Surgical Treatment of Acute Necrotizing Pancreatitis

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ALTHOUGH acute pancreatitis has been recognized as a discrete entity since its original description by Reginald Fitz in 1889,[9] it has remained a disease of incompletely undertsood pathophysiology and multiple apparent etiologies. Acute pancreatitis pursues an extraordinarily variable course ranging from mild edematous pancreatitis mimicking biliary colic or acute gastritis to a fulminant necrotizing process with hemorrhage, cardiovascular collapse and a rapidly fatal natural history.

Since the classic description of Opie in 1901,[18, 19] the generally accepted therapeutic program for acute pancreatitis has undergone substantial evolutionary development. Emergency operation with a direct attack upon the inflamed gland was advocated in the early years of the century. Moynihan[14] in 1925 advocated evacuation of fluid around the pancreas, incision of the pancreatic capsule, and cholecystectomy. DeTakats[7] in 1932 stressed dependent drainage through the left flank. An extraordinarily high mortality characterized these early efforts, however, and through the efforts and arguments of Mikkelsen[13] and others, a program of supportive, nonoperative therapy developed. Conservative management of acute pancreatitis has held sway, virtually unchallenged, to the present. The therapeutic regimen has been directed at restoration and maintenance of normal blood volume and electrolyte balance, suppression of pancreatic secretion, relief of pain, treatment of hypocalcemia, and prevention or treatment of infection with antibiotics.

While highly effective as a therapeutic regimen for the majority of cases of acute edematous pancreatitis, conservative therapy has been less than satisfactory in dealing with the more severe cases of fulminant, necrotizing pancreatitis accompanied by extensive fat necrosis, hypocalcemia, and progressive failure of multiple organ systems. Failure to cope successfully with this more severe variety of pancreatitis has led, in recent years, to a reappraisal of a surgical approach to selected cases of acute pancreatitis which fail to respond to a nonoperative therapeutic program. Howard and Jordan,[11] Pollock,[21] and Nardi[15] all have pointed out that exploratory laparotomy does not necessarily increase the mortality of acute pancreatitis and, in the least, enables one to avoid overlooking a potentially correctible alternative diagnosis, such as a perforated viscus, in a deteriorating patient. In 1968 Waterman *et al.*[25] reported encour-

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aging results from sump drainage of the lesser sac as a primary procedure for acute pancreatitis. These investigators also demonstrated beneficial effects of sump drainage in experimental acute pancreatitis in guinea pigs. Anderson[1, 2] has advocated early operative intervention in patients who do not respond within 12 hours of instituting medical therapy. He has stressed the goal of avoiding errors in diagnosis and treating complications of pancreatitis such as pseudocyst or abscess formation. Zinberg[27] as well has proposed an aggressive surgical approach to the deteriorating patient with acute pancreatitis and has emphasized the value of peritoneal lavage and appropriate drainage.

Over a period of 2 years we have operated upon a total of 15 desperately ill patients with pancreatitis in the acute phase. These patients represent approximately 5% of all patients with acute pancreatitis seen at the Massachusetts General Hospital during this period of time. The operative program (devised by W. M. D.) consisted of tube decompression of the biliary tree, gastrostomy, feeding jejunostomy, and sump drainage of the lesser sac and retroperitoneum.

In the first two patients in whom this procedure was carried out, laparotomy had been performed for uncertain diagnosis. At operation acute necrotizing pancreatitis was found and the procedures described above carried out. The results were so gratifying that the procedure was extended to patients with clearcut severe acute pancreatitis who were deteriorating and who had exhausted all non-operative therapeutic maneuvers. Decision to operate was made in most cases when the patient required pressors after adequate volume replacement or when endotracheal intubation and assisted ventilation were necessitated by respiratory failure. In comparable groups of patients reported by Foster[10] and Nugent,[16] mortality of 50 to 90% has attended supportive, non-operative therapy.

Methods

Before inducing anesthesia an arterial cannula was placed (if not already present) in the radial artery so that constant monitoring of arterial blood pressure and frequent determinations of arterial blood gases could be carried out. An adequate intra-thoracic central venous pressure line and a Foley catheter were already in place. Continuous electrocardiographic monitoring was also carried out.

The abdomen was opened through a midline incision, and the diagnosis of acute pancreatitis was confirmed. Extensive retroperitoneal phlegmon, often bloody, as well as extensive fat necrosis was encountered in every case (Figs. 1, 2). After evacuating any stones present in the gallbladder, a mushroom catheter was introduced, secured by a double purse-string of catgut and brought out through a stab wound in the abdominal wall. Adequacy of cholecystostomy as a technic for biliary decompression was assessed by the appearance of bile issuing from the cystic duct. A #20 straight catheter was inserted into the stomach and secured by a double purse-string of catgut. The gastrostomy tube was brought out through a stab wound, and the stomach fixed to the parietal peritoneum of the abdominal wall with non-absorbable sutures. A #18 straight catheter was introduced into the proximal jejunum as a feeding jejunostomy using the Witzel technic. This enterostomy tube was also brought out through a stab wound and secured to the parietal peritoneum with non-absorbable sutures (Fig. 3).

