The Cause and Treatment of Pancreatitis Associated with Pancreas Divisum

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Recurrent pancreatitis is more prevalent in the 4% of people with pancreas divisum (nonfused dorsal and ventral ducts), and it has been proposed that the pancreatitis is caused by stenosis at the orifice of the dorsal duct. We have treated 40 patients with pancreas divisum and proven or probable pancreatitis. The diagnoses were made by endoscopic pancreatography showing a foreshortened (<6 cm) ventral duct (Wirsung) and confirmed by postoperative pancreatograms showing the separate main duct (Santorini) emptying via the accessory papilla. Of these, 32 patients (25 men, 7 women, median age 30) had recurrent acute pancreatitis (22) or persistent pain (10) without chronic inflammation or fibrosis. Twenty-nine have been treated by transduodenal sphincteroplasty of the accessory papilla; 22 were stenotic (0.75 mm or less) and 7 nonstenotic. Among 25 patients observed for longer than 6 months after surgery, the relief of pain and pancreatitis has been good in 17, fair in 1, and poor in 7. There was no difference between accessory papillotomy alone (10-0-3) v papillotomies of both accessory and major papillae (7-1-4). Patients with stenosis (16-1-1) fared better ($p < 0.001$) than those without stenosis (1-0-6). Those presenting with discrete attacks (12-1-2) also fared better ($p < 0.05$) than those presenting with chronic pain (5-0-5). The other eight patients (two women, six men, median age 28) had chronic pancreatitis proven by pancreatography and surgical biopsy. In this group, treatment by sphincteroplasty of the accessory papilla failed, and seven patients eventually required a pancreaticojejunostomy (3), distal pancreatectomy (2), or total pancreatectomy (2). In pancreas divisum, pancreatitis is caused by stenosis at the accessory papilla of Santorini. There may be progression from recurrent acute pancreatitis to irreversible fibrosis in some cases. Sphincteroplasty is effective for recurrent acute pancreatitis, but ductal drainage or resection becomes necessary once chronic pancreatitis is established. A preoperative test for stenosis of the accessory papilla is needed to identify patients whose symptoms are genuinely caused by their pancreas divisum.

PANCREAS DIVISUM, an anomaly of pancreatic development in which the major ducts of the dorsal and ventral components fail to fuse, occurs in about 4% of the population.[2,4,5,12,14,17] Although some investigators attach no clinical significance to the anomaly,[10,13] most recent epidemiological studies have found an increased prevalence of pancreas divisum in persons with pancreatitis.[4–7,9,12,14,17] In our review of 519 patients undergoing cholangiopancreatography (ERCP), 12% of those with documented recurrent pancreatitis had pancreas divisum, in significant contrast to 2.9% of those being studied for biliary disease, and 3.3% of those with nonspecific chronic abdominal pain.[12]

The mechanism of the association between pancreas divisum and pancreatitis has not been established. Several papers have suggested that the orifice of the accessory papilla, through which the duct of Santorini must empty, might be inadequate when that duct carries the greater part of the pancreatic secretions, as is the case in pancreas divisum.[2,4,5,12] However, in the several small series reported thus far, treatment of the putative papillary stenosis by sphincteroplasty has successfully relieved symptoms and prevented recurrence of pancreatitis in fewer than 60% of patients.[2,4,6–8,12,16] Other approaches, such as distal pancreatectomy[16] or distal pancreaticojejunostomy, have generally been unsuccessful.

It is likely, in our opinion, that perhaps most of the treatment failures after sphincteroplasty of the accessory papilla occur because of poor patient selection. While the diagnosis of the pancreas divisum anatomical variant is relatively simple, there is no way at present to assess the adequacy of the accessory duct orifice or the intraductal pressures behind it. As a result, it is not possible to know whether the anatomical variant has any functional significance in an individual patient. The anomaly is often coincidental, and when it is not the cause of the patient's problems, any treatment directed at the accessory papilla will necessarily be futile.

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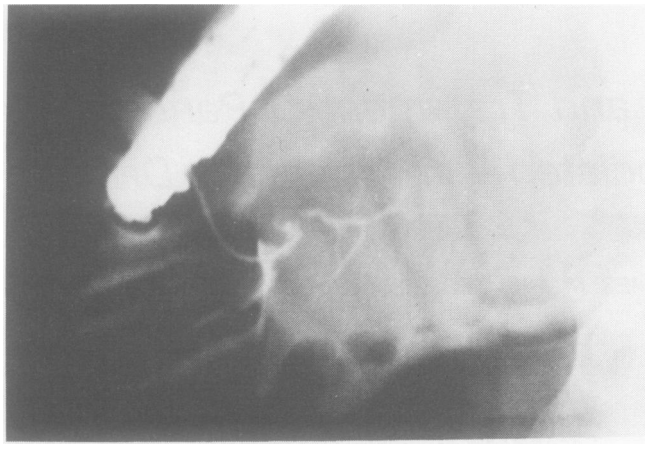

FIG. 1. Endoscopic pancreatogram via the major papilla in a patient with pancreas divisum. The duct of Wirsung is foreshortened and arborizes only within a portion of the pancreatic head.

