Surgical Sphincteroplasty in 446 Patients

James A. Madura, MD; James A. Madura II, MD; Stuart Sherman, MD; Glen A. Lehman, MD

**Hypothesis:** Pancreaticobiliary sphincter disease is reliably diagnosed by endoscopic and intraoperative manometry.

**Design:** Retrospective review of prospectively collected data.

**Setting:** A 400-bed urban university hospital.

**Patients:** Between May 1, 1978, and March 27, 2002, 446 patients were treated surgically for dysfunction of the pancreaticobiliary sphincters. There were 376 females and 70 males (mean±SD age, 41.6±12.5 years). There were 372 patients with sphincter of Oddi dysfunction, and 74 with pancreas divisum. Symptoms included abdominal pain (100.0%), nausea/vomiting (80.5%), back pain (57.2%), and pancreatitis (22.4%).

**Interventions:** Perfusion manometry has evolved as the gold standard for diagnosis, and intraoperative manometry was done in 214 patients. All patients underwent transduodenal sphincteroplasty and biopsies of the ampullae and transampullar septa.

**Results:** Excellent or good results were seen in 86.8% of the patients with sphincter of Oddi dysfunction and in 63.5% of the patients with pancreas divisum. Common duct and sphincter of Oddi pressures were 0 mm Hg in all patients after sphincteroplasty. Pancreatic duct and pancreatic sphincter manometry results were improved in 82.4% of the patients. Biopsy results of the main and accessory sphincters demonstrated inflammation and/or fibrosis in 33.9% of ampullae and 43.5% of transampullar septa, but this did not correlate with outcome. There was 1 death from a duodenal leak. Complications occurred in 34.8% of patients, with pancreatitis (8.8%), asymptomatic hyperamylasemia (6.0%), and wound/abdominal infection (7.1%) the most common. Predictive factors for good outcome were reduction in pancreatic duct and sphincter pressures following sphincteroplasty.

**Conclusion:** Good to excellent results may be achieved by surgical sphincteroplasty when careful patient selection by manometry is used.

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of these disorders. As technology developed, hypertensive and fibrotic pancreaticobiliary sphincters were not only identified but were treated therapeutically by endoscopists.

This report details the surgical approach to pancreatic and biliary sphincters during the past 2 decades of the 20th century, and presents an experience with many patients studied and treated by a team of gastroenterologists and a single surgeon (James A. Mudura).

## METHODS

### PATIENTS

Between May 1, 1978, and March 27, 2002, 446 patients underwent transduodenal sphincteroplasty for stenosis or other abnormalities of the pancreaticobiliary sphincters. There were 376 females and 70 males (mean ± SD age, 41.6 ± 12.5 years; range, 7-74 years). Of the 372 patients diagnosed as having sphincter of Oddi dysfunction (SOD), 100 patients who had previously undergone sphincterotomy or sphincteroplasty.

The patients' symptoms had been present for an average of 35 months, and included abdominal pain in 100.0%, nausea and/or vomiting in 80.5%, referred back pain in 57.2%, diarrhea in 25.8%, and documented pancreatitis in 22.4%. Other reported symptoms included weight loss in 17.7% and fatty food intolerance in 23.8%. Prior operations included cholecystectomy in 71.1%, gynecologic operations in 47.5%, and appendectomy in 41.3%. Of all patients, 81.6% underwent preoperative ERCP, but were inconsistent and frequently nondiagnostic. Transendoscopic pancreaticobiliary manometry eventually became the accepted diagnostic methods. As technology developed, hypertensive and fibrotic pancreaticobiliary sphincters were not only identified but were treated therapeutically by endoscopists.

In addition, most patients underwent numerous investigative studies preoperatively to rule out other intra-abdominal disease as a source for their symptoms.

Initially in this group of patients, the morphine-neostigmine provocative test, as described by Nardi and Acosta, was done in 290 patients. Seventy-one patients consented to a postoperative test. Intraoperative evaluation of the sphincter of Oddi was done using a modified Caroli apparatus, as described by White et al,3 of the sphincter of Oddi ablation either endoscopically (n = 73) or surgically (n = 27). In the 74 patients with pancreas divisum (PD), 33 underwent previous sphincterotomy or sphincteroplasty.

