TRANSDUODENAL SPHINCTEROPLASTY FOR RECURRENT PANCREATITIS

A PRELIMINARY REPORT*

S. AUSTIN JONES, M.D. AND LOUIS L. SMITH, M.D.
LOS ANGELES, CALIF.

FROM THE LOS ANGELES COUNTY GENERAL HOSPITAL, LOS ANGELES

THE OBJECT of this paper is to describe a surgical procedure which we feel will permanently interrupt the function of the ampulla of Vater and reduce the effect of the duodenal wall on the intramural portion of the common duct. This procedure may have been previously reported, but we have found no reference to it in the literature. The operation was devised for the treatment of patients with chronic relapsing pancreatitis and depends for its success on the presence of a common channel as the cause of the disease. We will present some of the theories of etiology of the disease, various surgical procedures that are being employed in its treatment, a description of the operation we have used, and a preliminary report of five cases on whom this operation has been done.

THEORIES OF ETIOLOGY OF PANCREATITIS

I. COMMON CHANNEL THEORY

There is good evidence, both clinical and anatomical, to show that when the pancreatic duct enters the common bile duct proximal to the ampulla of Vater, obstruction of the ampulla by spasm, edema, tumor, or stone may result in reflux of bile into the pancreatic ducts. Many feel that the majority of cases of pancreatitis are caused by this regurgitation.[36, 40] A brief summary of the arguments given for and against this theory is as follows:

A. Points in Favor of This Theory. 1. *Anatomical.* In autopsy dissections, the anatomical incidence of a common channel was 20 per cent of 200 cases as determined by Mann and Giordano.[32] Cameron and Noble reported figures on 354 dissections with an incidence of 76 per cent.[9] Howard found a common channel in 54 per cent of 150 dissections.[27] It has been argued that the pancreatic secretory pressure is greater than the combined pressures exerted by the liver and gallbladder, and that it would be impossible for bile to pass retrograde into the pancreas. However, it has been demonstrated that the duct of Santorini, which empties into the duodenum without a sphincter at its termination, connects with the duct of Wirsung in 36 per cent of dissections, thus nullifying the secretory pressure of the pancreas and permitting bile reflux.[20, 27]

2. *Cholangiography.* Using this method, a common channel was demonstrated in 17 per cent of normals and in over 50 per cent of individuals with pancreatitis.[27] In 35 cases having a common channel demonstrated by cholangiography, 100 per cent had a very distinct rise in blood amylase following reflux of the dye into the pancreatic duct.[16]

3. *Presence of Pancreatic Fluid in the Gallbladder.* By examining fluid aspirated from the gallbladder, Popper demonstrated the presence of pancreatic enzymes in 20 of 200 people without pancreatitis, and in 16 of 18 people with the disease.[37]

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4. *Combined Methods.* Doubilet and Mulholland use three methods of demonstrating this common channel. First, by cholangiography; second, by giving secretin intravenously when the ampulla is under direct vision and observing the discharge of pancreatic juice; and, third, by finding pancreatic enzymes in bile obtained from T-tube drainage after secretin administration. Using one or more of these three methods, they demonstrated the presence of a common channel in 48 of 49 cases of pancreatitis.[18]

5. *Clinical.* Transduodenal sphincterotomy, a method of preventing bile reflux, has been used by Mulholland, Doubilet and others. The results have been sufficiently successful to justify the continuance of this operation as a definitive method of surgical treatment for chronic relapsing pancreatitis.[15-19]

B. Points Against This Theory. It should be emphasized that the common channel does not satisfactorily explain all cases of pancreatitis. It is known that pancreatitis may occur only in the wedge of pancreas drained by the duct of Santorini, and can develop in a pancreas where there is no common channel.[2] Some feel that spasm of the ampulla is associated with constriction of the muscle fibers surrounding the opening of the pancreatic duct.[32] If true, this would prevent reflux of bile. It has also been stated that the pressure necessary to produce pancreatitis experimentally by the injection of bile into the pancreatic ducts is greater than can be physiologically developed by the gallbladder and liver.[32] This observation was made in the experimental animal, and may not apply to the human.

