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CAUDAL PANCREATICO-JEJUNOSTOMY FOR CHRONIC RELAPSING PANCREATITIS*

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MORE THAN HALF a century has elapsed since Opie[48] discussed the etiology of acute hemorrhagic pancreatitis by presenting the finding at autopsy of a calculus impacted within the ampulla of Vater. In the course of those 53 years a voluminous and sometimes confusing literature has accumulated on this subject, the great majority of which reflects an attempt to confirm or deny the implication of the common channel as *a sine qua non* of the disease. There can be little disagreement that the contributions in this direction have added immeasurably to our knowledge of the anatomy and physiology of the biliary-pancreatic system, and have seen great practical clinical application. On the other hand, it would appear that the concept of the common channel has by no means received universal acceptance as the *modus operandi* in pancreatitis. Indeed, evidence is already accumulating to suggest that the biliary tree may ultimately be acquitted as a necessary prerequisite for the onset of pancreatic inflammation.

THE BILE FACTOR IN PANCREATITIS

Since the views on pancreatitis as set forth in the following report are at variance with those that would implicate bile, the common bile duct, and the sphincter of Oddi as instruments of primary importance in the etiology of chronic pancreatitis, a prolonged discussion of the merits of the latter viewpoint is not in order. We recognize the probability that many individuals, with and without pancreatitis,[7, 29, 30] have a common channel;[43, 44] that bile salts have been extracted from pancreatic secretions;[24, 50] that the injection of bile[2] (as well as many other irritating substances) into the pancreatic ductal system can produce pancreatitis in the experimental animal, if the pancreatic acini are ruptured;[54, 55] that the common channel can become operative without biliary tract disease in response to drugs,[6, 59, 62] stress,[45] and neurogenic imbalances;[38] and that relief from pancreatitis has been recorded in many instances by ablation of the sphincter of Oddi.[15–18, 52] At the same time, the virtual abandonment of the common channel theory as proposed in this report, does not negate the valuable contributions of those who have proposed destruction of the sphincter of Oddi, or diversion of the biliary flow,[4] any more than it denies that the surgical relief of biliary tract disease should be the first concern of the surgeon who is confronted with the problem of pancreatitis.[25]

The solution of any jig-saw puzzle rests with the ability to show a continuity of pattern among the many component parts. An inspection of the multitude of facts that have accumulated on the subject of pancreatitis during the past half century will, at first glance, appear to result in a series of incompatibilities from which a workable pattern has not yet evolved. Many of these inconsistencies are readily apparent when one

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TABLE I. *Preoperative and Postoperative Secretin Stimulation Studies.*

			Pre-op	Post-op
Duodenal Drainage	Volume (100–200 cc)		123	125
	Amylase (300–1200u)*		79	822
	Lipase (7000–1400u)		62	1363
	Bicarbonate (90–130 M Eq/L)		None	15 M Eq/L
Secretin + Urecholine	#1 (Control)	Amylase (80–150u)	300	294
		Lipase (0.5–1.5u)	5.0	4.3
Blood Levels	#2 (20 Mins)	Amylase	354	288
		Lipase	4.9	3.7
	#3 (40 Mins)	Amylase	377	292
		Lipase	5.7	4.2
	#4 (60 Mins)	Amylase	396	274
		Lipase	6.3	3.5
	#5 (80 Mins)	Amylase	381	295
		Lipase	6.3	3.4
Abdominal pain after secretin			Very Severe	None

*Data enclosed in brackets represent the limits of normal in this laboratory.

reflects upon the problem as a whole. There are many cases of pancreatitis without concomitant disease of the biliary system.[26] There is almost no correlation between the incidence of anatomically demonstrable common channel with the sex of the patient,[44] yet biliary tract disease afflicts the female with four to five times the frequency it afflicts the male.[63] Chronic pancreatitis, on the other hand, has been cited by Waugh[65] as six times more common in men than women. There are cases of pancreatitis without demonstrable common channels, and there is no correlation between the apparent anatomical frequency of a common channel among the population at large, and the incidence of either biliary tract disease or pancreatitis.[44]