This report details our experience of 40 patients with pancreas divisum, referred for treatment of proven or presumed pancreatitis. The findings at surgery, correlated with the presenting clinical features and the long-term outcome of treatment, establish the importance of accessory papilla stenosis when pancreas divisum becomes symptomatic.

Patients and Methods

Between July 1975 and April 1983, 40 patients with pancreas divisum were considered for surgical treatment because of documented recurrent pancreatitis or severe refractory abdominal pain, suggesting pancreatitis. Patients in the latter circumstance required narcotic analgesia or repeated hospitalization. In every case but one endoscopic pancreatography[12] showed that the duct of Wirsung was short (2–6 cm), arborizing, and tapered at its distal end (Fig. 1). In the last case, the duct of Wirsung was absent. In 38 patients, there was no detectable communication between the ducts of the ventral and dorsal segments. The diagnosis was made by presuming that the major duct serving the distal pancreas emptied via a separate orifice at the accessory papilla. It was possible to verify that presumption before surgery by cannulating the accessory papilla for pancreatography in only 5 of the 38 cases (Fig. 2). The diagnosis has been confirmed at operation in 30 additional patients.

In the final two cases, a miniscule connection between the ducts of Wirsung and Santorini allowed a trickle of contrast medium to opacify the dorsal duct system when the injection pressure into the duct of Wirsung was increased enough to cause acinar filling (Fig. 3). Although these two patients with incomplete separation of the segments presented in a manner identical with the others, and are believed to have the same pathophysiology as those with complete separation, their period of observation after surgery is still too short to include them in the postoperative evaluation.

We have elected to treat this condition by transduodenal sphincteroplasty (surgical papillotomy) of the accessory papilla. In all cases, the gallbladder was removed, if still present, in order to be absolutely certain that undetected small stones were not at fault. Earlier in our experience, we also performed sphincteroplasties of the major papilla of Vater and duct of Wirsung because there was, at that time, too little experience to rely upon

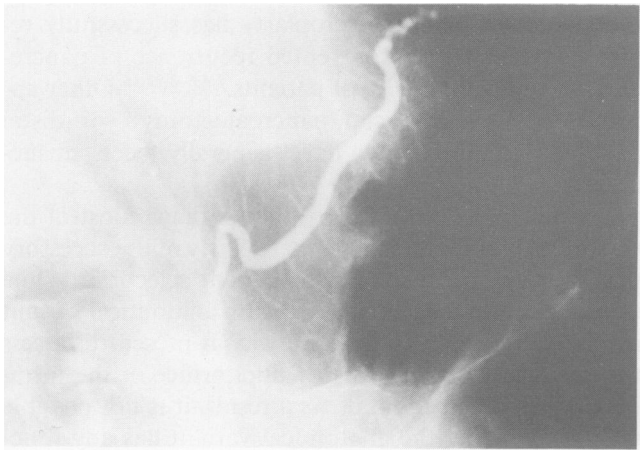

FIG. 2. Pancreatogram obtained through the accessory papilla (same patient as in Fig. 1) showing that the duct of Santorini is the major duct to the tail of the pancreas. It is normal in size and configuration except that it does not communicate with the duct of Wirsung in the pancreatic head.

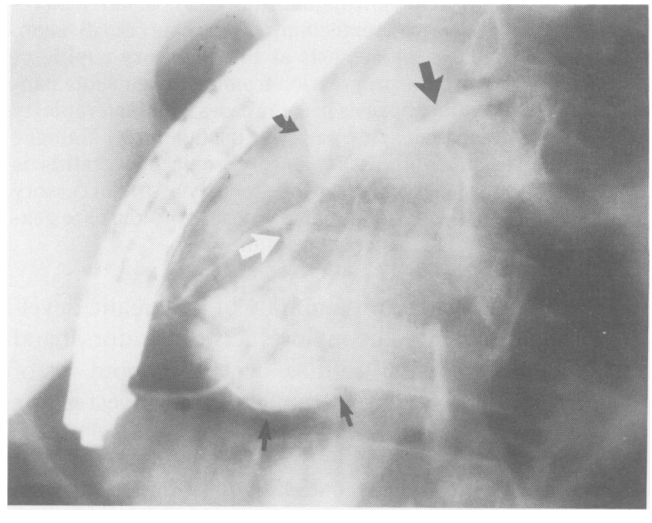

FIG. 3. Pancreatogram via the major papilla and duct of Wirsung. Contrast medium, injected with enough pressure to fill the acini in the pancreatic head (small arrows), opacifies the duct of Santorini (large arrow) through a tiny communication (white arrow). The common bile duct has also been injected (curved arrow).

accessory papillotomy alone.[15] As our confidence in the clinical significance of pancreas divisum has grown, we have abandoned the routine practice of sphincteroplasties of the bile duct and duct of Wirsung in these patients.

