Complications of Transjugular Intrahepatic Portosystemic Shunt: A Comprehensive Review[1]

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It is generally accepted that the transjugular intrahepatic portosystemic shunt (TIPS) procedure has lower morbidity and mortality rates than those of surgical shunting. Nevertheless, complications occur. The authors have reviewed their experience and that of other institutions in compiling an extensive list of complications. Complications are categorized according to those related to transhepatic needle puncture, transvenous access to the portal vein, portal venous cannulation, the stent, the puncture site, portosystemic shunting, and contrast material. Excluding hepatic encephalopathy and delayed stenosis or occlusion of the shunt, an overall complication rate of less than 10% can be expected for TIPS. The prevalence of aggravated or new cases of encephalopathy is 5%–35%, and over the long term, up to 75% of shunts may undergo stenosis or occlusion. The direct procedural mortality rate is less than 2%, and the 30-day mortality rate ranges from 4% to 45%, depending on several factors. The role to which TIPS is relegated will be influenced by the long-term success rate in the prevention of recurrent variceal hemorrhage.

Abbreviation: TIPS = transjugular intrahepatic portosystemic shunt

Index terms: Interventional procedures, complications, 76.458, 76.715, 95.44 • Liver, interventional procedure, 761.1229, 95.1268 • Shunts, portosystemic, 95.1268, 95.4532

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■ INTRODUCTION

Several reports of clinical series of patients undergoing the transjugular intrahepatic portosystemic shunt (TIPS) procedure have been published over the past several years (1–10). Additional case reports of unusual applications, problems, and technical notes have also been published (11–16). Numerous presentations on TIPS have taken place at major radiologic, surgical, and gastrointestinal meetings. However, to the best of our knowledge, there have been no published reports dedicated to addressing the complications that may occur as a result of the TIPS procedure. Although the relatively low invasiveness of TIPS offers many advantages over surgical portosystemic shunting, serious complications may still occur (1–34; Jones-Bey H. Radiologists exchange latest tips on TIPS. Diagnostic Imaging, Jan 1993, 23–30). Complications that are unique to TIPS, angiographic procedures, and portosystemic shunting in general may occur.

We have reviewed our experience and that of other investigators in an attempt to provide a comprehensive overview of the complications and the approximate frequencies with which they can be expected. The Table provides a summary of this overview and lists the complications categorized according to specific aspects of the TIPS procedure. In the remaining text, examples of many complications are followed by discussions dealing with their prevention and treatment in detail, whereas other complications are included for the sake of completeness. We hope that this review will create awareness of potential TIPS-related complications, provide clues to their prevention, and aid in their successful resolution, should they occur.

■ MORTALITY AND COMPLICATION RATES

Within the past few years, the TIPS procedure has become universally accepted as a tool in the management of complications associated with portal hypertension, particularly variceal bleeding. For the first time, a low-invasive procedure is available to reduce the portal venous pressures without requiring surgical laparotomy and its associated complications. The 30-day mortality rate for patients undergoing an emergency portacaval shunt is 40%–100%; the rate is 4%–20% for an elective procedure (37–41). By comparison, TIPS yields a favorable 30-day mortality rate of 45%, and in most cases the rate is substantially less (Table). Furthermore, the direct TIPS-related procedural mortality rate is less than 2%, with most deaths resulting from intraprocedural myocardial infarctions or intraperitoneal hemorrhage as a result of rupture of the portal vein or transcapsular puncture with the needle. In most cases, such complications would be expected to decline in frequency as more experience is gained with the procedure. Other potentially life-threatening complications include anaphylactic reactions to contrast material, acute renal failure related to contrast material, and uncontrollable encephalopathy. Most other complications that are reported, although serious, should not result in a fatal outcome.

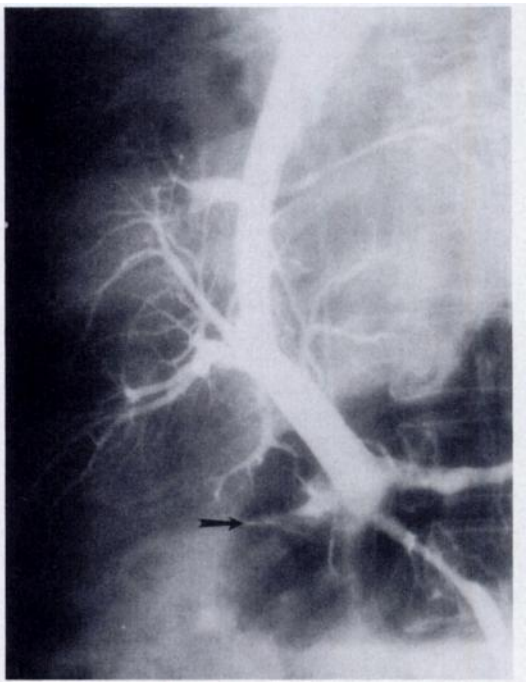
In our experience, the 30-day mortality rate in 59 patients who underwent a technically successful TIPS procedure was 20% (12 patients). Of the remaining five patients in our series in whom TIPS was technically unsuccessful (due to occlusion of the portal vein in one and prior portacaval surgery in the second), two died as a result of complications associated with continued variceal bleeding. All patients who died, with the exception of one, were Child class C patients. Twelve of the total 14 patients who died in the 30-day period following successful or failed TIPS were actively bleeding at the time of TIPS. Of the 23 (36%) actively bleeding patients in our series, 14 (61%) have survived 30 days after TIPS (39% mortality). In one patient who was urgently sent for TIPS, the procedure was technically impossible. However, she spontaneously stopped bleeding and is now undergoing chronic sclerotherapy. We have encountered only one (1.5%) direct procedural related death, which was secondary to intraperitoneal hemorrhage. The remaining 13 patients died of complications attributable to their end-stage liver disease, multisystem organ failure, or serious respiratory problems.

Complications Associated with TIPS

Category of Complication	UCSF	MVI	UH/UF	Other (%)*	MCV†
No. of patients with technical success/no. of total patients	96/100‡	76/76	111/120	91–100¶	59/64
Re-bleeding (%; mean follow-up in mo)	10; 4.7	4§	10; 8‖	6.7–36	11; 7
Direct procedural mortality (%)	None	None	<1	1–2	1.5
30-day mortality (%)	13	7	11	7.7–45	20
Procedural morbidity (%)	NR	15	NR	Up to 18	20
Related to transhepatic needle puncture					
Transcapsular puncture (%)	Many#	NR	NR	5	30
Biliary duct puncture (%)	Several**	NR	NR	<5	<5
Hepatic artery puncture (%)	NR	NR	NR	NR	1.5
Gallbladder puncture (%)	Several	NR	NR	<5	5–10
Right kidney puncture (%)	NR	NR	NR	NR	1.5
Related to the transvenous access to the portal vein					
Cardiac arrhythmias (%)	NR	NR	NR	NR	<5††
Right atrial puncture	NR	NR	NR	NR	None
Inferior vena caval puncture	NR	NR	NR	NR	None
Related to cannulation/dilation of the portal vein					
Portal venous rupture (%)	None	1.3	None	<1‡‡	None
Splenoportal venous thrombosis (%)	NR	5	NR	NR	9
Related to the stent					
Acute stent thrombosis (%)	NR	NR	NR	Up to 10	3
Delayed shunt occlusion (%)	9	3 at 1 y	5	2.2–15	5
Delayed shunt stenosis (stent/hepatic vein) (%)	6§§	36	30‖	4.4–77	60–75
Stent shortening/incomplete placement (%)	2	NR	NR	Up to 10	1.5
Stent extension/protrusion into the inferior vena cava (%)	NR	NR	NR	NR	5
Stent separation/dislodgment/migration (%)	1.1	1.3, Palmaz stent	NR	<1	3
Stent-induced hemolysis (%)	NR	NR	NR	NR	1.5‖‖
Related to the puncture site					
Hematoma/bleeding (%)	4.5	NR	4##	NR	1.5
Inadvertent carotid artery puncture (%)	NR	NR	NR	NR	3.0
Inadvertent tracheal puncture (%)	NR	NR	NR	NR	1.5
Related to portosystemic shunting					
New/worsened hepatic encephalopathy (%)	17	13	New: 5‖	13–35	25***
Fever/sepsis (%)	10	NR	<1	2.5–10	10†††
Deterioration of liver function	None	None, at 4 mo	None	Up to 7	None
Myocardial infarction (%)	1	NR	NR	Up to 2	None
Generalized coagulopathy (%)	NR	NR	4	Up to 3	None
Related to contrast material					
Allergic reaction	NR	NR	NR	NR	None
Nephrotoxicity/acute renal failure (%)	4	NR	NR	Up to 3	1.5

Note.—MCV = Medical College of Virginia (Richmond), MVI = Miami Vascular Institute (Baptist Hospital, Miami, Fla), NR = not reported, UCSF = University of California San Francisco, UH/UF = Ruprect-Karls-University (Heidelberg, Germany)/Albert Ludwig University (Freiburg, Germany). * Data acquired from smaller series, case reports, published abstracts, and meeting presentations from multiple centers (14–16,18, 19,22–31,34; Jones-Bey H. Radiologists exchange tips on TIPS. Diagnostic Imaging, Jan 1993, 23–30). † Informed consent was obtained before TIPS. ‡ TIPS was successfully performed in seven of 10 patients with portal venous occlusion. ¶ For these multiple sources, the numbers represent the technical success rate (%). § Mean follow-up was unspecified. ‖ From reference 32. # One patient with intraperitoneal hemorrhage required two units of red blood cells. ** Includes one patient with hemobilia. †† All cases of arrhythmia were transient and not fatal. ‡‡ Includes one case of portal vein dissection. §§ Includes cases with 50% or greater stenosis. ‖‖ From reference 35. ## Bleeding occurred at the percutaneous transhepatic site in four patients and at the transjugular site in one. *** From reference 36. ††† Includes only one documented case of TIPS-induced sepsis.

a.



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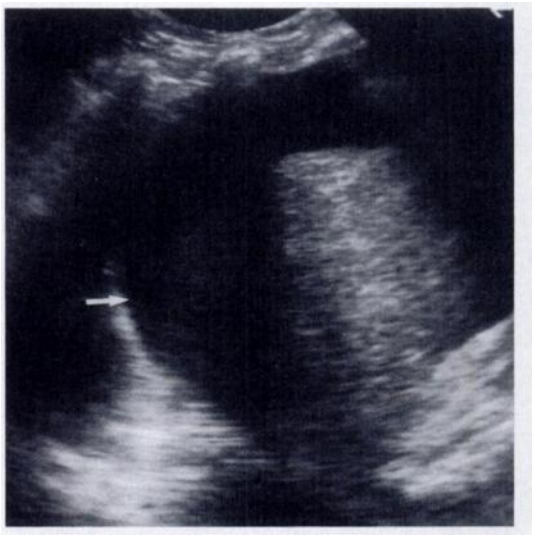


b.

Figure 1. Hemoperitoneum due to traversal of the liver capsule. **(a)** Portogram obtained minutes after injection through the Colapinto needle shows residual intraperitoneal and intrahepatic extravasated contrast material (arrows). The contrast material outlines the liver within the peritoneal cavity (arrowheads). Marked hypotension, occurring immediately after TIPS, prompted emergency sonography and repeat portography. **(b)** Sonogram reveals a large amount of blood (arrow). **(c)** Repeat portogram shows excellent blood flow through the shunt and no extravasation from the portal vein. A small amount of contrast material from the earlier injection into the peritoneal cavity persists (arrow).

The 30-day mortality rate after TIPS reported by most other investigators ranges from 7% to 45% (1–34). As in our experience, the majority of these deaths occurred in Child class C patients, many who were actively bleeding at the time of the procedure.

A review of results of multiple investigators, including all possibly related complications such as hepatic encephalopathy and delayed stenosis or occlusion of the shunt, reveals an overall procedural complication rate of approximately 4%–77% (1–34). However, for all practical purposes, when we exclude encephalopathy and delayed shunt stenosis or occlusion, the complication rate is 10% or less.

