SURGICAL TREATMENT OF CALCIFICATION OF THE PANCREAS*

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THE DEPOSITION of insoluble calcium salts in the ducts and acini of the pancreas appears to be a not uncommon consequence of recurrent pancreatitis.[1, 2] The direct cause of this chemical abnormality is not known. While the appearance of calcification on roentgen ray films is a visible evidence of pancreatitis, it in no way alters the course of the disease, nor should the treatment be different. It is a mistake to consider calcification of the pancreas out of its proper context, that is, an incident in the course of a disease entity. The occurrence of this incident does not change the pattern of pain, its rhythm, the progressive digestive symptoms due to external pancreatic deficiency, or the eventual outlook.

On the basis of Archibald's concept that the diversion of bile into the pancreatic ducts is brought about through a common biliary-pancreatic passageway by spasm of the sphincter of Oddi,[3] 73 patients with proved pancreatitis were treated by section of the sphincter of Oddi.[4-6] Among these were four with roentgenographic evidence of calcification of the gland. Herewith are abstracts of the clinical course of these patients:

Case 1.—F. J. (Bellevue Hospital, 4485–48) was a 50-year-old male with a history for 13 years of attacks of severe upper mid-abdominal pain, radiating to the epigastrium and left upper quadrant, lasting 2 hours to 2 days and occurring 2 to 3 times a month. These attacks usually followed a large meal and were most severe if food were taken after a prolonged alcoholic spree. There was no history of diarrhea. Roentgenograms of the abdomen revealed widespread calcification of the pancreas (Fig. 1); roentgen ray films of the gallbladder showed normal visualization of dye; the duodenum showed deformity, suggestive of duodenal ulcer. The glucose tolerance curve was normal. A secretin test showed moderate diminution of pancreatic function (total volume 105 cc.; total amylase 297 units). Since the symptoms were similar to those of non-calcified recurrent acute pancreatitis, and since he had adequate external and internal pancreatic function, it was decided to treat him as a case of recurrent acute pancreatitis.

At operation, February 9, 1948, only a few adhesions were present at the fundus and ampulla of the thin-walled gallbladder. The pancreas was markedly enlarged throughout, very hard, and had a cobblestone-like surface. The head of the pancreas pushed the duodenum forward and distorted it. A thin scar on the anterior duodenal wall was suggestive of a healed duodenal ulcer. A cholangiogram showed an enlarged common bile duct with distortion due to pressure from the pancreas, but the pancreatic duct was not visualized (Fig. 2). The gallbladder was removed and the common duct opened. Due to difficulty in passing the sphincterotome through the papilla, the duodenum was opened and the instrument passed through under vision. The blade was then opened,

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retracted and the sphincter sectioned. Two weeks after operation, cholangiographic studies again failed to visualize the pancreatic duct. Reflux studies[7] proved the presence of a common passageway. Kymographic studies showed only a slight response to acid, indicating possibly incomplete section of the sphincter, but the response to morphine showed excellent functional destruction. The patient was last seen March 27, 1950, more than 2 years after operation. Although he has continued to drink, he has had no

