Re-evaluation of the Treatment of Pancreatitis Associated with Biliary Tract Disease *

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ALMOST a century ago (1869) Langerhans [7] emphasized the complexity of the anatomical structure and the physiological function of the pancreas. Comparable investigations into the pathological lesions of the pancreas were reported by Opie [8] in 1901. Pancreatitis associated with biliary tract disease became an established clinical entity as a result of Opie's classic description of the common channel theory. Spasm of the sphincter of Oddi in animals was demonstrated by Archibald [1] in 1919 to provide a *common channel.* Metaplasia of the pancreatic ductal system causing obstruction led Rich and Duff [10] (1936) to assume a skeptical attitude toward the common channel theory and they believed it was rarely caused by biliary calculi. They reasoned that "the majority of cases of (hemorrhagic) pancreatitis result from partial obstruction to the outflow of the secretion causing distention and rupture of acini and ductules behind the obstruction, with resulting escape of pancreatic juice into the interstitial tissue." This stimulated considerable controversy and the publication of many papers. Then, in 1938, Warren Cole [3] in a comprehensive review concluded that 60 per cent of patients with pancreatitis had biliary tract disease usually of a calculous nature. Ivy and Gibbs [6] in 1939 likewise emphasized the frequency with which pancreatitis is associated with biliary tract disease as did Lester Dragstedt.[4]

The latter expressed the opinion that 60 per cent of individuals with pancreatitis (acute necrotic) had biliary tract disease but that only 10 per cent of these had a calculus in the ampulla of Vater that caused a common channel. In agreement with Archibald he believed that a *continuous* channel resulted through spasm of the sphincter of Oddi or edema of the papilla resulted from the disease of the biliary tract. John Howard,[5] long interested in the pancreas, has repeatedly called attention to the high incidence of gallstones found at autopsy in those whose death resulted from acute pancreatitis. It is also observed in patients who have had previous operations upon the biliary tract, some with a residual cystic duct remnant with or without calculi. Particularly in this group are also encountered those who have in addition a duodenal ulcer.

On the surgical service of The New York Hospital—Cornell Medical Center, pancreatitis associated with calculous biliary tract disease has been encountered with increasing frequency over the past three decades. The treatment accorded these patients as viewed in retrospect by the authors merits re-evaluation.

Clinical Material

Two hundred and twenty patients with pancreatitis and associated biliary tract disease observed over a 30-year period (1932–1962) have been reviewed as to diagnosis, clinical course, treatment and results. The

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TABLE 1. *220 Patients with Pancreatitis Associated with Biliary Tract Disease*

		No.	Deaths	Autopsies	Mort. Rate %
Group I	Treatment included operation	150	12	10	8
Group II	Treatment without operation	47	22	20	46.8
Group III	Pancreatitis followed operation upon the biliary tract	13	4	4	30.7
Group IV	Pancreatitis followed operation on other than the biliary tract	10	7	7	70
Totals		220	45	41	20.4

diagnosis was often difficult. The clinical picture was one of acute upper abdominal pain which in most instances radiated to the back. Nausea and vomiting occurred sometimes but not always. In the most severely ill prostration was evident. In our experience we found one of the most valuable examinations, contributing to diagnosis soon after admission to the hospital, to be a scout film of the abdomen which revealed a collection of gas in the stomach, proximal duodenum and jejunum just beyond Treitz' ligament, so well described by Poppel [9] in 1951. In our study those who recovered have been evaluated and the pathologic findings of those who have died have been correlated with the sequence of events during the terminal illness. The incidence of the disease (pancreatitis) appears to have increased appreciably during each of the three decades—1932–42, 1942–52, 1952–62—and while an actual increased incidence probably has occurred, it is also true that we are now more alert to the signs and symptoms. Furthermore, we have more efficient methods of establishing the diagnosis.

The presence or absence of biliary tract disease has also been more easily determined over this period. In our experience, an increasing proportion of patients with pancreatitis are being encountered who had previous diagnostic cholecystograms and some have had operations upon the biliary tract (Table 1).

The diagnosis of pancreatitis was confirmed at operation in Group I. The ten autopsies upon the 12 patients who died further demonstrated the virulent nature of the disease and in no instance provided evidence that the operation alone contributed to the fatal outcome. In Group II the autopsy findings in 20 of the 22 individuals who died revealed calculi in the biliary ductal system that could have been the precipitating factor in the pancreatitis and unrelieved the cause of the patient's death. The 13 patients in Group III who developed a pancreatitis following operation upon the biliary tract were recognized with reluctance. Only five were re-operated upon for the pancreatitis. Pancreatitis following operations unrelated to the biliary tract was fatal in seven of the ten patients.