II. BILIARY TRACT DISEASE

It is known that 70 to 85 per cent of patients with acute pancreatitis have associated biliary tract disease, with or without stones.[1] Over 50 per cent of cases of pancreatitis have biliary tract stones. Cholelithiasis is six times more common in autopsies on patients with pancreatitis than in a control group of autopsies on patients without pancreatic disease.[5] It must be noted that the majority of patients with cholelithiasis do not have pancreatitis, and also that cholecystectomy and common duct drainage for pancreatitis frequently fails to prevent further attacks of the disease.[22]

III. PANCREATIC DUCT OBSTRUCTION

Lium and Maddock have demonstrated that obstruction plus stimulation of the gland results in experimental pancreatitis, while neither obstruction nor stimulation alone will produce the disease.[30] Rich and Duff's theory of duct metaplasia as a cause of intra-pancreatic obstruction has been strongly questioned, as Yotuyanagi and others have demonstrated metaplasia in over 50 per cent of normal glands examined.[43, 52]

IV. ALCOHOL

A large percentage of patients with pancreatitis have a history of alcoholism. Of the cases of chronic pancreatitis reviewed by Edmondson, 38.7 per cent were alcoholics.[21] Alcohol in small amounts causes stimulation of both hydrochloric acid and pancreatic secretion. In higher concentration, however, it is inhibitory to both hydrochloric acid and pancreatic secretions, but causes marked irritation of the gastric and duodenal mucosa.[23] Richman and Colp state that alcohol *per se* in contact with duodenal mucosa produces large amounts of secretin.[44] Its exact role in the production of pancreatitis is not clear. Some state that alcoholism is more common with chronic relapsing pancreatitis than with acute pancreatitis, and postulate that these are two different disease entities.[39] The duodenitis produced by alcohol may cause occlusion of the ampulla

and intramural portion of the common duct, resulting in pancreatitis when a common channel is present.

V. HYPERVAGOTONUS

Overactivity of the vagus nerves may affect the pancreas in two ways. First, by direct action on the gland, producing an enzyme-rich secretion. Second, by the formation of hydrochloric acid, which, on contact with the duodenal mucosa, elaborates secretin, the main stimulator of water and bicarbonate secretion, and pancreozymin, another hormone produced by the proximal small bowel mucosa which stimulates enzyme secretion.[6, 24-26] Ripstein has reported a 72 per cent mortality from experimental pancreatitis produced in dogs. In a group protected by vagotomy, the mortality was only 24 per cent,[37, 45] Schaffarzick *et al.*, using the same animal, also demonstrated the beneficial effects of vagotomy on experimental pancreatitis.[46]

VI. MISCELLANEOUS CAUSES

These include trauma, systemic disease such as mumps, bacterial infection, direct or blood-borne, penetrating duodenal ulcer, and vascular accidents.

Surgical Procedures Employed in Therapy

1. Cholecystectomy and Common Duct Drainage. Most authorities agree that the first echelon of attack is the eradication of any existing biliary tract disease, followed by long armed T-tube drainage for six to 12 months. Approximately two-thirds of the patients so treated have recurrence of their disease.[22]

2. Procedures Interrupting the Function of the Ampulla of Vater. A. *Sphincterotomy,* as used by Mulholland and Doubilet, prevented further attacks of pancreatitis in 45 of their 51 cases.[18] They have discarded the transcholedochal approach in favor of incision of the sphincter under direct vision with the duodenum open. They then remove the gallbladder whether it is diseased or not, feeling that in human beings, as in dogs, the filling of the gallbladder is dependent on the integrity of the ampulla. When ampullary function is lost, there is no back pressure and distention of the gallbladder does not occur, the organ becoming a flaccid sac which may act as a nidus for infection.[18, 34] The results in our cases to be described later bear out this contention.

