Some Complications of Pancreatic Disease *

Illustrative Cases with Notes on Management

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CERTAIN COMPLICATIONS of pancreatic disease remain perplexing problems in surgical practice. This is due in part to the relative anatomic, physiologic, and roentgenologic inaccessibility of the organ (Fig. 1). It is the purpose here to review some of these complications and to present illustrative cases. The conditions to be considered have been arbitrarily grouped as follows:

I. *Complications of Acute Pancreatitis*
   a. Hemorrhage and electrolyte imbalance
   b. Pancreatic necrosis and abscess formation
   c. Pseudocyst formation
   d. Pancreatic fistula—acute and chronic
   e. Small bowel obstruction
   f. Biliary obstruction with jaundice
   g. Diabetes mellitus

II. *Complications of Chronic Relapsing Pancreatitis*
   a. Intractable pain
   b. Pancreatic calcification
   c. Malnutrition
   d. Most of those listed under I

III. *Complications of Pancreatic Trauma*
   a. Pancreatic ascites and peritonitis
   b. Certain of those listed under I and II

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COMPLICATIONS OF ACUTE PANCREATITIS

Hemorrhage and Electrolyte Imbalance

Shock. The etiology of shock in severe pancreatitis has been attributed to a variety of causes, and the irreversible form is often due to factors not yet fully understood. However, there is still need for an increased awareness of the large volume of blood, plasma, and other fluid that is lost into the peritoneal cavity and into the retroperitoneal space in this disease, if the oligemic shock is to be avoided. Although it has been emphasized that the circulating blood volume may be markedly diminished,[7] transfusional therapy is often delayed because the decline in the hemoglobin and hematocrit values may not occur until after the clinical evidence of shock is present, by which time metabolic changes may have occurred to render transfusion less effective. Unfortunately, one cannot always immediately differentiate between acute edematous pancreatitis and the far more serious hemorrhagic or necrotizing form of the disease. The presence of shock is usually an indication of the more severe condition.

The important therapeutic consideration is that a rapid weak pulse, with a declining blood pressure, is an indication that blood, plasma or plasma expander, plus other fluid, should be infused to replace what may amount literally to liters lost to the circulation. The following case is illustrative.

Case 1. *W. C. Acute Hemorrhagic Pancreatitis.** This 40-year-old white man was admitted to the University Hospital on May 5, 1956 (3:30 a.m.) with the chief complaint of severe abdominal pain of 2 days' duration, accompanied by nausea and diarrhea. A G. I. series performed on May 4, 1956 at another hospital had shown a "widened duodenal loop," and the admission diagnosis was that of pancreatitis. The past history was essentially non-contributory except that there had been a consumption of considerable amounts of alcohol and that "liver trouble" had been diagnosed as the cause of epigastric tenderness one year previously. *Physical examination* revealed a rapid pulse rate but a normal blood pressure. The abdomen was moderately obese and markedly distended, but there was no significant rigidity or palpable mass. Laboratory findings of significance were Hgb. 23.2 Gm. (hematocrit 51%), leukocytes 9,100, fasting blood sugar 327 mg.%, nonprotein nitrogen 51 mg.%, and serum amylase 439 Somogyi units.

His course in the hospital was brief. Approximately 2 hours after admission he went into shock which plasma expander and, later, blood transfusion could not reverse. Autopsy revealed the presence of several liters of grossly bloody fluid in the peritoneal and retroperitoneal spaces, a volume of loss sufficient to produce oligemic shock. Other findings were severe acute hemorrhagic pancreatitis, massive fatty metamorphosis of the liver, and lower nephron nephrosis.