The accessory papilla was located by visual identification and palpation at a point approximately 2–4 cm proximal and anterior to the papilla of Vater. Location of the accessory papilla and cannulation of the duct of Santorini was facilitated in a few difficult cases by administration of intravenous secretin (1 u/kg), which caused the mucous membrane covering the papilla to balloon out into hemispheric prominence. The size of the orifice was first evaluated by calibration with fine lacrimal duct probes. It was judged to be stenotic if it was less than 1 mm in diameter (barely admitting a #1 probe = 0.75 mm), and this judgment was entered in the operative records. With a severely stenotic papilla (the majority of those we found to be stenotic) the 0.75 mm probe could not be introduced through the pinpoint papillary orifice. It was necessary to puncture the mucous membrane of the papilla or even to cut down on the papilla by amputating it. The orifice was then widened by extending the papillotomy for at least 1 cm. The pancreatic mucosa was sutured to the duodenal mucosa with interrupted 4-0 chromic cat gut (Fig. 4). A #5 French pediatric feeding tube was threaded into the pancreatic duct to verify that the duct of Santorini extended to the tail and was not obstructed (Fig. 5). It was left in the pancreatic duct, exiting from double purse-string sutures, for postoperative stenting of the sphincteroplasty and drainage of pancreatic secretions.[18] The catheter, which can be used for pancreatography at a later time, was removed after 10 days.

For the purposes of this analysis, a distinction will be made between recurrent acute pancreatitis and chronic pancreatitis. Patients are considered to have recurrent acute pancreatitis if the gland appeared essentially normal or only slightly edematous at operation, if the pancreatogram of both segments was normal except for the division, and if there was no evidence of exocrine or endocrine insufficiency. The pancreatitis is considered