Sphincterotomy has not been universally successful in curing all cases of pancreatitis, even when a common channel is present. It is possible that in some cases a simple incision may heal with scarring, resulting in stricture or return of sphincter function. The muscular structure of the ampulla of Vater has been compared to the anal sphincter. While these structures are not entirely analagous, it should be stated that incision of the internal and external anal sphincters does not always result in permanent incontinence.[3, 4]

Following sphincterotomy, contraction of the duodenal wall produced by emotional stress,[35] or by drugs,[49] exerts a constricting effect on the lower end of the common duct as it passes obliquely through the duodenal wall, with resultant rise in the common duct pressure. This elevation of pressure has been shown to parallel a rise in duodenal tonus.[49] In the event that the pancreatic duct entered the common duct above this area of constriction, reflux could occur after sphincterotomy.

A long limb T-tube is used by some,[11, 38, 50] as a stint to maintain patency of the ampulla after sphincterotomy, and is left in place for a period of six to 12 months. Prolonged wearing of the T-tube, besides the obvious annoyance to the patient, is not entirely innocuous, since pancreatitis has resulted from its use.[48]

B. *Sphincteroplasty,* the plastic procedure we have used on the ampulla, is simply a method of obtaining a more complete

and permanent sphincterotomy, and will be discussed in detail later.

3. Vagotomy. Vagotomy may prove to be of real value, having a sound physiologic basis, as previously discussed. Vagotomy combined with sphincteroplasty may prove to give better results than either procedure used alone. In an effort to evaluate sphincteroplasty, we have elected to omit vagotomy except in one case where a coexisting duodenal ulcer made its use advisable. If vagotomy is done, some drainage procedure, preferably pyloroplasty, should accompany it.

4. Gastric Resection. This procedure has as its basis the reduction of acidity and hence the diminution of secretin formation by the intestinal mucosa. It has been said that more secretin is produced by the duodenum than by the jejunum, which would indicate the advisability of using a long loop anterior anastomosis. However, Comroe has stated that the relative quantities of secretin that can be formed in the duodenum and the jejunum never have been accurately worked out, and that certain foods may stimulate the formation of secretin when they come in contact with

FIG. 1

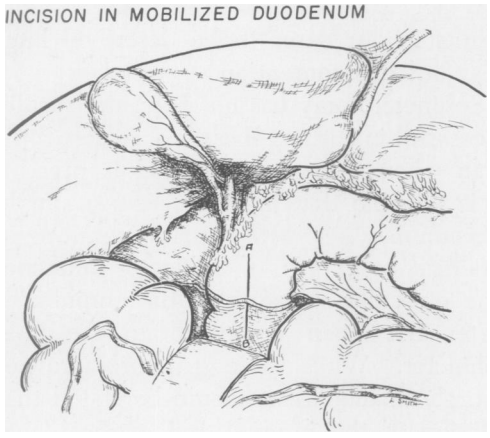
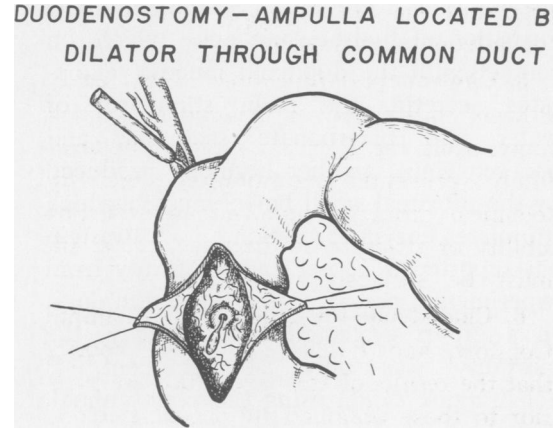