Studies on intrabiliary pressures under various conditions of stimulation have not recorded pressures of sufficient intensity to rupture pancreatic acini,[14] and indeed, patients so studied did not develop pncreatitis in response to the tests.[62] The incidence of pancreatitis associated with routine postoperative cholangiography is very low. Copher and Kodama[12] have stated that secretory pressures of bile are insufficient to force bile into the pancreatic tree; and Harms[28] has demonstrated that simultaneous mensuration of pressures within the pancreatic and common bile ducts reveals an excess in pressure of 38 to 150 mm. water of the pancreatic duct over the bile ducts. Judd[33] demonstrated the anatomy of the sphincter muscle to be such that its contracture closes both the common bile duct and the pancreatic duct, and Mann and Giordano[40] found the sphincter above the opening of the two ducts in most of their cases. Nordmann[47] ligated the ampulla in dogs with a common channel, showing that death ensued from biliary cirrhosis and that pancreatitis was not present at autopsy. By no means the least important evidence is just beginning to make its appearance in the form of failure of sphincterotomy to control pancreatitis,[21, 58] and of failure in 80 per cent of cases to control pancreatitis by complete diversion of the biliary stream.[57]

PANCREATIC DUCT OBSTRUCTION

It might seem apparent from the foregoing that a single concept of the etiology of pancreatitis must inevitably fall short of explaining all cases, and that the disease may have a number of primary causes. There can be little disagreement that the pancreatitis which is occasionally noted to follow mumps[5] represents a disease entity that has escaped accurate classification. The chronic form of pancreatitis may also come to be recognized as a disease secondary to any one of many primary etiologies. Further research will either support the latter possibility, or indeed, may indicate that a great majority of cases of chronic pancreatitis stem from a common etiology. To a certain

TABLE II. *Results of Fecal Fat Analysis on Fixed Fat Intake (Schmidt Diet), Case 1.*

	Pre-op	Post-op
Total Fat (25% of dry wt.)*	42%	21.4%
Neutral Fat (3–12% of dry wt.)	34.5%	13.1%
Split Fat (1–10% of dry wt.)	7.5%	8.3%

*Figures denote percentage of dry weight found in normals.

extent this conclusion can be reached today by retracing our steps to 1901 for a new look at Dr. Opie's original findings.[48]

Dr. Opie's patient died with an acute hemorrhagic pancreatitis. At autopsy a calculus was found impacted within the ampulla of

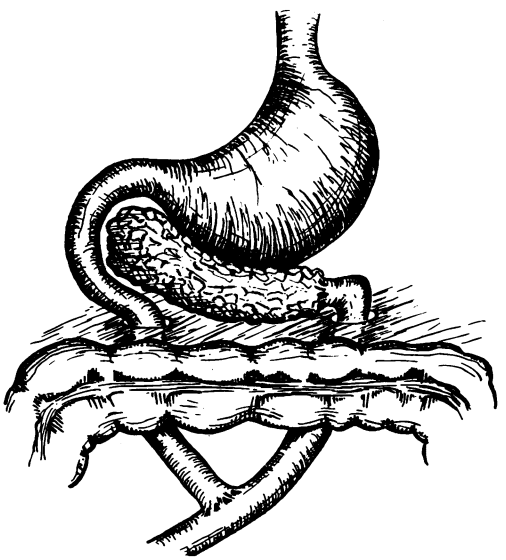

FIG. 1. Schematic representation of revised anatomy at conclusion of operation.

Vater. Had Dr. Opie not noticed that the presence of the calculus converted the terminal segment of the common bile duct into a common channel with the pancreatic duct, the hypothesis might have been advanced that the pancreas had been digested by its own ferments, which had no egress into the duodenum, because of the presence of the calculus at the ampulla of Vater. If this was in fact the cause of pancreatic digestion in that case, can we then ascribe all pancreatitis as secondary to the impedance of outflow of external pancreatic secretion? Has any evidence accumulated to suggest that pancreatic obstruction *per se* may be responsible for pancreatitis, irrespective of the presence of bile and/or a common channel?

In 1922, Priesel[51] demonstrated involvement of the pancreatic ducts by epithelial metaplasia. This finding was again recorded in 1927 as a basal cell metaplasia by Balo and Ballon.[3] In 1936, Rich and Duff[54] went on to demonstrate focal squamous metaplasia causing intrapancreatic obstruction, manifested by dilatation of small distal ducts and acini in 23 of their 24 cases, and involvement of the epithelium of the major duct in 13 cases. Leger[35] then described primary occlusion of the major pancreatic duct above the sphincter in 50 per cent of his cases, and similar instances have also been reported by Appleby[1] and Gillette.[27] Pancreatic calcifications have been shown by Edmundson, *et al.*,[22, 23] to lie within epithelial-lined cavities representing small pancreatic ductules, and parenchymal calcification was not present in this series. They concluded that these findings were explained by pancreatic duct obstruction followed by repeated attacks of pancreatitis.

