A 25-Year Experience with Total Portosystemic Shunts and Reappraisal of Colon Exclusion

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The results of 43 total shunt procedures for bleeding esophageal varices performed consecutively at two community hospitals from 1956 to 1981 are reviewed. Of 15 patients with immediate preoperative bilirubins greater than 2.0 mg/dl, 11 died following shunt surgery. Of 28 other shunted patients with immediate preoperative bilirubins of less than 2.0 mg/dl, there was only one in-hospital death, thus substantiating the contention that the last preoperative serum bilirubin value is the best predictor of operative mortality. Of ten patients with appreciable ascites verified at the time of operation, there were only two survivors, and both of these had preoperative bilirubins of less than 2.0 mg/dl. Twenty-nine of the 31 patients who left the hospital were still living at least one year after operation. All 23 patients operated on prior to 1977 were available for 5-year follow-up, and there were 14 survivors (60%). Thirteen of the 31 patients (42%) manifested some degree of hepatic encephalopathy, as interpreted by necessity for protein restriction and either Neomycin or Lactulose.

Incapacitating post-shunt hepatic encephalopathy developed in one patient who required recurrent hospitalizations for episodic coma. This patient underwent a total abdominal colectomy and ileorectal anastomosis, with elimination of all episodes of encephalopathy for the subsequent 4½ years. The previous 16 cases in the literature of surgical treatment of post-shunt encephalopathy are reviewed, and the efficacy of such colon exclusion is reassessed.

AS SURGEONS AND GASTROENTEROLOGISTS face multiple new medical and surgical options in the therapy of bleeding esophageal varices, it seemed timely to review our collective experience in two community hospitals with total shunt procedures performed over the past 25 years. Results were studied with particular reference to Malt's[10] contention that the last preoperative serum bilirubin value is the best predictor of operative mortality. Patients with immediate preoperative serum bilirubin less than 2.0 mg/dl were compared to those with immediate preoperative serum bilirubin greater than 2.0 mg/dl with respect to operative mortality, long-term survival, and incidence of post-shunt hepatic encephalopathy.

In addition, the management of refractory post-shunt hepatic encephalopathy required reassessment of the role of colon exclusion in the treatment of selected patients.

Methods

The study involved a retrospective review of all shunt procedures performed at two community hospitals from 1956 to 1981 for bleeding esophageal varices due to either alcoholic or post-hepatitic cirrhosis. The diagnosis of bleeding varices was based upon upper GI series, or endoscopy, or Blakemore balloon control of bleeding, or a combination of these modalities. In recent years, flexible endoscopy was utilized more frequently for preoperative confirmation of the source of the bleeding.

During the 25 years, 43 total shunts were performed. Thirty-nine were end-to-side portacaval, one was splenorenal, and three were mesocaval interposition shunts. One of five different general surgeons performed the respective procedure.

Thirty of the 43 patients had been hospitalized at least twice for variceal bleeding; ten of these 30 patients required a Blakemore balloon for control. Thirteen patients underwent shunt surgery during their initial admission for variceal bleeding; eight of these 13 patients required a Blakemore balloon for control. The necessity for Blakemore balloon control was considered a definite indication for surgery, preferably when the patient's clinical condition sufficiently improved to permit an elective shunt. The other five patients who underwent shunt surgery during their initial admission for variceal bleeding had non-alcoholic cirrhosis. This, likewise, was considered an indication for surgery, since there was no anticipated potential for spontaneous liver improvement.

The average age of the 43 patients was 55 years and

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ranged from age 20 to age 73. There were 30 alcoholic cirrhotic and 13 non-alcoholic cirrhotic patients, all of the latter being post-hepatitic.

The diagnosis of post-shunt hepatic encephalopathy in this study was based on the necessity for protein restriction and either Neomycin or Lactulose. No routine neurologic performance tests were conducted. Likewise, blood ammonia was not documented in all patients; even among those tested, no consistent correlation with clinical status could be verified.

