Treatment of bile duct lesions after laparoscopic cholecystectomy

J J G H M Bergman, G R van den Brink, E A J Rauws, L de Wit, H Obertop, K Huibregtsen, G N J Tytgat, D-J Gouma

Abstract
From January 1990 to June 1994, 53 patients who sustained bile duct injuries during laparoscopic cholecystectomy were treated at the Amsterdam Academic Medical Centre. There were 16 men and 37 women with a mean age of 47 years. Follow up was established in all patients for a median of 17 months. Four types of ductal injury were identified. Type A (18 patients) had leakage from cystic ducts or peripheral hepatic radicles, type B (11 patients) had major bile duct leakage, type C (nine patients) had an isolated ductal stricture, and type D (15 patients) had complete transection of the bile duct. Endoscopic retrograde cholangiopancreatography (ERCP) established the diagnosis in all type A, B, and C lesions. In type D lesions percutaneous cholangiography was required to delineate the proximal extent of the injury. Initial treatment (until resolution of symptoms and discharge from hospital) comprised endoscopy in 36 patients and surgery in 26 patients. Endoscopic treatment was possible and successful in 16 of 18 of type A lesions, five of seven of type B lesions, and three of nine of type C lesions. Most failures resulted from inability to pass strictures or leaks at the initial endoscopy. During initial treatment additional surgery was required in seven patients. Fourteen patients underwent percutaneous or surgical drainage of bile collections, or both. After endoscopic treatment early complications occurred in three patients, with a fatal outcome in two (not related to the endoscopic therapy). During follow up six patients developed late complications. All 15 patients with complete transection and four patients with major bile duct leakage were initially treated surgically. During initial treatment additional endoscopy was required in two patients. Early complications occurred in eight patients. During follow up seven patients developed stenosis of the anastomosis or bile duct. Reconstructive surgery in the early postoperative phase was associated with more complications than elective reconstructive surgery. Most type A and B bile duct injuries after laparoscopic cholecystectomy (80%) can be treated endoscopically. In patients with minor ductal injury (type C and D) reconstructive surgery is eventually required in 70%. Multidisciplinary approach to these lesions is advocated and algorithms for treatment are proposed.

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Keywords: cholecystectomy, laparoscopy, bile duct, endoscopy.

In the past four years laparoscopic cholecystectomy has gained widespread acceptance among surgeons and the public and has replaced conventional 'open' cholecystectomy as treatment of choice for symptomatic cholecystolithiasis. Several large multicentre studies have shown the efficacy and overall safety of laparoscopic cholecystectomy. Compared with 'open' cholecystectomy, laparoscopic cholecystectomy is associated with less postoperative pain, shorter stay in hospital and recovery, earlier return to work, and a better abdominal cosmetic outcome. Laparoscopic cholecystectomy does, however, carry an increased risk for biliary tract injury. Several series have been published describing the serious morbidity and mortality of these injuries. Comparatively few papers, however, have focused on treatment of bile duct injuries once they have occurred.

The aim of this study was to evaluate the diagnosis and treatment of bile duct lesions after laparoscopic cholecystectomy and to propose algorithms for treatment of these complications.

Methods

Patients
From January 1990 to June 1994, 53 patients were at least partially treated at the Academic Medical Centre, Amsterdam, the Netherlands, for bile duct lesions sustained during laparoscopic cholecystectomy. In 48 patients (91%) the laparoscopic cholecystectomy was performed in one of 32 referring hospitals, five patients (one transection and four bile leaks) had their laparoscopic intervention at the Amsterdam Academic Medical Centre. There were 16 men and 37 women with a mean age of 47 years (range 22–89).

Study analysis
The study included a retrospective evaluation of the initial laparoscopic procedure, presenting symptoms of the bile duct lesion, classification and site of the ductal injury, diagnostic procedures and therapeutic interventions before and after referral, and follow up to date.
Laparoscopic cholecystectomy was defined as any attempt at cholecystectomy begun as a laparoscopic procedure regardless of whether the procedure was converted to an open cholecystectomy or if the injury occurred after conversion. Laparoscopic bile duct injury was defined as any clinically evident damage to the biliary system (including the cystic duct), occurring at any time after laparoscopic cholecystectomy without any other probable cause. Exclusion criteria included patients with common bile duct stones or biliary neoplasia but without disruption of the biliary system.