TABLE 2. *Group I—Pancreatitis Associated with Biliary Tract Disease 150 Patients Treated by Operation*

Biliary Tract Disease	No.	Deaths	Autopsies	Mortality Rate (%)
Cholelithiasis	77	6	4	7.8
Cholelithiasis and choledocholithiasis	27	2	2	7.4
Acalculous cholecystitis	24	3	3	12.5
Previous operations on biliary tract	22	1	1	4.5

TABLE 3. *Type of Operation in 150 Patients*

Operation	No.	Deaths	Postmortem	Mortality Rate (%)
Cholecystostomy	20	6	5	30
Cholecystectomy	28	1	0	3.5
Cholecystectomy plus choledochotomy	50	1	1	2
Choledochotomy	17	0	0	—
Cholecystectomy plus choledocholithotomy	27	1	1	3.7
Choledocholithotomy	1	1	1	100
Miscellaneous	7	2	2	28.5

Calculi were present in 104 patients who had not been operated upon previously. These figures are evidence in support of the importance of biliary calculi in pancreatitis. As to those patients with acalculous cholecystitis, two possibilities should be kept in mind, first that calculi may have been overlooked in some and second that the pancreatitis may have preceded the cholecystitis. As has been pointed out by Bisgard and Baker [2] as well as others, pancreatic enzymes have been demonstrated in such circumstances. In only four of the 22 patients who had previous operations on the biliary tract were calculi present at the operation referred to above. We do not know how many of these had calculi at the primary operation. Those patients who had had previous operations upon the biliary tract provided a variety of findings. These included calculi in the common duct, cystic duct remnant with and without calculi, and occasionally a duodenal ulcer that had not been recorded at the primary operation.

Combined with the procedures listed in Table 3, duodenotomy was added in six patients and duodenotomy with sphincterotomy in nine patients.

Cholecystostomy alone in most instances was done because of the precarious condition of the patient. There were six deaths among the 20 patients undergoing cholecystostomy and postmortem examination was performed in five. Autopsy findings revealed a calculus in the gallbladder in only one patient. Cholecystectomy without common duct decompression and/or exploration was usually done in the early part of this three-decade period. The majority of the remaining 102 patients had surgical decompression of the common duct with or without additional procedures. Over the last 15 years this has become the main objective of the surgical approach.

Example Case—Group I. A 46-year-old man with known calculous biliary tract disease was admitted to the hospital in 1954 with severe abdominal pain and findings that led to the diagnosis of pancreatitis (amylase 329). Operation within 12 hours of admission revealed diffuse fat necrosis and a flagrant pancreatitis. A cholecystectomy for cholelithiasis and common duct exploration with removal of calculi was done. A T-tube was placed in the common duct. With heroic supportive measures the patient improved for a few days and then 13 days after the first operation an exploratory laparotomy was done for a spreading peritonitis and drainage of a subhepatic abscess. Forty-eight hours later the patient died. Postmortem examination revealed extensive necrosis of the pancreas, a generalized peritonitis and a well decompressed biliary ductal system.

The pancreatitis was advanced when operation was undertaken. Perhaps simple choledochotomy would have been preferable.

Analysis of 12 deaths, including the autopsy findings in ten, led to the following conclusions.

1. The average age of the 12 patients who died was 60 years with a range from 27 to 81.

2. Biliary tract disease had long been present and many patients had been operated upon previously.

3. Common duct calculi were overlooked and not removed in three patients.

TABLE 4. *Group II—47 Patients Treated Without Operation*

Biliary Tract Disease	No.	Deaths	Autopsies	Mortality Rate (%)
Cholelithiasis	28	16	14	57.1
Previous surgery on biliary tract	19	6	6	31.5
Totals	47	22	20	46.8

4. Operation was evidently undertaken too late in three patients because the pancreatitis had progressed to an extensive necrosis.

5. In addition to biliary tract disease, alcohol played an important role in two, perhaps even greater than the biliary calculi.

The clinical course of the 138 patients who survived operation was reviewed from the hospital records. Fifteen deaths are known to have occurred within 13 years of operation for pancreatitis. The cause was cancer in six, cardiovascular disease in four, and unknown in five. Of 49 patients followed for five years and more, 26 were symptom-free and 23 had complaints compatible with disease of the biliary tract or pancreas. Of 70 patients observed for less than five years, and these for the most part were treated in the last 15 years, 59 were symptom-free and 11 continued to have symptoms. In this group of 138 survivors all but four patients were observed in the follow-up clinic for more than one year.