The modified Caroli apparatus described by White et al3 was attached via sterile intravenous tubing to a Silastic ventricular catheter carefully positioned in the supra-ampullary common duct. Two 1-minute measurements of the rate of flow of isotonic sodium chloride solution through the common bile duct were obtained, followed by measurement of the height of the column of isotonic sodium chloride solution remaining in the tubing. Normal flow was accepted as a mean ± SD of 23 ± 7 mL/min of isotonic sodium chloride solution, and normal pressure as a mean ± SD of 11 ± 4 mL of isotonic sodium chloride solution.

Low-flow perfusion capillary manometry was done intraoperatively with equipment similar to that used in endoscopic biliopancreatic manometry. A triple-lumen side perfusion catheter was inserted into the biliary and pancreatic ducts and slowly withdrawn. It was attached to a low-pressure capillary perfusion device (Arndorfer Medical, Greenvale, Wis) that was connected to an 8-channel data recorder (model MMS 200; Narco Biosystems, Austin, Tex). Serial pressures were recorded in the biliary and pancreatic ducts and in their respective sphincters. Results were compared with published standard basal pressures. The accepted norm for the diagnosis of SOD is a mean basal pressure higher than 40 mm Hg in the sphincter of Oddi and the pancreatic sphincter. A pancreatic duct mean basal pressure of 24 mm Hg was accepted as the upper limit of normal in these patients.

Cholecystectomy was performed in all patients who had not undergone it previously. Transduodenal sphincteroplasty was performed in a standard fashion through a longitudinal duodenal incision centered over the major ampulla. The pancreatic orifice was identified and intubated with a metallic probe to prevent pancreatic ductal occlusion during the biliary sphincteroplasty. The choledochal and duodenal mucosa were approximated with fine absorbable sutures. Pancreatic duct and sphincter manometry was then performed, and data were recorded. If the initial pancreatic duct pressure was lower than 24 mm Hg, pancreatic duct sphincteroplasty was not done, which occurred in 11 patients. Next, the pancreatic ductal orifice was opened and its edges were sewn to the choledochal mucosa with synthetic nonabsorbable sutures. Finally, pancreatic duct and pancreatic sphincter zone pressure measurements were obtained again to ensure that these pressures had been appropriately reduced to normal.

In patients with PD, a “dual sphincteroplasty” was done. Following cholecystectomy, the major ampulla was opened and a biliary sphincteroplasty was done. The duct of Wirsung was not routinely opened because it is usually rudimentary in patients with PD. Next, the accessory ampulla was identified and intubated with a lacrimal duct probe. If identification was difficult, synthetic secretin was administered intravenously. Pressures were recorded if the manometry catheter was able to be freely inserted without impacting in the duct. The accessory ampulla was opened in a medial and cephalad direction, and the cut edges of the duct were approximated to the duodenal mucosa with synthetic nonabsorbable sutures.

Biopsy specimens of the ampullary sphincter, pancreatic duct sphincter (transampullar septum), and accessory sphincters (in the case of PD) were routinely obtained and submitted for histopathologic examination, and the results of the biopsies were reported as normal or abnormal with fibrosis and/or inflammation.

## PROCEDURES

The morphine-neostigmine test was performed in the manner described by Nardi and Acosta.3 Patients received an injection of 10 mg of morphine and 1 mg of neostigmine; venous blood samples were obtained before the injection and at 30-minute intervals thereafter for 4 hours. The samples were analyzed for amylase, lipase, aspartate aminotransferase, and alanine aminotransferase levels. The patients were also monitored for symptoms of typical upper abdominal pain, nausea, and/or vomiting. A result was considered positive if the enzyme levels increased by greater than 4 times the baseline values.

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Biopsy specimens of the ampullary sphincter, pancreatic duct sphincter (transampullar septum), and accessory sphincters (in the case of PD) were routinely obtained and submitted for histopathologic examination, and the results of the biopsies were reported as normal or abnormal with fibrosis and/or inflammation.
All patients experienced drainage of the right upper quadrant with a Penrose drain, which was removed when the patient resumed oral nutrition without evidence of an enteric or biliary leak.

Collected data were compared where appropriate statistically by $t$ test, $\chi^2$ analysis, or regression analysis.

### RESULTS

There was a single death in the entire group of 446 patients (0.2%). This occurred early in the series, following a duodenal leak, sepsis, and multisystem organ failure. Complications were seen in 34.8% of the patients, the most serious being pancreatitis, duodenal or bile leak, wound infection, and abdominal abscess, which occurred in 20.8%. Other less serious complications, such as urinary tract infection, atelectasis, deep venous thrombosis, asymptomatic hyperamylasemia, and prolonged ileus, occurred in 14.0% of the patients (Table 1).