Results

One hundred per cent follow-up was available for the 43 patients. Two of the three patients with mesocaval interposition, as well as the patient with a conventional splenorenal shunt, died after operation. The remaining mesocaval interposition shunt clotted at one year, and the patient required conversion to an end-to-side portacaval shunt. Thirty-nine end-to-side portacaval shunts were performed primarily. Consequently, the survival figures following hospital discharge reflect immediate and long-term results of end-to-side portacaval shunts.

Operative Mortality

Twelve of the 43 patients died during hospitalization for the shunt, an overall operative mortality rate of 28%. Twenty per cent of the shunt procedures were emergent; of these nine patients, six died (67%). In the group of 34 elective-shunt patients, six died (18%). Malt[10] reported similar operative mortality results in a comparable series of 115 total shunts performed in the years 1966 to 1973.

Operative mortality for Child's[3] Class A classification was zero of 11 (0%), for Child's[3] Class B—four of 21 (19%), for Child's[3] Class C—eight of 11 (73%). The average age of the 12 operative deaths was 56 years, as compared to 55 years for the 31 operative survivors. The operative mortality for the alcoholic cirrhotic patients was ten of 30 (33%), whereas the operative mortality for the non-alcoholic cirrhotic patients was two of 13 (15%). This difference in operative mortality reflected the fact that in the Child's[3] C classification there were ten alcoholic cirrhotic patients and only one non-alcoholic cirrhotic patient.

Survival

Thirty-one patients left the hospital, and 29 were still alive one year later. Both early deaths were Child's[3] Class B patients, and both died of hepatic failure. Twenty-three patients were operated on prior to 1977, and 14 (60%) lived for 5 years or more. Malt[10] reported a 5-year survival of 42% in a comparable series of mixed alcoholic and non-alcoholic cirrhotic patients, and Reynolds'[14] lifetable analysis of 37 alcoholic Child's[3] Class A operative survivors revealed a 52% 5-year survival.

Post-Shunt Rebleed

Two Class C patients, while still in the hospital, required reoperation for rebleeding esophageal varices. Both had undergone emergent shunts, one mesocaval and one end-to-side portacaval; at reoperations, portal pressures were found to have been reduced 190 and 180 mmH_2O, respectively, from pre-shunt levels. Both patients ultimately died, and at post-mortem examinations both were found to have patent shunts. Coagulation defects may play a role in the tendency of some post-operative cirrhotic patients to rebleed from varices in spite of reduction in portal pressure to near normal levels.

Of the 31 operative survivors, ten required rehospitalization for late rebleeding during the course of this study. Three were bleeding from duodenal ulcers, and in five the source was undetermined. Two patients had recurrent bleeding from esophageal varices documented at post-mortem examinations, and each had a patent shunt, 15 months and 2 years after respective surgeries. One of these patients had resumed drinking and also had evidence of extensive esophatitis at post-mortem examination. Although infrequent, these patients with ruptured varices and patent shunts demonstrate that such rebleeding is possible even after end-to-side portacaval shunts.

Redrinking

Attempts to assess the incidence of recidivism to alcohol in the 20 alcoholic cirrhotic survivors was impossible. Known redrinking could occasionally be documented, but unadmitted redrinking was the constant enigma.

Ascites

There were 21 patients with preoperative clinical ascites. Eleven of the patients responded to diuretic therapy, as measured by the absence of any appreciable ascites at the time of surgery, and seven of these 11 patients survived. Ten of the 21 patients with preoperative clinical ascites were still found to have significant ascites at surgery, and eight of the ten died. The two survivors both had an immediate preoperative bilirubin level of less than 2.0 mg/dl.