A fair comparison of overall complication rates among different institutions is extremely difficult for several reasons. First, the experience with TIPS is still limited and published reports are few. Second, there are substantial differences in the number of procedures performed, patient selection and evaluation, rec-

ognition and reporting of complications, and mean follow-up times. In addition, because the assessment of some complications such as hepatic encephalopathy is often subjective rather than objective and because other parameters such as laboratory values are not uniformly recorded at all institutions, complication rates may vary among different institutions.

For instance, marked thrombosis of the portal vein or stent is a major complication; however, a small, nonocclusive, clinically insignificant thrombus in the portal vein is a minor complication and may go unreported at some centers. New-onset hepatic encephalopathy may be considered either a complication or an accepted outcome of the procedure. It becomes apparent that the definition of what is a complication needs clarification. In our review, we have used an inclusive approach to define what represents a complication of TIPS in order to present a comprehensive list and to allow discussion of several important aspects of the procedure.

Additional data encompassing the experience of multiple institutions are necessary to completely evaluate the complication rates associated with the TIPS procedure. At the Miami Vascular Institute, a TIPS registry is being established. Data from multiple institutions are being collected, and it is hoped that some useful answers will be forthcoming.

■ COMPLICATIONS RELATED TO THE TRANSHEPATIC NEEDLE PUNCTURE

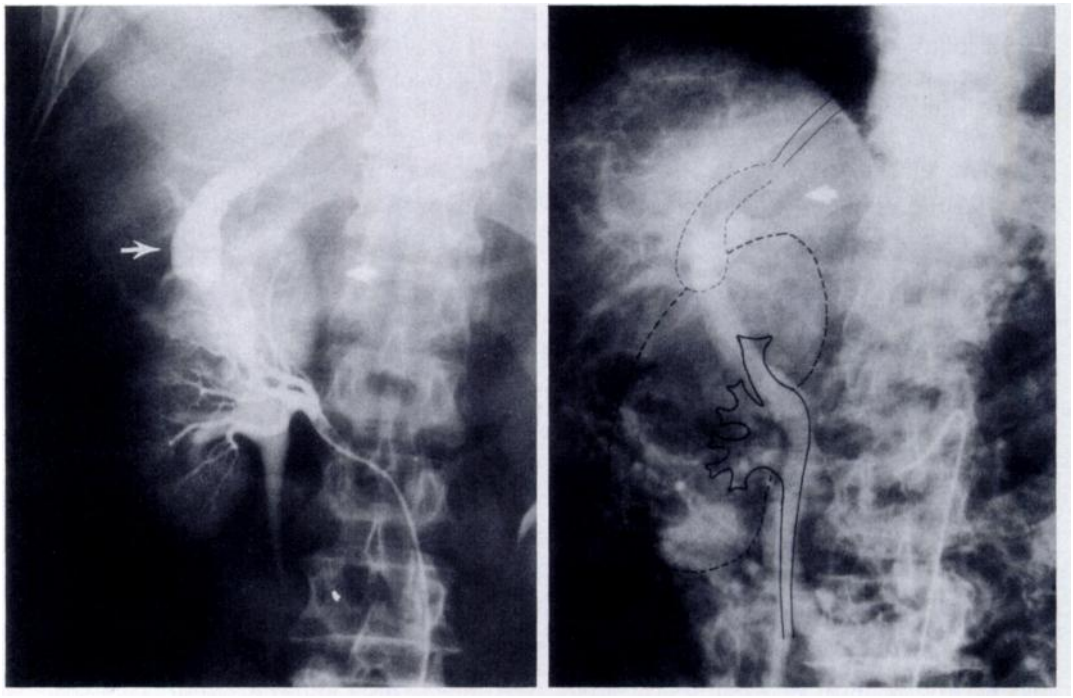
The most technically difficult and potentially the most crucial aspect of the TIPS procedure is the transhepatic puncture of the portal vein. In general, the fewer passes required to puncture the portal vein the safer the procedure.

Several different methods of localizing the portal vein have been used. The first few times TIPS was performed with initial percutaneous transhepatic cannulation and opacification of the portal vein (7). Interventional radiologists eventually became familiar enough with the transhepatic cannulation of the portal vein so that this initial opacification was no longer considered necessary. Today, only in instances of portal venous occlusion is percutaneous catheterization of the portal vein necessary (42). Instead, in most cases, TIPS can be performed by using wedged hepatic venography with retrograde opacification of the portal vein, a low-risk and technically easy maneuver. This technique is successful in opacifying the portal vein 76% of the time (43). Still, many investigators (including the authors) find arterial mesenteric portography to be safe, quick, and very useful in localizing the portal vein (2). Others rely primarily on ultrasound (US) guidance, sometimes augmented by insertion of a 0.018-inch guide wire (44) or a microembolization coil (45). Cannulation of a patent umbilical vein has also been described as a method of opacifying the portal venous system (46). Still other radiologists who have much experience with TIPS do not perform any procedures for prior localization of the portal vein, with the exception of fluoroscopy.

Traversal of the liver capsule with the needle is not uncommon, occurring in up to 30% of patients. Transcapsular puncture is particularly likely in patients with small livers or when multiple needle passes are required. We have encountered cases in which the optimal location for cannulation of the right portal vein lies only a centimeter or so away from the liver capsule. Ordinarily, traversal of the liver capsule with a 16-gauge Colapinto (Cook, Bloomington, Ind) needle is well tolerated and carries few adverse sequelae (1).

We have seen only one case of serious hemoperitoneum resulting from traversal of the liver capsule. In a patient with abnormal coagulation, a difficult transhepatic puncture of the portal vein led to multiple passes with the Colapinto needle (Fig 1) and traversal of the liver capsule twice. At emergency surgery, two holes in the liver capsule were found and repaired. Despite successful repair, the patient's debilitated condition led to eventual death.

When possible, it is reasonable to embolize the transcapsular needle tract with a pledget of gelatin sponge. Furthermore, we believe that prophylactic correction of clotting abnormalities with infusion of fresh frozen plasma and platelets is advisable before attempting the transhepatic cannulation puncture of the portal vein. In addition, because of risks of intraperitoneal bleeding, we do not recommend heparinization to prevent intraprocedural splenoportal thrombosis.



a. b.

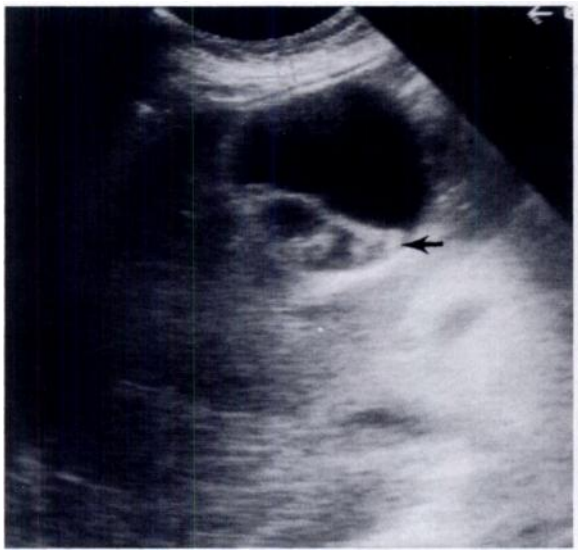
Figure 2. Puncture of the kidney and gallbladder. **(a)** Right renal arteriogram obtained after acute hematuria shows the gallbladder inadvertently opacified (arrow) by contrast material. There is no arterial injury. **(b)** On a later arteriogram, the gallbladder and the right kidney are outlined to illustrate the proximity of these organs to the bifurcation of the portal vein, which is due to the markedly decreased size of the liver. The right hepatic vein is also outlined. The large radiopaque arrow (in **a** and **b**) was a lead marker that was placed on the anterior skin of the patient and was used to assist the radiologist in entering the portal vein.

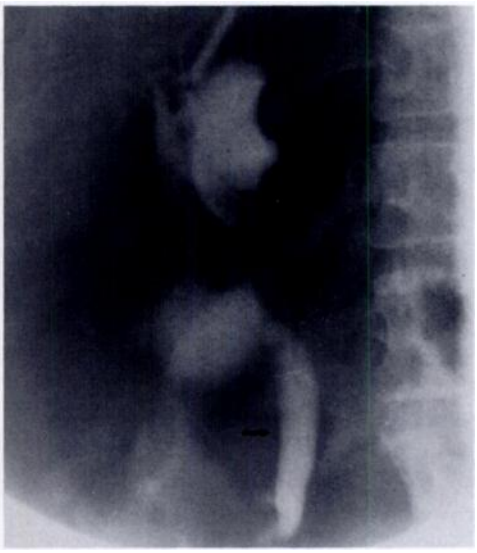
Structures close to the porta hepatis include the biliary ducts, hepatic arteries, gallbladder, and right kidney. All are prone to inadvertent puncture. In one patient in our series, both the right kidney and the gallbladder were entered with the Colapinto needle; the puncture was suspected due to self-limited gross hematuria (Fig 2). Theoretically, a very long tract between the hepatic and portal veins requiring straightening of the transhepatic needle may result in puncture or laceration of the right kidney, particularly if the kidney is in an anterior location.

Inadvertent puncture of the gallbladder or bile ducts (Figs 3, 4) is usually well tolerated; however, hemobilia or cholangitis may occasionally occur and has been reported (1,47). Intrabiliary blood clots can also result in biliary obstruction and then resolve spontaneously (Fig 5). In another patient, the left hepatic artery was punctured during attempted transhepatic puncture of the portal vein for placement of a second stent (Fig 6). There were no adverse sequelae.

Fistulas may also develop between any two vascular structures within the liver, including arteries, hepatic veins, portal veins, and bile ducts. One can imagine how easily fistulas may be initiated, since the transhepatic puncture traverses approximately 3–5 cm of liver parenchyma near the porta hepatis, the most vascular region of the liver.

The sequelae resulting from a fistula depend on which structures are involved. Fistulas between the hepatic vein and any arterial





3. 4.

Figures 3, 4. (3) Puncture of the gallbladder. Sonogram obtained shortly after TIPS reveals dependent echoes (arrow), indicating blood clot within the gallbladder following inadvertent puncture. (4) Puncture of a bile duct. Cholangiogram obtained unintentionally after injection of contrast material (arrows) as the transhepatic needle was retracted.

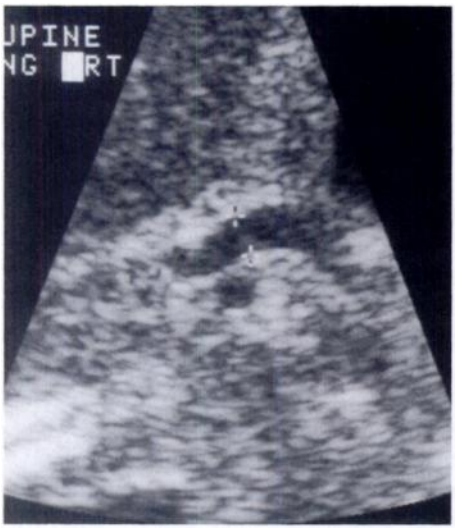


Figure 5. Puncture of a bile duct. Sonogram shows that the common bile duct has become distended to 8.5 mm due to obstruction from clot.

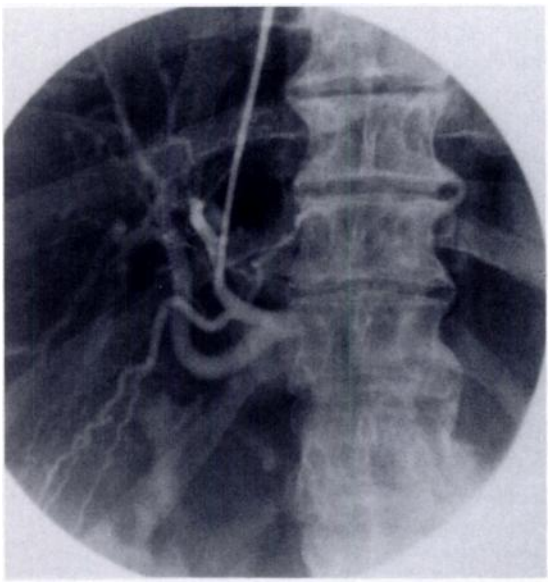


Figure 6. Puncture of the left hepatic artery. Portogram shows that the left hepatic artery is opacified after injection of contrast material through the Colapinto needle.

or portal vein branch will self-decompress into the systemic venous circulation. These fistulas are expected to be limited in size by the diameter of the transhepatic needle. Although such fistulas would most likely be clinically asymptomatic, percutaneous arterial embolization may be necessary to treat fistulas involving a hepatic artery.