Long-term outcome was considered excellent to good in 82.9% of all patients, and fair to poor in the remaining 17.1%. Of the patients with the usual anatomical features, 86.8% had an excellent or good outcome, while 63.5% of those with PD, either initially or at reoperation, achieved an excellent to good outcome. Patients who had not undergone previous sphincteric surgery, either surgical or transendoscopic, tended to do better, but not statistically so (Table 2). No restenosis of the major ampulla was seen during this experience, but restenosis of the pancreatic duct or accessory papilla was an occasional problem requiring either repeat ERCP with endoscopic sphincterotomy and stenting or reoperation. In the 100 patients who underwent repeat sphincteroplasty, 73 had previously undergone 1 or more transendoscopic sphincterotomies, while the other 27 had been treated surgically.

### EVOCATIVE TEST

Morphine-neostigmine evocative tests were done in 290 patients preoperatively and 71 patients postoperatively. Postinjection symptoms occurred in 91.6% of the patients tested, but only 46.7% had serum lipase levels elevated greater than 4-fold over the baseline value. Mean enzyme variables (amylase, lipase, aspartate aminotransferase, and alanine aminotransferase levels) were significantly reduced postoperatively when compared by the $t$ test ($P < .001$).

### CAROLI MANOMETRY AND DEBIMETRY

Caroli pressures were more than 15 cm of isotonic sodium chloride solution in 47.2% of the patients, while...
the corresponding 1-minute flows of isotonic sodium chloride solution were less than 15 mL/min in 19.0% of the patients. In the reoperative patients, however, all flows were in the normal range, suggesting that the previous surgical sphincteroplasty or endoscopic sphincterotomy had left a widely patent sphincter of Oddi. Neither pressure nor flow correlated well with a better result.

**PRESSURE RESULTS**

All patients undergoing major ampullary sphincteroplasty had manometric pressures reduced to 0 mm Hg (Figure). The pancreatic sphincter and ductal pressures were significantly reduced in 82.4% of the patients (Table 3). In 25 patients with small pancreatic ducts, post sphincterotomy pressures were actually increased by impaction of all 3 orifices of the triple-lumen pressure catheters, despite the orifices being located in different radial positions on the catheter tip. Anatomically, this was thought to occur at the pancreatic ductal genu, where the junction of the ventral and dorsal ducts occurred.

**PATHOLOGICAL RESULTS**

In 55.8% of the patients, the final pathological finding of the major ampullary sphincter was reported as negative for fibrosis and/or inflammation. In the transampullar septum, 56.6% of the specimens were observed to be normal. In patients with PD, 58.5% of accessory ampullary biopsy results were reported as normal (Table 4). An explanation for this many normal biopsy results might be sampling error, although that would be unusual in many cases done in the same way by a single surgeon. A more plausible explanation might be that not all of the patients had a fibrotic and stenotic sphincter, despite elevated pressure, but perhaps a hyperplastic or hypersensitive sphincter was the cause. In patients undergoing cholecystectomy as part of the procedure, chronic cholecystitis was seen in 78.3% of removed gallbladders. Interestingly, in patients with PD, 80.9% of the 22 excised gallbladders demonstrated evidence of chronic inflammation.

Abnormal histopathologic features did not correlate statistically with clinical outcome, suggesting that patients with negative histopathologic features did as well as the others because a fibrotic or inflamed sphincter was not necessarily the only cause for symptoms in these patients.

One patient undergoing sphincteroplasty during this time, but not included in this series, had a previously unrecognized small adenocarcinoma of the ampulla on the postoperative pathology report. He underwent pancreatoduodenectomy and has survived for 17 years postoperatively.

**COMMENT**

The cause of pancreaticobiliary sphincter dysfunction can be divided into several categories. The sphincter of the major ampulla is most frequently injured by the passage of gallstones from the gallbladder through the common bile duct and the ampulla of Vater. The impaction of stones in this ampulla may also cause inflammation and scarring in the orifice of the main pancreatic duct and its sphincter, resulting in increased pancreatic ductal pressure to overcome the obstruction caused by the inflammation and fibrosis. Alternatively, some sphincters might be hypersensitive to various noxious stimuli, such as alcohol, other drugs, or a hormonal secretion, resulting in elevated ampullary pressures, but without the characteristic fibrotic and inflamed histopathologic picture seen following the passage of stones. Only a few investiga-

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**Figure.** Mean basal pressure measured intraoperatively before and after sphincteroplasty in 144 patients with dysfunction of the sphincter of Oddi. P = .001 for the difference between all variables before and after sphincteroplasty. CBD indicates common bile duct; PD, pancreas divisum.

