Distal Splenorenal Shunt *Versus* Endoscopic Sclerotherapy in the Prevention of Variceal Rebleeding

First Stage of a Randomized, Controlled Trial

GIAN PAOLO SPINA, M.D.,* ROBERTO SANTAMBROGIO, M.D.,† ENRICO OPOCHER, M.D.,†
FELICE COSENTINO, M.D.,† ALESSANDRO ZAMBELLI, M.D.,‖ GIOVANNI RUBIS PASSONI, M.D.,*
GIOVANNI CUCCHIARO, M.D.,* MASSIMO MACRÌ, M.D, ELISABETTA MORANDI, M.D.,†
SAVINO BRUNO, M.D.,§ AND GIUSEPPE PEZZUOLI, M.D.,‡

In 1984 we started a prospective controlled trial comparing endoscopic sclerotherapy (ES) with the distal splenorenal shunt (DSRS) in the elective treatment of variceal hemorrhage in cirrhotic patients. The study population included 40 patients with cirrhosis and portal hypertension referred to our department from October 1984 to March 1988. These patients were drawn from a pool of 173 patients who underwent either elective surgery or endoscopic sclerotherapy during this time. Patients were assigned to one of the two groups according to a random-number table: 20 to DSRS and 20 to ES. During the postoperative period, no DSRS patient died, while one ES patient died of uncontrolled hemorrhage. One DSRS patient had mild recurrent variceal hemorrhage despite an angiographically patent DSRS. Four ES patients suffered at least one episode of gastrointestinal bleeding: two from varices and two from esophageal ulcerations. Five ES patients developed transitory dysphagia. Long-term follow-up was complete in all patients. Two-year survival rates for shunt (95%) and ES (90%) groups were similar. One DSRS patient rebled from duodenal ulcer, while three ES patients had recurrent bleeding from esophagogastric sources (two from varices and one from hypertensive gastropathy). One DSRS and two ES patients have evolved a mild chronic encephalopathy; four DSRS and two ES patients suffered at least one episode of acute encephalopathy. Two ES patients had esophageal stenoses, which were successfully dilated. Preliminary data from this trial seem to indicate that DSRS, in a subgroup of patients with good liver function and a correct portal-azygos disconnection, more effectively prevents variceal rebleeding than ES. However no significant difference in the survival of the two treatment groups was noted.

THE SELECTIVE DISTAL splenorenal shunt (DSRS) proposed by Warren et al.[1] in 1967 appeared to be the best procedure available for surgical decompression of patients with portal hypertension.[2-4] It seemed to combine the advantages of the definitive treatment of varices, typical of totally diverting shunts, without harmful loss of hepatopetal portal flow. This resulted in a better quality of life, as reported in several studies comparing the clinical results of selective and nonselective shunts.[3-9]

It has been shown that endoscopic sclerotherapy (ES) protects against variceal rebleeding when compared to a medical regimen[10-12] and improves long-term survival,[13] resulting in a possible alternative to shunt surgery.

To evaluate the efficacy of these treatments, we started a prospective controlled trial in 1984 comparing the efficacy of ES and DSRS in the prevention of variceal rebleeding in cirrhotic patients.[17]

Our study is similar to three recently published randomized controlled trials.[14-16]

Methods

The study population comprised 40 patients with cirrhosis and portal hypertension referred to our department from October 1984 to March 1988. These patients were drawn from a pool of 173 patients who underwent either elective surgery or endoscopic sclerotherapy because of portal hypertension during this time.

The criteria for inclusion into the study were (1) liver cirrhosis confirmed by biopsy in all patients; (2) endoscopic documentation of variceal hemorrhage (actively bleeding varix or nonbleeding varices without other lesions) requiring at least one unit of blood transfusion; (3) arrest of acute variceal hemorrhage either spontaneously

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or by use of intravenous vasopressin and/or somatostatin and/or balloon tamponade and/or hemostatic sessions of ES; (4) patient age of less than 70 years; (5) good or moderate liver function (Child's A and B class);[18] (6) patency of the splanchnic venous system and hepatopetal portal flow (according to Nordlinger's classification);[19] (7) eligible for either shunt or ES; (8) absence of life-threatening diseases (*i.e.*, tumors); and (9) willingness to return for regular sclerosis and follow-up. Patients bleeding from gastric varices were excluded. Figure 1 shows the reasons for the exclusion of 133 patients. Randomization was done when the patient was stabilized, no more than 24 hours before treatment. Patients were assigned to one of the two groups according to a random-number table. Informed written consent was obtained from all patients before their inclusion in the study. No patient refused the assigned treatment. Variceal rebleeding within 2 years of first treatment was considered the primary measure of patient outcome. The sample requirements to show a decrease in variceal rebleeding from 43% to 7% are about 20 patients for each group, applying standard power (90%), type I error (p < 0.05) and a two-tailed t test.[20]