Multiple drains were placed to the pelvis, left upper quadrant, right sub-diaphragmatic space, and right sub-hepatic space. A sump, either of the type designed by Waterman[25] or a commercially available * #28 sump, was introduced through a counter incision in the left flank and

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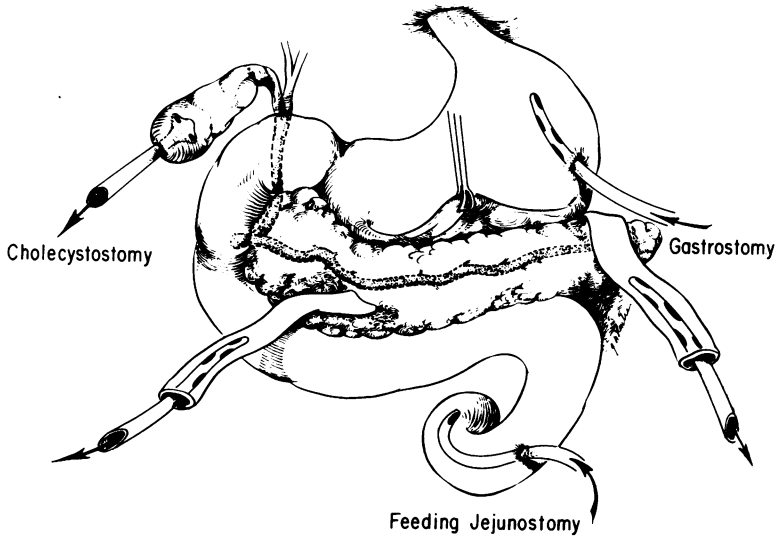

FIG. 3. Diagrammatic representation of surgical procedures applied to acute necrotizing pancreatitis. See text for description.

threaded retroperitoneally to the area of the tail of the pancreas.

Closure of the abdomen was effected by figure-of-eight #28 wires through anterior fascia and peritoneum as described by Spencer.[22] The wound was closed primarily in most instances, although delayed pimary closure was performed in several instances.

The vigorous supportive measures instituted preoperatively were continued. The postoperative patient is shown in Figure 4.

Results

The 15 patients selected for operative treatment of pancreatitis represented a variety of apparent etiologies. Eight were chronic alcoholics, three had biliary calculous disease, and in single instances pancreatitis followed trauma, previous elective surgery, and a recent viral illness.

Hypocalcemia and shock, generally accepted as ominous developments in pancreatitis, were prominent in the group, reflecting the severity of their disease. Edmondson[8] has noted that a serum calcium under 7 mg./100 ml. has been considered to indicate a hopeless prognosis. In this series hypocalcemia occurred in 13 of 15 patients (Fig. 5). Seven had values under 7 mg./100 ml. and one below 6. Foster and Ziffren[10] reported an 86.9% mortality in a group of 23 patients with pancreatitis in shock. Three of our patients presented with systolic blood pressures less than 80 mm. Hg, and 11 of 15 had systolic blood pressures below 90 mm. Hg develop prior to surgical intervention.

Serum amylase did not appear of prognostic value. While eight patients had amylase values above 150 Russell units (normal 0–25), the remainder had less elevated values, and one patient had a normal serum amylase on admission. Seven patients had serum lipase values over 4 cc. Leucocytosis was the rule, and eight of 15 patients had admission hematocrits over 45%, reflecting dehydration and hemoconcentration.

Radiologic findings were non-specific, but seven patients had pleural effusions and six had dilated loops of small bowel on abdominal films.

Of the 15 patients subjected to surgery, 11 survived and left the hospital well. This represents a mortality rate of 26% in this group of patients with extremely severe pancreatitis. Of those who succumbed, all survived the immediate postoperative phase and died at intervals of 11 to 35 days after operation. The 11 survivors had postoperative convalescences ranging from 11 to 124

