Factors Affecting Immediate and Long-term Survival After Emergent and Elective Splanchnic-Systemic Shunts

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The course of 121 shunted cirrhotic patients, managed according to a prospective protocol over a period of 10 years, was analyzed to determine predictors of 30-day and long-term survival. Forty-five per cent of the patients underwent emergent decompression within 12 hours of active bleeding, and 34% of the shunts were selective. Logistic regression linked early mortality to bilirubin and blood-urea nitrogen (BUN) ($p = 0.001$), and long-term survival to the presence of preoperative ascites and higher levels of alkaline phosphatase ($p = 0.027$), but neither variable set was a more accurate predictor than Child Class. Emergency shunt patients had greater risk of early death, 44% *vs.* 17% for patients shunted electively ($p = 0.001$), but beyond 30 days, their Kaplan-Meier survival curves were identical. Independently, angiographic prograde portal flow was favorably associated with short-term ($p = 0.003$) but not prolonged survival. The presence of Mallory bodies, fatty metamorphosis, and acute periportal inflammation, alone or in combination, had no prognostic value. Continued postoperative alcohol ingestion jeopardized long-term survival ($p = 0.017$). Survival of nonalcoholics was enhanced by selective as opposed to total splanchnic decompression ($p = 0.009$).

PREOPERATIVE PREDICTION of an individual's likelihood of enjoying fully functional survival after a splanchnic-systemic shunt has proven to be an elusive goal. Tests of liver function were identified early on as parameters of potential import,[1] and not very long thereafter, an appreciation of the clinical signs of advanced liver disease led Child[2] to propose the peculiar combination of subjective, semiquantitative, clinical assessments and numerical laboratory values which has become the "gold standard" for classifying patients with chronic liver disease. Child's simple A, B, and C classification was intended primarily to introduce uniformity into clinical reporting and was not really designed for prognosticating. Yet, it has often been used, at least retrospectively, as a putative survival predictor, even by Child[2] himself and by his successors.[3] More contemporary investigators, usually fortified with computer technology, have built upon the components of Child's criteria, making additions,[4] substitutions,[5-7,9] and deletions,[8-11] but offering little in the way of substantive improvement in predictive accuracy.[12-14]

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Prediction of postshunt survival is more complex and compelling than ever before. The choice of shunt circumstance, emergent or elective, is now compounded by a real choice of shunt type, total or selective,[14,15] which is apt to be much more influential than the moot issue of whether a shunt should be end-to-side, side-to-side, or some other totally diverting, hemodynamic equivalent.[16,17] The need for acurate preoperative risk assessment has also been stimulated by current, widespread interest in alternative therapies,[18,19] some championed as being competitive and safer methods for definitively treating variceal bleeding.[20,21]

Materials and Methods

One hundred twenty-one consecutive patients, cared for under the aegis of a single surgeon at one institution from 1973 to 1982, comprise the basis of this report. There were 73 men and 48 women, with a mean age of 50 ± 1 years (±S.E.M.) and an age range of 18–73 years. Eighty-nine patients had alcoholic cirrhosis documented by drinking histories varying from 6 to 58 years and confirmed by liver histology in 83 instances. The remaining 32 patients had chronic nonalcoholic liver diseases.

Eighty-four patients were initially admitted to our own institution, and 37 were transferred by ambulance directly from other hospitals. Actively bleeding patients were treated on arrival with continuous intravenous infusions of vasopressin originally beginning at 0.4 u min^{-1}, but after 1980, dosage was initiated on a weight-related basis, starting at 0.005 u kg^{-1} min^{-1}. A large-bore nasogastric tube was left in the stomach and lavage carried out through it with cold tap water to determine

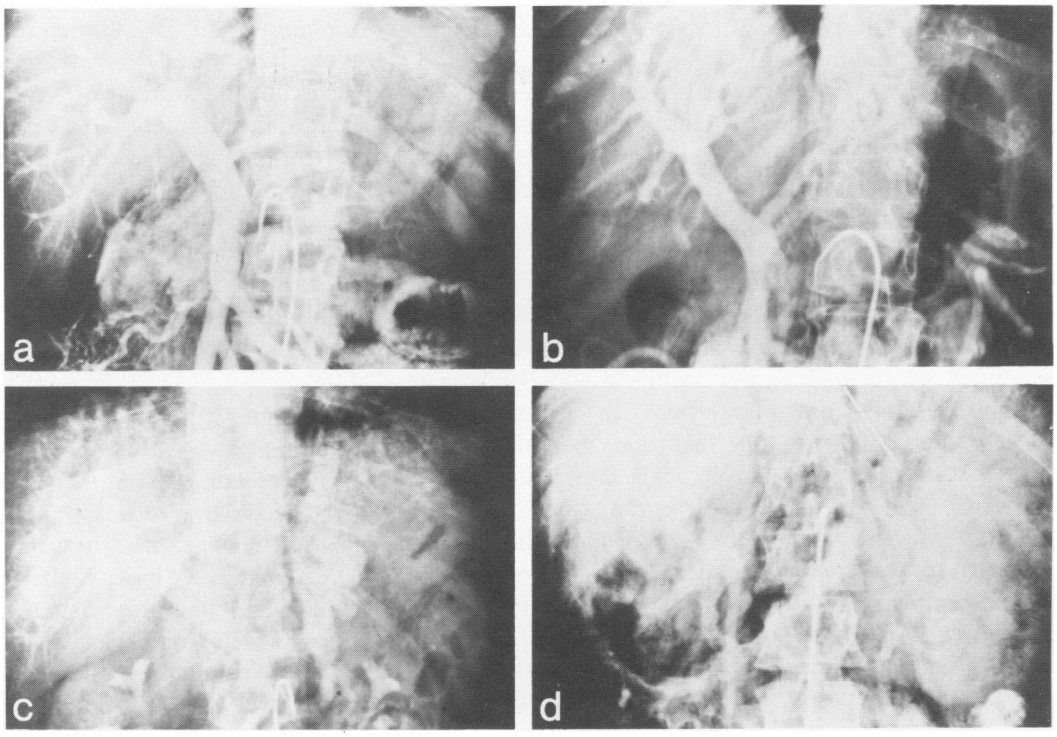

FIG. 1. Panels a–d show portal perfusion Grades 1 through 4 as seen on the venous phase of selective superior mesenteric arteriography. Grade 1 (a) shows well-preserved hepatopetal flow with each successive grade progressing towards complete hepatofugal flow as in (d).

if bleeding was stopped or continuing and to facilitate endoscopy. Persistent hemorrhage was treated by increasing the dose of vasopressin until a decrease in heart rate was noted, which most commonly occurred as the dosage approached 0.01 u kg^{-1} min^{-1}. Patients who continued to bleed despite maximal vasopressin therapy were emergently shunted, as were patients who rebled when their vasopressin infusions were gradually reduced over 72 hours.

