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Surgical Management in Portal Hypertension

Hiroshi Yoshida, Yasuhiro Mamada,
Nobuhiko Taniai, Takashi Tajiri and Eiji Uchida

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1. Introduction

Bleeding from esophagogastricvarices is a catastrophic complication of chronic liver disease. There are various treatments for esophagogastricvarices, such as endoscopic treatment, interventional radiology, and surgical procedure [1-3]. Recently, "General Rules for Recording Endoscopic Findings of EsophagogastricVarices [4]" were established and endoscopic treatment further improved survival rates [5].

Many years ago, operation was the only treatment available. A number of surgical procedures have been developed to manage esophagogastricvarices [6]. Broadly, these can be classified as shunting and nonshunting procedures.

We showed the surgical procedures for the treatment of esophagogastricvarices.

2. Operation technique

2.1. Shunting procedures

There are various shunting procedures for the treatment of esophagogastricvarices [7-25]. There are two types of shunting procedures, nonselective shunt and selective shunt. Nonselective shunts, such as portacaval or mesocaval shunts, reduce portal venous pressure and improve esophagogastricvarices. While nonselective shunt is associated with a high risk of hepatic encephalopathy secondary to the hyperammonemia that is caused by impaired protein metabolism in the liver [26-28].

Selective shunts, such as distal splenorenal shunt (DSRS) or left gastric venous caval shunt (Inokuchi shunt), maintain portal pressure and selectively reduce esophagogastricvariceal

pressure. Shunt surgery is the best procedure in terms of preventing recurrent bleeding [20-22], but carries a high risk of postoperative encephalopathy, especially after nonselective shunt [26-28]. Even in selective shunt, loss of shunt selectivity occurs occasionally, leading to postoperative encephalopathy [8, 14].

2.1.1. Nonselective shunt

2.1.1.1. Portacaval and mesocaval shunt

The mesocaval shunt was initially used to control bleeding from esophageal varices in children with congenital abnormalities of the hepatobiliary system. The procedure consisted of transposition of the divided inferior vena cava and the divided superior mesenteric vein, hence its name, mesocaval shunt. This operation was modified, and some reports have described a portacaval or mesocaval interposition shunt with a graft (H-graft mesocaval shunt) [9, 10, 29-33]. Millikan et al. [26] have reported that the incidence of hyperammonemia after nonselective shunt procedures was as high as 75%.

2.1.2. Selective shunt

2.1.2.1. Left gastric venous caval shunt (Inokuchi shunt)

To assure postoperative portal perfusion and to prevent Eck's syndrome, in 1967 Inokuchi designed a selective shunt, called the left gastric venous caval shunt [7, 19, 23-25].

After dilatation, and engorgement of the left gastric vein is confirmed by splenoportography, the gastrohepatic ligament is opened and the left gastric vein is identified, and dissected 2 cm towards its junction with the portal system or splenic vein. The vein dissection must be done carefully to avoid hemorrhage, since the wall of the left gastric vein is weak due to increased portal vein pressure. The anastomosis is then performed between the distal end of the transected left gastric vein and the inferior vena cava. The autograft, the great saphenous vein, is anastomosed to the inferior vena cava in an end-to-side fashion, and opposite end is pulled through the suprapancreatic space. After the anastomosis is completed, a splenectomy is done. If splenectomy is not indicated, short gastric vein ligation is necessary in order to decrease the collateral circulation from the greater curvature of the stomach. The selection of a caval anastomosis procedure depends upon anatomical individuality or operative difficulty or both. The left gastric venous-caval shunt can be modified in three ways, left gastric-spermatic (ovarian) shunt, left gastric-adrenal shunt, or left gastric-renal shunt.

Postoperative mean portal pressure was 335 mm of water, and although it is decreased when compared to 363 mm water at laparotomy, this may be the result of splenectomy. On the other hand, left gastric venous pressure decreased from 316 mm of water to 211 mm of water postoperatively [7].

2.1.2.2. DSRS

Original DSRS: The DSRS is a selective shunt that was developed by Warren (original DSRS) in 1967 [12] to preserve portal blood flow through the liver while lowering variceal pressure. The hope was that both bleeding and hyperammonemia would be prevented. DSRS effectively prevents rebleeding, but still carries a risk of hyperammonemia [14].

The procedure for DSRS consists of anastomosis of the distal end of the splenic vein to the left renal vein, and devascularization of left gastric artery and vein. The specific objectives of DSRS as stated in the original publication [12] were : 1) selective reduction of pressure and volume of flow through gastroesophageal veins; 2) maintaining portal venous perfusion of the liver; and 3) maintaining continual venous hypertension in the intestinal bed. These three objectives formed a basis for much subsequent work.

