

Peptic Ulcer Surgery in Patients with Liver Cirrhosis

Thomas Lehnert, M.D., and Christian Herfarth, M.D., F.A.C.S. (Hon.)

From the Department of Surgery, University of Heidelberg, Heidelberg, Germany

Objective

This study identified risk factors of surgical treatment for gastroduodenal ulcer disease in patients with liver cirrhosis.

Summary Background Data

Liver cirrhosis is frequently associated with complicated peptic ulcer disease. Surgery in liver cirrhotics has a high mortality and morbidity especially when abdominal operations are performed.

Methods

Sixty-nine patients undergoing surgery for gastroduodenal ulcer disease between 1972 and 1991 were studied, retrospectively.

Results

Ninety percent of patients required emergency surgery for bleeding ulcer ($n = 45$) or perforation ($n = 17$). Mortality was 29% for elective patients ($n = 7$), 35% for patients with perforation and 64% for patients with bleeding. Overall mortality of 69 patients was 54%. Only 15 of 69 patients (22%) had an uncomplicated postoperative course. Postoperative bleeding, septic complications, and renal failure were the most frequent postoperative complications. Bleeding and multiple organ failure were the leading causes of death in 70% of patients. A univariate analysis determined preoperative hemoglobin < 12 g/L ($p < 0.05$), systolic blood pressure < 100 mm Hg ($p < 0.025$), prothrombin time $< 60\%$ ($p < 0.05$) and the presence of portal hypertension ($p < 0.01$) as prognostic factors. No significant correlation with survival could be established for excretory liver function (serum bilirubin) and partial thromboplastin time.

Conclusions

To improve treatment results it is recommended (1) to substitute blood products (particularly coagulation factors) early and in sufficient quantities, (2) to diligently search for and to treat septic foci and administer antibiotics in a nonrestrictive manner, and (3) to restrict the operative procedure to the treatment required for control of the ulcer complication.

Since the mid-sixties a decline in the number of operations performed for gastroduodenal ulcer disease has

been reported from several centers. This decline concerns mainly elective surgical procedures while the number of operations performed for peptic ulcer emergencies such as perforation or bleeding has remained fairly constant. Even with the reduced number of elective operations our own experience indicates that the majority of elective procedures is now performed for complications

Address reprint requests to Thomas Lehnert, M.D., Department of Surgery, University of Heidelberg, Im Neuenheimer Feld 110, D-6900 Heidelberg, Germany.

Accepted for publication September 21, 1992.

of gastroduodenal ulcer such as gastric outlet obstruction or penetrating ulcer. Uncomplicated refractory ulcers have become rare in our current surgical practice.

Not surprisingly emergency operations and operations for complicated peptic ulcer disease are burdened with a higher postoperative complication rate and mortality. Treatment results will only be improved if risk factors of surgical treatment are recognized and amenable to treatment.

Operative procedures in patients with liver cirrhosis are known to have a particularly high postoperative mortality and morbidity. However, only limited information is available on the particular clinical problems associated with surgical treatment of peptic ulcer disease in patients with liver cirrhosis. This study evaluated clinical features and operative treatment as well as postoperative mortality and morbidity in these patients and identified an approach by which treatment results could possibly be improved.

PATIENTS AND METHODS

Clinical data of 69 patients with liver cirrhosis, surgically treated at the Department of Surgery, University of Heidelberg, between 1972–1991 for acute or chronic gastroduodenal ulcer disease, were recorded. Patients were included in the study only when liver cirrhosis had been documented histologically or when intraoperative findings were consistent with cirrhosis of the liver. Patients with gastroduodenal erosions without demonstrable ulcer, Mallory-Weis syndrome or gastric tumors were excluded from the study.

All except two patients required surgical treatment for a complication of ulcer disease (bleeding, perforation, gastric outlet obstruction, ulcer penetration). Sixty-two patients required emergency operations for bleeding or perforation and only seven operations were performed electively. Two of the elective operations were performed for suspected gastric cancer which could not be confirmed intraoperatively and on histologic examination of the resected specimens. No patient underwent operation for refractory but otherwise uncomplicated ulcer disease.

Postoperative mortality was determined including all deaths before discharge of the patient from the hospital. Deaths occurring more than 30 days after the operation or after the patient had been referred to other institutions for the treatment of concurrent medical problems are therefore included.

Statistical Analysis

Nonparametric data were tested by chi-square analysis with Yates' continuity correction of 2×2 tables. When

Table 1. ULCER LOCATION IN PATIENTS WITH LIVER CIRRHOSIS

| | n | Elective | Bleeding | Perforation |
|---------------|---------|----------|----------|-------------|
| Stomach | 22 (15) | 3 (2) | 17 (12) | 2 (1) |
| Pylorus | 3 (1) | 0 | 0 | 3 (1) |
| Duodenum | 32 (18) | 2 (0) | 20 (14) | 10 (4) |
| Anastomosis | 5 (1) | 1 (0) | 2 (1) | 2 (0) |
| V + D | 7 (2) | 1 (0) | 6 (2) | 0 |
| Sum | 69 (37) | 7 (2) | 45 (29) | 17 (6) |
| Mortality (%) | 53.6 | 28.6 | 64.4 | 35.3 |

Postoperative deaths in brackets.

V + D = stomach and duodenum.

the expected cell frequency was less than 5, Fisher's exact test was used. Parametric data were analyzed by the two-sided Student's *t*-test. Differences were considered significant at $p < 0.05$.