Flexible fiberoptic esophagogastroduodenoscopy was done on all but two patients. The purpose of endoscopy was to document the presence of esophagogastric varices and to eliminate the possibility of bleeding from a gastric or duodenal ulcer. This goal was accomplished in all 119 of the endoscoped patients, but active variceal bleeding was seen in only 31 of the 78 individuals who were endoscoped at a time proximate to their bleeding.

Injection sclerotherapy was attempted four times, always in vasopressin failures. It was impossible because of overwhelming bleeding in one instance and temporarily successful in two, although both eventually required emergency shunts to control their bleeding. One patient's hemorrhaging was controlled by three separate injections, enabling him to have a successful selective shunt, at a time of election, several weeks later.

Visceral angiography, including arterial and venous phase images of selective celiac, splenic, and superior mesenteric injections, was done in 102 patients. The celiac and splenic studies were filmed in the supine position, and the patient usually was turned into the right posterior-oblique position for the superior mesenteric injection, a position that favored the demonstration of hepatopetal flow.[22] An estimate of apparent portal flow was derived from the composite venous phases of all three arterial injections, and a Portal Perfusion Grade (Fig. 1) was assigned to each patient according to the following scale, which is similar to those devised by others.[22,23]

Grade 1: Portal vein visualized with quaternary branches on superior mesenteric and splenic venous phase arteriography with minimal collateral to the azygous runoff. *Grade 2:* Portal vein visualized on both superior mesenteric and splenic injections, but branches beyond the second generation not well seen, with substantial collateral runoff. *Grade 3:* Portal vein visualized, but the venous phase of the superior mesenteric injection opacified the splenic vein or large hepatofugal collaterals. *Grade 4:* Celiac arteriography revealed retrograde portal flow or the portal vein failed to visualize on both superior mesenteric and splenic venous phase imaging.

Liver biopsies, either needle core or wedge, were obtained at the start of the intraabdominal part of the operation in 115 patients. Sections were stained with hematoxylin and eosin, Masson's trichrome, and Wilder's reticulin stains. For the purpose of this report, all sections were reviewed retrospectively by an experienced observer who had no knowledge of the pathogenesis or clinical course. Specific attention was directed to the

presence of Mallory's hyalin, fatty metamorphosis, and acute portal inflammation.

Mallory bodies were detected by scanning at 100× to identify areas containing inclusions. Microscopically, the inclusions consisted of irregular, brightly eosinophilic, hyaline aggregates within the cell cytoplasm. When an area of inclusions was identified, it was examined at 400×. The mean number of cells with Mallory inclusions per high-power field was determined, based on five adjacent high-power fields. This process was then repeated until a maximum of ten separate foci were identified by medium-power scanning and counted at 400×. The final score thus reflected the mean number of cells containing Mallory inclusions per high-power field, based on 50 high-power fields sampled from ten separately selected foci. Cases scored as zero had no medium-power foci of cells with Mallory inclusions. Since, in some instances, the number of Mallory bodies was too numerous to count, a maximum Mallory score was set at 30 per high-power field.

Fatty metamorphosis was scored based on the overall percentage of hepatocyte area occupied by cells with vacuolated cytoplasm. Scoring varied in ten per cent increments from zero per cent to 60%.

The amount of acute inflammation in the portal areas was quantitated on a zero-to-four plus scale, based only on polymorphonuclear leukocytes present in the portal areas and in the immediate periportal hepatic parenchyma. Polymorphonuclear leukocytes within sinusoids were disregarded as a nonspecific, surgically related alteration.

The current status of all patients was known as of June 30, 1984, yielding a mean follow-up interval of 4 years, with the extremes being 18 months and 11 years. At the time of review, all data were entered into a computer program using software supplied by the S.A.S. Institute, Carey, North Carolina. Short-term survival comparisons were made by unpaired, two-tailed t-tests, Chi square analysis, or Fisher's exact test. When multiple variables were to be involved, Duncan's multiple range testing[24] was used for univariate comparisons. Multivariate relationships were explored by logistic regression and discriminant analysis. Long-term survival comparisons were based on Kaplan-Meier estimates and Gehan-Wilcoxon testing.[25]

Results

Bleeding History

Forty-seven per cent of the patients had had three or more distinct bleeding episodes. The mean interval between a first variceal hemorrhage and operative treatment of the underlying splanchnic venous hypertension was 7.6 ± 2 months. Five patients actually bled repeatedly for more than 3 years before their physicians referred them for surgical treatment. Yet neither the length of delay nor the number of bleeding episodes was a significant discriminating factor for operative survival.

Vasopressin, in conjunction with gastric pH control and blood component therapy, temporarily stopped bleeding in 105 of 109 patients (96%) for at least 8 hours. Fifty-nine individuals (56%) bled again at some time during the succeeding 4 days, of whom 55 underwent emergency splanchnic decompression.

