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Original article

Transduodenal sphincteroplasty and transampullary septectomy for sphincter of Oddi dysfunction

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***Background*: The diagnosis and management of sphincter of Oddi dysfunction are controversial issues. Both surgical and endoscopic series report modest success in the treatment of this condition. There is evidence from endoscopic series that the Milwaukee classification could predict the clinical outcome after sphincterotomy. We reviewed our long-term results of surgical sphincter ablation for sphincter of Oddi dysfunction, in order to correlate outcome with underlining pathology (biliary *versus* pancreatic) and Milwaukee biliary group classification.**

***Patients and Methods*: During a 10 year period (1987–1996), 36 patients with either biliary (n = 26) or pancreatic (n = 10) presentation of suspected sphincter of Oddi dysfunction were selected for surgery according to a standard protocol of investigation and management. All patients were classified according to the Milwaukee classification for the biliary group or its version for the pancreatic group and had transduodenal sphincteroplasty and transampullary septectomy.**

***Results*: Despite a trend towards a better outcome in the biliary group (good result 62%, moderate 23%, poor 15%) compared to the pancreatic (good result 40%, moderate 40%, poor 20%) the difference was not statistically significant (P = 0.48). Milwaukee classification for the biliary group correlated well with a favourable outcome (P <0.05).**

***Conclusions*: The modest outcome despite careful patient selection for surgery emphasises the need for more objective diagnostic tools. Milwaukee classification appears to be of good predictive value, and a good result can be anticipated in type I or even type II patients. The trend towards a better outcome in the biliary group may reflect the weakness of a drainage procedure to treat patients with parenchymal pancreatic disease.**

Key words: Sphincter of Oddi dysfunction – Sphincteroplasty – Postcholecystectomy syndrome

The diagnosis and management of sphincter of Oddi dysfunction is a challenging problem. Sphincter of Oddi dysfunction can cause symptoms of biliary or pancreatic origin presenting as biliary colic or recurrent pancreatitis, respectively. There is little doubt that the disorder exists, but controversy reigns as to how it can be diagnosed accurately and which patients would benefit from sphincter ablation. A combination of clinical, biochemical and radiological criteria,[1] ERCP manometry,[2–5] scintigraphic studies,[6–8] and provocation tests[8–10] have been proposed as criteria to select patients for sphincter ablation, but despite these modalities diagnosis remains often arbitrary and empirical.[11] Another

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area of controversy is how best to accomplish sphincter ablation – by a surgical procedure or endoscopically. The results from both endoscopic and surgical series are similar in outcome and morbidity with relatively high failure rates; this probably reflects the aforementioned difficulties in accurately diagnosing the disorder.[12] There is evidence from recent series that Milwaukee classification[1] could predict the outcome after endoscopic sphincterotomy in the biliary group of patients with sphincter of Oddi dysfunction.[13]

The aim of this study was to analyse the results of our 10-year experience as a referral unit for hepatobiliary and pancreatic surgery in Northern Ireland on transduodenal sphincteroplasty and transampullary septectomy for sphincter of Oddi dysfunction. We attempt to identify any correlation between long-term outcome and underlying pathology (biliary *versus* pancreatic) as well as Milwaukee classification.

Patients and Methods

Between 1987 and 1996, 36 consecutive patients (15 male, 21 female: median age, 46.4 years; range, 17–69 years) underwent transduodenal sphincteroplasty and transampullary septectomy for suspected sphincter of Oddi dysfunction. This group of patients represents a minority of the patients referred from other hospitals in Northern Ireland for investigation of pain of biliary/pancreatic origin and were carefully selected according to our protocol.