Immediate Preoperative Serum Bilirubin

In 1979, Malt[10] reported that the best predictor of operative morality is the last preoperative serum bilirubin value. He contended that the level of bilirubin in

TABLE 1. *Twenty-eight Patients with Immediate Preoperative Bilirubin Less than 2.0 mg/dl**

Albumin 3.0–3.5 (Child's B)	9
Albumin less than 3.0 (Child's C)	7
Blakemore balloon	8
Significant ascites at surgery	2
Preoperative encephalopathy	3

* Six patients had transient bilirubin 2.0–3.0 mg/dl (Child's B); four had transient bilirubin greater than 3.0 mg/dl (Child's C).

the serum is the best single indicator of hepatic function among routine laboratory tests.

The records of all 43 patients were carefully assessed for immediate preoperative serum bilirubin levels. Patients with immediate preoperative serum bilirubin levels of less than 2.0 mg/dl were compared to the group with preoperative bilirubin levels greater than 2.0 mg/dl with reference to operative mortality, 5-year survival, and incidence of post-shunt encephalopathy. The level of 2.0 mg/dl was chosen to correlate with Child's[3] requirement for Class A classification.

There were 15 patients with preoperative serum bilirubins greater than 2.0 mg/dl. Ten of these 15 patients had preoperative bilirubins in the 2.0 mg/dl to 3.0 mg/dl range, a level which often may not be clinically detectable. At least some of these patients may have benefited from a longer period of hepatic recovery prior to the elective shunt, since 11 of the 15 patients (73%) died following surgery. Malt[10] contended that the serum bilirubin reflects not only the hepatic function, but also other metabolic insults. He inferred that any treatment endogenously lowering the bilirubin levels improves the changes of survival after shunting. Therefore, prior to proceeding with elective shunt surgery, and especially if the patient has received any recent transfusions, the serum bilirubin should be rechecked routinely.

In 28 patients with preoperative serum bilirubin of less than 2.0 mg/dl (Table 1), there were likewise ten patients who had prior transient hyperbilirubinemia ranging from 2.0 mg/dl to 8.0 mg/dl. The timing of shunt surgery in all of these patients was delayed in order to allow maximum improvement in serum bilirubin levels. Of these 28 patients, 16 patients had hypoalbuminemia, eight required a Blakemore balloon, two had significant ascites, and three had preoperative encephalopathy; yet, the operative mortality was one in 28 (3.6%) (Tables 1 and 2). As compared to the 15 patients

TABLE 2. *Results in Twenty-eight Patients with Immediate Preoperative Bilirubin Less than 2.0 mg/dl*

Operative mortality	1/28 = 3.6%
1-year survival	26/27
1- to 5-year survival	7/8
5-year survival	13/19 = 68%
Post-shunt encephalopathy	11/27 = 41%

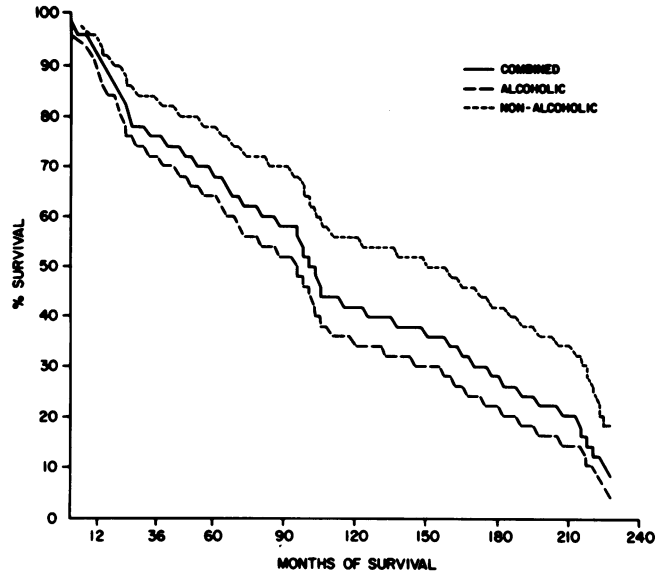

FIG. 1. Life table analysis of 27 patients with immediate preoperative bilirubin < 2.0 mg/dl.

with immediate preoperative bilirubin greater than 2.0 mg/dl, this difference in operative mortality is highly significant ($p < 0.0001$).