Sources of information
Information concerning the initial laparoscopic procedure was obtained from both the operative report and personal interview with the surgeon. Presenting symptoms of the bile duct injury and results of diagnostic or therapeutic interventions performed at referring centres, or both, were obtained from referral notes, local medical charts, and interviews with referring physicians. Patients were followed up at the outpatient clinic of our institution at regular intervals. If the patient had been discharged from further follow up or when follow up was performed by local physicians (in general for less serious bile duct injuries or after one to two year event free follow up) information was obtained from general practitioners and the patients themselves. Follow up was established up to August 1994 in all patients.

Classification of the bile duct injuries
We identified four types of bile duct injury: type A: cystic duct leaks or leakage from aberrant or peripheral hepatic radicles, type B: major bile duct leaks with or without concommitant biliary strictures, type C: bile duct strictures without bile leakage, and type D: complete transection of the duct with or without excision of some portion of the biliary tree. The site of the ductal lesion was determined by its most proximal border.

Definitions
Initial treatment was defined as all therapeutic interventions performed from diagnosis of the lesion until complete resolution of symptoms and discharge from the hospital. Early complications comprised all complications occurring during initial treatment. Late complications were defined as all complications occurring during follow up after initial treatment was established. Secondary treatment comprised all therapeutic interventions performed because of late complications.

Results

Laparoscopic cholecystectomy
The indication for laparoscopic cholecystectomy was uncomplicated symptomatic gall stone disease in most patients (Table I). Only a limited number of patients had an indication generally considered a risk factor for complications after laparoscopic cholecystectomy.13 'Uncomplicated' laparoscopic cholecystectomy was reported in 25 patients (47%), whereas in 28 patients technical problems were encountered during surgery (Table II). Of these 28 laparoscopic procedures with technical problems, only four (14%) were converted to an open technique because of these problems. We did not attempt to discover if in these cases the actual ductal lesion occurred before or after the conversion. In six additional cases the procedure was converted to an open cholecystectomy because iatrogenic lesions of the biliary tract were suspected. Therefore, the total number of conversions was 10 of 53 (19%). Intraoperative cholangiography was performed in two patients (4%).

Clinical presentation
Six bile duct injuries were identified at the time of the laparoscopic cholecystectomy. In the remaining 47 patients the ductal lesions presented postoperatively, one day to 93 weeks (median three days) after the initial laparoscopic procedure. Clinical presentation varied widely and was primary influenced by the type of injury (Table III). Bile leaks, either resulting from minor bile duct laceration (type A lesion), major bile duct laceration (type B lesion), or complete transection (type D lesion), tended to differ in clinical presentation from ductal strictures without bile leakage (type C lesion). This last group had a longer symptom free interval after the laparoscopic cholecystectomy (median of 57 days versus three days) and presented more often with signs of biliary obstruction (for example, jaundice, cholestatic liver function tests, dilatation of the proximal biliary tree on ultrasound, Table III). In contrast with isolated strictures, bile leaks presented in a less uniform way. Symptoms in the early postoperative phase were comparatively aspecific with general malaise, low grade fever, marginally increased liver function tests, and absence of dilatation on radiological imaging. More specific and severe symptoms such as jaundice, sepsis, and ileus were more frequently found in patients with bile leakage but these usually only became manifest several days after the initial operation.

<table>
<thead>
<tr>
<th>Table I</th>
<th>Indications for laparoscopic cholecystectomy in 53 patients who sustained bile duct injuries during laparoscopic cholecystectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication</td>
<td>n=53</td>
</tr>
<tr>
<td>Biliary colics</td>
<td>48</td>
</tr>
<tr>
<td>Acute cholecystitis</td>
<td>3</td>
</tr>
<tr>
<td>Previous bile duct stones</td>
<td>1</td>
</tr>
<tr>
<td>Adenomyomatosis</td>
<td>1</td>
</tr>
</tbody>
</table>

TABLE II In 53 patients with bile duct injury after laparoscopic cholecystectomy, technical problems occurred during the initial laparoscopic procedure in 28 patients. (Some patients had multiple problems)