The anatomical evidence suggesting pancreatic duct obstruction as the etiological agent in the production of chronic pancreatitis would seem to be confirmed by clinical experience. The clinical attack of pancreatitis is almost invariably precipitated by stimulation of the pancreatic secretion by food (especially fats), alcohol, hydrochloric acid, and emotional stress. The pain of pancreatitis is obstructive in nature, and unremitting, until the pancreatic tree decompresses. The ancillary complaints are invariably nutritional in nature, with marked weight loss despite a normal appetite, deficient small bowel function, and passage of fatty stools. The clinical course is one of progressive destruction of the gland with the appearance of cystic change (a likely manifestation of repeated attacks of back pressure), diabetes and steatorrhea.

Physiological support for the concept of pancreatic obstruction comes from quantitative measurement of undigested fat in feces on a controlled fat intake (Schmidt diet[60]); gelatin digestion by feces; elevation of serum amylase and lipase due to reversed absorption of the unexcreted pancreatic enzymes; and more recently, through duodenal

TABLE III. *Preoperative and Postoperative Secretin Stimulation Studies.*

			Pre-op	Post-op
Duodenal Drainage	Volume (100-200 cc)		126	33
	Amylase (300-1200u)*		771	636
	Lipase (7000-14000u)		1197	105.6
	Bicarbonate (90-130 M Eq/L)		50 M Eq/L	20 M Eq/L
Secretin + Urecholine	#1 (Control)	Amylase (80-150u)	441	118
		Lipase (0.5-1.5u)	2.0	0.9
	#2 (20 Mins)	Amylase	421	138
		Lipase	1.6	0.4
Blood Levels	#3 (40 Mins)	Amylase	441	131
		Lipase	1.6	0.8
	#4 (60 Mins)	Amylase	446	139
		Lipase	1.5	0.9
	#5 (80 Mins)	Amylase	474	121
		Lipase	2.0	0.8
Abdominal pain after secretin			None	None

*Data enclosed in brackets represent the limits of normal in this laboratory.

aspiration studies of the ability of the pancreas to excrete amylase, lipase and bicarbonate in response to stimulation by secretin. This latter mode of investigation has been well presented by Lagerlof,[34] Popper,[49] Myrhe,[46] Dreiling,[19, 20] Shingleton,[61] and others, and is worthy of a review by those interested in pancreatic function. Briefly stated, the administration of secretin to a patient with pancreatitis usually results in a diminished duodenal output of amylase, lipase and bicarbonate, in spite of a normal output of bile. At the same time, if the stimulation of the pancreas by secretin is sufficiently great, a clinical attack of pancreatitis may result, with its attendant serum response by elevation of blood amylase and lipase.

TREATMENT OF PANCREATITIS

The treatment of chronic relapsing pancreatitis has taken many forms during the recent years, most of which leave much to be desired when it is recalled that chronic pancreatitis is essentially a benign, crippling disease. Drainage of the biliary tree and resection of diseased gallbladders remain as the first focal point of attack,[25] although it is becoming evident that prolonged drainage of the common bile duct does not preclude the reappearance of symptoms of pancreatitis. Denervation of the splanchnic bed by splanchnicectomy,[13, 39] sympathectomy,[31, 53] and vagotomy[56] has been proposed. Elimination of pancreatic stimulation by hydrochloric acid through gastric resection, with and without vagotomy, is practiced.[10, 11] Subtotal[39] and total[42, 66] removal of the diseased pancreas is recommended by others. Diversion of the biliary system by choledochojejunostomy[4] and choledochoduodenostomy[9] has been claimed successful; and the response to the suggestion of Doubilet and Mulholland[15, 18] that sphincterotomy is the procedure of choice has been overwhelming.[32]

More recently, an approach directed at relief of an obstructed pancreatic outflow has been described, by direct intervention within the head of the pancreas, removing calculi impacted within the major duct,[9] or relocating the duct of Wirsung in the duodenum or loop of jejunum above the site of apparent obstruction.[8, 64] It is also more than likely that a great number of patients have been relieved of their pancreatitis by internal drainage or marsupialization of pancreatic cysts, which have been in communication with the pancreatic ductal tree.