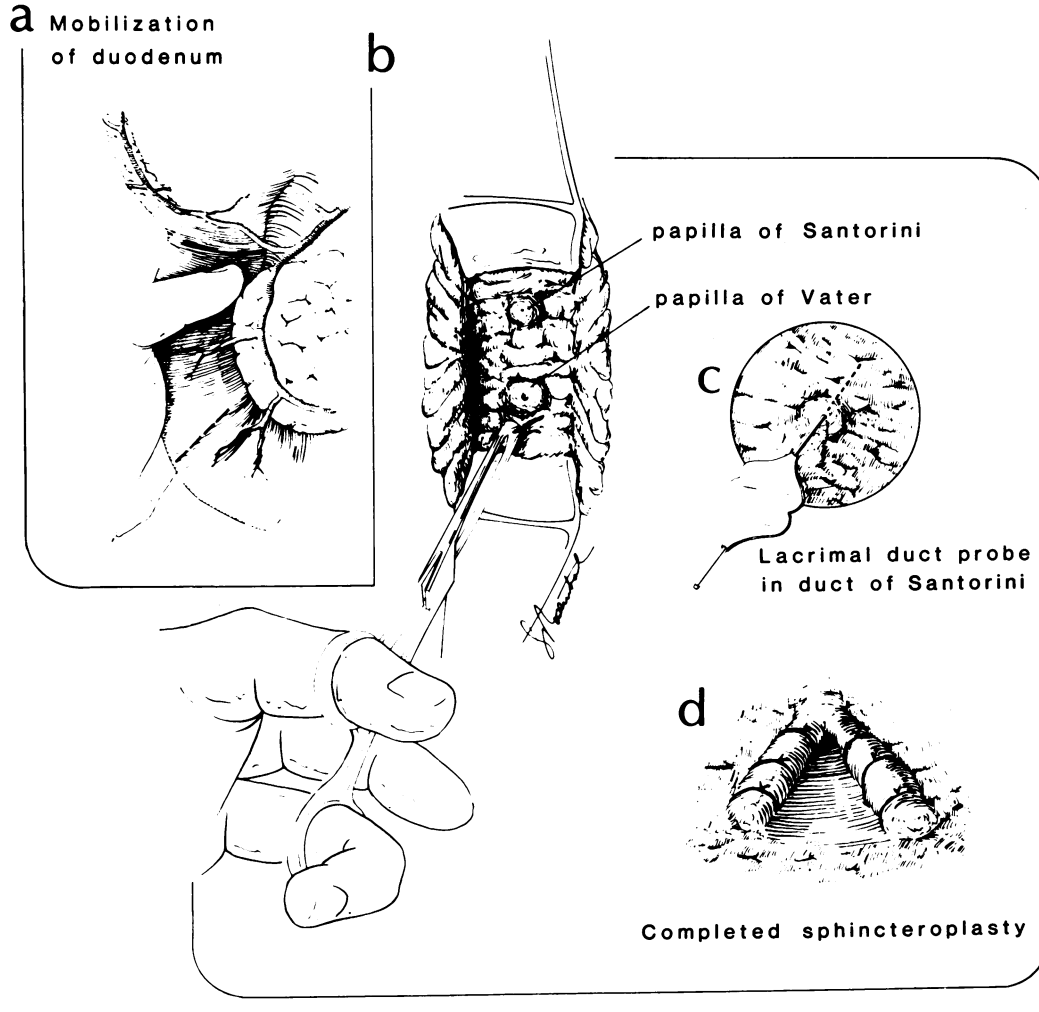

FIG. 4. Technique of surgical sphincteroplasty (papillotomy) of the accessory papilla of the duct of Santorini. (A) The duodenum is opened transversely opposite the major papilla. (B) The minor papilla is 3 cm proximal and slightly anterior to the major papilla. (C) A small probe is inserted into the orifice of the duct, and the papilla is incised using the probe as a guide. (D) The pancreatic duct mucosa is sutured to the duodenal mucosa for hemostasis and to promote healing without stricture.

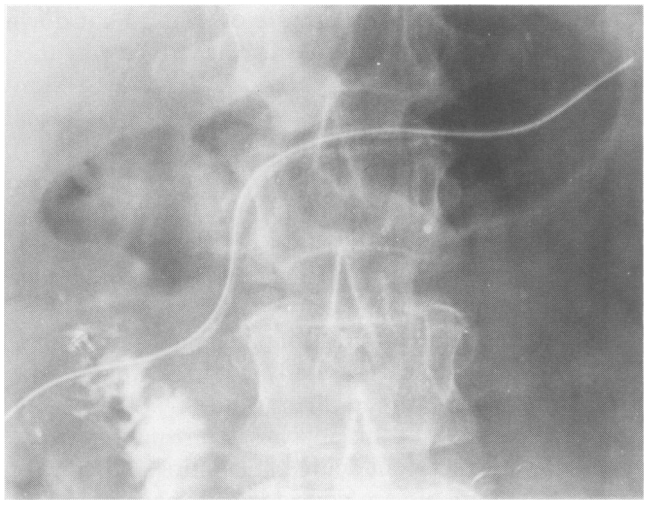

FIG. 5. A #5 radio-opaque catheter has been passed through the accessory papilla out to the tail of the pancreas. This maneuver proves that the duct of Santorini serves the major portion of the gland and has no point of obstruction. The catheter, left in the duct for postoperative drainage, can be used to obtain a pancreatogram later.

chronic if the gland was fibrotic on palpation or histological examination, if pancreatography showed either duct dilatation or constriction and pruning of branches, or if there was deficient function.

Statistical analyses were made by use of the chi-square in 2 × 2 Contingency Table.

Results

Acute pancreatitis. Thirty-two patients met the criteria for proven or possible recurrent acute pancreatitis (Table 1). There were 25 women and seven men with a median age of 30 years (range 16–76). Twenty-two patients had well-defined attacks of pain, consistent with pancreatitis: 18 of these had documented elevations of serum amylase and 10 of them had pancreatic edema shown by ultrasonography or CT scanning during such an attack. The other 10 patients had developed persistent epigastric pain, often radiating to the back and aggravated by eating. None of these 10 had documentation of hyperamylasemia or pancreatic edema. One of the 32 (#28) had gallstones at the time of presentation; gallbladders had been removed previously from nine and were removed during the current operation in the remainder. One of the 32 was known to drink alcohol to excess. No other antecedent risk factors for pancreatitis (drugs, hypercalcemia, hypertriglyceridemia, etc) were identified.

Twenty-nine of the 32 patients have been operated on. One is awaiting operation and two refused it. Of the 29, the pancreas appeared normal in 25 and slightly edematous in 4. Pancreatography of the duct of Santorini, performed via the catheter left through the accessory papilla at operation, showed a normal caliber and configuration of the main duct in all cases. The accessory papilla was judged to be stenotic in 22 of the 29 cases. Sphincteroplasty only of the accessory papilla was performed in 17 patients. In the other 12, sphincteroplasty was also performed on the sphincter of Oddi and the duct of Wirsung. There was one postoperative complication, a leak from a partially divulsed gastrostomy tube, causing a pelvic abscess. No clinically significant postoperative pancreatitis was recognized.

Of the 25 patients who have been followed for at least 6 months after operation, 17 have had a good result (no pain or pancreatitis, or minimal discomfort not requiring medication), one a fair result (much improved but still having mild attacks of pain), and seven have had

TABLE 1. *Clinical Features and Findings in Patients with Pancreas Divisum Associated with Recurrent Acute Pancreatitis*