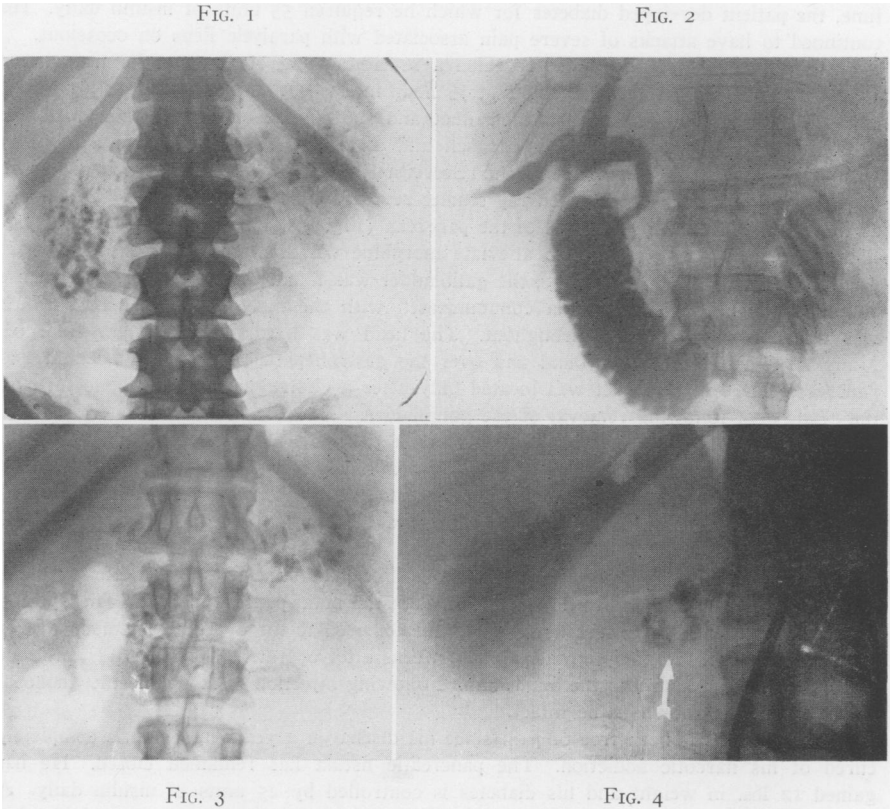

FIG. 1.—(Case 1) Preoperative roentgenogram showing widespread calcification of the pancreas.

FIG. 2.—(Case 1) Operative cholangiogram showing enlargement of the biliary tract with angulation and distortion of the common duct in its passage behind the pancreas. The pancreatic duct was not visualized.

FIG. 3.—(Case 1) Roentgenogram taken two years after sphincterotomy showed no evident change in the degree of calcification of the pancreas.

FIG. 4.—(Case 2) Preoperative examination showing a normally functioning gallbladder with calcification of the head of the pancreas (arrow). The rubber tube is draining the pancreatic fistula.

symptoms since operation, and has gained about 10 lbs. in weight. Roentgen ray films of the abdomen taken January 19, 1950, showed the pancreatic calcification unchanged (Fig. 3).

Case 2.—J. G. (Bellevue Hospital, 906–48) was a 36-year-old male admitted to the psychiatric division of Bellevue Hospital on February 24, 1948, in a semi-stuporous condition due apparently to insulin hypoglycemia. He had a history of attacks of severe

epigastric pain radiating to the back for 11 years. In 1940, cholecystostomy and drainage of a pancreatic cyst for obstructive jaundice due to the cyst, was performed at another hospital. After 2 years the attacks of severe epigastric pain recurred and he became a morphine addict. In 1945, also at another hospital, following studies which indicated marked diminution of pancreatic function, an attempt at a pancreatectomy was abandoned and a splenectomy and drainage of a pancreatic cyst was carried out. He developed a persistent pancreatic fistula. Attacks of epigastric pain persisted. At this time, the patient developed diabetes for which he required 55 units of insulin daily. He continued to have attacks of severe pain associated with paralytic ileus on occasions.

Studies at Bellevue Hospital revealed a severe diabetes (glucose tolerance curve: 173,235,333,308 mg. per 100 cc. sugar at ½ hour intervals). Injection of the pancreatic fistula visualized part of the pancreatic duct and showed free communication with the duodenum. Roentgenogram of the stomach and duodenum revealed a normal stomach with some obstruction of the duodenum. Secretin tests could not be done due to failure to pass a duodenal tube. Roentgen ray studies revealed a normal functioning gallbladder as well as calcification in the head of the pancreas (Fig. 4). He required frequent injections of Demerol for pain and to alleviate morphine withdrawal symptoms.

At operation on April 5, 1948, the gallbladder was found to be surrounded by dense adhesions. The pancreatic fistula communicated with the body of the pancreas which was thickened and fibrosed throughout. The head was hard, irregular and enlarged. Many large veins coursed around and over the gastrohepatic omentum, duodenum and pancreas. The common duct was located only after a cholangiogram was done through the cystic duct following removal of the gallbladder. The common duct was wide above and tortuous in its passage behind the head of the pancreas. The pancreatic duct was not visualized. The common duct was opened and a fine probe passed into the duodenum. The duodenum was opened and the sphincter of Oddi was cut over the probe. The pancreatic duct was seen to be emptying into the common bile duct 4 mm. above the papilla. A fine catheter was passed into the duodenum through the bile duct and brought out at the lateral angle of the wound. The pancreatic fistula was excised in its whole length from the skin to its connection with the main pancreatic duct. The opening which remained in the pancreatic duct was anastomosed to the overlying stomach over a small rubber tube. Cholangiograms taken 2 weeks after operation showed ready entrance of the contrast medium into the duodenum. Following injection of morphine the duodenal musculature was shown to be intact.