PANCREATIC DUCT DECOMPRESSION

If it is true that pancreatitis is a manifestation of obstruction to the outflow of external pancreatic secretion, then it follows that treatment should be directed at relief of the obstruction. Evidence has already been cited above, suggesting that the site of obstruction is probably in or about the major pancreatic duct within the terminal two or three centimeters of its termination at the duodenum. Since a more accurate formulation of the site and the nature of the obstruction is not yet available, it is more than likely that an attempt to relieve the obstruction at the terminus of the pancreatic duct will ulti-

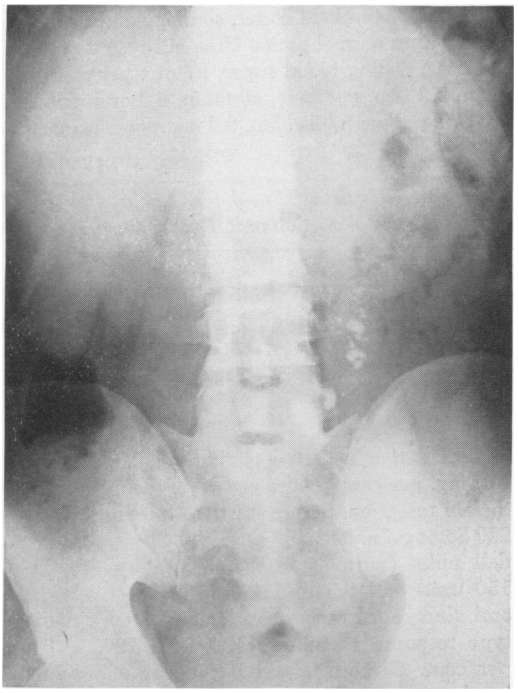

FIG. 2. Roentgenogram of abdomen showing calcification of pancreas (Case 1).

mately fail in a significant proportion of cases. It is because of this failure that the method of decompression of the pancreatic ductal system about to be described, was conceived.

It has been demonstrated that pancreatic juice can flow toward the tail of the pancreas as well as toward the head of the organ.[41] It follows that decompression of a pancreatic ductal system, obstructed within the head of the pancreas, can be achieved with equal facility *via* the tail. It is perhaps pertinent to emphasize that a surgical procedure directed at the tail of the pancreas assumes a greatly lessened morbidity and mortality than a comparable procedure directed at the head. Caudal pancreatostomy, as such, is not a new concept.

In 1911, Link[36] cited a case of chronic calcareous pancreatitis in which he dislocated the tail of the pancreas into the abdominal wall, thereby creating a permanent cutaneous pancreatic fistula, by means of which the pancreatic ductal system was permanently decompressed. This patient survived 30 years, and remained symptom free except on those occasions when transient interference with drainage from the fistula precipitated an attack of pancreatitis. Each of these attacks was promptly relieved by re-establishment of drainage.[37] There is, however, little reason to accept a permanent cutaneous pancreatic fistula with its attendant skin excoriation, fluid and electrolyte losses, and messy dressings, when a permanent, internal fistula can be created without significant increase in risk.

The cases about to be reported were treated by direct anastomosis of the caudal pancreatic duct to jejunum en Roux-Y, with no mortality, minimal morbidity, and without complication. To our knowledge, no similar procedure has heretofore been described. The operative technic consisted essentially of mobilization of the spleen and tail of the pancreas, transection of the pancreas at the junction of body and tail, and end-to-end anastomosis with defunctionalized limb of upper jejunum, brought behind the colon en Roux-Y. The anastomosis was effected between the pancreatic duct and jejunal mucosa, using small mattress sutures of fine catgut, and the greatly thickened pancreatic peritoneal capsule lent itself admirably to the outer layer of interrupted fine silk sutures. The revised anatomy at the completion of the procedure is illustrated diagrammatically in Figure 1.