FIG. 2



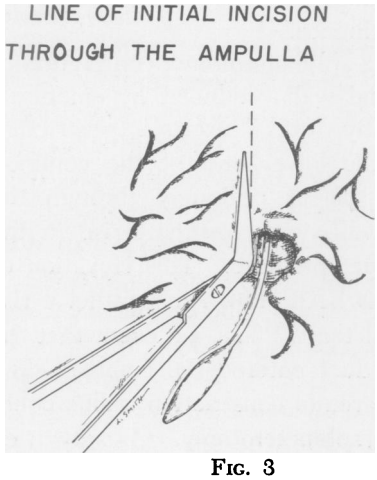


FIG. 3

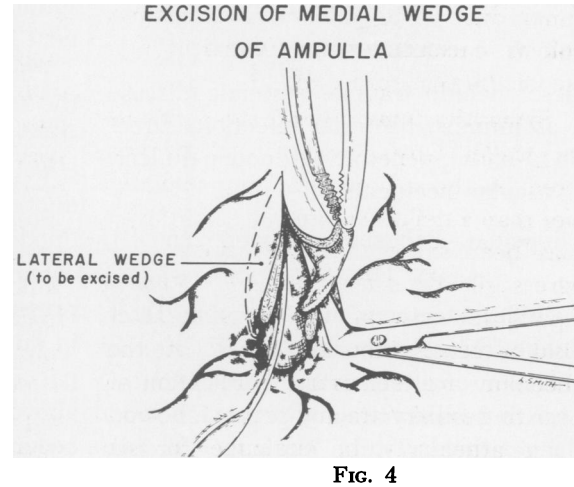


FIG. 4

the mucosa of the small intestine.[12] This would suggest that the length of the loop would be of more theoretical than practical value.

Another possible advantage of gastrectomy is the diversion of gastric contents away from the duodenum, resulting in a reduction of any existing duodenitis. The procedure has been used in too few cases for adequate evaluation.

5. Choledocho-jejunostomy, Roux en Y. Bowers' procedure of choledocho-jejunostomy, using the Roux en Y principle, absolutely prevents regurgitation, but the technical problems involved and the possibility of stricture at the anastomotic site must be seriously considered.[7, 8]

6. Choledocho-duodenostomy. Gambill, Comfort, and Baggenstoss have reported that the results of this procedure are superior to those obtained by cholecystectomy and prolonged T-tube drainage of the common duct. The rationale and technical problems encountered are identical with those of Bowers' procedure.[22]

7. Sympathectomy. Sympathectomy, despite Mallet Guy's contentions,[31] is considered by the majority to be a palliative rather than a definitive procedure.[13, 14, 28, 41] It has been shown that the disease may progress silently after the interruption of sympathetic nerve fibers. Long term appraisal indicates that the results of sympathectomy are temporary.[11] Ripstein was unable to demonstrate any protective action of sympathectomy in experimental pancreatitis produced in dogs.[45]

8. Miscellaneous Procedures. Pancreatico-jejunostomy,[10] or stripping of the choledochal nerves from the common duct,[42, 47] are technically complicated procedures. Partial or complete pancreatectomy are formidable operations but may be advisable in selected cases.[10, 11] Ligation of the duct of Wirsung has been done in too few cases for adequate evaluation.[33]

TECHNIC OF SPHINCTEROPLASTY WITH DISCUSSION

Feeling that the common channel is the most frequent cause of chronic pancreatitis, our approach to this problem has been common duct exploration, a transduodenal plastic procedure to permanently interrupt the function of the ampulla of Vater and duodenal wall, followed by long-armed T-tube drainage for a period of ten to 14 days. After considering the suggestion of Dr. Mulholland, and utilizing our brief experience, we are now impressed with the advisability of removing all gallbladders at the time of sphincteroplasty whether or not they are diseased. Should the patient develop a recurrence following the surgical procedure outlined above, we would consider definitive pancreatectomy or sympathectomy as a palliative measure.

