

Surgical Management of Injury to the Pancreas *

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IN THE COURSE of studies of diseases of the pancreas certain principles of treatment for injury by violence or at surgical operation have emerged.

The gland is deeply placed and although well protected it is a delicate fragile structure. The duct system is the vulnerable component of the organ, for when the ducts are vented by rupture or laceration, pancreatic secretion leaks out into adjoining spaces and dissects by fluid displacement. There are two major considerations in this situation: Loss of salt-rich fluid, and proteolytic enzymes activated out of place. When trypsinogen is activated to trypsin, the fluid is capable of digesting the pancreas itself and of damaging all structures in the path of flow.

Secretory pressure of acinar pancreatic cells is exerted into a closed-duct system. Intraductal pressure at any one moment is a resultant of secretory pressure and resistance to flow. In turn, under normal conditions, resistance to flow is maintained and is varied by the tonus of the sphincter of Oddi. When a path of lesser resistance is created by an abnormal opening in the duct system, flow of juice is through this relatively nonresistant opening. Such abnormal flow continues until it meets resistance equal to or greater than that maintained by the sphincter of Oddi.^{1, 2}

The variables in the dynamics of flow are, then: (1) Secretory pressure of pancreatic cells, influenced by the hormone, secretin, and by autonomic nervous stimuli, (2) Tonus of the sphincter of Oddi and, in some measure, musculature of the duodenal wall, and (3) In the event of rupture of the pancreatic ducts by disease or injury, resistance of structures encountered by escaping pancreatic juice. Pressure in the leaking fluid is exerted in all directions, and the route of flow may be to parts as distant as the pelvis or the thorax³ (Fig. 1, 2).

Brief summaries of the course of five patients are cited to demonstrate the development of the concept that destroying the resistance of the sphincter of Oddi by sphincterotomy is an essential part of the treatment of pancreatic trauma to prevent the flow of pancreatic juice retroperitoneally or into the peritoneal cavity.

Case 1. G. M. (Bellevue Hospital—38142-51), a 60-year-old man, was admitted to the hospital June 7, 1951, two days after having been kicked in the abdomen. He had severe generalized abdominal pain and an elevated serum amylase level (2,400 mg.%) (normal 100–200 mg.%). After four days of nonoperative treatment for what was thought to be acute pancreatitis, his symptoms subsided, and the serum amylase level returned to normal. When oral feeding was instituted, fluid gradually accumulated in the peritoneal cavity, and serum amylase rose over a symptomless period of five weeks to 7,000 mg. per cent. Paracentesis disclosed that the fluid was pure pancreatic juice, and at subsequent operation, 41 days after the original injury, five liters were removed. All peritoneal surfaces were glistening and edematous, presumably actively absorbing the fluid. The pan-

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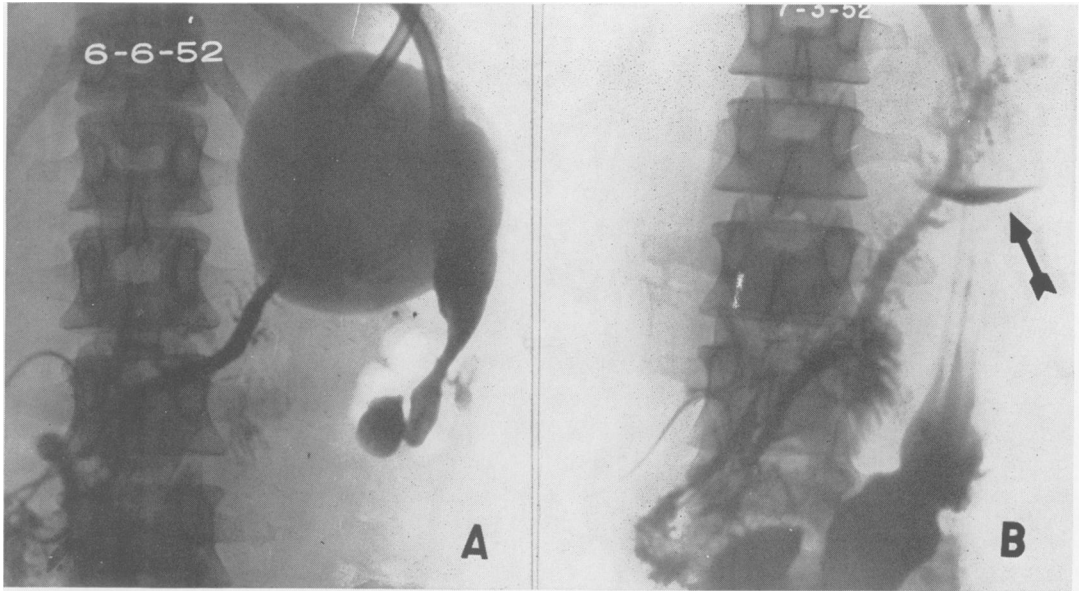


FIG. 1. Mechanism of pseudocyst formation following attack of acute pancreatitis. Operative pancreatogram (A) reveals radiopaque solution leaving main duct through an opening produced by necrosis, and filling a huge pseudocyst extending from the diaphragm to the sigmoid mesocolon. One month after sphincterotomy, which reduced the intraductal pressure, a pancreatogram (B) reveals almost complete disappearance of the pseudocyst, only a small space (arrow) remaining.