Henderson et al. [34] compared hemodynamics between alcoholic and nonalcoholic cirrhotic patients after DSRS. Portal perfusion and liver blood flow are maintained, both quantitatively and qualitatively, in nonalcoholic patients with cirrhosis, resulting in better hepatocyte function and improved survival.

Stenosis of a DSRS shunt may lead to inadequate variceal decompression, accompanied by a risk of rebleeding. Henderson et al. [35] reported that the patients with stenosis of a DSRS were successfully managed by balloon dilation. All of the shunts were patent, but showed a mean pressure gradient of 15 millimeters of mercury, which was reduced to a mean of 7 millimeters of mercury by dilation. Although, repeat angiography should be performed in patients with rebleeding or reappearance of varices after DSRS to determine the cause.

DSRS + splenopancreatic disconnection (SPD): Belghiti et al. [8] reported loss of shunt selectivity during long-term follow-up in patients who underwent original DSRS, confirmed via the pancreatic vein. Warren et al. [36] subsequently improved the DSRS procedure by adding SPD, i.e., skeletonization of the splenic vein from the pancreas to its bifurcation at the splenic hilum. The operation technique is as follows: The pancreas is approached through the lesser sac, with the additional takedown of the splenic flexure to improve access to the retropancreatic plane. The whole pancreas is mobilized along its inferior border from the superior mesenteric vein to the splenic hilum. The pancreatic perforating veins are ligated as they enter the splenic vein. It is imperative to sufficiently dissect the splenic vein from the pancreas and to carefully manipulate the junction between the splenic and superior mesenteric vein to ensure that skeletonization proceeds to the renal vein without kinking. The key to the entire procedure lies in accurate identification and ligation of the pancreatic perforating veins as they enter the splenic vein. The anastomosis should also be performed without tension or kinking of the splenic vein. Typically, the anastomosis lies just in front of the ligated adrenal vein on the left renal vein with continuous suture.

Moon et al. [37] examined the outcomes of DSRS+SPD in children to evaluate the usefulness of this operation. The platelet count and white cell count increased significantly after DSRS+SPD. Spleen size decreased significantly. No patient underwent subsequent transplantation or endoscopic treatment for esophagogastric varices after DSRS+SPD.

DSRS + SPD + gastric transection (GT): Loss of shunt selectivity was still observed via collateral pathways through the stomach [20]. We therefore modified DSRS by additionally performing SPD and GT to prevent loss of shunt selectivity. GT involved transection and anastomosis of the upper stomach with an autosuture instrument. The short gastric arteries and veins were spared. Katoh et al. [38] performed transection and re-suture of the seromuscular layer of the upper stomach to prevent loss of selectivity after DSRS + SPD. They called this procedure "superselective DSRS." We performed transection of all layers, whereas Katoh et al. transected only the seromuscular layer of the upper stomach.

We compared long-term results for three types of DSRS for the treatment of esophageal varices. Additional treatment for recurrent varices was required in the original DSRS group (9.1%), DSRS with SPD group (18.2%), and DSRS with SPD plus GT group (4.3%). All of the patients with recurrent varices had shunt stenosis within the first year after DSRS. The prevalence of hyperammonemia in the DSRS with SPD plus GT group was significantly lower than that in the original DSRS group and the DSRS with SPD group ($P < 0.01$). There were no significant differences in survival among the three groups. DSRS with SPD plus GT may reduce the incidence of postoperative hyperammonemia [14]. Kanaya et al. [39] have reported that the incidence of hyperammonemia after DSRS with SPD plus gastric disconnection (transection of only the seromuscular layer of the upper stomach) was 3.2%. We found that the prevalence of hyperammonemia after DSRS with SPD plus GT was 0% at 1 year, 9.1% at 5 years, and 9.1% at 10 years [14]. The loss of shunt selectivity promotes hyperammonemia and decreases portal blood flow. High serum ammonia concentrations result in encephalopathy. We previously reported that obliteration of portosystemic shunts followed by partial splenic embolization is beneficial in patients with portosystemic encephalopathy. Portal venous pressures were similar before and after treatment in patients who underwent embolization of portosystemic shunts followed by partial splenic embolization [40, 41]. In patients who had portosystemic encephalopathy after DSRS, however, elevated portal venous pressures after embolization of portosystemic shunts can not be reduced by partial splenic embolization. Fisher et al. [42] have reported normalization of hyperammonemia after administration of a solution enriched with branched chain amino acids. All patients with hyperammonemia in our study should receive branched chain amino acids [14]. However, patients with hyperammonemia require long-term nutritional support, negatively affecting their quality of life. Liver dysfunction was controlled with good nutritional support. We found no significant differences in cumulative survival among the original DSRS group, DSRS with SPD group, and DSRS with SPD plus GT group [14]. Kanaya et al. [39] have reported better 5- and 7-year survival rates after DSRS with SPD plus gastric disconnection than after standard DSRS.