Pancreatitis in patients with biliary tract disease is well treated by operation so far as averting an immediate fatal outcome, the mortality being 8 per cent. That 85 of 119 patients remained well during observations in the follow-up clinic for a year or more is evidence that the future of these individuals is unpredictable. There are many factors involved such as 1) persistent biliary tract disease; 2) obstruction to the pancreatic ductal system; 3) use of alcohol; and 4) duodenal ulcer and other conditions often unrecognized such as diaphragmatic hernia, diverticulitis, renal disease and coronary sclerosis. Although we lack specific data it is our impression that the use of alcohol is accounting for an increasing pro-

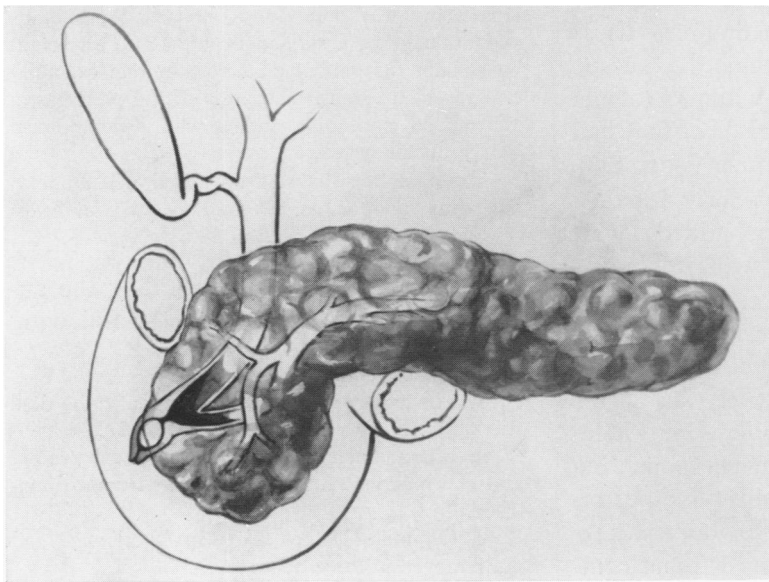

FIG. 1. Line drawing illustrating calculus, common channel and pancreatitis. Pancreatitis associated with biliary tract disease became established as a clinical entity as a result of Opie's classic description of the common channel theory (1901).

portion of *poor results*. The symptoms suggestive of biliary tract disease or chronic pancreatitis (experienced by 34 of 119 patients followed) were in some instances associated with a heavy alcohol intake. Such, however, in our opinion, was infrequent.

In reviewing the group of 47 patients treated without operation it should be emphasized that they were quite ill. They presented the clinical picture of acute upper abdominal pain, usually radiating to the back. Nausea and vomiting was a common but not invariable accompaniment. Almost without exception in the most ill, prostration was marked. Pallor and rapid pulse were noted in many and hypotension developed and preceded vascular collapse, particularly in those who died. Febrile response was variable, as was leucocytosis in the early phases. Serum amylase elevations ranged upward to 600 and in some instances of extensive pancreatic necrosis fell to low levels before death. On x-ray examination a scout film revealed in several Poppel's triad of a collection of gas in the stomach, proximal duodenum and just distal to Treitz ligament.

Because all these patients had actually had biliary tract disease demonstrated previously or had a history that was either unequivocal or strongly suggestive, the matter of a differential diagnosis was invariably present. Acute cholecystitis and perforation of a peptic ulcer required careful consideration. Clinically these were excluded or they would have been operated upon. The hospital records of many of these patients contain the statement that operation was contraindicated because of the pancreatitis, but that the biliary tract disease that probably was the cause should be corrected after the patient had recovered. And indeed this was generally done for those patients who recovered. It should be added that the hospital record and summaries of all the patients who died, as well as many who recovered, stressed that at the time of admission and for some time thereafter they were so ill, and their condition so grave, that they were considered unsuitable for any operative procedure.

TABLE 5. *47 Patients Treated Without Operation Over Three Decades*

	1932–1942	1943–1952	1953–1962
Cholelithiasis	2	10	16
Previous surgery on bilary tract	1	3	15
Totals	3	13	31
Deaths	2	9	11
Mortality rate	66.6%	69.2%	35.5%

There were 22 deaths amongst the 47 patients. They ranged in age from 20–75, the average age being 58. Autopsies confirmed the diagnosis in 20. In 12 of these, pancreatitis alone was the chief cause of death.

Example Case—Group II. A 72-year-old woman was admitted to the hospital in 1935. She was acutely ill with symptoms compatible with acute cholecystitis. Within three days she exhibited signs of a generalized peritonitis and a left pleural effusion, her temperature reached 40° C. and her leucocyte count 22,000 on the sixth hospital day. Two days later she died in cardiovascular collapse.

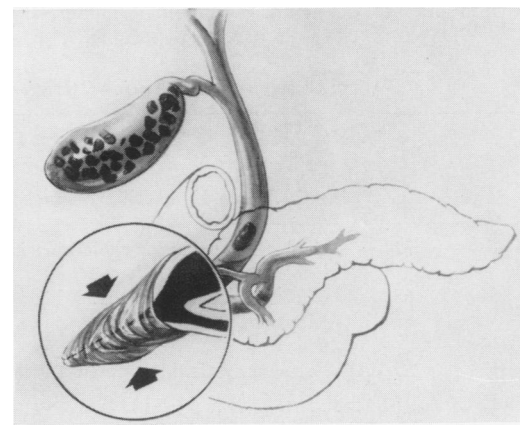

FIG. 2. Line drawing indicating spasm of the sphincter of Oddi associated with cholelithiasis. Archibald (1919) demonstrated the role of the sphincter in producing the common channel in animals.