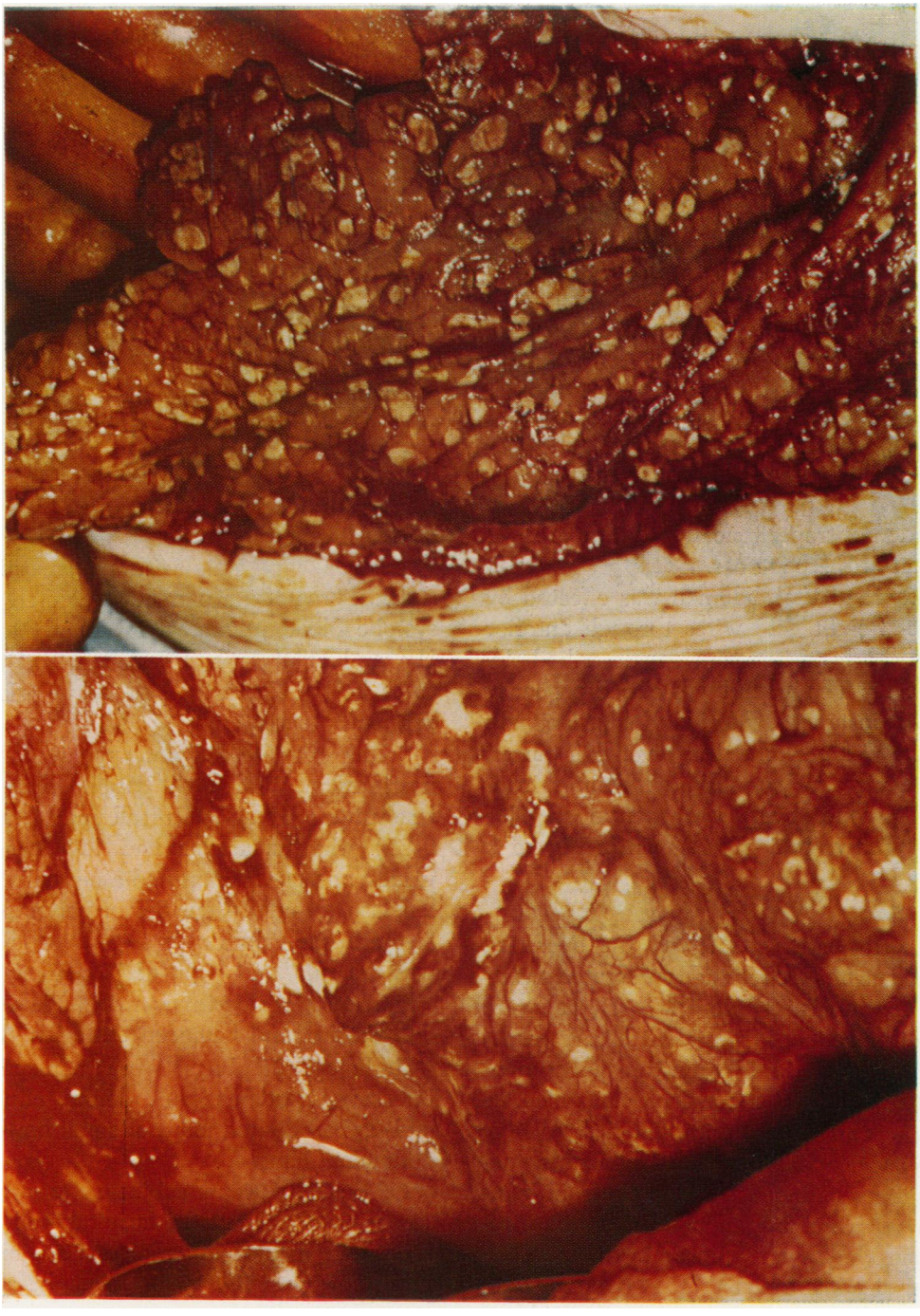

FIG. 1. (top) Retroperitoneal phlegmon with fat necrosis caused by acute necrotizing pancreatitis.

FIG. 2. (bottom) Extensive fat necrosis in greater omentum associated with severe acute pancreatitis.

days with a mean of 49 days. Although immediate improvement occurred in all patients, often with dramatic stabilization of cardiovascular and respiratory disorders, duodenal obstruction proved to be a long-term problem. Two patients had frank obstruction from duodenal compression, and five others had persistent widened duodenal "C loops" noted on upper gastrointestinal series. One patient required antral exclusion and vagotomy to restore gastrointestinal function, as passage of food through the duodenal C-lopo and apparent consequent hormonal stimulation repeatedly exacerbated the pancreatitis. In two other patients clamping of gastrostomy and/or cholecystostomy was followed by exacerbation of the pancreatitis, requiring further periods of gastrointestinal and bilious decompression. All patients appeared to benefit from enteric replacement and high caloric alimentation through the feeding jejunostomy.

Among the 11 survivors, staphylococcal or pseudomonas pneumonia was successfully treated in six patients. Seven patients had positive bile cultures taken at the time of operation which were gram negative organisms in all cases. One survivor discharged necrotic pancreas through a retroperitoneal sump site 2 months after operation.

Follow-up of surviving patients has revealed subsequent pancreatitis in five of 11 instances. In all cases these attacks have been far less severe than their initial episodes and have been easily managed by medical therapy. Three patients have undergone elective cholecystectomy and common duct exploration and two had sphincterotomies as well. Of the six alcoholics who survived, four have, not unexpectedly, returned to their habit in spite of psychiatric and social service care.

In all four patients who died, sepsis played a major role. Postmortem examinations were done on three of four patients and revealed massive pancreatic necrosis.