**Table 3. Intraoperative Manometric Pressures in the Pancreatic and Biliary Ducts and Sphincters in Patients Undergoing Transduodenal Sphincteroplasty**

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of Patients</th>
<th>Before Sphincteroplasty</th>
<th>After Sphincteroplasty</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common duct</td>
<td>144</td>
<td>9.9 ± 9.5</td>
<td>0</td>
</tr>
<tr>
<td>Ampulla</td>
<td>139</td>
<td>30.9 ± 29.4</td>
<td>0</td>
</tr>
<tr>
<td>Pancreatic duct</td>
<td>143</td>
<td>30.1 ± 28.2</td>
<td>17.7 ± 13.5</td>
</tr>
<tr>
<td>Pancreatic sphincter</td>
<td>141</td>
<td>55.3 ± 33.3</td>
<td>25.9 ± 32.3</td>
</tr>
</tbody>
</table>

**Table 4.** Intraoperative Manometric Pressures in the Pancreatic and Biliary Ducts and Sphincters in Patients Undergoing Sphincteroplasty

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of Patients</th>
<th>Before Sphincteroplasty</th>
<th>After Sphincteroplasty</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common duct</td>
<td>45</td>
<td>7.0 ± 6.7</td>
<td>0</td>
</tr>
<tr>
<td>Ampulla</td>
<td>43</td>
<td>18.4 ± 25.6</td>
<td>0</td>
</tr>
<tr>
<td>Pancreatic duct</td>
<td>49</td>
<td>45.2 ± 48.4</td>
<td>23.7 ± 17.0</td>
</tr>
<tr>
<td>Pancreatic sphincter</td>
<td>40</td>
<td>61.4 ± 44.4</td>
<td>31.0 ± 29.5</td>
</tr>
</tbody>
</table>

Abbreviation: SOD, sphincter of Oddi dysfunction. *P < .001, by t test, for all differences in pressure before and after sphincteroplasty.
tors have performed a biopsy of the ampulla in these patients, and have observed not only fibrosis and inflammation but also muscular hypertrophy in many of the studied specimens.\(^5\) -\(^7\) Several of these series reported many specimens with a normal histopathologic appearance, as was seen in the present group of patients.

In the 7% to 10% of patients who have PD with dorsal duct prominence and symptoms of pancreatitis, the orifice of the accessory duct may be congenitally insufficient to allow passage of the increasing amounts of pancreatic secretion seen as the individual grows and pancreas function increases proportionally. There certainly is a different mechanism than is seen in the major papilla, where gallstone transit accounts for much of the dysfunction. A small and tight minor papillary orifice is frequently observed when the accessory duct in a dominantly system can barely be located and intubation of its orifice can only be accomplished with the finest of lacrimal duct probes or the adjunctive use of secretin, despite a dilated dorsal duct. The reported results of surgical treatment for PD are not quite as favorable as for SOD, because the problem is congenital and may take longer to cause recurrent and undetected bouts of pancreatitis.

Endoscopic retrograde cholangiopancreatography and subsequent transendoscopic manometry studies have demonstrated hypertension in the biliary and pancreatic sphincters.\(^8\) Skilled endoscopists routinely perform such evaluations and are able to reliably apply these methods to diagnose conditions in patients with postcholecystectomy symptoms, unexplained upper abdominal pain, and idiopathic pancreatitis.

Early surgical approaches to the sphincter to remove stones impacted in the distal common duct were reported more than 100 years ago. In the mid 20th century, with the increasingly frequent removal of the gall-bladder, postcholecystectomy surgical symptoms similar to the original biliary pain occurred in 10% to 20% of patients and renewed interest in the pancreaticobiliary sphincters, initiating a search for accurate methods to diagnose and treat such symptoms. In the pre-ERCP era, no reliable techniques were available preoperatively to provide a reasonable certainty that these sphincters were indeed the source of the symptoms and, therefore, suggestions that potentially risky surgical procedures be done were met with much skepticism in the surgical and medical communities. During this era, surgeons examined symptomatic patients intraoperatively by palpation of the sphincter through the duodenal wall or by the inability to pass a 3-mm dilator through the ampulla into the duodenum. Others relied on the cholangiographic appearance of a tapered choledochal-duodenal junction or on a dilated common bile duct. Because these were largely subjective measures, the surgical approach to the sphincter of Oddi was not widely accepted, despite good results reported by respected researchers.\(^9\) -\(^13\) The evolution of sphincter ablation from sphincterotomy to sphincteroplasty to biliopancreatic sphincteroplasty seemed to result in better outcomes, yet a definitive preoperative selection tool remained elusive\(^14\) -\(^15\) (Table 5).