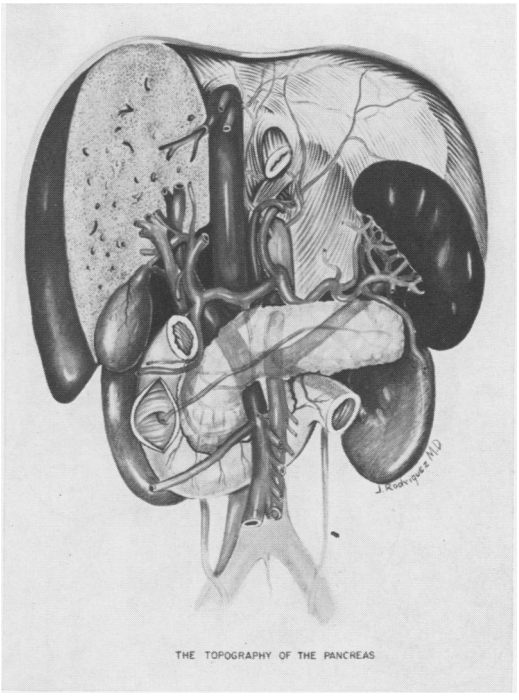

FIG. 1. Pancreatic complications are particularly difficult to manage because of the relative anatomic, physiologic, and roentgenologic inaccessibility of the organ.

Hypocalcemia. Various degrees of hypocalcemia may occur at any time during an attack of acute pancreatitis, but this complication most commonly appears during the first week and may last for from two to three weeks.[1] Large amounts of ionizable calcium are transferred from the serum to areas of fat necrosis to form soaps with fatty acids, and the magnitude of the calcium deficit parallels the degree of the pancreatic disease. Since this parallel does exist, the presence of hypocalcemia is of prognostic value in indicating the severity and thus the probable course of the attack. Rarely does hypocalcemic tetany occur in the absence of the necrotizing hemorrhagic type of the disease and, as indicated, its onset signifies serious disease. The treatment consists of liberal intravenous and oral calcium administration. In our own experience hypocalcemia has rarely constituted a troublesome problem.

Pancreatic Necrosis and Abscess Formation. A severe attack of pancreatitis is often associated with varying degrees of pancreatic necrosis and abscess formation. These collections of necrotic material may be scattered throughout the organ, which is usually markedly swollen, or a number of smaller abscesses may coalesce to form a single large one. Virtually the entire pancreas may become necrotic and slough, rendering the patient diabetic. When pancreatic necrosis with abscess formation is suspected, the alert and persistent examiner will occasionally be rewarded by palpating a mass in the left flank region. Such a retroperitoneal collection can often be most readily evacuated through a left flank incision (Fig. 2), and literally handfuls of necrotic material may be delivered. The

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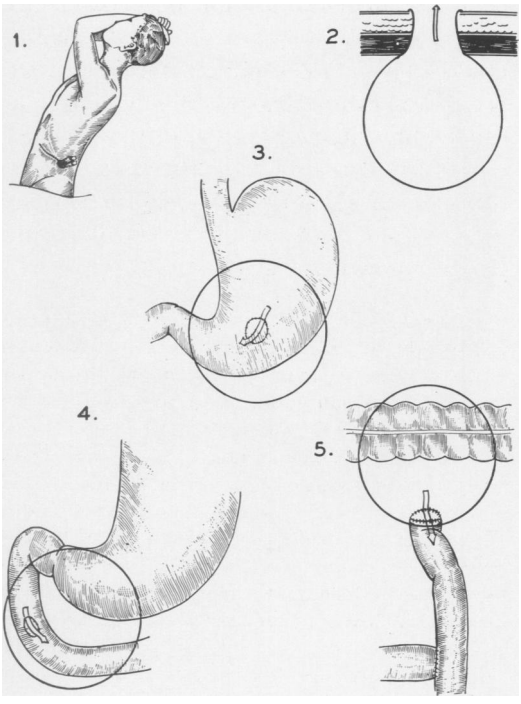

FIG. 2. The use of a left flank incision (1) for retroperitoneal drainage is frequently useful when a pancreatic abscess presents on this side. (2) Marsupialization, where applicable, is still an effective and safe procedure for the drainage of a pseudocyst. (3) Cystogastrostomy is readily achieved by an anterior gastrotomy, to permit an incision through the posterior wall of the stomach into the cyst. (4) Cystoduodenostomy is perhaps the least desirable operation, since a leak in the suture line may result in both a pancreatic and a duodenal fistula. (5) Cystojejunostomy (Roux-Y) usually represents a satisfactory method of internal drainage. If the anastomosis leaks, the continuity of the intestine is not affected, and nutrition can be more readily maintained than in the presence of a duodenal fistula.

initial mortality among such patients is high, and in those who survive diabetes mellitus may develop. The drainage of isolated scattered abscesses is rather unsatisfactory, in our experience.