Patient #	Age Sex	Attacks or chronic pain	Accessory papilla stenosis	Papillotomy S-Santorini W-Wirsung	Follow-up (months)	Results
1	40 F	pain	yes	S & W	103	good
2	20 F	attacks	yes	S & W	68	good
3	16 F	attacks	yes	S & W	67	good
4	55 F	pain	no	S & W	48	poor
5	30 F	pain	no	S & W	44	poor
6	76 F	pain	yes	S	41	good
7	58 F	attacks	yes	S & W	39	fair
8	20 F	attacks	no	S & W	28	poor
9	30 F	attacks	yes	S & W	27	good*
10	62 M	attacks	yes	S & W	26	good
11	35 F	pain	yes	S & W	25	poor
12	16 M	attacks	yes	S	19	good
13	31 F	pain	yes	S	18	good
14	41 M†	attacks	no	S	17	good
15	28 F	attacks	yes	S	15	good
16	29 F	attacks	yes	S	14	good
17	42 M	pain	yes	S & W	14	good
18	31 M	pain	no	S	14	poor
19	34 F	pain	yes	S	13	good
20	30 F	attacks	yes	S	12	good‡
21	24 F	attacks	no	S	11	poor
22	34 M	pain	no	S	11	poor
23	42 F	attacks	yes	S	9	good
24	47 F	attacks	yes	S	9	good
25	28 F	attacks	yes	S & W	7	good
26	23 M	attacks	yes	S	4	
27	29 F	attacks	yes	S	4	
28	20 F§	attacks	yes	S	3	
29	29 F	attacks	yes	S	3	
30	29 F	attacks				
31	32 F	attacks				
32	57 F	attacks				

* Repeat accessory papillotomy at 15 mos for restenosis.

† Alcoholic.

‡ Repeat accessory papillotomy and distal pancreaticojejunostomy at 5 mos for restenosis.

§ Small gallstones found in gallbladder at operation.

TABLE 2. *Clinical Features and Findings in Patients with Pancreas Divisum Associated with Chronic Pancreatitis*

Patient #	Age Sex	Attacks or chronic pain	Accessory papilla stenosis	Main pancreatic duct	Initial operation	Ultimate operation	Follow-up (months)	Results
33	16 M*	attacks	no	dilated	none		28	good
34	9 F	attacks	no	dilated	Puestow†		20	good
35	45 M	attacks	yes	obstructed	TDS (S),‡ Puestow		27	good
36	53 F	pain	yes	dilated	TDS (S & W)	Total pancreatectomy	34	poor
37	33 M	attacks	yes	obstructed	cyst-gastrostomy	TDS (S) & distal pancreatectomy	16	good
38	24 M*	pain	no	constricted	TDS (W) & distal pancreatectomy	TDS (S)	52	poor
39	21 M	pain	yes	constricted	TDS (S & W)	Total pancreatectomy	30	poor
40	28 M*	pain	unknown	dilated	Puestow		40	fair

* Alcoholic.

† Puestow = lateral pancreaticojejunostomy (Roux-en-Y).

‡ TDS = transduodenal sphincteroplasty of accessory papilla (S) ± papilla of Vater & duct of Wirsung (W).

a poor result (no change from preoperative status). There is no significant difference between those patients who had only the accessory papilla opened (10 good, 3 poor) v those who had sphincteroplasty to both the accessory papilla and the major papilla and duct of Wirsung (7 good, 1 fair, 4 poor).

There is a significant ($p < 0.001$) relation between the finding of a stenotic accessory papilla and the ultimate success of the operation. In the 18 patients with stenosis who are now more than 6 months since operation, the results have been good in 16, fair in 1, and poor in 1. In the seven without stenosis, the results have been good in one and poor in six.

The mode of presentation also was of importance. Of the 15 patients who presented with discrete acute attacks of pancreatitis, 12 had good results, one had a fair result, and two did poorly. In contrast, of the 10 patients presenting with persistent chronic pain, five had good results and five had poor results ($p < 0.05$). A patient with a stenotic papilla was slightly more likely than a patient with a nonstenotic papilla to have discrete attacks (17/22 v 3/7). The combination of a stenotic papilla and discrete attacks had a very high likelihood ($p < 0.001$) of successful outcome (11 good, 1 fair, 0 poor). The combination of a nonstenotic papilla and chronic pain had a very poor prognosis (0 good, 4 poor).

The success or failure of the operation was apparent within 3 months of surgery. All patients who had poor results had reverted to their preoperative symptomatic state by that time. All patients who had good results had improved immediately and remained well, except for two who developed proven restenosis of the accessory duct sphincteroplasty (at 15 and 5 months, respectively), had reoperation for repeat sphincteroplasty, and are again symptom free (11 and 8 months after the reoperation).

Chronic Pancreatitis. Eight patients with pancreas divisum proved to have chronic pancreatitis (Table 2). There were six men and two women with a median age of 26 years (range 9 to 53). Four presented with documented recurrent attacks of pancreatitis with asymptomatic intervals. Four had chronic pain with intermittent periods of exacerbation. At least three of these patients were judged to be alcoholics, including a 16-year-old boy who had been drinking heavily for 2 years. None had gallstones, hypercalcemia, hypertriglyceridemia, or other family members with pancreatitis.