Preoperative Clinical and Laboratory Data

Independently, Child Class and, with reasonable discriminant levels, ascites, BUN, creatinine, amylase, partial thromboplastin time, bilirubin, and serum glutamic oxaloacetic transaminase (SGOT) each had significant predictive value for early survival at the 0.05 level (Table 1). To achieve a more holistic analysis, all preoperative clinical and laboratory data, with the exception of Child Class, which would have represented duplication, were subjected to stepwise logistic regression with confidence limits at 95%. The program eliminated all variables except preoperative serum bilirubin and BUN, which together were significantly ($p = 0.001$) linked to a higher likelihood of postoperative death. The prognostic potential of this two-variable set was equally significant when applied just to emergency shunts. Stepwise logistic regression retained no variables of significance for the elective shunt group alone. Discriminant analysis, using preset levels of 3.5 mg/dl for bilirubin and 30 mg/dl for BUN, correctly classified 73% of all patients and 69% of emergently shunted patients as being either operative survivors or nonsurvivors.

Thirteen more patients died during the first year, between 50 and 340 days after their operations. They had significantly ($p < 0.02$) lower admission albumin levels, higher levels of alkaline phosphatase, a greater incidence of severe ascites (6/13), and a lesser likelihood of having had alcoholism as the cause of their cirrhosis compared to those surviving beyond the first year.

Child's Class significantly ($p = 0.001$) predicted long-term survival, both in terms of who would survive beyond the first year (Table 2) and of survival throughout the course of the study (Fig. 2). The data in Table 2 indicate that Child Class was equally as accurate in predicting greater than 1-year survival as it had been at indicating which patients would not survive the first month. Child Class was also just as accurate a classifier of nonalcoholic patients as it was for those with alcoholic liver disease.

In fact the predictive accuracy of Child Class could not be significantly surpassed for either short- or long-term prognostication by logistic regression-derived variable sets (Table 3). Stepwise logistic regression analysis

TABLE 1. *Prevalence and Potential Predictive Significance of Aberrant Values by Therapeutic Group*

Variable	Per cent Prevalence				
	Elective	Emergent	Total	Selective	All
(Number patients)	(66)	(55)	(80)	(41)	(121)
Age 60	21	27	26	20	24
Female	42	36	38	44	40
Nonalcoholic	35	16	20	39	26
Admission systolic pressure < 92 mmHg	9	18	15	10	13
Small liver	14	20	18	15	17
Heart disease	26	13	19	22	20
Diabetes mellitus	17	4	6	20	11
Chronic obstructive pulmonary disease	14	11	12	12	12
Ascites	18	44	38*	15	30*
Encephalopathy	5	7	6	5	6
Muscle wasting	12	29	22	15	20
Child Class C	23	62	51*	20	40*
Na < 126 mEq/L	6	11	11	2	8
K < 3.0 mEq/L	2	4	1	5	2
BUN > 30 mg/dl	2	26*	19*	0	12*
Cr > 1.4 mg/dl	8*	13	13*	5	10*
Amylase > 100 u/dl	8	13*	14*	3	10*
PT > 1.25 × control	15	25	22	15	20
PTT > 45 sec	11	15*	11	15	12
Albumin < 2.5 g/dl	8	17	15	5	12
Bilirubin > 3.5 mg/dl	12	40*	32*	10*	25*
SGOT > 100 IU/L	9	17	17*	5	13*
Alkaline phosphatase > 120 IU/L	32	13	22	27	24

* Indicates a significant ($p < 0.05$) potential predictor of 30-day mortality.

retained only ascites and alkaline phosphatase as the preoperative parameters having a significant ($p = 0.027$) association with long-term survival. These two variables combined were equal to, but no better than, Child Class with regard to the likelihood of surviving a year or more.

Angiographically Estimated Portal Blood Flow

Twenty-seven patients were estimated to have Grade 1 portal perfusion, 41 were judged to have Grade 2, 25 were assayed as Grade 3, and nine were thought to have Grade 4 perfusion. Patients with dominant hepatofugal flow, Grades 3 and 4, had a significantly ($p = 0.003$) greater chance of dying in the postoperative period than did those with Grade 1 or 2 hepatopetal flow (50 *vs.* 21%). This was in part attributable to the bloodier (12 ± 2 *vs.* 7.3 ± 1 units, $p = 0.017$) and longer (5 h, 36 min ± 21 min *vs.* 4 h, 41 min ± 12 min, $p = 0.016$) operations that were required to effect satisfactory shunts in Perfusion Grade 3 and 4 patients. Angiographic perfusion grading was not correlated with splanchnic pressures, and there were no differences in mean Perfusion Grade between nonalcoholics and alcoholics or between patients who were to undergo emergent or elective shunts. There was, by design, a significant ($p = 0.001$) differential favoring hepatopetal flow for the selective shunt group, wherein 90% of the patients had Grade 1 or 2 flow assessments.

Shunt Circumstance and Type

The 45% of patients shunted under emergent circumstances, defined as an operation within 12 hours of

TABLE 2. *Child's Class as a Discriminator of Early Postshunt Mortality Potential for Alcoholic and Nonalcoholic Patients*

Assessment		Alcoholics	Nonalcoholics	Combined
30-Day mortality	Sensitivity	20/26 (77)	3/9 (33)	23/35 (66)
	Specificity	40/63 (63)	20/23 (87)	60/86 (70)
	Accuracy	60/89 (67)	23/32 (72)	83/121 (69)
> 1-Year survival	Sensitivity	37/57 (65)	9/16 (56)	53/73 (73)
	Specificity	3/6 (50)	6/7 (86)	6/13 (46)
	Accuracy	40/63 (63)	15/23 (65)	59/86 (69)

Numbers in ( ) are percentages; no significant differences at 95% confidence level.