RESULTS

Age and Sex

The average age of all patients ($n = 69$) was 58.3 ± 13.7 years ($\bar{x} \pm SD$; men 58.1 ± 12.4 years, $n = 49$; women 58.9 ± 16.8 years, $n = 20$). The average age in patients with bleeding ulcer ($n = 45$) was 57.9 ± 13.9 years, in patients with perforated ulcer ($n = 17$) 61.5 ± 13.4 years and in elective patients ($n = 7$) 53.4 ± 13.0 years.

History of Ulcer Disease

A history of ulcer disease could be recorded in 35 of 69 patients (51%). Five of seven patients (71%) operated on electively had a history of peptic ulcer whereas the other two elective patients underwent operation for suspected malignancy, but were found to have benign gastric ulcer. Similarly 10 of 17 patients (59%) with perforated peptic ulcer had a history of peptic ulcer, whereas only 20 of 45 patients (44%) with bleeding ulcer had a history of ulcer disease. Less than half ($n = 25$) of the 62 patients (40%) receiving emergency surgical treatment had a history of ulcer disease exceeding more than 6 months.

Previous Surgical Procedures

Operations to treat ulcer disease had been performed at an earlier date in 11 of 69 patients (16%). In six patients, a Billroth-II partial gastrectomy had been performed some 3 to 26 years previously. In three patients, a perforated ulcer had been oversewn 3, 8, and 21 years

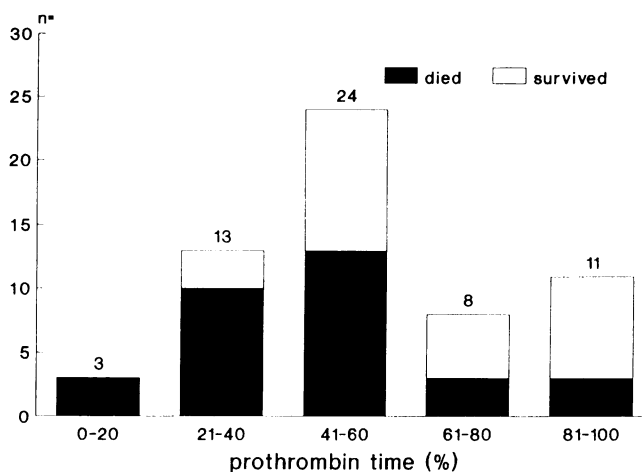


Figure 1. Postoperative mortality and prothrombin time. Only 11 patients had normal preoperative values (80–100%). At prothrombin time less than 60% mortality was 65% (26/40) compared with 32% (6/19) when prothrombin time was greater than 60% ($p < 0.05$).

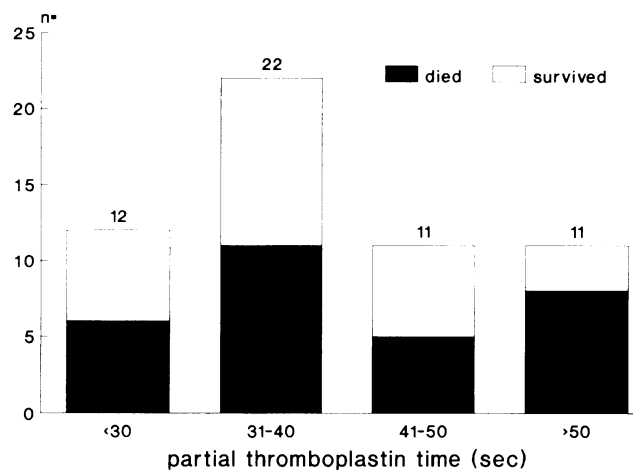


Figure 2. Postoperative mortality and partial thromboplastin time (PTT). Thirty four of 56 evaluable patients had normal preoperative values (<40 sec). Mortality of patients with prolonged PTT was 59% compared with 50% in patients with normal PTT (n.s.).

prior, respectively. One patient had gastrotomy for a bleeding ulcer 8 years previously. In the last patient, proximal gastric vagotomy with a satisfactory Burge-test had been performed. Five years later a Billroth-I gastrectomy was added because of recurrent ulcer. Another 6 years later, histologic examination of endoscopic biopsies from a giant anastomotic ulcer revealed liver tissue. To treat this recurrent penetrating ulcer, the gastroduodenostomy was converted to a gastrojejunostomy. Three years before this operation, liver cirrhosis was diagnosed.

Ulcer Location

The majority of ulcers (35/69; 51%) were located in the pyloric channel or in the duodenum (Table 1). Almost all gastric ulcers required operative treatment for bleeding (17/22; 77%). Perforated ulcers were mostly located in the pylorus or duodenum (13/17; 76%). One third of all gastric ulcers were located in the prepyloric antrum and 5 of 22 gastric ulcers (23%) were in the subcardial region. Five ulcers were anastomotic ulcers. Ten patients had penetrating ulcers and four patients had gastric outlet obstruction.

History of Cirrhosis

A diagnosis of liver cirrhosis was known or suspected preoperatively in 46 of 69 patients (67%). In the other 23 patients, liver cirrhosis was diagnosed only at the time of the operation. The cause of liver cirrhosis was alcohol abuse in 32 patients and hepatitis in 15 patients. In 22 patients, no cause could be elicited.