<table>
<thead>
<tr>
<th>Technical problems</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Unclear anatomy, difficult dissection Calot's triangle</td>
<td>10</td>
</tr>
<tr>
<td>Bleeding</td>
<td>7</td>
</tr>
<tr>
<td>Abdominal adhesions</td>
<td>6</td>
</tr>
<tr>
<td>Bile leakage</td>
<td>5</td>
</tr>
<tr>
<td>Short or wide cystic duct</td>
<td>6</td>
</tr>
<tr>
<td>Gall bladder removed in fragments</td>
<td>1</td>
</tr>
<tr>
<td>Removal of clip on common bile duct</td>
<td>1</td>
</tr>
</tbody>
</table>
TABLE III  Symptoms and signs in 47 patients with bile duct injuries after laparoscopic cholecystectomy that were noted postoperatively

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Overall (%) (Type A,B, and D,n=18)</th>
<th>Biliary stricture (%) (Type C, n=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jaundice</td>
<td>40 29 78</td>
<td>p&lt;0.05*</td>
</tr>
<tr>
<td>Cholestatic LFTs ultrasound</td>
<td>68 58 100</td>
<td>p&lt;0.05*</td>
</tr>
<tr>
<td>General malaise</td>
<td>29 13 89</td>
<td>p&lt;0.001*</td>
</tr>
<tr>
<td>Fever</td>
<td>72 84 33</td>
<td>p&lt;0.05*</td>
</tr>
<tr>
<td>Right upper abdominal pain</td>
<td>49 63 0</td>
<td>p&lt;0.01*</td>
</tr>
<tr>
<td>Fluid collections on tonography</td>
<td>78 84 56</td>
<td>p=0.17*</td>
</tr>
<tr>
<td>Ultrasound/computed tomography</td>
<td>63 79 0</td>
<td>p=0.001*</td>
</tr>
<tr>
<td>Sepsis</td>
<td>10 13 0</td>
<td>p=0.56*</td>
</tr>
<tr>
<td>Ileus</td>
<td>18 23 0</td>
<td>p=0.17*</td>
</tr>
<tr>
<td>Symptom-latency time (median, range)</td>
<td>3 (1-651) 3 (1-24)</td>
<td>p&lt;0.001†</td>
</tr>
<tr>
<td>Diagnosis-latency time (median, range)</td>
<td>8 (0-81) 8 (0-81)</td>
<td>p=0.68†</td>
</tr>
</tbody>
</table>

LFT = liver function test; symptom-latency time = time from operation until occurrence of first symptoms; diagnosis-latency time = time from occurrence of first symptoms until diagnosis of the ductal lesion. *Fisher’s exact test; †Mann-Whitney test.

**Diagnostic procedures**

Abdominal ultrasound was performed in 37 of 47 patients in whom the lesion was noted postoperatively and showed fluid collections in 63% and dilatation of the biliary tract in 29% of patients. In 10 patients fluid collections were punctured percutaneously under ultrasound guidance.

ERCP was performed in 45 patients and established the diagnosis in all type A, B, and C lesions. Although in type D lesions retrograde cholangiography showed a total stop and was thus of diagnostic importance, the exact proximal extent of the lesion could not be determined and required percutaneous transhepatic cholangiography or fistulography. Furthermore, in one patient with complete transection of the right segmental hepatic duct the retrograde cholangiogram was falsely interpreted as normal. Subsequent fistulography established the correct diagnosis.

**Type and level of the bile duct injury**

There were 18 minor bile duct leaks (type A lesions), 11 major bile duct leaks (type B lesions), nine bile duct strictures without concomitant bile leakage (type C lesions), and 15 patients with a complete transection of the duct (type D lesions). Table IV shows the localisation of the different types of bile duct injuries. After diagnosis of the bile duct lesion, initial treatment was performed at referring centres in 13 patients and after referral to our centre in 40 patients. Table V shows initial treatment and complications before and after referral.

**Minor bile duct leaks (type A lesions)**

**Initial treatment** (Table V) – all 18 patients with a type A ductal injury were diagnosed and treated endoscopically. Endoscopic treatment consisted of endoscopic sphincterotomy (nine patients) or insertion of a biliary endoprosthesis (nine patients) with elective stent removal after six weeks (Fig 1). Insertion of biliary endoprostheses was essentially attempted without sphincterotomy unless a precut sphincterotomy proved necessary to obtain access to the common bile duct or a sphincterotomy was required for removal of gall stones. Percutaneous or surgical drainage of bile collections, or both, was performed in nine patients (before ERCP in six, after endoscopy in three).

