23

Identification and Management of Bile Duct Injuries and Post-Operative Strictures

Arthur Richardson

Introduction

Bile duct injury (BDI) is associated with significant morbidity and mortality and may occur after gastric, pancreatic and biliary surgery. The vast majority of injuries occur following laparoscopic cholecystectomy (LC) and the diagnosis is often delayed with an associated increase in morbidity and mortality. The discussion will focus on the diagnosis and treatment of BDI following LC and will divide treatment into early and late injuries.

LC has become the treatment of choice for symptomatic gallstones since its introduction by Perissat, [1] Dubois and Colleagues [2] in France and Reddick and Olsen in the United States [3]. As LC rapidly replaced open cholecystectomy (OC) it became clear that the incidence of BDI was higher by a factor of about 2–3× [4–7]. It is now one of the most commonly performed general surgical operations and as such, although the frequency of BDI is low the prevalence is significant [8]. It was initially assumed by some authorities that the incidence of BDI would decrease with increasing surgeon experience [9–11] and although there is evidence to support this view [12], it does appear that the incidence of BDI remains at an unacceptable level.

The incidence of BDI following LC is difficult to ascertain. Up to one half of general surgeons may have been responsible for a BDI [13, 14] and a majority of surgeons may regard them as unavoidable and a legitimate complication of LC [14, 15]. In the author's opinion, this view is incorrect and BDI following LC should be regarded as an avoidable complication in most circumstances.

There is wide variation in the estimates of the incidence of BDI following LC. In the decade following the introduction of LC the incidence of BDI varied from 0.3% to 0.7%. [8, 10, 16–18]. More recent large population studies suggest that the incidence has declined and stabilised. A Swedish study analysing more than 150,000 cholecystectomies performed between 1987 and 2001 found a major BDI rate of 0.40% [19]. They found that the risk increased with older age and male gender and was decreased by the use of operative cholangiography. A more recent study from Sweden [20] looked at 55,134 cholecystectomies performed between 2007 and 2011 and found 174 BDI (0.3%) but only 30 patients required an hepatico-jejunostomy for reconstruction (0.05%).

A Finnish study [21] looked at 1616 OC and 6733 LC collected between 1997 and 2007. Altogether there were 75 BDI for an overall incidence of 0.9% which was divided into 20 BDI

A. Richardson, M.B.B.S. (SYD) D. Clin. Surg.
Department of Upper Gastrointestinal Surgery, Westmead Hospital, Westmead, NSW, Australia

University of Sydney, Sydney, NSW, Australia
e-mail: Arthur.richardson@sydney.edu.au

M.R. Cox et al. (eds.), *The Management of Gallstone Disease*,
https://doi.org/10.1007/978-3-319-63884-3_23

(1.24%) in the OC group versus 55 BDI in the LC group (0.82%). However, this included minor BDI as well as major BDIs and the majority of cases were managed endoscopically. If only cases requiring laparotomy were regarded as major injuries the incidence was much lower being 3 in the OC group (0.1%) and 18 in the LC group (0.2%).

There are reports of lower rates of BDI in the literature. A report from the Kaiser Permanente Northern California system looked at 83,449 patients who underwent LC between 1995 and 2008 [23]. They found 84 BDIs (0.10%) but of these more than half were cystic duct leaks. Only 34 (0.04%) of the injuries were major in that they required surgical reconstruction. There was a trend towards more severe proximal injuries and misinterpretation of the anatomy was cited by 92.9% of the surgeons as a contributing factor.

In one of the largest studies using the nationwide inpatient sample (NIS) in the United States, Worth et al. [24] analysed 3,741,260 LC between 2001 and 2011 and were able to show that the percentage of patients requiring a biliary reconstruction was 0.11% of LCs in 2001 versus 0.09% of LC in 2011, a statistically non-significant change. As this is an administrative database it is possible that this study underestimates the true incidence. Alternatively, a review of single incision laparoscopic cholecystectomy (SILC) suggested that the BDI rate was increased at 0.72%. However, it appears that the majority of these injuries were relatively minor being Strasberg A and as such the incidence of major bile duct injury was about 0.3% [25]. Only 13% of patients in this study had an operative cholangiogram. There appear to be methodological issues with this paper as pointed out by others [26, 27] and further investigation will be required to determine if there is an increased incidence of BDI with SILC versus conventional LC.