Preoperative Evaluation

A complete medical history was obtained in each patient with particular attention to previous episodes of gastrointestinal bleeding and evidence of either primary or posthemorrhagic hepatic failure (jaundice, ascites, or edema). Physical examination concentrated on the assessment of the nutritional state and the presence of hepatosplenomegaly, jaundice, ascites, and edema. Routine laboratory tests were performed to evaluate liver function (Table 1). An overall assessment of the severity of liver disease (hepatic score index) was made at the time of patient inclusion in the study to complete Child's classification and adequately compare the two groups of patients.[3] A score from 1 to 3 according to the level of severity was assigned to each clinical and laboratory parameter. The total of the scores, the maximum of which is 27, was called the hepatic score sum. It is often impossible to compare different hepatic score sums because of missed parameters in some patients. Thus the hepatic score index was expressed as the ratio of the hepatic score sum to the highest possible hepatic score sum. Serum alfafeto-protein and ultrasonography were routinely obtained to screen the presence of hepatic neoplasm. The etiology of cirrhosis was determined from clinical history, serum markers of viral hepatitis and liver biopsy. The presence of esophageal varices was assessed through endoscopic examination. Criteria used for classifying the endoscopic findings were based on the General Rules for Recording Endoscopic Findings on Esophageal Varices compiled by the Japanese Research Society for Portal Hypertension.[21]

Cerebral function was assessed through a complete neurologic examination, taking into account the mental state, asterixis, electroencephalographic findings (EEG), and the trail making test.[21,23] All parameters were assessed
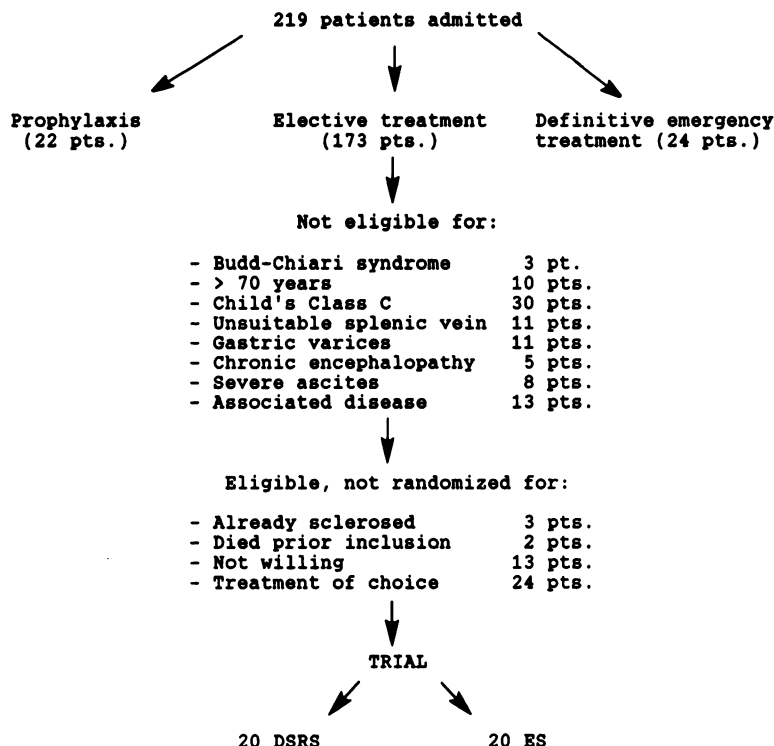

FIG. 1. Reasons for exclusion.