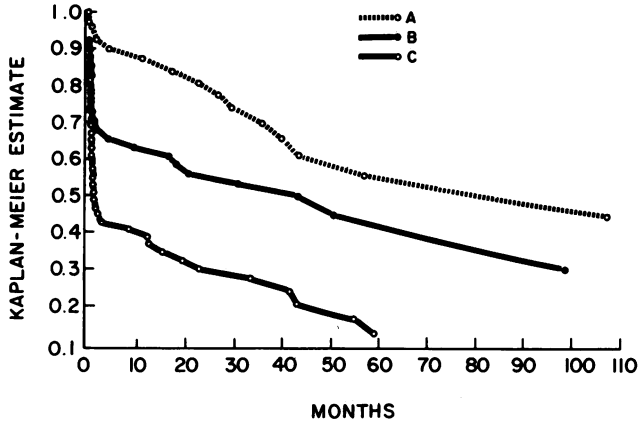

FIG. 2. Postoperative survival according to Child Class.

active bleeding, were two and one-half times more likely to die in the first 30 postoperative days than those whose shunts were performed electively (Table 4). As a consequence of the self-selective process of continuing to bleed or rebleeding, patients coming to emergency shunt differed significantly in many aspects from those destined for elective procedures (Table 5). However, of the 14 identifiable differentiating features, only four (ascites, Child Class C, BUN, and preoperative serum bilirubin) were significantly ($p < 0.05$) associated with postoperative mortality.

Figure 3 shows the identical probability of prolonged survival which was shared by emergently and electively shunted cirrhotic patients once they passed the immediate postoperative hazards. The curves are a deliberate distortion of the facts, in that they disregard operative mortality for both groups to emphasize the similarity of their subsequent potential for longevity.

Shunt Type and Liver Disease Etiology

The differences between patients who had total and selective shunts are enumerated in Table 6. Many of the same factors appear again, because almost two-thirds of the total shunts were done under emergency circumstances. The variables with a significant relationship to postoperative mortality, at the 0.05 confidence level, included amylase as well as those tabulated for emergency *vs.* elective shunts. The mortality differential between selective and total shunts reflected this bias.

TABLE 3. *Comparative Accuracy of Stepwise Logistic Regression-Derived Discriminators and Child's Class*

Assessment	Bilirubin and BUN	Ascites and Alkaline Phosphatase	Child's Class	Null Probability
30-Day mortality	88/120 (73)		83/121 (69)	0.252
> 1-Year survival		63/85 (74)	59/86 (69)	0.265

Numbers in ( ) are percentages.

TABLE 4. *Shunt Circumstance and Type, and Related Operative Mortality*

Shunt Circumstance	Shunt Type: Total N	Total Mortality (%)	Selective N	Selective Mortality (%)	All N	All Mortality (%)
Emergent	51	43	4	43	55	44*
Elective	29	28	37	8	66	17*
All	80	37	41	12	121	29

* $p = 0.001$ emergent *vs.* elective.
† $p = 0.048$ elective total *vs.* elective selective.

Liver disease etiology was not a factor in determining 30-day mortality, but long-term survival for nonalcoholic patients was related to shunt type. The significant ($p = 0.009$) advantage of selective shunting for the nonalcoholic population is illustrated in Figure 4. Comparison with the overall survival probability curve for the entire group of 121 patients (Fig. 5) shows that the apparent advantage of selective shunting was more attributable to an adverse effect of total diversion on nonalcoholic patients than to a positive protective influence afforded by selective decompression.

Liver Histology

Use of the computer permitted a comprehensive search for significant relationships potentially attributable to mean Mallory scores, periportal polymorphonuclear leukocyte infiltrates, and the extent of steatosis. One hundred four biopsies were wedge excisions and 11 were needle cores, but there did not appear to be any sampling impediment imposed by the smaller core specimens or the more superficial origin of the wedge biopsies. The livers of 13 patients had Mallory cores greater than 10, the histological sections from 15 individuals showed fatty metamorphosis exceeding 20% of the total hepatocyte area, and 22 livers had periportal polymorphonuclear infiltrates (Fig. 6). Despite these fortuitous distributions, the following negative results accrued:

1. Mean Mallory scores for alcoholic and nonalcoholic patients were not statistically distinguishable.
2. No relation could be found between SGOT and Mallory scores, periportal polys, extent of steatosis, any paired combination, or all three histologic findings together.
3. Neither Mallory scores, periportal polymorphonuclear infiltrates, nor fatty metamorphosis were significantly associated with recent alcohol abuse.
4. High Mallory scores, periportal polys, and extensive steatosis, individually or in combination, were

TABLE 5. *Significant Differences Between Emergently and Electively Shunted Patients*

Variable	Emergent Group	Elective Group	Null Probability
Alcoholic cirrhosis	84%	65%	0.024
Ascites	44%	18%	0.007
Muscle wasting	20%	6%	0.001
Child Class (1 = A, 3 = C)	2.5 ± 0.1	1.9 ± 0.1	0.001
Distinct bleeding episodes	3.3 ± 0.2	2.5 ± 0.2	0.029
Potassium	3.9 ± 0.09 mEq/L	4.1 ± 0.06 mEq	0.023
BUN	24 ± 2 mg/dl	14 ± 1 mg/dl	0.001
Prothrombin time	1.23 ± 0.02 × control	1.14 ± 0.02 × control	0.002
Albumin	3.0 ± 0.07 g/dl	3.2 ± 0.06 g/dl	0.046
Bilirubin	3.9 ± 0.5 mg/dl	2.1 ± 0.3 mg/dl	0.001
SGOT	74 ± 11 IU/L	43 ± 4 IU/L	0.004
Portal pressure	32 ± 1 mmHg	28 ± 1 mmHg	0.002
Blood transfused in O.R.	13 ± 2 units	6 ± 1 units	0.001

not more frequently observed in the livers of emergency shunt patients and portended no specially adverse prognosis for these individuals.

5. Whether analyzed on the basis of mean values or potentially facilitative discriminant levels, no significant mortality liability could be detected consequent to the presence of any or all of the assayed histologic variables.

Control of Bleeding and Shunt Patency

Excepting those with profound liver failure who bled as an agonal event, only five patients (4%) ever experienced recurrence of upper gastrointestinal bleeding. Two patients bled fatally, one within 36 hours of his shunt and the other after almost 4 years. Both shunts must be assumed to have thrombosed, as postmortem examinations were not permitted in either instance. Angiographic proof of patency was obtained for the other three patients, two of whom subsequently proved to have duodenal ulcer disease.