CRITERIA FOR OPERATION

To present material based only on concepts, and without objective documentation, is not justifiable. Therefore, a program was established at this hospital for cases of chronic relapsing pancreatitis which was designed to demonstrate physiologically the following points: (1) Elevation of serum amylase and lipase during an acute attack; (2) an abnormal quantity of undigested fecal fat on a measured fat intake (Schmidt

diet[60]); and (3) a diminished duodenal output of amylase, lipase and bicarbonate, after stimulation of the pancreas by secretin and urecholine. The establishment of these criteria constituted the major portion of the preoperative work-up, and was undertaken by the Medical Service.*

In addition to the criteria noted above, further documentation of the presence of obstruction of pancreatic outflow was sought at the operating table, and consisted of the following: (4) On section of the pancreas at the junction of the body with the tail, the pancreatic duct should be dilated. (5) Injections of radiopaque fluid material (70 per cent Diodrast®) into the pancreatic duct system, followed by roentgenographic examination, should not reveal egress of the dye into the duodenum. (6) Stimulation of the pancreas by secretin should result in a rise in intraductal pressure well above the resting normal. This was accomplished by affixing the catheterized major pancreatic duct to a manometer filled with isotonic saline solution.

Case 1. The patient was a 44-year-old distillery worker. He had been subject to recurrent attacks of knife-like epigastric pains associated with sweating and vomiting for over 9 years. The attacks had often been precipitated by greasy foods or alcohol, and varied up to 3 days in duration. A diagnosis of pancreatitis was first made at laparotomy during an acute attack in 1947. The patient entered our hospital because of the increasing severity of the pain and a loss in weight of 20 pounds. He had been a relatively heavy beer drinker but had not resorted to the regular use of narcotics.

Physical examination revealed a chronically ill white male, who appeared older than the stated age and showed evidence of moderate weight loss. Vital signs were normal and blood pressure was 140/80. There were no remarkable physical findings except for mild epigastric tenderness deep to a healed transverse incision.

A complete blood count, serology and urinalysis were within normal limits. Urea nitrogen was 9 mg. per cent; fasting blood sugar, 87 mg. per cent; calcium, 9.6 Gm. per cent; albumin, 4.9 and globulin, 2.0 Gm. per cent; bilirubin, 0.6 mg. per cent; cephalin flocculation, negative; cholesterol, 240 and esters, 178 mg. per cent. There was 5 per cent bromsulfathalein retention. Serum amylase on admission was 299 Somogyi units, and lipase, 4.0 Cherry and Crandall units (normals in this laboratory are 80 to 150 units and 0.5 to 1.5 units, respectively). An oral glucose tolerance test demonstrated a diabetic type response as follows 104, 127, 224, 280 mg. per cent. Glycosuria did not occur at any time. Stool examinations were negative for blood, ova and parasites.

TABLE IV. *Results of Fecal Fat Analysis on Fixed Fat Intake (Schmidt Diet), Case 2.*

	Pre-op	Post-op
Total Fat (25% of dry wt.)*	53.3%	42.1%
Neutral Fat (3–12% of dry wt.)	23.6%	14.4%
Split Fat (1–10% of dry wt.)	29.7%	27.7%

*Figures denote percentage of dry weight found in normals.

A study of the pancreatic response to secretin and urecholine was carried out. The results of this test are recorded in Table I, and it is evident from the figures that there was a greatly diminished duodenal content of pancreatic ferments and bicarbonate (although the output of bile was normal). At the same time, the patient experienced a very severe clinical attack of pancreatitis, and the serum content of amylase and lipase showed considerable elevation.

Roentgenograms of the chest were negative. A gastro-intestinal series showed widening of the duodenal swing and flattening of the inner margin. Cholecystograms revealed a normally functioning gallbladder. The degree of pancreatic calcification is readily apparent in Figure 2. Quantitative analysis of fecal fat was carried out while the patient was maintained on a measured fat intake (Schmidt diet). The results are recorded in Table II. The percentages listed indicate percentage of dry weight, and reveal a marked inability of the intestinal tract to digest fats.

On February 10, 1954, under general anesthesia, caudal pancreatico-jejunostomy was performed in accordance with the above description. The patient received one blood transfusion of 500 cc. and tolerated the surgery well. Nasogastric decompression was instituted for 48 hours postoperatively, after which period a regular diet was permitted. He was ambulatory from the first day after operation, and

* Acknowledgment is due to Dr. Julius Wolf, Dr. Wallace Epstein and Dr. Abraham Goldminz, for the supervision and conduct of this investigation, and the study of pancreatic function tests by their Service will be the subject of a separate report.