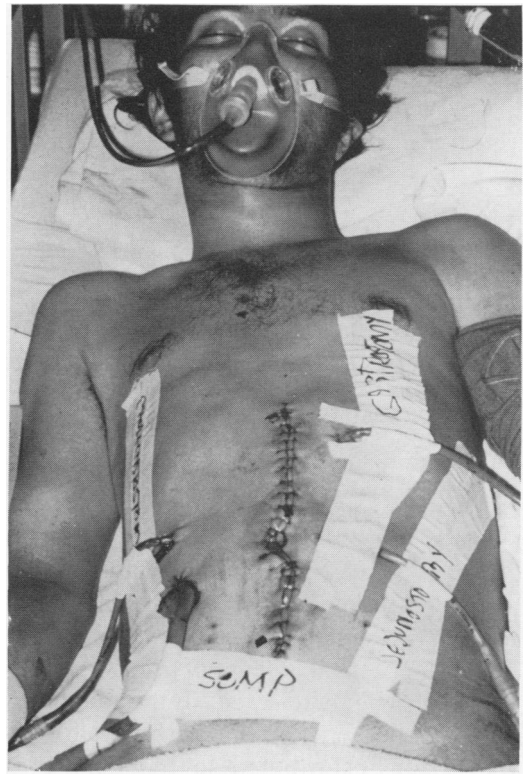

FIG. 4. Postoperative patient with severe acute pancreatitis following gastrostomy, cholecystostomy, feeding jejunostomy, and drainage.

Severe bilateral pneumonia was present in each instance. Each of these patients had survived the early phase of the disease characterized by hypovolemia and cardiorespiratory failure as described by Nugent,[17] only to succumb to late sepsis, dying 11, 24,

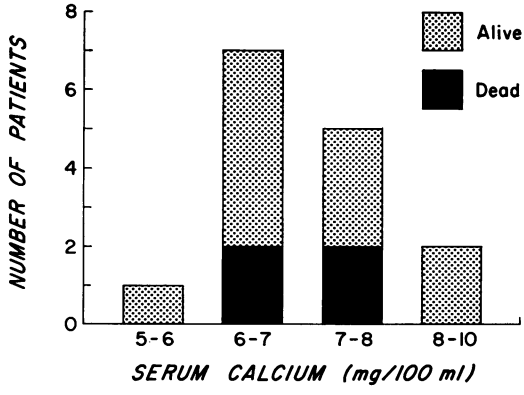

FIG. 5. Serum calcium levels in patients subjected to operation for severe acute pancreatitis.

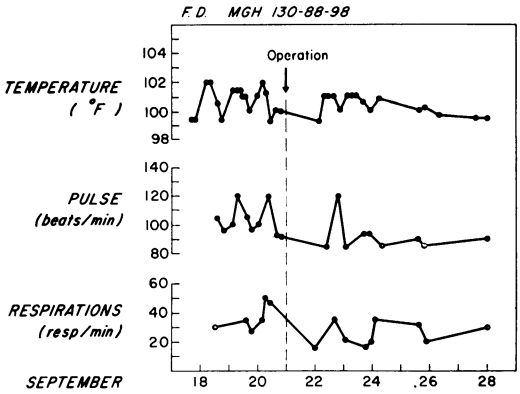

FIG. 6. Vital signs in patient 3 before and following operation for acute necrotizing pancreatitis. Respiratory failure necessitated endotracheal intubation and assisted ventilation 48 hours prior to operation.

26, and 35 days after operation. In each instance necrotic pancreas with secondary infection, presumably bloodborne, provided a retroperitoneal focus of sepsis which could not be eradicated. Intra-abdominal and retroperitoneal hemorrhage from necrotic tissue contributed to the demise of two of the four patients.

Laboratory data on admission, radiologic findings, or etiologic background of pancreatitis did not seem to carry prognostic significance in determining a satisfactory response to the described program of operative management.

Case Reports

Case 1. C. D. A 46-year-old woman entered the M.G.H. because of severe left lower quadrant pain of several hours' duration. She had been in previous good health with no history of alcoholism, pancreatitis, or biliary disease. There was no familial incidence of hyperlipemia.

Admission physical examination revealed a silent, diffusely tender abdomen with rebound tenderness maximal in the left upper quadrant. Laboratory data revealed an amylase of 174 Russell units (normal 0–25), calcium of 6.7 mg./100 ml. (normal 9–11), and marked hyperlipemia with total lipids of 7,870 mg./100 ml., total serum fatty acids of 1,550 mg./100 ml., and serum triglycerides of 910 mg./100 ml. X-rays on admission revealed a small left pleural effusion but were otherwise negative.

A conservative regimen for acute pancreatitis was initiated including antibiotic agents, nasogastric suction, analgesics, anticholinergics, and appropriate volume replacement in the form of plasma, blood, and intravenous fluids. In spite of this program the patient deteriorated with persistent abdominal tenderness, fever, tachycardia, increasing volume requirement, and failing ventilatory function, necessitating endotracheal intubation and ventilatory assistance.