One type of fistula may occur between the stent and biliary system (Fig 7). It has been suggested that such a fistula may result in early occlusion of the stent due to marked pseudointimal hyperplasia (48). In the illustrated case, a 2-month-old TIPS had occluded, and injection of contrast material into the lumen of the stent to determine the cause of the shunt failure resulted in inadvertent opacification of biliary ducts.

All of these examples reinforce the fact that several structures in the region of the portal venous confluence lie close to each other. Puncture or compression of these structures is occasionally unavoidable. As more experience with the technique of transhepatic portal venous puncture is gained, the frequency of these inadvertent punctures should decrease.

To improve further the safety of the transhepatic portal venous puncture, some operators prefer using a smaller diameter needle such as the Brockenborough needle (USCI Division, Bard, Billerica, Mass), an 18-gauge needle that takes a 0.018-inch guide wire, instead of the 16-gauge Colapinto needle that takes up to a 0.045-inch guide wire. Disadvantages of the Brockenborough needle, however, are its decreased stiffness and the use of an 0.018-inch guide wire, both of which may limit successful passage of a catheter into the portal vein in some cirrhotic livers. Smaller gauge transhepatic needles have been developed specifically for TIPS and may reduce the prevalence of complications attributable to the needle even further (49). One of these systems has a 0.038-inch trocar stylet (Rosch-Uchida set; Cook). This system provides adequate needle stiffness with a potentially safer portal venous puncture.

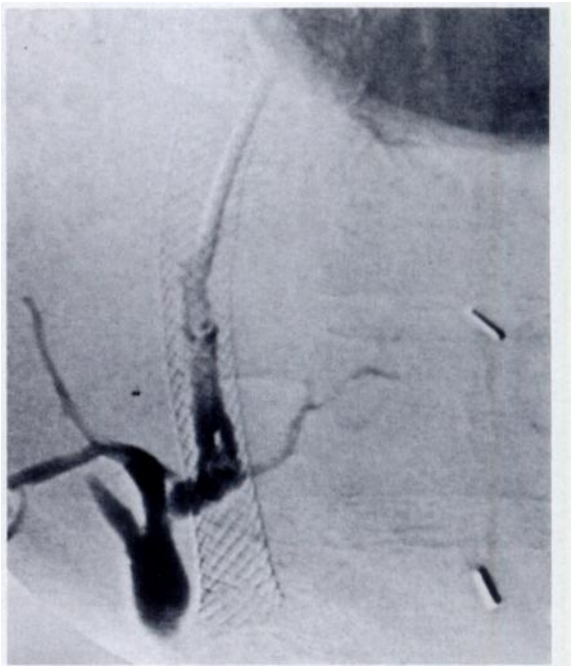
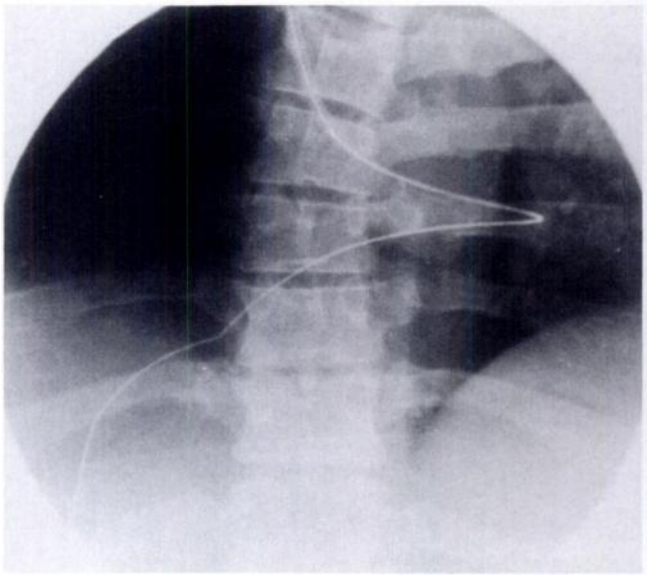


Figure 7. Fistula between the stent and bile ducts. Subtraction portogram obtained to determine the cause of stent occlusion shows contrast material in the biliary system.

■ COMPLICATIONS RELATED TO THE TRANSVENOUS ACCESS TO THE PORTAL VEIN

Because the approach to the liver is through the venous system, the catheter and guide wire system must traverse the right atrium, in addition to the superior and inferior venae cavae. Irritation of the conducting system of the heart may occur if a guide wire or catheter should buckle within the right atrium. This is more likely to occur when the liver parenchyma is very hard and substantial effort is needed to advance catheters into the portal vein. Ectopic atrial or ventricular beats or tachyarrhythmias may occur. In the illustrated case, prolapse of the guide wire resulted in a complex arrhythmia over several seconds (Fig 8a, 8b). Theoretically, fatal ventricular tachycardia, arteriovenous nodal block, or right bundle branch block may result. A preexistent left bundle branch block may necessitate prophylactic insertion of a temporary pacemaker, since there is a 5% chance of inducing a fatal right bundle branch block.

If the guide wire buckles, one may use the Check Flow II introducer, which is part of the Ring Transjugular Intrahepatic Access Set (Cook). This 40-cm-long sheath provides additional stiffness and may lessen the chances of guide wire and catheter prolapse (Fig 8c).



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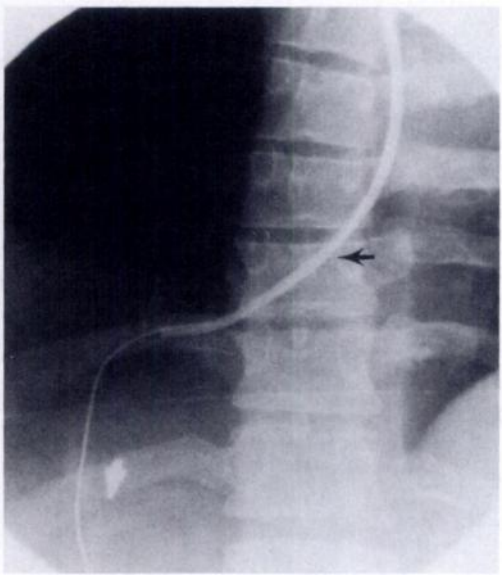

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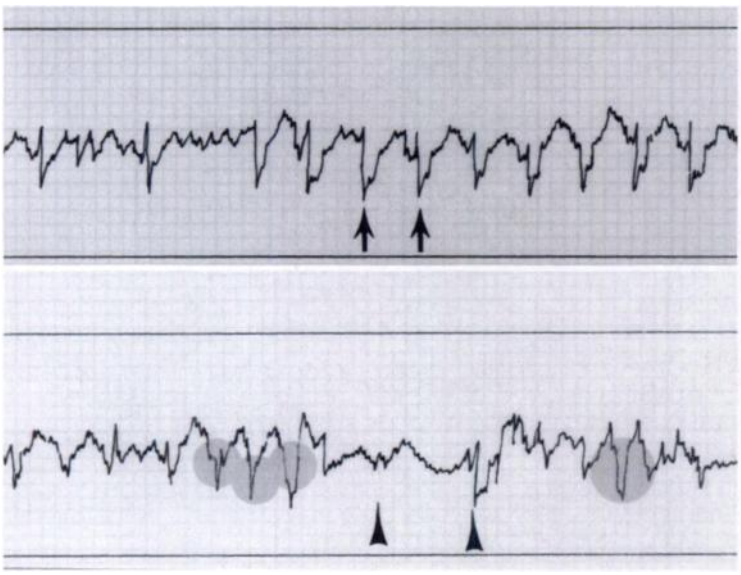
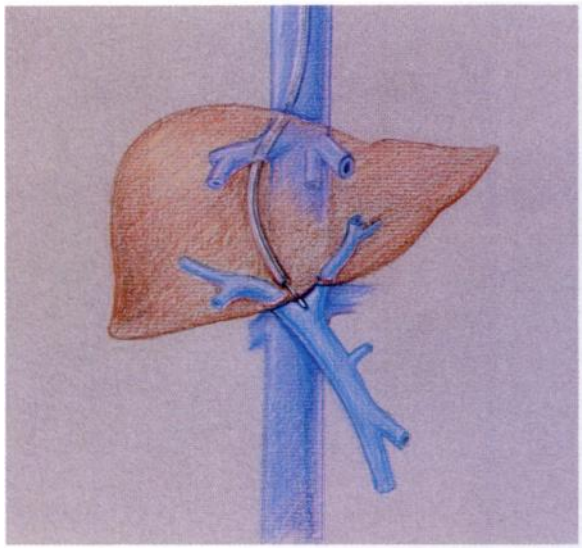


b.

Figure 8. Complex arrhythmia. **(a)** Spot radiograph reveals buckling of the guide wire and catheter into the right atrium and ventricle. **(b)** Simultaneous electrocardiographic recording demonstrates a new transient block of the right bundle branch (arrows), three ectopic ventricular beats (overlapping circles), and then several fusion beats (arrowheads), followed by another premature ventricular beat (single circle). **(c)** Use of the Check-Flow introducer prevents guide wire prolapse into the heart (arrow).

The Rosch-Uchida set also provides additional stiffness to the transjugular needle, wires, and catheters, as well as further protection of the heart and vena cava (18).

A potential theoretic complication, which has not yet been reported, is puncture or laceration of the right atrium or vena cava with the Colapinto (or other transhepatic) needle. The needle should always be advanced through a guiding sheath over a stiff guide wire, under fluoroscopic guidance. This is safer and makes needle passage easier. Furthermore, the needle should never project beyond the tip of the guiding sheath, unless it is inside the liver.