Patients were divided as having either biliary (n = 26: 8 male and 18 female: median age, 47.5 years; range, 17–69 years) or pancreatic (n = 10: 7 male and 3 female: median age, 43.6 years; range, 24–57 years) type pathology on the basis of clinical presentation and investigation. The biliary group consisted of patients with 'postcholecystectomy syndrome' (n = 14), in whom choledocholithiasis had been excluded, or patients with typical biliary colic and an intact gallbladder with no evidence of gallstones or abnormal gallbladder function on ultrasonography, oral cholecystogram and scintigraphy. These patients were further classified according to Milwaukee classification[1] (Table 1) as: type I, 11 patients; type II, 11 patients; or type III, 4 patients. The pancreatic group included patients with recurrent idiopathic acute pancreatitis (n = 5) or patients with chronic pancreatitis only if they had a dilated pancreatic duct (n = 5). Three of those with chronic pancreatitis had previously undergone distal pancreatectomy and splenectomy for pain control with poor outcome. Patients with gallstones, pancreas divisum, or chronic pancreatitis in the absence of a dilated pancreatic duct were not included in this series. Patients of the pancreatic group were classified according to the classification proposed by Sherman *et al.*[14] which is similar to the biliary classification (Table 2) and all were classified as type II.

Table 1 *Milwaukee biliary group classification (Hogan & Geenen[1])*

	Biliary type pain	Abnormal LFTs	Dilated CBD	Delayed drainage
Group I	+	+	+	+
Group II	+	With one or two of the above		
Group III	+	None of the above		

Abnormal LFTs: ALP and AST more than twice normal value, on at least two occasions.
Dilated CBD: > 12 mm on ultrasound scan (or 10 mm on ERCP film).
Delayed drainage: > 45 min on ERCP (supine position).
This classification was slightly modified in that PTC was performed instead of ERCP in a few cases (same criteria were used as for ERCP: delayed drainage of contrast when more than 45 min in supine position).

Table 2 *Pancreatic group classification (Sherman* et al.*[14])*

	Recurrent pancreatitis and/or typical pancreatic pain	Abnormal enzymes	Dilated PD	Delayed drainage
Group I	+	+	+	+
Group II	+	With one or two of the above three		
Group III	+	None of the above three		

Abnormal enzymes: > 1.5–2 times the upper normal limit.
Dilated PD: > 6 mm in the head, > 5 mm in the body, on ERCP film
Delayed drainage: > 9 min.

According to our selection protocol, all patients had significant and at times incapacitating pain for at least one year prior to operation, had no benefit from medical therapy including spasmolytic agents and smooth muscle relaxants, and many of them eventually required strong analgesia including opioids. Other upper gastrointestinal tract pathology was excluded in every case with endoscopy and/or radiological contrast studies. All patients had liver and pancreatic enzymes checked on several occasions, abdominal ultrasound scan and either ERCP or PTC. Patients with their gallbladder intact and negative ultrasonography had additionally oral cholecystogram and HIDA scintigraphy. Late in this series, the morphine-prostigmine provocation test was added to the protocol, although this did not influence any decision by itself. The test was defined as positive when the original pain was reproduced along with either pancreatic (amylase, lipase) or liver (AP, ALT) enzyme elevation 4 times above baseline values, provided the elevation exceeded the upper normal value. Other tests used as single or in combination on an individual basis included intravenous cholangiogram, CT

scan (especially done in all patients in the pancreatic group) and small bowel series. Endoscopic manometry was not performed in any patient of this series.

The decision to proceed to surgical management was taken on the basis of strong evidence of sphincter of Oddi dysfunction from the pre-operative investigations, in combination with a significant disruption of normal life-style caused by the duration and severity of symptoms. Transduodenal sphincteroplasty and transampullary septectomy were always performed by the senior author (BJR) in order to standardise the procedure and has been described in detail previously.[15] Cholecystectomy was routinely added when the gallbladder was intact, despite being normal on pre-operative work-up. By this policy, microlithiasis or chronic inflammation of the gallbladder wall were excluded as a cause of the symptoms in every case.