Santambrogio et al. [43] compared endoscopic injection sclerotherapy (EIS) with DSRS for the prevention of recurrent variceal bleeding in cirrhotic patients who underwent long-term follow-up. They concluded that DSRS with a correct portal-azygos disconnection more effectively prevents variceal rebleeding than EIS in a subgroup of patients with good liver function. However, this positive effect did not influence long-term survival because other factors (e.g., hepatocellular carcinoma) were more important determinants of the outcomes of the cirrhotic patients with portal hypertension.

Rikkers et al. (13) performed a prospective, randomized trial to evaluate the effectiveness of DSRS for the treatment of cirrhotic patients who previously had bleeding from esophageal varices. A total of 55 patients were randomly assigned to receive a DSRS (26 patients) or a nonselective shunt (29 patients). Three operative deaths occurred in each group. Early postoperative angiography revealed preservation of hepatic portal perfusion in 14 of 16 selective patients (88%), but in only 1 of 20 nonselective patients ($p < 0.001$). Quantitative measures of hepatic function (maximal rate of urea synthesis and Child's score) were similar to preoperative values in the selective shunt, but had significantly decreased in the nonselective shunt on the first postoperative evaluation. Encephalopathy has not developed in any patient with continued portal perfusion, as compared with 45% of patients without portal flow ($p < 0.05$). No significant differences between selective and nonselective shunt have been detected with respect to total cumulative mortality (10 selective, 38%; 8 nonselective, 28%), shunt occlusion (2 selective, 10%; 5 nonselective, 18%), or recurrent variceal hemorrhage (1 selective, 4%; 2 nonselective, 8%). Overall, postoperative encephalopathy has developed in significantly fewer selective patients (3 selective, 12%; 15 nonselective, 52%; $p < 0.001$). Therefore, they conclude that the DSRS, especially when its objective of maintaining hepatic portal perfusion is achieved, results in significantly less morbidity than nonselective shunt.

Warren et al. (44) reported the metabolic basis of portosystemic encephalopathy and compared the effects of selective vs. nonselective shunts. Metabolic studies were done in the Clinical Research Unit during a 14-day stay under carefully controlled dietary conditions. Maximal rate of urea synthesis did not change in patients with DSRS, but decreased significantly in those with nonselective shunt. Likewise, ammonium chloride tolerance, defined as the smallest dose required to produce a 40- $\mu\text{g}/\text{dL}$ rise in the plasma ammonia concentration, was unchanged in the DSRS group, but significantly worsened in the nonselective shunt group.

Galambos et al. [45] compared nonselective shunt with selective shunt for the treatment of bleeding esophageal varices in a randomized controlled trial. A total of 48 patients were randomly assigned to receive a nonselective shunt (24 patients) or a selective shunt (24 patients). Mortality rates, the frequencies of shunt occlusion, and the frequencies of recurrent gastrointestinal bleeding were similar. Encephalopathy developed more often after a nonselective shunt than after a selective shunt. Nonselective shunts consistently diverted the hepatopetal mesenteric-portal flow from the liver. Deterioration of hepatic function was greater after nonselective than selective shunt.

2.2. Nonshunting procedures

Historically, nonshunting procedures were developed in an attempt to decrease the high rates of encephalopathy associated with portosystemic anastomoses. An alternative to total shunt was developed by Sugiura and Futagawa in 1973 [46]. Esophageal transection (ET) disrupts the blood supply to esophagogastric varices. ET solves the problem of hepatic encephalopathy; unfortunately, however, varices can recur because portal pressure remains high.

Various nonshunting procedures, such as the Hassab operation, ET, splenectomy, or terminal esophago-proximal gastrectomy, have been developed to treat esophagogastric varices

[46-49]. All nonshunting procedures perform splenectomy. Portal vein thrombosis is not a rare complication of splenectomy and can be fatal in patients with hypersplenism. Kawana-ka et al. reported that low antithrombin 3 activity and further decreases in this activity are associated with portal vein thrombosis after splenectomy in cirrhotic patients, and that treatment with antithrombin 3 concentrates is likely to prevent the development of portal vein thrombosis in these patients [50].

2.2.1. Splenectomy

Splenectomy was one of the earliest nonshunting procedures. It was found to be generally ineffective for preventing recurrent variceal bleeding [51]. Despite elimination of the splenic component of the portal circulation, portal hypertension is maintained after simple splenectomy, and the risk of continued bleeding via the splenic venous branches is high.