TABLE 6. *Pancreatitis Following Operation Upon the Biliary Tract for Calculous Disease*

		No. Patients 13	Survivors 9	Deaths 4
Survivors Age	Sex	Type of Operation (Primary)		Re-operation for Pancreatitis
70	M	Cholecystectomy for cholelithiasis		Exploratory Laparotomy
27	F	Cholecystectomy for cholelithiasis		
56	F	Cholecystectomy for cholelithiasis		
73	M	Choledocholithotomy*		Gastrojejunostomy
23	F	Cholecystectomy for cholelithiasis		
70	F	Cholecystostomy for cholelithiasis		Cholecystectomy (cholelithiasis) Choledochotomy
50	F	Cholecystectomy for cholelithiasis Choledochotomy		
64	F	Cholecystectomy for cholelithiasis Choledochotomy		
37	F	Cholecystectomy for cholelithiasis		

* Patient underwent cholecystectomy for cholelithiasis and choledocholithotomy 3 years previously.

At postmortem examination anterior to the pancreas throughout its length there was a cavity containing a mass of soft necrotic material which was evidently changed fat. This cavity was formed by a wall that was indurated and was made up of stomach and omentum. The surface of the pancreas in contact with the cavity was in places gray and in places somewhat yellowish and opaque. A sinus communicating with this cavity included a small pocket filled by necrotic material behind the superior mesenteric artery and extended downward in the perirenal fat. In this fat were opaque chalky white spots of fat necrosis measuring 2 to 3 mm. in diameter. The cavity ended at the parietal peritoneum where the ileum was adherent by fibrous adhesions and in this area there was perforation into the ileum 3 mm. in diameter. In the surrounding fat there were a moderate number of areas of fat necrosis similar to those described.

Dr. Eugene Opie [8] then Professor of Pathology examined and described the following findings.

"Pancreas: The pancreas is much smaller than normal and it is not distinctly outlined. It is dark gray in color, mottled with large areas of light grayish white. These latter areas can be easily separated from the rest of the pancreatic tissue. No normal pancreatic tissue is seen. The pancreas is bound to the surrounding structures by greenish red fibrinous adhesions. Cut section of the pancreas was not made at this time." [Later the microscopic examination revealed complete necrosis.]

"On opening the duodenum the bile papillae is conspicuous but just above is a high elevation

TABLE 7. *Pancreatitis Following Operation Upon the Biliary Tract for Calculous Disease*

		No. Patients 13	Deaths 4	Autopsies 4	Mortality Rate 30.7%	
Sex	Age	Operation Upon Biliary Tract		Operation for Pancreatitis		Cause of Death
F	72	Cholecystectomy for cholelithiasis Choledochotomy		None		Pancreatitis
M	65	Cholecystectomy for cholelithiasis Biopsy of pancreas		(1) Drainage of pancreatic cyst (2) Drainage of intra-abdominal abscess		Pancreatitis Peritonitis
F	59	Excision cystic duct remnant Choledocholithotomy Duodenotomy		Exploratory laparotomy with drainage for peritonitis		Pancreatitis
F	51	Choledochotomy**		None		Pancreatitis

* Cholecystectomy 31 years previously.
** Cholecystectomy 2 years previously.

TABLE 8. *Pancreatitis Following Operation Unrelated to the Gallbladder or Ductal System*

No. Patients	Survivors	Deaths
10	3	7

Survivors		Primary and Unrelated	
Sex	Age	Operation	Operation for Pancreatitis
F	39	Ureterolithotomy	Cholecystectomy (cholelithiasis) Choledochotomy
M	7	Splenectomy*	None
F	53	Duodenotomy** Biopsy of pancreas	(1) Gastrojejunostomy (2) Dismantling of gastrojejunostomy Jejunostomy

* 9 months later patient underwent elective cholecystectomy for cholelithiasis noted at time of splenectomy.
** Cholecystectomy for cholelithiasis 17 years previously.

measuring 12 × 8 mm., raised above the surface 7 mm. On opening the common duct it was dilated and found to contain more than 40 rounded stones ranging from 3 to 10 mm. Where the bile duct enters the intestinal wall, dilatation ceases. Following the duct by dissection a pocket is found 9 mm. from its duodenal orifice and in this pocket causing the projection of the mucosal described above is a gallstone 9 × 7 × 4 mm. The pancreatic duct joins the bile duct 3 mm. from its duodenal opening and the latter is 2 mm. in diameter. The hepatic ducts 2 cm. within the liver contain gallstones.