Liver Function

Since most patients were operated as emergencies or were referred from other hospitals evaluable data were not available for all patients. Synthetic liver function was impaired in 48 of 59 patients when determined by prothrombin time (81%, Fig. 1) and in 22 of 56 patients when determined by partial thromboplastin time (39%, Fig. 2). Excretory liver function as determined by preoperative serum bilirubin levels was impaired in 33 of 47 patients (70%, Fig. 3).

Portal Hypertension

A diagnosis of portal hypertension was made when ascites or esophageal or fundic varices were demonstra-

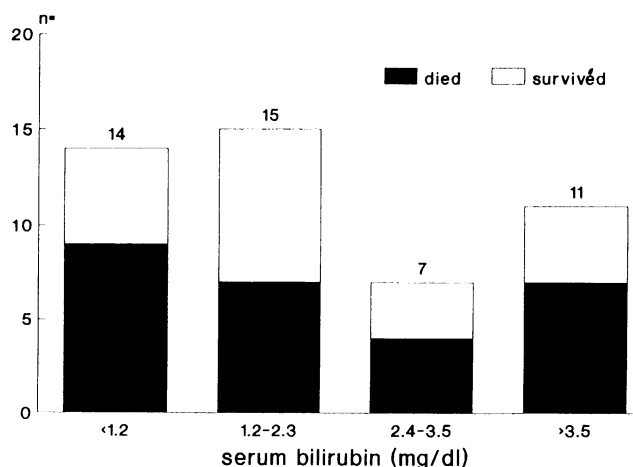


Figure 3. Postoperative mortality and serum bilirubin. No significant correlation can be established between postoperative mortality and preoperative serum bilirubin.

Table 2. PREEXISTING DISEASES IN ULCER PATIENTS WITH LIVER CIRRHOSIS

| | n | Died | Per cent |
|--------------------------------|----|------|----------|
| Heart-vascular | 32 | 19 | 59 |
| Pulmonary (tuberculosis n = 4) | 18 | 11 | 61 |
| Pancreas | | | |
| Diabetes | 11 | 5 | 45 |
| Chron. pancreatitis | 4 | 2 | 50 |
| Kidney | 10 | 9 | 90 |
| Bone | 5 | 2 | 40 |
| Irregular antibodies | 2 | 2 | |
| Haematopoetic system | 2 | 1 | |
| Brain | 2 | 1 | |
| Prostrate | 2 | 1 | |
| Biliary tract | 2 | 2 | |
| Intestinal | 2 | 1 | |
| Thyroid | 1 | 1 | |
| Adrenals | 1 | 1 | |
| Zollinger-Ellison-syndrome | 1 | 1 | |

ble. When these criteria are applied, portal hypertension was present in 49 of 69 patients (71%). Portal hypertension was demonstrable in 14 of 22 patients with gastric ulcer and in 25 of 35 patients with pyloric or duodenal ulcer.

Child's Classification

For the estimation of operative risk and long-term prognosis assessment of the patients according to Child's criteria would have been desirable. However, because most patients presented as emergencies essential criteria of the Child's classification such as the presence of neurologic symptoms or the response of ascites to conservative treatment could not be evaluated.

Indication for Surgical Treatment

With the exception of two patients, all operations were performed for complications of peptic ulcer disease. Sixty-two patients required emergency treatment for bleeding (n = 45) or perforated (n = 17) ulcer. Only seven patients had elective procedures. In two of these seven patients bleeding ulcers had responded to conservative treatment thus allowing for preoperative preparation of the patient. One patient had a recurrent anastomotic ulcer following proximal gastric vagotomy and subsequent Billroth-I partial gastrectomy penetrating into the liver. Two other patients were operated upon for suspected malignancy.

Table 3. OPERATIVE PROCEDURES IN ULCER PATIENTS WITH LIVER CIRRHOSIS

| | n | Elective n | Bleeding n | Perforation n |
|---------------------------|---------|---------------|---------------|------------------|
| Resectional procedures | | | | |
| BI | 5 (3) | 2 (0) | 3 (3) | 0 |
| BII | 23 (12) | 2 (0) | 21 (12) | 0 |
| BI-BII-Conv. | 1 (1) | 1 (1) | 0 | 0 |
| Gastrectomy | 2 (2) | 1 (1) | 1 (1) | 0 |
| Sum | 31 (18) | 6 (2) | 25 (16) | 0 |
| Nonresectional procedures | | | | |
| Suture | 23 (12) | 0 | 11 (7) | 12 (5) |
| Excision | 6 (2) | 1 (0) | 2 (1) | 3 (1) |
| TV | 8 (5) | 0 | 7 (5) | 1 (0) |
| PGV | 1 (0) | 0 | 0 | 1 (0) |
| Sum | 38 (19) | 1 (0) | 20 (13) | 17 (6) |
| Total | 69 (37) | 7 (2) | 45 (29) | 17 (6) |

Postoperative deaths in brackets.

Concurrent Disease

Additional diseases requiring treatment were recorded in 52 of 69 patients (75%, Table 2). The majority of patients had concomitant vascular or myocardial disease, pulmonary disease, or diseases of the pancreas. Preexisting renal insufficiency was recorded in 10 patients (14%) and only one of these patients survived. Characteristically of the overall reduced physical status of these patients, four patients had tuberculosis in the past. No additional disease was recorded in 17 patients. Ten of these 17 patients died (59%) and 5 patients died within 3 days of hospital admission indicating a foudroyant course possibly interfering with adequate diagnostic procedures.