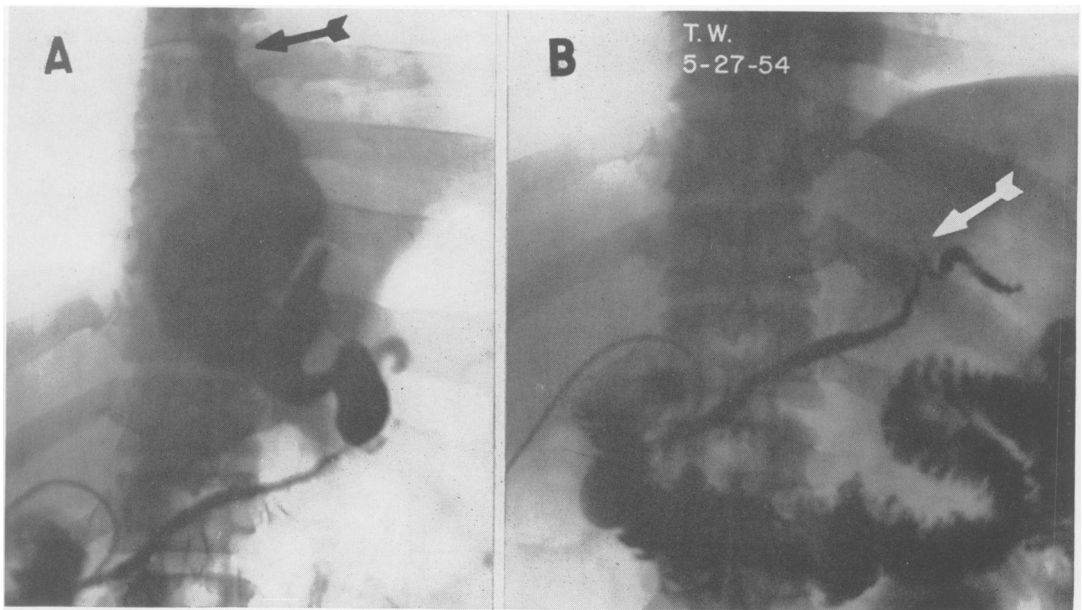


FIG. 2. Visualization by operative pancreatogram of a posterior mediastinal pseudocyst which resulted from an attack of acute pancreatitis. It extended as high as the 6th thoracic vertebra (arrow). In this patient the first sign suggestive of such a pseudocyst was the finding of a high concentration of amylase in the fluid present in the left pleural space. One month after sphincterotomy, a pancreatogram (B) showed complete disappearance of the pseudocyst, only a small linear space indicating the original point of rupture in the pancreatic duct (arrow).

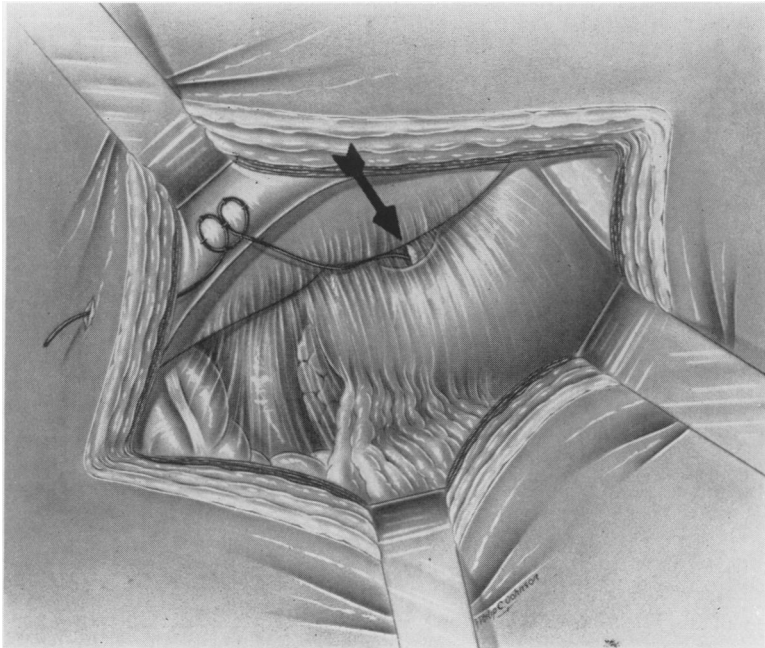


FIG. 3. Case 1. Drawing illustrating the glassy transparent appearance of the peritoneum, due to continual absorption of inactivated pancreatic juice over a 41-day period. The edematous condition of the tissues permitted only insertion of a plastic tube into the ruptured duct (arrow).