Pancreatograms demonstrated deformities of chronic pancreatitis in the duct of Santorini in all eight patients (dilated main ducts in four, constricted and obliterated ducts in two, obstructed main ducts in the body of the pancreas in two, with distal dilatation in one, and obliteration in the other). Endoscopic cannulation of the duct of Santorini for pancreatography was accomplished only in the two children. The remaining pancreatograms were obtained at or after operation. Histological confirmation of chronic pancreatic inflammation and fibrosis was obtained at operation in all seven patients treated surgically. One patient (#36) was diabetic at presentation, and two (#33 and 36) had evidence of steatorrhea.

Four of these patients were judged at operation to have stenosis of the accessory papilla. Three others were judged not to be stenotic; two had easy endoscopic cannulation of the accessory papilla with deep penetration by the cannula and free-filling of contrast material (#33 and 34); one (#38) had a papillary orifice which easily admitted a 5F. catheter at operation. The orifice of patient #40 was not examined.

Transduodenal sphincteroplasty of the accessory papilla was performed in five patients (accessory papilla alone in #35 and 37; both the accessory papilla and the major papilla and duct of Wirsung in #36, 38, and 39). When performed as the sole initial maneuver, it failed in two patients, both of whom were later treated by total pancreatectomy for unremitting pain; both continued to have pain. In a third patient, transduodenal sphinc-

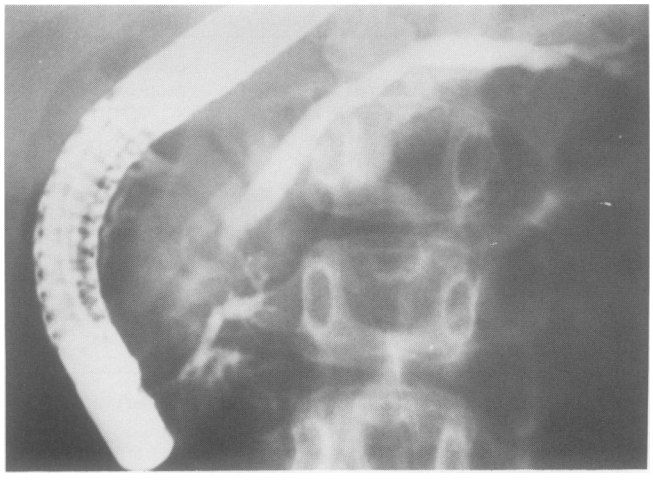

FIG. 6. Endoscopic pancreatogram in a 9-year-old girl with chronic pancreatitis. The duct of Santorini and Wirsung have each been injected with contrast medium separately and do not communicate with each other. Note the diffuse dilatation of the ducts.

teroplasty of the accessory papilla was combined with pancreaticojejunostomy to the obstructed distal segment of pancreas with excellent results. Two patients had transduodenal accessory sphincteroplasty as second operations. One, an alcoholic, was treated first by distal pancreatectomy and sphincteroplasty of the major papilla and duct of Wirsung without benefit. He had temporary improvement after the second operation at which the accessory papilla was opened, but he has continued to drink and has been hospitalized repeatedly for pain. The fifth patient had drainage of a pseudocyst which developed after an attack of acute pancreatitis. Six months later, he had accessory papillotomy and resection of the obstructed pancreatic tail because of recurrent attacks of pancreatitis, and subsequently, has been asymptomatic.

Two patients who presented with diffuse dilatation of the main pancreatic duct (Fig. 6) were treated primarily by the modified Puestow procedure (side-to-side or lateral pancreaticojejunostomy). One patient has done well; the other takes one to two Percodan tablets per day, but works regularly and has not been rehospitalized.

Thus, of four patients who are currently well, none has had only accessory papillotomy. Papillotomy in this group of patients has been associated with good results only when combined with pancreatic resection or distal drainage.

Discussion

Pancreas divisum occurs in at least 3 to 7% of the population.[4,10,12–14,17] Most of the millions of people with pancreas divisum have no related symptoms or disease; the anomaly is of no clinical importance in them. Nonetheless, the weight of the epidemiological evidence strongly suggests that some individuals with pancreas divisum are more likely to develop pancreatitis than people with fused pancreatic ducts.[4,12,14,17] It has been hypothesized by several authors[2,5,8,12] that the problem might be a relative stenosis at the accessory papilla, which in affected persons is the sole outflow tract for most of the pancreatic secretions (up to 2,000 ml/day). The findings of this study provide strong support for that view. Impressive pin-point stenosis (0.75 mm or less) was indeed demonstrable in a high proportion (22/29) of our patients with acute pancreatitis and pancreas divisum. The size of the orifice of the accessory papilla in this subgroup was smaller than we have ever seen at the duct of Wirsung in patients with benign ampullary stenosis. More important, relief of that stenosis by accessory papillotomy was highly effective treatment in 17 out of 18 patients; but, predictably and by way of further confirmation, the operation was ineffective in relieving symptoms in 6 of 7 cases when the papilla was not stenotic. Even the recurrence of symptoms in two patients with restenosis of the accessory papilla tends to support the causality of the papillary stenosis in these patients.