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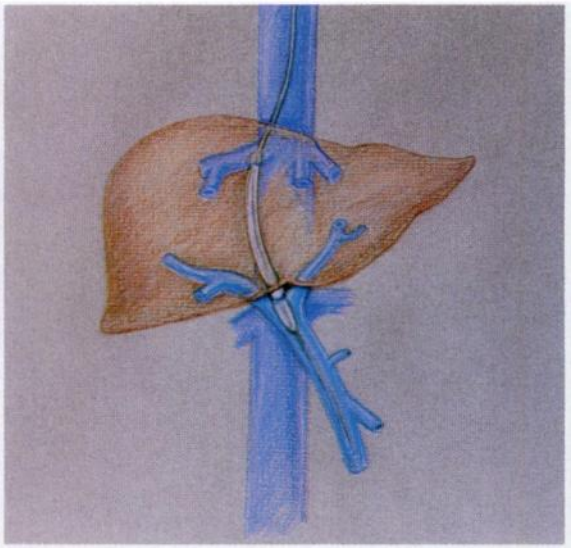

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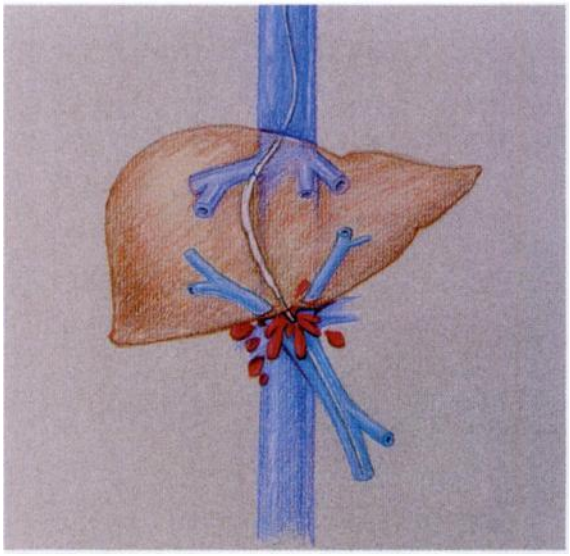


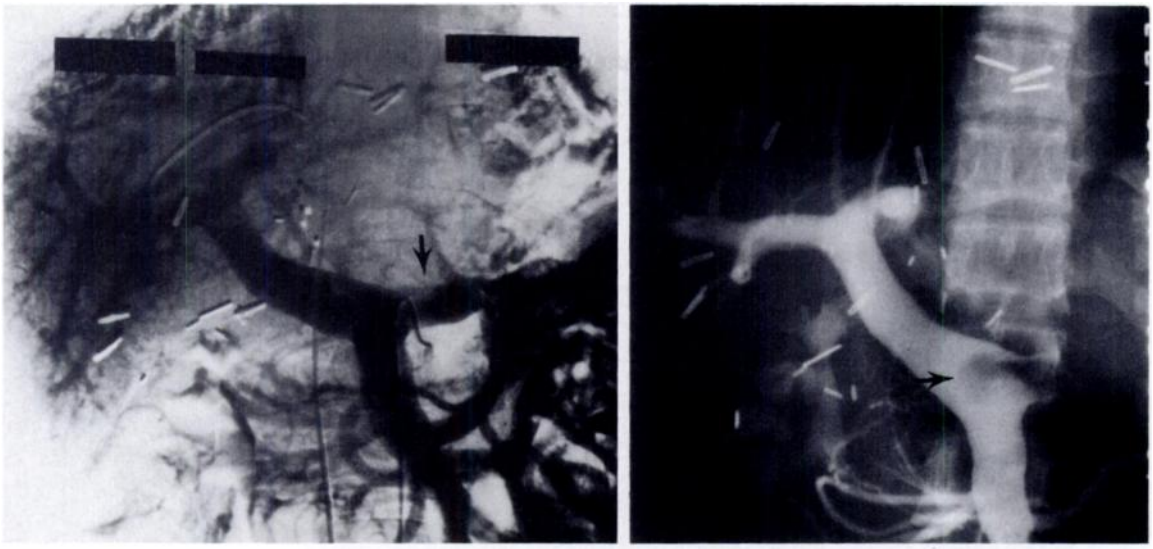
c.

Figure 9. Extrahepatic puncture of the portal vein. **(a)** Drawing illustrates the possible extrahepatic location of the main portal vein, in comparison to the intrahepatic location of the right and left branches of the portal vein. **(b)** The balloon dilates the extrahepatic puncture site of the main portal vein. **(c)** Massive hemoperitoneum results immediately following deflation of the balloon.

■ COMPLICATIONS RELATED TO CANNULATION AND DILATION OF THE PORTAL VEIN

The most serious and potentially fatal complication of TIPS is intraperitoneal hemorrhage resulting from rupture of the portal vein (50). Portal venous rupture results from extrahepatic puncture of the portal vein and subsequent balloon dilation. This may result in immediate massive hemoperitoneum and probable exsanguination (Fig 9).

The portal vein divides into right and left branches in the hilum of the liver. The left portal vein is constant, whereas the right is variable and may be short or even absent in 16% of patients, making puncture of the right portal vein much more difficult. Because it is imperative that the puncture of the portal vein be entirely intrahepatic, only cannulation of the right or left portal vein branches should be used to create the intrahepatic parenchymal tract. After the portal vein is cannulated, but before the parenchymal tract is dilated,



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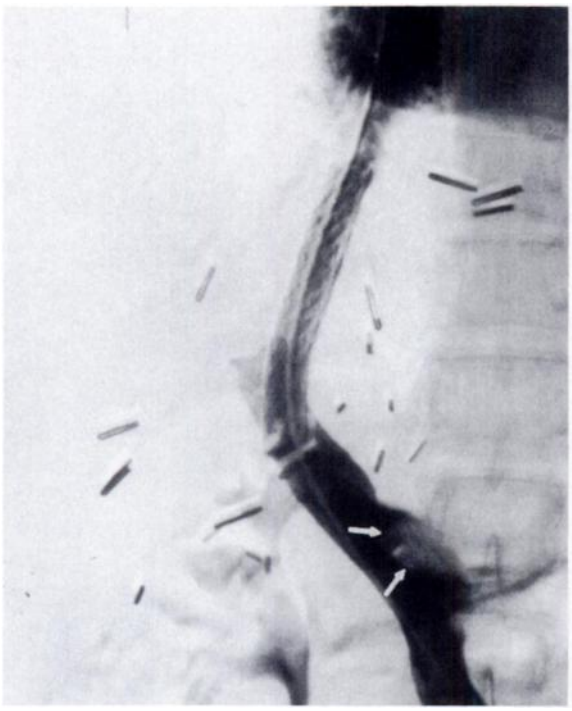


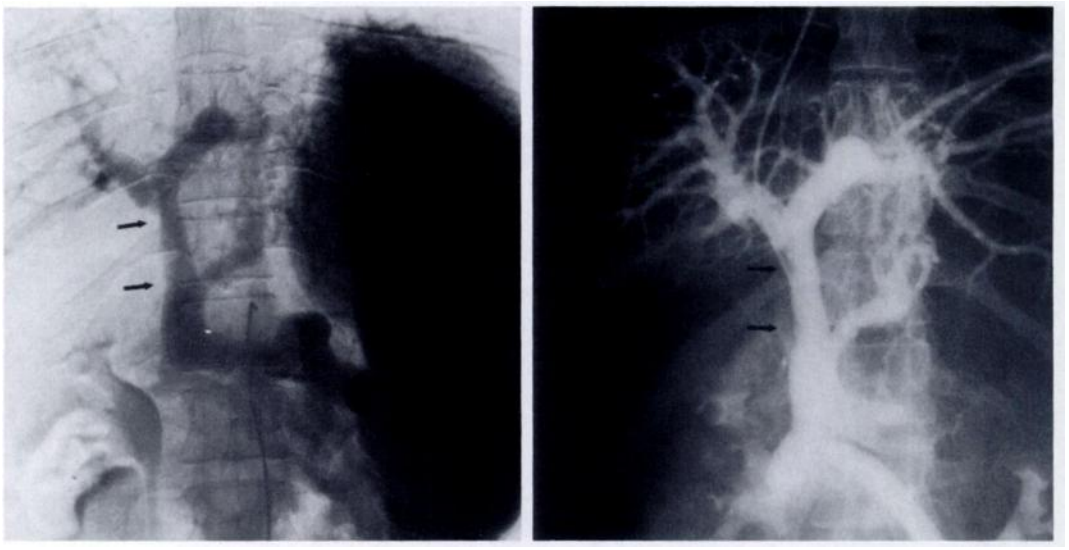
c.

Figure 10. Splenic venous thrombosis. **(a)** Mesenteric arteriogram obtained 2 weeks before the TIPS procedure shows a patent splenic vein (arrow). **(b)** Angiogram obtained during TIPS shows fresh thrombus (arrow) extending into the portal vein from the splenic vein. **(c)** Final post-TIPS subtraction portogram is normal, except for the presence of residual blood clot (arrows) in the splenic vein.

careful assessment of the entrance site into the portal vein is recommended. This may require imaging in oblique projections to confirm an adequate intrahepatic entrance site.

Portal venous rupture has been described previously, and in one instance was successfully managed by emergent placement of a Wallstent (Schneider, Minneapolis, Minn) to tamponade the intraperitoneal hemorrhage (Zemel G, oral communication, 1992).

Splenoportal venous thrombosis is another complication related to cannulation of the portal vein and is more likely to result from extended cannulation and manipulation. We have seen the prevalence of this complication decrease substantially, since less time is required to perform the procedure. Portal venous thrombus may also form before the procedure, as portal hypertension alone is a risk factor for splenoportal venous thrombosis. In one patient, for example, splenic venous thrombosis developed during the 2-week interval between mesenteric arteriography and the TIPS procedure (Fig 10). In another patient, organized mural thrombus in the por-



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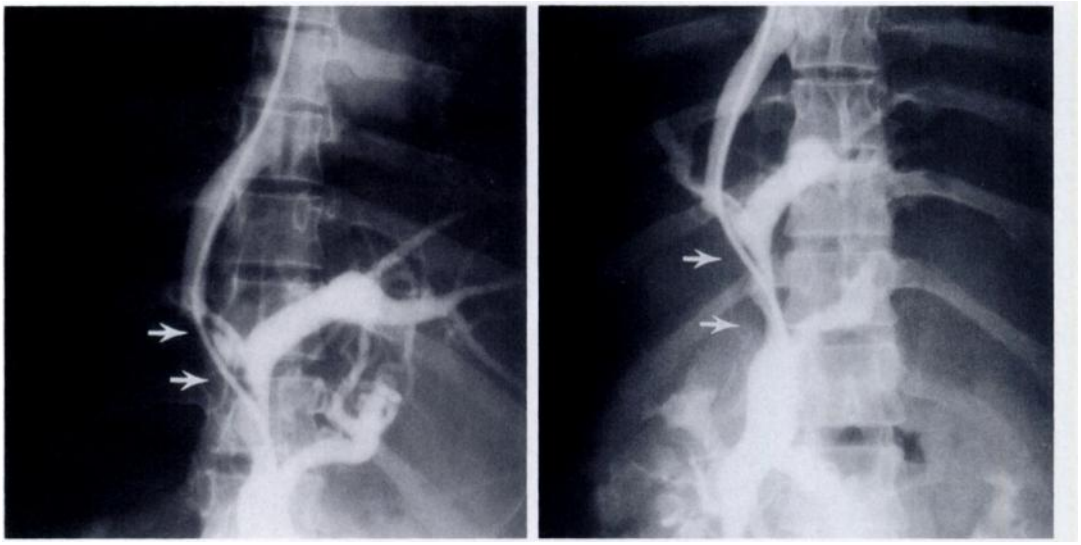

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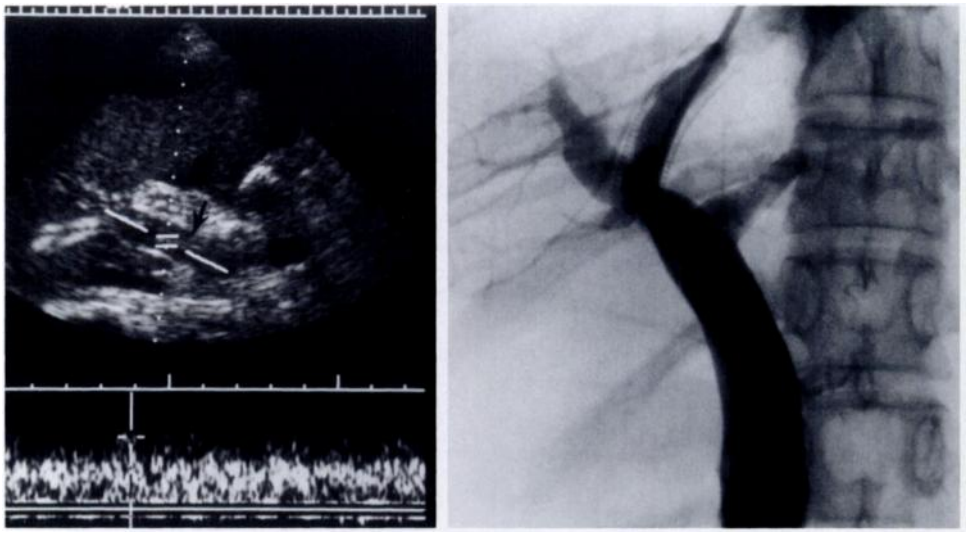


e. f.