SPINA AND OTHERS

TABLE 1. *Characteristics of Patients*

Characteristic	DSRS (n = 20)	ES (n = 20)
Age (years)	46.9 ± 10.3	51.2 ± 8.2
Sex M/F	11/9	16/4
Etiology*		
Alcoholic	2	13
Nonalcoholic	18	7
Child's class		
A	8	7
B	12	13
Hepatic score	0.52 ± 0.05	0.58 ± 0.08
Prothrombin time (%)		
>80	2	5
80–50	15	11
<50	3	4
Bilirubin (mg/dL)		
<1.2	13	9
1.2–3	7	8
>3	0	3
Albumin (gr/dL)		
>3	18	18
3–2.5	2	2
<2.5	0	0
Portal perfusion		
Degree I	36%	27%
II	53%	40%
III	12%	33%
N. of previous bleeding	2.3 ± 1.7	1.9 ± 1.0
N. of blood unit	3.1 ± 2.3	2.2 ± 2.1
Interval between bleeding and treatment (months)	3.2 ± 2.3	3.1 ± 2.4
N. of previous encephalopathy episodes	5	5
Easily controlled preoperative ascites	2	3

* Alcoholic *versus* nonalcoholic: p = 0.0031.

using the 0 to 4 rating system proposed by Conn.[24] The "Cancelling A's" test was also used[25] and rated with the same method. Each parameter was arbitrarily weighted in proportion to its importance. Mental state was assigned a factor of 4, asterixis and EEG a factor of 1, and each of the others a factor of 0.5. An overall score for hepatic encephalopathy (HE) was calculated from the sum of the values for each of the five parameters.[4] We called HE "acute" if it was precipitated by gastrointestinal bleeding, heavy drinking, pharmacologic or dietary imbalances, of brief duration and easily controlled with elimination of precipitating cause. We called HE chronic if it was spontaneous, of long duration, and more difficult to manage. Naturally preoperative HE was excluded in all patients.

A visceral angiogram was obtained by selective catheterization of celiac axis and superior mesenteric artery. The degree of hepatic perfusion was evaluated according to Nordlinger's criteria.[19] The rate of contrast (sodium and meglumine ioxaglate, Hexabrix 320—Byk Gulden, Milan, Italy) infusion was 6 mL/seconds × 10 seconds.

Operative Management

All shunts were performed by a single experienced surgical team.[26] DSRS was constructed according to the technique described by Warren[27] (15 cases). In five cases a splenopancreatic disconnection[28] was performed as a technical addition to DSRS, offering the optimum surgical therapy to each patient.

Endoscopic variceal sclerosis was conducted by two endoscopists with extensive experience in this field.[29] All patients were given 5 to 10 mg diazepam premedication. ES was performed using an Olympus GIF IT (Lorenzatto, Torino, Italy) flexible endoscope or an electronic Welch Allyn videoendoscope (Coresea, Milan, Italy). At each session 10 to 50 mL of polidocanol (0.5% to 1%) (Athoxysclerol, Creusller) and 0.5% methylene blue were injected using a flexible injection needle in the area 5 to 7 cm above the esophagogastric junction. Methylene blue allowed visual confirmation of intravariceal and paravariceal injections.

Postoperative Evaluation

In the evaluation of the hospital mortality rate and early complications, we defined the first 30 days after the initial treatment as the postoperative period. In the ES group, the events that occurred during the interval between the first session and eradication were also recorded and evaluated.

In the postoperative period, esophageal endoscopy was performed in each patient. A visceral angiography was performed on the 10th average postoperative day only in patients having DSRS. Shunt patency was verified in venous phase of angiograms in the DSRS group. In the ES group, the number of further sessions depended on the findings obtained at endoscopy performed 1 week after the first sclerosis session. As soon as the eradication was achieved, the patient was included in the follow-up program. The remaining ES patients underwent further sclerosis sessions if they were free of complications such as mucosal ulcerations, symptomatic stricture, severe esophagitis, fever, and pneumonia. In the presence of complications, an upper endoscopy was performed at intervals of seven to ten days and further ES sessions were considered only when complications were resolved. During the follow-up period, DSRS patients were checked the first, third, and sixth month after discharge and then at least twice yearly, on an outpatient basis. Follow-up endoscopy was scheduled 4 to 6 weeks after the last session and then at 6-month intervals, unless recurrent hemorrhage occurred.

At each visit, liver function was evaluated after a complete medical examination and laboratory tests. Longitudinal assessment of liver function was quantified using

an arbitrary score calculated on the basis of several standard laboratory tests. Synthesis activity (albumin and prothrombin time determinations), excretion (bilirubin and alkaline phosphatase levels) and the presence of hepatocellular necrosis (serum glutamic-oxaloacetic transaminase determinations) were quantified by the same method previously described for the preoperative assessment of global functional hepatic reserve.[3] This index was obtained yearly after treatment and compared each time with preoperative values. It was only used for the longitudinal assessment of liver function and termed longitudinal hepatic score index. Child's classification and the preoperative hepatic score index were not suitable to follow-up because some parameters could not be assessed over a long period of time.