**Early complications** – complications of the endoscopic treatment occurred in two patients. One patient with a cystic duct leak and distal obstruction caused by a stone in the cystic duct remnant (Mirrizi’s syndrome) suffered from bleeding after precut sphincterotomy and recovered after blood transfusions. She underwent surgical resection of the cystic duct remnant. An 89 year old patient with a cystic duct leak died seven days after ERCP. He presented 15 days after surgery with multi organ failure and respiratory insufficiency that proved irreversible after stent insertion.

**Late complications and secondary treatment** – during a median follow up period of 14 months (range 1–57) three late complications occurred. One patient developed colicky abdominal pain and underwent laparotomy for removal of a second gall bladder. A second patient developed a stricture of the common hepatic duct, six months after laparoscopic cholecystectomy and was successfully treated endoscopically. Re-evaluation of previous cholangiograms showed that this stenosis was overlooked at the initial ERCP at which time a short endoprosthesis was placed bypassing only the biliary sphincter but not bridging the stenosed part of the bile duct. Finally a third patient with an injury to an aberrant bile duct developed atrophy of hepatic segment 6–7 and a distal bile duct stricture. She was treated endoscopically. None of the remaining 14 patients developed any late complications or showed abnormal liver function tests at follow up to date.

**Major bile duct leaks (type B lesions)**

**Initial treatment** (Table V) – there were 11 patients with bile leakage from the common bile duct or the main hepatic duct. Four

TABLE IV  Classification and localisation of 53 patients with bile duct injuries during laparoscopic cholecystectomy

<table>
<thead>
<tr>
<th>Minor bile duct leaks (Type A lesions)</th>
<th>Major bile duct leaks (Type B lesions)</th>
<th>Strictures (Type C lesions)</th>
<th>Complete transection (Type D lesions)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common bile duct</td>
<td>0</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Bifurcation &lt; 2 cm</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Bifurcation</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Left and/or right hepatic duct</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Cystic duct</td>
<td>12</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Peripheral biliary radicle</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Aberrant bile duct</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>11</td>
<td>9</td>
</tr>
</tbody>
</table>
patients underwent suture repair over a T tube at the site of the leakage (two patients during cholecystectomy). ERCP and insertion of biliary endoprostheses was attempted in seven patients. Endoscopic treatment generally consisted of insertion of an endoprosthesis at the first endoscopic procedure. After six weeks the lesion was evaluated endoscopically. In the case of absence of leakage and no signs of secondary ductal stenosis the endoprosthesis was removed. If a stricture of the bile duct was present, however, the patient entered the endoscopic treatment regimen for benign ductal stenosis: two 10 Fr endoprostheses were placed and left in situ for one year with elective stent exchange every three months to prevent cholangitis from clogging. Stent insertion failed in two patients with a large defect in the wall of the common hepatic duct. The guidewire selectively passed through the defect and could not be advanced into the proximal biliary system. Further treatment in these patients consisted of hepaticojejunostomy (n=1) and surgical T tube insertion followed by endoscopic placement of an endoprosthesis (n=1). Drainage of bile collections was performed in five patients, all before (n=1) or during initial treatment (n=4).

Early complications – early complications occurred in two patients: a 74 year old patient died four days after stent insertion of a myocardial infarction and a second patient with bile leakage after removal of the T tube was treated endoscopically with biliary endoprostheses.

Late complications and secondary treatment – during a median follow up of 17 months (range 0–33) three patients (all initially treated with suture repair and T tube insertion), developed late ductal stenosis. In two a hepaticojejunostomy was performed after endoscopic treatment had failed, the third patient was successfully treated with biliary stenting for one year. All patients initially treated with biliary endoprostheses are free of symptoms with normal liver function parameters, 4 to 32 months (median 20) after laparoscopic cholecystectomy (6 to 11 months after stent removal). Two patients still have stents in situ.

Bile duct strictures (type C lesions)

Initial treatment (Table V) – all nine patients with ductal strictures had their diagnosis confirmed and insertion of endoprostheses attempted at ERCP. In four patients insertion of the endoprostheses failed because the stricture could not be passed by a guidewire. Three subsequently underwent surgery; hepaticojejunostomy (n=2), removal of hemoclips (n=1). The fourth patient had complete disappearance of all symptoms before any further treatment. She remains free of symptoms with normal liver function tests, 18 months after the stenosis was diagnosed. The most probable explanation is that she has developed a biliary-digestive fistula that bypasses the biliary stricture. Insertion of a stent was successful in the remaining five patients but one patient