There is evidence to suggest that the rate of BDI has stabilised. Khan et al. [28] looked at the rate of BDI referred to a tertiary referral ERCP service over an 8.5-year period. Of 17,684 records they identified, 183 patients had a BDI. However, only 17 of these cases were common bile duct (CBD) or right hepatic duct (RHD) transections. They concluded that the frequency, anatomic distribution and rate per 100 ERCPs was static and this finding has been supported by other reports [29, 30].

When one critically reviews all of the recent available literature it would appear that the rate of major BDI requiring surgical reconstruction in countries with well-developed surgical care is of the order of 0.05–0.2% which equates to one case every 500–2000 LC. The weight of evidence would suggest that the figure is closer to one major BDI per 600–1000 LC and it appears to have remained stable over the past decade. There appears to be a trend towards more severe proximal injuries in the LC era [23].

No matter how one calculates the incidence it remains at an unacceptable level. Sedlack [31] analysed the issue from a quality improvement point of view. Specifically, if an incidence of one major BDI per 1500 LC is accepted as the rate of injury and if one applies six sigma design this equates to 95 defects per million opportunities (DPMO) and 5.25 sigma. If this rate were applied to the airline industry this would equate to 20 commercial airline crashes per day in the United States alone. Sedlack goes on to point out that if a DPMO of 3.4 is the accepted best practice [32] then at this rate the incidence of major BDI should be one case per 45,000 LC.

Classification of BDI

Multiple classification systems have been proposed over many years which often make comparison between surgical series confusing and misleading. The original classification was proposed by Bismuth and Corlette [33] in the era prior to LC and related to bile duct strictures (Table 23.1). This system is relatively simple to use and is based on how proximally the injury occurred and the length of the healthy proximal bile duct stump. This does tend to correlate with the ease of repair and with the ultimate outcome of repair [34]. However, it does not include any associated vascular injury.

A simple classification dividing injuries into major and minor was proposed by McMahon

[35] but this has limited usefulness in the era of LC. Likewise, the Csendes [36] classification attempted to simplify the grading of BDI but is rarely quoted in the contemporary literature. In an attempt to account for injuries in the LC era, Stewart and Way [37] developed a classification to try and describe the whole spectrum of injuries but does not take account of associated vascular injuries.

In Europe there have been multiple classification systems proposed some of which are used in the literature. The Amsterdam classification [38] was proposed as a more simple system in 1996 and this is again perhaps too simple for the era of LC but is not infrequently used in predominantly European publications. Neuhaus [39] proposed a system but it does not describe associated vascular injuries, is complicated and is infrequently used. Likewise a classification from Hong Kong [40] has not received widespread adoption.

Table 23.1 Bismuth's classification

Type	Criteria
1	Low CHD stricture, with a length of the common hepatic duct stump of >2 cm
2	Proximal CHD stricture-hepatic duct stump <2 cm
3	Hilar stricture, no residual CHD, but the hepatic ductal confluence is preserved
4	Hilar stricture, with involvement of confluence and loss of communication between right and left hepatic duct
5	Involvement of aberrant right sectorial hepatic duct alone or with concomitant stricter of CHD

The Strasberg classification [41] (Fig. 23.1) is probably the most widely accepted system and builds on the Bismuth classification. It defines major duct injuries as well as the causes of bile leaks (Chap. 22). It is relatively simple to remember and use (Table 23.2). It is divided into five classes (A–E), with five subclasses in E (E1–5). Type A represents a bile leak from the cystic duct or an accessory duct but where there is no loss of continuity with the common bile duct. Type B is a divided sector or segmental duct where there is no continuity with the common bile duct but no associated bile leak. Type C represents a leak from a sector or segmental bile duct but no continuity with the common bile duct. Type D is a partial section of the major bile duct but it is still in continuity with the biliary system. Type E is a complete transection of the major bile duct and the five subtypes (E1–5) relate to the stump length remaining. It appears that long term results can be correlated with the Strasberg grade of injury [42].

