Improved Survival in 45 Patients with Pancreatic Abscess

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The reported mortality due to pancreatic abscesses after acute pancreatitis has been 30 to 50%, a statistic that has remained unchanged for decades. This is a report of 45 patients treated over 10 years, showing a dramatic improvement in survival during that period. They represent 2.5% of admissions at the Massachusetts General Hospital for acute pancreatitis. The identifiable antecedents included alcohol (38%), gallstones (11%), and surgical trauma (16%), or were unknown in 24%. Computerized tomography (CT) was clearly the best means of specific diagnosis (unequivocal evidence in 74%, suggestive in 21%). Treatment in 44 patients was surgical debridement and catheter drainage, and in one it was resection of the pancreatic head. Multiple abscesses were present at the first operation in 21 patients. Seven had second drainage procedures for additional abscesses. In the first 5 years (1974–1978), 10 of 26 patients died (38%). In the second 5 years (1979–1983), one of 19 died (5%) ($p < 0.01$). Postoperative complications (84%) included wound hemorrhage (9 of 26 *vs.* 1 of 19), systemic sepsis (7 of 26 *vs.* 1 of 19), pancreatic fistula (14/45, 13 of which closed spontaneously), colonic perforation (4), duodenal perforation (2), and gastric perforation (1). The causes of death were renal and respiratory failure with sepsis (7), hemorrhage (3), and pulmonary emboli (1). Analysis of the findings shows in the second 5-year period more frequent use of CT to certify the diagnosis of pancreatic abscess earlier, a more aggressive attitude producing earlier surgical intervention, and more extensive drainage and debridement of associated necrotic tissue. Transcatheter arterial embolization was used successfully to control postoperative hemorrhage from the abscess cavity. CT-guided percutaneous catheter drainage was used occasionally for drainage of recurrent abscesses. Neither open packing of major pancreatic abscesses nor lavage of the abscess cavity, as recently advocated, was necessary.

PANCREATIC ABSCESSES ARE in most instances the consequence of infection of necrotic tissues that are products of severe acute pancreatitis[1,2] and have been observed after 1 to 9% of cases.[3–10] Diagnosis of an abscess may be obscured and delayed because its manifestations blend with those of the pre-existing illness. The destructive effects of the abscess combine with enzymatic and ischemic injuries from the pancreatitis to produce a reported mortality of 30 to 50%,[3,4,6–8,10,11] a figure that has remained largely unchanged for decades. In response to this, recent reports have suggested innovations to therapy, including open packing of the abscess cavity[10] and multiple operations for dressing changes,[12] or continuous lavage of the cavity.[13,14] We reviewed the experience with pancreatic abscesses at the Massachusetts General Hospital during the past 10 years and have found a dramatic increase in survival. This report is our analysis of the factors that appear to contribute to that improvement.

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Materials and Methods

During the 10-year period from 1974 to 1983, 1818 cases of acute pancreatitis were treated at the Massachusetts General Hospital. Forty-five of them (2.5%) developed a pancreatic abscess, confirmed at operation at this institution. Only patients whose abscess was the direct consequence of antecedent acute pancreatitis have been included. Excluded are all patients with infected pseudocysts, those with only histological evidence of microabscess, peripancreatic and lesser-sac abscesses not consequent to pancreatitis, and patients whose surgical drainage was performed elsewhere. The records of these 45 patients comprise the data base for this study.

Results

Patients

There were 30 males and 15 females, ranging in age from 21 to 79 years (mean: 44). Identifiable antecedents to acute pancreatitis are shown in Table 1. The seven postoperative cases followed biliary tract procedures in five, transduodenal pancreatography in one, and subtotal gastrectomy in one. Twenty-six cases occurred in the first 5-year period of the study and 19 in the second. The incidence relative to total cases of acute pancreatitis during the two periods was not significantly different.

Presented at the 105th Annual Meeting of the American Surgical Association, April 25–27, 1985, New Orleans, Louisiana.

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Submitted for publication: April 29, 1985.

TABLE 1. *Antecedents to Pancreatitis in 45 Patients Going on to Pancreatic Abscess*

Alcohol	17
Gallstone	5
Abdominal operation	7
Renal transplantation	2
Trauma	1
Islet cell tumor	1
Carcinoma of pancreas	1
Idiopathic	11

Clinical Course

Twenty-six patients had a fulminant course, continuous with their original acute pancreatitis. These patients tended to have high fevers and prominent signs of abdominal sepsis. In this group, the mean interval between onset of pancreatitis and drainage of the abscess was 18 days.