Note: The gallstone does not occupy the diverticulum of Vater, but lies in a pocket formed by the mucosa of the duodenum. The lesion of the pancreas is best explained by assuming there has been necrosis of . . . the pancreas with formation of an overlying cavity containing necrotic material."

In the remaining eight patients the primary immediate cause of death was listed as due to cardiovascular disease in four, hepato-biliary disease in three, and renal disease in one. Pancreatitis was listed as a secondary cause in all eight. In the authors' opinion surgical therapy upon the common duct could have been expected to have provided an opportunity for survival with the exception of those who were moribund when admitted and who were demonstrated to have extensive pancreatic necrosis at postmortem.

From the data in Table 5 two facts appear significant: 1) an increasing incidence of patients with pancreatitis treated without operation; and 2) a decreasing mortality rate that is indeed high.

Group III. *Thirteen Patients Who Developed Pancreatitis Following Operation upon the Biliary Tract for Calculous Disease.* As a complication following biliary tract operation, pancreatitis of the severity that we have included in this study was encountered in only 13 patients of a total of over 5,600. The development of pancreatitis following an operation upon the biliary tract, regardless of whether it was a primary or secondary procedure, seems to have been the result of trauma. Exploration

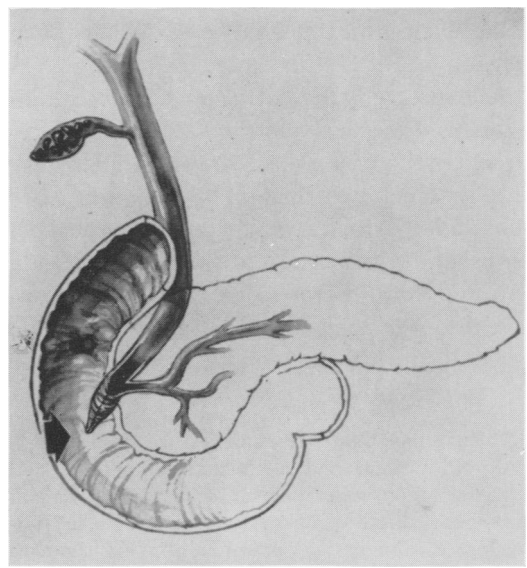

FIG. 3. Line drawing of cystic duct remnant containing calculi and duodenal ulcer in a patient who developed acute pancreatitis three years after primary operation for cholelithiasis.

TABLE 9. *Pancreatitis Following Operation Unrelated to the Gallbladder or Ductal System*

No. Patients	Deaths	Autopsies	Mortality Rate
10	7	7	70%

Sex	Age	Primary and Unrelated Operation	Operation for Pancreatitis	Autopsy Findings
F	69	Ileostomy	None	Pancreatitis Calculi in gallbladder and common duct
M	62	Prostatectomy	None	Pancreatitis Calculi in gallbladder and common duct
M	59	Bowel Resection	None	Pancreatitis Acute cholecystitis Calculi in gallbladder and common duct
F	77	Ureterolithotomy	None	Pancreatitis Cholelithiasis
F	75	Thyroidectomy*	None	Pancreatitis No biliary calculi
M	44	Pyelolithotomy**	None	Pancreatitis No biliary calculi
F	68	Ureterolithotomy***	None	Pancreatitis No biliary calculi

* Cholecystectomy and common duct exploration 9 years previously.
** Cholecystectomy 7 years previously.
*** Cholecystectomy 6 years previously.

of the common duct with considerable instrumentation directed toward the duodenal portion was done in seven patients. In the latter disturbance, retraction upon the pancreas, which may have been the site of some degree of inflammatory reaction that went unrecognized, could have been an important factor. However, if trauma is the cause of pancreatitis in these circumstances then we should see it more frequently than we do. Operative cholangiography which was done at operation in three of these patients may have produced a subsequent spasm of the sphincter of Oddi, an important factor in the etiology of pancreatitis. A long distal arm of a T-tube passed into the duodenum in two of these three patients.

Example Case—Group III. In 1962 a 59-year-old woman had a choledocholithotomy and duodenotomy with removal of impacted calculi from the distal common duct. During the procedure a false passage was made in the common duct. A long arm T-tube was passed into the duodenum. Within a few days she had developed pancreatitis. A flagrant pancreatitis was verified at operation. The T-tube was replaced by simple common duct decompression and a transabdominal gastrostomy performed. The patient's condition failed to improve and she died five days later. At postmortem the pancreas was necrotic and there was a diffuse fat necrosis throughout the abdomen. The biliary ductal system was not dilated and contained no calculi.

Extensive instrumentation probably contributed to the subsequent pancreatitis. The placing of the distal arm of the T-tube through the ampulla into the duodenum may have also been a factor.

