two pound weight (eg, a 500 mL intravenous fluid bag) can be used to maintain tension on the tube. This is often sufficient to stop the variceal hemorrhage.

If bleeding continues despite inflation of the gastric balloon, the esophageal balloon (if present) should be inflated to 30 to 45 mmHg. While the esophageal balloon is inflated, the pressure should be checked periodically (at least once per hour). It is important not to overinflate the esophageal balloon as it puts the patient at risk for esophageal necrosis or rupture. Once the bleeding is controlled, the pressure in the esophageal balloon should be reduced by 5 mmHg to a goal pressure of 25 mmHg. If bleeding resumes, the pressure is increased by 5 mmHg.

The tube can be left in place for 24 to 48 hours. The gastric balloon (along with the esophageal balloon if used) should be deflated every 12 hours to check for rebleeding. If the bleeding has ceased, the tube can be left in place with the balloons deflated. The balloons can then be reinflated if bleeding resumes. If the bleeding resumes upon deflation of the balloon(s), the balloon(s) should immediately be reinflated. As mentioned above, balloon tamponade is a temporizing measure and definitive treatment should be arranged for ongoing or recurrent bleeding.

Rebleeding and complications — One of the main problems with balloon tamponade is the high risk of rebleeding following deflation of the balloon. In addition, balloon tamponade is associated with significant complications, the most lethal of which is esophageal rupture.

Major complications have been observed in approximately 14 percent of patients, occurring more frequently in series in which tubes were inserted by relatively inexperienced staff [46]. Thus, these tubes should only be used in settings in which experienced providers are available.

The tubes should also be used cautiously in patients with respiratory failure, cardiac arrhythmias, or a hiatal hernia [51]. The airway must be protected in all patients receiving such treatment due to impaired ability to clear oral secretions and high risk for aspiration.

An American Association for the Study of Liver Diseases survey with 234 respondents found that the majority (89 percent) believed that balloon tamponade should be used for stabilization before TIPS placement. The reasons cited for not using balloon tamponade were: complications (23 percent), low benefit (11 percent), high success rate of endoscopy alone (29 percent), or all of the above (37 percent) [52]. Importantly, none of the trainee respondents were comfortable with this technique.

MANAGEMENT OF ESOPHAGEAL VARICES

Bleeding esophageal varices are typically managed with endoscopic therapy ([algorithm 1](#)). Endoscopic variceal ligation is generally preferred due to its high efficacy and low complication rate. Endoscopic sclerotherapy is an alternative that is also highly effective, but it is associated

with higher complication rates than endoscopic variceal ligation ([table 1](#)). If endoscopic therapy fails, treatment options include transjugular intrahepatic portosystemic shunt (TIPS) placement or creation of a surgical shunt.

Initial management — Endoscopic therapy is the definitive treatment of choice for active variceal hemorrhage [53]. The goal should be to perform an upper endoscopy within 12 hours of presentation [4,14]. [Erythromycin](#) can be given prior to the procedure to help clear the stomach of blood [14] (see "[Approach to acute upper gastrointestinal bleeding in adults](#)", [section on 'Prokinetics'](#)). Endoscopic therapy can be performed at the same time as diagnostic endoscopy by virtually all trained gastroenterologists.

Endoscopic variceal ligation and endoscopic sclerotherapy — Two forms of endoscopic treatment are commonly used: endoscopic variceal ligation (EVL) and endoscopic sclerotherapy (ES). EVL is generally preferred as initial treatment [4,14]. If initial attempts at treatment with EVL fail, ES can be tried [54]. Esophageal stents are also now being used by some endoscopists for the treatment of bleeding esophageal varices. (See '[Esophageal stents](#)' below.)

- EVL is similar to hemorrhoidal banding; it involves placing small elastic bands around varices in the distal 5 cm of the esophagus ([picture 1](#)). (See "[Endoscopic variceal ligation](#)".)
- ES involves injection of a sclerosant solution into the varices using an injection needle that is passed through the accessory channel of the endoscope. A number of sclerosant solutions are available that are all effective, including [sodium morrhuate](#) and ethanolamine. The volume and frequency of injections vary widely among endoscopists and the particular situation. (See "[Prevention of recurrent bleeding from esophageal varices in patients with cirrhosis](#)".)

EVL and ES are initially successful in 70 to 100 percent of patients, with many studies reporting success rates around 90 percent. However, it is important to note that these are old trials so their relevance in current practice is unclear. In a 1995 meta-analysis of seven randomized trials with 547 patients, EVL was similar to ES with regard to bleeding cessation (89 versus 88 percent; odds ratio [OR] 1.14, 95% CI 0.44-2.90) [55]. However, EVL was superior to ES for the outcomes of rebleeding (31 versus 47 percent; OR 0.52, 95% CI 0.37-0.74), death (24 versus 32 percent; OR 0.67, 95% CI 0.46-0.98), and stricture formation (0 versus 11 percent; OR 0.10, 95% CI 0.03-0.29).

Regardless of the initial treatment, we use band ligation for subsequent elective endoscopic treatment sessions. (See "[Prevention of recurrent bleeding from esophageal varices in patients with cirrhosis](#)".)

Both esophageal varices and gastroesophageal varices types I (along the lesser curvature of the stomach) and II (along the greater curvature of the stomach) can be treated by sclerotherapy or band ligation. However, isolated gastric varices do not usually lend themselves to these modalities of treatment ([picture 2](#)). (See "[Pathogenesis of variceal bleeding in patients with cirrhosis](#)", section on '[Location of varices](#)' and '[Management of gastric varices](#)' below.)

Combined data from a number of studies suggest that complications with EVL are substantially less than with ES, presumably because of the shallower tissue injury (cumulative complication rate of 11 versus 25 percent) [55-64]. Complications of EVL include complications from overtube placement, esophageal ulceration, and possible worsening or development of portal hypertensive gastropathy or gastric varices. (See "[Endoscopic variceal ligation](#)", section on '[Complications](#)'.)

Complications of ES may be categorized as local, regional, or systemic ([table 1](#)) [65,66]:

- Local complications: Ulceration, bleeding, dysmotility, stricture formation, and portal hypertensive gastropathy
- Regional complications: Esophageal perforation and mediastinitis ([image 1](#))
- Systemic complications: Sepsis and aspiration with ventilation perfusion mismatch and hypoxemia

All of these complications occur more frequently when endoscopy is performed emergently rather than electively. In a meta-analysis, a much lower rate of esophageal stricture formation was the major advantage of band ligation in terms of complications [55]. However, there are no reliable data comparing sclerotherapy and band ligation for adverse events specifically in the setting of acute variceal hemorrhage. Both variceal ligation and sclerotherapy have the potential to worsen portal hypertensive gastropathy following obliteration of varices ([picture 3](#)), which could result in bleeding. (See "[Endoscopic variceal ligation](#)", section on '[Portal hypertensive gastropathy](#)'.)

Esophageal stents — Self-expanding metal stents (SEMS) have been used for the treatment of acute refractory esophageal variceal bleeding [14,44,67-69]. A specially designed covered SEMS for treatment of esophageal varices is introduced over a guidewire during endoscopy and does not require fluoroscopy.

Several small studies have reported promising initial results and meta-analyses have also been published [44,67-70]. In the most recent meta-analysis, technical success was achieved in 97 percent of cases and bleeding was controlled in 96 percent. However, all studies have reported adverse events related to the SEMS placement, such as ulceration and stent migration. Until more data are available, the use of SEMS for treating refractory variceal bleeding should be

restricted to clinical trials or centers with substantial expertise in the use of these stents for refractory variceal hemorrhage.

Management if endoscopic therapy fails — Emergent endoscopic treatment fails to control bleeding or bleeding recurs in 10 to 20 percent of patients [1]. Patients in whom hemostasis cannot be achieved are at high risk for exsanguination and other complications related to active bleeding. If one endoscopic modality fails to control bleeding, it is reasonable to try using a different treatment modality (eg, band ligation for failed sclerotherapy) [54,71] ([algorithm 1](#)). In patients with rebleeding following initially successful endoscopic therapy, a second attempt at endoscopic therapy may be carried out, though data are lacking regarding the best approach for patients with early rebleeding [4]. For those with severe bleeding, proceeding with stabilization and TIPS may be more appropriate.

If bleeding is not quickly and effectively stopped endoscopically, or if rebleeding occurs a second time, more definitive therapy (TIPS placement or surgery) is required [14]. Balloon tamponade or esophageal stent placement can be performed as a temporizing measure. TIPS is generally preferred as definitive therapy because it is associated with a high success rate (90 to 100 percent of patients will achieve hemostasis). While surgery is also highly effective, it is associated with a high mortality rate (up to 50 percent). (See '[Balloon tamponade](#)' above and "[Overview of transjugular intrahepatic portosystemic shunts \(TIPS\)](#)", section on '[Indications](#)' and '[Surgery](#)' below.)

Most patients who fail endoscopic treatment have already received a trial of pharmacologic treatment by the time a diagnosis of failed endoscopic treatment is established. There are no data to support the use of higher doses of [octreotide](#) or somatostatin in those who have failed endoscopic treatment.