The procedure we have used consists of a transduodenal approach (Figs. 1 and 2) and a plastic procedure on the sphincter, excising a wedge-shaped portion of common duct, duodenal wall, and sphincteric muscle (Figs. 3 and 4), followed by an approximation of the duodenal to the common bile duct mucosa with interrupted sutures of fine silk. This results in the formation of a wide trough-like stoma which should completely interrupt the function of the sphincter and heal with minimal scarring (Fig. 5). We have left a long-armed T-tube in the common duct to prevent obstruction by early edema, and have removed it after ten to 14 days (Fig. 6). It should be noted that the initial incision through the ampulla is 1½ cm. in length and is made in its anterolateral aspect to avoid injury to the pancreatic duct which may enter on the medial side (Fig. 3).

The plastic procedure described, with mucosa-to-mucosa approximation, will minimize the possibility of scarring. The wide excision of ampullary and duodenal musculature should minimize the constricting

action of the duodenal wall on the lower end of the common duct. There is no need for prolonged T-tube drainage, and in future cases we plan to use a short-limbed tube for ten to 12 days to act as a stint and permit postoperative studies.

While it might be felt that such complete destruction of ampullary function would result in ascending cholangitis, Waltman Walters[51] and others have pointed out that obstruction rather than reflux is the major factor in causing this complication. We feel that obstruction should not occur after a plastic procedure such as the one described. Also, it must be pointed out that the object of the procedure is *complete* and *permanent* destruction of sphincteric action, and sphincteroplasty should accomplish this more completely than would simple severance of the sphincteric muscle bundle.

CASE REPORTS

Case 1.—(995-136) This was a 43-year-old Negro male, a chronic alcoholic who had been treated on 6 previous entries for attacks of pancreatitis. During these episodes his complaints were upper abdominal pain which radiated into the back and which was associated with nausea and vomiting. During these attacks, epigastric and left upper quadrant tenderness were the usual findings. His blood amylase studies ranged between normal and 720 units.

On August 13, 1951, he entered the hospital with epigastric pain radiating to the back, nausea, and vomiting. On examination, he had generalized abdominal tenderness. The blood amylase was 243 and blood calcium was normal. He was treated with sedation, antibiotics, atropine, fluids, and suction. When his symptoms subsided, laboratory studies demonstrated a normal liver function. Gastric analysis showed low acidity and a gallbladder dye series revealed good concentration without stones. Flat film demonstrated no pancreatic calcifications.

At operation on September 24, 1951, the external surface of the liver had a fine mottled fibrotic appearance, which on biopsy was reported to be mild ascending cholangitis. On the anterior surface of the prepyloric area of the stomach, adjacent to the greater curvature, there was a firm nodule 2 cm. in diameter. On opening the stomach and inspecting the mass from inside, a duct opening was noted entering normal gastric mucosa, which on pressure discharged a serous secretion. Rapid frozen section confirmed the clinical impression of heterotopic pancreatic tissue. The head of the pancreas was firm and fibrotic and there were adjacent inflammatory lymph nodes. The ectopic pancreatic tissue was excised, necessitating pyloroplasty to facilitate closure. The gallbladder was thin walled and contained no stones. The common duct, which was normal in size, was opened and explored and no stones found. A transduodenal sphincteroplasty was then done and a long-limbed T-tube placed in the common duct and passed into the duodenum.

His recovery objectively was uncomplicated; however, the patient complained of epigastric pain and nausea after eating until several days prior to discharge from the hospital, at which time his complaints suddenly and completely disappeared. During this period of discomfort, amylase and diastase studies were entirely normal. Upper gastro-intestinal series prior to discharge showed antral spasm as well as reflux of barium into the common duct. At no time did the patient have jaundice, chills, fever, or other evidence of cholangitis. The T-tube was removed two weeks after operation.

He left the hospital on November 11, 1951, and was seen two weeks later in the clinic completely free of symptoms, and having gained some 20 pounds. He has returned to the hospital twice since this time with vague abdominal complaints without clinical or laboratory evidence of pancreatitis. It is felt that this patient's psychologic make-up and homosexual tendencies have made the results of sphincteroplasty difficult to evaluate. To date he has had no postoperative clinical or laboratory evidence of pancreatitis. A cholecystogram taken three and one-half months after operation showed a non-functioning gallbladder.