Reynolds[14] reported an operative mortality in 40 Class A alcoholic patients as 7.5%, and in Johnston's[9] personal series of 54 end-to-side portacaval shunts, only 8% of which were emergencies, the operative mortality was 4%.

Of the four operative survivors with immediate preoperative serum bilirubins greater than 2.0 mg/dl, only one lived 5 years. This was in contrast to life table analysis of the 27 operative survivors with bilirubin less than 2.0 mg/dl, as shown in Figure 1. Projected survival at 5 years was 68%, at 10 years was 43%, and at 15 years was still almost 30%. Of these patients with preoperative bilirubin less than 2.0 mg/dl, there was no statistical difference in survival between non-alcoholic and alcoholic cirrhotic patients. Thus, a low immediate preoperative serum bilirubin level appears to be a favorable indicator of long-term survival as well as low operative mortality. However, no such correlation could be established between the level of preoperative serum bilirubin and the incidence of post-shunt encephalopathy.

Post-Shunt Hepatic Encephalopathy

Post-shunt encephalopathy is a manifestation of portal-systemic encephalopathy as described by Sherlock.[16] Warren[19] defines this as neuropsychiatric abnormalities, occuring in a patient with stable liver disease which usually clears rapidly with withdrawal of all protein from the diet and the institution of ancillary measures such as oral catharsis, multiple enemas, and the use of non-

absorbable antibiotics to reduce the bacterial flora of the gastrointestinal tract.

The etiology of post-shunt hepatic encephalopathy remains obscure, but Hanna[6] describes several factors that seem to be involved. First, a toxic substance or substances from the gastrointestinal tract (possibly ammonia, among others) bypass their normal metabolic site in the liver and reach the brain at high concentrations, where they produce cerebral dysfunction. Second, total portosystemic shunts divert portal venous flow away from the liver, contributing to hepatocellular damage and hence impaired metabolism of ammonia and other toxins that are delivered to the liver through the hepatic artery. Third, total portosystemic shunts decompress the splanchnic bed, which may increase ammonia or other toxin absorption from the intestine. The major source of these nitrogenous substances is the colon, where bacterial biotransformation of protein occurs.[20]

Post-shunt hepatic encephalopathy developed in 13 of the 31 operative survivors (42%). Encephalopathy was considered mild in one patient who never required hospitalization, moderate in six patients, each of whom required at least one hospitalization, and severe in six patients who required multiple hospitalizations. At least some degree of pre-shunt encephalopathy had existed in four of the 13 patients. Three patients became encephalopathic within the first year, four within 2 years, two within 3 years, and four over 5 years after respective shunts. However, of the 23 patients followed over 5 years, the incidence of post-shunt encephalopathy remained the same (43%).

There was no appreciable difference in the instance of post-shunt encephalpathy in the alcoholic and nonalcoholic survivors. Of the 20 alcoholic survivors, eight (40%) developed encephalopathy. Of the 11 non-alcoholic survivors, five (45%) developed encephalopathy.

The average age of 13 patients with post-shunt encephalopathy was 55 years, compared to 54 years in 18 patients who did not manifest post-shunt encephalopathy. However, only two of nine patients under 50 years of age developed post-shunt encephalopathy, as compared to 11 of 22 over age 50. Johnston[9] found that encephalopathy was unusual in his patients under age 50, but over 50% of those over age 50 at the time of operation were affected. His subsequent policy has been to reserve shunt surgery for Child's[3] Class A patients who are under the age of 50 and without a past history of encephalopathy. Reynolds[14] limited shunt procedures to those under age 60, and in 37 Class A alcoholic survivors, he reported development of post-shunt encephalopathy in 48%.