Shunt patency was not systemically studied, but in every instance where it was investigated, the shunt was found to be patent. Twenty-eight shunts were proven to be patent by autopsy examination, 13 were shown to be open by contrast-enhanced computerized tomographic scanning or real-time ultrasound, seven were found to be patent by angiography, and six to be patent at the time of secondary operations. In all, patency was established by one mean or another in 54 instances and thrombosis indicated by inference twice.

Cause of Death

Thirty-five patients (29%) died during the first 30 postoperative days. Fifteen had complete postmortem examinations. Liver failure accounted for 18 of the deaths. The second most common cause of death was uncontrollable intraabdominal bleeding, which occurred in eight patients. Six of the eight had undergone emergency shunts, but two developed generalized bleeding during or immediately following elective shunts. In three instances, after exhaustive use of all the usual surgical hemostatic measures and multiple infusions of platelet concentrates and fresh-frozen plasma, the abdominal cavities were tightly packed with gauze rolls and closed. The patients were returned to the surgical intensive care unit in the hope that the hemorrhage would be tamponaded for a sufficient period of time to correct the hemostatic defect with more platelets and fresh-frozen plasma. This goal was never accomplished. The abdomens became so tight that urine output ceased and ventilation became compromised. No patient survived who was reexplored under any circumstance for continuing intraabdominal hemorrhage.

Sepsis, related to indwelling monitoring catheters or to primary tracheobronchial contamination, was the cause of three deaths. Two other individuals died of cardiorespiratory failure, and two developed fatal renal failure. One instance of kidney failure appeared to be contrast-induced, and the other was acute tubular necrosis attributable to preoperative and intraoperative hypotension. Renal failure also complicated most of the deaths from liver failure and sepsis. Two patients died

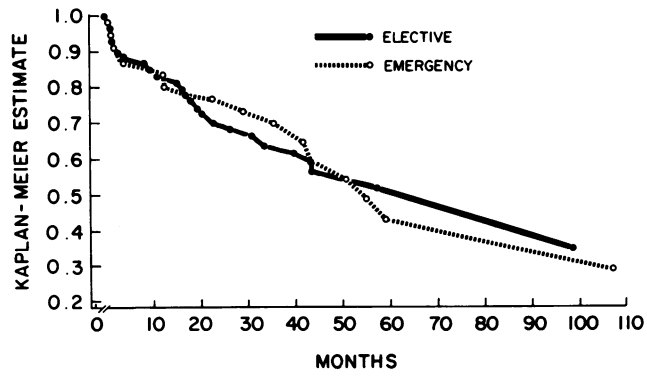

FIG. 3. Comparative survival curves for emergency and elective shunts after eliminating 30-day hospital deaths.

TABLE 6. *Significant Differences Between Totally and Selectively Shunted Patients*

Variable	Total Group	Selective Group	Null Probability
Alcoholic cirrhosis	80%	61%	0.025
Ascites	25%	15%	0.009
Child Class (1 = A, 3 = C)	2.3 ± 0.1	1.8 ± 0.1	0.002
Portal perfusion grade	2.4 ± 0.1	1.8 ± 0.1	0.001
BUN	21 ± 1 mg/dl	14 ± 1 mg/dl	0.001
Prothrombin time	1.20 ± 0.02 × control	1.14 ± 0.02 × control	0.043
Bilirubin	3.4 ± 0.4 mg/dl	1.9 ± 0.3 mg/dl	0.008
SGOT	65 ± 8 IU/L	40 ± 5 IU/L	0.029
Amylase	70 ± 8 u/dl	45 ± 5 u/dl	0.016
Blood transfused in O.R.	11 ± 1 units	6 ± 1 units	0.005

of acute hemorrhagic pancreatitis, in both instances after elective distal splenorenal shunts.

The Kaplan-Meier survival estimate for the whole group (Fig. 5) indicates that the probability of surviving for 5 years was 0.35, and for surviving 9 years, 0.24. It also shows that a high rate of mortality characterized the entire first year. With a mean follow-up of 4 yrs ± 3 mos, 39 patients had died from 50 days to 9 years after their shunt operations. Postmortem examinations were allowed in only 13 instances, but these patients' clinical courses indicated that liver failure continued to be the principal cause of death, accounting for 29 (74%) of the delayed mortalities. One patient, with an end-to-end renosplenic shunt which must have occluded, died of massive upper gastrointestinal bleeding nearly 4 years after his operation. Three of the other nine deaths were at least indirectly related to alcoholism, a self-inflicted gunshot wound of the head, a motor vehicle accident, and squamous cell carcinoma of the esophagus. The remaining deaths were caused by cerebral strokes (two), lymphoma, leukemia, lung cancer, breast cancer, and intestinal infarction.

Continued Alcohol Abuse

Figure 7 shows the significant ($p = 0.017$) detrimental effect of continued drinking on the survival probability for patients with alcoholic liver disease. Twenty-seven surviving alcoholics abstained and 27 continued to drink heavily, both groups having been followed for similar periods of time (Table 7). The sample size was too small to determine if the incidence of encephalopathy was influenced, but clear differences in the assessments of liver function were apparent.

Encephalopathy

Seven of 73 patients who survived a year or more had had encephalopathy before their operations, but only two of them were bothered by chronic encephalopathy after their shunts. In all, 12 patients (16%) developed postshunt encephalopathy. Nine were among the 44 survivors of total shunts, and three were from the group of 29 patients who lived a year or more after having a selective shunt. This twofold differential incidence, favoring selective decompression, was not statistically significant.