The patient has been free of pain since his discharge 2 years ago, and is completely cured of his narcotic addiction. The pancreatic fistula has remained closed. He has gained 12 lbs. in weight and his diabetes is controlled by 25 units of insulin daily. A roentgenogram taken on March 31, 1950, showed no obvious change in the calcification of the pancreas (Fig. 5).

Case 3.—D. R. W. (Beth Israel Hospital, Newark, N. J.—operation with Dr. A. Abrams) was a 40-year-old white male with a 12-year history of attacks of severe epigastric pain radiating to the back and to both upper quadrants, occasionally accompanied by vomiting. The attacks lasted from 2 hours to 2 days and at first occurred about every 6 months, but in the last few years the severe attacks occurred every few weeks and he had pain after eating almost every day. Ten years ago a diagnosis of perforated ulcer was made but no operation was done. Between 1940-1944 he was admitted to 4 different military hospitals. Many roentgenograms showed a normal gallbladder and normal stomach and duodenum. He was finally discharged from the army February 11, 1944, because of incapacitating pain. No diagnosis was made. Examination by a civilian physician in 1947 revealed calcification of the pancreas (Fig. 6). A cholecystostomy was performed but by request of the patient the pancreas was not removed. He had no pain as long as the cholecystostomy was functioning. As soon as the drainage tube was removed the attacks of pain recurred. A secretin test done on

April 12, 1948, revealed marked impairment of the pancreas (total volume 120 cc.; total amylase 65 units). At operation on April 16, 1948, the pancreas was found to be large and very hard with an irregular knobby surface. The gallbladder was removed. An operative cholangiogram showed dilatation and distortion of the biliary tract with marked narrowing in the retro-duodenal part. The pancreatic duct was visualized

FIG. 5

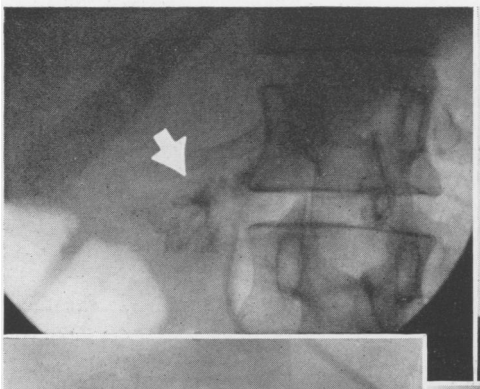

FIG. 6

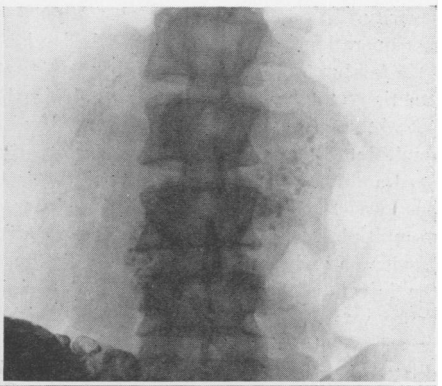

FIG. 7

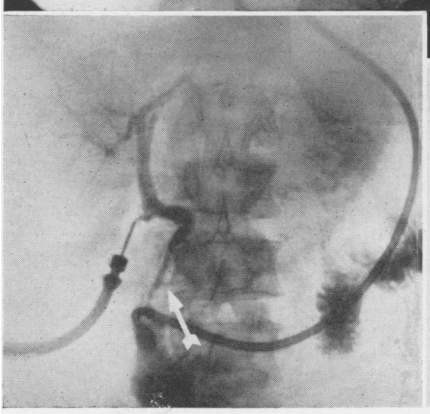

FIG. 8

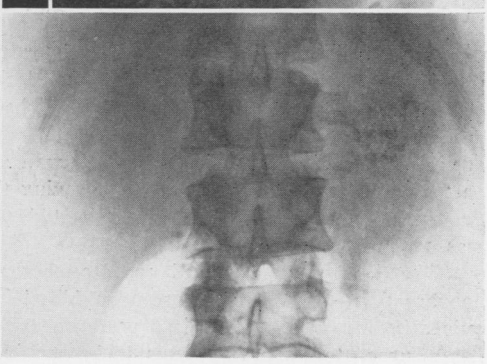

FIG. 5.—(Case 2) Two years after sphincterotomy (arrow) the degree of calcification of the head of the pancreas has remained unchanged.