Severe post-shunt hepatic encephalopathy can post a difficult management dilemma, as the medical history of one such patient will reveal. Within two months of undergoing an end-to-side portacaval shunt for variceal hemorrhage in 1973, this alcoholic cirrhotic patient, then 53 years of age, began to manifest mild hepatic encephalopathy. Before operation, he had been noted to be tremulous, but never confused or in coma. In May 1976, the patient underwent vagotomy and antrectomy as well as Hill repair of a hiatal hernia for blood loss anemia attributed to duodenal ulcer and acute esophagitis.

TABLE 3. *Walker's[18] Contraindications to Colon Exclusion*

Contraindications	Current Patient with Subtotal Colectomy
Bilirubin > 2 mg/dl	1.5 mg/dl
Albumin < 3 g/dl	3.6 g/dl
Protime > 4 seconds	14.5 seconds (control—11.0 seconds)
Ascites unresponsive to salt restriction	None
Active progressive cirrhosis	Stable cirrhosis

This operation alleviated intestinal bleeding, but between November 1976 and April 1977, five additional hospitalizations were required for more frequent post-shunt encephalopathy manifested by either semicoma or coma.

The patient had not been drinking since his original portacaval shunt and had been adhering as strictly as possible to a 40-g protein diet and as much as 6 g Neomycin by mouth (po) daily plus Lactulose, but he would sometimes go to bed quite well at night, only to be unarousable the next morning. Fear of aspiration was paramount when he was admitted in frank coma in April 1977. Consequently, after review of the surgical literature and personal communication with Resnick,[12] colon exclusion was offered to the patient. Preoperative liver function tests were acceptable by Walker's[18] criteria (Table 3). After reversing the current episode of encephalopathy, a subtotal colectomy with anastomosis of the ileum to the low rectum was performed on May 4, 1977. In the succeeding 4½ years, the patient did not experience any further episodes of hepatic coma. He did not require either dietary protein restriction or Neomycin or Lactulose. He did experience approximately six to eight semi-loose bowel movements per day. At one point, an attempt to reduce stool frequency with Lomotil produced exacerbation of confusion; subsequently, no antidiarrheal medications were prescribed.

In 1981, the patient again began drinking alcohol and ultimately died with jaundice, delirium tremens, and progressive hepatic failure on January 22, 1982.

Colon Exclusion

In 1960, Atkinson[1] reported treatment of a case of recurrent hepatic coma by total abdominal colectomy

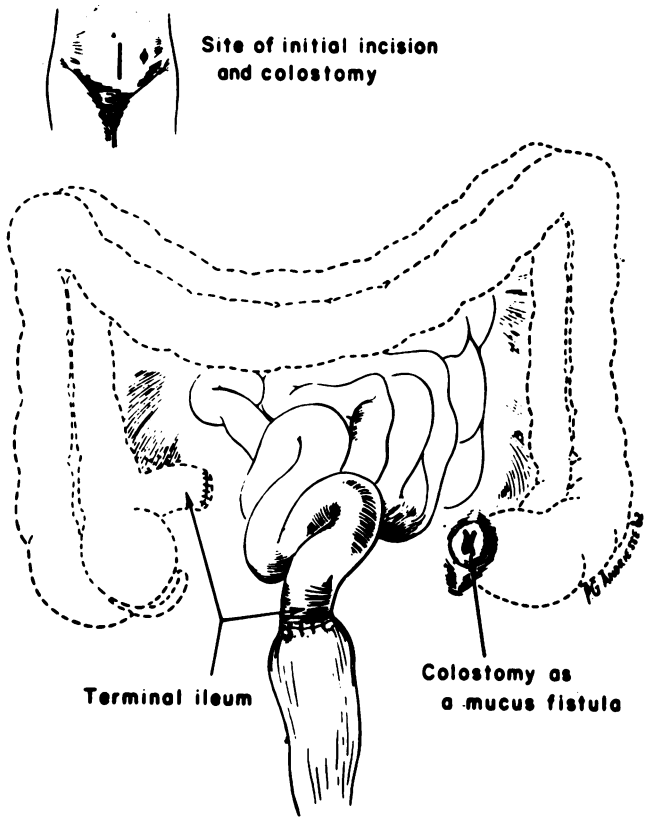

FIG. 2. McDermott's[11] procedure for defunction of the colon.