Pseudocyst Formation. Pancreatic pseudocysts may follow trauma or pancreatitis, or they may arise silently with no history of previous disease or injury. Clinics having a large volume of trauma report a relatively higher proportion of pseudocysts arising following injury than following pancreatitis, while the reverse is true in reports from hospitals where few cases of trauma are admitted.

The wall of the pseudocyst consists of "fibrous" inflammatory membrane, where the wall of a contiguous organ such as the stomach does not serve as one boundary of the pseudocyst. True cysts arise from distention of pancreatic ducts and thus have an epithelial lining. Pseudocysts comprise 60 to 70 per cent of pancreatic cysts, which may be small or may fill almost the entire abdomen. The lesion commonly presents as a silent mass in the upper abdomen, but cysts may be associated with any of the variety of symptoms and signs characteristic of pancreatic disease.

Forms of surgical treatment of pancreatic cysts are:

1. Incision and simple drainage (not often used)
2. Excision
3. Marsupialization
4. Internal drainage

When possible, complete excision of the cyst should be carried out at the initial operation. In the instance of true cysts excision is particularly desirable because of the possibility of malignancy arising from the cells of the epithelial lining.[2] However, this desideratum is feasible in only approximately one-third of the pseudocysts encountered, due to the dense fibrous attachment to surrounding structures such as blood vessels, the common bile duct, and the liver.

Marsupialization, where applicable, still has a definite place in the management of pseudocysts, though recently internal drainage has enjoyed widespread use. Some surgeons prefer marsupialization (Fig. 2) as the initial procedure, followed by internal drainage later if a fistula persists. The three procedures most commonly employed for internal drainage are cystogastrostomy, cystoduodenostomy, and Roux-Y cystojejunostomy (Fig. 2).

The results of internal drainage are usually satisfactory, especially if the cyst is anastomosed to a defunctionalized loop of jejunum. However, occasionally ulceration and necrosis of the cyst wall result in a leak at the cystojejunal anastomosis. Direct anastomosis of the cyst to the stomach or to the duodenum is attended by the largely theoretical hazard of chronic sepsis due to the passage of alimentary material into the cyst. Actually, this usually does not occur —at least barium given by mouth cannot be demonstrated to enter the cyst.

Case 2. *Pancreatitis with Pseudocyst: Cystoduodenostomy.* T. W., a 50-year-old man, was first admitted to the University Hospital on Oct. 2, 1956 with the complaint of severe pain in the epigastrium. An elevated serum amylase level and other findings suggested the diagnosis of pancreatitis, and there was a questionable mass in the epigastrium extending to the right. Cholecystogram was negative, and a positive history of alcoholic intake was not obtained. He improved, declined operation, and went home on Oct. 13, 1956. However, 2 days later the pain recurred and on Oct. 30 he was readmitted. The serum amylase level was again elevated, and an epigastric mass was again palpated. The abdomen was explored and a grapefruit sized pancreatic pseudocyst was found, bounded by the common bile duct, the posterior surface of the duodenum, the duct of Wirsung, and other contiguous structures. After considerable dissection, during which an operative cholangiogram was performed to identify the location of the common bile duct, the operator decided it was unwise further to attempt excision and—as the most expeditious procedure in a patient whose condition was precarious—anastomosed the cyst to the duodenum. The size of the cyst did not permit marsupialization, since it would not extend to the anterior abdominal wall.

The postoperative course was complicated by the development of duodenal and pancreatic fistulas which required further drainage of the subhepatic space. Following this, however, the fistula closed in a very few days.