Table 4. POSTOPERATIVE COMPLICATIONS IN ULCER PATIENTS WITH LIVER CIRRHOSIS*

| | n | Per cent | Died n | Died (%) |
|--------------------|----|----------|-----------|-------------|
| Bleeding | 27 | 40 | 22 | 81 |
| Pulmonary failure | 13 | 20 | 10 | 77 |
| Renal failure | 12 | 18 | 11 | 92 |
| Peritonitis | 9 | 14 | 8 | 89 |
| Liver failure | 8 | 12 | 7 | 88 |
| Anastomotic leak | 6 | 9 | 5 | 83 |
| Myocardial failure | 4 | 6 | 3 | 75 |
| Wound infection | 4 | 6 | 1 | 25 |

* More than one complication per patient possible. Fifteen patients without recorded complications (22%).

Table 5. CAUSES OF DEATH

| | n | Per cent |
|------------------------|----|----------|
| Multiple organ failure | 13 | 35.1 |
| Bleeding/coagulopathy | 13 | 35.1 |
| Sepsis | 5 | 13.5 |
| Myocardial failure | 3 | 8.1 |
| Liver failure | 2 | 5.5 |
| Pulmonary embolism | 1 | 2.7 |
| Sum | 37 | 100.0 |

Operative Treatment

Resectional treatment was performed in 31 patients (Billroth-I n = 5; Billroth-II n = 23; BI-to-BII-conversion n = 1; total gastrectomy n = 2; Table 3). One patient had proximal gastric vagotomy and eight patients had truncal vagotomy. In four of these patients, a Billroth-II partial gastrectomy had been performed 3 to 26 years previously.

Postoperative Morbidity

A total of 83 postoperative complications were recorded in 54 patients (Table 4). Infectious complications (peritonitis, pneumonia, wound infections; n = 26) and bleeding complications (n = 27) were most frequent. Only 17 patients with a complicated postoperative course survived (31%). Only 15 of 69 patients (21%) had an uncomplicated course.

Postoperative Mortality

Thirty-seven of 69 patients did not survive (54%, Table 1). The highest mortality was recorded in patients with posthepatic (9/15; 60%) or alcoholic cirrhosis (19/32, 59%) compared with patients with cryptogenic cirrhosis (9/22, 41%). Mortality was highest in patients with bleeding ulcer (29/45, 64%) and accordingly in patients with gastric ulcer (15/22, 68%). The mortality of patients with perforated ulcers was 35% (6/17). The most frequent cause of death was multiple organ failure and bleeding complications (26/37, 70%). Five patients died of septic complications despite initially preserved function of vital organs (Table 5).

Prognostic Factors

The mortality of patients with preexisting renal insufficiency was 90% (9/10). Apart from this no significant influence of comorbidity on outcome could be determined. Mortality of 52 patients with coexisting disease

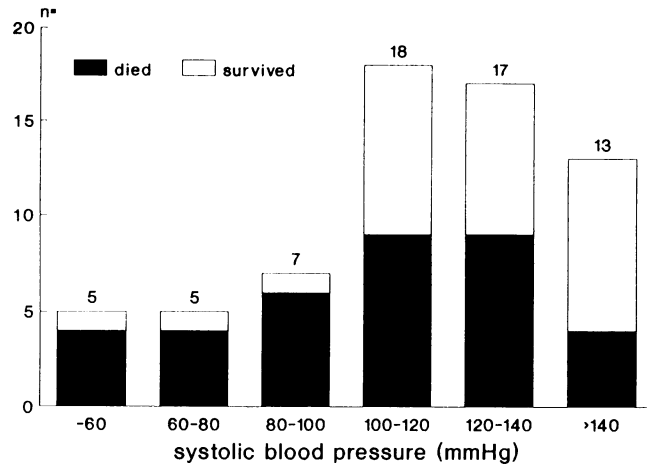


Figure 4. Postoperative mortality and preoperative systolic blood pressure. In patients with blood pressure < 100 mm Hg mortality was 82% compared with 46% in patients with blood pressure not falling below 100 mm Hg (p < 0.025).

was 52% compared with 59% for patients without proven concomitant diseases. It must be taken into account though that some of the latter patients had a foudroyant course not allowing for adequate diagnostic evaluation.

The mortality was increased in patients with preoperative systolic blood pressures less than 100 mm Hg (p < 0.025; Fig. 4), prothrombin time of less than 60% (p < 0.05; Fig. 1), preoperative hemoglobin less than 12 g/L (p < 0.05; Fig. 5) and clinical evidence of portal hypertension (p < 0.01; Fig 6). No significant differences were demonstrable when resectional treatment was compared with nonresectional treatment (18/31, 58% vs. 19/38, 50%).