The morphine-neostigmine evocative test was an early attempt to select such patients, but was subsequently proved too nonspecific by several investigators.\(^16\) -\(^17\) Nevertheless, several recent reports suggest using this evocative test as a screening tool in patients with typical postcholecystectomy symptoms. Caroli\(^7\) and White et al\(^1\) published their efforts to define abnormal sphincteric function (radiomanometry) and select patients for sur-

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**Table 4. Histopathologic Findings of the Biliopancreatic Ampullae and Sphincters in Patients Undergoing Sphincteroplasty**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Biopsies/No. of Patients</th>
<th>Histopathologic Findings*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Ampulla of Vater</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Those with SOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial procedure</td>
<td>255/272</td>
<td>155 (60.8)</td>
</tr>
<tr>
<td>Reoperation</td>
<td>87/100</td>
<td>50 (57.5)</td>
</tr>
<tr>
<td>Those with pancreas divisum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial procedure</td>
<td>40/41</td>
<td>29 (72.5)</td>
</tr>
<tr>
<td>Reoperation</td>
<td>18/33</td>
<td>15 (83.3)</td>
</tr>
<tr>
<td><strong>Transampullary Septum</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Those with SOD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial procedure</td>
<td>234/272</td>
<td>135 (57.7)</td>
</tr>
<tr>
<td>Reoperation</td>
<td>84/100</td>
<td>34 (40.5)</td>
</tr>
<tr>
<td>Those with pancreas divisum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial procedure</td>
<td>36/41</td>
<td>27 (75.0)</td>
</tr>
<tr>
<td>Reoperation</td>
<td>12/33</td>
<td>11 (91.7)</td>
</tr>
<tr>
<td><strong>Accessory Ampulla</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Those with pancreas divisum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial procedure</td>
<td>29/41</td>
<td>19 (65.5)</td>
</tr>
<tr>
<td>Reoperation</td>
<td>24/33</td>
<td>12 (50.0)</td>
</tr>
</tbody>
</table>

Abbreviation: SOD, sphincter of Oddi dysfunction.

*Data are given as number (percentage) of each group. Percentages may not total 100 because of rounding. Denominators used were the number of biopsies for each group.
gical sphincterotomy. Other investigators using these techniques duplicated these studies, but acknowledged that these measurements did not really address the pancreatic sphincter and duct. However, it was not until endoscopic retrograde access to the pancreaticobiliary system was available in humans that the diagnosis of stenotic or hypertensive sphincteric diseases began to be accepted. Classen and Demling and Kawai et al are credited with independently describing endoscopic sphincterotomy in 1974, but the technique was primarily used to extract retained common duct stones. Low-pressure perfusion catheters and recording devices were used to identify patients with symptoms of pancreaticobiliary disease, who had findings of dysfunction that could be addressed. Hogan and Geenen created a classification system and demonstrated that patients with symptoms of partial obstruction to biliary and pancreatic flow who had elevated sphincteric pressures obtained significant relief from sphincterotomy, when compared with patients who had symptoms but no objective findings. During the past decade, increasing experience has led skilled endoscopists at major referral centers to perform more transendoscopic sphincterotomies for SOD, and their results are similar to those of surgical transduodenal sphincteroplasty (Table 6).

The prevalence of sphincteric dysfunction in the general population is not really known because most reports are from a few centers specializing in the diagnosis and management of pancreaticobiliary disease. It is reported that more than 10,000 endoscopic sphincterotomies have been done, with more than 95% done for common duct stone extraction; and it has become clear that patients undergoing procedures for SOD do not do as well as patients undergoing endoscopic sphincterotomies for stone retrieval, especially when the procedures are done by endoscopists with limited experience. Despite the popularity and acceptance of this nonoperative approach, a significant complication rate is acknowledged. Pancreatitis rates up to 20% have been reported, and several early series reported 1% to 2% mortality. In addition, recurrent stenosis is reported in up to 25% to 33% of patients. Other significant complica-