The assessment of the neurologic status was performed using the above-mentioned criteria. An EEG was obtained at least once a year. A return to drinking was based on patient's statements, our own assessment, and information from relatives. Continued drinking was defined as daily consumption in excess of 1 L of wine and/or spirits. All patients were on a 10-meq sodium and protein-balanced diet (1 g protein/Kg body weight) and undergoing lactulose prophylactic treatment: the initial dose was 60 g/day in three separate doses and adjusted thereafter to induce at least one bowel movement per day.

Definitions

Eradication was defined as absence of varices or presence of F1 white varices. Rebleeding was defined as hemorrhage due to esophagogastric varices and/or congestive gastropathy that required at least one unit blood transfusion and was designated as being from varices if supported by endoscopic findings. The ideal treatment of variceal rebleeding was emergency sclerotherapy. Chronic rebleeding from congestive gastropathy was treated with beta-blocking therapy.[30] Rebleeding due to peptic ulcer was recorded separately.

The risk of hospitalization for HE was defined by taking the number of late hospital admissions due to episodes of HE and dividing it by the total number of patients evaluated in the follow-up period.

Death, whatever the cause, was considered failure of therapy in both groups. Failure of shunt was defined as shunt thrombosis on follow-up angiographic evaluation. Sclerotherapy failure was defined as a change to surgery due to recurrent hemorrhage or severe complications that prevented further ES. This decision was taken by the endoscopists and surgeon and was not based on a specific number or degree of severity of the recurrent bleeding episodes.

Data Management and Statistical Analysis

Initial evaluation and subsequent follow-up data were collected on databases (Excel, Microsoft Corp., Cologno, Monzese, Italy) for computer input (Epson PC AX computer, Seiko Epson Corporation) and subsequent analysis (Microstat, Ecosoft Inc., Indianapolis, IN). Survival and therapy failures were analyzed by the Kaplan–Meier method and were compared by the log-rank test.[31] Comparison between groups was made by chi square test for proportions and Student's t test for the means.

Results

In the ES group, varices were completely eradicated in 19 patients (95%): one patient died before eradication. The number of injection sessions were 3.1 ± 1.1 (95% CI from 2.6 to 3.6 months), occurring over 4.8 ± 3.7 months (95% CI from 3.2 to 6.4 months) and the mean amount of polydocanol required for eradication was 89.4 ± 43.6 mL (95% CI from 70 to 108.7 mL).

During the postoperative period, no DSRS patient died, while one ES patient died of uncontrolled hemorrhage after the first sclerosis session. One DSRS patient had mild recurrent variceal hemorrhage controlled by conservative therapy, despite an angiographically patent shunt. Four ES patients suffered at least one episode of gastrointestinal bleeding. Two were from varices requiring emergency variceal sclerotherapy and two from esophageal ulcerations, which were managed conservatively. No patient had episodes of hepatic encephalopathy. Five ES patients developed transitory dysphagia due to esophageal ulcerations. One ES patient suffered pleural space effusion.