Transjugular intrahepatic portosystemic shunt — TIPS placement starts with passing a needle catheter (Colapinto catheter) via the transjugular route into the hepatic vein and wedging it there ([figure 1](#)). The needle is then extruded and advanced through the liver parenchyma to the intrahepatic portion of the portal vein and a stent is deployed. A TIPS functions like side-to-side surgical portacaval shunt but does not require general anesthesia or major surgery for placement. Ninety to 100 percent of patients will achieve hemostasis following TIPS placement [1].

The early use of TIPS has been associated with lower all-cause mortality, lower rates of failure to control bleeding, and less rebleeding [72]. In addition, early TIPS placement following endoscopic therapy may improve transplantation-free survival in patients with advanced cirrhosis [73]. (See "[Overview of transjugular intrahepatic portosystemic shunts \(TIPS\)](#)".)

Absolute contraindications to TIPS placement include heart failure, polycystic liver disease, severe pulmonary hypertension, uncontrolled systemic infection or sepsis, and severe tricuspid regurgitation. Relative contraindications include hepatocellular carcinoma (particularly if central), portal vein thrombosis, and severe coagulopathy or thrombocytopenia. Complications of TIPS placement include portosystemic encephalopathy, technical complications (eg, cardiac arrhythmias, traversal of the liver capsule), and TIPS stenosis. (See ["Transjugular intrahepatic portosystemic shunts: Postprocedure care and complications"](#) and ["Overview of transjugular intrahepatic portosystemic shunts \(TIPS\)"](#).)

In early studies, hemostasis was achieved following TIPS placement in the vast majority of patients with refractory variceal hemorrhage despite endoscopic treatment [74,75]. However, interpretation of the efficacy of TIPS in the setting of active hemorrhage was confounded by the heterogeneous patient populations and failure to define the nature of active bleeding.

A later study described patients who were actively bleeding despite emergent endoscopic treatment within the previous 72 hours and considered to be at high risk of dying from emergent surgery (defined by the presence of sepsis or deep coma, pneumonia, multiorgan failure, renal failure, severe comorbid conditions) [76]. The patients were stabilized by balloon tamponade and had TIPS performed within 12 hours. Hemostasis was achieved in 90 percent of patients, and the 30-day survival rate was 63 percent. In those without pulmonary compromise, the 30-day survival rate was above 90 percent. These data are far superior to those obtained with surgery for patients in whom a 10 to 20 percent survival would have been expected. Other studies have confirmed these results. (See ["Overview of transjugular intrahepatic portosystemic shunts \(TIPS\)"](#).)

The role of TIPS in prevention of rebleeding is discussed separately. (See ["Prevention of recurrent bleeding from esophageal varices in patients with cirrhosis"](#).)

Surgery — Emergency shunt surgery is extremely effective in arresting hemorrhage and preventing rebleeding, but it is associated with up to a 50 percent mortality rate [77-79]. It is not commonly used because TIPS has lower complication rates.

Many patients die of liver failure and complications of surgery, despite achievement of hemostasis. Those with severe hemorrhage, tense ascites, deep coma, aspiration pneumonia, renal failure, or sepsis are at particular risk from surgery [79]. The ideal patient for surgical therapy is one with well-preserved liver function who fails emergent endoscopic treatment and has no complications from the bleeding or endoscopy. Surgery may also be required for patients who are not good surgical candidates but have a contraindication to TIPS placement, such as heart failure.

The choice of surgery depends on the training and expertise of the surgeon. There are two basic types of operations: shunt operations and nonshunt operations. Shunt operations can be categorized as follows:

- Nonselective – Those that decompress the entire portal tree and divert all flow away from the portal system, such as portacaval shunts.
- Selective – Those that compartmentalize the portal tree into a decompressed variceal system while maintaining sinusoidal perfusion via a hypertensive superior mesenteric-portal compartment, such as a distal splenorenal shunt.
- Partial – Those that incompletely decompress the entire portal tree and thereby also maintain some hepatic perfusion.

Nonshunt operations generally include either esophageal transection (in which the distal esophagus is transected and then stapled back together after varices have been ligated) or devascularization of the gastroesophageal junction (Sugiura procedure).

Experimental approaches

Hemostatic nanopowder — In addition to the standard endoscopic therapies, there is interest in novel endoscopic therapies such as the endoscopic application of hemostatic powder. The powder becomes cohesive and adhesive when it comes into contact with moisture, forming a stable mechanical barrier at the site of bleeding. The powder is delivered through a catheter and sprayed onto the bleeding site under endoscopic guidance without the need for direct tissue contact. Hemostatic nanopowder may have a role as bridging therapy if definitive therapy cannot be performed initially (eg, because resources are not available or the endoscopist does not have expertise in endoscopic treatment of variceal hemorrhage) [14]. However, definitive therapy will still be required. In addition, hemostatic nanopowder is not approved by the US Food and Drug Administration for this indication.

Several studies have supported a role for hemostatic powder in the treatment of acute variceal bleeding [80-84]. In a randomized trial with 86 patients with cirrhosis and acute variceal bleeding, 43 patients were randomized to receive endoscopy with hemostatic powder application within two hours of admission, followed by early elective endoscopy the next day (within 12 to 24 hours of admission) for definitive treatment. The remaining 43 patients received early elective endoscopy the day after admission. Both groups received **octreotide** starting at admission. Among the patients treated with hemostatic powder, four did not achieve hemostasis with the hemostatic spray and required rescue therapy for spurting bleeding. An additional patient initially achieved hemostasis but developed recurrent hematemesis two

hours later and died before rescue endoscopy. The remainder of the patients treated with hemostatic powder achieved clinical hemostasis and had follow-up elective endoscopy after a median of 18 hours that confirmed endoscopic hemostasis. In the group assigned to undergo early elective endoscopy only, 13 patients required emergency endoscopy for hemostasis prior to elective endoscopy for recurrent bleeding. The remaining 30 patients underwent early elective endoscopy after a median of 16 hours and all had active bleeding at the time of endoscopy. All of the patients in both groups received definitive therapy at the elective endoscopy (endoscopic variceal ligation, glue injection, or combination therapy). Overall, patients treated with hemostatic powder were less likely to require rescue endoscopy (12 versus 30 percent) and had lower mortality at six weeks (7 versus 30 percent).

MANAGEMENT OF GASTRIC VARICES

General approach — Bleeding intragastric varices should be treated with [octreotide](#) (or somatostatin or [terlipressin](#)) and balloon tamponade followed by cyanoacrylate injection [85-90], transjugular intrahepatic portosystemic shunt (TIPS) placement, or surgery. Bleeding gastric varices can be technically difficult to treat and cyanoacrylate injection is the preferred initial approach, where available, for most gastric varices (it is not widely available in the United States) [2,4,14]. Endoscopic variceal ligation is also an option for patients with gastroesophageal varices along the lesser curvature of the stomach (GOV1) [4,14].

Successful hemostasis and obliteration of gastric varices has also been reported with variceal band ligation and with intravariceal injections of sclerosant, absolute alcohol, and fibrin glue [87,88,91-96]. There has also been a case report of treating bleeding gastric varices with hemostatic nanopowder [81]. However, gastric varices frequently rebleed despite initially successful endoscopic therapy and as a result, with the exception of treatment with cyanoacrylate, the use of endoscopic treatment should be limited to clinical trials [97]. (See "[Overview of the treatment of bleeding peptic ulcers](#)", section on 'Hemostatic sprays'.)

Emergency TIPS appears to be as effective for short-term control of bleeding gastric varices as surgery [98]. However, TIPS may be less effective than surgery in patients with bleeding gastric varices who have spontaneous splenorenal collaterals. Early TIPS is an alternative to endoscopic therapy in patients at high risk of failing endoscopic therapy (eg, patients with Child-Pugh class C) [4]. (See '[Surgery](#)' above and "[Overview of transjugular intrahepatic portosystemic shunts \(TIPS\)](#)", section on '[Gastric varices](#)'.)

Cyanoacrylate injection — Cyanoacrylate can be used in centers where it is available. When introduced into an aqueous medium (such as the lumen of a varix), cyanoacrylate glue

immediately polymerizes into a firm clot. In the United States, it is used in only a few centers under research protocols and its use is not approved by the US Food and Drug Administration. However, it can provide a valuable alternative for patients with life-threatening bleeding in whom TIPS is not an option.

In two small randomized trials, cyanoacrylate injection appeared to be more effective and safer than band ligation [88] or alcohol injection [45]. In a retrospective study that included 19 patients who underwent endoscopic ultrasound-guided cyanoacrylate injection, obliteration of the gastric varices was achieved in 18 patients (95 percent) [89].

In addition, cyanoacrylate may prevent rebleeding. In one trial, 77 patients who had bled from gastric varices were assigned to receive either cyanoacrylate or a beta-blocker for secondary prophylaxis [90]. After a median follow-up of 26 months, cyanoacrylate was associated with a lower rebleeding rate compared with beta-blockers (15 versus 55 percent) and was also associated with a lower mortality rate (3 versus 25 percent).

The addition of beta-blockers to treatment with cyanoacrylate does not appear to decrease the risk of rebleeding. In a randomized trial, 95 patients with bleeding gastric varices that were successfully treated with cyanoacrylate were assigned to receive treatment with beta-blockers plus repeated cyanoacrylate injections (every three to four weeks until the varices were obliterated) or repeated cyanoacrylate injections alone [99]. Rebleeding rates were similar between those who received combination therapy and those treated with cyanoacrylate alone at six months (89 versus 94 percent), one year (77 versus 77 percent), two years (62 versus 58 percent), and three years (52 versus 47 percent). Mortality rates were also similar between the groups.