with ileorectal anastomosis. Subsequently in 1962, McDermott[11] utilized bypass of the colon in the treatment of hepatic encephalopathy in three patients; henceforth, in the United States, this operation was known eponymically as "The McDermott Procedure" (Figure 2). Subsequently, his procedure was modified to involve exteriorization of the distal ileum along with the sigmoid mucous fistula to allow cross irrigation of the residual colon with enemas.[7]

What has been the subsequent experience with colon exclusion by removal or bypass? Does this procedure still have a place in the management of selected patients with significant post-shunt hepatic encephalopathy?

From 1962 to 1968 there were reports of eight additional colon bypass cases[4,5,7,15,18] as well as two abdominal colectomies[8,17] for post-shunt encephalopathy. All 12 cases with operative survivors reported improvement in clinical manifestations of hepatic encephalopathy, but the maximum follow-up was limited to 23 months in a single patient (Table 4).

It was Walker's[18] conclusion that the operation, although not a panacea for the management of liver failure in terminal cirrhosis, did have a place in selected patients with relatively good hepatic function and disabling encephalopathy. He warned that the operation may not be advisable if (1) hyperbilirubinemia exceeded 2.0 mg/dl, (2) the serum albumin level was below 3 g/dl, (3) fluid retention was unresponsive to dietary salt restriction alone, (4) the prothrombin time was increased by more than 4 seconds, or (5) there was active cirrhosis with rapidly deteriorating liver function (Table 3).

Walker[18] felt that the beneficial effect of the operation may be likened to extreme purgation without its ill effect. The same connotation was expressed in Atkinson's[1] original report when he concluded that the ultimate prognosis may depend largely upon whether, and to what extent, the bacterial population of the small intestine increases in the future. To reduce potential for this likelihood, Dudrick[5] performed his bypass procedure with preservation of the ileocecal valve in order to minimize colonization of the small bowel with ammonia-forming organisms from the distal rectum.

Willson[20] described a patient with non-cirrhotic portal hypertension who had an end-to-side portacaval shunt in 1958 and subsequent right colectomy, then subtotal colectomy and eventual ileostomy in 1976 for management of recurrent encephalopathic episodes. There was improvement in encephalopathy with each of the operative procedures, especially the initial right colectomy in 1966 and the ileostomy in 1976. Because of ileostomy dysfunction, the patient in 1978 underwent reanastomosis of the ileum to 20 cm of rectosigmoid, and this resulted in recurrence of refractory encephalopathy. Consequently, a second ileostomy was performed in January 1980, and the patient again had a favorable clinical response and marked improvement in psychomotor testing. It is postulated that the effect of any colon exclusion procedure is related to alteration in bacterial flora or gut transit time.[13,20] Thus, the 20-cm residual length of rectosigmoid in this particular patient may have been the critical determinant.

On the other hand, Cameron[2] reported a case with severe chronic portal-systemic encephalopathy treated successfully by resection of only the right colon with anastomosis of the ileum to the distal transverse colon. Follow-up was limited to 15 months.

In 1968, Resnick[13] and the Boston Inter-Hospital Liver Group dramatically dampened any incipient enthusiasm for colon exclusion. Resnick reported a controlled trial in which 19 patients underwent a colon bypass procedure and 19 were treated medically by random selection. Both long-term mortality and relief of encephalopathy were similar for the operated and medically treated series, and there was a 26% operative mortality in the surgical group. Subsequent reference[6] to the treatment of post-shunt hepatic encephalopathy dismissed colon exclusion by the statement that in a controlled study of Resnick's, no real benefit was shown.