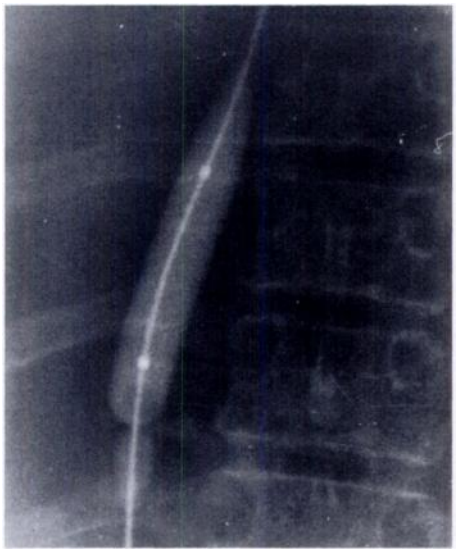
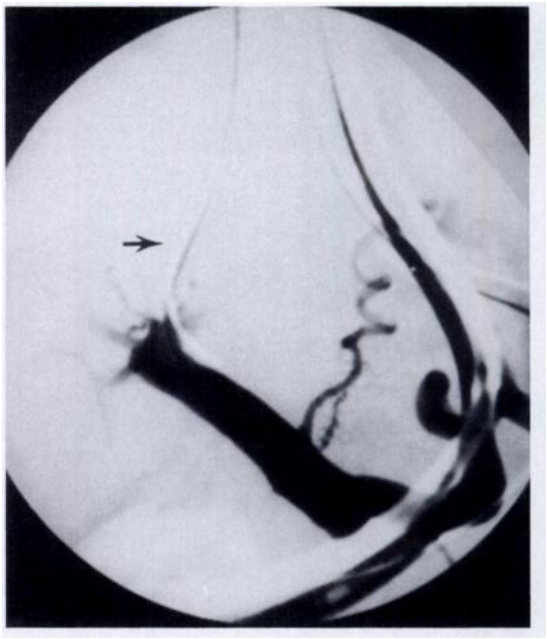


Figure 12. Balloon location. Portogram shows the angioplasty balloon, with its "waist" at the level of the portal vein. Only a small length of balloon projects into the portal vein to intentionally decrease trauma to the portal vein.

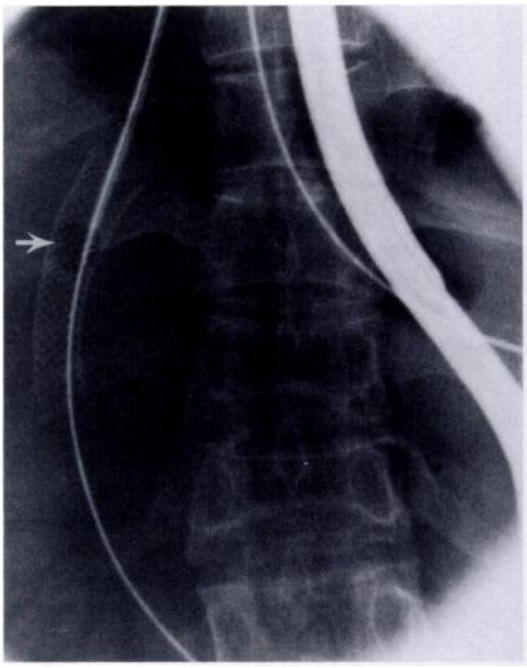
tal vein was not recognized at the pre-TIPS studies. During the course of the TIPS procedure, as a result of manipulation of the portal vein with catheters and guide wires, the thrombus propagated, resulting in substantial narrowing and decreased flow through the shunt (Fig 11).

Catheterization of the portal vein is not benign, and a minimum amount of catheter and guide wire manipulation is desirable. Furthermore, when balloon dilation of the parenchymal tract is performed, the shortest possible length of angioplasty balloon should be allowed to project into the portal vein (Fig 12).

◀ **Figure 11.** Portal venous thrombosis. **(a)** Subtraction superior mesenteric portogram obtained before TIPS reveals minimal irregularity of the main portal vein due to organized mural thrombus (arrows). **(b)** Direct portogram obtained immediately following catheterization of the portal vein shows mural portal venous thrombus (arrows). **(c)** Sequential portogram obtained immediately after stent placement reveals marked, increased clot formation (arrows). **(d)** After balloon dislodgment, there is partial reduction of the thrombus (arrows). **(e)** Follow-up duplex US scan reveals increased echogenicity of the adherent nonocclusive mural thrombus (arrow) within the portal vein, with preservation of hepatopedal flow. The patient was followed up with clinical and sonographic examinations, which revealed complete resolution of her esophageal varices. **(f)** Follow-up portogram obtained after 6 months shows excellent resolution of the thrombus. Note the presence of stent narrowing due to neointimal hyperplasia.



a.



b.

c.

Figure 13. Stent thrombosis. **(a)** Subtraction portogram obtained 2 days after TIPS reveals acute thrombosis of the stent (arrow). A duplex US scan (not shown) had revealed absence of flow in the stent. **(b)** Angiogram shows air (arrow) within an inflated occlusion balloon as it is withdrawn back through the stent to dislodge the thrombus. **(c)** Final subtraction portogram reveals good flow through the widely patent shunt.

■ COMPLICATIONS RELATED TO THE STENT

Acute thrombosis of the stent may occur during or shortly after the TIPS procedure. Early occlusion is often due to a technical factor, such as incomplete bridging of the parenchymal tract or prolonged cannulation of the stent after deployment. Stent thrombosis may also occur as a result of a high-grade stenosis of the hepatic vein.

Thrombosis of a stent can be successfully treated by means of balloon dislodgment with an occlusion balloon (Fig 13). When feasible, it is preferable to "bulldoze" the thrombus into the coronary vein, or a large varix, simul-

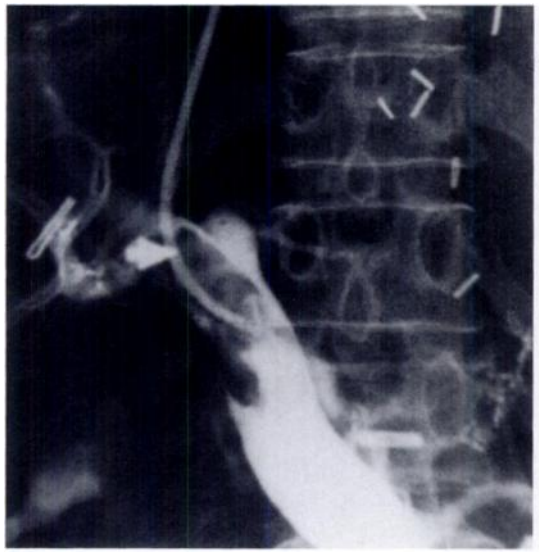
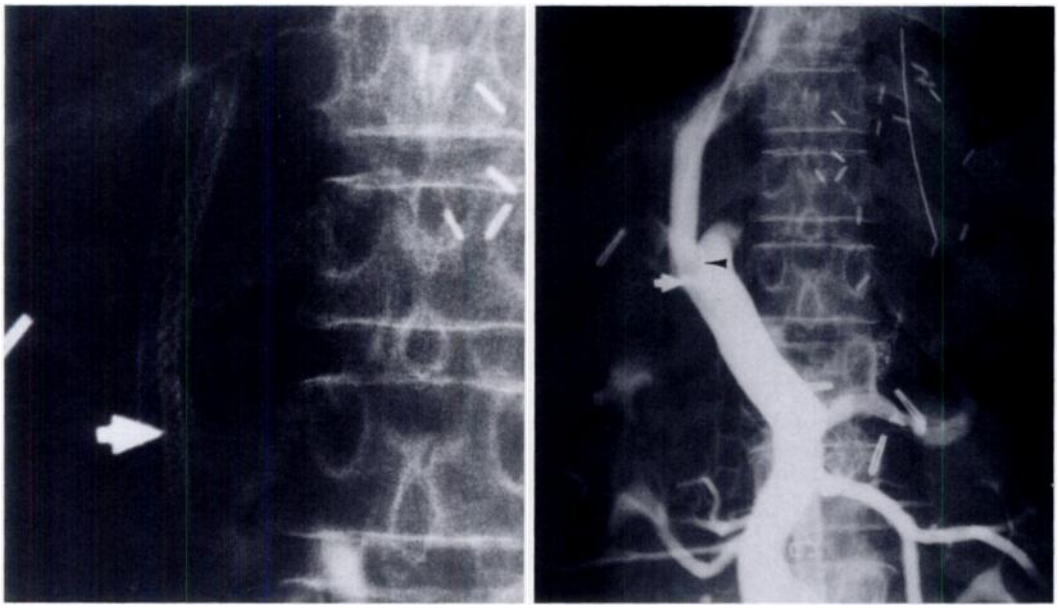


Figure 14. Suction embolectomy of portal venous thrombosis. **(a)** Angiogram obtained immediately following cannulation of the portal vein shows acute thrombus formation, which is related to the trauma associated with puncture and cannulation of the portal vein. The initial mesenteric portogram revealed no thrombus. **(b)** Angiogram shows the Colapinto guiding catheter in position before suction has been applied to aspirate the clot. **(c)** Final portal venogram reveals a widely patent shunt, with minimal residual nonocclusive thrombus (arrowhead). (See legend to Fig 2 for an explanation of the large radiopaque arrow in **a**–**c**.)

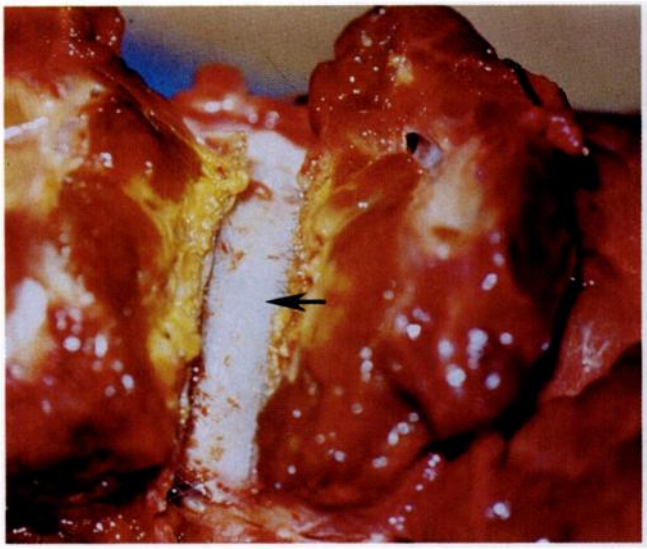
a.



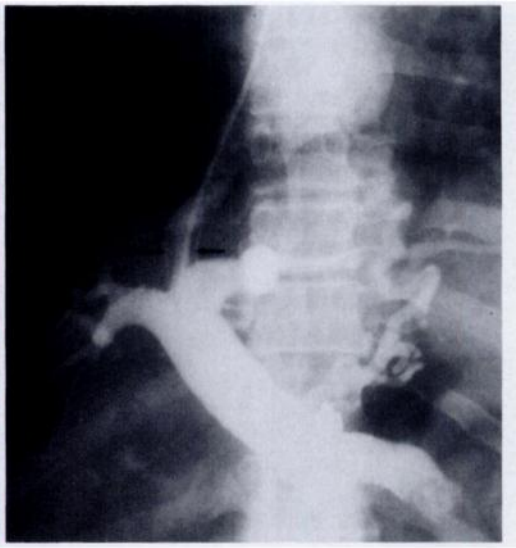
b. **c.**

taneously occluding the varices. In practice, this may be impossible. Alternatively, the balloon may be pulled through the stent; this maneuver, which will result in pulmonary embolization, is contraindicated in patients with right-to-left cardiac shunts, with severe lung disease, or in whom even a small pulmonary embolus would not be tolerated. Another way of dealing with acute thrombosis of either the portal vein or the stent is by suction embolectomy with a large end-hole catheter. The Colapinto (9-F) guiding catheter is well suited for this purpose. The tract is dilated with a balloon, and the Colapinto guiding catheter is then advanced directly into the clot. Suction is applied, and the catheter is carefully withdrawn from the patient. Because the stent is already deployed, the procedure is easily performed and results in quick, safe, and effective resolution of the thrombosis (Fig 14).