Complications from cyanoacrylate injection were reported in a series of 753 patients [100]. The complications included rebleeding due to extrusion of the glue cast (4.4 percent), sepsis (1.3 percent), distant emboli (pulmonary, cerebral, splenic; 0.7 percent), gastric ulcer formation (0.1 percent), major gastric variceal bleeding (0.1 percent), and mesenteric hematoma associated with hemoperitoneum and bacterial peritonitis (0.1 percent). The complication-related mortality rate was 0.5 percent.

To decrease the risk of glue embolization, some have proposed combining cyanoacrylate injection with endoscopic-ultrasound guided coil placement. The idea is to place the coil immediately prior to injection of the cyanoacrylate so that the coil (which has synthetic fibers attached to it) may act as a scaffold for the glue, preventing embolization. This was shown to be feasible in a study of 30 patients [101]. Whether it decreases this risk of embolization requires further study.

Alternative treatments — Alternative treatments that have been studied include variceal band ligation, early TIPS, thrombin injection, use of bands and snares, balloon occluded retrograde transvenous obliteration (BRTO), vascular plug and gelatin sponge-assisted retrograde transvenous obliteration, and coil embolization with gelatin sponge injection.

- **Variceal band ligation** – Variceal band ligation was examined in a study of 27 patients with gastric varices [102]. Band ligation was successful in stopping acute bleeding in 16 of 18 patients (88 percent) [102]. However, 6 of the 27 patients (22 percent) died, and bleeding recurred in 5 patients (19 percent). Many of these patients had noncirrhotic portal hypertension due to schistosomiasis. Thus, it is uncertain if similar results would be obtained in patients with cirrhosis. (See "[Endoscopic variceal ligation](#)".)
- **Early TIPS** – Early TIPS is an alternative to endoscopic therapy in patients at high risk of failing endoscopic therapy (eg, patients with Child-Pugh class C) [4]. TIPS is also an option for patients who fail endoscopic therapy.
- **Thrombin injection** – A promising approach is the intravariceal injection of thrombin [103-106]. It appears to be similarly effective when compared with cyanoacrylate injection, possibly with fewer side effects. One of the largest series looking at the use of thrombin included 52 patients with bleeding gastric varices treated with intravariceal injections of bovine thrombin (average of 1070 int. unit administered during two treatment sessions) [104]. Initial hemostasis was achieved in 94 percent. Bleeding related mortality within 72 hours of the index bleed was 6 percent. After six weeks of follow-up, 9 of 49 surviving patients (18 percent) rebled, and an additional patient died.

A subsequent randomized trial compared thrombin injection with cyanoacrylate injection in 68 patients [106]. The rates of hemostasis at 48 hours were similar (94 percent for thrombin injection and 97 percent for cyanoacrylate injection, $p = 0.60$). Gastric ulcers were more common in patients treated with cyanoacrylate injection (37 percent) compared with patients treated with thrombin injection (0 percent). Complications overall, including gastric ulcer formation, ulcer bleeding, fever, bacteremia/sepsis, abdominal pain, urinary tract infection, and spontaneous bacterial peritonitis (SBP) were more common in those treated with cyanoacrylate injection (51 percent) compared with those treated with thrombin injection (12 percent). In addition, the complications seen in the thrombin injection group included fever (two patients), abdominal pain (one patient), and UTI (one patient) but did not include ulcer formation/bleeding, bacteremia/sepsis, or SBP.

- **Elastic bands and detachable snares** – Another report described the successful use of elastic bands and detachable snares in controlling acute rebleeding and achieving gastric

variceal eradication [107].

- **BRT0** – BRT0 is a procedure that has been used for bleeding gastric varices as well as ectopic varices (eg, small bowel varices). BRT0 is an interventional radiologic technique that involves occluding blood flow by inflation of a balloon catheter within a draining vessel, followed by instillation of a sclerosant proximal to the site of balloon occlusion. BRT0 requires the presence of a spontaneous shunt into which a balloon catheter is retrogradely introduced. In the case of gastric varices, there frequently is a spontaneous gastrosplenic shunt. Small bowel varices usually drain into dilated collateral vessels that connect to the portal vein through systemic shunts.

Observational studies suggest that patients with gastric varices treated with BRT0 have good long-term bleeding control (90 percent in one study [108]), but technical failure occurs in approximately 10 percent of cases [108,109]. BRT0 may increase portal pressure and lead to the development or worsening of esophageal varices and ascites. In addition, systemic vein thrombosis has been described [110]. Combining BRT0 with TIPS may improve outcomes. In a series of 39 patients, BRT0 combined with TIPS was compared with BRT0 alone. The combination of BRT0 and TIPS was associated with higher rates of being ascites/hydrothorax free (100 versus 29 percent at two years) and lower rates of recurrent hemorrhage (0 versus 21 percent at two years) though no difference in survival was seen [111]. While commonly performed in Japan, this procedure is not widely practiced in the United States.

- **Vascular plug and gelatin sponge-assisted retrograde transvenous obliteration** – Another interventional radiologic technique that has been described to treat gastric varices is vascular plug and gelatin sponge-assisted retrograde transvenous obliteration. A vascular plug is placed in the left adrenal vein or gastrosplenic shunt. Embolization of the gastrosplenic shunt and gastric varices with a gelatin sponge is then performed. In a series of 20 patients, vascular plug placement and gelatin sponge embolization were technically successful in all patients, with no procedure-related complications [112]. One week later, computed tomographic (CT) scanning revealed that all patients had complete thrombosis of the gastrosplenic shunts and gastric varices. In addition, clinical symptoms of hepatic encephalopathy resolved in the seven patients who had hepatic encephalopathy prior to the procedure. There were no cases of variceal bleeding during follow-up (mean 422 days); however, worsening of esophageal varices was noted in 4 of 18 patients (22 percent) who underwent follow-up endoscopy at a mean follow-up of 9.4 months.
- **Coil embolization with gelatin sponge injection** – Coil embolization with gelatin sponge injection has been described as an alternative to cyanoacrylate injection. It is performed

using endoscopic ultrasound [113]. The varix is punctured and embolization coils are deployed. This is followed by the injection of [absorbable gelatin sponge](#) to occlude the varix.

MANAGEMENT OF ECTOPIC VARICES

Varices occasionally develop at sites other than the stomach and esophagus and come to clinical attention when they bleed. Examples are duodenal, rectal, and peristomal varices (which develop around the stoma in patients who have a colostomy). The optimal treatment for patients with bleeding ectopic varices is unclear. Endoscopic treatment is often unsuccessful and traditional treatment has been surgery. Our approach is to start with transjugular intrahepatic portosystemic shunt (TIPS) placement, reserving surgery for patients who fail TIPS or who have a contraindication to TIPS placement. (See '[Transjugular intrahepatic portosystemic shunt](#)' above.)

Case reports have noted that the bleeding can also be controlled by TIPS [114-117]. It is unlikely that enough cases will be seen by any center to generate "hard data" upon which to base therapy.

A 2017 guideline from the American Association for the Study of Liver Diseases suggests a multidisciplinary approach to treatment, with treatment options including endoscopic variceal ligation, cyanoacrylate injection (where available), endosonographic coil placement, TIPS with or without embolization, and balloon occluded retrograde transvenous obliteration (BRTO) [2]. However, the quality of evidence supporting this recommendation is low.

As with gastric varices, small bowel varices can be treated with BRTO. In a study of seven patients, six were candidates for the procedure and were treated by balloon inflation followed by an injection of 5 percent [ethanolamine oleate iopamidol](#) into the draining vessels [118]. Bleeding was controlled in all of the patients, with one patient experiencing rebleeding after 30 months. (See '[Alternative treatments](#)' above.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "[Society guideline links: Gastrointestinal bleeding in adults](#)".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Beyond the Basics topics (see "[Patient education: Esophageal varices \(Beyond the Basics\)](#)")

SUMMARY AND RECOMMENDATIONS

• Initial management

- The initial management of patients with acute variceal hemorrhage includes hemodynamic resuscitation (while avoiding over transfusion) and ensuring the patient's airway is protected. Patients with cirrhosis should also receive prophylactic antibiotics such as a fluoroquinolone or third-generation cephalosporin ([algorithm 1](#)). These issues are discussed in detail elsewhere. (See "[Overview of the management of patients with variceal bleeding](#)".)
- Balloon tamponade can be used to control bleeding while arrangements are being made for definitive treatment in patients with severe bleeding or in whom definitive treatment may be delayed. (See '[Balloon tamponade](#)' above.)

• Pharmacologic therapy

- In patients with upper gastrointestinal bleeding who have varices or are at risk for having varices, we recommend pharmacologic treatment with a vasoactive medication in addition to endoscopic treatment, rather than endoscopic treatment alone ([algorithm 1](#)) (**Grade 1B**). Pharmacologic therapy should be started at the time of presentation and should **not** be held pending confirmation of the diagnosis. Vasoactive

medications (eg, [terlipressin](#), somatostatin, [octreotide](#)) decrease portal blood flow and, as a group, have been shown to decrease mortality and improve hemostasis in patients with acute variceal bleeding. However, terlipressin is the only agent individually shown to reduce mortality. Terlipressin may also have more sustained hemodynamic effects than octreotide. Pharmacologic therapy should be continued for three to five days. (See '[Pharmacologic therapy](#)' above.)