Recently, laparoscopic splenectomy is widely accepted as a standard treatment for hematologic disorders such as idiopathic thrombocytopenic purpura. Laparoscopic splenectomy is improved safety in liver cirrhosis patients with portal hypertension [52].

2.2.2. Hassab operation

In 1967, Hassab [47] reported a successful technique for gastroesophageal decongestion and splenectomy, developed in Egypt. Most of his patients had schistosomiasis. The operation entailed removal of the spleen as well as devascularization of the cardiac portion of the stomach and abdominal portion of the esophagus, including the suprarenic veins. By ligating the left gastric artery and splenic artery, portal blood flow was also decreased, thereby decompressing the portal system. Recently, the Hassab operation has been employed in patients with varices limited to the stomach.

2.2.3. Terminal esophago-proximal gastrectomy

Terminal esophago-proximal gastrectomy involves proximal gastric transection and autostomy proximal gastrectomy in association with extensive devascularization and splenectomy [49].

2.2.4. ET

Among non-shunting procedures for the treatment of esophagogastric varices, ET has been the most popular operation. ET in Japan was first performed in 1967 [53], using a modification of Walker's procedure for transthoracic ET [54]. The procedure was then refined by Sugiura and Futagawa in 1973 [46]. ET consists of paraesophageal devascularization, esophageal transection and reanastomosis, splenectomy, and pyloroplasty. First, splenectomy with devascularization of the greater curvature was performed. Devascularization of the lesser curvature was done from the angle to the esophagogastric junction, and the left gastric artery was ligated and divided. The esophagus and cardia were devascularized from the lesser to the greater curvature. Then, the vagal nerve and paraesophageal vessels were ligated and divided. The esophagus was completely transected above the esophagogastric junction, and the mucosa was anastomosed with interrupted sutures, performed recently with

an autosuture instrument. ET was done using three different approaches, transthoracic, thoracoabdominal, and transabdominal. Devascularization of the esophagus and the stomach is most extensive and complete in the thoracoabdominal approach; however, this is the most drastic procedure.

Sugiura et al. [55] reported on 636 patients with portal hypertension in whom ETs with paraesophagogastric devascularization were performed to manage esophageal varices. The operative mortality rates were as follows: emergency cases 13.7%, elective cases 3.2%, prophylactic cases 4.3%, and overall 5.2%. There were no deaths among the 233 patients in Child's class A; the 232 patients in class B had a 2% mortality rate, and the 171 patients in class C had a 17% mortality rate. The 10-year actuarial survival rates in patients with cirrhosis were 55% in emergency cases, 72% in prophylactic cases, and 72% in elective cases. In patients without cirrhosis, the corresponding survival rates were 90%, 96%, and 95%, respectively. The recurrence rate of variceal bleeding or varices was less than 5%. They concluded that the Sugiura procedure is safe and effective for controlling esophageal varices and prolongs the long-term survival of patients with portal hypertension.

In our study, however, the recurrence rate of varices after ET was high [21]. We examined hemodynamic changes associated with recurrent esophageal varices after ET and evaluated the effectiveness of EIS for their treatment. Nineteen patients with recurrent esophageal varices after ET were treated by EIS. Endoscopic varicealography during injection sclerotherapy (EVIS), following oral blockage of flow by a balloon, identified three patterns: type 1 (common type), continuous filling by the feeder vessel of the varix; type 2 (retrograde disappearing type), confirmed hepatofugal flow; and type 3 (immediate washout type), immediate washout of contrast medium. Angiography showed that the hepatofugal feeder vessel was the right gastric vein in all cases. Recurrent esophageal varices were classified as type 1 in 14 patients (73.7%), type 2 in 4 (21.1%), and type 3 in 1 (5.3%). Fewer treatment sessions were required in type 1 than in type 2 varices ($p < 0.005$). Recurrent varices were completely eradicated in all patients except the patient with type 3 disease. Cumulative re-recurrence rates at 5 and 10 years were higher in type 1 than in type 2 varices without significance (28.6% and 71.4% vs. 25.0% and 25.0%, respectively). Cumulative survival rates after EIS at 5 and 10 years also were similar for type 1 and type 2 varices (77.1% and 66.1% vs. 66.7% and 66.7%). EIS was thus effective for the management of recurrent esophageal varices after ET, excluding type 3 disease [56].