creas was found partially transected at its neck, and the main pancreatic duct gaped open. A small plastic catheter was placed into the duct in the distal gland and brought out through the abdominal wall (Fig. 3). The proximal duct was tied. Thereafter, serum amylase levels returned to normal (Fig. 4). Drainage from the catheter was collected so that the patient could drink it. After

a short period of convalescence, a tuberculous pulmonary lesion was found to be active, and the patient was transferred to a medical ward for chest diseases. He remained there for two years and five months, all the time collecting and drinking pancreatic juice from the distal gland. Finally, he recovered sufficiently to permit anastomosis of the distal draining pancreatic duct to the stomach.

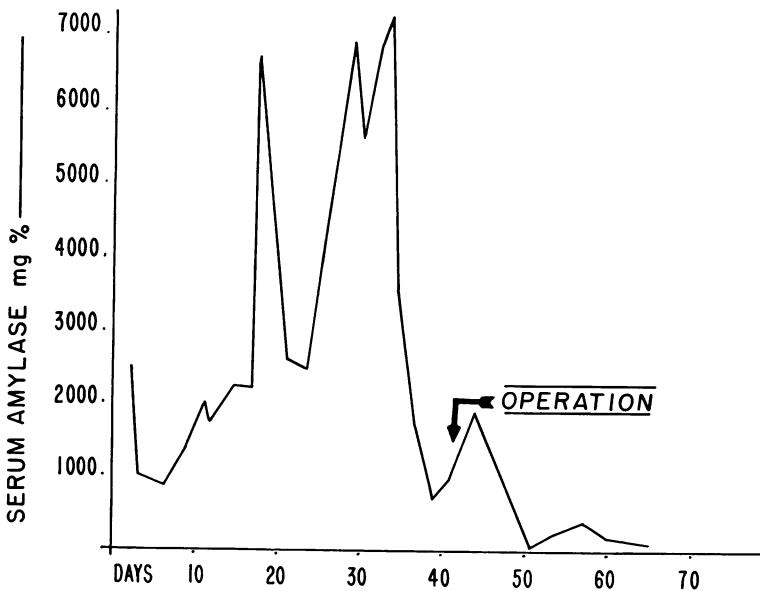
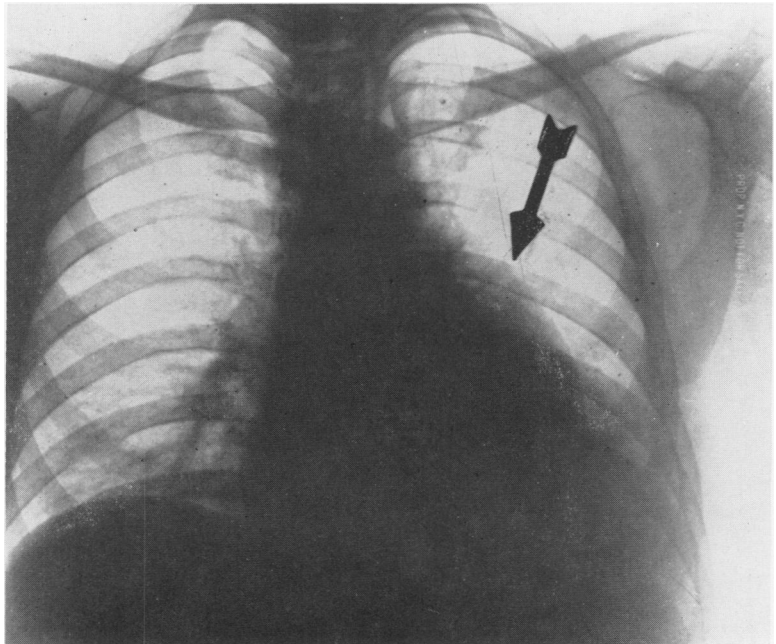


FIG. 4. Case 1. Concentration of serum amylase as a result of rupture of the pancreatic duct into the peritoneal cavity. Following admission, treatment by nasogastric suction and atropine lowered the serum amylase, but when feeding was started the amylase rose to over 7,000 mg.%. Nasogastric suction for three days again resulted in a fall of the serum amylase to less than 1,000 mg.%, and operation dropped it to normal.

FIG. 5. Case 2. Chest plate three days before sphincterotomy, showing outline of pseudocyst behind heart shadow (arrow) and fluid in left pleural space.



The instructive aspects in this patient's course were:

1. The insidiousness with which pancreatic juice collects in a free space, in this case the peritoneal cavity. A huge quantity poured out undetected until, to all intents, a pseudocyst had been created of the entire peritoneal cavity. The metabolic defect brought about by water and electrolyte loss was compensated by oral intake throughout this period.

2. Lack of tissue reaction to pure inactivated pancreatic juice.

3. The persistence of a free-flowing fistula for two years and five months, or for just as long a period as no resistance to flow is met.