- In countries where it is available (it is not available in the United States), we suggest pharmacologic therapy with [terlipressin](#) rather than somatostatin or [octreotide](#) (**Grade 2C**). It is initially given as 2 mg intravenous (IV) bolus every four hours. Once the bleeding is controlled, it can be titrated down to a dose of 1 mg IV every four hours. Octreotide is available in the United States and is given as a 50 mcg IV bolus, followed by a continuous infusion at a rate of 50 mcg per hour. Octreotide is probably only effective when used in conjunction with endoscopic therapy. (See '[Terlipressin](#)' above and '[Somatostatin and its analogs](#)' above.)

• Endoscopy and TIPS

- Upper endoscopy should be performed for diagnosis and possible treatment in patients with suspected esophageal or gastric variceal bleeding within 12 hours of presentation. (See "[Overview of the management of patients with variceal bleeding](#)" and '[Initial management](#)' above.)
- The approach to definitive treatment will depend on the type(s) of varices present:
 - **Esophageal varices:** We recommend that bleeding esophageal varices initially be treated with esophageal band ligation rather than sclerotherapy (**Grade 1B**). While both methods are successful in approximately 80 to 90 percent of patients, endoscopic variceal ligation is preferred over endoscopic sclerotherapy primarily because it is associated with fewer complications. (See '[Initial management](#)' above.)
 - **Gastric varices:** For patients with bleeding gastric varices, we suggest endoscopic treatment with injection of the tissue adhesive cyanoacrylate (if available) rather than TIPS placement (**Grade 2B**). However, using cyanoacrylate for treating varices is not a labeled indication for the drug in the United States. Small trials suggest cyanoacrylate injection is effective and it is less invasive than TIPS placement. TIPS placement is an alternative in areas where cyanoacrylate is not available (it is not widely available in the United States). (See '[Management of gastric varices](#)' above.)

- **Ectopic varices:** For patients with bleeding ectopic varices, we suggest initial treatment with TIPS placement rather than endoscopic therapy or surgery, provided there are no contraindications to TIPS placement (**Grade 2C**). The optimal treatment for patients with bleeding ectopic varices is unclear. Endoscopic treatment is often unsuccessful, and traditional treatment has been surgical. However, case reports have noted that the bleeding can also be controlled by TIPS placement and TIPS placement is less invasive than surgery. (See '[Management of ectopic varices](#)' above.)

• Rebleeding

- For most patients who have rebleeding following initial control of bleeding with endoscopic therapy, we suggest a second session of endoscopic treatment rather than proceeding to transjugular intrahepatic portosystemic shunt (TIPS) or surgery ([algorithm 1](#)) (**Grade 2C**). However, for those with severe bleeding, proceeding to stabilization and TIPS may be more appropriate. If bleeding recurs after a second attempt at endoscopic therapy or if the bleeding cannot be stopped with endoscopic therapy, treatment options include TIPS and surgery. (See '[Management if endoscopic therapy fails](#)' above.)
- In patients who require salvage therapy, we suggest TIPS placement rather than surgery, provided there are no contraindications to TIPS placement and the patient does not have a history of portosystemic encephalopathy (**Grade 2B**). TIPS is effective in controlling bleeding in 90 to 100 percent of patients. While surgery is also effective, it is associated with a high mortality rate (up to 50 percent). Absolute contraindications to TIPS placement include heart failure, severe pulmonary hypertension, uncontrolled systemic infection or sepsis, and severe tricuspid regurgitation. Relative contraindications include hepatocellular carcinoma (particularly if central), portal vein thrombosis, and severe coagulopathy or thrombocytopenia. (See '[Transjugular intrahepatic portosystemic shunt](#)' above.)
- Surgical options for patients who cannot undergo TIPS placement or in whom it is not successful include shunt operations (eg, distal splenorenal shunt) and nonshunt operations (eg, esophageal transection). (See '[Surgery](#)' above.)

Use of UpToDate is subject to the [Terms of Use](#).

REFERENCES

1. Habib A, Sanyal AJ. Acute variceal hemorrhage. *Gastrointest Endosc Clin N Am* 2007; 17:223.
2. Garcia-Tsao G, Abraldes JG, Berzigotti A, Bosch J. Portal hypertensive bleeding in cirrhosis: Risk stratification, diagnosis, and management: 2016 practice guidance by the American Association for the study of liver diseases. *Hepatology* 2017; 65:310.
3. Hwang JH, Shergill AK, Acosta RD, et al. The role of endoscopy in the management of variceal hemorrhage. *Gastrointest Endosc* 2014; 80:221.
4. de Franchis R, Baveno VI Faculty. Expanding consensus in portal hypertension: Report of the Baveno VI Consensus Workshop: Stratifying risk and individualizing care for portal hypertension. *J Hepatol* 2015; 63:743.
5. Tripathi D, Stanley AJ, Hayes PC, et al. U.K. guidelines on the management of variceal haemorrhage in cirrhotic patients. *Gut* 2015; 64:1680.
6. Garcia-Tsao G, Sanyal AJ, Grace ND, et al. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. *Hepatology* 2007; 46:922.
7. de Franchis R, Baveno V Faculty. Revising consensus in portal hypertension: report of the Baveno V consensus workshop on methodology of diagnosis and therapy in portal hypertension. *J Hepatol* 2010; 53:762.
8. Prandi D, Rueff B, Roche-Sicot J, et al. Life-threatening hemorrhage of the digestive tract in cirrhotic patients. An assessment of the postoperative mortality after emergency portacaval shunt. *Am J Surg* 1976; 131:204.
9. Graham DY, Smith JL. The course of patients after variceal hemorrhage. *Gastroenterology* 1981; 80:800.
10. Burroughs AK, Jenkins WJ, Sherlock S, et al. Controlled trial of propranolol for the prevention of recurrent variceal hemorrhage in patients with cirrhosis. *N Engl J Med* 1983; 309:1539.
11. Burroughs AK, McCormick PA. Prevention of variceal rebleeding. *Gastroenterol Clin North Am* 1992; 21:119.
12. Wells M, Chande N, Adams P, et al. Meta-analysis: vasoactive medications for the management of acute variceal bleeds. *Aliment Pharmacol Ther* 2012; 35:1267.
13. Ioannou G, Doust J, Rockey DC. Terlipressin for acute esophageal variceal hemorrhage. *Cochrane Database Syst Rev* 2003; :CD002147.
14. Gralnek IM, Camus Duboc M, Garcia-Pagan JC, et al. Endoscopic diagnosis and management of esophagogastric variceal hemorrhage: European Society of Gastrointestinal Endoscopy (ESGE) Guideline. *Endoscopy* 2022; 54:1094.

15. Kim HR, Lee YS, Yim HJ, et al. Severe ischemic bowel necrosis caused by terlipressin during treatment of hepatorenal syndrome. *Clin Mol Hepatol* 2013; 19:417.
16. Baik SK, Jeong PH, Ji SW, et al. Acute hemodynamic effects of octreotide and terlipressin in patients with cirrhosis: a randomized comparison. *Am J Gastroenterol* 2005; 100:631.
17. Abid S, Jafri W, Hamid S, et al. Terlipressin vs. octreotide in bleeding esophageal varices as an adjuvant therapy with endoscopic band ligation: a randomized double-blind placebo-controlled trial. *Am J Gastroenterol* 2009; 104:617.
18. Bloom SR, Polak JM. Somatostatin. *Br Med J (Clin Res Ed)* 1987; 295:288.
19. Escorsell A, Bandi JC, Andreu V, et al. Desensitization to the effects of intravenous octreotide in cirrhotic patients with portal hypertension. *Gastroenterology* 2001; 120:161.
20. Albillos A, Colombato LA, Lee FY, Groszmann RJ. Octreotide ameliorates vasodilatation and Na⁺ retention in portal hypertensive rats. *Gastroenterology* 1993; 104:575.
21. McCormick PA, Biagini MR, Dick R, et al. Octreotide inhibits the meal-induced increases in the portal venous pressure of cirrhotic patients with portal hypertension: a double-blind, placebo-controlled study. *Hepatology* 1992; 16:1180.
22. Spahr L, Giostra E, Frossard JL, et al. A 3-month course of long-acting repeatable octreotide (sandostatin LAR) improves portal hypertension in patients with cirrhosis: a randomized controlled study. *Am J Gastroenterol* 2007; 102:1397.
23. Chen L, Groszmann RJ. Blood in the gastric lumen increases splanchnic blood flow and portal pressure in portal-hypertensive rats. *Gastroenterology* 1996; 111:1103.
24. Ludwig D, Schädel S, Brüning A, et al. 48-hour hemodynamic effects of octreotide on postprandial splanchnic hyperemia in patients with liver cirrhosis and portal hypertension: double-blind, placebo-controlled study. *Dig Dis Sci* 2000; 45:1019.
25. Bañares R, Albillos A, Rincón D, et al. Endoscopic treatment versus endoscopic plus pharmacologic treatment for acute variceal bleeding: a meta-analysis. *Hepatology* 2002; 35:609.
26. Gotzsche PC. Somatostatin or octreotide for acute bleeding oesophageal varices. *Cochrane Database Syst Rev* 2000; :CD000193.
27. Villanueva C, Ortiz J, Sàbat M, et al. Somatostatin alone or combined with emergency sclerotherapy in the treatment of acute esophageal variceal bleeding: a prospective randomized trial. *Hepatology* 1999; 30:384.
28. Kravetz D, Bosch J, Terés J, et al. Comparison of intravenous somatostatin and vasopressin infusions in treatment of acute variceal hemorrhage. *Hepatology* 1984; 4:442.