Nineteen patients had a more indolent presentation, characterized by persistent low-grade fever, leucocytosis, and a pancreatic inflammatory mass. Seven of them had an interval of seeming well-being between the pancreatitis and relapse with abscess. The average interval between onset of pancreatitis and drainage of the abscess in this group was 49 days (range: 30–120 days).

There was no significant difference in the distribution of the two patterns in the two time periods.

Symptoms and Signs

Epigastric pain was the most common symptom, but it was absent in nearly 20% of patients. Fever, the most common sign, was absent in 13%. The individual features or combinations of features of the history and physical exam were too inconstant to be reliable for diagnosis of a pancreatic abscess (Table 2).

Laboratory Findings

The principal laboratory findings are shown in Table 3. In most patients, they were nonspecific and were frequently normal. Of note, the white blood count was most commonly between 10,000 and 15,000. Six patients (13%) had white cell counts under 10,000. The serum amylase was increased in fewer than half the patients at the time of diagnosis of the pancreatic abscess. Tests of liver function were abnormal in 38 to 51%. Hypoalbuminemia and hypocalcemia were also common.

TABLE 2. *Presenting Symptoms and Signs of Pancreatic Abscess in 45 Patients*

Fever	39 (87%)
Abdominal pain	37 (82%)
Abdominal tenderness	28 (62%)
Palpable mass	22 (50%)
Nausea or vomiting	21 (47%)
Distention	12 (27%)
Jaundice	7 (16%)
Systemic sepsis	3 (7%)
Pulmonary failure	2 (4%)
GI bleeding	1 (2%)

TABLE 3. *Laboratory Findings at Diagnosis of Abscess (45 Patients)*

Test		Number
White blood count/mm^3	>20,000	6
	15–20,000	7
	10–15,000	26
	<10,000	6
High serum amylase (>25 Russell u.)		19
High alkaline phosphatase (>40 international u.)		18
High SGOT (>40 u.)		23
High bilirubin (>1.2 mg/dl)		17
Low calcium (<8.5 mg/dl)		25
Low albumin (<3 gm/dl)		20

Serum ribonuclease (RNAse) was measured in 14 patients as part of our prospective evaluation of this test as an index of pancreatic necrosis.[15,16] The serum RNAse was elevated above normal values in 11 of 14. All of those 11 had necrotic tissue requiring debridement from the abscess cavity. Two of the three patients with normal levels of RNAse had pus but no residual necrotic debris in the cavity; the third did not have the RNAse measured until 2 months after the onset of pancreatitis.

Bacteriologic Findings

Cultures were taken from the abscess in all patients and were positive in 40 (Table 4). Grain stains showed organisms in the other five. Twenty abscesses grew out only one organism, but 20 cultures contained a polymicrobial mixture of enteric flora: two and three bacterial species were found in eight patients each, and four species were found in four patients. *Escherichia coli,* Enterococcus, and Staphylococcus were most prevalent. *Candida albicans* was the sole agent found in three abscesses, but no

TABLE 4. *Results of Microbiological Cultures from 45 Pancreatic Abscesses**

Escherichia coli	22
Enterococcus	17
Staphylococcus	16
Klebsiella	6
Proteus	4
Candida albicans	3
Pseudomonas	3
Streptococcus	2
Torulopsis glabrata	1
Hemophilus parainfluenzae	1
Diphtheroids	1
Serratia marcescens	1
Negative culture (positive gram stain for organisms on smear)	5

* More than one organism grew from 20 specimens.

TABLE 5. *CT and US Findings in Patients with Pancreatic Abscess*

Findings	CT Scan	US Scan
Diagnostic of abscess	14 (74%)	12 (35%)
Nonspecific abnormalities	4 (21%)	18 (53%)
Pseudocyst	2	6
Pancreatic mass	1	8
Pancreatic swelling	1	4
Negative	1	2
Unsuccessful	0	2
Total	19	34

other anaerobic organisms (Bacteroides or Clostridia) were identified despite the general employment of anaerobic culture techniques and media.