### Table 5. Results of Transduodenal Sphincteroplasty Reported Between 1975 and 1996

<table>
<thead>
<tr>
<th>Source</th>
<th>No. of Patients</th>
<th>Excellent or Good Result, %</th>
<th>Morbidity, %</th>
<th>Mortality, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kozloff and Joseph, 1975</td>
<td>65</td>
<td>82</td>
<td>NR</td>
<td>3.0</td>
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<tr>
<td>Partington, 1977</td>
<td>138</td>
<td>79</td>
<td>NR</td>
<td>2.9</td>
</tr>
<tr>
<td>Madura et al, 1981</td>
<td>50</td>
<td>88</td>
<td>28</td>
<td>2.0</td>
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<tr>
<td>Bagley et al, 1981</td>
<td>67</td>
<td>64</td>
<td>9</td>
<td>6.0</td>
</tr>
<tr>
<td>Nardi et al, 1983</td>
<td>95</td>
<td>48</td>
<td>NR</td>
<td>4.2</td>
</tr>
<tr>
<td>Anderson et al, 1985</td>
<td>56</td>
<td>77</td>
<td>28</td>
<td>5.7</td>
</tr>
<tr>
<td>Hasabouka et al, 1986</td>
<td>22</td>
<td>60</td>
<td>18</td>
<td>0</td>
</tr>
<tr>
<td>Stephens and Burdick, 1986</td>
<td>81</td>
<td>68</td>
<td>7</td>
<td>1.2</td>
</tr>
<tr>
<td>Duca, 1989</td>
<td>70</td>
<td>97</td>
<td>7</td>
<td>1.4</td>
</tr>
<tr>
<td>Nussbaum et al, 1989</td>
<td>29</td>
<td>62</td>
<td>38</td>
<td>0</td>
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<tr>
<td>Mooy et al, 1991</td>
<td>86</td>
<td>75</td>
<td>23</td>
<td>0.8</td>
</tr>
<tr>
<td>Azabache Puente and Saavedra, 1992</td>
<td>58</td>
<td>95</td>
<td>25</td>
<td>1.7</td>
</tr>
<tr>
<td>Kelly and Rowlands, 1996</td>
<td>20</td>
<td>65</td>
<td>25</td>
<td>0</td>
</tr>
<tr>
<td>Touli et al, 1996</td>
<td>26</td>
<td>58</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>Overall</td>
<td>863</td>
<td>74.1</td>
<td>20.0</td>
<td>1.9</td>
</tr>
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</table>

Abbreviation: NR, data not reported.

### Table 6. Results of Endoscopic Sphincterotomy Reported Between 1981 and 2003

<table>
<thead>
<tr>
<th>Source</th>
<th>No. of Patients</th>
<th>Excellent or Good Result, %</th>
<th>Morbidity, %</th>
<th>Mortality, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rosch et al, 1981</td>
<td>37</td>
<td>86</td>
<td>4.6</td>
<td>1.3</td>
</tr>
<tr>
<td>Riemann et al, 1983</td>
<td>25</td>
<td>80</td>
<td>13.0</td>
<td>0</td>
</tr>
<tr>
<td>Tanaka et al, 1985</td>
<td>12</td>
<td>66</td>
<td>0</td>
<td>0</td>
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<tr>
<td>Neoptolemos et al, 1987</td>
<td>30</td>
<td>70</td>
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<td>Thatcher et al, 1987</td>
<td>51</td>
<td>77</td>
<td>15.6</td>
<td>0</td>
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<td>Guerjude, 1988</td>
<td>17</td>
<td>65</td>
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<td>23</td>
<td>65</td>
<td>8.7</td>
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<td>Weitemeyer, 1994</td>
<td>247</td>
<td>82</td>
<td>28.3</td>
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<td>Hwang et al, 1996</td>
<td>24</td>
<td>71</td>
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<td>Tzovaras and Rowlands, 1998</td>
<td>36</td>
<td>84</td>
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<td>37</td>
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<td>Cicala et al, 2002</td>
<td>14</td>
<td>93</td>
<td>NR</td>
<td>NR</td>
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<tr>
<td>Park et al, 2003</td>
<td>313</td>
<td>75</td>
<td>15.0</td>
<td>0</td>
</tr>
<tr>
<td>Overall</td>
<td>866</td>
<td>77.0</td>
<td>16.8</td>
<td>0.12</td>
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Abbreviation: NR, data not reported.
tions include duodenal perforation, intraluminal hemorrhage at the sphincterotomy site, and pancreatic duct strictures. Restenosis of the pancreatic duct orifice has been a problem reported after long-term stenting with polyethylene or other prostheses, and has led to focal sclerosing pancreatitis in the head of the pancreas, requiring subsequent pancreaticoenteric anastomosis or resection in some patients.48,49