29. Jenkins SA, Baxter JN, Corbett W, et al. A prospective randomised controlled clinical trial comparing somatostatin and vasopressin in controlling acute variceal haemorrhage. *Br Med J (Clin Res Ed)* 1985; 290:275.
30. Saari A, Klvilaakso E, Inberg M, et al. Comparison of somatostatin and vasopressin in bleeding esophageal varices. *Am J Gastroenterol* 1990; 85:804.
31. Huang CC, Sheen IS, Chu CM, et al. A prospective randomized controlled trial of sandostatin and vasopressin in the management of acute bleeding esophageal varices. *Changgeng Yi Xue Za Zhi* 1992; 15:78.
32. Hwang SJ, Lin HC, Chang CF, et al. A randomized controlled trial comparing octreotide and vasopressin in the control of acute esophageal variceal bleeding. *J Hepatol* 1992; 16:320.
33. Planas R, Quer JC, Boix J, et al. A prospective randomized trial comparing somatostatin and sclerotherapy in the treatment of acute variceal bleeding. *Hepatology* 1994; 20:370.
34. D'Amico G, Politi F, Morabito A, et al. Octreotide compared with placebo in a treatment strategy for early rebleeding in cirrhosis. A double blind, randomized pragmatic trial. *Hepatology* 1998; 28:1206.
35. Primignani M, Andreoni B, Carpinelli L, et al. Sclerotherapy plus octreotide versus sclerotherapy alone in the prevention of early rebleeding from esophageal varices: a randomized, double-blind, placebo-controlled, multicenter trial. *New Italian Endoscopic Club. Hepatology* 1995; 21:1322.
36. Sung JJ, Chung SC, Yung MY, et al. Prospective randomised study of effect of octreotide on rebleeding from oesophageal varices after endoscopic ligation. *Lancet* 1995; 346:1666.
37. Besson I, Ingrand P, Person B, et al. Sclerotherapy with or without octreotide for acute variceal bleeding. *N Engl J Med* 1995; 333:555.
38. Avgerinos A, Nevens F, Raptis S, Fevery J. Early administration of somatostatin and efficacy of sclerotherapy in acute oesophageal variceal bleeds: the European Acute Bleeding Oesophageal Variceal Episodes (ABOVE) randomised trial. *Lancet* 1997; 350:1495.
39. D'amico G, Criscuoli V, Fili D, et al. Meta-analysis of trials for variceal bleeding. *Hepatology* 2002; 36:1023.
40. Seo YS, Park SY, Kim MY, et al. Lack of difference among terlipressin, somatostatin, and octreotide in the control of acute gastroesophageal variceal hemorrhage. *Hepatology* 2014; 60:954.
41. Imperiale TF, Teran JC, McCullough AJ. A meta-analysis of somatostatin versus vasopressin in the management of acute esophageal variceal hemorrhage. *Gastroenterology* 1995; 109:1289.

42. Blei AT, Groszmann RJ. Vasopressin and vasoconstrictors. In: *The Physiology of the Intestina I Microcirculation*, Shepherd AP, Granger DN (Eds), Raven Press, New York 1984. p.377.
43. Roberts LR, Kamath PS. Pathophysiology and treatment of variceal hemorrhage. *Mayo Clin Proc* 1996; 71:973.
44. Hubmann R, Bodlaj G, Czompo M, et al. The use of self-expanding metal stents to treat acute esophageal variceal bleeding. *Endoscopy* 2006; 38:896.
45. D'Amico G, Pagliaro L, Bosch J. The treatment of portal hypertension: a meta-analytic review. *Hepatology* 1995; 22:332.
46. Chojkier M, Conn HO. Esophageal tamponade in the treatment of bleeding varices. A decadel progress report. *Dig Dis Sci* 1980; 25:267.
47. Hunt PS, Korman MG, Hansky J, Parkin WG. An 8-year prospective experience with balloon tamponade in emergency control of bleeding esophageal varices. *Dig Dis Sci* 1982; 27:413.
48. Fort E, Sautereau D, Silvain C, et al. A randomized trial of terlipressin plus nitroglycerin vs. balloon tamponade in the control of acute variceal hemorrhage. *Hepatology* 1990; 11:678.
49. Paquet KJ, Feussner H. Endoscopic sclerosis and esophageal balloon tamponade in acute hemorrhage from esophagogastric varices: a prospective controlled randomized trial. *Hepatology* 1985; 5:580.
50. Pitcher JL. Safety and effectiveness of the modified Sengstaken-Blakemore tube: a prospective study. *Gastroenterology* 1971; 61:291.
51. Minocha A, Richards RJ. Sengstaken-Blakemore tube for control of massive bleeding from gastric varices in hiatal hernia. *J Clin Gastroenterol* 1992; 14:36.
52. Bajaj JS, Ananthakrishnan A, Saeian K. Survey of attitudes of AASLD members toward balloon tamponade. *Hepatology* 2005; 41:1435.
53. Grace ND. Diagnosis and treatment of gastrointestinal bleeding secondary to portal hypertension. American College of Gastroenterology Practice Parameters Committee. *Am J Gastroenterol* 1997; 92:1081.
54. Saeed ZA, Michaletz PA, Winchester CB, et al. Endoscopic variceal ligation in patients who have failed endoscopic sclerotherapy. *Gastrointest Endosc* 1990; 36:572.
55. Laine L, Cook D. Endoscopic ligation compared with sclerotherapy for treatment of esophageal variceal bleeding. A meta-analysis. *Ann Intern Med* 1995; 123:280.
56. Stiegmann GV, Goff JS, Michaletz-Onody PA, et al. Endoscopic sclerotherapy as compared with endoscopic ligation for bleeding esophageal varices. *N Engl J Med* 1992; 326:1527.
57. Lo GH, Lai KH, Cheng JS, et al. Emergency banding ligation versus sclerotherapy for the

- control of active bleeding from esophageal varices. *Hepatology* 1997; 25:1101.
58. Laine L, el-Newihi HM, Migikovsky B, et al. Endoscopic ligation compared with sclerotherapy for the treatment of bleeding esophageal varices. *Ann Intern Med* 1993; 119:1.
 59. Lo GH, Lai KH, Cheng JS, et al. A prospective, randomized trial of sclerotherapy versus ligation in the management of bleeding esophageal varices. *Hepatology* 1995; 22:466.
 60. Sarin SK, Govil A, Jain AK, et al. Prospective randomized trial of endoscopic sclerotherapy versus variceal band ligation for esophageal varices: influence on gastropathy, gastric varices and variceal recurrence. *J Hepatol* 1997; 26:826.
 61. Hou MC, Lin HC, Kuo BI, et al. Comparison of endoscopic variceal injection sclerotherapy and ligation for the treatment of esophageal variceal hemorrhage: a prospective randomized trial. *Hepatology* 1995; 21:1517.
 62. Gimson AE, Ramage JK, Panos MZ, et al. Randomised trial of variceal banding ligation versus injection sclerotherapy for bleeding oesophageal varices. *Lancet* 1993; 342:391.
 63. Avgerinos A, Armonis A, Manolakopoulos S, et al. Endoscopic sclerotherapy versus variceal ligation in the long-term management of patients with cirrhosis after variceal bleeding. A prospective randomized study. *J Hepatol* 1997; 26:1034.
 64. Baroncini D, Milandri GL, Borioni D, et al. A prospective randomized trial of sclerotherapy versus ligation in the elective treatment of bleeding esophageal varices. *Endoscopy* 1997; 29:235.
 65. Schuman BM, Beckman JW, Tedesco FJ, et al. Complications of endoscopic injection sclerotherapy: a review. *Am J Gastroenterol* 1987; 82:823.
 66. Sanowski RA, Waring JP. Endoscopic techniques and complications in variceal sclerotherapy. *J Clin Gastroenterol* 1987; 9:504.
 67. Wright G, Lewis H, Hogan B, et al. A self-expanding metal stent for complicated variceal hemorrhage: experience at a single center. *Gastrointest Endosc* 2010; 71:71.
 68. Müller M, Seufferlein T, Perkhofer L, et al. Self-Expandable Metal Stents for Persisting Esophageal Variceal Bleeding after Band Ligation or Injection-Therapy: A Retrospective Study. *PLoS One* 2015; 10:e0126525.
 69. Marot A, Trépo E, Doerig C, et al. Systematic review with meta-analysis: self-expanding metal stents in patients with cirrhosis and severe or refractory oesophageal variceal bleeding. *Aliment Pharmacol Ther* 2015; 42:1250.
 70. McCarty TR, Njei B. Self-expanding metal stents for acute refractory esophageal variceal bleeding: A systematic review and meta-analysis. *Dig Endosc* 2016; 28:539.