Pancreatic Fistula—Acute and Chronic. Pancreatic fistulas may develop under a variety of circumstances. Operative injury is a common cause, as is acute pancreatitis. As noted above, another cause of pancreatic fistula is the chronic type which may result from the marsupialization of a pancreatic cyst, particularly when the cyst communicates with a major pancreatic duct. Acute pancreatic fistulas may justifiably be treated expectantly, since the majority will close. A chronic fistula, on the other hand, is less likely to close, and may require surgical intervention. Such an instance is illustrated by the following case.

Case 3. *G. W. Marsupialization of Cyst Leading to Chronic Fistula: Anastomosis of Drainage Tract to Roux-Y Jejunal Loop.* This 45-year-old woman was admitted to the University Hospital on Mar. 15, 1956 with the chief complaint of anorexia and abdominal pain. Three years previously she had begun to experience intermittent epigastric pain and soon after had noted a fullness in this region. At this point a diagnosis of diabetes mellitus was made. In October of 1954 she again experienced a feeling of fullness in the epigastrium and a pancreatic pseudocyst was marsupialized. Postoperatively she did well except for the occasions when drainage from the cyst ceased due to stenosis of the fistulous opening. At such times she became febrile, and her diabetic state, generally mild, required much more insulin. Dilatation of the tract with a Kelly hemostat usually re-established flow.

At laparotomy on Mar. 25, 1956 the drainage tract was freed and anastomosed to a defunctionalized jejunal loop (Fig. 3). It had been planned to excise the sinus with the left portion of the pancreas, but the presence of an abscess with dense surrounding inflammation rendered this unwise. The fistula entered the main pancreatic duct. Convalescence was uneventful, and thus far the patient has remained well.

Small Bowel Obstruction. Mechanical obstruction of the duodenum or jejunum near the ligament of Treitz is a not uncommon complication of pancreatitis (Fig. 1), and we have encountered this problem in three patients during the past year. The obstruction may also occur at the level of the second portion of the duodenum with widening of the duodenal loop due to external pressure. In our patients the bowel was kinked by subacute pancreatic inflammation.

Paralytic Ileus. Grollman [4] has emphasized the importance of the "sentinel loop" which represents a distended segment of