Cleva et al. [57] compared the systemic hemodynamic effects of DSRS with those of esophagogastric devascularization and splenectomy in patients treated for schistosomal portal hypertension. The hyperdynamic circulatory state observed in Manson's schistosomiasis was corrected by esophagogastric devascularization and splenectomy, but persisted in patients who underwent DSRS. Similarly, the elevated mean pulmonary artery pressure resolved after esophagogastric devascularization and splenectomy, but persisted after DSRS. They concluded that esophagogastric devascularization and splenectomy seems to be the most physiologic operation for patients with schistosomal portal hypertension.

We compared the long-term results of DSRS and ET in cirrhotic patients with complete variceal eradication who were followed up for at least 3 years. There was no recurrent varix in the DSRS group. The cumulative recurrence rates of varices in the ET group were 31.6% and

52.5% at 5 and 10 years, respectively. The cumulative rates of hyperammonemia at 5 and 10 years were significantly higher in the DSRS group (30.4%, 30.4%) than in the ET group (0%, 5.6%) ($p=0.009$). The cumulative survival rates in the DSRS group vs. the ET group were 90.9% vs. 94.7% at 5 years and 85.2% vs. 81.7% at 10 years (NS). These results suggest that DSRS is more effective than ET in preventing recurrence of esophageal varices, but is associated with a higher incidence of hyperammonemia [21]. In that study, no patient who underwent DSRS with complete eradication had recurrent varices. When collateral pathways to the esophagus develop after DSRS, flow is via the short gastric veins, the splenic vein, and the left renal vein. After ET, collateral pathways to the esophagus develop across the transection site and generate new varices. Most of the recurrent varices in the ET group were supplied by the right gastric vein across the transection site [56]. However, collateral flow in the DSRS group decreased hepatic blood flow and led to the development of postoperative hyperammonemia. Rikkers et al. [58] reported that patients with no hepatic portal perfusion had the worst survival and greatest morbidity after DSRS.

Idiopathic portal hypertension (IPH) is a disease of unknown etiology characterized by splenomegaly, anemia, and portal hypertension. This disorder develops in the absence of liver cirrhosis, extrahepatic portal vein occlusion, schistosomiasis, or any other identifiable cause [59, 60]. We evaluated the results of shunting and nonshunting procedures for the treatment of esophagogastric varices in patients with IPH. Esophagogastric varices were completely eradicated in 3 (75.0%) patients in the shunting group and 4 (80.0%) in the nonshunting group. Additional endoscopic treatment (one session) was performed in 2 patients with incompletely eradicated varices. There was no recurrence in the shunting group. In the nonshunting group, esophagogastric varices recurred in all 4 patients with completely eradicated varices. All recurrent esophageal varices were completely eradicated. Postoperative platelet counts ($\times 10^4/\mu\text{L}$) were significantly lower in the shunting group (10.0 ± 2.6) than in the nonshunting group (42.0 ± 14.0) ($p=0.0029$). The increase in the platelet count after operation was significantly lower in the shunting group (1.7 ± 0.2 times) than in the nonshunting group (5.8 ± 2.9 times) ($p=0.0267$). No patient received anticoagulants postoperatively. Portal venous thrombus did not develop in the shunting group, but appeared in 4 patients (80.0%) in the nonshunting group. No patient had loss of shunt selectivity or portal-systemic encephalopathy. One patient in the nonshunting group died of cerebral hemorrhage; all others are alive. Shunting procedure, DSRS, was suggested to be useful for the management of esophagogastric varices in patients with IPH [61].

Author details

Hiroshi Yoshida^{1*}, Yasuhiro Mamada², Nobuhiko Taniai², Takashi Tajiri² and Eiji Uchida²