In 2007 the group from Hannover [43] attempted to devise a system that took account of the weaknesses and strengths of other classification systems. They pointed out that the injury

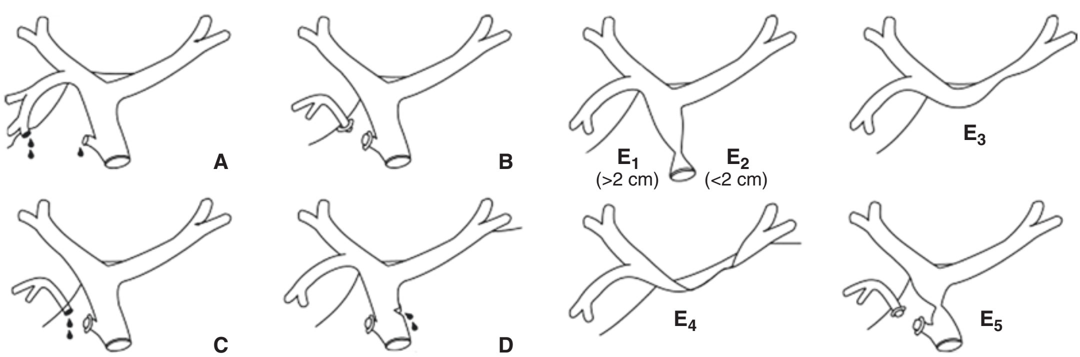

Fig. 23.1 Strasberg classification. (**a**) Bile leak from cystic duct or minor biliary radical in gallbladder fossa. (**b**) Occluded right posterior sectoral duct. (**c**) Bile leak from divided right posterior sectoral duct. (**d**) Bile leak from main bile duct without major tissue loss. (**e1**) Transected main bile duct with a stricture more than 2 cm from the hilus. (**e2**) Transected main bile duct with a stricture less than 2 cm from the hilus. (**e3**) Stricture of the hilus with right and left ducts in communication. (**e4**) Stricture of the hilus with separation of right and left ducts. (**e5**) Stricture of the main bile duct and the right posterior sectoral duct (from Strasberg SM et al. An analysis of the problem of biliary injury during laparoscopic cholecystectomy. J Am Coll Surg 1995;180:105)

Table 23.2 Comparison of Bismuth and Strasberg classifications

Biliary anatomy	Bismuth	Strasberg
Cystic duct leak or leak from small ducts in liver bed	–	A
Occlusion of an aberrant RHD	–	B
Transection without ligation of an aberrant RHD	–	C
Lateral injury to CBD (<50% circumference)	–	D
CHD stricture, stump >2 cm	Type 1	E1
CHD stricture, stump <2 cm	Type 2	E2
Hilar stricture, no residual CHD, confluence is preserved	Type 3	E3
Hilar stricture, involvement of confluence, loss of communication between RHD and LHD	Type 4	E4
Stricture of low-lying right sectorial duct (alone or with concomitant CHD stricture)	Type 5	–
Injury to an aberrant RHD plus injury in the hilum	Type 5	E5

CBD common bile duct, *CHD* common hepatic duct, *LHD* left hepatic duct, *RHD* right hepatic duct

in relation to the confluence of the hepatic duct and any associated vascular injury were major determinants of the outcome of surgical repair and as such these factors need to be accounted for in any comprehensive classification system. They contended that the major weaknesses of the other classification systems were that the Strasberg and Neuhaus systems did not consider vascular involvement and that the Stewart-Way and Neuhaus systems did not discriminate between injuries involving the confluence of the hepatic duct. Although the Hannover system is comprehensive it has not gained widespread use due to its complexity.