It is especially noteworthy that this is the first sizeable experience in which sphincteroplasty of the accessory papilla alone,[8] without complementary sphincteroplasty of the major papilla of Oddi and duct of Wirsung, has been found to be effective treatment for pancreatitis associated with pancreas divisum. We believe that accessory papillotomy should be the recommended treatment for recurrent pancreatitis due to pancreas divisum.

The duct of Santorini and its outflow through the accessory papilla often become atretic and disappear after fusion of the dorsal and ventral pancreatic segments.[10] Perhaps the stenosis occurring in some persons with pancreas divisum is a consequence of the beginnings of that process of atresia, necessarily halted because the usual alternative outflow tract of the duct of Wirsung has not developed. It would be reasonable to expect, if such were the case, a spectrum of severity of stenosis from none to severe, and, therefore, a spectrum of severity of consequent damage to the pancreas. Relatively severe stenosis might present earlier in life with duct dilatation and secondary chronic damage to the pancreas, while milder degrees of stenosis might present later with lesser, more reversible injuries.

Several cases presented in this series and in others,[6,14,17] suggest that the effects of the pancreas divisum can lead from recurrent acute pancreatitis to chronic fibrotic pancreatitis. If such a progression does occur in this disease, it may be the only solid example of true acute pancre-

atitis evolving to true chronic pancreatitis. Biliary pancreatitis has not been shown to become chronic (except as a consequence of major scarring and duct destruction due to necrotizing pancreatitis), and alcoholic pancreatitis is widely believed to be chronic at the time of its first acute manifestations. However, that inference should be drawn with caution. It is possible that the pancreas divisum is coincidental rather than causative in some of our patients with chronic pancreatitis, especially since three of the eight were alcoholics, three of the eight had no demonstrable stenosis, and the sex ratio was reversed in comparison with the acute pancreatitis group. There are no epidemiologic studies of chronic pancreatitis and pancreas divisum comparable to those of acute pancreatitis.

Although transduodenal sphincteroplasty was highly effective in treating recurrent pancreatitis when the gland was not fibrotic, that operation was clearly inadequate for patients with this form of chronic pancreatitis. As is the case with chronic pancreatitis in general, surgical treatment should be tailored to fit the type of pancreatic duct abnormality. If there is a specific point of obstruction along the duct, the gland distal to the point of obstruction must be resected or, if the obstructed duct has dilated, decompressed by anastomosis to jejunum. If the main pancreatic duct is dilated throughout its length, a Puestow pancreaticojejunostomy[20] will be appropriate, despite the fact that only the dorsal duct system receives the benefit of the decompression in someone with pancreas divisum. Total pancreatectomy may be considered, in extreme cases, unmanageable by medical means when the pancreatic duct is constricted by fibrosis rather than dilated. If acute pancreatitis does evolve to chronic pancreatitis in patients with pancreas divisum, there would seem to be a great advantage to operating early in the disease, while sphincteroplasty is likely to be effective, and before major resections become necessary.

The recognition of pancreas divisum is usually easy to infer from the endoscopic pancreatogram, which shows the foreshortened duct of Wirsung (<6 cm), tapered at its distal end, and failing to extend out to the pancreatic body and tail. The major *caveat* is that pancreas divisum must be distinguished from a block of Wirsung's duct due to inflammatory injury[17,19] or tumor. Final confirmation of the proper diagnosis requires radiographic demonstration of the major pancreatic duct, continuous with the duct of the duct of Santorini or passage of a catheter through the accessory papilla out to the pancreatic tail.

Unfortunately, endoscopic cannulation of the accessory papilla is difficult even when the papilla is not stenotic, and likely to be impossible when it is stenotic. The accessory papilla was successfully cannulated for pancreatography of the duct of Santorini in only 5 of our 40 patients, a failure rate that closely matches that of other experienced endoscopists.[4,5,13,16,17] It is hard to avoid the conclusions that: (1) failure to cannulate the accessory papilla does not confirm either the presence of pancreas divisum or stenosis of the papilla; (2) successful cannulation of the papilla suggests that there is not significant stenosis and, therefore, that pancreas divisum is probably not the cause of the symptoms being investigated; and, (3) endoscopic papillotomy by electrosurgical cutting wire or by balloon dilatation of the accessory papilla is least likely to be possible when the papilla is truly stenotic and is unnecessary when it is not. Although Cotton has reported two such cases,[3] we have not yet been able to succeed in inserting an endoscopic papillotomy wire into accessory papilla in a patient with symptomatic pancreas divisum. The only case we have seen treated by endoscopic balloon dilatation of the accessory papilla was not stenotic to begin with and developed protracted acute pancreatitis after the procedure.