*Address all correspondence to: hiroshiy@nms.ac.jp

1 Department of Surgery, Nippon Medical School Tama Nagayama Hospital, Japan

2 Department of Surgery, Nippon Medical School, Japan

References

- [1] Yoshida H, Mamada Y, Taniai N, Tajiri T. New methods for the management of esophageal varices. *World J Gastroenterol*. 2007 Mar 21;13(11):1641-5.
- [2] Yoshida H, Mamada Y, Taniai N, Tajiri T. New methods for the management of gastric varices. *World J Gastroenterol*. 2006 Oct 7;12(37):5926-31.
- [3] Yoshida H, Mamada Y, Taniai N, Yoshioka M, Hirakata A, Kawano Y, et al. Treatment modalities for bleeding esophagogastric varices. *J Nippon Med Sch*. 2012;79(1):19-30.
- [4] Tajiri T, Yoshida H, Obara K, Onji M, Kage M, Kitano S, et al. General rules for recording endoscopic findings of esophagogastric varices (2nd edition). *Dig Endosc*. 2010 Jan;22(1):1-9.
- [5] Yoshida H, Mamada Y, Taniai N, Yamamoto K, Kawano Y, Mizuguchi Y, et al. A randomized control trial of bi-monthly versus bi-weekly endoscopic variceal ligation of esophageal varices. *Am J Gastroenterol*. 2005 Sep;100(9):2005-9.
- [6] Yoshida H, Mamada Y, Taniai N, Tajiri T. New trends in surgical treatment for portal hypertension. *Hepatol Res*. 2009 Oct;39(10):1044-51.
- [7] Inokuchi K, Kobayashi M, Kusaba A, Ogawa Y, Saku M, Shiizaki T. New selective decompression of esophageal varices. By a left gastric venous-caval shunt. *Arch Surg*. 1970 Feb;100(2):157-62.
- [8] Belghiti J, Grenier P, Nouel O, Nahum H, Fekete F. Long-term loss of Warren's shunt selectivity. Angiographic demonstration. *Arch Surg*. 1981 Sep;116(9):1121-4.
- [9] Shields R. Small-diameter PTFE portosystemic shunts: portocaval vs mesocaval. *HPB Surg*. 1998;10(6):413-4.
- [10] Mercado MA, Morales-Linares JC, Granados-Garcia J, Gomez-Mendez TJ, Chan C, Orozco H. Distal splenorenal shunt versus 10-mm low-diameter mesocaval shunt for variceal hemorrhage. *Am J Surg*. 1996 Jun;171(6):591-5.
- [11] Paquet KJ, Lazar A, Koussouris P, Hotzel B, Gad HA, Kuhn R, et al. Mesocaval interposition shunt with small-diameter polytetrafluoroethylene grafts in sclerotherapy failure. *Br J Surg*. 1995 Feb;82(2):199-203.
- [12] Warren WD, Zeppa R, Fomon JJ. Selective trans-splenic decompression of gastroesophageal varices by distal splenorenal shunt. *Ann Surg*. 1967 Sep;166(3):437-55.
- [13] Rikkers LF, Rudman D, Galambos JT, Fulenwider JT, Millikan WJ, Kutner M, et al. A randomized, controlled trial of the distal splenorenal shunt. *Ann Surg*. 1978 Sep;188(3):271-82.

- [14] Tajiri T, Onda M, Yoshida H, Mamada Y, Tani ai N, Umehara M, et al. Long-term results of modified distal splenorenal shunts for the treatment of esophageal varices. *Hepatogastroenterology*. 2000 May-Jun;47(33):720-3.
- [15] Stipa S, Balducci G, Ziparo V, Stipa F, Lucandri G. Total shunting and elective management of variceal bleeding. *World J Surg*. 1994 Mar-Apr;18(2):200-4.
- [16] Klein AS, Fair JH, Cameron JL. Suprarenal mesocaval shunt. *SurgGynecol Obstet*. 1991 Oct;173(4):319-22.
- [17] Sato Y, Hatakeyama K. Left gastric venous caval direct shunt in esophagogastric varices. *Hepatogastroenterology*. 2002 Sep-Oct;49(47):1251-2.
- [18] Inokuchi K, Beppu K, Koyanagi N, Nagamine K, Hashizume M, Sugimachi K. Exclusion of nonisolated splenic vein in distal splenorenal shunt for prevention of portal malcirculation. *Ann Surg*. 1984 Dec;200(6):711-7.
- [19] Inokuchi K. Selective decompression of esophageal varices by a left gastric venacaval shunt. *SurgAnnu*. 1978;10:215-36.
- [20] Henderson JM, Warren WD, Millikan WJ, Galloway JR, Kawasaki S, Kutner MH. Distal splenorenal shunt with splenopancreatic disconnection. A 4-year assessment. *Ann Surg*. 1989 Sep;210(3):332-9; discussion 9-41.
- [21] Tajiri T, Onda M, Yoshida H, Mamada Y, Tani ai N, Yamashita K. Comparison of the long-term results of distal splenorenal shunt and esophageal transection for the treatment of esophageal varices. *Hepatogastroenterology*. 2000 Nov-Dec;47(36):1619-21.
- [22] Rikkers LF. Definitive therapy for variceal bleeding: a personal view. *Am J Surg*. 1990 Jul;160(1):80-5.
- [23] Inokuchi K, Beppu K, Koyanagi N, Nagamine K, Hashizume M, Iwanaga T, et al. Fifteen years' experience with left gastric venous caval shunt for esophageal varices. *World J Surg*. 1984 Oct;8(5):716-21.
- [24] Inokuchi K, Kobayashi M, Ogawa Y, Saku M, Nagasue N. Results of left gastric vena caval shunt for esophageal varices: Analysis of one hundred clinical cases. *Surgery*. 1975 Nov;78(5):628-36.
- [25] Inokuchi K. A selective portacaval shunt. *Lancet*. 1968 Jul 6;2(7558):51-2.
- [26] Millikan WJ, Jr., Warren WD, Henderson JM, Smith RB, 3rd, Salam AA, Galambos JT, et al. The Emory prospective randomized trial: selective versus nonselective shunt to control variceal bleeding. Ten year follow-up. *Ann Surg*. 1985 Jun;201(6):712-22.
- [27] Rikkers LF, Jin G. Variceal hemorrhage: surgical therapy. *GastroenterolClin North Am*. 1993 Dec;22(4):821-42.
- [28] Rikkers LF, Sorrell WT, Jin G. Which portosystemic shunt is best? *GastroenterolClin North Am*. 1992 Mar;21(1):179-96.