4. The absorption of pancreatic enzymes from peritoneal surfaces into the blood stream.

5. Altered dynamics of flow created by a partially divided duct opening into the peritoneal cavity as distinct from the dynamics of flow through a hole in the side of a main duct opening into the retroperitoneal space.

This latter situation which occurs typically as a result of necrosis during an attack of acute pancreatitis, can also be created as a result of trauma. The following case illustrates this principle.

Case 2. T. S. (Bellevue Hospital—59368-56), a 38-year-old man, attempted suicide by stabbing himself at several sites in the left upper abdomen. He was operated upon, and the spleen and left kidney, both badly lacerated, were removed. He progressed well for seven days and then became dyspneic. Fluid was found in the left pleural space and in the mediastinum. This fluid was identified as pancreatic juice by its high enzyme content (1,200 mg.%) and the collection behind the heart shadow considered to be a pancreatic pseudocyst. At a second operation on the 14th day, cholecystectomy, sphincterotomy and intubation of the pancreatic duct were performed. Pancreatographic studies demonstrated rupture of the pancreatic duct (Fig. 6A). Thereafter, x-ray films of the chest showed rapid shrinkage of the pseudocyst. Pancreatographic studies demonstrated partial healing (Fig. 6B) and finally on the fourteenth day, complete healing of the pancreatic duct (Fig. 6C).

In this patient the insidiousness with which inactivated pancreatic juice flows retroperitoneally from a ruptured duct into

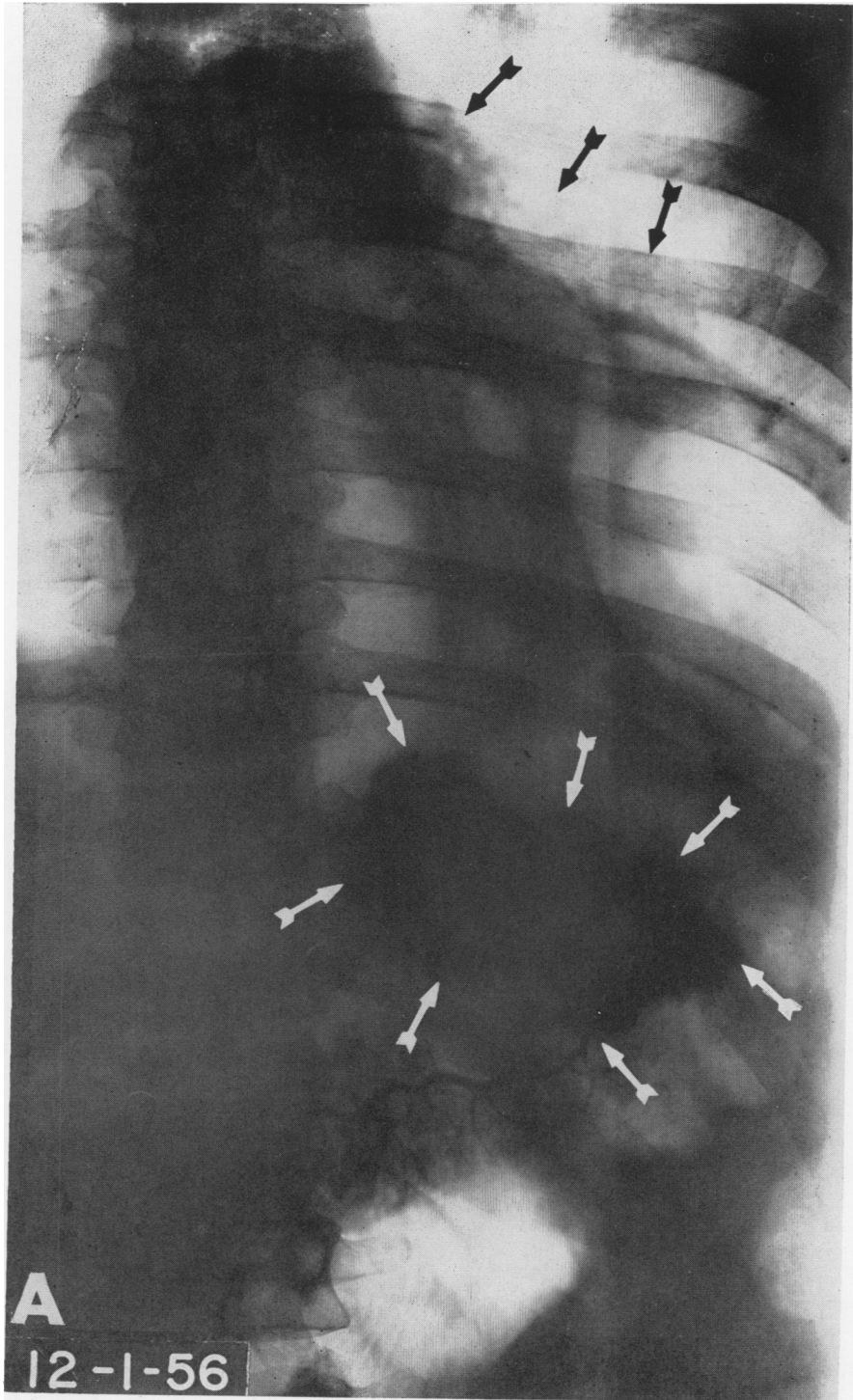


FIGURE 6A.