Two of the three patients who were encephalopathic after having had selective shunts had had chronic encephalopathy before their operations. All three individuals were studied by selective visceral arteriography and by transhepatic portography. One patient had a thrombosed portal vein. All three had developed large collaterals between the portal-mesenteric and splenic veins

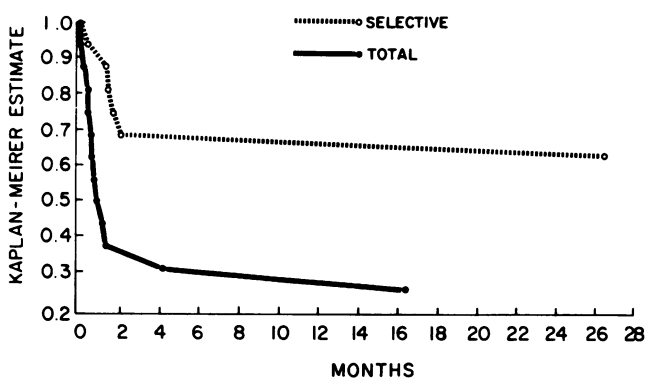

FIG. 4. Survival of patients without alcoholic liver disease after total and selective splanchnic-systemic shunts.

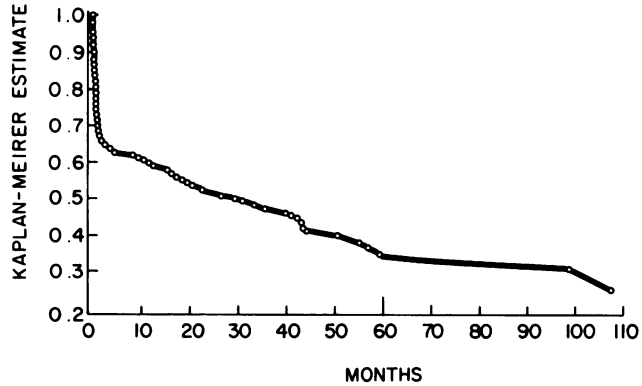

FIG. 5. Kaplan-Meier estimate of survival for the entire group of 121 patients.

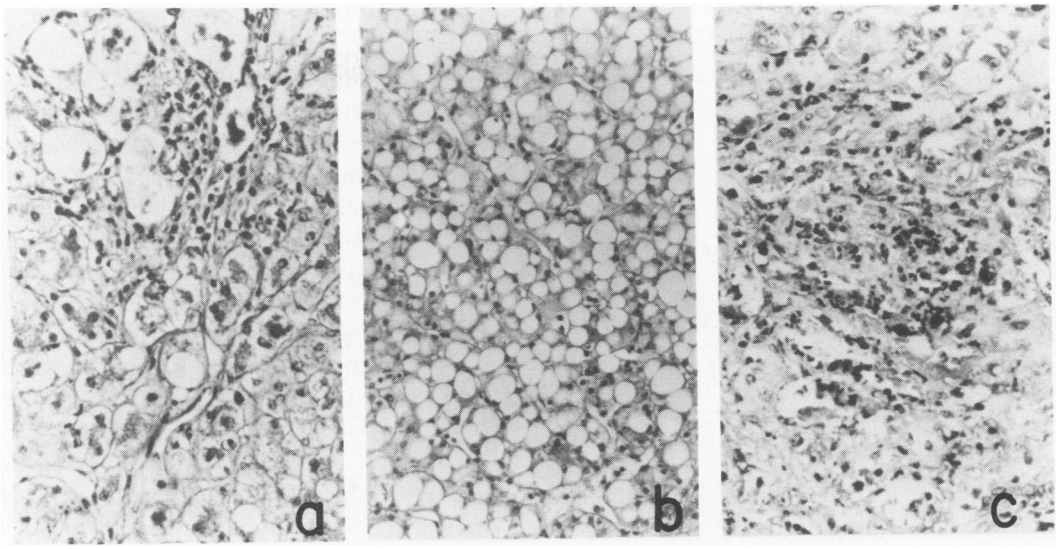

FIG. 6. Liver biopsy specimens showing (a) many Mallory inclusions; (b) fatty metamorphosis in excess of 20% of the hepatocyte area; and (c) 3+ periportal polymorphonuclear leukocyte infiltration.

(Fig. 8). In one instance, transhepatic coil and Gelfoam (Upjohn Company, Kalamazoo, MI) embolizations were done, but other collaterals took over the flow, and the patient continued to be encephalopathic. Operative obliteration was attempted in the patient whose portogram is illustrated in Figure 8. The large collaterals were dorsal and cephalad to the pancreas, approximating the course of the left gastric vein which had been ligated at its juncture with the portal vein. When these large collaterals were tied off, the mesenteric veins instantaneously became turgid, and petechiae and edema rapidly developed in the intestinal mesentery and bowel wall. The abdominal fascia could not be closed, so only the skin was approximated, in the anticipation that the mesenteric venous drainage might improve; it did not, and the patient died with an infarcted intestine. Two patients[26] with total shunts and severe chronic encephalopathy had successful conversions to selective shunts, with complete and lasting resolution of their encephalopathy.

Discussion

Several authors have indicated their discontent with the Child Class system of segregating patients.[6-9] The A, B, and C categories have been libeled as being imprecise and difficult to fit. How, for example, should a patient, who is a B in terms of easily controllable ascites, be classed when his serum bilirubin is 4.5 mg/dl, well up into the C category? This issue has been addressed by classifying patients on the basis of their worst criterion[4] or by establishing numerical categories separately for each criterion and then averaging them to yield one mean value that determines the class.[13] Child's criteria have been criticized for encompassing variables of little import, which diffuse the impact of those that are important,[8-11] and they have also been attacked on the grounds that they ignore significant discriminators.[9] Nevertheless, the Child Class system remains the benchmark against which all others must be judged.

Table 8 presents the results of 12 studies, including the present one, which were directed at defining the preoperative parameters of exceptional liability for dying in the first postoperative month or of a low probability for long-term survival. Since there was no valid way to construct composite means and to test their significance, each study was merely accorded "votes" proportional to the number of patients it encompassed. "Votes" for bromsulfophthalein sodium excretion were disregarded on the basis that the test is now rarely performed, as were grudging admissions of the value of Child Class. Bilirubin received the plurality among short-term predictors, followed closely by ascites and Child Class C. Albumin and ascites tied for first among the factors believed to indicate diminished probability of prolonged survival but, again, Child Class was close to the top.