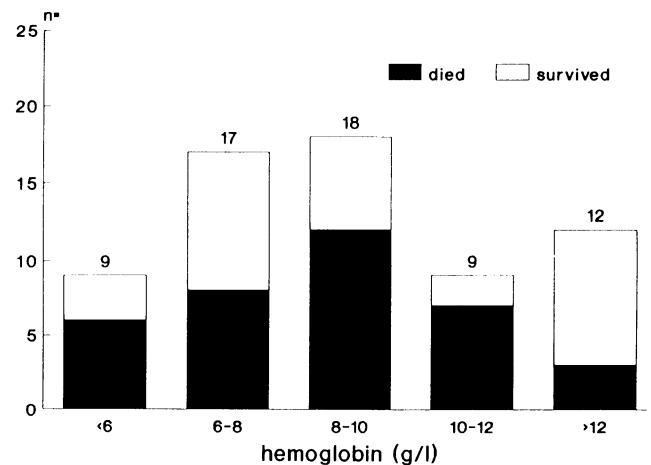


Figure 5. Postoperative mortality and preoperative hemoglobin. In patients with hemoglobin less than 12 g/L mortality was 62% as compared with 25% in patients with preoperative hemoglobin values less than 12 g/L (p < 0.05).

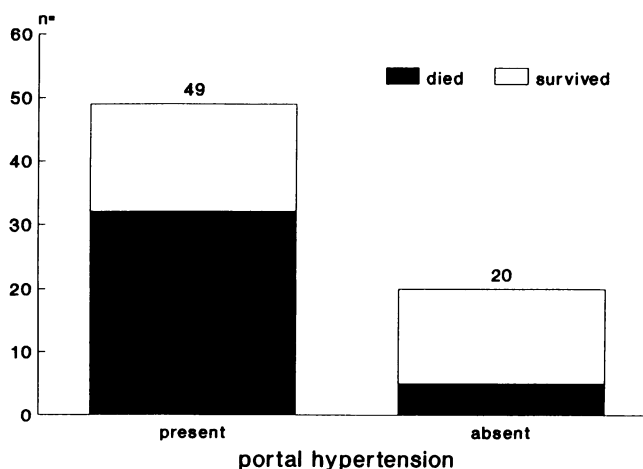


Figure 6. Postoperative mortality and portal hypertension. Mortality was 65% when signs of portal hypertension were recorded (32/47) and 25% when portal hypertension was absent (5/20; $p < 0.001$).

DISCUSSION

Experimental as well as clinical studies have provided a wealth of information serving to explain why liver cirrhosis might predispose to the development of peptic ulcer disease. Patients with liver cirrhosis may have increased serum levels of gastrin and histamin¹⁻³ and the presence of helicobacter microorganisms can be demonstrated in the gastric mucosa of almost 50% of patients with liver cirrhosis.⁴ In helicobacter positive patients the gastrin-feedback mechanism is interrupted.⁵ In addition to this, helicobacter produces proteases and by degradation of urea ammonia which both could facilitate ulcer development by altering the properties of the mucus gel covering the mucosa^{6,7} or via direct cytotoxicity against superficial gastric epithelial cells.⁸ While prostaglandins are cytoprotective in the stomach 6-keto-prostaglandin F₁ α levels are reduced in helicobacter-positive patients which is unusual for inflammatory tissues.⁹

Patients with cirrhosis may also have increased duodenogastric reflux¹⁰ and impaired gastric emptying of solids.¹¹ In patients with advanced cirrhosis and portal hypertension oxygen-saturation of duodenal mucosa is reduced¹² and gastric mucosal prostaglandin levels are reduced.^{13,14}

The incidence of gastroduodenal ulcer in patients with liver cirrhosis has been reported between 8 and 20%.¹⁵⁻¹⁷ It remains to be determined, however, whether the prevalence of peptic ulcer in patients with cirrhosis is truly increased when other factors such as alcohol and nicotine abuse or portal hypertension are taken into account as confounding variables.

The incidence of liver cirrhosis appears to be increased vice versa in patients with gastroduodenal ulcers. In ulcer patients 65 years or older the incidence of liver

cirrhosis has been recorded as 8.4%.¹⁸ In our own patients (1981-1990; $n = 876$) the incidence of liver cirrhosis is 9% in patients with bleeding ulcer, 6% in patients with perforated ulcer, and 8% in patients 65 years or older. In a study of 104 patients operated upon for gastroduodenal ulcer disease intraoperative liver biopsies revealed a histologic diagnosis of liver cirrhosis in 13% of patients.¹⁹ Several long-term follow-up studies confirm a threefold risk of gastroduodenal ulcer patients to die of liver cirrhosis compared with a normal population.²⁰⁻²³

The few reports indicate that surgical treatment of patients with liver cirrhosis is burdened with a particularly high postoperative mortality (Tables 6, 7). Mortality of intraabdominal operations has been recorded as 34.8% compared with a 9.1% mortality of extraabdominal procedures²⁴ and the mortality of emergency procedures is higher than the mortality of electively performed operations.²⁵⁻²⁷

Most experience has been accumulated in patients with biliary tract surgery. While postoperative mortality in elective patients is below 1%, mortality exceeds 20% in patients with liver cirrhosis (Table 7). A compilation of 87 patients with colon surgery revealed a postoperative mortality of 41% and mortality of 9 patients with operations of the small bowel was 67%. Experience with gastroduodenal ulcer surgery in cirrhotics is similarly limited. Mortality of emergency procedures for perforation or bleeding has been recorded at 42 and 49%, respectively (Table 6).

These observations prompt the question whether medical prophylaxis in ulcer patients with liver cirrhosis may be indicated and beneficial. More than half of our patients had no history of ulcer disease and this is confirmed by reports in the literature indicating that 61% of patients did not have a history of peptic ulcer disease.²⁸ It would seem, therefore, that the majority of patients are not candidates for medical prophylactic treatment.