FIG. 6A. Case 2. Operative pancreatogram showing extravasation of radiopaque solution from ruptured pancreatic duct (white arrows). Outline of pseudocyst in chest can be seen (black arrows). FIG. 6B, C. Pancreatogram two days later shows lessened

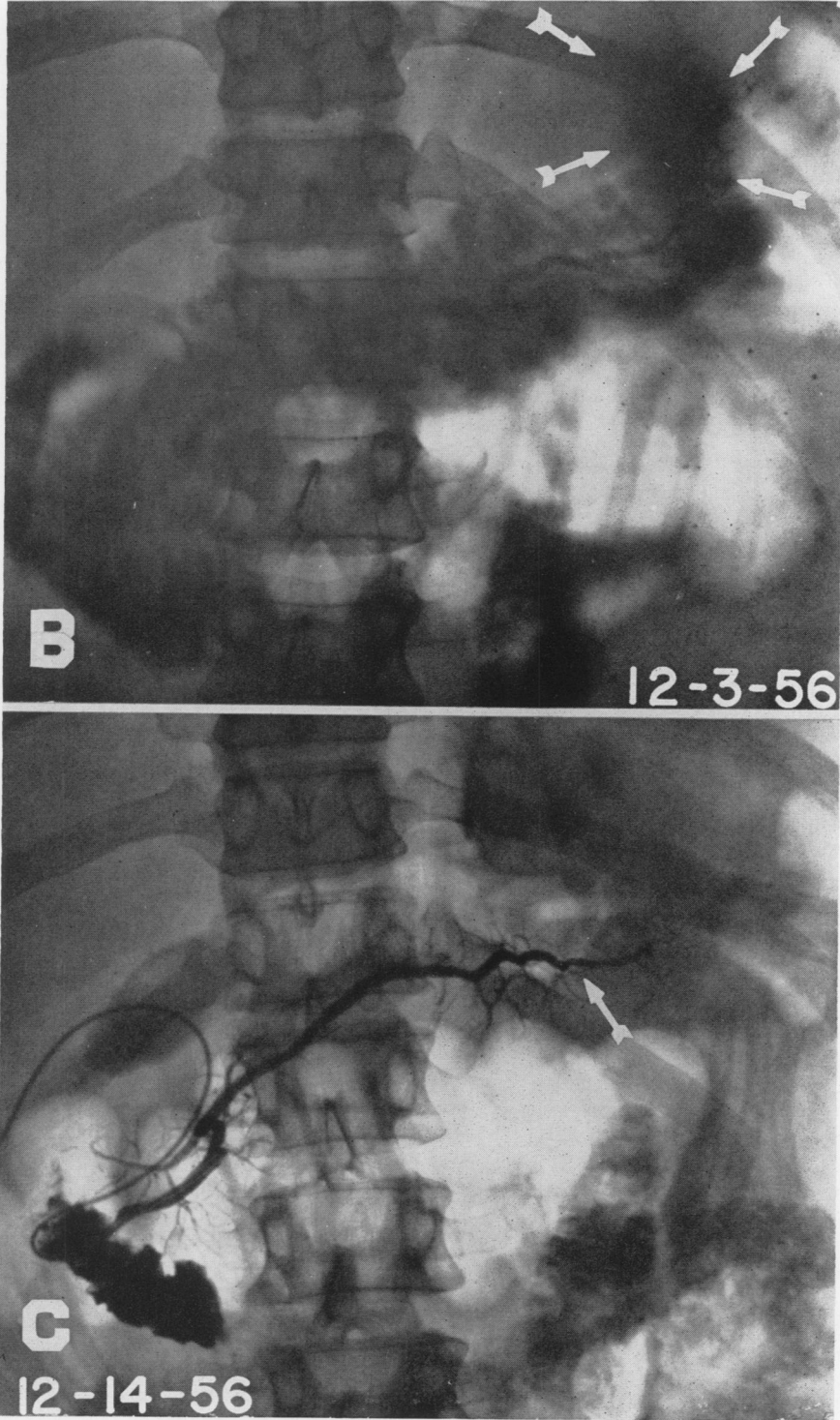


FIGURE 6B, C.

extravasation (white arrows). The whole pancreas is opacified. Two weeks after operation (C), before removal of the plastic drainage tube, pancreatic duct is shown to be healed (arrow).

channels of low resistance (as distant as the posterior mediastinum to the level of the sixth thoracic vertebra and thence by permeation of the thin pleura into the left pleural space) is again demonstrated. The important consideration, however, is collapse of the pseudocyst when resistance to flow through the sphincter of Oddi is abolished by sphincterotomy. This effect had been demonstrated previously in pseudocysts which arise as the result of pancreatitis (Fig. 1, 2).