A careful perusal of Resnick's[13] report indicates that most patients did not meet Walker's[18] criteria for op-

TABLE 4. *Surgical Treatment of Post-Shunt Hepatic Encephalopathy: Review of Case Reports*

Reference/Year	Years of Age	Type of Cirrhosis	Previous Shunt	Procedure	Follow-up
Atkinson[1]/1960	68	Not Stated	Side-to-side portacaval (August 1954)	Colectomy with ileorectal anastomosis (June 1959)	6 months
McDermott[11]/1962	56	Postnecrotic	Double portacaval (May 1959)	Exclusion of colon with ileosigmoidostomy (September 1961)	1 month (died)
	52	Not known	Double portacaval (July 1959)	Exclusion of colon with ileostomy (October 1961)	6 months
	47	Alcoholic	Splenorenal (1950)	Exclusion of colon with ileosigmoidostomy (November 1961)	1 month
Dienst[4]/1964	n/g	Not given	Splenorenal (1957)	Exclusion of colon with ileorectal anastomosis (March 1963)	6 months
Johnston[8]/1965	55	Postnecrotic	End-to-side portacaval (February 1963)	Colectomy with ileorectal anastomosis (July 1964)	6 months (died)
Singer[17]/1965	66	Alcoholic	End-to-side portacaval (October 1963)	Colectomy with ileorectal anastomosis (February 1965)	8 months
Walker[18]/1965	45	Cryptogenic	Type of and date of shunt not specified	Exclusion of colon with ileosigmoidostomy	18 months
	47	Cryptogenic	Type of and date of shunt not specified	Exclusion of colon with ileosigmoidostomy	Died
	51	Alcoholic	Type of and date of shunt not specified	Exclusion of colon with ileosigmoidostomy	Died
Hume[7]/1966	65	Not known	Portacaval shunt (February 1960)	Exclusion of colon with ileosigmoidostomy (September 1963)	23 months
Sher[15]/1966	43	Postnecrotic	Side-to-side portacaval (December 1964)	Exclusion of colon with ileorectal anastomosis (April 1963)	8 months
	61	Alcoholic	End-to-side portacaval (1964)	Exclusion of colon with ileorectal anastomosis (July 1965)	6 months
Dudrick[5]/1968	46	Postnecrotic	End-to-side portacaval (1958)	Exclusion of colon with cecorectosigmoid anastomosis (December 1966)	4 months
Cameron[2]/1968	56	Alcoholic	End-to-side portacaval (1965)	Right colectomy with ileum to distal transverse colon (April 1966)	15 months
Willson[20]/1981	62	Non-cirrhotic portal hypertension	End-to-side portacaval (1958)	Right hemicolectomy (1966) Subtotal colectomy (1974) Ileostomy (1976) Ileosigmoidostomy (1978) Ileostomy (1980)	4 years 2 years 1 year
Talman/1982	57	Alcoholic	End-to-side portacaval (October 1973)	Colectomy with ileorectal anastomosis (April 1977)	4.5 years

erability and that many were chronically incapacitated end stage cirrhotics with Child's[3] Class B or C liver function. Most significantly, some, if not most, of Resnick's patients had never had a shunt procedure. Consequently, included in his report were patients who underwent the colon bypass not for post-shunt encephalopathy, but for encephalopathy that was a manifestation of progressive liver failure.

In spite of these shortcomings, the effect of Resnick's study was to deem the procedure ineffective. During the next 14 years, there was only one report[20] of colon exclusion by either bypass or removal for the treatment of post-shunt hepatic encephalopathy.

An often overlooked aspect of Resnick's[13] paper was its own conclusion that the results to date neither support general use of the operation nor assignment of colon bypass to the limbo of discarded surgical procedures. The same advice would still apply today, along with the admonition that colon exclusion must be reserved for those with relatively good hepatic function and severe episodic post-shunt encephalopathy.