71. Grace ND, Groszmann RJ, Garcia-Tsao G, et al. Portal hypertension and variceal bleeding: an AASLD single topic symposium. *Hepatology* 1998; 28:868.
72. Zhou GP, Jiang YZ, Sun LY, Zhu ZJ. Early transjugular intrahepatic portosystemic shunt for acute variceal bleeding: a systematic review and meta-analysis. *Eur Radiol* 2021; 31:5390.
73. Lv Y, Yang Z, Liu L, et al. Early TIPS with covered stents versus standard treatment for acute variceal bleeding in patients with advanced cirrhosis: a randomised controlled trial. *Lancet Gastroenterol Hepatol* 2019; 4:587.
74. LaBerge JM, Ring EJ, Gordon RL, et al. Creation of transjugular intrahepatic portosystemic shunts with the wallstent endoprosthesis: results in 100 patients. *Radiology* 1993; 187:413.
75. Rössle M, Haag K, Ochs A, et al. The transjugular intrahepatic portosystemic stent-shunt procedure for variceal bleeding. *N Engl J Med* 1994; 330:165.
76. Sanyal AJ, Freedman AM, Luketic VA, et al. Transjugular intrahepatic portosystemic shunts for patients with active variceal hemorrhage unresponsive to sclerotherapy. *Gastroenterology* 1996; 111:138.
77. Villeneuve JP, Pomier-Layrargues G, Duguay L, et al. Emergency portacaval shunt for variceal hemorrhage. A prospective study. *Ann Surg* 1987; 206:48.
78. Orloff MJ, Bell RH Jr, Hyde PV, Skivolocki WP. Long-term results of emergency portacaval shunt for bleeding esophageal varices in unselected patients with alcoholic cirrhosis. *Ann Surg* 1980; 192:325.
79. Rikkers LF, Jin G. Emergency shunt. Role in the present management of variceal bleeding. *Arch Surg* 1995; 130:472.
80. Ibrahim M, El-Mikkawy A, Mostafa I, Devière J. Endoscopic treatment of acute variceal hemorrhage by using hemostatic powder TC-325: a prospective pilot study. *Gastrointest Endosc* 2013; 78:769.
81. Holster IL, Poley JW, Kuipers EJ, Tjwa ET. Controlling gastric variceal bleeding with endoscopically applied hemostatic powder (Hemospray™). *J Hepatol* 2012; 57:1397.
82. Ibrahim M, El-Mikkawy A, Abdalla H, et al. Management of acute variceal bleeding using hemostatic powder. *United European Gastroenterol J* 2015; 3:277.
83. Stanley AJ, Smith LA, Morris AJ. Use of hemostatic powder (Hemospray) in the management of refractory gastric variceal hemorrhage. *Endoscopy* 2013; 45 Suppl 2 UCTN:E86.
84. Ibrahim M, El-Mikkawy A, Abdel Hamid M, et al. Early application of haemostatic powder added to standard management for oesophagogastric variceal bleeding: a randomised trial. *Gut* 2019; 68:844.

85. Huang YH, Yeh HZ, Chen GH, et al. Endoscopic treatment of bleeding gastric varices by N-butyl-2-cyanoacrylate (Histoacryl) injection: long-term efficacy and safety. *Gastrointest Endosc* 2000; 52:160.
86. Lee YT, Chan FK, Ng EK, et al. EUS-guided injection of cyanoacrylate for bleeding gastric varices. *Gastrointest Endosc* 2000; 52:168.
87. Lo GH, Lai KH, Cheng JS, et al. A prospective, randomized trial of butyl cyanoacrylate injection versus band ligation in the management of bleeding gastric varices. *Hepatology* 2001; 33:1060.
88. Sarin SK, Jain AK, Jain M, Gupta R. A randomized controlled trial of cyanoacrylate versus alcohol injection in patients with isolated fundic varices. *Am J Gastroenterol* 2002; 97:1010.
89. Romero-Castro R, Ellrichmann M, Ortiz-Moyano C, et al. EUS-guided coil versus cyanoacrylate therapy for the treatment of gastric varices: a multicenter study (with videos). *Gastrointest Endosc* 2013; 78:711.
90. Mishra SR, Chander Sharma B, Kumar A, Sarin SK. Endoscopic cyanoacrylate injection versus beta-blocker for secondary prophylaxis of gastric variceal bleed: a randomised controlled trial. *Gut* 2010; 59:729.
91. Sarin SK. Long-term follow-up of gastric variceal sclerotherapy: an eleven-year experience. *Gastrointest Endosc* 1997; 46:8.
92. Trudeau W, Prindiville T. Endoscopic injection sclerosis in bleeding gastric varices. *Gastrointest Endosc* 1986; 32:264.
93. Soehendra N, Nam VC, Grimm H, Kempeneers I. Endoscopic obliteration of large esophagogastric varices with bucrylate. *Endoscopy* 1986; 18:25.
94. Kind R, Guglielmi A, Rodella L, et al. Bucrylate treatment of bleeding gastric varices: 12 years' experience. *Endoscopy* 2000; 32:512.
95. Datta D, Vlavianos P, Alisa A, Westaby D. Use of fibrin glue (beriplast) in the management of bleeding gastric varices. *Endoscopy* 2003; 35:675.
96. Seewald S, Ang TL, Imazu H, et al. A standardized injection technique and regimen ensures success and safety of N-butyl-2-cyanoacrylate injection for the treatment of gastric fundal varices (with videos). *Gastrointest Endosc* 2008; 68:447.
97. Jutabha R, Jensen DM. Management of upper gastrointestinal bleeding in the patient with chronic liver disease. *Med Clin North Am* 1996; 80:1035.
98. Chau TN, Patch D, Chan YW, et al. "Salvage" transjugular intrahepatic portosystemic shunts: gastric fundal compared with esophageal variceal bleeding. *Gastroenterology* 1998; 114:981.

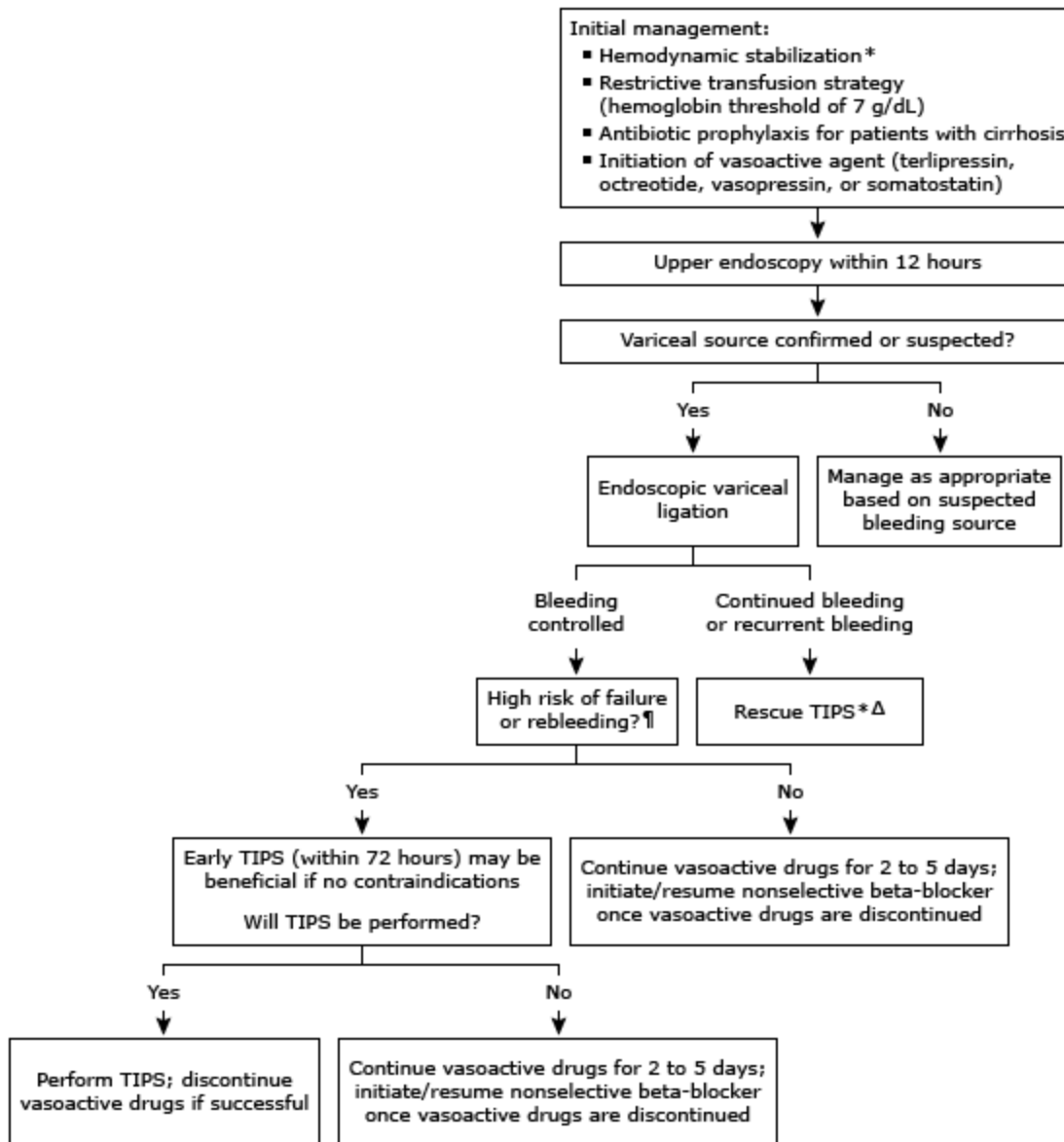
99. Hung HH, Chang CJ, Hou MC, et al. Efficacy of non-selective β -blockers as adjunct to endoscopic prophylactic treatment for gastric variceal bleeding: a randomized controlled trial. *J Hepatol* 2012; 56:1025.
100. Cheng LF, Wang ZQ, Li CZ, et al. Low incidence of complications from endoscopic gastric variceal obturation with butyl cyanoacrylate. *Clin Gastroenterol Hepatol* 2010; 8:760.
101. Binmoeller KF, Weilert F, Shah JN, Kim J. EUS-guided transesophageal treatment of gastric fundal varices with combined coiling and cyanoacrylate glue injection (with videos). *Gastrointest Endosc* 2011; 74:1019.
102. Shiha G, El-Sayed SS. Gastric variceal ligation: a new technique. *Gastrointest Endosc* 1999; 49:437.
103. Ramesh J, Limdi JK, Sharma V, Makin AJ. The use of thrombin injections in the management of bleeding gastric varices: a single-center experience. *Gastrointest Endosc* 2008; 68:877.
104. Przemioslo RT, McNair A, Williams R. Thrombin is effective in arresting bleeding from gastric variceal hemorrhage. *Dig Dis Sci* 1999; 44:778.
105. Yang WL, Tripathi D, Therapondos G, et al. Endoscopic use of human thrombin in bleeding gastric varices. *Am J Gastroenterol* 2002; 97:1381.
106. Lo GH, Lin CW, Tai CM, et al. A prospective, randomized trial of thrombin versus cyanoacrylate injection in the control of acute gastric variceal hemorrhage. *Endoscopy* 2020; 52:548.
107. Lee MS, Cho JY, Cheon YK, et al. Use of detachable snares and elastic bands for endoscopic control of bleeding from large gastric varices. *Gastrointest Endosc* 2002; 56:83.
108. Akahoshi T, Hashizume M, Tomikawa M, et al. Long-term results of balloon-occluded retrograde transvenous obliteration for gastric variceal bleeding and risky gastric varices: a 10-year experience. *J Gastroenterol Hepatol* 2008; 23:1702.
109. Cho SK, Shin SW, Lee IH, et al. Balloon-occluded retrograde transvenous obliteration of gastric varices: outcomes and complications in 49 patients. *AJR Am J Roentgenol* 2007; 189:W365.
110. Yoshimatsu R, Yamagami T, Tanaka O, et al. Development of thrombus in a systemic vein after balloon-occluded retrograde transvenous obliteration of gastric varices. *Korean J Radiol* 2012; 13:324.
111. Saad WE, Wagner CC, Lippert A, et al. Protective value of TIPS against the development of hydrothorax/ascites and upper gastrointestinal bleeding after balloon-occluded retrograde transvenous obliteration (BRTO). *Am J Gastroenterol* 2013; 108:1612.