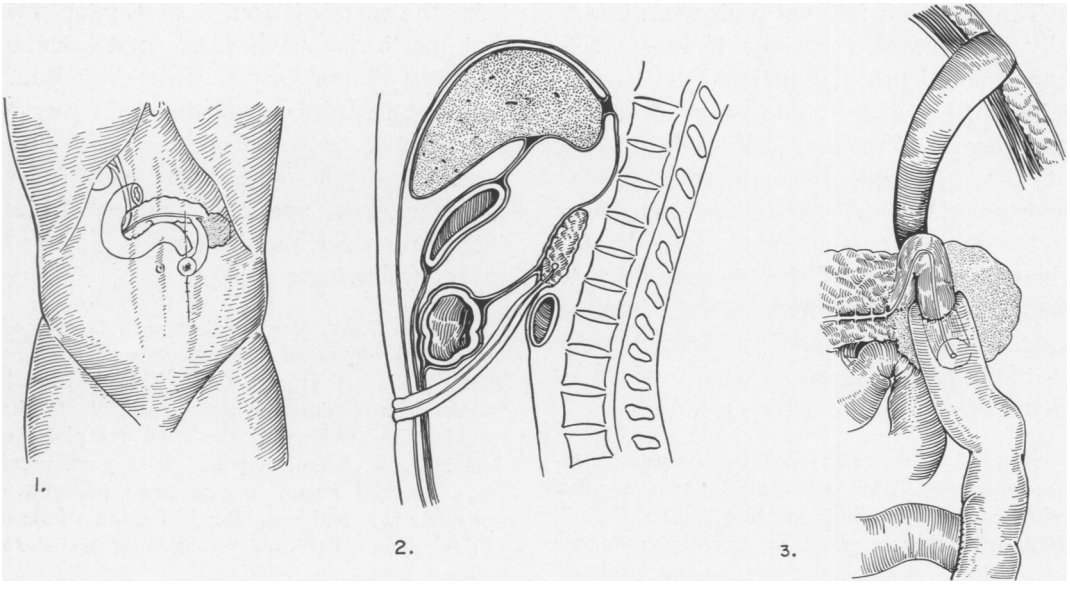

FIG. 3. Persistent fistula following marsupialization of pseudocyst. In the first sketch (1) is shown the draining fistula which followed marsupialization of a pseudocyst almost two years previously. The fistula arose from the main pancreatic duct near the tail of the pancreas, where an incidental abscess was encountered. 2. Sagittal view of pancreatic fistula. The fistulous tract passed in front of the jejunum but behind the colon. 3. The 2.5 cm. thick sinus tract, with the 3 mm. lumen in its center, was telescoped into the defunctionalized jejunal loop when the abscess rendered pancreatic resection inadvisable (Case 3). Anastomosis of mobilized fistulous tract to Roux-Y loop.

jejunum or ileum, usually seen in the upper left quadrant on a scout film of the abdomen. This is thought to represent an area of paralytic ileus due to the irritating pancreatic ferments. Paralytic ileus in this condition may simulate mechanical obstruction to a striking degree.

Case 4. *S. L. Jejunal Obstruction from Pancreatitis.* This 67-year-old woman was admitted to the University Hospital on Dec. 18, 1955 with the chief complaint of abdominal pain which she had had intermittently for years but which in the past 6 weeks had progressed to severe left upper quadrant pain radiating through to the back. Almost all oral intake was vomited. Physical examination revealed an emaciated and dehydrated elderly woman in no acute distress but who appeared acutely and chronically ill. Some examiners thought they felt a mass in the epigastrium. As evidence of fluid disequilibrium, the plasma chloride level was 58 mEq./L., the sodium level 119 mEq./L., the carbon dioxide combining power 36 mEq./L., and the nonprotein nitrogen level 71 mg.%.

The dehydration was corrected, and then an upper gastro-intestinal series was performed with thin barium. It was revealed that the jejunum was obstructed near the ligament of Treitz (Fig. 4). At laparotomy the duodeno-jejunal junction was freed from a very firm inflammatory mass which proved to represent chronic pancreatitis on three separate biopsies. Convalescence was uneventful except for transient hyperglycemia. She has since gained weight and has remained well.

Biliary Obstruction with Jaundice. The jaundice associated with pancreatitis may, of course, be due to a common duct stone, for approximately one-third of the cases of acute pancreatitis are associated with gall stones and another third with alcoholic intake. Common duct stone should always be excluded. Nevertheless, jaundice may appear when there are no stones, and this may be due to compression of the common duct by the acutely inflamed pancreas, or to fibrotic or calcareous changes in the chronically diseased organ. In the chronic disease it may prove exceedingly difficult,