At this point laparotomy was carried out revealing 2,000 cc. of brownish ascitic fluid. The pancreas was enveloped in a retroperitoneal phlegmon obliterating the lesser sac. Cholecystostomy, jejunostomy, and gastrostomy tubes were placed as described, multiple drains were inserted, and sumps placed both at the base of the pancreatic phlegmon as well as retroperitoneally by a lateral counter incision.

Postoperatively, the patient was weaned from ventilatory assistance and extubated on her third postoperative day. Jejunostomy feedings were instituted by the end of the first week. She was discharged well on her 34th postoperative day. One month later she was re-admitted for an episode of thrombophlebitis, while feeling well systemically. During her hospital stay she extruded a large amount of necrotic debris with fat necrosis from her retroperitoneal drain site. She has remained well over a follow-up period of 1 year.

Case 2. T. S. A 33-year-old alcoholic man entered the M.G.H., transferred from another hospital because of failure to respond to a medical program for acute pancreatitis. Serum amylase on admission to that hospital 48 hours earlier was 3,000 Somogyi units.

On admission to the M.G.H. he was noted to be extremely dyspneic and cyanotic. Vital signs included a pulse of 160, respiratory rate of 40, and a temperature of 40.1° C. He was judged to be in high output respiratory failure and was promptly intubated and placed on mechanical ventilatory assistance with 100% oxygen. Initial laboratory values included an amylase of 285 Russell units, calcium of 6.8 mg./100 ml., and bilirubin of 2.7 mg./100 ml.

After appropriate volume replacement and antibiotic therapy, he was taken to the operating room 12 hours after admission where laparotomy revealed acute pancreatitis with extensive retroperitoneal hemorrhage and fat necrosis throughout the peritoneal cavity. The omentum was indurated, and the mesentery of the small bowel and transverse mesocolon was foreshortened and edematous. The lesser sac was completely obliterated by the inflammatory process. Cholecystostomy, gastrostomy, and feeding jejunostomy were placed, as

well as multiple drains and a sump to the base of the pancreatic phlegmon.

Over the next 3 days the patient improved significantly and was weaned from his endotracheal tube. On the fifth postoperative day a sizable left pleural effusion developed. Thoracentesis revealed fluid with a high amylase concentration. He demonstrated a persistent colloid and crystalloid requirement necessitating administration of 5,000 to 6,000 cc. daily. In spite of close monitoring of urine output and central venous pressure, the patient became azotemic with a BUN of 100 mg./100 ml.

By the 8th hospital day respiratory failure had once again become a problem, and tracheostomy was carried out. On the 19th postoperative day he developed a temperature of 39.1° C., and blood cultures grew *Staphylococcus aureus*. Radiographs of the abdomen revealed a mottled, "moth-eaten" appearance in the left upper quadrant. The diagnosis of a septic retroperitoneal collection of necrotic pancreas was entertained. Failure to control the sepsis led to re-operation; exploration of the retroperitoneum revealed a necrotic, foul smelling, purulent mass completely replacing the pancreas. It was possible to evacuate this material but with considerable blood loss.

Postoperatively the patient required pressors, became anemic, and progressive hypoxia supervened. In spite of maximal ventilatory support, death ensued on the 25th hospital day. Postmortem examination revealed acute hemorrhagic pancreatitis with pancreatic necrosis and extensive fat necrosis, bilateral pneumonia, and acute tubular necrosis of the kidneys. The biliary tree was normal. The pancreatic duct of Wirsung joined the common bile duct 3 mm. before a common entrance into the duodenum.

Case 3. F. D. A 34-year-old man entered the M.G.H. with abdominal pain and vomiting of 1 day's duration. A chronic alcoholic, he had been drinking steadily for 3 weeks prior to admission. Initial examination revealed him to be in shock with blood pressure of 70/40, pulse of 120, and respiratory rate of 20. There was diffuse abdominal pain with rebound tenderness greatest in the upper abdomen. The abdomen was silent.

Admission laboratory data included an hematocrit of 53% and a white blood count of 21,300 mm^3. Amylase was 275 Russell units, BUN was 48 mg./100 ml., and calcium was 8.4 mg./100 ml., dropping to 6.9 mg./100 ml. over the first 12 hours.