The ineffectiveness of common duct decompression established at operation in three of the 13 patients is disconcerting. At autopsy a common channel was demonstrated in all three and no calculi were present.

Group IV. *Ten Patients with Biliary Tract Disease Who Developed Pancreatitis Following Operations Unrelated to the Gallbladder or Ductal System.* Various gradations of pancreatitis have been reported following trauma and surgical procedures to all parts of the body. These range from elevation of the serum amylase

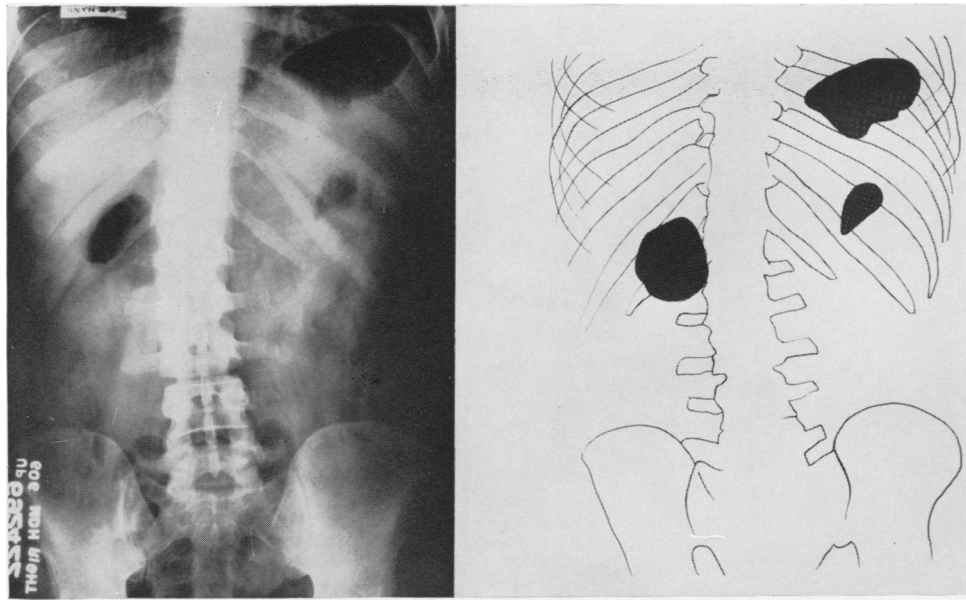

FIG. 4. X-ray and drawing of a scout film revealing gas accumulation in stomach, duodenum and jejunum beyond the ligament of Treitz in patient with flagrant pancreatitis (Poppel's triad).

and other pancreatic enzymes to massive necrosis of the pancreas. The occurrence of pancreatitis in patients with biliary tract disease following surgical procedures unrelated to the gallbladder or ductal system is rare indeed. In our experience over a 30-year period with over 35,000 general surgical procedures, we encountered it in only ten patients.

In all probability patients who would be placed in this category have been overlooked. In only three of the ten patients was the diagnosis of pancreatitis established. All three recovered. The remaining seven were discovered at autopsy.

Example Case—Group IV. A 59-year-old man was admitted in 1956 with severe arteriosclerotic heart disease. Following resection of 150 cm. of the distal ileum for mesenteric thrombosis he experienced a number of minor postoperative complications from which he was recovering when he began to have severe abdominal pain and became jaundiced. He was known to have gallstones. The serum amylase level was 216 and decreased over the succeeding several days as his condition became progressively worse. Death occurred ten days after onset of upper abdominal pain and jaundice. Autopsy revealed the immediate cause of death to be extensive pancreatic necrosis. Cholelithiasis and five calculi obstructing the terminal common duct had provided a common channel. In addition there were findings of advanced cardiovascular disease.

At all times following admission the patient's condition was grave. There were several contraindications to any operation after the onset of the jaundice. The pancreatitis was believed to be the cause of the jaundice and even though it was recognized that calculi might be obstructing the common duct it was elected to withhold operation in anticipation that it would subside.

The mortality rate is indicative of the gravity of the pancreatitis that occurs during the immediate postoperative period following any operation. The implication of biliary calculi in the gallbladder or ductal system is obvious. The role of the pre-existing calculous disease that had been treated surgically previously is problematical.