Delayed occlusion of the stent is more likely to occur as a result of pseudointimal proliferation or a technical factor such as incomplete coverage of the parenchymal tract with the stent, rather than due to thrombosis alone. There is likely to be some superimposed thrombosis as well. Thrombolysis and stent modification have been successfully performed; however, the procedures require extreme caution in patients with previous variceal hemorrhage (17).



15a.



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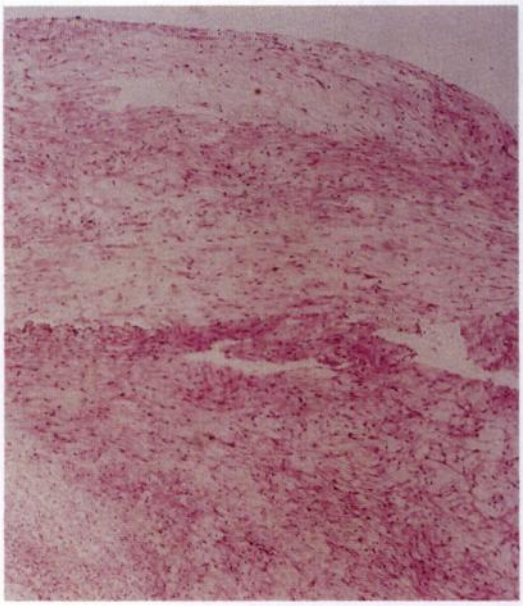

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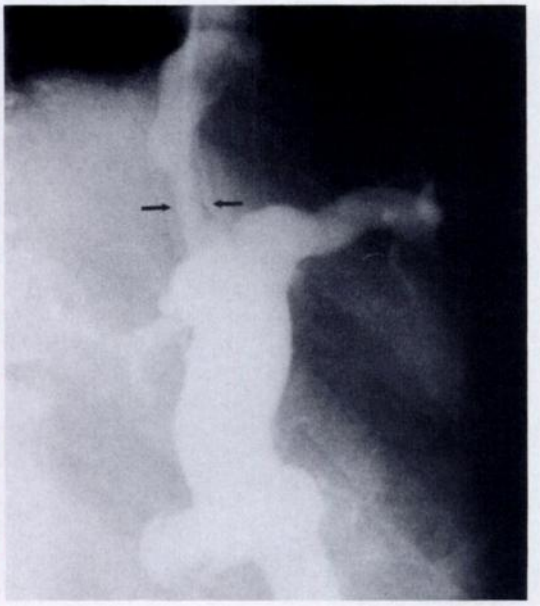
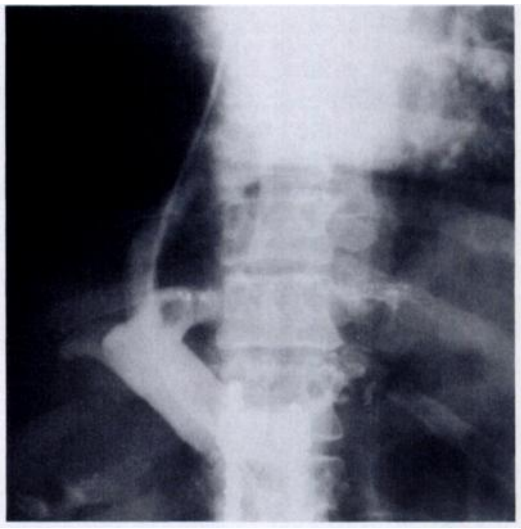
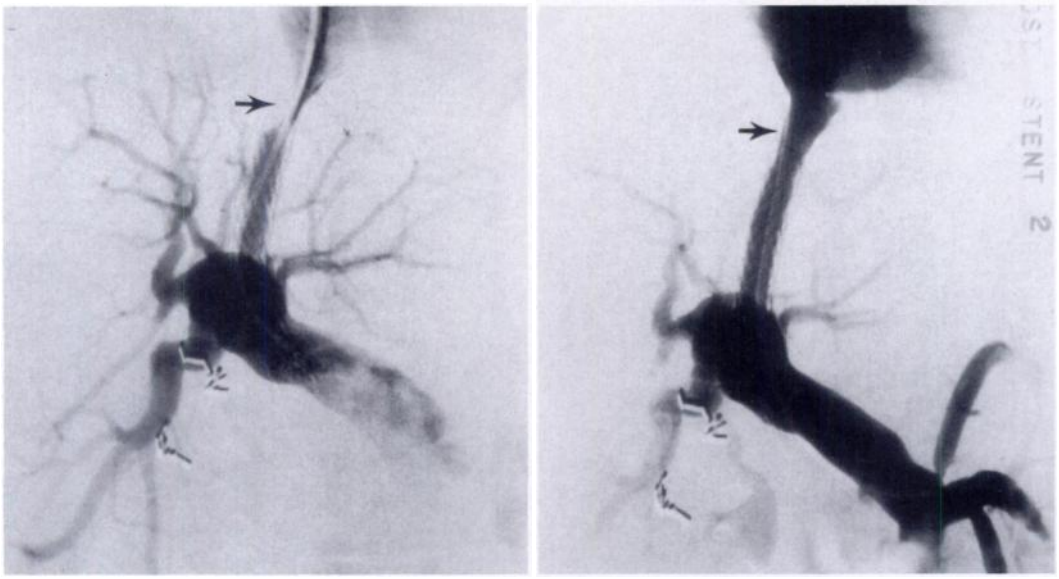


16b.



16c.

Figures 15, 16. Pseudointimal hyperplasia. **(15a)** Photograph of a gross specimen shows a pearly white coating lining the inside of a Wallstent (arrow). The lining represents collagenous material, also known as pseudointimal hyperplasia. **(15b)** Low-power photomicrograph (hematoxylin-eosin stain) shows collagenous material. **(16)** Anteroposterior **(a)** and lateral **(b)** radiographs reveal stent narrowing due to pseudointimal hyperplasia (arrows). **(c)** Radiograph obtained after balloon dilation demonstrates marked improvement in the lumen of the stent.



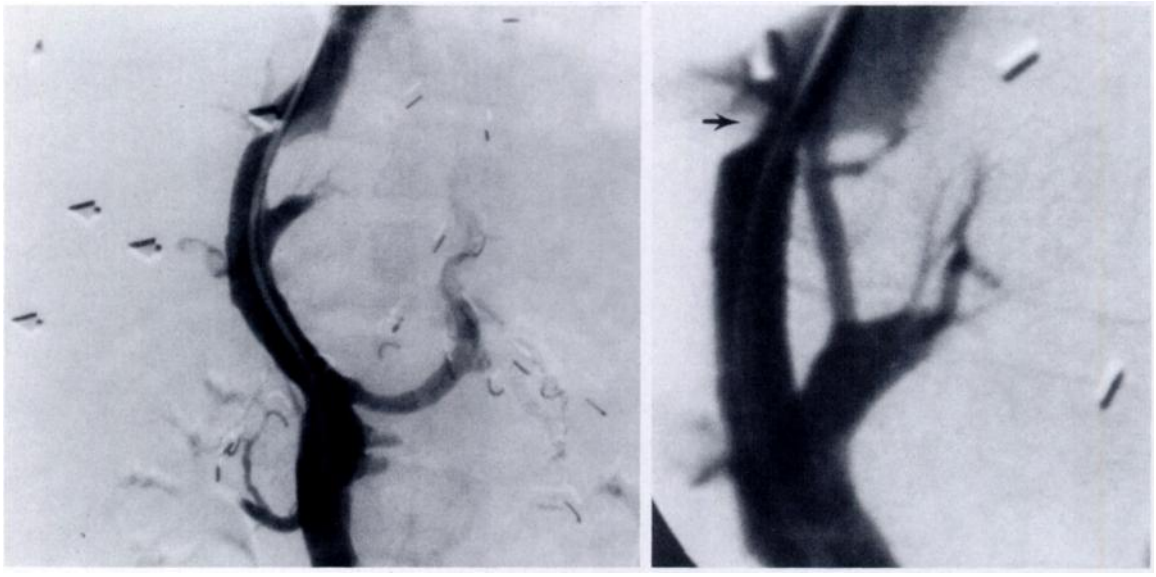
a. b.

Figure 17. Hepatic venous stenosis. **(a)** Follow-up subtraction portogram obtained 6 months after TIPS reveals that the hepatic venous stenosis is now more substantial (arrow). There was diminished flow through the stent and increased flow into the peripheral portal vein branches. Clinically, the patient's ascites had recurred. The hepatic vein was redilated with a new 42-mm Wallstent. **(b)** Subtraction portogram shows that flow through the shunt is substantially improved (arrow). The portosystemic pressure gradient decreased from 18 to 10 mm Hg, and the patient's ascites resolved completely.

Pseudointimal proliferation results in the growth of collagenous tissue between the stent and the endothelial surface of the stent lumen (Fig 15) and has been well documented (48). In one of our patients, delayed narrowing of the stent was seen 6 months following TIPS and was successfully treated with balloon dilation, resulting in hemodynamic improvement of the shunt and sustained regression of the varices (Fig 16).

Pseudointimal proliferation results in shunt stenosis involving either the stent or the hepatic vein (48). This probably occurs to some extent in all patients following TIPS and is hemodynamically significant only when the stenosis exceeds 50% narrowing. A review of our experience and that of others suggests that narrowing of the stent or hepatic vein occurs in approximately 55% of patients with the Wallstent and over 70% with the Gianturco Z-stent (18). Because the experience with TIPS is still early, it is not unreasonable to expect chronic narrowing of the stent resulting in shunt failure to occur in 70% or more of patients. It is this problem that will undoubtedly be the greatest challenge to the long-term patency of TIPS and is likely to result in recurrent variceal bleeding. For this reason, we recommend close follow-up of patients after TIPS with serial duplex US examinations and direct portal venography with pressure measurements every 6–12 months.

Stenosis of the hepatic vein after TIPS has been related to utilization of a small hepatic vein (19). Initial selection of the largest hepatic vein is the best guarantee against this problem, and it behooves the operator to carefully evaluate all of the hepatic veins before the transhepatic puncture. Placement of stents all the way to the inferior vena cava is another way to avoid hepatic venous stenosis following TIPS (Fig 17). However, this may result in other problems (discussion to follow).



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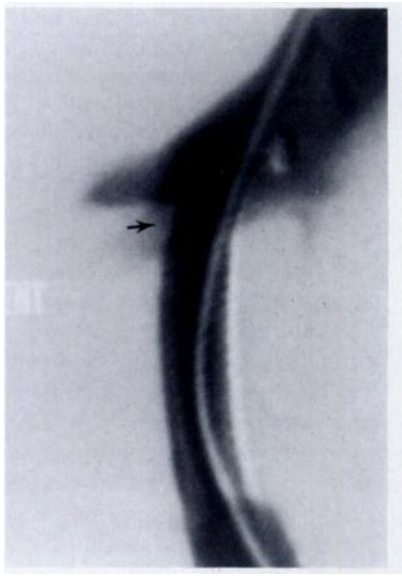
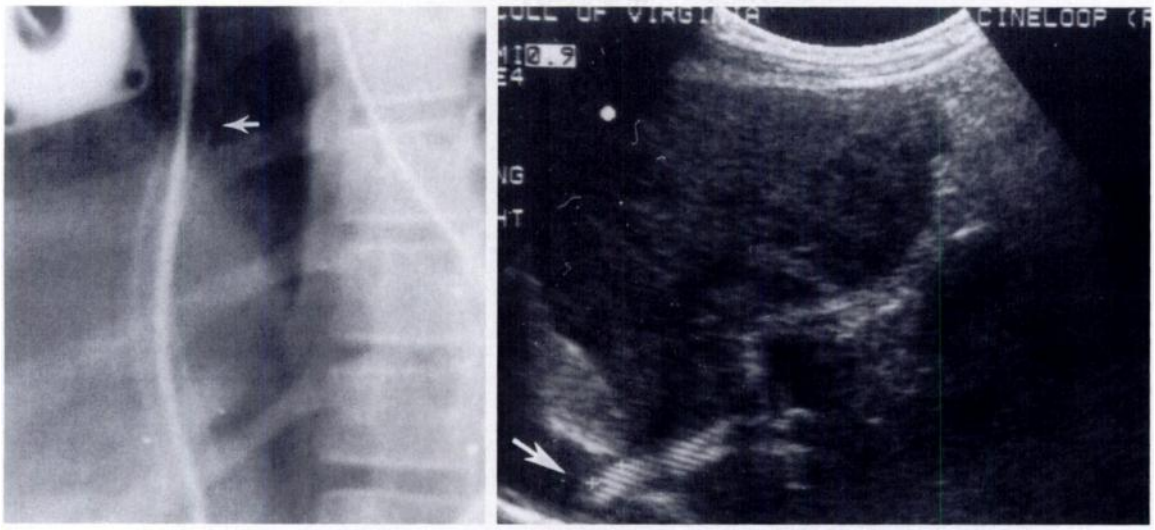


c.