Case 2.—(94522—Private case of S. A. J.) This 36-year-old white male, a chronic alcoholic for over 5 years, had had repeated attacks of epigastric pain aggravated by alcohol over a 15-month period. Gastro-intestinal series at the onset of his illness showed a probable duodenal ulcer. His amylase had been elevated during two of his episodes of pain and was not taken on the others. He had had no jaundice, hematemesis, or melena. On 2 occasions his symptoms were of such severity that he had required treatment for shock.

The present illness began September 26, 1951, when he developed severe epigastric pain, radi-

ating through to the back, vomiting, and all the signs and symptoms of severe shock. He was admitted to the hospital as an emergency, where, on examination, he had absent peristalsis and marked rebound tenderness and rigidity in the mid-epigastrium. The amylase was 400 units.

Following treatment with suction, atropine, blood, and intravenous procaine, he improved rapidly and was asymptomatic within 3 days. A gallbladder dye series showed normal gallbladder function and a second upper gastrointestinal series showed no evidence of gastric or duodenal disease. Liver studies were normal.

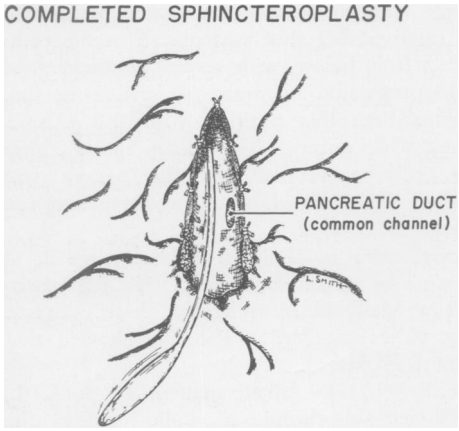


FIG. 5

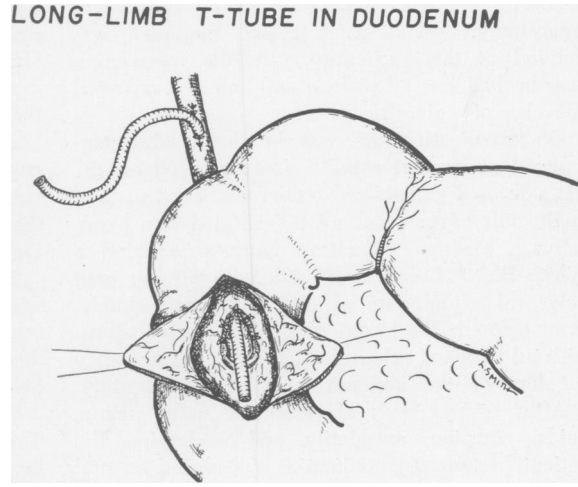


FIG. 6

At operation on October 4, 1951, the pancreas was found to be firm and nodular throughout its entire length. The first portion of the duodenum was indurated consistent with an old healed duodenal ulcer. The gallbladder and the liver were grossly normal. The common duct was not enlarged and contained no stones.

Because of the past roentgen ray evidence and present findings of duodenal disease, a vagotomy was done, followed by a pyloroplasty. The common duct and duodenum were then opened, a transduodenal sphincteroplasty performed, and a long-armed T-tube placed through the common duct into the duodenum.

His postoperative course was uneventful except for a sinus tachycardia demonstrated by electrocardiogram, which cleared spontaneously after 48 hours. The T-tube was removed on the twelfth postoperative day.

Since the operation he has gained 8 pounds and has been entirely free of any symptoms. A repeat gallbladder dye series done 4 months postoperatively demonstrated no function of this organ.

Case 3.—(518-896 This was a 34-year-old Negro housewife who entered the hospital on November 5, 1951, with constant epigastric pain and severe vomiting of one week's duration. She gave a history of "stomach trouble" for years with epigastric pain aggravated by eating. The patient also admitted chronic alcoholism. Review of her old hospital record revealed that she had had 3 previous major attacks of pancreatitis, 1 of which was associated with an electrolytic imbalance including a severe hypopotassemia.