More complicated problems arise when, for some reason or other, the pancreatic juice is activated and proteolytic activity is added to flow pressure in the advance of the pseudocyst, as exemplified in the following summary.

Case 3. F. S. (Bellevue Hospital—16794-58), a 32-year-old man, was stabbed in the left flank. An operation was performed nine hours after injury at which time two lacerations of the transverse colon and a small tear in the diaphragm were repaired. The left kidney was lacerated on its surface, and a small amount of blood was evacuated from the area of this wound. The spleen and pancreas were inspected and found to be unharmed. The first ten days after operation were increasingly troubled. High fever, pain and the appearance of fluid in the left pleural cavity led to speculation concerning sub-phrenic abscess or pancreatic duct rupture and formation of a pseudocyst in the posterior mediastinum. A sample of pleural fluid contained a large quantity of pancreatic enzymes, and the serum amylase level was elevated. On the 12th day, at reoperation, the left sub-phrenic space was found clear but dense inflammatory reaction about the colon and a swollen, hard pancreas confirmed the impression that free activated pancreatic juice was flooding the area. The patient's condition was too precarious for extended operation at this time, so a drain was placed into the area of the swollen pancreas. Shortly thereafter a copious flow of active pancreatic juice poured through the drain sinus, and the pleural effusion subsided. It was impossible to control the digestive action of the pancreatic juice. The wound site disrupted, a fecal fistula developed, severe bleeding from eroded vessels ensued; electrolyte loss, oliguria and infection all combined to make the situation desperate. Finally, on the 25th day, which was the 13th day after the second operation, cholecystectomy, transduodenal sphincterotomy, drainage of

the main pancreatic duct and right transverse colostomy were performed. Improvement was striking within 24 hours and he progressed to eventual recovery.

The course of this patient illustrates the destructive properties of activated pancreatic juice. It is not entirely clear how activation of trypsinogen is accomplished when no enterokinase is present. Necrotic tissue and bacterial contamination are assumed to be the activating principle, yet suppuration can occur in areas of pancreatic juice extravasation without trypsinogen activation. Furthermore, careful experimental observations indicate that trypsin cannot digest a healthy living cell.⁴ Some injury to the cell must accompany the process and precede proteolysis.

In any case, the process is rapidly devastating, and preventive measures should be taken before irreparable local damage and metabolic disorder overwhelm the patient.

The following summary is an example of primary definitive conservative management of an injury to the pancreas.

Case 4. D. B. (Bellevue Hospital—73646-56), a 21-year-old man, was stabbed in the abdomen on January 24, 1958. At operation, two hours later, a large quantity of blood in the peritoneal cavity was found to have arisen from a severed splenic artery, which was ligated. The same knife thrust had also transected the pancreas through its body. Cholecystectomy and transduodenal sphincterotomy exposed the main pancreatic duct in the ampulla of Vater. An attempt to thread a catheter through this opening and out to the distal transected duct was unsuccessful. It was then introduced through the proximal cut end of the duct, out through the duodenum, up the common bile duct, and through the cystic duct and abdominal wound to the outside. The other end of the catheter was inserted into the distal pancreatic duct, bridging the division and acting as a stent. In the post-operative period there was some leakage of juice about a drain placed to the tail of the pancreas but free flow through the catheter shortly controlled the fistula. After 20 days the catheter was removed. Pancreatographic studies on the eighth and twenty-eighth day demonstrated the restitution of the pancreatic duct without loss of any part of the organ (Fig. 7).