Figure 18. Stent shortening. **(a)** Anteroposterior subtraction portogram fails to demonstrate incomplete coverage of the transhepatic tract by a Wallstent. (See legend to Fig 2 for an explanation of the large radiopaque arrows.) **(b)** Right anterior oblique projection demonstrates incomplete bridging of the hepatic parenchyma by the stent, as seen by a space between the proximal portion of the stent and the hepatic vein (arrow). **(c)** Another right anterior oblique view obtained after deployment of a second Wallstent shows it completely covering the parenchymal tract (arrow).

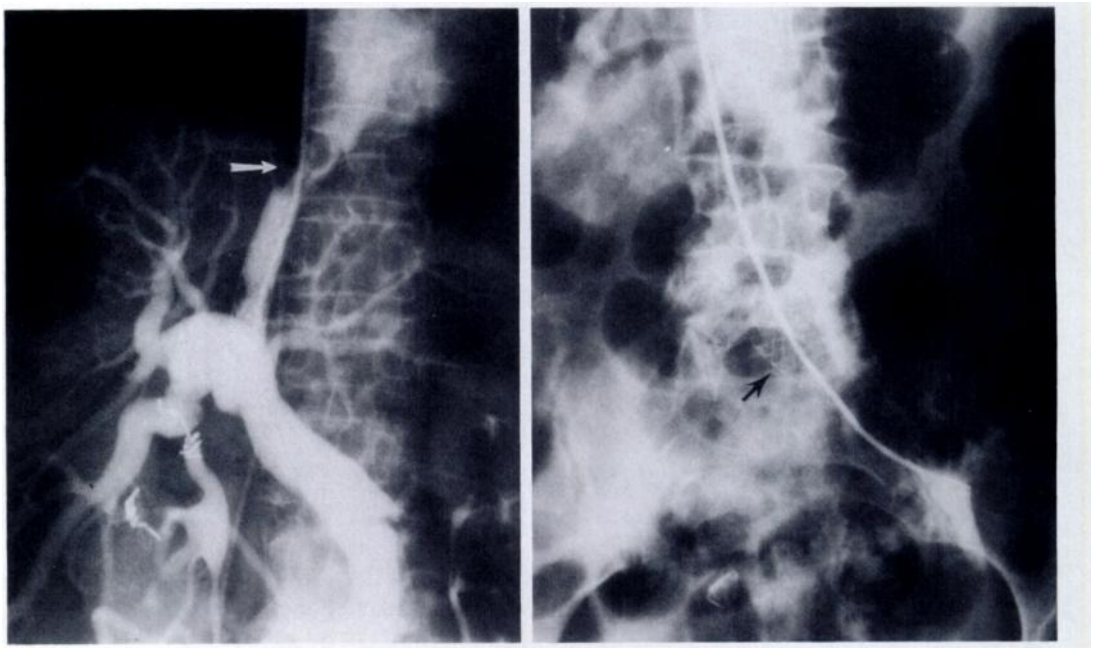


a. b.

Figure 19. Excessively long stent. **(a)** Angiogram shows a Wallstent extending well into the inferior vena cava (arrow). **(b)** Sonogram reveals the stent projecting into the inferior vena cava (arrow).

One potential problem with the Wallstent is persistent shortening of the stent so that it no longer completely bridges the parenchymal tract (51). This may occur if there is insufficient protrusion of the Wallstent into the portal or hepatic veins and if the stent is initially expanded to less than its maximum diameter of 10 mm. As the stent fully springs back to its intrinsic diameter of 10 mm, it may shorten completely out of the portal or hepatic vein, resulting in shunt occlusion. For this reason, deployment of the Wallstent should allow for at least several millimeters protrusion into the portal and hepatic veins. Subtraction portography, performed with a right anterior oblique projection, can be very useful for confirming that the transhepatic tract is completely covered by the stent (Fig 18).

The opposite of excessive stent shortening may occur. A stent may project too far into either the portal vein or the hepatic vein and inferior vena cava (Fig 19). Although shunt function is not altered, the excess length of the stent may result in clot formation and subsequent difficulty in cannulation of the stent. In addition, the exposed wires may constitute a hazard to the surgeon's hand, as well as interfere with subsequent liver transplantation. Despite these drawbacks, there is an important advantage in having the stent extend all the way to the inferior vena cava: Hepatic venous stenosis is prevented. Furthermore, some surgeons do not find that stent extension into the inferior vena cava creates much of a problem. As more experience is accumulated, answers to these technical questions are likely to be found.



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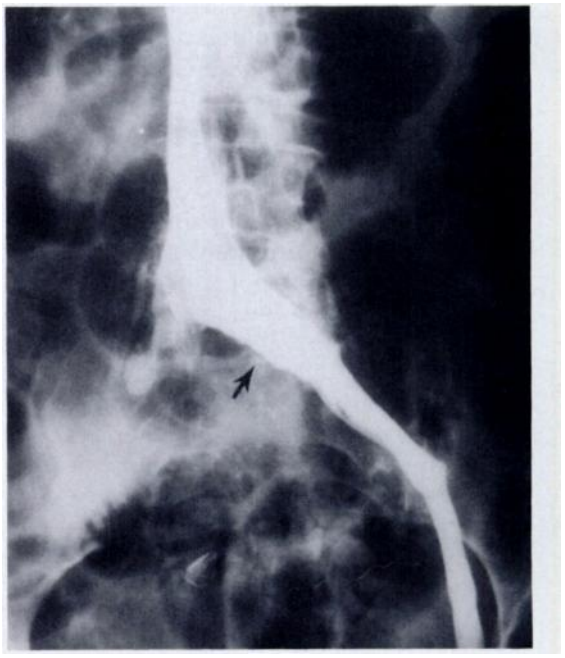
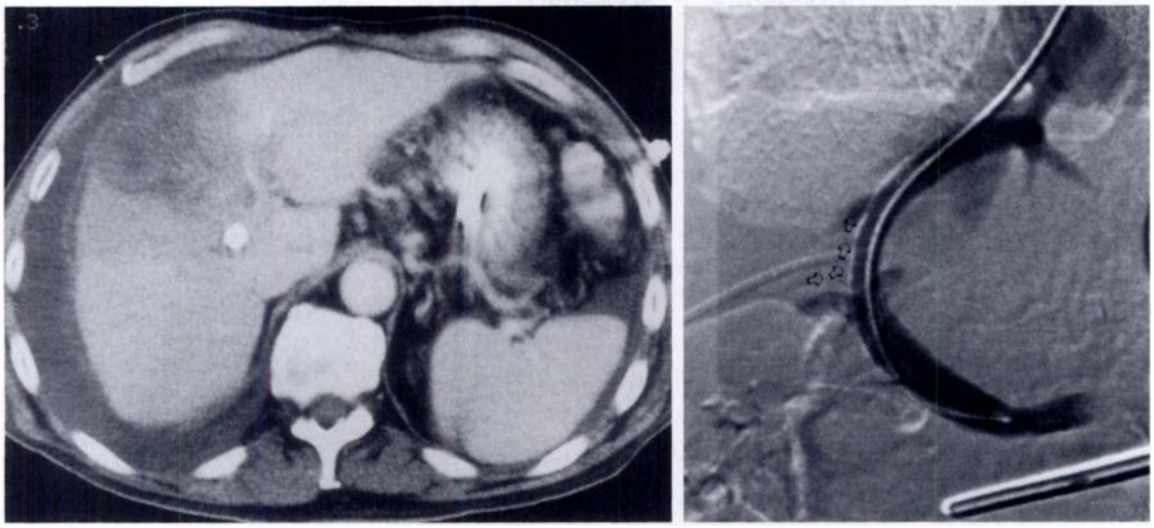


c.

Figure 20. Stent migration. **(a)** Angiogram obtained during the initial TIPS procedure in the same patient as in Figure 17 shows narrowing of the hepatic vein at its orifice with the inferior vena cava (arrow). Attempts to place an additional Palmaz stent resulted in migration of the stent. An alternative deployment location was required. The stent was tethered onto the inflated balloon catheter and guided into the common iliac vein, where it was deployed and further dilated to 15 mm. **(b)** Scout image shows the new location of the dislodged Palmaz stent (arrow). **(c)** Venogram obtained 6 months later shows that the left iliac vein is widely patent. Arrow = the stent.

Stents may undergo separation, dislodgment, or migration (5). In one of our patients during the TIPS procedure, two Palmaz stents separated just as the patient took a deep breath, while an additional stent was being dilated from 8 to 10 mm. When the balloon was deflated, the stent migrated proximally onto the shaft of the balloon catheter (Fig 20).

Dislodgment or migration of the Wallstent should occur less frequently than with the Palmaz stent because the former is longer and exerts self-retaining radial forces. Still, we have seen an instance of separation of Wallstents in the portal vein, and migration of a Wallstent in the biliary system has been described (52). Adequate overlap between two stents is necessary, and balloon catheters,



a. b.

Figure 22. Compression of the hepatic artery. **(a)** Computed tomographic (CT) scan shows a low-attenuation, wedge-shaped region in the right hepatic lobe. It is believed to represent an infarction. **(b)** Subtraction portogram demonstrates excellent flow through the shunt, which results in a "steal" of blood flow from the right portal vein (tandem arrows).

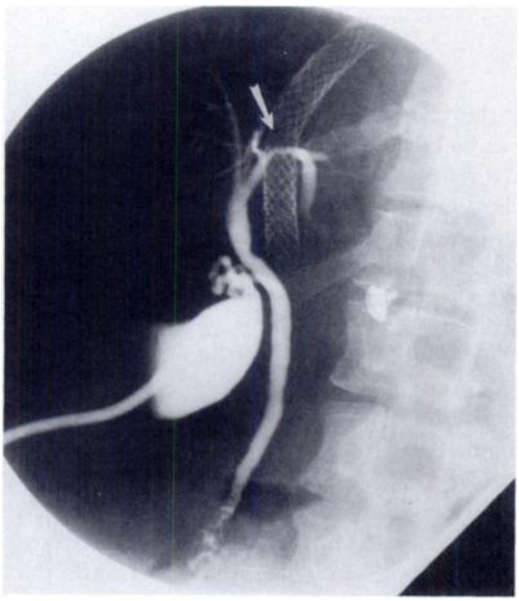


Figure 21. Compression of a bile duct. Cholecystogram obtained 2 months after TIPS shows subtle compression of the left biliary radical by the stent (arrow).

guide wires, etc, should be withdrawn very carefully when working with stents. Salvage of a shunt, when stent separation occurs, has also been reported and can be performed by deployment of another stent to "bridge the gap" (16).

As the Wallstent expands to its full diameter, adjacent arteries, veins, and bile ducts may be compressed. Histologic studies have shown that the stents exert a minimum of compression on the hepatic artery and bile ducts (48). One of our patients, who developed cholecystitis after TIPS, proved to have slight compression of the left bile duct (Fig 21) but otherwise had no proved adverse sequelae from this compression (long-term follow-up was not possible, however, because the patient underwent liver transplantation soon after cholangiography). In another case, compression of the hepatic artery by the Wallstent was blamed for interruption of the hepatic arterial blood supply to the liver, resulting in a focal hepatic infarction (Fig 22a). Fortunately, ischemia of the liver is rare due to the dual blood supply of the hepatic artery and portal vein. For the preceding undocumented case, we theorized that reversal of right portal vein blood flow resulted from excellent shunt function and prevented compensation for the reduction in arterial perfusion (Fig 22b). Additional examples of compression of vascular structures by the stent will likely be recognized as experience with TIPS increases.