Patients were followed-up as out-patients every 3 months for the first year, every 6 months for the second year and yearly up to 5 years. Patients who were discharged from the clinic were contacted by telephone to obtain follow-up information beyond 5 years. If indicated, patients with poor or moderate outcome were seen more frequently than the above plan. The parameters evaluated were 30 day mortality and morbidity, length of operative time, length of hospital stay and long-term outcome. The latter was estimated on a simple scale as good (patient pain-free/spectacularly improved and no further need for hospital admission/ analgesia), moderate (some improvement of symptoms but still analgesia required, though less often than pre-operatively) and poor (no change/ worse). The outcome was correlated to the patient's pathology (biliary *versus* pancreatic) as well as to Milwaukee classification.

Statistical analysis

Correlation between outcome and patient's pathology group (biliary *versus* pancreatic) and between outcome and type of disorder in the biliary group (types I–III) was analysed using the chi-square test. As all patients in the pancreatic group were classified in the same type (type II), such correlation was not possible for the pancreatic group.

Results

The mean duration of the procedure was 150 min (range, 70–260 min). No death occurred in this series. There were 12 minor complications that did not affect the length of hospital stay and 4 major complications which resulted in prolonged hospital stay (Table 3). One patient with a history of COAD developed acute respiratory failure during the second postoperative day and needed ICU support for 4 days. Another patient developed severe pancreatitis (>3 Ranson's criteria) which settled on conservative treatment. One patient developed a pseudocyst which required an elective cyst-gastrostomy and a fourth needed limited small bowel resection for adhesive obstruction. The median hospital stay was 12.5 days (range, 8–45 days).

Table 3 Complications after transduodenal sphincteroplasty and transampullary septectomy

Minor	*n*	Major	*n*
Atelectasis	4	Acute respiratory failure	1
Transient hyper-amylasemia (< 48 h)	7	Frank pancreatitis	1
		Pancreatic pseudocyst	1
Wound infection	1	Small bowel obstruction	1

Table 4 Outcome according to group classification and Milwaukee classification

	Good	Moderate	Poor
Biliary group			
Type I ($n = 11$)	9	2	0
Type II ($n = 11$)	7	2	2
Type III ($n = 4$)	0	2	2
Total ($n = 26$)	16 (62%)	6 (23%)	4 (15%)
Pancreatic group			
*Type II ($n = 10$)	4 (40%)	4 (40%)	2 (20%)
Overall ($n = 36$)	**20 (56%)**	**10 (28%)**	**6 (16%)**

*According to the classification proposed by Sherman *et al.*[14]

Median follow-up was 54 months (range, 9–120 months). The outcome in each group, type and overall is shown in Table 4. There appears to be a trend towards a better outcome in the biliary group compared to the pancreatic one (62% *versus* 40%) but the difference did not reach statistical significance ($P = 0.48$). Interestingly, in the biliary group there was a good correlation between the Milwaukee classification and the outcome ($P < 0.05$).

Repeat ERCP was performed in 11 patients (6 with poor and 5 with moderate outcome) 12–16 months postoperatively, in order to exclude re-stenosis as the cause of persistent symptoms. In all cases both sphincteroplasties were found wide open.

A morphine-prostigmine provocation test was performed pre-operatively in 11 patients late in the series. Using the above mentioned definitions, the test was positive in 7 cases: three had good, three had moderate and one had poor outcome. Interestingly, all three patients with positive provocation test and good outcome belonged to the type I biliary group. Of the four patients with equivocal or negative test, one had good, one moderate and two had poor outcome. None of the patients with negative provocation test belonged to the type I group.

Discussion

Sphincter of Oddi dysfunction is an uncommon but debilitating condition. The modest number of patients in this series during a 10 year period, from a well defined area of Northern Ireland, reflects the rarity of the disorder. Its incidence in the general population or following cholecystectomy is impossible to estimate, mainly because most patients were tertiary referrals, reflecting the authors' special interest in this condition.