Radiological Findings

Radiological abnormalities were common, but most were nonspecific for abscess, especially in differentiating abscess from other forms of pancreatic inflammation. The plain film of the abdomen showed signs of ileus or small bowel dilatation ("sentinel loop") in 46%, but the classical "soap-bubble sign" (mottled lucencies in the retroperitoneal tissues) or gas bubbles were noted in only four patients (9%). An upper gastrointestinal series showed displacement or outlet obstruction of the stomach and extrinsic compressions of the duodenum in 25 of 29 patients. In seven selected patients, barium enema examination demonstrated displacement or narrowing of the colon, or both, in six and a communication between the colon and the abscess in one. Thirty-two patients had an abnormal chest x-ray, including pleural effusions, atelectasis, or pneumonitis.

Ultrasound (US) and computed tomographic (CT) scanning were more specific and effective (Table 5). US was used in 34 patients before surgery, yielded a specific diagnosis in 12 (35%), showed nonspecific pancreatic abnormalities in 18 (53%), and was negative or unsuccessful in four (12%). Nineteen patients were studied with US in the first period and 15 in the second.

CT was performed in 19 patients and gave a specific diagnosis in 14 (74%). Four (21%) studies were suggestive but not specific, and only one (5%) was negative. Seven of 26 patients (27%) in the first 5 years and 12 of 19 (63%) in the second 5 years had a CT examination.

Serial US or CT examinations, carried out in 6 patients, clearly showed the evolution from pancreatic phlegmon, to liquefaction necrosis, to abscess (Fig. 1).

Percutaneous needle aspiration and percutaneous catheter drainage of abscesses under CT guidance were tried in one patient each. Sampling of the pus was successful in both, but both patients subsequently required definitive surgical drainage.

Surgical Treatment

Surgery was performed on all 45 patients. Although most of the abscesses were in the pancreas and immediate peripancreatic region, including the lesser sac, they were

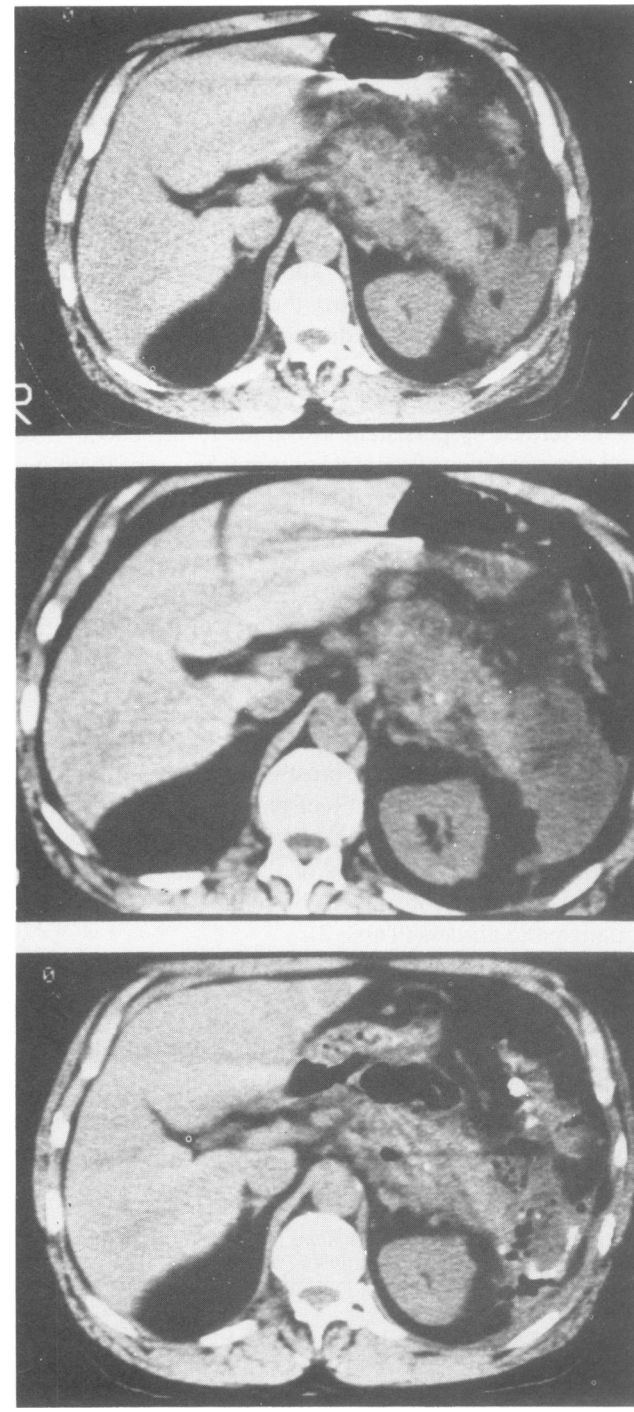

FIG. 1. Serial CT scan of the pancreas at 10, 20, and 30 days after onset of acute pancreatitis. Progressive changes are evident: phlegmonous swelling and edema (*top*); irregular lucent areas consistent with liquefaction necrosis, along with extension to the left perirenal space (*middle*); gas bubbles within a pancreatic abscess (*bottom*).