In view of all these difficulties with classification systems and in light of the difference in type of BDI in the LC era—the injuries seem to be more proximal, they more often present as bile leaks rather than strictures and frequently there appears to be an associated vascular injury often with loss of tissue, the European Association for Endoscopic Surgery (EAES) proposed a classification integrating the best features of other classifications [44]. It includes three categories and is referred to as AToM (Table 23.3). The three categories are A (for anatomy), To (time of) and M (mechanism). Additionally, the description includes the anatomic characteristics of the injury: NMBD for non-main bile duct or MBD for main bile duct (followed by a number 1–6 corresponding to the level of the injury on the main bile duct, followed by Oc (for occlusion), or D (for division), P (partial) or C (for complete), LS (loss of substance), VBI (vasculo-biliary injury), and the vessel when known (RHA, LHA, CHA, PV, MV); time of detection: Ei (early intraoperative), Ep (early post-operative-defined as less than 7 days) or L (late); and mechanism of injury: Me (mechanical) or ED (energy driven). Additionally, the EAES proposed a classification matrix for the time of occurrence of the BDI (Table 23.4). This is the most comprehensive classification system for BDI and is likely to replace other systems. Certainly, from the point of view of accurate reporting and comparison of cases this is the preferable system.

Prevention of BDI

The technique of LC and how to deal with the difficult case has been covered in Chaps. 13, 16, and 19.

BDI should be regarded as preventable in the majority of circumstances. Although inexperience may play a part in some injuries it is sobering that up to half of BDI are caused by experienced surgeons. Although this may be related to experienced surgeons doing the most difficult cases [13, 45]. In the majority of instances it appears that major injuries arise from misidentification of the anatomy rather than aberrant anatomy—most typically the common bile duct for the cystic duct [23, 46–48]. Certainly, it appears that more difficult cases with fibrosis and inflammation may be at higher risk of a BDI but the author's experience is that many of these cases occur in young women with no particular features of increased risk. Often, the surgeon will describe the case as straightforward and there may be no appreciation that a BDI has occurred.

Table 23.3 ATOM classification matrix for bile duct injuries (from Fingerhut et al. ATOM, the all-inclusive, nominal EAES classification of bile duct injuries during cholecystectomy. Surg Endosc 2013; 27:4608-4619)

	Anatomical characteristics						Time of detection			Mechanism	
Anatomic level	Type and extent of injury					Vasculobiliary injury (yes = VBI+) and name of injured vessel (RHA, LHA, CHA, PV, MV) (no = VBI−)	Ei (de visu, bile leak, IOC)	Ep	L	Me	ED
	Occlusion		Division								
	C	P[a]	C	P[a]	LS[b]						
MBD											
1											
2											
3											
4											
5											
6											
NMBD											

For each injury, the surgeon fills in the following matrix: (1) single injury (yes/no); (2) multiple injuries (yes/no). Then one matrix is filled in for each injury, as appropriate. For example, an injury made by an energy-driven (ultrasonic) dissector involving the superior biliary confluence with interruption or the right and left hepatic ducts, detected (intraoperatively) during the operation by the presence of bile would be classed as MBD 4 C VBI Ei, ED, The Connor Garden E6 injury is in fact a type 4 with LS: MBD 4 LS

EAES European Association for EndoSCOpic Surgery, *MRD* main biliary duct, *NMBD* nonmain biliary duct (Luschka duct, aberrant duct, accessory duct), *level 1* ≥2 cm from lower border of superior biliary confluent, *level* 2 <2 cm from lower border of Superior biliary confluent, *level 3* involves the superior biliary confluent but communication right left is preserved, *level 4* involves superior biliary confluent but communication right left is interrupted, *level 5a* right or left hepatic duct, *level 5b* right sectorial duct but bile duct still in continuity, *C* complete, *P* partial, *LS* loss of substance. *Me* mechanical, *ED* energy driven, *VBI* vasculobiliary involvement, *RHA* right hepatic artery, *LHA* left hepatic artery, *CHA* common hepatic artery, *PV* portal vein, *MV* marginal vessels, *Ei* early intraoperative, *Ep* early postoperative, *L* late, *OC* intra-operative cholangiogram