The disorder may present with symptoms from either the biliary or the pancreatic tree. There is a significant overlap in clinical presentation which is not surprising, given the anatomy of the sphincter and the functional inter-relationship between its segments.[16] The ideal test to diagnose accurately sphincter of Oddi dysfunction has yet to be identified. Endoscopic manometry has been proposed by some authors as the gold standard for diagnosis of sphincter of Oddi dysfunction.[2–5] However, the test has been criticised by others: it is invasive, requires sedation, is done under relatively unnatural conditions[17] and is associated with a higher incidence of pancreatitis compared to diagnostic ERCP,[18,19] especially when the pancreatic segment of the sphincter is evaluated.[20] Furthermore, previous studies[21,22] failed to show a correlation between manometric findings and outcome after sphincterotomy. It seems that manometry is redundant for type I patients, as the vast majority have uniformly increased pressure and for type III patients, as most of them have normal pressures.[12] Its role is probably limited for type II patients where it may provide useful information and identify a subgroup that would benefit from sphincter ablation.[12] Interestingly, in a recent prospective trial[13] including patients with type III and III biliary dysfunction, where manometric findings were used to select patients for endoscopic sphincterotomy, manometry did not correlate with outcome or Milwaukee classification. The latter, in contrast, correlated well with a favourable outcome. Endoscopic manometry was not performed in any of our patients, but Milwaukee classification was used and analysis showed there was a good correlation between this classification and surgical outcome. To our knowledge, there is no other surgical series in which this correlation has been tested, previously.

As endoscopists have become more adept at sphincterotomy, treatment seems to have shifted from surgical to less invasive endoscopic methods;[23] still there is no evidence to suggest superiority over the surgical sphincteroplasty. Literature lacks prospective randomised comparative trials and cohort studies are difficult to compare because of material heterogeneity and differences in methodology. Poor results remain high with both endoscopic sphincterotomy (12–39%)[5,13,21,22,24–27] and surgical sphincteroplasty (7–29%) (Table 5).[28–35] However, follow-up is significantly longer in surgical series. As mucosa-to-mucosa apposition and, therefore, minimisation of the scar tissue can be achieved with sphincteroplasty, the incidence of re-stenosis is anticipated to be lower than after sphincterotomy. This was actually tested in the long-term status many years ago.[36,37] Originally, the procedure included division of the sphincter and suturing of bile duct mucosa to duodenal mucosa in order to prevent narrowing of the opening and extravasation of biliopancreatic secretions. Later, involvement of the transampullary septum in the fibrotic process causing obstruction of the pancreatic outflow, was thought by some authorities to be responsible for recurrent symptoms and, therefore, septectomy was added to the original operation.[9,28] Indeed, separate recordings of pancreatic sphincter pressure in patients with recurrent pancreatitis will often demonstrate high pressure even after endoscopic ablation of the biliary sphincter.[38,39] Moody *et al.* described 12 of 17 patients with poor symptomatic outcome following adequate sphincteroplasty who obtained pain relief after excision of the septum.[40] The surgical approach has the advantage of securing adequate drainage from both biliary and pancreatic ducts.

A distinction between biliary and pancreatic pathology is important, as there is evidence that results could differ significantly between the two groups. In the present series, there was certainly a trend towards a better result in the biliary group, although the difference was not significant due to the small numbers in each group. This is consistent with previous reports,[28,30,34] although the inclusion criteria vary considerably between series. A poorer result in the pancreatic group, especially in the absence of chronic pancreatitis, has been attributed to the presence of underlying parenchymal disease with early minimal changes, not apparent in conventional imaging.[34] Our data support this hypothesis, as sphincteroplasty was found wide open on repeat endoscopy in patients with a poor result and none of the patients with recurrent acute pancreatitis experienced further episodes of pancreatitis. Toouli *et al.*[35] reported recently a series of surgical sphincteroplasty on patients with idiopathic recurrent pancreatitis. This series is unique in that manometry was used for pre-operative evaluation, although manometric findings were not always used to select patients for intervention nor have the manometric results been correlated with the outcome. Interestingly, patients initially treated with conventional endoscopic sphincterotomy had poor results, and 5 out of 7 needed further treatment with surgical sphincteroplasty and septectomy. Overall, 15 of their 26 patients with transduodenal sphincteroplasty and septectomy were cured and only three had a poor result. The outcome does not seem to be as good in patients with chronic pancreatitis though.[31] Williamson[33] reported his results on a mixed group and concluded that sphincteroplasty has a limited role to play when used in isolation in chronic pancreatitis, but could be useful as an adjunct to other procedures. We were not able to