On examination, the patient was in marked shock. Her abdomen was distended, tender, rigid, and silent. Blood amylase was 569 units. She received vigorous anti-shock therapy with blood and oxygen, as well as treatment for pancreatitis including analgesia, atropine, fluids, suction, and antibiotics.

Following recovery, the liver function studies were as follows: Albumin 4.0, globulin 4.1, icteric index 11, cephalin flocculation 2 Plus, and thymol turbidity 3. Gastric acids were low and a cholecystogram showed normal function.

At operation on November 29, 1951, there was old and recent evidence of right upper quadrant inflammation with dense fibrous adhesions. The liver and gallbladder were grossly normal, and the pancreas was firm and enlarged one and a half times normal size. Exploration of the common duct showed it to be small in diameter but patent and free of stones. Transduodenal sphincteroplasty was done and, since no small long-limb T-tube was available, a No. 12

Robinson catheter was passed through the common duct into the duodenum and brought out through a stab wound.

The patient made an uneventful recovery. The catheter was removed on the ninth postoperative day. The patient left the hospital on the twelfth postoperative day, and has had no recurrence of symptoms.

Case 4.—(1300-834) This was a 45-year-old Caucasian male who had been a chronic alcoholic for 11 years. He entered the hospital on October 10, 1951, complaining of intermittent left lower quadrant pain of 2 week's duration, associated with anorexia but with no nausea or vomiting. One year previously, at another hospital, a diagnosis of acute pancreatitis was established at laparotomy. The right upper quadrant was drained at this procedure. In the intervening year he had lost 12 pounds and had had repeated episodes of epigastric pain.

On examination, he was in shock with generalized abdominal rigidity, more marked on the left side, and generalized tenderness, most marked in the left lower quadrant. Peristalsis was hypoactive. Positive laboratory findings included a White Blood Count of 19,400 with 80 per cent polys, blood amylase of 405 units, and cloudy, straw-colored fluid on abdominal paracentesis, with an amylase value of 3550 units. Emergency flat film of the abdomen was non-contributory.

After several days of treatment with suction, fluids, atropine, antibiotics and analgesics, the patient became asymptomatic, was placed on oral feedings, and ambulated. On October 19, 1951, he developed severe epigastric pain, and an amylase at this time was 545 units. Three days later a palpable epigastric mass was discovered which was consistent with a pancreatic pseudocyst. He complained of almost continual abdominal pain, ate poorly, and developed peripheral edema on October 30, 1951, at which time he had an albumin of 2.8 and a globulin of 3.3. Following supportive measures consisting of blood and supplementary feedings for 2 weeks, albumin was 3.3, globulin 3.4 and peripheral edema had disappeared. At this time his icteric index was 7, thymol turbidity 3, and cephalin flocculation 2.

On November 14, 1951, an upper gastrointestinal series showed moderate reflex spasm of the third portion of the duodenum, but no widening of the loop. A barium enema on November 13 and a cholecystogram on November 26 showed no abnormalities. Gastric analysis showed low acids. The abdominal mass was no longer palpable.

At operation on December 3, 1951, marked inflammatory changes in the region of the hepatoduodenal ligament and in the region of the head of the pancreas were noted. This organ was enlarged to one and one-half times normal size, and was firm throughout its entire length. The liver and the gallbladder showed no apparent pathologic change. The common duct could not be isolated in the dense mass of inflammatory tissue described. There was local evidence of fat necrosis but no pancreatic cyst. As it was felt too dangerous to incise the grossly edematous hepatoduodenal ligament, the common duct was searched for with a needle on a syringe. This method was not successful. Accordingly, the duodenum was opened and the ampulla of Vater located by injecting a weak solution of methylene blue into the gallbladder, compressing this structure, and visualizing the dye as it entered the duodenum. A transduodenal sphincteroplasty was then performed, and a small rubber stint was placed in the common duct from below, with several centimeters of the tube projecting downward into the duodenum, and sutured in this position with 4-0 chronic gastro-intestinal suture. The postoperative course was entirely uneventful and the patient was discharged from the hospital asymptomatic on December 14, 1951.