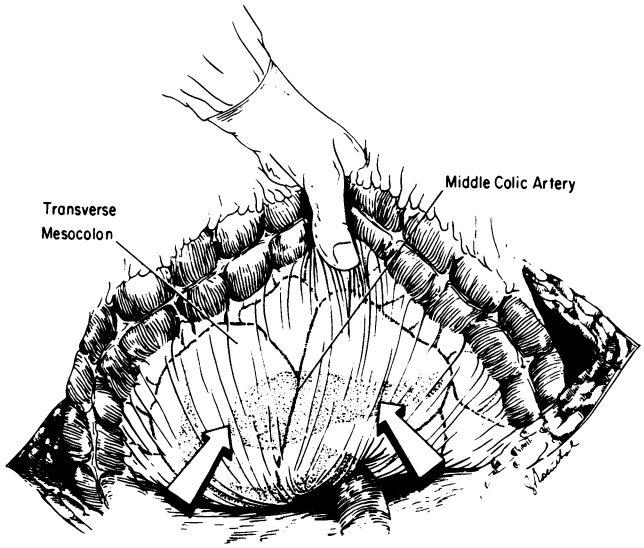

FIG. 2. Diagrammatic view of a recommended access to pancreatic debridement and drainage. This approach is often simpler and less hazardous to the stomach and colon than a dissection through the omentum into the lesser sac.

found in every quadrant of the abdomen. Twenty-one patients had more than one abscess at the time of initial drainage.

Our operative technique has evolved to a relatively uniform practice in the last 5 years. In most cases, we used a midline abdominal incision to allow wide and unrestricted exploration of the entire abdomen. Access to the abscess was often easiest and most direct through the transverse mesocolon (Fig. 2), but other approaches were tailored to the location of the palpable foci of induration. Debridement of softened devitalized tissue, largely by means of finger dissection, was as complete as possible. The cavity with its extensions resulting from drainage and debridement was filled and packed with a combination of stuffed rubber drains and suction drains. These were grouped broadly out to the abdominal wall but traversed the abdominal wall through individual incisions (Fig. 3). The drains were removed in stages, starting after a week. We believe that this technique provides the virtues of packing and large paths for slough and egress of tissue and yet minimizes the risk of incisional hernia. Primary closure of the abdominal incision was utilized in all 27 patients operated on by the senior author.

In 41 cases, the abscess was drained with multiple sump catheters, stuffed Penrose drains, or closed-suction drains. Single drains were used mainly when the abscess was thought to be a simple cavity without solid necrotic tissue. In two of the 42 cases, the tail of the pancreas and spleen was resected as part of the debridement. One patient also had a left colectomy for colonic necrosis and perforation. In two other cases, the cavity was packed open. In one case, the cavity was anastomosed to the stomach for internal drainage. In the final case, the abscess cavity in the head of the pancreas was excised with a duodenal fistula by pancreaticoduodenectomy (Whipple procedure). Table 6 lists other operative procedures carried out during the primary drainage operation.

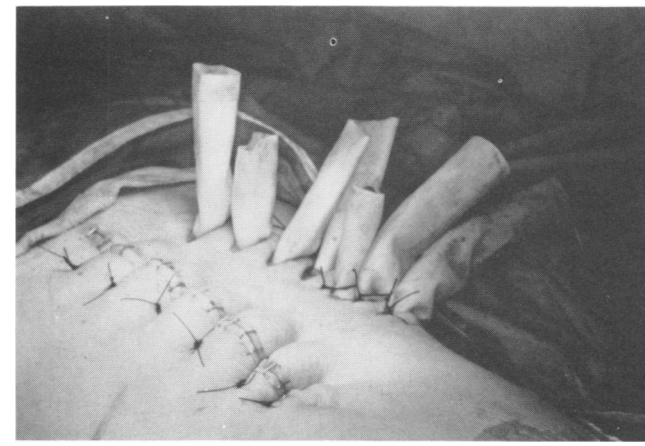

FIG. 3. Exit from the abdomen of the drains used to pack and drain a large pancreatic abscess cavity.