Table 5 *Results of surgical treatment*

Source	Year	*n*	Follow-up	Result	Morbidity
Moody *et al.*[28]	1983	83	1–10 years	Good, 43%; fair, 33%; poor, 24%	21%
Nardi *et al.*[29]	1983	89*	5–20 years	Pain free, 50%	N/A
Anderson *et al.*[30]	1985	28	67.5 months (mean)	Good, 71%; poor, 29%	23%
Stephens *et al.*[31]	1986	81	6 months	Pain free, 68%; fair, 25%; poor, 7%	7%
Hastbacka *et al.*[32]	1986	22		Good, 59%; moderate, 23%; poor, 18%	18%
Williamson[33]	1988	20	6–100 months	Pain free, 60%; improved, 25%; poor, 15%	20%
Nussbaum *et al.*[34]	1989	29	22 months (mean)	Ex/good, 62%; fair, 14%; poor, 24%	38%
Toouli *et al.*[35]	1996	26*	9–105 months	Pain free, 58%; mild, 30%; poor, 12%	8%
Present series		36	54 months (9–120 months)	Good, 56%; moderate, 28%; poor, 16%	11%

*For recurrent acute pancreatitis exclusively.

detect any difference in our pancreatic group for two reasons: first, we included patients with chronic pancreatitis only when there was dilated pancreatic duct, which makes our group highly selected. Second, 3 out 5 of our patients had undergone pancreatic resection in the past (Table 5).

Morphine-prostigmine provocation test has been used as diagnostic tool,[9,10,33] although it has been criticised for lack of specificity.[41,42] Dynamic hepatobiliary scintigraphy has been tested as well as non-invasive diagnostic methods for the biliary group and there are recent reports which have shown high sensitivity.[6–8] The small number of patients who had these tests in our series precludes any conclusion about their usefulness. Interestingly though, we detected three patients of the type I biliary group with abnormal HIDA scintigraphy and positive provocation test. All experienced a good result. This may suggest that even if these methods in isolation lack any predictive value for the outcome, when combined with Milwaukee classification they could expand its high predictive value.

Conclusions

Our results are in agreement with previous reports; the constantly reported modest outcome after sphincter ablation mainly reflects the difficulty in accurate diagnosis of the disorder. Alternatively, it may indicate that sphincter of Oddi dysfunction is sometimes part of a more generalized motility disorder of the gastrointestinal tract and, therefore, targeting to the sphincter only is not the solution to the problem.[12,43] In any case, the decision for any procedure on the sphincter should be taken cautiously, after meticulous investigation and patient selection; Milwaukee biliary group classification has certainly an important role to play in this selection process.