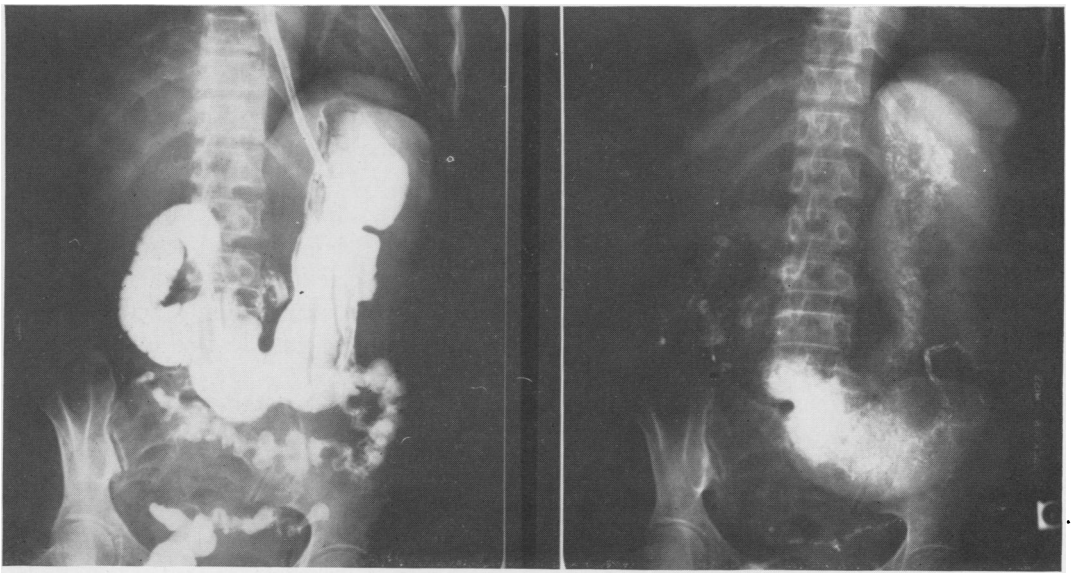

FIG. 4. Small bowel obstruction. Note the obstruction near the duodeno-jejunal junction, with associated dilatation of the proximal duodenum and stomach (Case 4).

even with biopsy at operation, to exclude malignancy as a cause of the jaundice.

Icterus of some degree may occur in about one-fourth of the cases of acute pancreatitis and may become manifest within a matter of hours after the onset of pain. In most cases the jaundice subsides as the pancreatitis subsides.

Case 5. *E. C. Pancreatitis—Biliary Calculi—Sphincterotomy.* This 50-year-old woman was admitted on Mar. 14, 1956 with the chief complaint of jaundice of one week's duration. Prior to the onset of the jaundice she had experienced nausea, vomiting, and epigastric pain, and while under observation in the hospital she exhibited chills and fever. The gallbladder was not visualized on 2 cholecystograms, tried despite the mild jaundice. The serum amylase level was elevated initially but declined to within normal limits over the next few days. The diagnoses of cholelithiasis, choledocholithiasis, and associated pancreatitis were considered likely.

At operation a gallbladder containing many stones was removed, as were numerous stones from the markedly dilated common bile duct. However, after all detectable stones had been removed neither instrument nor catheter could be passed through the sphincter of Oddi. Accordingly, the duodenum was opened through a longitudinal incision and the papilla of Vater examined. Only a very minute opening was present, surrounded by dense fibrous tissue. Sphincterotomy was performed and this then permitted the facile passage of a 6 mm. Bakes dilator (Fig. 5). The common duct was then flushed several times with saline and a T-tube inserted. There was clinical evidence of pancreatic inflammation of moderate degree.

While operative cholangiography is in almost routine use in our clinic, it was not available to us on this particular day. As luck would have it, the postoperative cholangiogram revealed a stone at the lower end of the common duct. Fortunately, five days later a second cholangiogram revealed that the stone had passed. Cole [3] has observed the passage of residual common duct stones in seven of nine patients followed over a period of 18 months. We have encountered this in one additional case.

The patient has thus far remained well.

Diabetes Mellitus. We have noted that pancreatic inflammation may give rise to either temporary or permanent diabetes. In the acute, severe attack hyperglycemia and glycosuria may be observed which subside as recovery progresses. However, in necrotizing pancreatitis so much pancreatic tissue may slough as to leave insufficient islet cells to secrete an adequate supply of insulin. In the course of his attack, one patient with acute hemorrhagic necrotizing

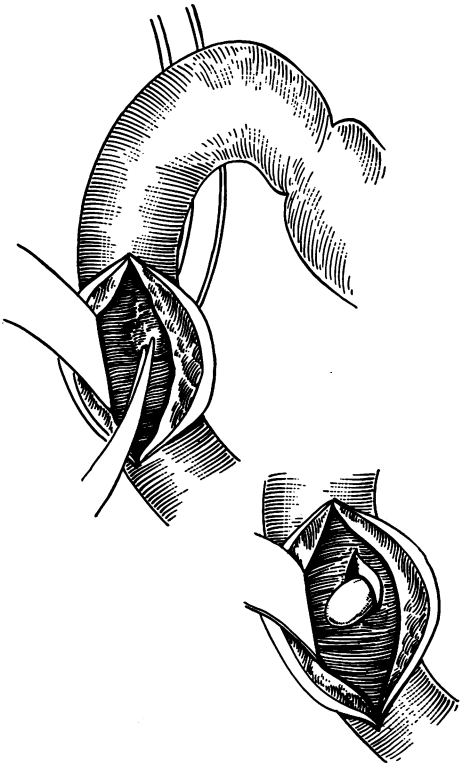

FIG. 5. Sphincterotomy (see Case 5).