![Figure 1: Example of a type A lesion (minor bile duct leakage). Left hand side: retrograde cholangiogram showing leakage of contrast through the cystic duct remnant (percutaneous drain in situ). Middle: short 10 Fr straight polyethylene endoprosthesis (Amsterdam type) inserted. Right hand side: retrograde cholangiogram after elective removal of the endoprosthesis six weeks after insertion; no more leakage of contrast from the cystic duct remnant, some contrast in the duodenum.](http://gut.bmj.com/)

<table>
<thead>
<tr>
<th>Table V</th>
<th>Initial treatment and complications of 53 patients with bile duct injuries during laparoscopic cholecystectomy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor bile duct leaks</td>
<td>Major bile duct leaks</td>
</tr>
<tr>
<td>Initial treatment before referral</td>
<td>4</td>
</tr>
<tr>
<td>Surgery</td>
<td>0</td>
</tr>
<tr>
<td>Endoscopy</td>
<td>4</td>
</tr>
<tr>
<td>Early complications (%)</td>
<td>0</td>
</tr>
<tr>
<td>Late complications (%)</td>
<td>3 (75)</td>
</tr>
<tr>
<td>Initial treatment after referral</td>
<td>14</td>
</tr>
<tr>
<td>Surgery</td>
<td>1*</td>
</tr>
<tr>
<td>Endoscopy</td>
<td>14</td>
</tr>
<tr>
<td>Early complications (%)</td>
<td>2 (14)</td>
</tr>
<tr>
<td>Late complications (%)</td>
<td>0</td>
</tr>
<tr>
<td>Overall</td>
<td>n=18</td>
</tr>
<tr>
<td>Surgery</td>
<td>6*</td>
</tr>
<tr>
<td>Endoscopy</td>
<td>18</td>
</tr>
<tr>
<td>Drainage bile collections</td>
<td>n=5</td>
</tr>
<tr>
<td>Early complications (%)</td>
<td>2 (11)</td>
</tr>
<tr>
<td>Late complications (%)</td>
<td>3 (17)</td>
</tr>
<tr>
<td>Deaths (%)</td>
<td>1 (6)</td>
</tr>
</tbody>
</table>

*After failed endoscopic stone extraction, †for bile leakage after removal of T tube, ‡after failed stent insertion, §after failed stent insertion in three patients, because of treatment preference in one patient, ¶for bile leakage after end to end anastomosis of the common hepatic duct.
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underwent hepaticojejunostomy because of treatment preference of the referring physician.

**Early complications** — no complications occurred during initial treatment of type C lesions.

**Late complications and secondary treatment** — during a median follow up of 20 months (range: 5–33) there were three late complications and one unrelated death. Two patients developed an episode of fever after endoscopic stent exchange that resolved with conservative treatment. In one patient the stricture persisted after one year stenting. He subsequently underwent uncomplicated hepaticojejunostomy. The patients who underwent successful endoscopic treatment are free of symptoms with normal liver function tests 10 and 19 months after removal of the stents. One patient still has stents in situ.

**Complete transaction of the bile ducts (type D lesions, Fig 2)**

**Initial treatment** (Table V) — all 15 patients with a type D bile duct injury eventually underwent reconstructive surgery. The following reconstructive surgical procedures were performed: Roux-en-Y hepaticojejunostomy at the confluence or common hepatic duct (n=11), separate hepaticojejunostomies to both the right and left main hepatic duct (n=2) or to the right hepatic duct only (n=1), and end to end anastomosis of the common hepatic duct (n=1). A Rodney Smith’s mucosal graft procedure was performed on one patient. Of 14 hepaticojejunostomies, two were performed at the initial cholecystectomy, five were performed in the early postoperative phase mainly during diagnostic laparotomy, and seven were performed electively after 8–12 weeks. In the last group drainage of bile collections was initially established percutaneously or surgically, or both, to stabilise the patient’s condition. At a second stage, in most cases 24 hours before the reconstruction, percutaneous transhepatic cholangiography and drainage (PTCD) (Fig 2) was performed to investigate the proximal extent of the lesion and drains were inserted to guide the surgeon during the reconstructive procedure.

**Early complications** — reconstructive surgery was complicated by bile leakage in seven patients (before referral in four patients (80%), after referral in three (30%), Table V). Three of these patients required surgery and cardiorespiratory supportive measures, one patient was treated with biliary endoprosthesis, and three patients were treated by external drainage and antibiotics.