Conclusion

Review of a 25-year community hospital experience with total portosystemic shunts provides a benchmark with which to compare current and future alternatives in the treatment of bleeding esophageal varices. A subset has been developed consisting of patients with immediate preoperative serum bilirubin levels of less than 2.0 mg/dl. In 28 such patients, the operative mortality was 3.6%, and the 5-year survival was 68%. The incidence of post-shunt hepatic encephalopathy was 41%, of which half were severe.

Thus, in patients undergoing total shunts for bleeding esophageal varices, immediate preoperative serum bilirubin less than 2.0 mg/dl was a very favorable predictor of low operative mortality, and it also appeared to be a favorable indicator of increased 5-year survival. However, there was no correlation between low preoperative serum bilirubin and the incidence of post-shunt hepatic encephalopathy. For those patients who do develop severe episodic post-shunt encephalopathy, colon exclusion by either bypass or removal may offer effective amelioration.

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Discussion

DR. GARDNER W. SMITH (Baltimore, Maryland): I recently had the opportunity to review a manuscript reporting 12 cases of colonic resection for portal-systemic encephalopathy. The conclusion of those authors was precisely the same as that of Dr. Talman and his associates. The operative mortality is high, varying from 26% to 50%, and the survival of these patients is not prolonged. However, in otherwise good-risk patients with incapacitating encephalopathy, the quality of life clearly is improved. The operation simply is contraindicated in patients with end-stage liver disease.

There are several aspects of the total shunt experience which deserve comment, and some of them I have drawn from the opportunity to read the manuscript. I think Dr. Talman did not have a chance to present all of his data.

In the first place, there was a 32% incidence of recurrent bleeding among the survivors in this group and a 6% incidence of variceal hemorrhage in patients with patent shunts. Both of those figures are a little unusual. The usual figures for a portacaval shunt would be about 16% and 3%, respectively. The two patients who bled from varices with a patent shunt both bled in the immediate postoperative phase after emergency operation, and one of them actually had an interposition mesocaval shunt. I guess one can speculate about the possibility of compartmentalization as an explanation for this phenomenon.

He also commented on the better long-term survival for nonalcoholic as compared to alcoholic patients, and it is interesting that this is consistent with the same observation that has been made by both Dr. Warren and Dr. Zeppa after selective shunts.

Perhaps the most striking aspect of this report is the preponderance of end-to-side portacaval shunts. This probably reflects the fact that the series goes back to 1956, but it may also represent some resurgence of enthusiasm for Eck's original fistula. In those situations where a total shunt is appropriate, the end-to-side portacaval shunt actually is comparable to the more modern interposition mesocaval one as regards operative mortality, the risk of encephalopathy, and long-term survival. It has the singular advantage of a much lower risk of late shunt thrombosis.

I do have a few questions for Dr. Talman. I note that 40% of the patients who developed post-shunt encephalopathy did so more than 5 years after their operation. I would ask, therefore, whether it is possible that this problem, at least in part, is related simply to a progression of the underlying cirrhosis, rather than exclusively being a complication of the shunt.

Secondly, Dr. Talman noted the correlation between preoperative hyperbilirubinemia and both operative risk and long-term survival. Another factor which others have commented on as regards operative risk is the presence of active liver cell necrosis, as manifested by increased serum transaminases. I would ask him whether that was also noted to be a factor in this particular series.

And finally, he and his associates have accomplished these total shunts with an overall operative mortality for Class A and B patients of about 13% and an actual 5-year survival of 60%. Given these good results, I wonder if the end-to-side portacaval shunt is still their operation of choice, or do they occasionally use a selective shunt as well?

DR. JULIUS A. MACKIE, JR. (Philadelphia, Pennsylvania): The authors raised several questions, one of which concerned whether or not these operations still have a place in patients with post-shunt encephalopathy.

At the time the exclusion techniques were developed in the 1960s, ammonia intoxication of the central nervous system was the accepted explanation for hepatic encephalopathy, occurring in patients with severe liver disease and after shunts. Although elevated serum ammonia is involved in the pathogenesis of encephalopathy, we now know that there are other factors included in the problem. Therefore,