112. Gwon DI, Ko GY, Yoon HK, et al. Gastric varices and hepatic encephalopathy: treatment with vascular plug and gelatin sponge-assisted retrograde transvenous obliteration--a primary report. *Radiology* 2013; 268:281.
113. Bazarbashi AN, Wang TJ, Jirapinyo P, et al. Endoscopic Ultrasound-Guided Coil Embolization With Absorbable Gelatin Sponge Appears Superior to Traditional Cyanoacrylate Injection for the Treatment of Gastric Varices. *Clin Transl Gastroenterol* 2020; 11:e00175.
114. Bernstein D, Yrizarry J, Reddy KR, et al. Transjugular intrahepatic portosystemic shunt in the treatment of intermittently bleeding stomal varices. *Am J Gastroenterol* 1996; 91:2237.
115. Allgaier HP, Ochs A, Haag K, et al. [Recurrent bleeding from colonic varices in portal hypertension. The successful prevention of recurrence by the implantation of a transjugular intrahepatic stent-shunt (TIPS)]. *Dtsch Med Wochenschr* 1995; 120:1773.
116. Sort P, Elizalde I, Llach I, et al. Duodenal variceal bleeding treated with a transjugular intrahepatic portosystemic shunt. *Endoscopy* 1995; 27:626.
117. Deipolyi AR, Kalva SP, Oklu R, et al. Reduction in portal venous pressure by transjugular intrahepatic portosystemic shunt for treatment of hemorrhagic stomal varices. *AJR Am J Roentgenol* 2014; 203:668.
118. Hashimoto N, Akahoshi T, Yoshida D, et al. The efficacy of balloon-occluded retrograde transvenous obliteration on small intestinal variceal bleeding. *Surgery* 2010; 148:145.

Topic 1260 Version 49.0

GRAPHICS

Management of acute variceal hemorrhage



TIPS: transjugular intrahepatic portosystemic shunt; CTP: Child-Turcotte-Pugh.

* Temporizing measures to acutely control hemorrhage include balloon tamponade and endoscopic esophageal covered stent placement (for patients with uncontrolled bleeding despite endoscopic therapy). Balloon tamponade is associated with significant complications and rebleeding upon balloon deflation. If balloon tamponade is chosen, the patient should be intubated prior to balloon placement.

¶ CTP class C cirrhosis or CTP class B with active bleeding at time of endoscopy.

Δ If bleeding is modest, repeat endoscopic therapy may be attempted.

Reference:

1. *Garcia-Tsao G, Sanyal AJ, Grace ND, Carey W, Practice Guidelines Committee of the American Association for the Study of Liver Diseases, Practice Parameters Committee of the American College of Gastroenterology. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. Hepatology 2007; 46:922.*
-

Graphic 129181 Version 1.0

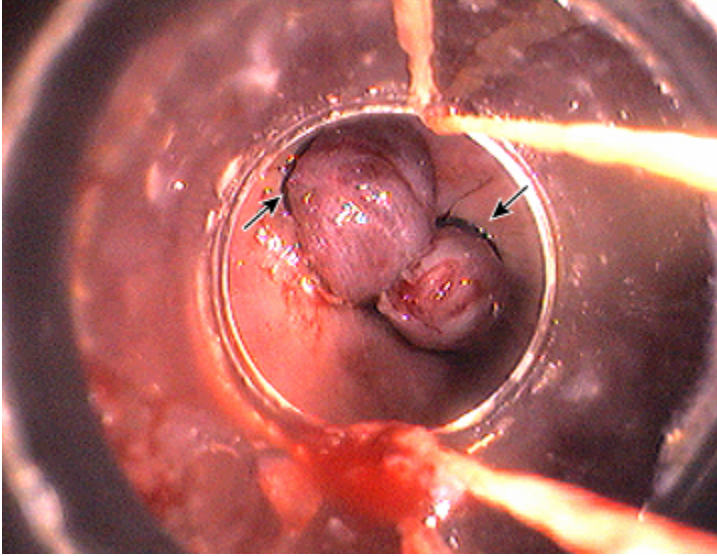
Complications in patients undergoing emergent or elective sclerotherapy for variceal bleeding

Local complications
Ulcers
Bleeding
Stricture
Esophageal dysmotility
Pain
Odynophagia
Laceration
Regional
Esophageal perforation
Mediastinitis
Pleural effusions
Acute gastric dilation
Systemic
Sepsis
Aspiration
Spontaneous bacterial peritonitis and candidemia
Ventilation-perfusion mismatch (hypoxia)
Adult respiratory distress syndrome
Portal vein thrombosis
Complications to physician
Sclerotherapist's eye

Adapted from Sanyal AJ. Semin Liver Dis 1993; 13:4.

Graphic 71037 Version 3.0

Esophageal varix band ligation

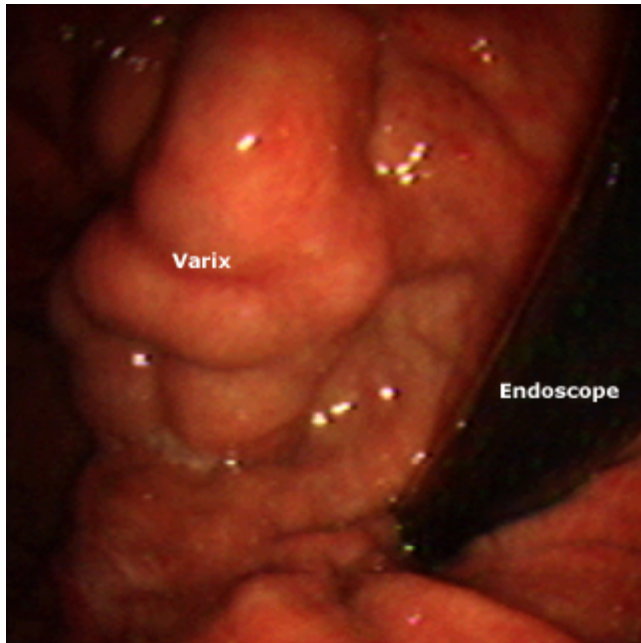


Endoscopy shows two varices in the distal esophagus that have been banded. The black bands are indicated with the arrows. The two strings in the right of the field are connected to the trigger device used to deploy the bands.

Courtesy of Laurence Bailen, MD.