Discussion

Generally speaking pancreatitis is poorly understood. When it is associated with biliary tract disease we have something tangible to consider. The common duct channel concept set forth by Opie and championed by many investigators since the turn of the century seems plausible in-

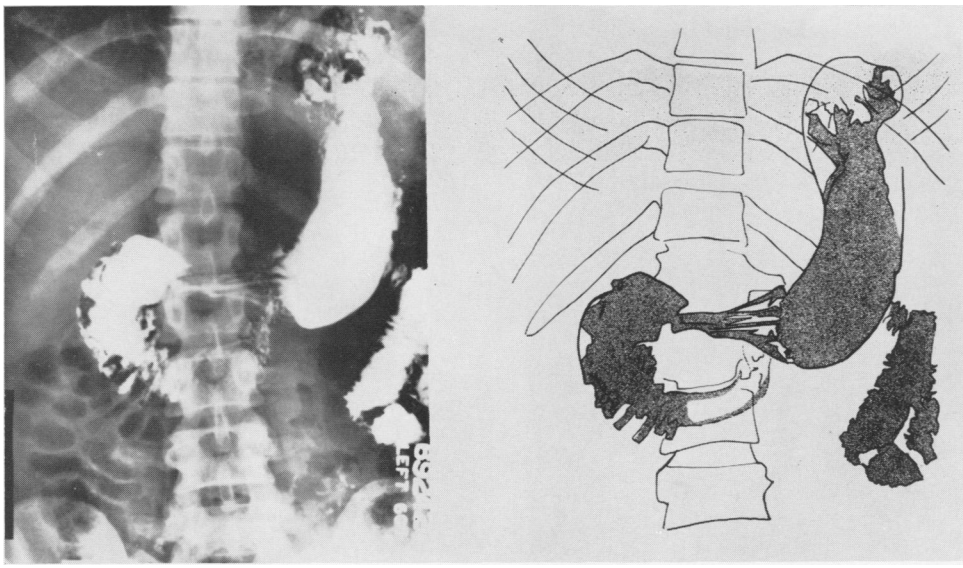

FIG. 5. Roentgenogram and line drawing of barium in stomach, duodenum and jejunum of a patient with advanced pancreatitis.

deed. Since upward to 60 per cent of patients with pancreatitis have calculous biliary tract disease it is logical to assume a causal relationship between the two. Because biliary tract disease can so often be demonstrated to have preceded the pancreatitis by a matter of years, the latter may be looked upon as a sequela of the former. The tendency has been to further postpone the definitive surgical treatment of the biliary tract disease when acute pancreatitis develops. This is justifiable indeed, but decompression drainage of the common duct becomes imperative under these circumstances. Admittedly there may be other factors involved in the etiology of pancreatitis, even in the presence of biliary tract disease, and, therefore, over-simplification of the mechanisms is to be guarded against. Assuming the dominant role of biliary tract disease in the etiology of pancreatitis when they co-exist, the therapy to be employed in avoiding a fatal outcome merits critical evaluation.

The pancreas when obstructed and/or infected is in a diseased state. It is an understatement to say that it is fragile and extremely vulnerable to trauma or disturbance of any kind. Concomitantly, a continuation of the etiologic factors precipitating a pancreatitis causes further injury that may be catastrophic. A mild pancreatitis may thus develop into complete necrosis of the pancreas. Ivy [6] has elaborated on the mechanisms described by Opie, whereby a common channel becomes established in the presence of biliary tract disease. These range from obstruction of the ampulla of Vater by a calculus to spasm of the sphincter of Oddi. Perhaps there are others. The pancreas in an acute pancreatitis thus produced is readily injured by any manipulation, instrumentation and even handling. Any direct attack on the pancreas is admittedly contraindicated. To leave the pancreas unmolested is desirable in order to prevent further injury, but this does not reduce the precipitating factors—obstruction, increased pressure within the ductal system and possible bile regurgitation. When pancreatitis is thus produced, decompression offers the best opportunity for its interruption. This requires an operative procedure. Sometimes the procedures undertaken for

this purpose are not effectual. For example, a cholecystostomy for an acute cholecystitis, if there is obstruction within the cystic duct, may not decrease the pressure within the ductal system or reduce the spasm of the sphincter. However, in the majority of instances some degree of decompression is obtained and when it is the further distention of the pancreatic ductal system is prevented and presumably the irritating and damaging influence of bile and/or pancreatic juice reduced. Under these circumstances one can anticipate that decompression without molesting the pancreas is, on a theoretical basis at least, the ideal treatment. This is indeed actually supported by our clinical experience.

Of course, one of the most important aspects of this problem is how much of the trauma of the operation upon the biliary tract, directly or indirectly, has an effect upon the pancreas and how can this be minimized or avoided. Granted this becomes a bit of a philosophical consideration for discussion but practically it is of critical importance in this group of patients.

The ultimate lesion in pancreatitis is a necrosis of the walls of arteries and veins resulting from the action of pancreatic juice (enzymes) that has escaped from the ductal system. It is reasonable to consider that thinned-out acini, proximal to some form of obstruction, rupture and are thus the source. The pancreatic juice when combined with bile and bacteria may contribute to a more rapid interruption of the integrity of the acini than pancreatic juice alone and likewise be more deleterious to the interstitial structures. Hemorrhage into the interstitial tissues causes further injury. The process may be limited or extensive, dependent upon the nature and duration of the etiologic factors.