The use of intraoperative manometry allowed us to assess the patients with suspected dysfunctional pancreaticobiliary sphincters in the major and minor papilla. In addition, immediate postsphincteroplasty manometry ensured that the abnormal pressures were in fact reduced to normal levels. As previously mentioned, once the choledochal sphincter was ablated, the pancreatic duct and its sphincter could be tested for increased pressures, and if the pressures were normal or subnormal, as they were in some cases, then no pancreatic sphincteroplasty was done. Because both ducts drain through the main ampulla in 90% of patients, a more distal sphincter obstruction could affect both orifices, and the pressures in the pancreas and biliary tree could be diminished by sphincter ablation of the major ampulla alone.

In centers with skilled endoscopists, few sphincteroplasties are referred to surgeons. The technical revolution limiting biliary tract surgical experience to choledochojunostomy has eliminated routine intraoperative cholangiography, and no efforts are made to evaluate the sphincteric mechanism. There do remain situations in which the biliopancreatic ampullae are unreachable by endoscopy, such as following gastric restrictive procedures for obesity and in patients who have undergone gastric resection with Billroth II or Roux-en-Y reconstruction. These patients are unable to be diagnosed endoscopically, and endoscopic sphincterotomy is impossible as well. In this situation, the surgeon may be called on to approach this area via open laparotomy, and, given lack of experience and the equipment necessary to measure ductal and sphincteric pressures, he or she may be only able to do a “blind” transduodenal sphincteroplasty without subjective evidence before or after the procedure.

The therapy of pancreaticobiliary sphincter dysfunction is still evolving. Transendoscopic diagnostic methods can identify with certainty patients with symptoms and abnormal manometry results. The endoscopic results of treatment in carefully selected patients are uniformly good, at least in the short-term (Table 4). Transendoscopic sphincterotomy is done much more frequently than surgical sphincteroplasty ever was; however, the pitfalls observed in surgical sphincterotomy remain (ie, without suturing the ducts open, restenosis will occur in many patients). The routine placement of pancreatic duct stents in major and minor papillae after endoscopic sphincterotomy seems to contribute to recurrent pancreatic ductal obstruction and symptomatic chronic pancreatitis. In conclusion, patients with primary or postcholecystectomy symptoms of upper abdominal pain, nausea, and/or vomiting may elude diagnosis of their problem. Many of these patients may be referred for ERCP with manometry results diagnostic of dysfunction of the sphincter of Oddi. Most of these patients will be treated by endoscopic sphincterotomy, but there is significant morbidity and occasional pancreatitis-related mortality. In addition, recurrent stenosis of the pancreatic duct occurs with greater frequency than was seen in the surgical sphincteroplasty era. Surgeons may then be called on to treat these difficult clinical problems. In areas in which skilled endoscopic therapists are not available, surgeons may be required to be the primary therapist and should be prepared to study these patients with intraoperative manometry, not only to ensure that the diagnosis is correct but that surgical treatment is adequate. Preparation for these operations requires equipment and knowledge of its use. If done appropriately, transduodenal sphincteroplasty can be done reliably, with minimal morbidity and mortality, with equal or better results than those observed endoscopically.

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have to say that this is sphincter spasm, congenital hypertrophy of the muscular layer, or some other unknown cause resulting in sphincter dysfunction. When biopsying, you must also remember there are several articles reporting that there are a few patients who have an undetected neoplasm of the ampulla. We had one such patient, who is not included in this series, that had an adenocarcinoma of his ampulla, and we did a Whipple on him 17 years ago, and he is still alive and well.

The pressure studies are a little more complex because we were measuring several anatomical sphincters, but 80% of the patients had a reduction in sphincter pressure and this did correlate with a better outcome. Ten percent had no change in pressure, but these were patients who had normal pressures in the pancreatic duct, so we did not do a pancreatic sphincteroplasty, thinking we could not improve that, and those patients all did quite well. The other 10% of patients, mostly with pancreatic divisum, whose pressures actually increased post sphincterotomy (but I think that was a mechanical problem with the triple-lumen catheter that seemed to get impacted in the duct, occluding ≥1 lumina and giving falsely elevated pressure), these patients did about the same as the other pancreas divisum patients.