The patient was resuscitated with six units of plasma, 2,000 cc. of Ringer's lactate and placed on a maximal conservative regimen for pancreatitis. Vital signs initially stabilized but fever, epigastric

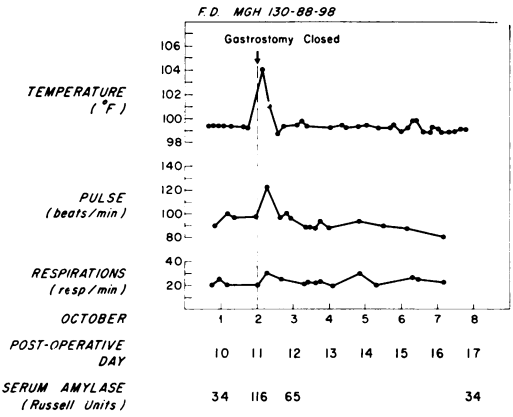

FIG. 7. Patient 3 in whom gastrostomy was clamped on 11th postoperative day followed by immediate exacerbation of pancreatitis. Several similar episodes occurred over several weeks.

tenderness, and rebound persisted. On the third hospital day the patient developed severe respiratory distress and required tracheal intubation and assisted ventilation. Progressive deterioration under conservative therapy was taken as an indication for operation. Exploratory laparotomy on the fifth hospital day revealed acute pancreatitis with a large retroperitoneal phlegmon and hemorrhage. Cholecystostomy, gastrostomy, and jejunostomy tubes were placed as well as multiple drains and a sump to the pancreatic bed.

Postoperatively, clinical improvement was striking (Fig. 6), and the patient required only a small additional amount of plasma. He required 2 days of ventilatory assistance and was then weaned from the mechanical ventilator and extubated. By the sixth postoperative day he was alert, stable, and taking tube feedings via the jejunostomy.

On the 11th postoperative day after initiation of oral feedings, the gastrostomy tube was clamped. The patient abruptly developed severe abdominal pain, hypotension, and respiratory distress requiring re-insertion of an endotracheal tube (Fig. 7). These events occurred over a 3-hour period subsequent to clamping the gastrostomy tube. Amylase rose to 116 Russell units. Appropriate volume replacement was given, and the gastrostomy was placed to gravity. The patient rapidly recovered over the next 12 hours. He then resumed his benign course with jejunostomy feedings.

An upper gastrointestinal series revealed extrinsic compression of the second portion of the duodenum causing partial obstruction (Fig. 8). One week after the episode noted above, he was again started on oral fluids, but again developed epigastric pain necessitating continued gastrostomy drainage and jejunostomy feedings. Several similar

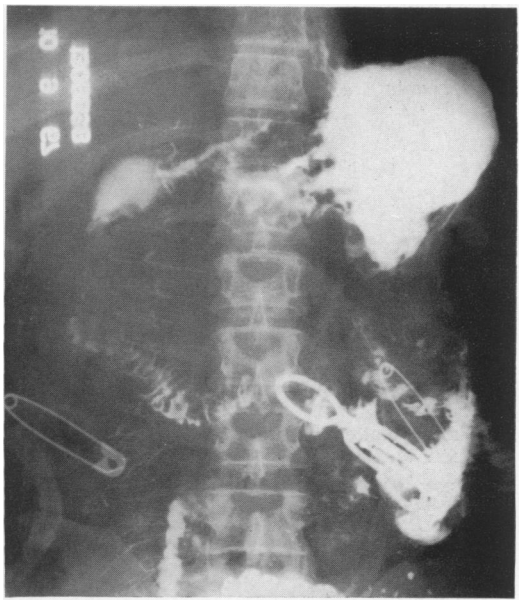

FIG. 8. Partial duodenal obstruction with retention of gastric content in patient 3 secondary to pancreatic phlegmon.

episodes occurred despite subsequent documentation by upper G.I. series (Fig. 9) of resolution of the partial duodenal obstruction. Therefore, 2 months after initial operation, laparotomy was again carried out at which time antral exclusion and vagotomy were effected. At operation the pancreatic phlegmon had resolved, and an indurated but recognizable gland was present.

The postoperative course was benign. He was able to tolerate oral feedings without difficulty and was discharged to a convalescent home. Subsequent follow-up has revealed no further clinical pancreatitis or pancreatic insufficiency. He has, unfortunately, resumed his alcoholic habits.

Discussion

Although the majority of occurrences of acute edematous pancreatitis can be successfully treated by medical means, there is a minority group of instances which go on to pancreatic necrosis and frequently hemorrhage from enzymatic and septic digestion of local vasculature. Little improvement has been effected in the treatment of this group of patients over the past three decades, and mortality rates of 50 to 90% have been consistently reported. While considering surgical intervention in the acute phase an unnecessary insult in the milder forms of pancreatitis, we have applied an aggressive approach of drainage and biliary and gastrointestinal decompression in this more severe group. In this preliminary report a mortality rate of 26% is reported for surgical therapy applied to 15 patients with acute fulminant, necrotizing pancreatitis.