Persistent or recurrent abscesses requiring further treatment occurred in seven patients (26%), four in the first period and three in the second. Four had had minimal debridement and use of only a single drain. In two cases during the second period, recurrent abscesses were successfully drained with percutaneous catheters placed under US or CT guidance.

Morbidity and Mortality

Complications occurred in 38 of the 45 patients (84%) (Table 7). There was a marked reduction of life-threatening complications in the second period. Major hemorrhage developed in nine of 26 patients between 1974 and 1978, but in only one of 19 in the next 5 years. Major sepsis and multisystem failure occurred in 27% during period 1, compared with 5% in period 2. There were six infections in the abdominal incision (one among the 27 primary wound closures by the senior author) and one subsequent incisional hernia.

Postoperative pancreatic fistulas occurred in 14 of the 45 patients. All except one closed spontaneously within 2 months; the last one required a distal pancreatectomy

TABLE 6. *Other Surgical Procedures During Drainage of Pancreatic Abscess in 45 Patients*

Cholecystostomy	2
Gastrostomy	4
Feeding jejunostomy	4
Gastrostomy, feeding jejunostomy	8
Gastrostomy, jejunostomy, cholecystostomy	4
Cholecystectomy	2
Choledochojejunostomy	2

TABLE 7. *Morbidity and Mortality in 45 Patients with Pancreatic Abscess*

Complications	1974–1978	1979–1983	1974–1983	Died
Hemorrhage				7
Pancreatic fistula	9	1	10	1
Colonic necrosis	8	6	14	1
Colonic fistula	1	1	2	0
Duodenal fistula	1	1	2	1
Gastric fistula	1	1	2	0
Systemic sepsis	1	0	1	8
Recurrent abscess	7	1	8	1
Renal failure	4	3	7	4
Respiratory failure	3	1	4	3
Pulmonary emboli	3	0	3	1
Pneumonia	1	0	1	
Wound infection	6	3	9	
Permanent diabetes	5	1	6	
Splenic vein thrombosis	2	1	3	
	1	0	1	
Incisional hernia	0	1	1	
Total patients with complications	24 (92%)	14 (74%)	38 (84%)	
Total patients dying	10 (38%)	1 (5%)	11 (24%)	

after 6 months. Colon perforation (2) or colocutaneous fistula (2) developed in four patients. One of the former died with a necrotic left colon, and the other survived a resection of the splenic flexure and later re-anastomosis. Neither of the two fistulas closed, and both required staged colon resection and re-anastomosis. The only gastric fistula also persisted and necessitated urgent gastrectomy to manage recurrent hemorrhage. Of two duodenal fistulas, the lone survivor needed a Whipple resection. No persistent fistulas or other complications occurred as a consequence of cholecystostomy, gastrostomy, or feeding jejunostomy tubes in 22 patients.

Eleven patients (24%) died, 10 of 26 (38%) between 1974 and 1978, and one of 19 (5%) between 1979 and 1983. Seven died with renal and respiratory failure due to overwhelming sepsis, three from massive intra-abdominal hemorrhage, and one from pulmonary emboli. Eight of 26 patients (31%) with early fulminant presentation died. Three of 19 (16%) with late indolent presentation died; one of these deaths was due to breakdown of an ill-advised[17] anastomosis of the abscess cavity to the stomach.

Ten patients developed major hemorrhage (>2000 ml) from the abscess cavity in the postoperative period. Five were directly re-explored, successful control being accomplished in four. Arteriographic examinations identified the bleeding point in all four patients in whom it was tried. In those four patients, transcatheter embolization permanently stopped the hemorrhage in one, produced temporary control (allowing orderly surgical re-exploration and debridement) in two, and failed in one. The tenth patient ceased bleeding spontaneously.

The mean duration of hospitalization after operation was 49 days.

Discussion

Pancreatic abscess is a late complication of acute pancreatitis, usually arising from secondary infection of necrotic pancreatic and peripancreatic tissues.[1,2,18] What Fitz recognized nearly 100 years ago[19] has had to be relearned. At present, the incidence of abscess after acute pancreatitis is less than 4%,[1,3] 1.7% in the recent large series from the Mayo Clinic,[8] and 2.5% in our own.