FIG. 6.—Widespread calcification of the pancreas first noted 11 years after the onset of severe attacks of pancreatitis.

FIG. 7.—(Case 3) Operative cholangiogram showed distortion of the common duct with marked narrowing in the retro-duodenal part due to pressure of the enlarged pancreas. The pancreatic duct (arrow) was visualized.

FIG. 8.—(Case 3) Roentgenogram taken 21 months after sphincterotomy showed no change in the degree of calcification of the pancreas.

(Fig. 7). The common duct was about twice its normal size, and very tortuous, owing to its passage behind the enlarged irregular head of the pancreas. The common duct was opened, the sphincterotome passed into the duodenum and sphincterotomy done. The common duct was sutured and a small drain placed down to the line of suture. The drain was removed in 48 hours. In spite of occasional bouts of alcoholism, he has had no pain since operation 2 years ago. However, he requires 6 Gm. of pancreatin daily to prevent bulky diarrhea. He has gained about 15 lbs. in weight. Roentgenogram of the abdomen on January 7, 1950, revealed no change in the degree of calcification (Fig. 8).

Case 4.—P. D. (Bellevue Hospital, 387664-49) was admitted for the first time to the Third Surgical Division on November 24, 1949, complaining of recurrent attacks of epigastric pain of 20 years duration, and loss of 78 lbs. in weight in the past 2 years. The attacks were brought on originally by nervousness or excitement and lasted 1 to 2 days. Since 1937, when he began to drink excessively, the attacks became much more severe, radiated to the back, and lasted 1 to 2 weeks. In spite of a number of operations at various hospitals (cholecystectomy, 1939; repair of a para-esophageal hernia, June, 1949, and exploratory laparotomy, August, 1949), the recurrent attacks increased in severity. In addition, for the past 2 years, pain occurred after every meal. Prior to the last operation in August, 1949, calcification of the head of the pancreas was noted (Fig. 9). A secretin test on November 29, 1949, revealed marked impairment of pancreatic function (total volume, 80 cc.; total bicarbonate, 20 cc. and total amylase 56 units).

At operation, December 1, 1949, hard dense adhesions were found binding the liver, small intestine and transverse colon to the diaphragm. The pancreas was very large, hard and irregular, and had pushed the duodenum upward and to the right. The common duct was found to be thin-walled and narrow. A cholangiogram could not be done due to multiple needle holes produced in the efforts to locate it. A probe was passed into the duodenum and the sphincter sectioned transduodenally. The pancreatic duct, located after the injection of secretin, was found to join the bile duct 6 mm. above the papilla. A probe was passed 4 cm. up the duct without encountering an obstruction. A fine tube was inserted and the pancreatic duct visualized by means of Diodrast (Fig. 10). It was dilated and tortuous but not obstructed. The duodenum was closed and a T-tube inserted in the common duct.

Cholangiographic studies two weeks later, revealed that the T-tube was lying in the common hepatic duct, and that the choledochus lay behind the duodenum and the enlarged head of the pancreas (Fig. 11). The cystic duct and a small remnant of the gallbladder was still present. The dye ran readily into the duodenum. Following the administration of morphine the duodenal wall could be seen to compress the intramural part of the common duct (Fig. 12), proving that the one-way valve action to prevent duodenal reflux was intact. Manometric study showed that the resistance to flow of bile was 120 mm. water.

The T-tube was removed on December 28, 1949, and the patient discharged on January 6, 1950. When last seen on March 28, 1950, he was asymptomatic and had gained 30 lbs. in weight. His appearance and outlook have improved remarkably. He has returned to work and seems to be strikingly rehabilitated. Roentgenogram of the abdomen on March 2, 1950, showed no change in the degree of calcification (Fig. 13).

DISCUSSION

Many procedures heretofore performed for the surgical management of pancreatitis with calcification have been directed not at the disease but at one incidental manifestation. Pancreatectomy has been advocated.[8] This operation is very difficult because of the fixation of the gland and the occasional presence of a surrounding venous dilatation. The poor results in terms of mortality, resultant diabetes, and digestive disturbances overbalance the relief of pain which some patients have exhibited.

Removal of the calcific deposits is not a logical attack on the disease.