Dr Pickleman asked why we did a dual sphincteroplasty in the pancreas divisum patients and if it was necessary. We were measuring pressures, flows, and manometric pressures in these patients, and found that many of these patients actually had abnormal sphincter of Oddi function. Another reason we did dual sphincteroplasty is that early in our experience we were not always sure of the diagnosis of pancreas divisum, so once we identified the ventral duct a pancreatogram would clinch the diagnosis for us. We, therefore, did a procedure that was originally described in the early 1970s in which we opened both sphincters. Reviewing published pancreas divisum articles, it is inconsistent what procedures were done and the reasons for them. Some did dual sphincteroplasty; some did only the accessory papilla, while others left gallbladders intact. We thought that if we eliminated all chances for recurrent pain we would be more successful, and so we did dual sphincteroplasties routinely. So it is my opinion that opening both ducts appears to be better than accessory ductoplasty alone.

The question of the large number of patients seen and operated upon has been raised. These patients were not all from Indiana, but referred from across the central part of the United States. It reflects the interest in this disease process at our medical center and the skill and proficiency of our endoscopy team. In reviewing the endoscopic literature, there are a very small handful of centers, mostly in the Midwest and on the East Coast, that do sizable numbers of ERCPs, manometries, and endoscopic sphincterotomies. It was estimated a few years ago that over 10 000 endoscopic sphincterotomies were done around the world, and 93% were done for stones. Only 5% of endoscopic sphincterotomies were done for sphincteric dysfunction, and these are done in a few places that have developed ERCP manometric investigation and safe methods of sphincterotomy. In spite of the thousands of ERCPs and manometries that Drs Lehman and Sherman have done in Indiana, the largest series that they have reported thus far is 300 cases in 2003. The endoscopy service has conferences to demonstrate what they do. I do not think it teaches unskilled endoscopists to be skilled, but it opens new lines of referral to our center.

You asked for technical tips to avoid a hamburger-appearing sphincter. They do look like that when you first start doing them, but Dr Robert Hermann of The Cleveland Clinic was a visiting professor when I first started doing these and taught me a few important techniques. The idea is to get good exposure and do the operation at skin level if the patient is not excessively large. We perform a generous Kocher maneuver and pass a small Bakes dilator through the common duct into the duodenum, palpate it, and make as small a longitudinal incision as possible over the sphincter itself. The most important thing I learned from Dr Hermann was to place a 3-0 silk traction suture about 1 cm distal to the ampulla and retract it through the duodenal opening and attach it to the patient's skin or the drapes, then you have a wonderfully stable and accessible field in which to work. In this way, you can avoid duodenal trauma from forceps and other grasping instruments. Magnification is probably important for beginners or people who wear glasses to help locate the orifice of the pancreatic duct. If you cannot find the pancreatic duct, the use of intravenous secretin is helpful, since it will localize and intermittently dilate the ductal orifice. When identifying the accessory duct, it is usually about 1 cm cephalad and 1 cm medial to the major papilla. We used absorbable suture routinely on the choledochal sphincteroplasty, and on the pancreatic duct we used polypropylene so that the endoscopists would have “landing lights” should they ever need to restudy the ducts. Finally, we always closed the duodenum longitudinally. We initially used T tubes, but quickly abandoned that, and we always used an old-fashioned Penrose drain in the right upper quadrant. So that is how our treatment evolved in doing this procedure.

Jeffrey Landercasper, MD, LaCrosse, Wis: Dr Madura, I commend you on such low surgical morbidity and mortality on over 400 patients undergoing operations on the duodenum and sphincter. I have a question for you, also about indications. Do you have any data, do you have any information, on a similar cohort of patients diagnosed during this same time with manometrically proven sphincter of Oddi dysfunction who did not undergo either endoscopic sphincterotomy or surgical sphincterotomy, but rather chose to undergo no treatment or medical treatment and how they did in long-term follow-up to compare to your group?

Dr Madura: I do not have such information from our center, but there are 2 randomized controlled studies in the literature, one by Geenen in Milwaukee and the other by Touuli of Australia. In both of those reports, they did manometric studies in all patients and then they did a sham sphincterotomy in one group and a real endoscopic sphincterotomy in the other, and there was definitely a therapeutic advantage to having a sphincterotomy in their patients. And while these are quoted as landmark studies, they contain only 40 or 30 patients in each study, but that is what the endoscopy literature considers significant.