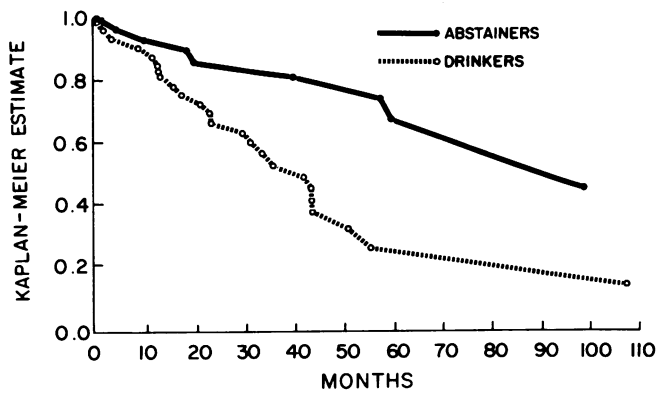

FIG. 7. Adverse influence of continued alcohol abuse on survival probability for shunted patients with alcoholic liver disease.

TABLE 7. *Effect of Continued Alcohol Abuse*

Parameter	Drinkers	Abstainers	Null Probability
Number patients	27	27	
Follow-up interval (months)	42 ± 5	53 ± 6	0.169
Number encephalopathic	6	3	0.511
Number working	13	20	0.054
Albumin (g/dl)	3.2 ± 0.1	3.6 ± 0.1	0.001
Prothrombin time (× control)	1.19 ± 0.03	1.08 ± 0.03	0.006
Bilirubin (mg/dl)	2.53 ± 0.33	1.62 ± 0.33	0.059
Alkaline phosphatase (IU/L)	148 ± 13	112 ± 13	0.059
SGOT (IU/L)	52 ± 8	34 ± 8	0.126

The limits of this popularity polling technique of analysis are highlighted by the tabulation of gender, which was mentioned by three groups of investigators, encompassing 30% of the surveyed patient population. Two of the three studies found it disadvantageous to be male,[4,9] whereas the other investigation alleged similar liability for being female.[12]

The current study corroborated the collective wisdom by documenting that stepwise logistic regression-derived variable sets had no better predictive accuracy than did Child Class. It also defined a special subset of patients who probably should not be shunted until their bilirubin and BUN abnormalities have at least partially abated. Since this prohibition is based on a predictive formula having only 73% accuracy, the admonition cannot be absolute, but it should make an emergency shunt less attractive and enhance the appeal of injection sclerotherapy for such individuals.[21,27]

Survival was clearly affected by the choice of an emergency or an elective shunt. The mortality of emergency shunting was due both to selective bias and to linking the ill effects of bleeding and operation together, but there may have been no real therapeutic alternative for these individuals. Those championing emergency shunts have pointed out that variceal bleeding is so lethal that it is unlikely that a large segment of the bleeding cirrhotic population ever becomes eligible for elective shunting.[6] No advocate of elective shunts has refuted this statement, armed with a knowledge of exactly how many bleeding cirrhotics presented to his institution and knowing what proportion of those individuals survived temporizing treatments to be shunted later at a time of election.

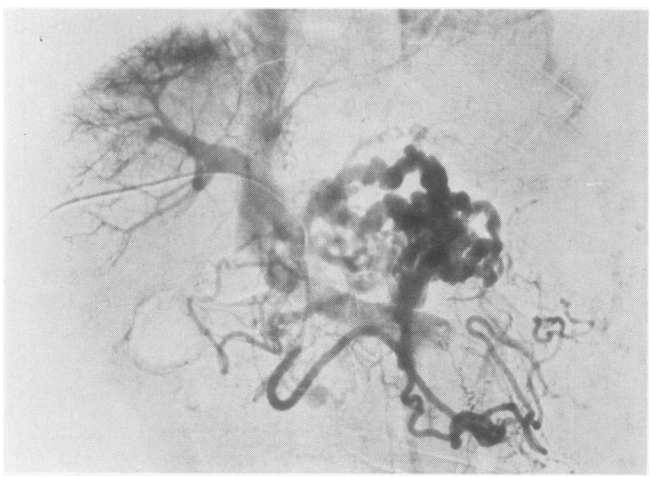

FIG. 8. Transhepatic portogram 4 months after selective distal splenorenal shunting showing failure to effect gastrosplenic—portomesenteric disconnection.

A significant survival advantage favoring selective shunting for patients with nonalcoholic cirrhosis has been demonstrated previously.[14,28] A reasonable global probability estimate for 5-year survival following any splanchnic-systemic shunt is 0.30–0.45.[3,6,8,10,14] Previous reports have noted that selectively shunted nonalcoholic patients fare much better than this, but the current study emphasizes a more negative aspect, demonstrating that nonalcoholic cirrhotic patients have a lesser probability of prolonged survival after total shunts. Since the total shunt group overlapped the emergency shunt group, this negative aspect might be taken to indicate just a poorer prognosis for emergently shunted nonalcoholic patients. However, a patient's being shunted as an emergency was unlikely to have been the important factor since there was no survival differential for nonalcoholics and alcoholics in the analysis of 30-day mortality, where the adverse impact of emergency shunting was directed.