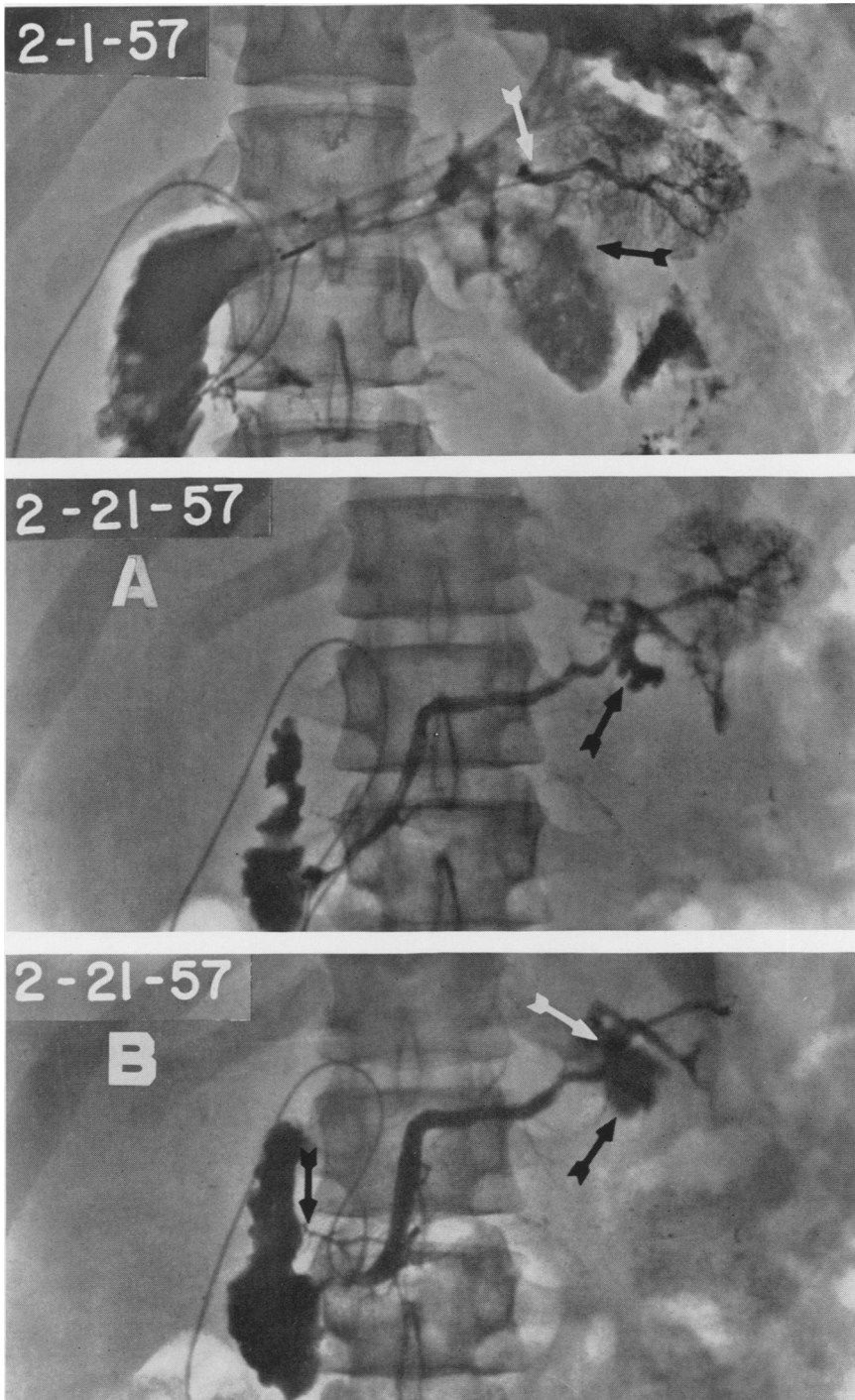


FIG. 7. Case 4. Serial pancreatograms showing healing of a transected duct, held together by a plastic tube acting as a stent. Pancreatogram on the eighth postoperative day (2-1-57) showed point of transection (arrow). The pressure of injection of the radiopaque solution caused some extravasation (black arrow), but at this time there was no seepage of pancreatic juice along the drain site. Two weeks later the final pancreatographic study showed the stent still in place (A), with a slight extravasation due to back pressure. When the tube was withdrawn partly (B), the duct could be seen almost completely healed (white arrow), although some extravasation (black arrow) due to pressure of injection was still seen.