[a]Indicate percentage of circumference, if known

[b]Indicate length, if known

Table 23.4 EAES classification matrix for moment of occurrence of BDI

Item	Check if present
Before identification of cystic triangle elements	
During identification (dissection) of Cystic triangle elements	
After identification of Cystic triangle elements	
(clipping, energy-driven or ligation of cystic artery or duct, opening of the cystic duct for IOC); misinterpretation of above mentioned structures)	
Before cholecystectomy	
During cholecystectomy	
After cholecystectomy	
During dissection or manoeuvers for stone extraction from main bile duct via cystic duct	
During dissection or manoeuvers for stone extraction from main bile duct via common bile	
During IOC (opening the cystic duct or what is thought to be so), introduction of catheter or instrument for IOC)	
During other manoeuvers (hepaticoenterostomy)	
After IOC (withdrawal of catheter or instrument)	
Mechanical or energy-driven injury for elective haemostasis or ligation	
Mechanical or energy-driven injury for unexpected bleeding	

From EAES (http://www.eaes.eu)

EAES European Association for Endoscopic Surgery, *BDI* bile duct injury, *IOC* intraoperative cholangiogram

The science of technical human error is increasingly important in the practice of surgery to limit the frequency and severity of complications. Cognitive psychology attempts to explain how these errors occur and how they may be perpetrated by experienced practitioners despite strong evidence that a mistake has been made in identification. As explained by Way et al. [47] this can mean that the surgeon mistakes an abnormal situation using a mental and familiar recollection of the normal anatomy. This is then further complicated by cognitive fixation [49] and plan continuation meaning that the situation is not questioned and the error is continued. This may result in devastating consequences for the patient. Dekker [48] wrote of the situation when a variety of sensory, visual and tactile inputs interact to promote action i.e. continuing the operation. The situation may be made worse by other traps and errors such as the gallbladder not being retracted appropriately such that the cystic duct remains "hidden" [50]. Clearly, surgery has a lot to learn from this science.

Early in the era of LC it was clear that there was a problem with an increased rate of BDI and there was an attempt to develop guidelines to prevent BDI. At the National Institute of Health consensus conference in the early 1990s [51], seven critical steps were described to accurately identify the anatomy. It is not clear how widely these recommendations were adopted and there did not appear to be any improvement in the BDI rate during the late 1990s. Further preventative descriptions were proposed [6, 52] and these mostly involved dissection close to the gallbladder, avoidance of diathermy close to the common bile duct, careful identification of the cystic duct/common bile duct junction but without unnecessary dissection and the use of a 30° laparoscope. As pointed out by Connor [15] however, the above assumes that the anatomical identification and interpretation is appropriate which is often not the case. There are however, anatomical landmarks which may be helpful before dissection is begun. The author finds identification of Rouviere's sulcus helpful (Chaps. 1 and 16, Fig. 16.16) as dissection above this point will be safe. That said, this is not a constant anatomical finding because it is absent in up to 25% of patients [49].

One of the most commonly used techniques for biliary anatomical identification is the critical view of safety (CVS) as initially proposed by Strasberg and colleagues in 1995 [41]. More recently, CVS was re-described in detail [53] and contains three requirements. First, all fat and fibrous tissue must be cleared from the cholecystohepatic triangle (Figs. 16.8 and 16.9). An understanding of the anatomy of the fibrous connections of the liver and in particular the cystic plate (Chap. 1) is required to fulfil the second requirement. This second requirement requires the inferior portion of the gallbladder to be separated from the cystic plate to open the cholecystohepatic triangle (Fig. 16.10). Lastly, the third requirement is that this dissection should leave two and only two structures, being the cystic duct and cystic artery entering the gallbladder (Fig. 16.17). Once these three requirements have been met a CVS (Fig. 16.15) has been achieved. Photo or video documentation may be helpful for peer review, training and medico-legal purposes. Achievement and documentation of a CVS is mandatory in some countries [54] and is certainly strongly recommended [55–57]. In the author's opinion, achieving a CVS is essential in the performance of LC but, as it appears that a significant number of injuries occur while the surgeon is trying to achieve a CVS [46], it is not by itself sufficient to prevent BDI.