Even when an attempt at medical prophylaxis is made using H₂-receptor antagonists in the majority of patients, no sufficient reduction of acid secretion can be achieved.²⁹ Even on maintenance therapy with H₂-receptor blockers more than half of the patients experience ulcer recurrence within 12 months.³⁰ Better results may possibly be achieved by using maintenance therapy with H⁺/K⁺-pump blockers,³¹ but current experience with 27 patients only appears to be too limited to allow for general conclusions to be drawn. These findings indicate that the efficacy of medical treatment is currently limited at best. They emphasize the need to improve the results of surgical treatment by identifying and correcting pre- and postoperative risk factors.

In our study, prothrombin time, preoperative systolic blood pressure, hemoglobin, renal function, and portal hypertension proved to be prognostic parameters. The

Table 6. POSTOPERATIVE MORTALITY FOLLOWING PEPTIC ULCER SURGERY IN PATIENTS WITH LIVER CIRRHOSIS

| Author | Ref | Period | n | Died | Per cent |
|-----------------------------|------|-----------|-----|------|----------|
| Elective Operations | | | | | |
| Branicki | (37) | 1985-1987 | 8 | 0 | 0.0 |
| Sirinek | (27) | 1981-1985 | 3 | 0 | 0.0 |
| Zarski | (34) | n.a. | 2 | 0 | 0.0 |
| present study | | 1972-1991 | 7 | 2 | 28.6 |
| Sum | | | 20 | 2 | 10.0 |
| Emergency Operations | | | | | |
| Bleeding Ulcer | | | | | |
| Palmer | (38) | 1946-1968 | 8 | 2 | 25.0 |
| Wara | (39) | 1971-1978 | 5 | 3 | 60.0 |
| Garrison | (25) | 1975-1982 | 12 | 4 | 33.3 |
| Sirinek | (27) | 1981-1985 | 9 | 1 | 11.1 |
| Branicki | (37) | 1985-1987 | 12 | 5 | 41.7 |
| Zarski | (34) | n.a. | 3 | 2 | 66.6 |
| present study | | 1972-1991 | 45 | 29 | 64.4 |
| Sum | | | 94 | 46 | 48.9 |
| Perforated Ulcer | | | | | |
| Garison | (25) | 1975-1982 | 11 | 5 | 45.5 |
| Zarski | (34) | n.a. | 3 | 0 | 0.0 |
| Mosnier | (43) | 1979-1987 | 22 | 11 | 50.0 |
| present study | | 1972-1991 | 17 | 6 | 35.3 |
| Sum | | | 53 | 22 | 41.5 |
| Total | | | 167 | 70 | 41.9 |

prognostic significance of PT, PTT, and serum albumin as parameters of synthetic function of the liver has been established by other studies.^{24-27,32} Ascites has also been determined to indicate a poor prognosis.^{24,26,32,33} In contrast to our findings others have determined increased serum bilirubin levels as a parameter of excretory liver function to be of prognostic significance.^{24,33} Renal function has only been examined in one study.³⁴ These authors also conclude that compromised renal function correlates with a poor prognosis.

The clinical relevance of these findings deserves some critical comment. Firstly practical applicability must be questioned. In our experience more than 90% of all operations had to be carried out as emergency procedures. Therefore subtle diagnostic procedures such as aminopyrine breath test or MEGX-test to better evaluate liver function may not be possible and even the simpler tests such as serum bilirubin or serum albumin measurements may not be available in emergency situations. More importantly, these prognostic parameters (bilirubin, albumin, SGOT, aP) cannot be influenced in an emergency.

Our univariate analysis of clinical and metabolic parameters indicates that preoperative systolic blood pressure below 100 mm Hg ($p < 0.025$), hemoglobin below 12 g/L ($p < 0.05$), and prothrombin time of less than 60% ($p < 0.05$) as well as the presence of portal hyperten-

sion ($p < 0.01$) correlate with a poor prognosis. Early and sufficient replacement of clotting factors and blood products are important to counteract an impending deficit of these compounds. In patients with portal hypertension intraoperative infusion of vasopressin has been recommended to reduce bleeding from venous collaterals.²⁷ Favorable results of such treatment have been reported in terms of mortality and morbidity, but relate mostly to elective operations.

Our finding that resectional treatment has a somewhat worse prognosis in terms of mortality (Table 3) and morbidity may be of practical value, even though differences did not achieve significant levels, since the choice of the operative procedure can obviously be influenced. In elective surgery for peptic ulcer, resectional treatment was followed by disturbances of liver function parameters³⁵ more frequently than nonresectional treatment.³⁶ These findings would suggest therefore, that resectional treatment be avoided in patients with liver cirrhosis since opening of an intraabdominal hollow organ and the resulting greater wound area may give way to the development of septic or bleeding complications.