Interruption of the autonomic nerve fibers transmitting pain sensation is a successful maneuver for relief of the pain.[2, 9, 10, 11] However, the accompanying diarrhea and fat intolerance, the diabetes, if present, and general nutritional deterioration may progress.[10]

Recurrent attacks of acute pancreatitis may not be accompanied by overt pain or the symptoms may be so vague as soon to be forgotten. The damage with each attack may eventually convert the gland into a fibrosed organ, oocasionally complicated by calcification. It is not surprising therefore, that cases of calcification are reported without any previous history of pain. Such a case noted on our service is outlined:

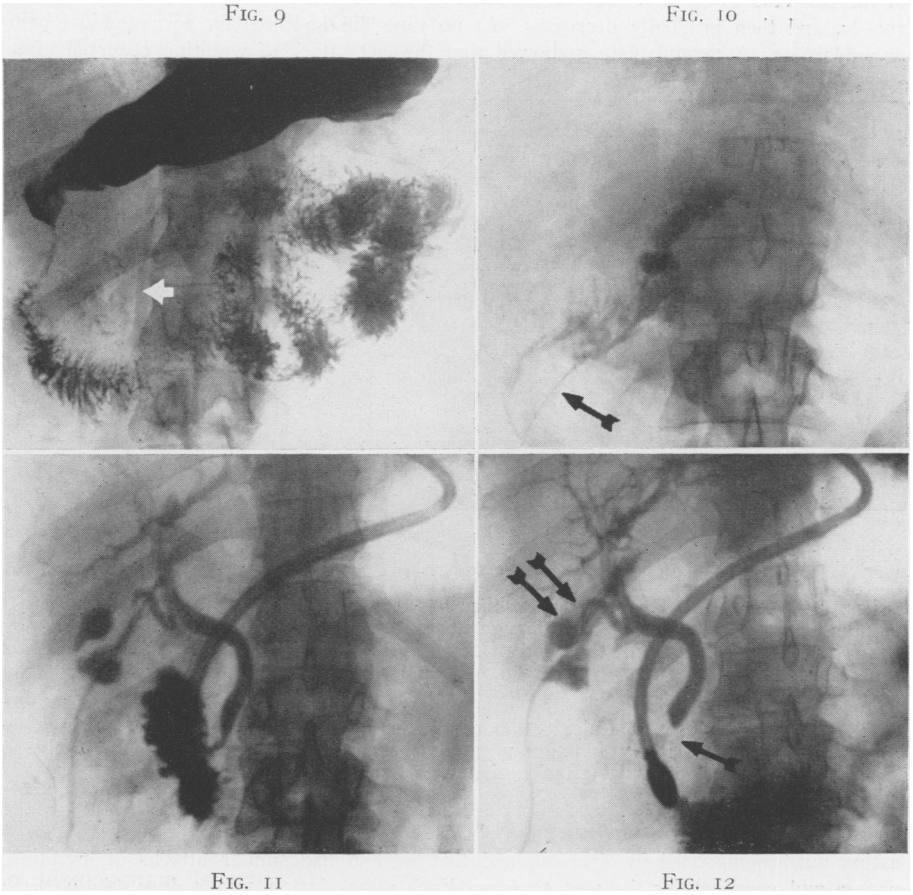

FIG. 9.—(Case 4) Preoperative roentgen ray studies showed calcification of the head of the pancreas (arrow) with marked narrowing and distortion of the duodenum.

FIG. 10.—(Case 4) Operative pancreatogram showed the proximal half of the main pancreatic duct to be markedly dilated and tortuous but not obstructed. Visualization was achieved by injecting Diodrast through a fine tube (arrow) inserted into the pancreatic duct.

FIG. 11.—(Case 4) Postoperative cholangiogram showed the T-tube to be lying in the common hepatic duct. The common duct and the cystic duct (left behind at a previous operation) were clearly visualized by the dye which ran readily into the duodenum.

FIG. 12.—(Case 4) Following the administration of morphine, the postoperative cholangiogram showed compression of the intra-mural part of the common duct (arrow), demonstrating that the one-way valve action of the duodenal wall was intact. The cystic duct could be seen to communicate with a very small remnant of the gallbladder (two arrows).