**Late complications and secondary treatment** — median follow up in the 15 patients with a type D injury was 25 months (range 6–38) and late complications occurred in seven patients (47%). Four patients had recurrent cholangitis caused by stenosis of the biliodigestive anastomosis and were treated with percutaneous transhepatic balloon dilatation (n=3) or reconstruction of the hepaticojejunostomy (n=2), or both. Two patients had signs of biliary obstruction that resolved spontaneously. Diagnostic imaging in these patients showed no evidence of stenosis and an expectant policy was followed. Finally, one patient with episodes of recurrent pancreatitis caused by sphincter stenosis after previous precut sphincterotomy was treated endoscopically. At follow up to date all patients are free of symptoms but seven patients (47%) have cholestatic liver function parameters (>3 times the upper limit).

**Figure 2:** Example of a type D lesion (complete ductal transaction). Left hand side: retrograde cholangiogram showing only filling of the distal bile duct with complete obstruction at the site of operation clips. (Note: minor leakage of contrast). Right hand side: percutaneous cholangiogram through the left hepatic system to delineate the proximal extent of the injury: ischaemic stricture at the site of the bifurcation, leakage of contrast into a subhepatic cavity drained by a percutaneous pigtail catheter

**Algorithm for treatment of bile duct injuries after laparoscopic cholecystectomy.**

PTCD: percutaneous transhepatic cholangiography and drainage.

HJS: hepaticojejunostomy.

**Figure 3:** Algorithm for treatment of bile duct injuries after laparoscopic cholecystectomy.
Early complications and late stenosis of the anastomosis occurred more often in patients who underwent reconstructive surgery in the early postoperative phase: in the five patients who underwent reconstructive surgery in the early postoperative phase, four complications were seen (80%) whereas in the seven patients who underwent elective hepaticojejunostomy only one complication occurred (14%), (p=0.07 Fisher’s exact test).

Secondary reconstructive surgery
Five patients (two type B lesions, one type C lesion, and two type D lesions) underwent a secondary hepaticojejunostomy because of ductal (re)stenosis. All these secondary procedures were performed without early complications and no patient has developed any further late complications during a median follow up of nine months (range 1–22).

Discussion
This study shows that the diagnosis of bile duct injuries after laparoscopic cholecystectomy is usually straightforward in patient with an isolated ductal stricture (Table III), but may be difficult in patients with bile leakage as symptoms are frequently absent or aspecific in the early postoperative phase. Early diagnosis in these patients is, however, important because the clinical condition may rapidly deteriorate after three to five days when ileus, peritonitis, and sepsis develop. Several authors have, therefore, emphasised the importance of early aggressive investigation in patients with diffuse abdominal pain, fever, malaise or liver function abnormalities after laparoscopic cholecystectomy. First step is to perform an abdominal ultrasound to investigate the presence of ductal dilatation or fluid collections. The last are often located in the lower abdominal cavity. Biliary dilatation is often absent (in our series in 71%) because the biliary system is decompressed by the leak. In the event of fluid collections, percutaneous needle aspiration may differentiate an abscess from a biloma. When ductal dilatation is present or needle aspiration yields bile, an ERCP is the next diagnostic procedure. In our study, ERCP established the diagnosis in all type A, B, and C lesions and attributed to the diagnosis and further treatment in patients with type D lesions. However, the last group required percutaneous cholangiography to delineate the proximal extent of the injury. Early diagnostic laparotomy without classification of the injury and therapeutic plan should be avoided.

In our series endoscopic treatment proved effective in 89% of the patients with leakage from cystic ducts or peripheral hepatic radicles (type A ductal injury) though 16% required additional external drainage of biloma. These results are comparable with those recently published by Kozarek et al. Others have advocated the use of endoscopic sphincterotomy for minor bile duct leaks. However, endoscopic sphincterotomy is not without risks and possible longterm sequelae of loss of sphincter function are of concern in younger patients. We, therefore, treat these patients with biliary endoprostheses to bypass the leak and, more importantly, to lower the pressure of the biliary system by bypassing the biliary sphincter (Fig 3). The stent is preferably inserted without prior endoscopic sphincterotomy unless this is necessary to extract bile duct stones or gain biliary access. Although insertion of an endoprosthesis gives the patient the burden of a second endoscopic intervention for removal of the stent, we feel that this is outweighed by preventing a sphincterotomy.