The mean follow-up in the DSRS group was 29.2 ± 11.4 months (95% CI from 24.2 to 34.1 months) and the mean follow-up in the ES group was 23.8 ± 14.8 months (95% CI from 23.8 to 30.3 months). Two-year survival rates for shunt (95%) and ES (90%) groups were similar (Fig. 2). One DSRS patient died of intestinal obstruction, one of liver cancer and one of heart failure. One ES patient died of hepatic failure and one of unknown causes. Failure of therapy was at that moment equal to the death rate: no patient had shunt thrombosis or changed treatment. In the ES group, varices reformed in 5 of 19 eradicated patients (26%) after 8.6 ± 7.4 months (95% CI from 2 to 15.3 months). They were successfully re-eradicated. No DSRS patient had variceal rebleeding (one DSRS patient rebled from duodenal ulcer), while two ES patients had recurrent hemorrhage from varices and one from hypertensive gastropathy. The global percentage of patients who rebled was 5% and 35%, respectively (p = 0.04). Actuarial curves of patients free from esophagogastric rebleeding, for the two groups, confirmed the efficacy of DSRS (Fig. 3). One DSRS and two ES patients developed mild chronic
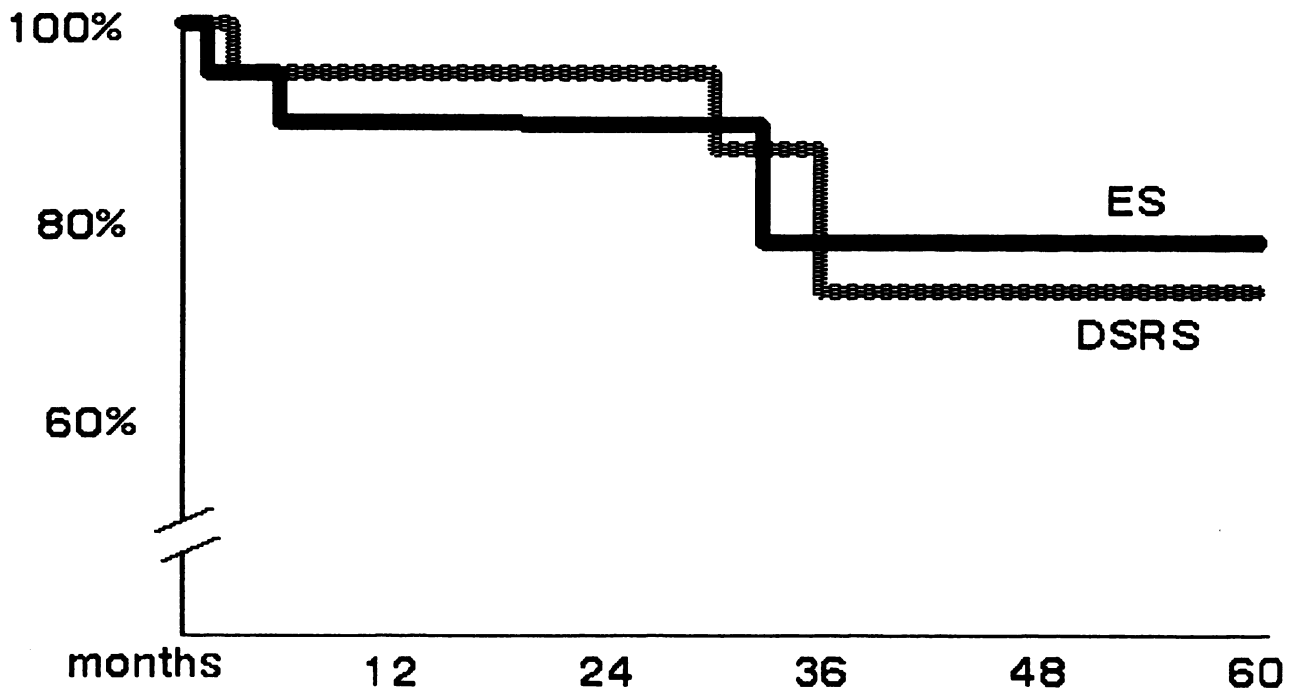
FIG. 2. Actuarial survival curves for distal splenorenal shunt (DSRS) and endoscopic sclerotherapy (ES). The vertical axis indicates the survival rate. No significant difference was noted using the log-rank test.