Case 5.—A. M. (Bellevue Hospital, 75767–49) was a 57-year-old man admitted to the 3rd (New York University) Surgical Division on October 8, 1949, with a 2-day history of severe vomiting and diarrhea, following the eating of mushrooms picked in a field. Although the white blood count was 16,250, there were no abdominal signs or symptoms except moderate distention. The vomiting stopped after one day in the hospital. However, a routine serium amylase on October 10, 1949, was 815 mg. per 100 cc. Close examination of the roentgen-ray films of the abdomen showed calcification of the head of the pancreas (Fig. 14). The serum amylase on October 11, 1949, was 1100 mg. per 100 cc. and then gradually decreased. At no time did he complain of abdominal pain or tenderness. A careful history showed that the last attack of vomiting occurred after

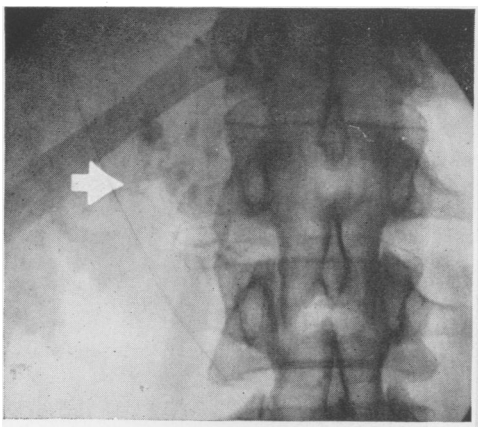

FIG. 13

FIG. 13.—(Case 4) Roentgenogram three months after operation reveals no change in the degree of calcification of the head of the pancreas (arrow).

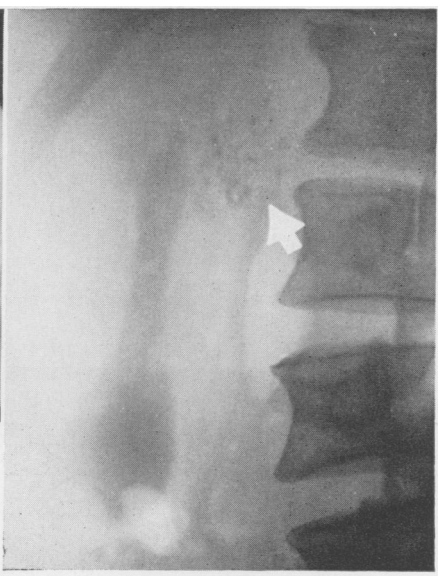

FIG. 14

FIG. 14.—(Case 5) Calcification of the head of the pancreas (arrow) in a patient with a normally functioning gallbladder (dye concentrated). The only symptoms were occasional attacks of nausea and vomiting.

eating mushrooms fried in olive oil, and that he had had similar attacks of sweating, nausea and vomiting for the past 3 years. Each attack lasted 3 to 4 days. At no time was abdominal pain or tenderness present.

Roentgen ray examination of the gallbladder, stomach and duodenum showed no abnormality except for a small duodenal diverticulum. A secretin test revealed normal function (volume 165 cc. bicarbonate 98 cc. and amylase 1084 units).

Although the patient refused operation, we believe that serious consideration should be given to operative section of the sphincter of Oddi even in the absence of pain. Prevention of further reflux of bile and recurrent episodes of pancreatitis will stop the progress of the disease.

The abolition of pain and the gain in weight following sphincterotomy in the four patients described above proves that the pain in calcification of the pancreas is due to recurrent pancreatitis and not to the presence of calculi.

Although this operative procedure in advanced cases may not improve function already lost, it should prevent further deterioration of the gland.

SUMMARY

Calcification of the pancreas occurs as an occasional incident in the development of chronic pancreatitis. Pain, if present, will be abolished, and recurrent severe attacks of pancreatitis prevented, by section of the sphincter of Oddi. This simple procedure will arrest the progress of the disease, and salvage the residual external and internal pancreatic function.

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DISCUSSION.—DR. LESTER R. DRAGSTEDT: I have been interested in this problem of the pathogenesis of acute pancreatitis or acute pancreatic necrosis for many years.

It is possible to produce the disease experimentally in animals in a great variety of ways, but of all of these experimental methods for producing pancreatitis, the one most likely to occur in human pathology, it has always seemed to me, is the reflux of bile into the pancreatic duct, as postulated by Opie and by Archibald.

I think this recent work of Dr. Mulholland and Dr. Doubilet has provided us with very important evidence confirming the view of Archibald and Opie. They have clearly demonstrated the existence of a common channel in patients with recurrent acute pancreatitis.

Not only do we have that positive evidence with respect to the relation of reflux of bile to the disease, but we also have negative evidence from the very large amount of surgery on the pancreas, both in man and in animals, that has been done in recent years.