pancreatitis sloughed out most of his pancreatic tissue, which presented as a left flank mass and was drained through a flank incision. He became permanently diabetic and required both dietary control and insulin. Incidentally, *bilateral paravertebral sympathetic block* produced dramatic relief of pain in this patient. The same end result of pancreatic destruction may follow repeated attacks of non-necrotizing disease which produces a generalized fibrosis with or without calcification. The diabetes caused by pancreatitis is usually a mild form, as is that which follows total pancreatectomy.

COMPLICATIONS OF CHRONIC RELAPSING PANCREATITIS

Any of the complications which have been described for the initial attack of acute pancreatitis may of course occur in subsequent attacks, which in the aggregate eventually produce the sinister condition known as "chronic pancreatitis" or "chronic relapsing pancreatitis."[5] However, there are several features of the chronic disease which deserve separate mention. Among these are intractable pain, calculus formation, and malnutrition. Zollinger, Keith and Ellison[7] feel that at least three criteria are required for the diagnosis of chronic pancreatitis: three verified previous attacks, pancreatic calcification, and a positive biopsy. However, patients with obvious chronic pancreatitis do not invariably exhibit calcification.

Intractable Pain. Without question the most distressing complication which develops in connection with chronic pancreatitis is that of intractable pain. While not excruciating initially, it has a constancy and gradually increasing severity which frequently lead to utter demoralization, opiate addiction, and occasionally to suicide.

Treatment is largely unsatisfactory. Of course, if biliary stones are found and removed, or if alcoholic intake can be abolished, then the recurrent attacks of pancreatitis may cease. Even so, all too frequently neither these measures, nor vagotomy-gastrectomy, choledochojejunostomy, or sphincterotomy is successful. Total pancreatectomy rarely improves the situation and, while bilateral sympathectomy may afford much pain relief, it is only palliative and not curative.

Pancreatic Calcification. Pancreatic calculi are present in approximately 50 per cent of cases of chronic pancreatitis, but are rarely seen before the age of 40 years.[6] The deposits may be either ductal or parenchymal; the latter are more commonly encountered, and are frequently so extensive as to outline the extent of the pancreas. Although calculi themselves need not produce symptoms, the chronic inflammation of which they are a result is characterized by the boring, often constant epigastric pain mentioned above. Some authors recommend pancreatic ductal exploration and removal of the stones, but in our experience this is a rather unsatisfactory effort at "pick-

ing raisins out of a bun." In fact, following one extensive procedure at which numerous stones were removed from pancreatic ducts and parenchyma, the radiologist reported that from the postoperative film it appeared that "some of the previously reported pancreatic calculi may have been removed." In any event, the patient's symptoms were not improved by the procedure.

Malnutrition. One of the outstanding features of chronic pancreatitis is the extremely poor nutritional state which may develop. Among the reasons for this are that the pain may be aggravated by eating, causing many patients to refuse food, and that the digestive disturbances result from a diminished enzyme secretion by the pancreas. A sprue-like condition may appear, associated with the loss of increased amounts of protein and fat in the stools. Weight loss, avitaminosis, hypoproteinemia, and edema and ascites may all attest the deranged nutritional metabolism. To effect an improvement in the general nutritional state represents scarcely less of a problem than does the relief of pain.

Case 6. *L. K. Chronic Relapsing Pancreatitis Associated with Alcoholism.* This 50-year-old male construction worker was first admitted to the University Hospital on May 27, 1956 complaining of fever and right upper quadrant pain. The previous history revealed that he had been a heavy drinker for the past 15 years, and for 13 years had had 3 or 4 "gallbladder" attacks per year consisting of nausea, vomiting, pain in the right upper quadrant, and fever. In 1950 the diagnosis of diabetes mellitus had been made, and the patient had since been taking 20 units of protamine zinc insulin and 30 units of regular insulin per day. In 1951 he had been jaundiced for one month. In February 1956, he had experienced gross hematemesis on 4 consecutive mornings, for which he was hospitalized and given 1500 ml. of blood.