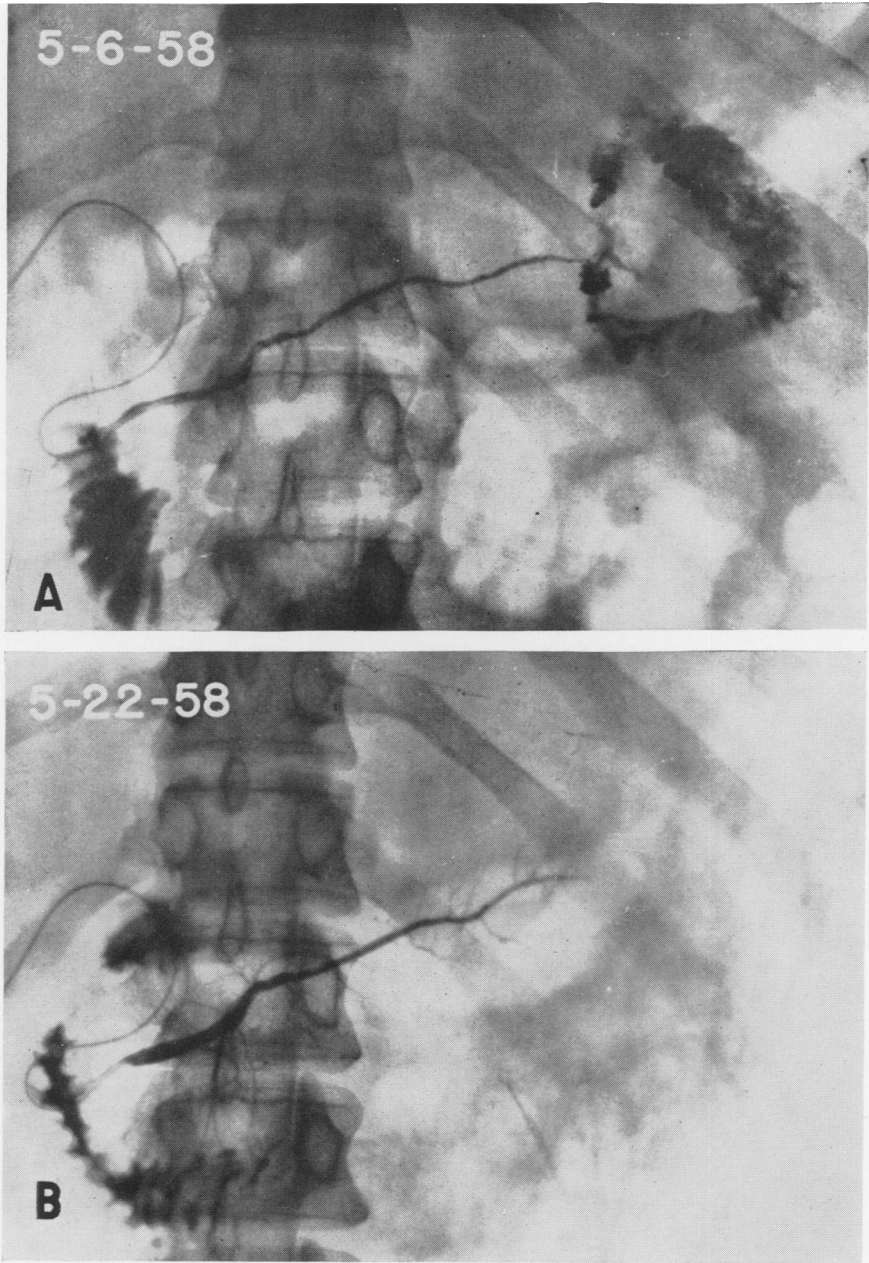


FIG. 8. Case 5. Pancreatographic study was performed fifteen days after operation (A). At this time the patient had no symptoms and the serum amylase was normal indicating that there was no seepage into the peritoneal cavity. However, the end of the duct was not closed since the radiopaque solution, as a result of the increased injection pressure, entered into the space previously occupied by the amputated tail. The final pancreatic study (B) performed 31 days after operation showed complete healing of the end of the duct.

The events in this patient's course demonstrate that the principle of unresisted flow may be utilized through a catheter

stent in a divided pancreatic duct. Also, that definitive provision for such flow can be made at a primary operation designed

to accomplish permanent relief in one stage.

The course of one more patient is cited for its interest regarding blunt abdominal injury and its uneasy evidences of severe disease.

Case 5. H. J. (Bellevue Hospital—20948-58), a 39-year-old man, was brushed by a car while riding a bicycle and knocked to the ground. He was seen at a hospital emergency room and sent home. Two days later because of increasing abdominal pain he entered Bellevue Hospital. A serum amylase level of 1,280 mg. per cent was the basis for the diagnosis of acute pancreatitis, for which he was treated. When the pain did not subside, needle aspiration of the peritoneal cavity produced pure blood. Operation was then performed (on April 21, 1958) 20 hours after admission and three days after injury. The bleeding was presumed to be from the spleen which was removed promptly, but on examination was intact. On further exploration the tail of the pancreas was found transected at its junction with the body, displaced and fixed posteriorly by swollen inflamed adjacent tissue. The distal separated pancreas was removed and bleeding points secured. The open end of remaining pancreas was so friable that satisfactory closure of the duct could not be accomplished. To forestall leakage of pancreatic juice through this non-resistant path, sphincterotomy and pancreatic duct intubation were carried out. The catheter was brought to the exterior through the cystic duct. This provided for flow sufficiently free to prevent leakage from the damaged end of the pancreas which healed (Fig. 8).

Aside from the dynamics of extravasations of pancreatic juice and the rationale of treatment, the matter of whether or not escaping juice is activated is of great im-

portance and interest. When pure unactivated juices escapes, the evidences in injury are obscure. A slowly developing collection may first manifest itself by distention of the parieties or by the formation of a pseudocyst. On the other hand, activated juice causes severe inflammatory reaction, erosion of tissues, and hemorrhage. The precise mechanism of activation is not known. Necrotic tissue and suppuration are assumed to play a role. There are sufficient instances, however, of sudden activation after a period of quiet, to justify aggressive preventive measures in the treatment of simple extravasations.

Summary

The principle is exemplified that in trauma to the pancreas sphincterotomy and drainage of the duct of Wirsung is an effective treatment. This is true with escape of pancreatic juice that is not activated. The presence of activated pancreatic juice requires emergency treatment.

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