encephalopathy. Four DSRS and 2 ES patients suffered at least 1 episode of acute encephalopathy (p was not significant): 2 (1 DSRS and 1 ES) due to pharmacologic imbalance, 2 (1 DSRS and 1 ES) due to heavy drinking, 1 DSRS due to dietary abuse and 1 DSRS due to severe hyperglycemia. The results of trail-making test (TMT), cancelling A's test and electroencephalography (EEG) confirmed the similar outcome in the two groups of pa-
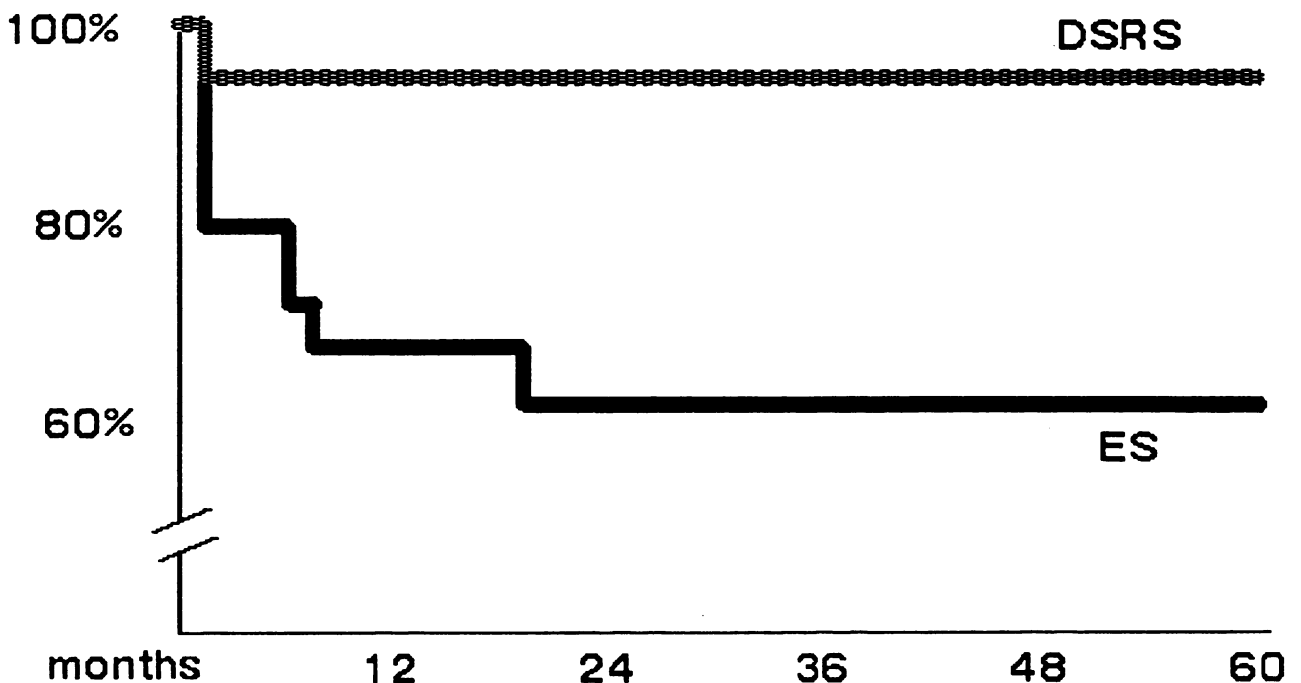

FIG. 3. Actuarial curves of patients free from gastroesophageal rebleeding for distal splenorenal shunt (DSRS) and endoscopic sclerotherapy (ES). The vertical axis indicates the rate of patients free of gastroesophageal rebleeding. The DSRS group has significantly prevented gastroesophageal rebleeding compared to ES (p < 0.05) using the log-rank test.

TABLE 2. *Comparison of the Results of TMT,*
Cancelling A's Test, and EEG

Period	Preoperative	Long-Term Follow-up
TMT (sec.)		
DSRS	50.8 ± 20.5	52.1 ± 21.3
ES	61.9 ± 17.9	60.2 ± 24.5
A's test*		
DSRS	4.4 ± 3.1	3.5 ± 3.3
ES	5.8 ± 4.5	5.2 ± 3.5
EEG (% abnormal)†		
DSRS	15%	20%
ES	15%	21%

* Refers to the number of A's the patients have omitted to cancel in the test.

† p value not significant for all comparisons.

tients on yearly examination (Table 2). The risk of hospitalization for HE was similar: 0.25 ± 0.9 and 0.21 ± 0.7 after DSRS and ES, respectively (p was not significant). One DSRS patient (5%) and four ES patients (21%) went back to drinking (p was not significant). Two ES patients had esophageal stenosis that were successfully dilated. Ascites developed in two DSRS patients (5%) and in ten ES patients (53%; p = 0.01). Longitudinal data of liver function tests were available on 18 ES and 19 DSRS patients at 1 year. ES did not cause changes in hepatic function (from 0.53 ± 0.14 to 0.53 ± 0.14), whereas after the DSRS procedure there was significant impairment in function at 1 year (from 0.54 ± 0.10 to 0.61 ± 0.11; p = 0.012).

Discussion

The pattern of a randomized controlled trial (RCT), comparing surgical treatment with a conservative therapy like ES, is often a complicated process requiring a choice of end-point patient selection criteria, treatment schedules, and methods of patient evaluation. Three RCTs,[14–16] in addition to our study, have been published comparing DSRS to ES. They show different approaches to these issues.