Physical examination on May 27, 1956 revealed the liver palpable three fingerbreadths below the right costal margin; prominent veins were visible on the left side of the abdomen. However, the bromsulfalein retention, total serum protein level, and cholecystogram were indicative of no serious hepatic pathology. A gastro-intestinal series did not demonstrate esophageal varices, but a suggestion of irregularity in the region of the duodenal bulb was noted; calcification was seen in the general distribution of the pancreas.

He was subsequently discharged, but was readmitted on June 23, 1956 because of severe abdominal pain. He had lost 35 pounds, his current weight being 97 pounds. A gastro-intestinal series revealed considerable widening of the duodenal loop and at laparotomy a 10×10 cm. mass was found in the region of the pancreas, adherent to both stomach and colon. The operator took an adjacent lymph node for biopsy, and the frozen section diagnosis was that of probable carcinoma of the pancreas. The liver was grossly normal. Unfortunately, from the standpoint of definite diagnosis, permanent sections revealed only chronic inflammation of a lymph node; the pancreatic mass itself should have been biopsied. However, the patient improved considerably in the subsequent months, and the diagnosis of chronic pancreatitis would appear to have been the correct one.

COMPLICATIONS OF PANCREATIC TRAUMA

Even though the pancreas is sheltered by virtue of its deep retroperitoneal position, it still may be subjected to various forms of trauma. This may result from injury to the pancreas during surgical operations upon other organs such as the stomach, duodenum, biliary tract, spleen, or kidney; or it may result from blunt or penetrating injury in accidents.

Following pancreatic trauma, shock and prostration do not occur with the frequency seen in acute pancreatitis. Although pancreatic necrosis, hemorrhage, and cyst formation may be encountered, these have been considered above, and it is the matter of blunt external trauma producing pancreatic fistula with ascites which we wish to mention here. The irritating pancreatic juice gives rise to a chemical peritonitis, and one of the most difficult problems in our recent experience with pancreatic disease was that presented by pancreatic juice ascites in a young girl, the result of blunt trauma. As in other pancreatic disease, epigastric pain and an elevated serum amylase level were the most helpful findings in reaching the diagnosis.

Case 7. *D. R. Blunt Trauma with Pancreatic Peritonitis.* During March, 1956, this 5-year-old girl fell about 5 feet and landed on her abdomen across a plank. She was not unconscious, and there was no break in the skin. Several hours later she began to vomit and was hospitalized near her home for a week, during which time the vomiting ceased. There was no hematemesis or melena. One week following discharge her abdomen became distended and she was readmitted to the hospital but soon was discharged. She was later hospitalized a third time because of vomiting, fever, and abdominal distention, and an abdominal exploration was carried out, drains being placed in the region of the pancreas. Following this a serous material drained for a few days, but this then ceased and the abdomen enlarged rapidly.

She was transferred to the University Hospital on May 25, 1956. *Physical examination* revealed a chronically ill child whose abdomen was considerably distended, probably with fluid. Paracentesis netted 1,800 ml. of greenish-tinged serous fluid, with a high amylase level. As repeated taps became necessary, to allow for improved respiration and food intake, a limited abdominal exploration was performed with the hope of establishing an external pancreatic fistula, for the irritant effect of the pancreatic juice was causing a rapidly downhill course. Many loculated fluid collections within the abdomen were found but no definite cysts. The loculations were opened, the fluid was aspirated, and a drain was placed in the lesser sac and beneath the mesocolon. A fistula was thus established, she rapidly improved, and after a few weeks drainage ceased entirely. She has remained well.

SUMMARY

Among the complications of pancreatic disease that have been reviewed are hemorrhage and electrolyte imbalance, pancreatic necrosis and abscess formation, pseudocysts, pancreatic fistula, intestinal obstruction, obstructive jaundice, diabetes mellitus, intractable pain, pancreatic calcification, malnutrition, and pancreatic ascites.

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