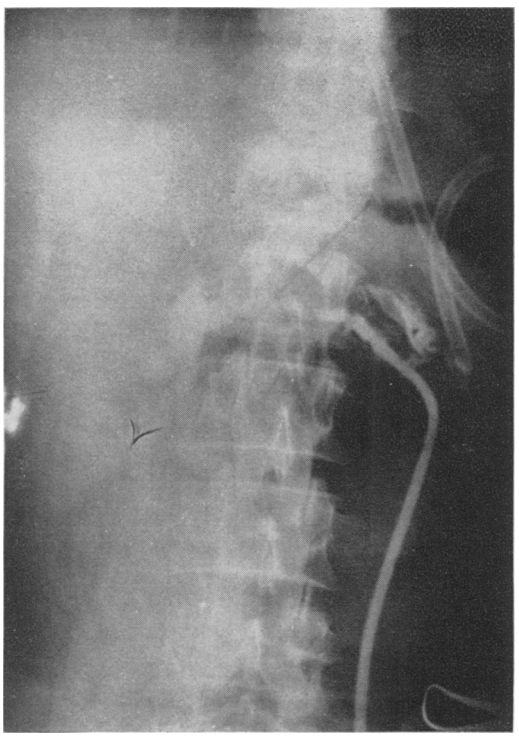

FIG. 3. Pancreatogram at operation (Case 2).

his wound healed satisfactorily. He was encouraged to take those foods at mealtime which had been denied him for the previous 9 years, which he did with much enthusiasm and without distress.

Two weeks after operation the jejunum was intubated with a long intestinal decompression tube and the pancreas again stimulated with secretin and urecholine. It is noteworthy that the patient experienced no abdominal pain with the test postoperatively. The chemical analyses are recorded in Table I, and reveal a markedly improved pattern when compared with that prior to operation. Most notable were the lack of elevation of serum amylase and lipase and the appearance in the small bowel of ten times the preoperative quantity of amylase and twenty times that of lipase. The failure of lipase and bicarbonate to appear in normal concentrations is indicative of long-standing inflammatory disease of the pancreas, and is to be expected under these circumstances.[19] It is planned to reinvestigate these values and repeat the glucose tolerance test at a later date in an effort to show some degree of reversal of pancreatic damage. Postoperative stool analysis for fat digestion indicates a significant improvement in ability to digest fat, as evidenced in Table II.

At the conclusion of the study period the patient was discharged, with instructions to avoid indulgence in alcoholic beverages.

COMMENT

Here is a patient with a documented history of chronic relapsing pancreatitis. That he fulfilled the three preoperative criteria of elevation of serum amylase and lipase, diminished gastro-intestinal utilization of fats, and poor response to pancreatic stimulation by secretin, is evident. The operative criteria were fulfilled as follows: The pancreatic duct at the junction of the body and tail measured 6 mm. in diameter. Radiopaque material did not gain access to the duodenum when instilled into the pancreatic duct under pressure. (This radiograph is technically unsuitable for photographic purposes, but is comparable with the film reproduced below with Case 2.) Administration of secretin under anesthesia resulted in a rapid rise in intraductal pressure to 490 mm. of water. Over a 30-minute period of observation the pressure fell more than half way to normal, with a final reading of 290 mm. Observations beyond this point were deemed inadvisable, since the operative procedure still lay ahead.

Case 2. The patient, a 37-year-old bartender, had experienced excellent general health until one year prior to his admission to our hospital, when he began having rather severe, cramping, epigastric pains. These attacks of pain were usually aggravated by ingestion of fatty foods, and were frequently alleviated by vomiting, sometimes as long as 4 to 6 hours after an attack began. He had lost 100 pounds during the one-year history, although his appetite had remained unaffected until just prior to admission. There had been no noticeable change in bowel habits.

Physical examination revealed a chronically ill and wasted white male who appeared older than his stated age of 37 years. Blood pressure was 110/70, and the remaining vital signs were normal. There were no abnormal physical findings except for a moderate tenderness over the entire epigastrium.

Laboratory data are recorded as follows: A complete blood count, urinalysis and serology were within normal limits. Total protein was 5.9 Gm. per 100 cc., with 4.0 Gm. albumin and 1.9 Gm. globulin. Urea nitrogen was 7 mg. per cent; fasting

blood sugar, 81 mg. per cent; serum calcium, 10.7 Gm. per cent. Cephalin flocculation was negative; thymol turbidity, 0.7; bilirubin, 0.3; cholesterol, 176 with esters, 134 mg. per cent.