It is true that survival is the primary end-point in prospective studies. In two studies[14,16] the definition of major end-point was not specified (survival, prevention of recurrent bleeding, and maintenance of hepatic function) and no statistical method for determining trial size was used. The study of Terés et al.[15] was designed to observe an increase in survival. However the expected increase seems to be too optimistic. On the basis of the available data,[2,5,10–13] the sample requirements to show a realistic increase in survival at 5 years of the 10% is about 470 patients per group, applying standard power (80%), type I error (p < 0.05) and a two-tailed t test.[20]

Because gastroesophageal rebleeding was the most life-

threatening complication in ES, we wanted to verify if DSRS was more effective in the prevention of rebleeding than ES and evaluate if this improvement affected survival. We limited the necessary recruitment to 20 patients per group, so the trial needed only one surgical team. This is a general problem in surgical multicenter trials, as the surgeon's skill is a major factor in the outcome of the patients, especially when a technically complex operation like DSRS is used.[32] Our trial was undertaken after the team had acquired experience with 80 selective shunts, to ensure a fairly good standardization of the surgical approach and intraoperative and postoperative treatment.[5] However the patient's inclusion has not been stopped at 20 cases for each group. In fact the random choice in our trial does not prevent a problem reported in Table 1, which shows a statistical difference in etiology between the two groups. Although our experience did not attribute a prognostic role to etiology,[3,33] some studies indicated a more favorable prognosis for survival in nonalcoholics compared to alcoholics.[34,35] We then decided to increase the number of patients to be recruited either to eliminate the difference or carry out a subgroup analysis.

This study also differs from the other RCTs[14–16] because of the trial design, particularly if we consider the criteria of inclusion and exclusion and the number of patients seen and rejected. Figure 1 shows the criteria of exclusion used in our trial. Some choice in the study design caused an increase in the number of patients rejected. In fact 219 patients were admitted to our department: 22 patients were considered to have had no previous variceal bleeding and had prophylactic treatment, and 24 patients had active variceal bleeding that was treated on an emergency basis. They were excluded because they received definitive treatment (portocaval shunt, esophageal transection) or were lost or died after hemostatic session of sclerotherapy. Ninety-one of 173 patients evaluated were not eligible for inclusion in the study. Finally 13 patients were unwilling to return for regular sclerosis and 24 patients were not included because they had not undergone preoperative angiography or were sent to us with previous indications to DSRS or sclerosis. Our selection criteria excluded 77% of patients with previous variceal bleeding. This value was similar to that reported in another study (83%).[14] Other studies[15,16] excluded fewer patients (44% and 62%, respectively). However Terés et al.[15] had additional exclusions after randomization (16%) due to technical reasons.

The results of these trials[14–17] confirm previous results showing that DSRS is more effective in preventing variceal rebleeding than ES (Table 3). Gastroesophageal rebleeding after DSRS can be caused by either gastroduodenal lesions or esophageal varices. The most common cause of variceal rebleeding is shunt thrombosis, but hemorrhage can also occur when the shunt is patent because of renal vein hypertension.[36]

TABLE 3. *Two-Year Survival and Rebleeding Rate in Randomized Controlled Trials Comparing DSRS with ES*

	Warren[14]	Terés[15]	Rikkers[16]	Our Data
Two-year survival rate after DSRS	59%	71%	65%	95%
Two-year survival rate after ES	84%	68%	61%	90%
Child's C patients	43%	0	33%	0
Interval between bleeding and treatment (days)	>3–5	10–15	NR	90*
Rebleeding rate after DSRS	3%†	14%†	19%†	5%†
Rebleeding rate after ES	53%†	37%†	57%†	35%†
Variceal eradication rate	NR	46%	63%	95%
Failure to salvage rebleeders	17%	33%	47%	14%
Shunt for ES failure	31%	6%	7%	0
Global mortality rate due to rebleeding	3%	14%	27%	5%
Global mortality rate due to other causes	13%	16%	13%	10%

NR, not reported.   † p < 0.05.
* Mean value.

The problem of rebleeding in the ES group is complex and is related to factors such as the number of patients with eradicated varices, the time interval required for eradication, the incidence of sclerosis-induced mucosal ulcerations of the esophageal wall, the variceal relapses after eradication, and the incidence of hypertensive gastropathy.[37] Our incidence of rebleeding (35%) is similar to that reported by Teres et al.,[15] but lower compared to other studies.[14,16] The importance of the different techniques to explain these differences is not clear.[38,39] It is obvious that a more aggressive technique of ES, characterized by endoscopic examinations at shorter intervals or by more ES sessions, could be more effective in preventing variceal rebleeding. However this potential benefit could be cancelled by a higher risk of rebleeding from sclerosis-induced mucosal ulcerations. Our sclerotherapy regimen did not follow a rigid time protocol. Because of the unpredictability of individual responses, it is unlikely that an optimal schedule can be suitable to all patients. The different rate of rebleeding in these RCTs can not be explained only by the failure in varices eradication. The different interval between hemorrhage and beginning of the surgical or endoscopic treatment can be an important factor. Different treatment intervals have been shown to affect the incidence of variceal rebleeding.[40] Finally it is a common experience that early rebleeding occurs frequently in Child's C patients[14] and the number of Child's patients differed in all these RCTs.