More recently, the use of checklists have been proposed by Gawande and others for a number of surgical interventions with an overriding objective to reduce error rates and improve outcomes [58]. A number of surgical procedures may be appropriate for this type of checklist and LC is an ideal surgical procedure as there are a number of critical steps involved.

Connor and colleagues [59] have described a five step approach which embraces the major safety measures to prevent BDI in a systematic way. They are:

1. To confirm the gallbladder lies in the hepatic plane and is retracted to the 10 o'clock position (Fig. 16.4).
2. Confirm Hartman's pouch is lifted up and toward the segment 4 pedicle (Figs. 16.6a, c, 16.7a).

3. Identify Rouviere's sulcus (Figs. 1.18 and 16.16).
4. Confirm the release of the posterior leaf of the peritoneum covering the hepato-biliary triangle (Figs. 16.7, 16.8).
5. Confirm the critical view of safety (Figs. 1.19 and 16.15) with or without operative cholangiography.

A key part of establishing the critical view of safety is dissecting from the cystic plate toward the cystic duct. When this cannot be achieved, a subtotal cholecystectomy should be performed (Chaps. 16 and 18). Failure to do so and dissecting more distally away from the gallbladder leads to misidentification of the CBD as the cystic duct.

There remains a controversy over the usefulness or otherwise of routine operative cholangiography (Chaps. 1, 16, 17, 18 and 20). It is fair to say that around the world the rate of routine operative cholangiography appears to be declining although this varies between countries. Certainly, in Australia there are surgeons who perform intra-operative cholangiography (IOC) routinely, selectively and not at all. Using large administrative databases, it is possible to get some idea of the rate of IOC. Amongst 20,307 patients between 2006 and 2009 in Denmark the rate of cholangiography being performed was less than 10% [22]. In Switzerland the rate of IOC declined from 37.1% in 1995 to 30.1% in 2005—a statistically significant decline [60].In a recent survey of surgeons in the United States IOC was only used in 16% of cases [61]. Using the United States nationwide inpatient sample between 2004 and 2009, 111,815 cases of cholecystectomy were analysed and only surgeons performing more than 10 cases per year were included. Even so, the average number of cholecystectomies per surgeon per year was still only 23.6. The average rate of IOC per surgeon was only 30% and the authors concluded that there was no difference in mortality or BDI rate but that the group having routine IOC received more additional procedures and incurred higher costs [62].

The major controversy with regard to routine IOC is whether it helps to avoid BDI. The issue has unfortunately been bedeviled by poor quality research on both sides and is unlikely ever to be resolved to every surgeon's satisfaction. The issue of routine IOC and the role of IOC has been discussed at length in Chap. 17. In summary, the author and the editors strongly support the use of routine IOC. Clearly, the technique of operative dissection and correct identification is the main factor in prevention of BDI. The use of routine IOC augments good dissection as demonstrated by the studies looking at large data bases where there is a significant reduction in the rate of BDI when IOC has occurred [12, 19, 63–67]. Secondly the correct performance and correct interpretation of the IOC reduces the extent of the injury and diagnoses the injury at the time of operation allowing for an immediate repair with improved outcomes [65, 67–70].

Management

The management of bile leaks and BDI can be divided into early and delayed according to the type of injury, using the Strasberg classification (Fig. 23.1 and Table 23.2).

Early Recognition of BDI

Type A

Most Type A injuries will present as a bile leak. It does appear that there has been an increase in the occurrence of bile leaks with LC [73, 74]. Occasionally bile leaks will be diagnosed intraoperatively but the majority will present within 24h of the operation [75, 76].