Insertion of an endoprosthesis proved successful in 71% of patients with leakage from major bile ducts (type B lesions). This success rate is comparable with the 76–79% success rate reported by others. Three patients treated with suture repair and T tube insertion developed late stenosis (75%). Although this probably reflects the referral bias this study suffers from, it points towards an important late complication of this type of lesion; secondary stenosis at the site of the leak. Woods et al described 27 patients with major bile leaks or strictures, or both. Primary suture repair was in patient with four of these 11 needing additional endoscopic stenting for secondary strictures and another four requiring biliodigestive bypass. Surgical treatment with placement of a T tube is generally advocated for these patients if the injury is detected during cholecystectomy.

Although this will successfully treat the bile leakage, the duration of T tube placement may be too short to effectively prevent secondary stenosis. We, therefore, prefer to remove the T drain after six weeks and treat patients in whom the leak is detected postoperatively with primary endoscopic stenting (Fig 3). Insertion of an endoprosthesis not only adequately seals the bile leak, it also allows prompt and early diagnosis and treatment of secondary ductal stenosis. In case the endoscopic stent insertion fails we first attempt to drain the bile duct with PTCD, before resorting to surgical placement of a T tube (Fig 3).

In contrast with the results of endoscopic treatment in patients with a type A or type B lesion, the results of endoscopic treatment of isolated biliary strictures were disappointing. Overall, successful treatment of strictures was accomplished in three of eight patients (38%) in whom endoscopic treatment was attempted as definitive treatment (one patient with stents in situ). However, most failures (80%) resulted from inability to pass the stricture at the first endoscopic session, which was performed primarily as a diagnostic procedure and no adverse effects were noted from attempts at stent insertion. There are no randomised studies available comparing endoscopic stenting with surgical treatment for patients with bile duct strictures after cholecystectomy. We recently published a retrospective study in which these two treatment regimens were compared and concluded that surgery and endoscopy were equivalent. Because surgery is still available when endoscopy fails whereas vice versa is impossible once a
Roux-en-Y loop has been constructed, we prefer to attempt endoscopic treatment first (Fig 3).

All 15 patients with complete transection of the bile duct eventually underwent reconstructive surgery. Early morbidity, late stenosis of the anastomoses, and abnormal liver function tests at follow up to date all attest to the severity of the injury and the difficulty of adequate treatment. Most early and late complications occurred in patients initially treated at referring centres (Table IV). The outcome of surgical treatment of these lesions is influenced by a variety of factors including: proximal extent of the injury, type of reconstructive procedure performed, experience of the performing surgeon, timing of intervention, presence of proximal dilatation and local inflammation at the time of the procedure, condition of the patient, and the length of follow up. The numbers in this series are too small to perform a multivariate analysis for evaluation of these factors. An important factor determining the outcome of reconstructive surgery is the timing of the procedure. We observed that early complications and late anastomotic stenosis occurred in 80% of patients treated with early reconstructive surgery whereas these complications were observed in 17% of patients who underwent elective surgery after 8–12 weeks. Reconstructive surgery in the acute postoperative phase, often started as a diagnostic procedure in a patient with peritonitis, ileus or sepsis, is at risk for leakage and stenosis because of the absence of proximal dilatation and the presence of severe inflammatory changes of the tissue. Adequate drainage for 8–12 weeks allows for the acute local inflammatory reaction to subside and enables the surgeon to establish the exact proximal extent of the injury before surgery. In most patients 24 hours before the reconstruction, a PTD was performed to delineate the proximal anatomy and to insert a biliary catheter. These percutaneous catheters may be very helpful at surgery for identification of the injured duct and for subsequent stenting of the anastomosis if necessary.

Laparoscopic cholecystectomy has shown its overall safety and efficacy. Although most centres performing laparoscopic cholecystectomy may now be well beyond the ‘learning curve’ phase, the incidence of bile duct injuries will probably stay increased compared with conventional cholecystectomy. The incidence and severity of the lesions warrant a systematic approach concerning diagnosis and treatment. Although differences in local expertise concerning interventional radiology, therapeutic endoscopy12 and reconstructive surgery9 may lead to modifications of the proposed algorithms, the principles outlined in this article may be helpful for the clinician in the treatment of bile duct lesions after laparoscopic cholecystectomy.

Treatment of bile duct lesions after laparoscopic cholecystectomy.

J J Bergman, G R van den Brink, E A Rauws, L de Wit, H Obertop, K Huibregtse, G N Tytgat and D J Gouma

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