Particularly good results have been reported in patients receiving dietary treatment preoperatively and in which antibiotics were administered freely.²⁷ The importance of early and sufficient antibiotic treatment together with an aggressive search and treatment of septic

**Table 7. MORTALITY FOLLOWING
INTRAABDOMINAL SURGERY IN PATIENTS
WITH LIVER CIRRHOSIS**

| Author | Ref | Period | n | Died | Per cent |
|----------------------|------|-----------|-----|------|----------|
| Biliary Tract | | | | | |
| Schwartz | (40) | 1965-1980 | 33 | 9 | 27.3 |
| Aranha | (41) | 1971-1979 | 55 | 14 | 25.5 |
| Doberneck | (24) | 1972-1982 | 20 | 7 | 35.0 |
| Garrison | (25) | 1975-1982 | 39 | 8 | 20.5 |
| Sirinek | (27) | 1981-1985 | 47 | 3 | 6.4 |
| Zarski | (34) | n.a. | 18 | 4 | 22.2 |
| Sum | | | 212 | 45 | 21.2 |
| Small Bowel | | | | | |
| Aranha | (26) | 1971-1984 | 2 | 2 | 100.0 |
| Garrison | (25) | 1975-1982 | 5 | 3 | 60.0 |
| Zarski | (34) | n.a. | 2 | 1 | 50.0 |
| Sum | | | 9 | 6 | 66.6 |
| Colon | | | | | |
| Metcalfe | (32) | 1970-1984 | 54 | 22 | 40.7 |
| Aranha | (26) | 1971-1984 | 5 | 4 | 80.0 |
| Garrison | (25) | 1975-1982 | 9 | 5 | 55.6 |
| Griffen | (42) | 1977-1982 | 6 | 3 | 50.0 |
| Sirinek | (27) | 1981-1985 | 1 | 0 | 0.0 |
| Zarski | (34) | n.a. | 12 | 2 | 16.7 |
| Sum | | | 87 | 36 | 41.4 |

foci is confirmed by the high incidence of septic complications in our experience. Although to date there are no reliable data available, the addition of selective bowel decontamination may be useful in cirrhotic patients undergoing abdominal surgery.

In conclusion there may be three different approaches by which the results of surgical treatment for gastroduodenal ulcer disease in patients with liver cirrhosis may be improved:

- (1) In an elective situation, careful evaluation of liver and renal function and possible preoperative recompensation appear to be of utmost importance. In the emergency situation, early and sufficient substitution of clotting factors and blood products appears to be crucial.
- (2) The high rate of septic complications would make perioperative antibiotic prophylaxis a minimum requirement. In an emergency situation, in patients with additional risk factors, and especially after resectional treatment administration of antibiotics for a prolonged period, and selective bowel decontamination should be considered.
- (3) In the emergency situation, surgical treatment should not be aimed at the treatment of peptic ulcer disease, but should be limited to what is required to control bleeding or perforation. Whenever justifi-

able, resectional treatment should be avoided in these patients.

- (4) In patients with a known combination of peptic ulcer disease and liver cirrhosis, long-term maintenance treatment with omeprazole may be beneficial and maintenance treatment may also be recommended for patients with nondefinitive surgical treatment of a bleeding or perforated ulcer.

References

1. Sato T, Imamura M, Sasaki I, et al. Gastric acid secretion and gastrin and gastric inhibitory polypeptide release in cirrhotic patients. *Am J Gastroenterol* 1985; 80:163-169.
2. Irvine WT, Duthie HL, Ritchie HD, et al. The liver's role in histamine absorption from the alimentary tract: its possible importance in cirrhosis. *Lancet* 1959; I:1064-1069.
3. Stopnik D, Hampel KE, von Kleist D. Endogenes plasma-histamin und 'hepatogenes' gastroduodenales ulcus bei lebercirrhose. *Dtsch Med Wochenschr* 1977; 102:932-933.
4. Kim H, Park C, Jang WI, et al. The gastric juice urea and ammonia levels in patients with campylobacter pylori. *Am J Clin Pathol* 1990; 94:187-191.
5. Levi S, Beardshall, Haddad G, et al. Campylobacter pylori and duodenal ulcers: the gastrin link. *Lancet* 1989; I:1167-1168.
6. Sarosiek J, Bilski J, Murty VLN, et al. Colloidal bismuth subcitrate (de-nol) inhibits degradation of gastric mucus by campylobacter pylori protease. *Am J Gastroenterol* 1989; 84:506-510.
7. Sidebotham RL, Baron JH. Hypothesis: helicobacter pylori, urease, mucus and gastric ulcer. *Lancet* 1990; 335:193-195.
8. Smoot DT, Mobley HLT, Chippendale GR, et al. Helicobacter pylori urease activity is toxic to human gastric epithelial cells. *Infect Immunol* 1990; 58:1992-1994.
9. Goren A, Fotherby KJ, Shorthouse M, et al. Campylobacter and acid secretion. *Lancet* 1989; II:613.
10. Kliems G, Feld W, von Bergmann K. Lebercirrhose und duodeno-gastrischer Reflux: zum Problem des hepatogenen Ulcus. *Langenbecks Arch Chir* 1983; 360:109-118.
11. Abiazov AA, Dudarev AL, Iakovleva LA: Gastric emptying rate in healthy subjects and patients (a radionuclide study). *Med Radiol Mosk* 1991; 36:18-20.
12. Tanaka M, Inatsuchi S, Terasaki T, et al. Duodenal mucosal hemodynamics in patients with liver cirrhosis. *Acta Med Okayama* 1990; 44:273-277.
13. Arakawa T, Satoh H, Fukuda T, et al. Endogenous prostaglandin e2 in gastric mucosa of patients with alcoholic cirrhosis and portal hypertension. *Gastroenterology* 1987; 93:135-140.
14. Weiler H, Weiler C, Gerok W. Gastric mucosal prostaglandin e2 levels in cirrhosis and portal hypertension. *J Hepatol* 1990; 11:58-64.
15. Sonnenberg A. Concordant occurrence of gastric and hypertensive disease. *Gastroenterology* 1988; 95:42-48.
16. Giacobbe A, Facciorusso D, Conoscitore P, et al. Peptic ulcer in liver cirrhosis. *Minerva Dietol Gastroenterol* 1990; 36:223-226.
17. Rabinovitz M, Yoo YK, Schade RR, et al. Prevalence of endoscopic findings in 510 consecutive individuals with cirrhosis evaluated prospectively. *Dig Dis Sci* 1990; 35:705-710.
18. Seitz W, Rothmund M, Kraushaar G. Die chirurgische Behandlung des Gastroduodenalulcus beim alten Menschen. *Langenbecks Arch Chir* 1982; 356:95-103.
19. Zittel RX, Weyand H, Weyand F. Zur Bedeutung pathologischer