- [29] Smith RB, 3rd, Warren WD, Salam AA, Millikan WJ, Ansley JD, Galambos JT, et al. Dacron interposition shunts for portal hypertension. An analysis of morbidity correlates. *Ann Surg.* 1980 Jul;192(1):9-17.
- [30] Sarfeh IJ, Rypins EB. The emergency portacaval H graft in alcoholic cirrhotic patients: influence of shunt diameter on clinical outcome. *Am J Surg.* 1986 Sep;152(3):290-3.
- [31] Sarfeh IJ, Rypins EB, Fardi M, Conroy RM, Mason GR, Lyons KP. Clinical implications of portal hemodynamics after small-diameter portacaval H graft. *Surgery.* 1984 Aug;96(2):223-9.
- [32] Sarfeh IJ, Rypins EB, Mason GR. A systematic appraisal of portacaval H-graft diameters. Clinical and hemodynamic perspectives. *Ann Surg.* 1986 Oct;204(4):356-63.
- [33] Sarfeh IJ, Rypins EB, Raiszadeh M, Milne N, Conroy RM, Lyons KP. Serial measurement of portal hemodynamics after partial portal decompression. *Surgery.* 1986 Jul;100(1):52-8.
- [34] Henderson JM, Millikan WJ, Jr., Wright-Bacon L, Kutner MH, Warren WD. Hemodynamic differences between alcoholic and nonalcoholic cirrhotics following distal splenorenal shunt--effect on survival? *Ann Surg.* 1983 Sep;198(3):325-34.
- [35] Henderson JM, El Khishen MA, Millikan WJ, Jr., Sones PJ, Warren WD. Management of stenosis of distal splenorenal shunt by balloon dilation. *SurgGynecol Obstet.* 1983 Jul;157(1):43-8.
- [36] Warren WD, Millikan WJ, Jr., Henderson JM, Abu-Elmagd KM, Galloway JR, Shires GT, 3rd, et al. Splenopancreatic disconnection. Improved selectivity of distal splenorenal shunt. *Ann Surg.* 1986 Oct;204(4):346-55.
- [37] Moon SB, Jung SE, Ha JW, Park KW, Seo JK, Kim WK. The usefulness of distal splenorenal shunt in children with portal hypertension for the treatment of severe thrombocytopenia and leukopenia. *World J Surg.* 2008 Mar;32(3):483-7.
- [38] Katoh H, Shimosawa E, Kojima T, Tanabe T. Modified splenorenal shunt with splenopancreatic disconnection. *Surgery.* 1989 Nov;106(5):920-4.
- [39] Kanaya S, Katoh H. Long-term evaluation of distal splenorenal shunt with splenopancreatic and gastric disconnection. *Surgery.* 1995 Jul;118(1):29-35.
- [40] Yoshida H, Mamada Y, Taniai N, Yamamoto K, Kaneko M, Kawano Y, et al. Long-term results of partial splenic artery embolization as supplemental treatment for portal-systemic encephalopathy. *Am J Gastroenterol.* 2005 Jan;100(1):43-7.
- [41] Yoshida H, Mamada Y, Taniai N, Tajiri T. Partial splenic embolization. *Hepatol Res.* 2008 Mar;38(3):225-33.
- [42] Fischer JE, Rosen HM, Ebeid AM, James JH, Keane JM, Soeters PB. The effect of normalization of plasma amino acids on hepatic encephalopathy in man. *Surgery.* 1976 Jul;80(1):77-91.