Since Ranson developed his criteria for semiquantitating the severity of pancreatitis,[20] several studies have used his or like criteria to stratify pancreatic abscess patients as well.[6,8,12] The probability of a pancreatic abscess seems clearly related to the severity of the antecedent pancreatitis. This correlation re-emphasizes that the pathogenesis of pancreatic abscesses is rooted in the ripe culture medium produced by the combination of ischemic necrosis[1,15,21,22] and enzymatic injury to tissues by escaped pancreatic enzymes. When both factors are prominent, the abscess tends to manifest earlier, as part of a continuum of ongoing necrotizing pancreatitis.[5,10,12,23] When the enzymatic component is more finite, the attack may even seem to be ended, only to re-emerge in a new stage of infection.[4,10,18] In our experience, the early abscesses (10–20 days after onset) occurred in sick, unstable, toxic patients and carried a mortality rate double that of the patients who presented several weeks later, often with an intervening period of apparent well-being.

Adding to the problem is the fact that the symptoms and signs of pancreatic abscesses are nonspecific and variable, and routine laboratory and radiographic tests are not very helpful. It is frequently difficult to distinguish the developing abscess from the continuing phlegmon, a noninfected pancreatic inflammatory mass. Some patients may have little or no fever or leucocytosis. The classic "soap-bubble sign" on plain abdominal radiographs was seen in only 9% of cases. The correct diagnosis and treatment instituted because of it are therefore delayed inordinately, allowing further problems and complications.

Ultrasonography has added little to the sensitivity or accuracy of diagnosis of pancreatic abscess; while 88% of patients with a proven abscess had abnormal US examinations, only 35% had abnormalities specific for an abscess. In contrast, CT scanning demonstrated abscess-specific changes in 74% of patients and nonspecific signs of pancreatic inflammation in an additional 21%. Other recent studies have had identical findings.[7,23,24]

The results of treatment for pancreatic abscesses reported to date have been discouraging. If those series that include infected pseudocysts, post-traumatic abscesses, and causes of peripancreatic infection other than acute pancreatitis are excluded, the mortality of surgically treated pancreatic abscesses is commonly 30 to 50%.[3,4,6–8,10,11,25] The causes of death are a combination of ongoing

destruction of tissues, progressive sepsis, hemorrhage, and multiple organ failure. Antibiotics have not had a convincing positive effect in reducing the incidence of abscesses after pancreatitis, and have never cured one. At best, they limit bacteremia. At worst, the indiscriminate use of antibiotics selects out resistant organisms, including Candida,[18,26] from the polymicrobial pool of enteric candidates.

The traditional method of treatment of pancreatic abscesses has been surgical drainage, usually by inserting one or more nontraumatic drains of some type in the abscess cavity. The route of access to the cavity has been a subject of discussion, some favoring a transperitoneal approach and others entering it *via* the flank and retroperitoneum. The failure of these efforts has at least in part been due to the reluctance to expose the cavity widely and to debride the necrotic components aggressively. Extension of the injury after conservative drainage and debridement and recurrence of the abscess and sepsis account for most of the deaths.

In a logical effort to meet that challenge, Bradley has championed more aggressive debridement with open packing of the cavity, as previously suggested by Bolooki and his colleagues.[10] This method requires frequent reoperation for dressing changes at first, although the repacking can eventually be done in the patient's room without general anesthesia. Bradley and Fulenwider[12] most recently have reported a 14% mortality rate (3 of 21 patients) treated thus. Stone et al.,[27] reporting from a different service at the same institution, stated that five of 14 of their patients (36%) died after open packing (three patients eviscerated). They advocated subtotal distal pancreatectomy and then packing, and reported 9% mortality (2 of 22). However, few of the patients (11%) treated by Stone et al. had abscesses consequent to acute pancreatitis. Most (81%) followed trauma to the pancreas. Becker et al.,[8] have reported disappointing experience with open packing at the Mayo Clinic hospitals. The average length of hospitalization after open packing was 76 days in Bradley's series,[12] compared with 49 days in this one.

Büchler et al., concerned by the difficulty of adequately debriding necrotic tissues by surgical means, have used continuing local lavage *via* catheters left in the cavity after debridement. Compared with the historical controls, mortality in patients with pancreatic necrosis was reduced from 28 to 6%.[13]

In the group of patients included in this report, we have observed a striking increase in survival with pancreatic abscesses after acute pancreatitis. Over the 10 years of our study, mortality has fallen from 38% (1974–1978) to 5% (1979–1983). In our analysis, we cannot attribute this change to any one factor such as a particular surgical technique. For example, the open packing method used by Bradley and Fulenwider[12] was basically the same as that used by Bolooki et al.[10] 15 years earlier; yet the later series had a much lower death rate. Certainly, better antibiotics are not the answer. The mortality rate in patients treated by antibiotics without good drainage is essentially 100%,[3,8,27] and the net effect of new generations of antibiotics is to promote the growth of resistant organisms from the available pool, as is well recognized and again exemplified by our own findings. Rather we feel that our success is due to a combination of factors:

1. Greater awareness of the natural progression of acute pancreatitis through specific phases of ischemic and enzymatic injury, necrosis, and secondary infection.[1,28]
2. Better means of detecting pancreatic abscesses and the antecedent liquefaction necrosis by CT scanning.[24] We continue our interest in the use of serum ribonuclease as an early marker for pancreatic necrosis[15,16,22] and observed increased ribonuclease levels in 11 of 14 patients.
3. A more aggressive attitude toward early debridement. With the understanding that substantial amounts of necrotic tissue are better evacuated and not left to become infected and that infection in necrotic tissues may have few manifestations at first, we prefer not to wait for signs of overt sepsis. It has been our experience that the longer the destructive process is allowed to continue, the more likely are additional complications, including vascular and visceral injuries.
4. More complete debridement. The mere placement of drains in the cavity is inadequate in many cases. We debride as much of the regional necrosis as can be removed by blunt dissection, best done with fingers to distinguish firm vital tissue from stringy, mushy dead material. The cavity is often irregular, with pseudopods extending widely, surrounding major vessels, and frequently multiloculated or multifocal. A complete search of the abdomen is advisable in most primary operations, and exposure should be adequate to allow control of points of hemorrhage. The transmesocolic approach (Fig. 2) is often easiest, most expeditious, and safest in avoiding damage to the transverse colon or stomach when they are densely adherent to the abscess. This route is of particular value in reoperating on patients who have had prior gastric surgery, have a gastrostomy tube anchoring the stomach, or who have recurrent abscess after previous drainage through a different place.

Presumably because of the multiple factors listed above, fewer lethal complications were encountered in the second period of this study. We infer that the more responsive, aggressive approach being advocated left less time for the regional necrotizing process to extend to adjacent organs and blood vessels and also left less dead tissue in the field of injury. In addition, new techniques have been introduced to detect and manage those serious complications that occur notwithstanding.

Recurrent abscesses—perhaps *additional* abscesses—occurred in seven of 45 patients (16%). This figure is considerably lower than the 30 to 40% recurrence rate in other series,[7,25,30] and may reflect our practice of thorough abdominal exploration and maximal debridement at the first operation. All seven of these patients had at least one reoperation for surgical drainage, but two of them had one or two other abscesses drained by CT-guided percutaneous catheters when the abscess was small or the surgical access was hazardous because of the residual of previous operations. In contrast to the limited (60%) success of percutaneous catheter drainage as primary therapy for pancreatic abscess,[31] our small experience with its adjunctive use in this situation has been successful. Application of this technique has been advocated also as a temporizing measure in very ill patients to improve their ability to withstand more formal surgical drainage subsequently.

Hemorrhage from the abscess cavity is second only to progressive sepsis as the cause of death from pancreatic abscesses. In this series, 10 patients had one or more major episodes of bleeding after the initial operation. Because of the recognized difficulties in defining and obtaining access to the bleeding site by operation, even before considering the additional injuries to viscera caused in the process and the high likelihood of rebleeding, we have increasingly turned to angiographic means for precise diagnosis and transcatheter arterial embolization for stabilization and control.[32] The bleeding vessel was identified in all four patients in whom it was used, and the bleeding was stopped in three of the four. Transcatheter embolization is not definitive therapy, but a means to convert a situation out of control into one allowing semi-elective re-exploration, debridement, and repacking. Four of five patients who were re-explored for active bleeding died, whereas only one of four died when bleeding was controlled first by embolization.

Fistulas after drainage and debridement of a pancreatic abscess are common because of necrosis from the disease and possibly from injury by the surgeon or the drains. Pancreatic fistulas were seen in 14 of our patients (31%). As has been generally true,[8,12,33] the vast majority of these closed spontaneously within weeks or months. In only one patient was distal pancreatectomy necessary to cure a persistent (7 months) pancreatic fistula.

Enteric fistulas were much more troublesome.[4,34–39] One patient died after segmental infarction and leak from the transverse and descending colon. Colocutaneous fistulas in two other patients persisted and eventually required colostomy, segmental resection of the drained colon, and later re-anastomosis. Neither of two duodenal fistulas healed, and the lone survivor was treated by a right pancreaticoduodenectomy. The lone gastric fistula precipitated urgent gastrectomy to control recurrent hemorrhage, but the patient died.