- Leberbefunde beim Magen-Duodenalulcus und beim Ulcus pepticum jejuni. *Dtsch Med Wochenschr* 1967; 92:791-793.
20. Hirohata T. Mortality from gastric cancer and other causes after medical treatment or surgical treatment of gastric ulcer. *J Natl Cancer Inst* 1968; 41:895-908.
 21. Bonnevie O. Causes of death in duodenal and gastric ulcer. *Gastroenterology* 1977; 73:1000-1004.
 22. Ross AHM, Smith MA, Anderson JR, et al. Late mortality after surgery for peptic ulcer. *N Engl J Med* 1982; 307:519-522.
 23. Watt PCH, Patterson CC, Kennedy TL. Late mortality after vagotomy and drainage for duodenal ulcer. *Br Med J* 1984; 288:1335-1338.
 24. Doberneck RC, Sterling WA, Allison DC. Morbidity and mortality after operation in non-bleeding cirrhotic patients. *Am J Surg* 1983; 146:306-309.
 25. Garrison RN, Cryer HM, Howard DA, et al. Clarification of risk factors for abdominal operations in patients with hepatic cirrhosis. *Ann Surg* 1984; 199:648-655.
 26. Arahna GV, Greenlee HB. Intra-abdominal surgery in patients with advanced cirrhosis. *Arch Surg* 1986; 121:275-277.
 27. Sirinek KR, Burk RR, Brown M, Levine BA. Improving survival in patients with cirrhosis undergoing major abdominal operations. *Arch Surg* 1987; 122:271-273.
 28. Teres J, Bordas JM, Bru C, Diaz F, Bruguera M, Rodes J. Upper gastrointestinal bleeding in cirrhosis: clinical and endoscopic correlations. *Gut* 1976; 17:37-40.
 29. Walker S, Krishna DR, Klotz U, et al. Frequent non-response to histamine H₂-receptor antagonists in cirrhotics. *Gut* 1990; 30:1105-1109.
 30. DiMario F, Gottardello L, Burra P, et al. Studio clinico-endoscopico sulla cicatrizzazione della lesione e sulla prevenzione delle recidive in pazienti con cirrosi epatica e malattia ulcerosa. *G Clin Med* 1990; 71:259-266.
 31. Brunner GH, Lamberts R, Creutzfeldt W. Efficacy and safety of omeprazole in the long-term treatment of peptic ulcer and reflux oesophagitis resistant to ranitidine. *Digestion* 1990; 1:64-68.
 32. Metcalf AMT, Dozois RR, Wolff BG, et al. The surgical risk of colectomy in patients with cirrhosis. *Dis Colon Rectum* 1987; 30:529-531.
 33. Tinkoff G, Rhodes M, Diamond D, et al. Cirrhosis in the trauma victim. *Ann Surg* 1990; 211:172-177.
 34. Zarski JP, Bichard P, Bourbon P, et al. La chirurgie digestive extra-hepatique chez le cirrhotique: mortalite, morbidite, facteurs pronostiques pre-operatoires. *Gastroenterol Clin Biol* 1988; 12:43-47.
 35. Herfarth C. Enzymologische Untersuchungen zur Frage der postoperativen Leberschädigung. *Bruns Beitr Klin Chir* 1968; 216:504-516.
 36. Otto GF, Brückner WL. Leberenzymmuster im Serum nach Magenoperationen. *Med Klin* 1971; 66:1603-1607.
 37. Branicki FJ, Coleman SY, Fok PJ, et al. Bleeding peptic ulcer: a prospective evaluation of risk factors for rebleeding and mortality. *World J Surg* 1990; 14:262-270.
 38. Palmer ED. The vigorous diagnostic approach to upper-gastrointestinal tract hemorrhage. *JAMA* 1969; 24:1477-1480.
 39. Wara P, Berg V, Amdrup E. Factors influencing mortality in patients with bleeding ulcer. *Acta Chir Scand* 1983; 149:775-785.
 40. Schwartz SI. Biliary tract surgery and cirrhosis: a critical combination. *Surgery* 1981; 90:577-583.
 41. Aranha GV, Sontag SJ, Greenlee HB. Cholecystectomy in cirrhotic patients: a formidable operation. *Am J Surg* 1982; 143:55-60.
 42. Griffen WO, Jr. Discussion. *Ann Surg* 1984; 199:648-655.
 43. Mosnier H, Farges O, Vons C, et al. Gastroduodenal ulcer perforation in the patient with liver cirrhosis. *Surg Gynecol Obstet* 1992; 174:297-301.