- [43] Santambrogio R, Opocher E, Costa M, Bruno S, Ceretti AP, Spina GP. Natural history of a randomized trial comparing distal spleno-renal shunt with endoscopic sclerotherapy in the prevention of variceal rebleeding: a lesson from the past. *World J Gastroenterol*. 2006 Oct 21;12(39):6331-8.
- [44] Warren WD, Rudman D, Millikan W, Galambos JT, Salam AA, Smith RB, 3rd. The metabolic basis of portasystemic encephalopathy and the effect of selective vs nonselective shunts. *Ann Surg*. 1974 Oct;180(4):573-9.
- [45] Galambos JT, Warren WD, Rudman D, Smith RB, 3rd, Salam AA. Selective and total shunts in the treatment of bleeding varices. A randomized controlled trial. *N Engl J Med*. 1976 Nov 11;295(20):1089-95.
- [46] Sugiura M, Futagawa S. A new technique for treating esophageal varices. *J Thorac Cardiovasc Surg*. 1973 Nov;66(5):677-85.
- [47] Hassab MA. Gastroesophageal decongestion and splenectomy in the treatment of esophageal varices in bilharzial cirrhosis: further studies with a report on 355 operations. *Surgery*. 1967 Feb;61(2):169-76.
- [48] Hassab MA. Gastro-esophageal decongestion and splenectomy GEDS (Hassab), in the management of bleeding varices. Review of literature. *Int Surg*. 1998 Jan-Mar; 83(1):38-41.
- [49] Yamamoto S, Hidemura R, Sawada M, Takeshige K, Iwatsuki S. The late results of terminal esophagoproximal gastrectomy (TEPG) with intensive devascularization and splenectomy for bleeding esophageal varices in cirrhosis. *Surgery*. 1976 Jul;80(1): 106-14.
- [50] Kawanaka H, Akahoshi T, Kinjo N, Konishi K, Yoshida D, Anegawa G, et al. Impact of antithrombin III concentrates on portal vein thrombosis after splenectomy in patients with liver cirrhosis and hypersplenism. *Ann Surg*. 2010 Jan;251(1):76-83.
- [51] Smith GW. Splenectomy and coronary vein ligation for the control of bleeding esophageal varices. *Am J Surg*. 1970 Feb;119(2):122-31.
- [52] Kawanaka H, Akahoshi T, Kinjo N, Konishi K, Yoshida D, Anegawa G, et al. Technical standardization of laparoscopic splenectomy harmonized with hand-assisted laparoscopic surgery for patients with liver cirrhosis and hypersplenism. *J HepatobiliaryPancreat Surg*. 2009;16(6):749-57.
- [53] Idezuki Y, Sugiura M, Sakamoto K, Abe H, Miura T, Hatano S, et al. Rationale for transthoracic esophageal transection for bleeding varices. *Dis Chest*. 1967 Nov;52(5): 621-31.
- [54] Walker RM. Transection operations for portal hypertension. *Thorax*. 1960 Sep; 15:218-24.

- [55] Sugiura M, Futagawa S. Results of six hundred thirty-six esophageal transections with paraesophagogastricdevascularization in the treatment of esophageal varices. *J Vasc Surg.* 1984 Mar;1(2):254-60.
- [56] Yoshida H, Onda M, Tajiri T, Toba M, Umehara M, Mamada Y, et al. Endoscopic injection sclerotherapy for the treatment of recurrent esophageal varices after esophageal transection.. *Dig Endosc.* [original]. 2002;14:93-8.
- [57] de Cleve R, Herman P, D'Albuquerque L A, Pugliese V, Santarem OL, Saad WA. Pre- and postoperative systemic hemodynamic evaluation in patients subjected to esophagogastricdevascularization plus splenectomy and distal splenorenal shunt: a comparative study in schistosomal portal hypertension. *World J Gastroenterol.* 2007 Nov 7;13(41):5471-5.
- [58] Rikkers LF, Cormier RA, Vo NM. Effects of altered portal hemodynamics after distal splenorenal shunts. *Am J Surg.* 1987 Jan;153(1):80-5.
- [59] Boyer JL, Sen Gupta KP, Biswas SK, Pal NC, BasuMallick KC, Iber FL, et al. Idiopathic portal hypertension. Comparison with the portal hypertension of cirrhosis and extrahepatic portal vein obstruction. *Ann Intern Med.* 1967 Jan;66(1):41-68.
- [60] Okuda K, Kono K, Ohnishi K, Kimura K, Omata M, Koen H, et al. Clinical study of eighty-six cases of idiopathic portal hypertension and comparison with cirrhosis with splenomegaly. *Gastroenterology.* 1984 Apr;86(4):600-10.
- [61] Yoshida H, Mamada Y, Taniai N, Mineta S, Kawano Y, Mizuguchi Y, et al. Shunting and nonshunting procedures for the treatment of esophageal varices in patients with idiopathic portal hypertension. *Hepatogastroenterology.* 2010 Sep-Oct;57(102-103): 1139-44.