Hypertensive or congestive gastropathy is a serious complication due to raised portal pressure. DSRS reduces the gastric blood flow and prevents this complication. In the ES group two patients (one had also a variceal rebleeding) had five episodes of bleeding due to gastropathy. The evident temporal relationship between the use of beta-blockers and hemorrhage control supports its therapeutic efficacy. This concomitant therapy in the ES group was used only after digestive hemorrhage. Patients with endoscopic findings of congestive gastropathy, without hemorrhage, were not prophylactically treated with beta-blocker agents.

The 2-year survival rate ranged from 59% to 95% in DSRS and from 61% to 90% in the ES group (Table 3). Why are there these great differences?

The different incidence in these studies of two prognostic factors (Child's class C patients and the interval between hemorrhage and treatment) can explain these results.[41] However a more important factor is present in the ES group: the fate of the patient after variceal rebleeding (Table 3). The fact that some studies[15,16] do not have a good therapeutic option in the case of ES failure can influence these results. The best survival rate reported in Warren's study[14] seems to be due to the large number of ES patients submitted to shunt surgery (10 of 36 patients) with a low operative mortality rate (10%). This advantage was not shown in the other studies.[15,16] This can be explained by the fact that shunt surgery compromises liver function, thus death due to liver failure becomes the major cause of death instead of hemorrhage.[33] In fact, in our study, DSRS patients had significantly decreased liver function 1 year after shunt surgery, when compared to the preoperative values (0.54 ± 0.10 *versus* 0.61 ± 0.11). The mean difference was 0.07, with a 95% confidence interval from −0.001 to 0.141. We do not know the actual clinical significance of this difference, even if statistically significant. The ES patients, on the other hand, did not show any impairment in liver function. This might be explained by the fact that patients in the ES group with alcoholic cirrhosis eliminated alcohol abuse, resulting in improved liver function.

The main complication for the shunted patient was chronic HE that little affected ES patients. In the four RCTs the incidence of HE was higher in the DSRS group than in the ES group. This trend became significant in Teres' study,[15] probably due to the modified technique used, which did not associate portal-azygos disconnection with splenorenal shunt. In fact the entity of collaterals

after DSRS seems to be an important factor influencing the incidence of HE.[42-45] In Rikkers'[16] and Warren's[14] studies chronic HE was probably attributed to the hepatic failure, and that was due to the fact that both Child's C patients[14,16] and total shunts[16] were taken in consideration. In our study,[17] the low rate of chronic HE in the DSRS group could be due to the recruitment of patients with good liver function and a shorter follow-up period than in other studies. The results of the psychometric tests and EEG confirmed the similar outcomes in the two groups of patients at 1-year follow-up evaluation.

ES was more often complicated by ascites than was DSRS. However ascites almost always responded to standard diuretic therapy. The complication rate of ES was low. In our experience,[17] only two patients had esophageal stenosis. This was the only complication that affected the quality of life in the ES group. Similar rates were shown in two RCTs[15,16] (10%), while Warren et al.[14] had no complications after ES.

In conclusion DSRS seems to be more effective than ES for preventing gastroesophageal rebleeding. However this improvement was not followed by an increase in patient survival. Indeed early survival was significantly improved by ES only when shunt surgery was available for uncontrolled bleeding.[46] Whether shunt rescue is an artefact of analysis or a valid phenomenon is not known. Further investigations are necessary to confirm if ES might be the first stage of a therapeutic regimen completed by definitive therapy, *i.e.*, surgery.

Which is the best treatment? In some patients the choice is easy because it is mandatory. ES is indicated for patients with unsuitable splenic vein or high risk of HE. DSRS is indicated for patients with gastric variceal bleeding or recurrent hemorrhage, despite sclerotherapy. In other cases the decision whether to shunt or to perform ES should be made only after having carefully studied all characteristics of every patient. To achieve good results, it is important that every time we make our decision we take patient compliance into account. The patients should be informed that shunt surgery is more traumatic but more definitive for bleeding and that sclerotherapy has a minimal impact on hepatic functional reserve but is less effective in preventing rebleeding and demands a rigorous follow-up.

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