Surgical therapy, while in vogue in the early experience with acute pancreatitis, was discarded by most surgeons in the 1930's and 1940's because of an unacceptable mortality rate attending its application. With improved technics of monitoring and anesthesia and a better understanding and therapy of the metabolic derangements to be encountered, it seems possible today to intervene surgically and reverse the course of the disease by decompression of the biliary tree and gastrointestinal tract and intraperitoneal and retroperitoneal drainage. In the group of patients herein reported, there was no immediate mortality associated with surgery, and all patients were improved after operation, although four patients eventually succumbed to late sepsis. Three of the four patients who died had had clinical pancreatitis for over 72 hours before they sought medical care, while in the group of survivors, none had symptoms for that long, and six had been ill for less than 24 hours.

The benefits of operative therapy appear to be multiple. Nasogastric suction to inhibit pancreatic secretion has long been an integral part of the conservative therapy of acute pancreatitis. This can be accomplished more effectively, for a longer time, and with fewer respiratory complications by the use of gastrostomy. Gastric decompression prevents gastric juice from acidifying duodenal contents and thus initiates the secretin mechanism; and as well, gastric decompression, by preventing antral distension, inhibits the release of gastrin, a direct pancreatic secreatogogue.[20] The importance of gastric decompression was dramatically exhibited in one surgically treated

patient (Case 3) in whom signs of acute pancreatitis re-appeared in a matter of hours after the gastrostomy was clamped. Two other patients demonstrated an exacerbation of the disease following gastrostomy and/or cholecystostomy tube clamping.

Drainage and decompression of the biliary tree has also been practiced for many years and by itself has not been considered to influence the course of acute pancreatitis.[10, 23] However, cholecystostomy has obvious potential benefit in situations of common duct calculi or biliary sepsis. Although pancreatitis has often been considered a sterile, enzymatic inflammatory process until late secondary sepsis occurs, it is of interest that operative bile cultures were positive for gram negative organisms in seven of the 15 patients. It may be that bacterial superinfection occurs at an earlier time in the course of severe necrotizing pancreatitis than is currently appreciated.

Intubation of the small bowel by jejunostomy serves immediately at operation to decompress dilated small bowel. Its main benefits become apparent later in the patient's course. In many of the patients in this report, long-term functional or mechanical duodenal obstruction occurred, and the jejunostomy served as access for refeeding biliary and gastric secretions as well as for high caloric alimentation when intestinal function has returned.

Acute drainage of the peritoneal cavity eliminates fluid collections which cause elevation of diaphragms with attendant contribution to high output respiratory failure.[4] An alternative method for removal of such intraperitoneal fluid collections has been taken by Bolooki and Gliedman [3] who have reported beneficial effects on severe acute pancreatitis of peritoneal dialysis alone. The therapeutic role of multiple intraperitoneal drains and peripancreatic sumps described herein is perhaps less clear than decompression of the gastrointestinal and biliary tracts. These drains provide a route

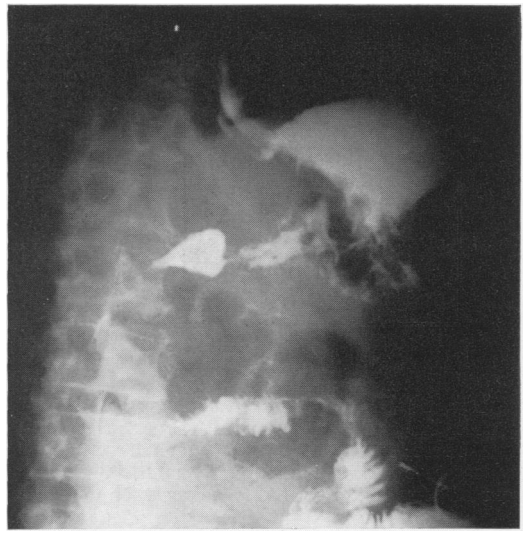

FIG. 9. Resolution of mechanical duodenal obstruction in patient 3 as a result of decrease in size of pancreatic phlegmon which still displaces duodenum. Gastric residuals were low at this time, but clamping of the gastrostomy still repeatedly exacerbated signs of pancreatitis.

of egress for enzymatic-rich peritoneal fluid and may establish a tract through which purulent collections may point. In one instance (Case 1) retroperitoneal sump drainage proved of benefit when the patient was able to slough out frankly necrotic pancreatic tissue through a sump site. On the negative side, however, they serve as a possible source of retrograde infection of potentially sterile abdominal and retroperitoneal spaces.

Much significance has been attached to differentiating "hemorrhagic" from "edematous" pancreatitis. In some instances a clinical diagnosis of hemorrhagic pancreatitis has been made on the basis of the severity of the illness rather than available pathologic material. In the present series of patients "hemorrhagic" pancreatitis was judged to be present by gross inspection at operation in eight of the 15 patients (Fig. 10). There did not, however, appear to be any correlation between mortality, severity of the illness, or extent of retroperitoneal necrosis and the presence or absence of hemorrhage as observed at operation.