References

1. Hogan WJ, Geenen JE. Biliary dyskinesia. *Endoscopy* 1988; **20**: 179–83.
2. Gilbert DA, DiMarino AJ, Jensen DM, Katon R, Kimmey MB, Laine LA *et al.* Status evaluation: sphincter of Oddi manometry. *Gastrointest Endosc* 1992; **38**: 757–9.
3. Thune A, Scicchitano J, Roberts-Thompson I, Toouli J. Reproducibility of endoscopic sphincter Oddi manometry. *Dig Dis Sci* 1991; **10**: 1401–5.
4. Lans JL, Parikh NP, Geenen JE. Application of sphincter of Oddi manometry in routine clinical investigations. *Endoscopy* 1991; **23**: 139–43.
5. Geenen JE, Hogan WJ, Dodds WJ, Toauli J, Venu RP, Wagner Jr HN. The efficacy of endoscopic sphincterotomy after cholecystectomy in patients with sphincter of Oddi dysfunction. *N Engl J Med* 1989; **320**: 82–7.
6. Sostre S, Kalloo AN, Spiegler EJ, Camargo EE. A non-invasive test of sphincter of Oddi dysfunction in postcholecystectomy patients: the scintigraphic score. *J Nucl Med* 1992; **33**: 1216–22.
7. Persson B, Axelson B, Jacobsson H. Cholesintigraphy in the diagnosis and assessment of benign papillary stenosis. *Eur J Nucl Med* 1993; **20**: 770–5.
8. Madascy L, Velosy B, Lonovics J, Csernay L. Evaluation of results of the prostigmine-morphine test with quantitative hepatobiliary scintigraphy: a new method for the diagnosis of sphincter of Oddi dyskinesia. *Eur J Nucl Med* 1995; **22**: 227–32.
9. Nardi GL, Acosta JM. Papillitis as a cause of pancreatitis and abdominal pain: role of evocative test, operative pancreatogram and histologic evaluation. *Ann Surg* 1966; **164**: 611–21.
10. Buntain WL, Buonocore E, Royal SA. Beneficial use of an evocative test in chronic relapsing pancreatitis in children. *Am J Surg* 1986; **152**: 628–37.
11. Moody FG, Vecchio R, Galabuig R, Runkel N. Transduodenal sphincteroplasty with transampullary septectomy for stenosing papillitis. *Am J Surg* 1991; **161**: 213–8.
12. Tzovaras G, Rowlands BJ. Diagnosis and treatment of sphincter of Oddi dysfunction. *Br J Surg* 1998; **85**: 588–95.
13. Wehrmann T, Wiemer K, Lembcke B , Yung M. Do patients with sphincter of Oddi dysfunction benefit from endoscopic sphincterotomy? A 5-year prospective trial. *Eur J Gastroenterol Hepatol* 1996; **8**: 251–6.
14. Sherman S, Troiano FP, Hawes RH, O'Connor KW, Lehman GA. Frequency of abnormal sphincter of Oddi manometry compared with the clinical suspicion of sphincter of Oddi dysfunction. *Am J Gastroenterol* 1991; **86**: 586–90.
15. Kelly SB, Rowlands BJ. Transduodenal sphincteroplasty and transampullary septectomy for papillary stenosis. *HPB Surgery* 1996; **9**: 199–207.
16. Boyden EA. The anatomy of the choledohoduodenal junction in man. *Surg Gynecol Obstet* 1957; **104**: 641–52.
17. Blades EW, Sivak MV. Sphincter of Oddi manometry: how far is the leap of faith? *Gastrointest Endosc* 1993; **39**: 593–5.
18. King CE, Kalvaria I, Sninsky CA. Pancreatitis due to endoscopic biliary manometry: proceed with caution. *Gastroenterology* 1998; **94**: A227.