Contrary to the reported experience of others,[40] we did not encounter any persistent fistulas or other major complications from gastrostomy or jejunostomy tubes placed for drainage or enteral feeding in 20 patients. With these findings, we join those who feel that *selective* use of feeding and draining enterostomy tubes is justifiable and advantageous in some patients.[18,23,33,41] For example, we advocate their use when prolonged gastroduodenal obstruction is anticipated (or has already been present) or when a high enteric fistula makes nutrition a long-term problem. We do not advocate either routine or indiscriminate use.

It has become axiomatic that adequate nutrition is essential to survive an extended catastrophic catabolic illness such as a pancreatic abscess.[6,7,23,34] Total parenteral nutrition was used for a time in 76% of our patients without undue complication and undoubtedly contributed to their survival. Because infection of central venous catheters remains a hazard in the septic patient and further confounds the evaluation of fever sources, we try to avoid intravenous hyperalimentation until the sepsis appears to be under control. An enteral feeding catheter may be particularly welcome when subclavian venous catheters have had to be removed or exchanged repeatedly.

Patients with acute pancreatitis die in one of two principal ways: either in the first week from the shock of fulminant pancreatitis, or weeks to months later from infection and its concomitants. In considering his seeming success with peritoneal lavage in keeping patients with fulminant pancreatitis alive, Ranson was discouraged to note that the ultimate death rate was undiminished because so many patients salvaged initially went on to die at a later time from sepsis.[42] Studies such as the present one must serve to restore a note of encouragement. By applying our best therapy at each stage of acute pancreatitis—early-phase circulatory and pulmonary dysfunction, middle-phase necrosis, late-phase infection—we can reasonably hope to increase overall survival substantially.

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Discussion

DR. JOHN M. HOWARD (Toledo, Ohio): I should like to pay tribute to a wonderful presentation.

It seems to me that one of the important things is the prevention of abscess. As we are improving at carrying patients through the acute episode, we are salvaging patients that would have died a number of years ago, and we are seeing patients with massive necrosis who survive without abscess.

I wonder perhaps if prophylactic antibiotics, during the time when we have many portals of entry, are playing a role in prevention.

(Slide) Here is a patient. This is a debrided necrotic tissue, necrosis without abscess, debridement of almost the entire pancreas.

(Slide) This is a necrotic pancreas debrided 5 months after the acute onset. We thought we were going in to drain a pseudocyst, but there was no pseudocyst. The tissue was dried, almost like a peat bog, with this necrotic pancreas.

In my experience, the things that have predisposed to infection are early operation or ERCP in the presence of necrotic pancreas. As we are not draining pancreases early, we are not seeing abscess very often.

(Slide) Finally, we have recently surveyed our experience over a period from 1962 with nonoperative management of acute pancreatitis excluding posttraumatic, postoperative drug-induced pancreatitis, and our mortality rate is 0.3%.

DR. GABRIEL A. KUNE (Melbourne, Australia): I very much enjoyed Andy Warshaw's paper, and although his view of the world is from Boston, this is kind of our view of how we visualize the world (Slide). Thus, there is a difference in our geography.

(Slide) But the interesting thing is that if I had to give that paper, it would have been in almost exactly the same way as he has done it, right down to the last decimal point.

We have also experienced a continual improvement in the survival of pancreatic abscesses, and this is our experience in the last 15 years, let's say not a quantum leap but a quantum pull with the advent of nutritional support and CT scanning. Thus, we, as have they, have experienced an incredible, improvement in the survival of these people.

At the same time, the number of complications and the number of reoperations that are needed are almost exactly the same as are his.

The other comment I would like to make is that percutaneous drainage of the original abscess has been recommended in several places, but we really would be against that because we have been as unsuccessful as his group has at this procedure, mainly because the original abscess is really an infected slough. This type of situation would be rather difficult to remove with a needle, and, therefore, we would advocate an open operation in exactly the same way that he performed it.

For postabscess recurrence, we also have had good luck with percutaneous drainage, but not with the original abscess. That is my comment, and my question is the following (and this has also been alluded to by Dr. Howard). In the last 5 years, we have started systematically to do necrosectomies and sequestrectomies in the severe cases. It is our impression that, although we do not have sufficient data and sufficient numbers to support it, we have decreased the number of subsequent abscesses with this type of operation. Our problem is that we cannot