Graphic 54194 Version 3.0

Gastric varix

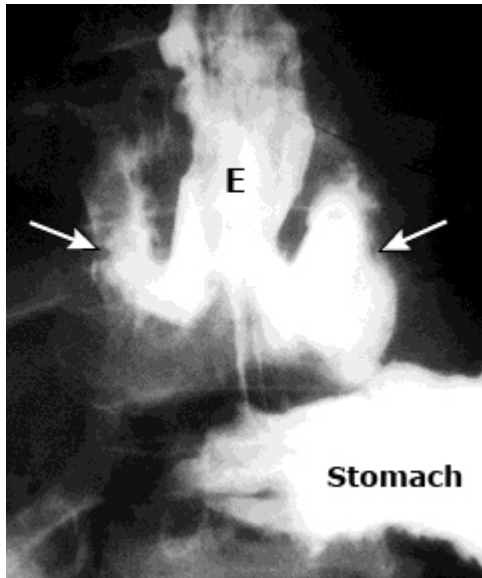


Upper endoscopy of a gastric varix in the fundus of the stomach. Gastric varices can arise in conjunction with esophageal varices. They can also be isolated when they result from segmental portal hypertension due to obstruction of the splenic vein by pancreatic carcinoma or chronic pancreatitis.

Courtesy of Rome Jutabha, MD, and Dennis M Jensen, MD.

Graphic 58644 Version 1.0

Esophageal perforation



This barium swallow study was performed in a 45-year-old man who presented with severe chest pain following sclerotherapy for esophageal varices. The study demonstrates two large perforations (arrows) arising from the distal esophagus (E).

Courtesy of Jonathan Kruskal, MD, PhD.

Graphic 82026 Version 3.0

Portal hypertensive gastropathy

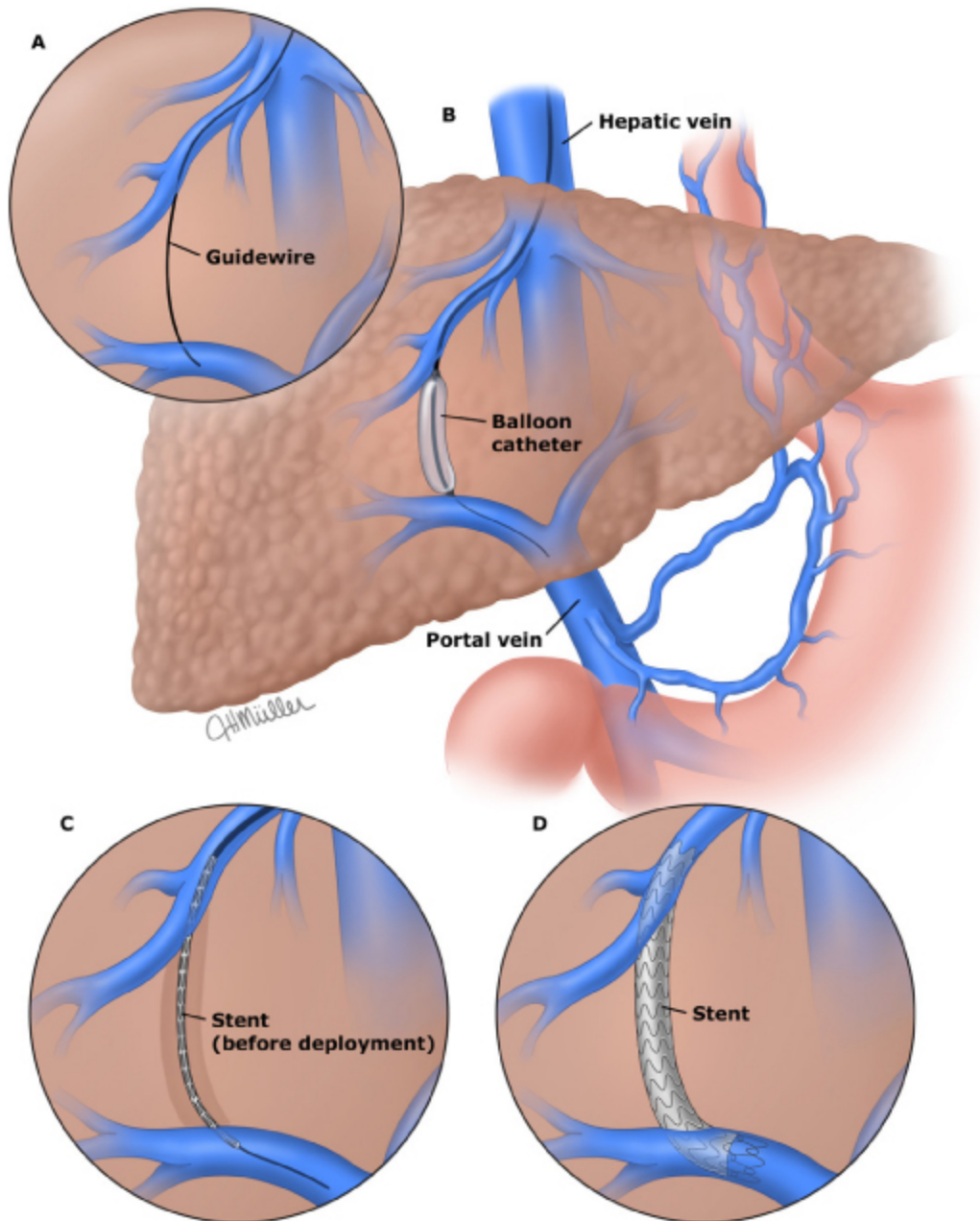


Endoscopy in portal hypertensive gastropathy reveals a characteristic fine white reticular pattern separating areas of pinkish mucosa, giving the gastric mucosa a "snakeskin" appearance.

Courtesy of Rome Jutabha, MD and Dennis M Jensen, MD.

Graphic 75725 Version 1.0

Transjugular intrahepatic portosystemic shunt



A transjugular intrahepatic portosystemic shunt (TIPS) is created by passing a needle catheter via the transjugular route into the hepatic vein and wedging it there. The needle is then extruded and advanced through the liver parenchyma to the intrahepatic portion of the portal vein and a stent is placed between the portal and hepatic veins. A TIPS functions like side-to-side surgical portacaval shunt, but does not require general anesthesia or major surgery for placement. (A) Passage of a guidewire between the hepatic vein and the portal vein. (B) Inflation of a balloon catheter within the liver to dilate the tract between the hepatic vein and the portal vein. (C) Deployment of the stent. (D) Stent in its final position.

Graphic 72311 Version 2.0

Contributor Disclosures

Jasmohan S Bajaj, MD Grant/Research/Clinical Trial Support: Bausch [hepatic encephalopathy]; Grifols [hepatic encephalopathy]; Mallinckrodt [hepatic encephalopathy]; Sequana [hepatic encephalopathy]. Consultant/Advisory Boards: Galecto [hepatic encephalopathy]; Merz [hepatic encephalopathy]; Seres [hepatic encephalopathy]. All of the relevant financial relationships listed have been mitigated. **Arun J Sanyal, MD** Equity Ownership/Stock Options: Durect [NASH]; Exhale NZ [Helicobacter pylori]; Genfit [NASH]; HemoShear [Rare liver diseases]; Indalo [NASH]; NorthSea [NASH]; Rivus [NASH]; Sanyal Bio [Animal testing]; Tiziana [NASH]. Grant/Research/Clinical Trial Support: Alnylam [NASH]; Amgen [NASH]; Boehringer Ingelheim [NASH]; Bristol Myers [NASH]; Covance [Lipoproteins]; Echosens Sandhill [NASH]; Fractyl [NASH]; Genentech [NASH]; Gilead [NASH]; HistoIndex [NASH]; Immuron [Alcoholic hepatitis]; Inventiva [NASH]; Lilly [NASH]; Madrigal [NASH]; Mallinckrodt [Portal hypertension]; Merck [NASH]; Novartis [NASH]; Novo Nordisk [NASH]; Owl [NASH]; Path AI [NASH]; Pfizer [NASH]; ProSciento [NASH]; Regeneron [NASH]; Roche [NASH]; Salix [Hepatic encephalopathy]; Second Genome [Microbiome]; Siemens [NASH]. Consultant/Advisory Boards: 89 Bio [NASH]; Albireo [NASH]; Amgen [NASH]; Amra [Metabolism]; AstraZeneca [NASH]; BiocellVia [NASH]; Boehringer Ingelheim [NASH]; Bristol Myers [NASH]; Conatus [NASH]; Fractyl [NASH]; Galectin [NASH]; Genentech [NASH]; Genfit [NASH]; Gilead [NASH, COVID-19]; HemoShear [Rare diseases]; HistoIndex [NASH]; Immuron [Alcohol-associated liver disease]; Intercept [NASH]; Janssen [NASH]; Lilly [NASH]; Madrigal [NASH]; Mallinckrodt [Portal hypertension]; Merck [NASH]; NGM Bio [NASH]; NorthSea [NASH]; Novartis [NASH]; Novo Nordisk [NASH]; PathAI [NASH]; Perspectum [NASH]; Pfizer [NASH]; Poxel [NASH]; ProSciento [NASH]; Regeneron [NASH]; Roche [NASH]; Salix [Cirrhosis]; Sanofi [NASH]; Sequana [Cirrhosis]; Siemens [NASH]; Takeda [NASH]; Terns [NASH]. All of the relevant financial relationships listed have been mitigated. **John R Saltzman, MD, FACP, FACG, FASGE, AGAF** No relevant financial relationship(s) with ineligible companies to disclose. **Anne C Travis, MD, MSc, FACG, AGAF** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

[Conflict of interest policy](#)

→