He was seen in clinic in February, 1952, at which time he was asymptomatic. He had a nonfunctioning gallbladder on repeat cholecystogram done at this clinic visit. The film showed that his stint had passed.

Case 5.—(15451—Private patient of S. A. J.) This 38-year-old chronic alcoholic entered the hospital on January 27, 1952, with a one-day history of epigastric pain radiating through to the back, and vomiting. Past history included 6 entries at the Los Angeles County General Hospital between September, 1948, and May, 1951, with a diagnosis on each entry of acute pancreatitis. Studies made during these attacks showed amylase values varying from 460 to 832 units, and a urinary diastase value on one occasion of 38,000. Blood calciums ranged from 7.7 to 9.3 mg. per 100 cc. Gallbladder studies and upper gastro-intestinal series were normal. Liver function tests showed no evidence of disease.

On examination, the patient had moderate tenderness in the mid-epigastrium with slight rigidity and rebound in the same area. Peristalsis was diminished. On the day of entry the amylase was 75 units, calcium 9.2 mg. per 100 cc., albumin 4.7, globulin 2.5. The following day a urinary diastase was normal. The amylase was repeated on January 29 and was again normal. On January 30, the bromsulphalein showed 14 per cent retention in 45 minutes, cephalin flocculation was negative, the gallbladder study showed poor function, and the upper gastro-intestinal study was negative.

His initial diagnosis was acute gastritis, and the immediate treatment consisted of suction and mild sedation for 2 days, which resulted in a complete cessation of symptoms. Because of the many past attacks of proved pancreatitis, it was decided to do definitive surgery at this entry.

At operation on February 6, 1952, he was found to have a pancreas that was slightly enlarged and very fibrotic throughout its entire length. There was moderate inflammatory reaction at the junction of the head of the pancreas with the hepatoduodenal ligament. The gallbladder contained no stones, and the liver appeared grossly normal. The common duct was explored and no stones were found. A transduodenal sphincteroplasty was performed, and a long-limb T-tube placed in the common duct and passed into the duodenum. A cholecystectomy was then done.

In this case, the long limb of the T-tube was tied with a silk ligature to permit a postoperative cholangiogram. The postoperative course was entirely uncomplicated. Cholangiogram nine days postoperatively showed no obstruction and the T-tube was withdrawn, the patient being discharged the following day.

He has subsequently had no complaints whatsoever.

SUMMARY

1. While the etiology of pancreatitis is not clear, the weight of evidence at present favors the common channel theory as the best explanation in the majority of cases.

2. Hypervagotonus may prove to be of significance in the etiology of this disease.

3. Sphincteroplasty, the surgical procedure we have described, is a method of producing a more complete and permanent sphincterotomy. We believe that the simplicity of the procedure, the complete destruction of the ampullary function, the mucosa to mucosa approximation, the reduction of duodenal wall constriction on the lower end of the common duct, and the short period of T-tube drainage required are distinct advantages.

4. From the clinical and experimental evidence available, it seems advisable to remove the gallbladder in cases on which sphincteroplasty is done, whether the organ is diseased or not. This will be confirmed if the nonfunctioning gallbladders described following sphincteroplasty become diseased.

5. Sphincteroplasty is applicable to any situation where there is an obstruction of the lower end of the common duct requiring sphincterotomy for its relief.

6. Vagotomy combined with sphincteroplasty may prove more successful than either procedure used alone in the treatment of chronic pancreatitis. When sphincteroplasty *per se* has been evaluated, we plan to combine the two procedures.

7. It should be strongly emphasized that the period of follow-up on the patients reported is inadequate for any final conclusions. This study is a preliminary report.

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