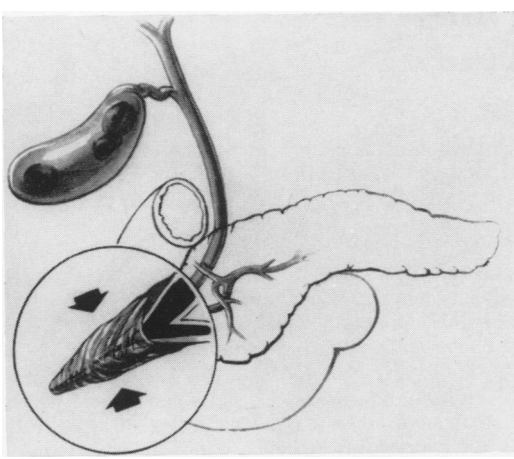

FIG. 6. Line drawing of calculi in the gallbladder and common bile duct together with spasm—a fairly frequent finding in pancreatitis associated with biliary tract disease. (Example case—Group I.)

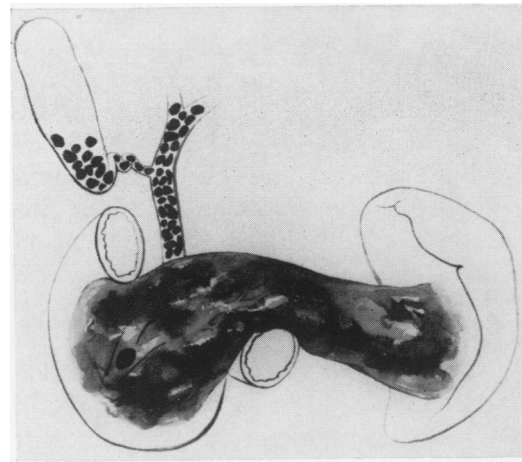

FIG. 7. Line drawing of acute necrotic pancreatitis in a 72-year-old woman examined at postmortem by Dr. Eugene Opie (1935). There were many calculi in the ductal system with one obstructing the terminal common duct.

Conclusion

The present and widely accepted treatment for acute pancreatitis, regardless of its etiology, provides for avoidance of operation in anticipation that it will subside. For those who have associated biliary tract disease, and upwards to 60 per cent are estimated to have this condition, surgical correction is recommended to be done later. Based upon an experience with 220 patients with pancreatitis associated with biliary tract disease, and the findings at autopsy in 41, it seems to us that operation to decompress the common bile

duct as early as feasible after the diagnosis has been established offers the optimum opportunity for recovery. Definitive operations for biliary tract disease may be expected to add trauma to the involved pancreas. We believe such procedures are contraindicated. Perhaps this has contributed to the tendency to avoid operation in the past two decades. If an acute fulminating pancreatitis is present when operation is done, little will be accomplished. Preoperative evaluation to determine the extent or degree of the pathologic process in pancreatitis is difficult and unsatisfactory. The persistently high mortality of pancreatitis associated with biliary tract disease, even with modern supportive measures, is indicative of the inadequacies of the current methods of management.

Pertinent to our recommendation that the surgical treatment of pancreatitis associated with biliary tract disease be re-evaluated is the statement of Eugene Opie [8] (1901) on the treatment of acute hemorrhagic and gangrenous pancreatitis: "What has been said concerning the etiology and pathology of acute inflammation of the pancreas demonstrates the futility of medical treatment directed to the palliation of the lesion."

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Discussion

Dr. Warren H. Cole (Chicago): The exact cause of pancreatitis is not known, but it is well known that in many cases the disease is related to biliary tract disease. In Dr. Glenn and Dr. Frey's report they estimate that this relationship may be as high as 60 per cent; I think that corresponds very closely to our experience.

It is rather unique that in 6 per cent of their cases the pancreatitis followed the cholecystectomy, and in 5 per cent it followed some other type of operation. What could be the cause of this? Often the pancreatitis occurs just a few days after an operation (usually celiotomy). Is it trauma, or is it spasm of the sphincter of Oddi which takes place after cholecystectomy? I think it may be either; at least we must keep both in mind.

It is well known that the disease occurs with varying degrees of severity, and of the 221 cases they report 47 were severely ill, and in that group they did not operate; naturally the mortality rate was high in this group.

We do have to emphasize that now and then some of those sick patients will be improved under chemotherapy and supportive therapy and become operable. Operation will usually be urgent if suppurative cholangitis is present.

They also reported that in 150 cases of the less severe type operation was performed. Up to date I have taken a more conservative attitude in acute pancreatitis without jaundice, usually postponing operation until the patient is over the acute phase. If cholecystography reveals gallbladder disease the operation will be an elective cholecystectomy with or without common duct drainage depending upon indications.