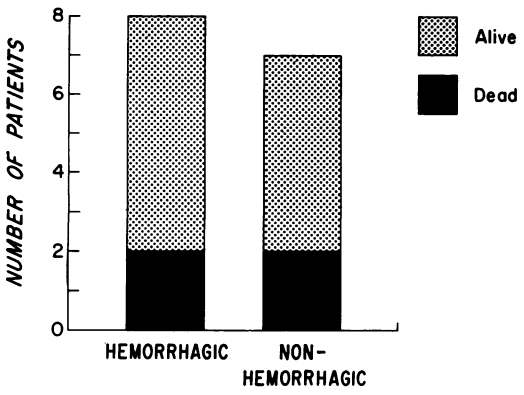

FIG. 10. Hemorrhagic versus non-hemorrhagic pancreatitis as judged by gross inspection at operation. Presence or absence of hemorrhage did not appear to correlate with severity of the illness, extent of retroperitoneal tissue necrosis, or mortality.

Nugent[16, 17] has described the course of acute pancreatitis as divided into a hypovolemic, a cardiorespiratory, and a septic phase beginning at about 2 weeks after the onset of the disease. Analogous to progress in burn therapy, it now seems possible to maintain almost all patients with the most severe pancreatitis through the first two of these phases. Late sepsis, however, remains a difficult clinical problem.[6] All four deaths in this series were due to late sepsis. In no instance did significant pseudocyst or frank abscess formation occur. In those who died, however, a necrotic, septic retroperitoneal mass of cheesy consistency was encountered. This material was not readily amenable to drainage, but the question arises whether early excision might have been the only hope of salvage for these four patients. Watts[26] and Khedroo and Casella[12] have reported subtotal pancreatectomy with success in situations of complete pancreatic necrosis. This technic possibly merits further consideration, although it would appear very difficult to select, while still salvageable, the rare patient who may benefit from such radical therapy.

Summary

Supportive, non-operative therapy has been found effective for most cases of acute pancreatitis. However, conservative therapy has a low salvage rate in patients with fulminant necrotizing pancreatitis which still carries a mortality rate of 50 to 90%.

A group of 15 patients with severe pancreatitis treated surgically is reported. Many of these patients had hypocalcemia and shock prior to operation. Except for two patients explored for diagnostic uncertainty, all patients either required endotracheal intubation for respiratory distress or pressors for blood pressure maintenance prior to surgical intervention.

The operative program consisted of tube decompression of biliary and gastrointestinal systems by cholecystostomy, gastrostomy, and jejunostomy. Multiple intraperitoneal drains and sump drainage to peripancreatic and retroperitoneal areas were placed. Mortality in this group was 26%. All four patients who died succumbed to late sepsis, and in the three examined by autopsy essentially complete pancreatic necrosis was found.

This preliminary report recommends operative intervention in patients with severe pancreatitis who deteriorate under maximal conservative therapy.

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Discussion

Dr. Dan W. Elliott (Pittsburgh): I suspect that I have asked to say a few words primarily because in the past I have been publicly very much against early emergency operations for acute pancreatitis.

This view was based most objectively upon the experience with acute pancreatitis at Ohio State in the 15 years up to 1965. At that time, 31 patients were operated upon during the first week of illness, largely at emergencies, resulting in a mortality rate of 33%, which we thought was prohibitive. This was opposed to an operative mortality rate of 1.67% for 120 patients operated upon electively for gallstones or the drainage of acute abscesses during the second or third weeks of illness, and after stabilization with vigorous supportive therapy. The experience at West Penn Hospital in Pittsburgh during the past 5 years has been the same—an over-all mortality of 7½% and one third of the patients who underwent emergency operation succumbed.

Dr. Daggett's mortality rate of 26% is perhaps a greater improvement over our earlier 33% than it sounds, because their 15 patients had the earmarks of truly fated acute pancreatitis which not all of our earlier patients had.

The rationale for operation is nicely presented. We have often used gastrotomy with local anesthesia for long-term gastric suction. The rationale for drainage of the gallbladder appears logical, particularly if the common channel theory of etiology is accepted. Certainly, I still believe that this is one of the best explanations for acute disease. However, the evidence which Dr. Williams, Dr. Zollinger, and I presented concerning the common channel etiology of acute pancreatitis a number of years ago suggested that the severity of the attack was established within the first hour by the chemical and enzymatic nature of the initial bile and pancreatic mixture flooding the pancreas.

There has been no evidence since this time that continuing pressure from the biliary tree is a problem after the initial insult. However, the presence of *E. coli* in the bile in seven of Dr. Dr. Daggett's fifteen patients suggests that drainage of the gallbladder could be important. Dr. Alan Thompson has certainly presented beautifully documented experimental evidence that *E. coli* can increase the penetration and the toxicity of bile flooding the pancreas in acute pancreatitis.

If the common channel theory is important and if drainage of the gallbladder will help recovery, then the amylase levels in the bile which drains out those catheters in the gallbladder should be elevated for several days after operation. We know that the initial bile amylases taken