We have previously reported a case in which intravascular hemolysis and secondary hyperbilirubinemia developed after an otherwise unremarkable TIPS procedure (Fig 23) (35). A peripheral blood smear revealed damaged red blood cells. One reason for the hemolysis may be due to excessive wear and tear on blood cells caused by projection of stent wires into the portal or hepatic vein or by flow turbulence along the sides of the stent. We have seen this complication only once. Perhaps by minimizing the projection of the stent into the portal or hepatic vein or by optimizing the angle of the stent with the portal vein wall, the amount of blood flow through the stent interstices could be reduced, thereby preventing this complication. Deploying the stent so that it points directly into the main portal vein, rather than at an angle, may improve the flow dynamics of the shunt.

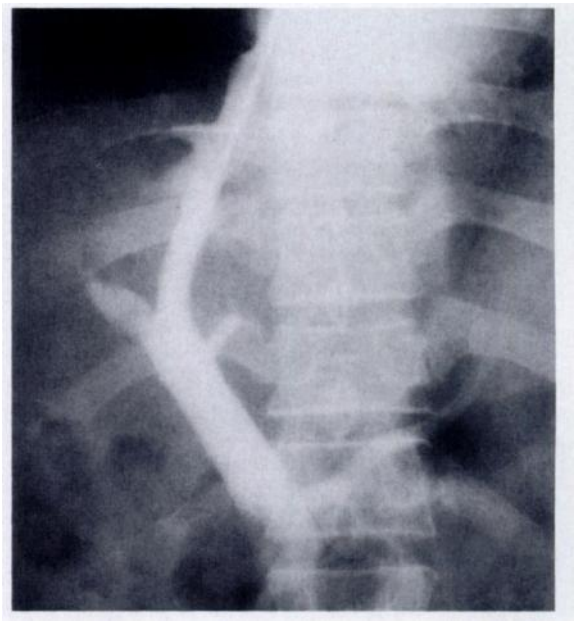


Figure 23. Intravascular hemolysis. Completion portogram reveals excellent shunting through the stent. The stent is well positioned and does not extend too far into either the portal or hepatic vein. Despite this, hemolysis occurred.

■ COMPLICATIONS RELATED TO THE PUNCTURE SITE

As with any invasive vascular procedure, there is always a chance of bleeding or hematoma at the access site. Hematomas or bleeding at the venous puncture site are rare, despite placement of 9- or 10-F sheaths in patients who often have coagulopathy and thrombocytopenia. We and others have seen limited hematomas at the internal jugular venous puncture site (1,6). None required transfusion or surgical repair. Other complications, including arteriovenous fistulas or pseudoaneurysms, may occur at the jugular venous or common femoral arterial puncture (from mesenteric arterial portography) sites.

Various structures in the neck, in particular the carotid artery, are subject to injury as a result of proximity to the internal jugular vein. We have inadvertently punctured the carotid artery on two occasions. For this reason, some operators prefer an anterior approach to the internal jugular vein, enabling palpation of the carotid artery. The thoracic duct may also be injured if a left-sided jugular venous puncture is performed. In a 14-year-old patient in our series, inadvertent puncture of the trachea occurred during an internal jugular venous puncture, resulting in transient bleeding from the endotracheal tube. Fortunately, there were no adverse sequelae.

Several things may help increase the safety of the puncture. To decrease the number of attempts required for jugular venous puncture, we perform a quick duplex US examination of the right side of the neck to identify the internal jugular vein. Underlying coagulation and platelet abnormalities should be corrected as much as possible. Use of a micropuncture system with a 21-gauge needle and 0.018-inch guide wire (Cook) will also increase the safety of the puncture, particularly in patients with short necks. We have successfully used both internal jugular veins and the right external jugular vein for the procedure. We prefer to use the external jugular vein whenever possible, since there is increased safety and ease of puncture if a 21-gauge system is used. The course of the right external jugular and subclavian veins is not as straight as that of the internal jugular vein. However, once the sheath is inserted, these veins will straighten sufficiently to enable performance of the TIPS procedure.

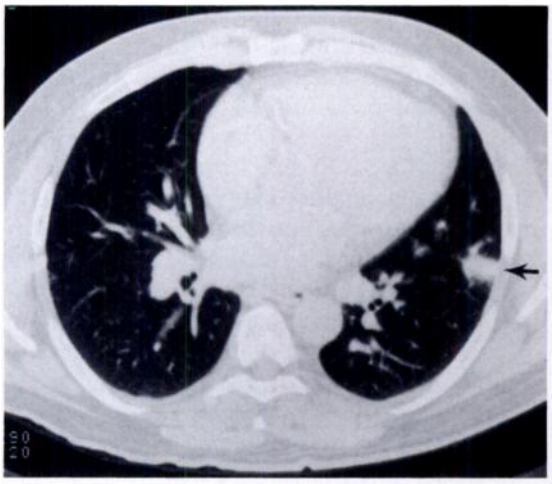


Figure 24. Septic pulmonary emboli. CT scan demonstrates an ill-defined area of high attenuation in the periphery, which is believed to represent a septic embolus (arrow).

■ COMPLICATIONS RELATED TO PORTOSYSTEMIC SHUNTING

Physiologically, TIPS most closely resembles a "side-to-side" portacaval shunt (8). Theoretically, this permits some portal blood flow into the liver, thereby reducing hepatic encephalopathy. Factors that have been noted to result in an increase in encephalopathy include increased stent diameter, patient age greater than 62 years, and advanced liver disease.

Hepatic encephalopathy has been seen in 5%–35% of patients following TIPS (1–34,36). The German group reported a 2.2%–5% de novo rate and 17% overall prevalence of hepatic encephalopathy following TIPS (17,32). This compares favorably with the varying prevalence of 5%–67% seen after surgical shunting (38). In our series, post-TIPS encephalopathy was seen in up to 25% of patients, most of whom had a history of hepatic encephalopathy prior to TIPS (36). In patients without preexistent hepatic encephalopathy, less than 10% developed it following TIPS. Furthermore, the condition was easily treated with diet and lactulose alone in all except one patient, who underwent urgent liver transplantation. The varying prevalence of post-TIPS encephalopathy may be due to differences in the clinical assessment and Child classification of patients among institutions.

The most reliable parameters for the measurement of hepatic encephalopathy include mental status, measurement of asterixis grade, and the trail making test. This test, also known as the "number connection test," is an objective way to monitor encephalopathy and can be administered serially to patients before and after TIPS. Magnetic resonance spectroscopy may also be useful in detecting subclinical encephalopathy (53). Careful assessment of the presence of encephalopathy is essential and should be based on clinical grounds and the patient's history.

We have encountered post-TIPS fever ($< 102°F$ [38.9°C] in all cases) in up to 10% of patients. One of these patients was febrile before the initial TIPS procedure; *Staphylococcus aureus* was cultured from samples of the patient's blood and taken from the central venous catheter tip. Although ideally patients should be afebrile before TIPS, an emergent TIPS was performed in this patient because of uncontrolled variceal bleeding. The patient has done well following treatment with intravenous antibiotics. In another patient, shaking chills and fevers to 104°F (40°C) occurred within a few days after balloon dilation of the original stent and placement of a second stent. Five sets of blood cultures were positive for *Klebsiella* organisms. A CT scan (Fig 24) obtained 2 weeks after the TIPS revision demonstrated septic pulmonary emboli. The patient's fever responded well to a 1-month course of intravenous antibiotics.

The fevers in our other patients after TIPS have been self-limited, lasting 3–5 days. The cause may be due to sudden "seeding" of bacteria or pyrogens from the mesenteric portal circulation into the systemic blood stream or from a reaction of adjacent liver tissue to the metallic stent. Other possible causes include reabsorption of "factors" contained in ascites or low-grade infections elsewhere in the body.

All of our patients routinely undergo broad-spectrum antibiotic prophylaxis with cephalosporins at the time of TIPS. We do not, however, routinely prepare the bowel with cathartics or antibiotics. To date, there have been no reports of stent infection after TIPS.

It is well documented that liver function deteriorates after portal flow diversion from surgical portosystemic shunts (38,54). However, to date, there have been few reports of substantial liver compromise following TIPS (1,3,17,22,28,55). All of our patients had some hepatic impairment 6 months after TIPS, and those patients with the most severe hepatic compromise before TIPS (Child class C)

were most affected. It may be possible that by sparing blood flow to the liver, minimal hepatic compromise results. Further long-term evaluation is needed. In patients with rapid deterioration of liver function after TIPS, it is reasonable to study the hepatic arterial supply to exclude a stenosis of either the celiac or hepatic arteries. Diminished hepatic arterial supply may prevent arterial flow compensation to the liver when portal blood flow has been diverted into the shunt. Angioplasty or surgical bypass could be performed in cases of a substantial stenosis.

Intraprocedural myocardial infarction has been described by several investigators and has occurred in less than 2% of patients (1,22). One may argue that these debilitated and acutely ill patients would likely have suffered a similar result during surgical shunting procedures requiring general anesthesia. A few investigators have noted that coagulopathy may worsen following TIPS (3,22). In two patients, uncontrollable coagulopathy resulted and led to death (22). Aggravated coagulopathy may be secondary to rapid reabsorption of fibrinolytic substances from ascites. Other factors may contribute to increased bleeding after TIPS, namely, decreased hepatic synthetic function or destruction of platelets by the stent (35).

■ COMPLICATIONS RELATED TO CONTRAST MATERIAL

We have not encountered any cases of contrast material–related allergies. TIPS may be performed in patients with a contrast material allergy as long as the indications are carefully reviewed. Use of steroid prophylaxis and nonionic contrast material is highly recommended.

Scant reports of renal failure associated with use of contrast material have been described (1). In our experience, despite occasional administration of over 250 mL of nonionic contrast material to several patients, nephrotoxicity is uncommon. In patients with end-stage liver disease, it may be difficult to differentiate between nephrotoxicity from contrast material and hepatorenal syndrome as a cause of renal failure. Interestingly, early reports suggested that TIPS may reverse hepatorenal syndrome. LaBerge et al (1) reported a 1-mg/dL (88.4-μmol/L) decrease in the level of serum creatinine in a patient treated with TIPS for hepatorenal syndrome. As in most angiographic studies, our patients are routinely well hydrated before and during the procedure. Routine bladder catheterization is also performed to monitor fluid status during and after the procedure.

■ CONCLUSION

The wide discrepancy in complication rates among institutions may be attributable to differences in reporting of complications, patient selection, and management of patients in the intensive care unit rather than the operator. Although most of the complications described are serious, most are not life-threatening. Potentially the most dangerous technical aspect of the procedure is the transhepatic puncture of the portal vein. Next, the most important thing to watch for is that the entrance site of the portal vein is entirely intrahepatic. Optimization of coagulation parameters increases the safety of the procedure. Working quickly and efficiently also enables the procedure to be performed in a few hours or less, thereby lessening the risk of splenoportal venous thrombosis. Although our list of complications is extensive, it is in no way inclusive. Additional complications will undoubtedly be described.

We remain optimistic that TIPS will occupy a prominent place in the treatment of variceal hemorrhage, ascites, and other complications of portal hypertension. The success rate of TIPS in the treatment of acute variceal hemorrhage and the relatively low invasiveness of the procedure speak for themselves. The role to which TIPS is relegated over the long term will be influenced by the long-term clinical success rate in the prevention of recurrent variceal hemorrhage. This clinical success rate in turn may be limited by neointimal hyperplasia–induced narrowing of the stent and hepatic vein. As elsewhere in the vascular system, solutions to this ubiquitous problem are needed.

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