The mediocre results of sphincteroplasty of the accessory papilla for pancreatitis associated with pancreas divisum[6,16] have been discouraging to some commentators and have caused them to question whether the association is valid and the treatment justifiable.[1,11,16] It is our view to the contrary that the problem lies not in whether the diagnosis exists or how it should be operated upon, but in properly selecting those patients whose pancreas divisum should be treated. Our findings indicate that if the accessory papilla of a patient with recurrent pancreatitis and pancreas divisum is stenotic, relief of pancreatic duct obstruction by accessory papillotomy is highly likely to succeed in ameliorating the recurrent pancreatitis and pancreatic pain. At present, unfortunately, we can only make the determination of stenosis at the operating table.

Even with the most carefully applied clinical criteria for selecting patients for surgery, (such as nonalcoholic young women presenting with documented discrete attacks of pancreatitis), we will continue to include some patients who have no stenosis and who will not be helped by papillotomy, and certainly to exclude some patients who might benefit from the operation. A preoperative test for stenosis of the accessory papilla is very much needed to identify patients whose symptoms are genuinely caused by their pancreas divisum. We are currently investigating the use of ultrasonography to see if the pancreatic duct dilates behind the stenosis when the pancreas is stimulated; our experience thus far with more than 35 patients is quite promising and will be reported elsewhere.

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Discussion

Dr. Charles F. Frey (Sacramento, California): Dr. Warshaw, you postulate that the pain in these patients results from a relative imbalance of draining a large portion of the gland through a small duct which has become strictured; yet many of these patients are in their forties and fifties. Why have they not had symptoms earlier? If their pain is due to a late development of a stricture, what is it that is causing the stricture in these patients at this time in their life?

You further postulate, if left untreated, these patients will go on to develop chronic pancreatitis. If this is so, why did one half of your patients in the group with chronic pancreatitis not have an ampullary stricture?

The second question is: Why are your results so outstanding in this group of patients—in fact, better than those reported with any other operation for chronic pancreatitis? Perhaps this group of patients did not have pancreatitis. And the third question is: Pancreatitis is a disease of exacerbations and remissions. How long do you feel these patients should be followed before you conclude treatment is a success?

Dr. William V. McDermott, Jr. (Boston, Massachusetts): This is not a newly-recognized anomaly, certainly. Opie was the first to describe this in 1903, although he is much better known for his famous case of the impacted stone, which he reported with Halsted and which led to the long-standing but eventually moribund concept of the common channel theory of pancreatitis.

Interestingly enough, this did appear in the surgical literature in an article by Dr. Rienhoff in 1945. Other than that, all references to this have been rather abstruse comments from anatomical studies in nonsurgical, nonclinical journals.

The recent resurgence of interest in this anomaly dates from the introduction of endoscopy, when it was possible for the first time to correlate clinical findings—or, at least, attempt to correlate clinical findings with the existence of this anomaly through endoscopic pancreatography. And with the reports of Gregg in this country and Cotton in England in 1977, the floodgates were opened, and, obviously, considerable interest has developed in this presumed syndrome. Dr. Warshaw had a previous report with Richter and his colleagues in 1981 and Dr. Carey of this society also reported, with Cooperman and colleagues, in 1982, on a small operative series.

Last year, we gave reports of our observations on Pancreas Divisum before the New England Surgical Society, and I shall touch briefly on these in this discussion.

As Dr. Warshaw said, the incidence of this anomaly ranges somewhere, by studies available, between 4 and 7%; yet, clearly, there are not that many people walking around with clinical syndromes.

(Slide) The operation we have used in a surgical approach to this problem has not been limited only to the lesser sphincter, but has involved sphincteroplasties of the major and minor papillae, mainly because we were never certain as to what the disease was, and which of the two separate ductal systems it involved.

The total number in our series was 19. (slide) These were selected out of a series of 70 patients who had the association of recurrent epigastric pain and the anomaly of pancreas divisum, and were recommended for surgery because of the severity and intractability of their symptoms. This group comprised mostly women, as seems to have been true in other series; the patients were in the younger age group, the oldest being in the 40s, although the onset of symptoms in all began before the age of 40, at a median age of 26.

I call your attention to the fact that in our series only seven of the cases had any chemical, microscopic, radiological or morphological findings at operation to suggest concurrent pancreatitis, a finding which others have noted as well.

(Slide) The results are somewhat equivocal. Of the 18 patients available for follow-up, one had excellent initial results, although there were four cases in whom some recurrence of symptoms developed in the weeks or months ensuing. This left only 11 patients with good long-term results.

Of the seven patients who had persistent or recurrent problems, further operation were carried out in six, involving a variety of procedures—resection and distal drainage, further papillotomy or 90% resection, with some improvement in 3 cases.

Thus, the results of surgery have not been conclusive to us. I personally have been unable to tell whether this minor sphincter is stenotic, or is just tiny, which it is. It is very difficult to say whether, in the absence of objective findings, there is a definite syndrome of pain associated with relative stenosis, leading to some dilatation of the duct. We have only seen one case of ectasia of the duct, and one other with slight dilatation.

I would leave the audience with this caution and question: Is this a true entity, or chance association? Objective evidence of disease or of clinical pancreatitis associated with the anomaly is sparse.