During his hospital stay he experienced two attacks of pancreatitis, at which time the serum amylase rose to 518 and 492 Somogyi units; the serum lipase rose to 5.2 and 3.6 Cherry and Crandall units, respectively. An oral glucose tolerance test revealed a diabetic-type response, the figures being 83, 142, 132 and 167 mg. per cent. The patient spilled no sugar in his urine during his hospitalization. Stools were negative for blood, ova and parasites.

Roentgenograms of the chest showed only a mild degree of emphysema. A gastro-intestinal series revealed marked irritability of the gastric antrum and duodenum, with some retention after four hours. Cholecystography demonstrated a functioning gallbladder without evidence of calculi. Plain roentgenograms of the abdomen demonstrated diffuse calcification throughout the entire length of the pancreas.

Studies of pancreatic functions were carried out by means of a secretin-urecholine test, and by quantitative analysis of fecal fat on a measured fat intake. These results are recorded in Tables III and IV, respectively. The depression of pancreatic output of lipase and bicarbonate is evident in Table III, although the amylase was normal. At the same time, the serum response of these enzymes to stimulation of the pancreas by secretin was manifested by a flat curve (at an elevated level). We believe that the failure to achieve a significant serum response may be correlated with the fact that the test did not provoke, in this instance, an attack of acute pancreatitis. The output of bile, determined by chemical analysis of the duodenal contents during the test, was normal. A review of the data recorded in Table IV indicates a marked impairment of ability to digest fat.

On March 10, 1954, caudal pancreaticojejunostomy was performed through a left subcostal incision. He received 500 cc. of blood during the operative procedure and tolerated the surgery well. He was ambulatory from the first postoperative day and his wound healed satisfactorily. Nasogastric decompression was maintained for 72 hours, after which an unrestricted diet was permitted.

Two weeks after operation the pancreatic function tests were repeated. The results are recorded in Tables III and IV.

COMMENT

The second patient also fulfilled the three preoperative criteria of pancreatic dysfunction. The operative criteria were also fulfilled in this case. At the junction of the body of the pancreas with the tail, the major pancreatic duct measured 7 mm. in diameter. A pancreatogram, using 4 cc. of 70 per cent Diodrast®, revealed a dilated major pancreatic duct, obstructed within the head of the pancreas, and no egress into the duodenum (Fig. 3). The intraductal pressure responded to the administration of secretin by rising to 250 mm. of water within five minutes from a resting pressure of 120 mm.

DISCUSSION

In the absence of proof that pancreatitis is a disease due to obstruction of the pancreatic ductal system, fulfillment of the six criteria outlined above has been accepted as presumptive evidence that obstruction does exist. If quantitative analysis of the output of pancreatic secretion indicates subnormal values, then one of two assumptions is justified: either pancreatic acini are not functioning, or the collecting system is obstructed. Taken individually, the preoperative criteria cannot make this distinction. Elevation of serum amylase and lipase during an acute attack, however, is acceptable evidence that pancreatic acini are functioning. Coupled with documentary evidence of diminished pancreatic output, it is tenable to assume that obstruction exists. The operative criteria, being more direct and mechanical in nature, confirm this hypothesis in the cases reported. Finally, demonstrable increase in pancreatic output following the operative decompression leaves little doubt that obstruction had been present.

The presentation of an isolated surgical experience is not intended as proof of the thesis that chronic pancreatitis is a disease of primary obstruction to pancreatic outflow. It is entirely possible that the relief afforded these patients will be transient. If this is so, many factors may be responsible for the ultimate failure, not the least of which may be the specific technics employed. These ques-

tions will be answered in due course. They do not detract, however, from the objective in presentation of this material, namely, to urge a re-evaluation of the approach to the problem of chronic pancreatitis, and to bring to light a simple, apparently effective means for decompression of the obstructed pancreatic tree, carrying almost no morbidity or mortality, and imposing no significant physiological penalty. It is hoped that by so doing, those surgeons in a position to see larger groups of these cases will be enjoined to explore its widest possibilities and application.

SUMMARY

1. The entity of chronic pancreatitis has been discussed in terms of obstruction to the outflow of external pancreatic secretion.

2. A method for decompression of an obstructed pancreatic duct by caudal pancreatico-jejunostomy en Roux-Y has been presented for evaluation.

3. Clinical and laboratory criteria have been presented to justify the surgical approach herein advocated.

4. Two case reports are presented, in which the proposed technic was used successfully.

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