The literature is profuse and polarized regarding the prognostic import of liver histology (Table 9). Proponents[29–35] argue that some or all of the histologic features of acute alcoholic hepatitis augur almost certain death, particularly if an emergency shunt were to be done. As a corollary, many advocate screening by preliminary liver biopsy.[30,31,33–35] The detractors[36–38] deny any import based on their own experience and point to the highly selected, small number of fatalities on which the advocates base their brief.[36,38] It has also been suggested that nonalcoholic cirrhotics were overrepresented in the better-prognosis groups cited by the proponents,[38] and that almost all positive data came from emergency shunt patients.[36]

When the data presented in Table 9 were analyzed separately for both sides, it was found that the positive studies segregated 19% (86/455) of their patients as being at high risk, and the negative studies segregated 22% (118/525), indicating statistically identical selectivity. The negative reports were based on a higher percentage of emergency shunts, 40% *vs.* 24%, and on a greater

TABLE 8. *Favored Preoperative Clinical and Laboratory Prognosticators of Postshunt Mortality*

Authors	Study Population Weighting* 30-day or Hospital	Long-term	30-day or Hospital Mortality		Diminished Long-term Survival	
Foster et al.[12]	0.13	0.09	Child C		Female gender	
Campbell et al.[13]	0.05	0.03	Child C		Child C	
Malt et al.[8]	0.13		Bilirubin, ascites			
Simert et al.[9]	0.14	0.13	Bilirubin, ascites		Albumin, male gender, bilirubin, heart disease, ascites	
Malt and Malt[10]		0.09			Albumin	
Cello et al.[4]	0.11	0.08	Child C, hematocrit		Male gender, malnutrition	
Orloff et al.[6]	0.19		Ascites, SGOT, hypokalemic alkalosis, transfusions, recent alcohol			
Kerstein and Stevenson[5]	0.05		Bilirubin, albumin, transfusions			
Warren et al.[14]		0.49			Child C, albumin, ascites, transfusions, prothrombin time, SGOT	
Talman et al.[11]	0.05		Bilirubin			
Waxman and Shoemaker[7]	0.02		Transfusions, shock			
Chandler et al.	0.13	0.09	Bilirubin and BUN, Child C		Ascites and alkaline phosphatase, Child C	
ALL	1.00	1.00	Bilirubin	0.50	Albumin	0.71
			Ascites	0.46	Ascites	0.71
			Child C	0.42	Child C	0.61
			Transfusions	0.26	Gender	0.30
			Plus others listed only once		Plus others listed only once	

* Decimals indicate the portion of all represented patients (940 for hospital mortality and 951 for long-term survival) encompassed by a specific study.

proportion of patients with alcoholic liver disease, 91% (294/324)* *vs.* 63% (230/363).† Both differences were significant ($p = 0.001$) and indicated that the negative studies actually were better positioned to detect an adverse influence of histology if emergently shunted alcoholics were to be the prime targets.

Our own results are included in the analysis. They indicate no association of Mallory bodies, acute portal inflammatory infiltrates, and extensive fatty metamorphosis with chronic alcoholism or with recent alcohol abuse. Moreover, they confirm the lack of association with elevated SGOT levels noted by Bell and his colleagues.[38]

Mallory bodies, or Mallory bodies in large numbers, were no more common in the livers of patients with alcoholic cirrhosis than they were in livers that were cirrhotic from some other cause. Although Mallory[39] initially regarded hyaline inclusions as indicative of alcoholic liver disease, they are now known to be much less specific and not even confined to the liver.[40]

The weight of evidence denigrates any prognostic value of liver histology, and certainly does not support preliminary liver biopsies. On the other hand, the positive reports cannot be dismissed on the basis that they represent only a super-selected small number of bad cases. The ambivalence in the literature is exemplified by a study linking liver histology to poor prognosis[35] and by another report, based on some of the same patients, which denies any association between alcoholic hepatitis as evidenced by liver biopsy and operative mortality.[4]

Finally, two features involved in our early mortalities deserve mention. Packing and tight closure of the abdomen to gain time to restore hemostasis, although limited to three instances, was convincingly less efficacious in the cirrhotic bleeder than it has been reported to be in trauma victims with coagulopathies.[41] Uncontrollable intraoperative bleeding remains an unsolved and unpredictable problem which was responsible for 23% of the operative mortality in this series. The two deaths from acute necrotizing pancreatitis, both following elective distal splenorenal shunts in patients whose pancreases appeared to be normal at operation, also warrant emphasis. Postoperative pancreatitis may be an underre-

* Rousselot and his colleagues, omitted for lack of data.

† DiCarlo and his co-workers, omitted for lack of data.

TABLE 9. *Liver Histology as a Prognosticator of Early* Postshunt Mortality*

Authors	Criteria Selected	Proportion of Patients Segregated	Shunt Circumstance	Mortality: Segregated Group (%)	Mortality: Remainder (%)	Null Probability
Gall and Keirle[29]	Steatosis, PMN bile stasis and alcoholic hyalin	5/63	32% Emergent	100*	24*	<0.002
Mikkelsen et al.[30]	PMN, necrosis and hyaline bodies	23/54	52% Emergent	83	23	<0.001
Rousselot et al.[36]	Hyaline necrosis	27/201	All elective	15*		
Mikkelsen[31]	PMN, necrosis and hyaline bodies involving > 5% of cells	3/28	39% Emergent	67*	4*	s.t.s.
Kanel et al.[37]	Variable degree of steatosis, PMN and Mallory bodies	20/45	All elective	80	88	n.s.
DiCarlo et al.[32]	Mallory bodies, steatosis, necrosis, PMN, Councilman bodies, pleomorphism	24/92	40% Emergent	67*	12*	<0.001
Eckhauser et al.[33]	Mallory body clusters at 100× scanning	10/124	86% Elective	90	72	<0.001
Lang et al.[34]	Hyaline bodies in 10% of hepatocytes	6/41		100*	0*	<0.001
Grendell et al.[35]	Panlobular fat	15/53	Elective	73	26	<0.01
	Alcoholic hyalin	15/53		67	29	<0.025
Bell et al.[38]	Necrosis, PMN and Mallory bodies	49/164	All emergent	51	57	n.s.
Chandler et al.	>20% steatosis	15/115	45%	60	39	n.s.
	PMN	22/115	Emergent	59	37	n.s.
	>10 Mallory bodies/ h.p.f.	13/115		46	40	n.s.

PMN = polymorphonuclear leukocyte infiltration; s.t.s. = sample too small; n.s. = not significant.

* 30 days, otherwise percentages indicate 1-year mortality.

ported hazard of selective shunting. The only other two reported cases also involved normal-appearing pancreatic glands,[42] and the risk of manipulating a normal pancreas to perform a Warren shunt may be greater than it would be if the pancreas were fibrotic, as are those of many patients with alcoholic cirrhosis.

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