19. Sherman S, Hawes RH, Troiano FP, Lehman GA. Pancreatitis following bile duct sphincter of Oddi manometry: utility of the aspirating catheter. *Gastrointest Endosc* 1992; **38**: 347–50.
20. Lehman GA. Endoscopic sphincter of Oddi manometry: a clinical practice and research tool. *Gastrointest Endosc* 1992; **37**: 490–2.
21. Thatcher BS, Sivak MV, Tedesco FJ, Vennes JA, Hutton SW, Ashkar EA. Endoscopic sphincterotomy for suspected dysfunction of the sphincter of Oddi. *Gastrointest Endosc* 1987; **33**: 91–5.
22. Roberts-Thompson IC, Toouli J. Is endoscopic sphincterotomy for disabling biliary-type pain after cholecystectomy effective? *Gastrointest Endosc* 1985; **31**: 370–3.
23. Burton FR. Postcholecystectomy syndrome. How to determine if the sphincter of Oddi is the cause. *Postgrad Med* 1992; **91**: 255–8.
24. Riemann JF, Lux G, Forster P, Altendorf A. Long-term results after endoscopy papillotomy. *Endoscopy* 1983; **15**: 165–8.
25. Neoptolemos JP, Bailey IS, Carr-Locke DL. Sphincter of Oddi dysfunction: results of treatment by endoscopic sphincterotomy. *Br J Surg* 1988; **75**: 454–9.
26. Seifert E. Long term follow-up after endoscopic sphincterotomy. *Endoscopy* 1988; **20**: 232–5.
27. Bozkurt T, Orth KH, Butsch B, Lux G. Long-term clinical outcome of postcholecystectomy patients with biliary-pain: results of manometry, non-invasive techniques and endoscopic sphincterotomy. *Eur J Gastroenterol Hepatol* 1996; **8**: 245–9.
28. Moody FG, Becker JM, Potts JR. Transduodenal and transampullary septectomy for postcholecystectomy pain. *Ann Surg* 1983; **197**: 627–36.
29. Nardi GL, Michelassi F, Zannini P. Transduodenal sphincteroplasty. *Ann Surg* 1983; **198**: 453–61.
30. Anderson TM, Pitt HA, Longmire Jr WP. Experience with sphincteroplasty in pancreatobiliary surgery. *Ann Surg* 1985; **201**: 399–406.
31. Stephens RV, Budrick GE. Microscopic transduodenal sphincteroplasty and transampullary septoplasty for papillary stenosis. *Am J Surg* 1986; **152**: 621–7.
32. Hastbacka J, Jarvinen H, Kivilaakso E, Turunen MT. Results of sphincteroplasty in patients with spastic sphincter of Oddi. *Scand J Gastroenterol* 1986; **21**: 516–20.
33. Williamson RCN. Pancreatic sphincteroplasty: indications and outcome. *Ann R Coll Surg Engl* 1988; **70**: 205–11.
34. Nussbaum MS, Wamer BW, Sax HC, Fischer JE. Transduodenal sphincteroplasty and transampullary septotomy for primary sphincter of Oddi dysfunction. *Am J Surg* 1989; **157**: 38–43.
35. Toouli J, Di Francesco V, Saccone G, Kollius J, Schloithe A, Shanks N. Division of the sphincter of Oddi for treatment of dysfunction associated with recurrent pancreatitis. Br J Surg 1996; **83**: 1205–10.
36. Eiseman B, Brown WH, Virabutr W, Gottesfeld S. Sphincterotomy: evaluation of its physiologic rationale. *Arch Surg* 1959; **79**: 294–303.
37. Jones SA, Steedman RA, Keller TB, Smith LL. Transduodenal sphincteroplasty (not sphincterotomy) for biliary and pancreatic disease: indications, contra-indications and results. *Am J Surg* 1969: **118**: 292–306.
38. Funch-Jensen P, Kruse A. Manometric activity of the pancreatic duct sphincter in patients with total bile duct sphincterotomy for sphincter of Oddi dyskinesia. *Scand J Gastroenterol* 1987; **22**: 1067–70.
39. Rolny P, Arleback A, Funch-Jensen P, Kruse A, Jamerot G. Clinical significance of manometric assessment of both pancreatic duct and bile duct sphincter in the same patient. *Scand J Gastroenterol* 1989; **24**: 751–4.
40. Moody FG, Calabuing R, Vecchio R, Runkel N. Stenosis of the sphincter of Oddi. *Surg Clin North Am* 1990; **70**: 1341–54.
41. Steinberg WM, Salvato RF, Toskes PP. The morphine-prostigmine provocative test: is it useful for making clinical decisions? *Gastroenterology* 1980; **78**: 728–31.
42. LoGiudice JA, Geenen JE, Hogan WJ, Dodds WJ. Efficacy of the morphine-prostigmine test for evaluating patients with suspected papillary stenosis. *Dig Dis Sci* 1979; **24**: 455–8.
43. Soffer EE, Johlin FC. Intestinal dysmotility in patients with sphincter of Oddi dysfunction. A reason for failed response to sphincterotomy. *Dig Dis Sci* 1994; **39**: 1942–6.