The presentation of bile leaks due to a Type A injury and to some extent their subsequent management is dependent on whether a drain was inserted at the time of LC. In those patients who have a drain there may be bile in the drain immediately post operatively (Fig. 23.11).

Where no drain has been placed, the immediate post-operative bile leak presents as severe post-operative pain or a patient that does no progress well after surgery. Any patient that is not well and able to be discharged within 24h of surgery or has severe abdominal pain should be considered to have a bile leak until proven otherwise. Delayed recognition and failure to manage

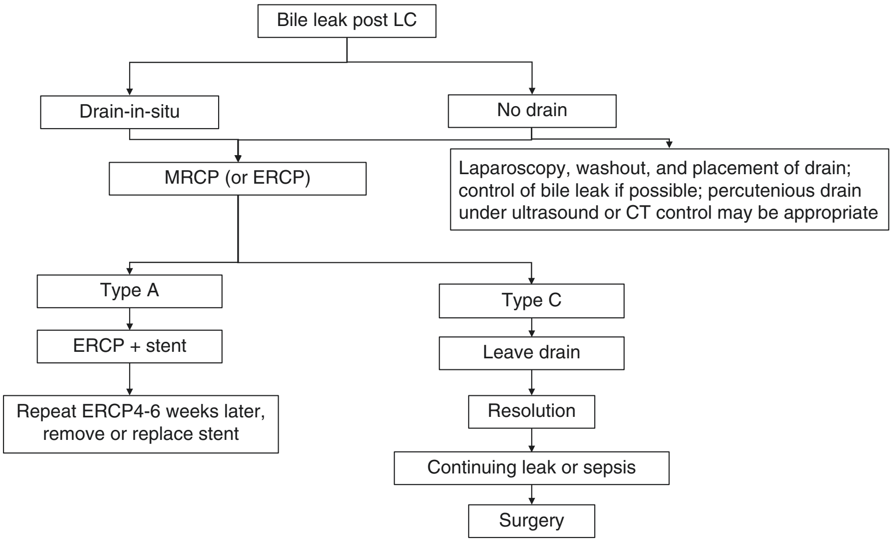

Fig. 23.2 Algorithm for the management of bile leak post LC (Strassberg Types A, C)

promptly results in the development of sepsis and poor outcomes. The investigation and treatment of Type A injuries and associated biliary peritonitis is discussed in Chap. 22 (Figs. 22.1 and 23.2).

Type B injury

Variations in biliary anatomy, particularly of the right hepatic duct system (Chap. 1) is a known risk factor in LC. There may be aberrant anatomy in up to 24% of patients undergoing LC [77]. Anomalous drainage of the right posterior sectoral bile duct into either the cystic duct or common hepatic duct (Figs. 1.9b and 1.9e) is one of the most common aberrations and occurs in 2–5% of patients [78–80] (Chap. 1). This injury is not covered by the Bismuth classification but is a type B Strasberg injury when it is occluded without leakage, usually by clips. Note these patients do not present with a bile leak as it involves occlusion of the sectorial duct system.

Not all of these injuries are diagnosed early after the LC. As they do not present with a bile leak or jaundice they may not be detected for a prolonged period of time resulting in atrophy of segments 6 and 7. Some patients, however, do present with vague symptoms with abnormal cholestatic liver function tests or recurrent episodes of cholangitis [81–83]. In the past it was not uncommon for these injuries to be misdiagnosed due to misinterpretation of a “normal” ERCP although in specialist centres with MRCP the diagnosis should not be missed. In the author’s experience PTC is rarely required for diagnosis or treatment.

In the past, surgery was often advocated for the treatment of these injuries to prevent the development of cholangitis or abscess formation [79, 80, 83]. More recently, however, management has evolved with non operative management of this type of injury the preferred initial approach [84]. Operative management which may involve liver resection should be reserved for patients who develop infective complications (cholangitis or liver abscess).

Type C Injury

Type C injuries are more problematic than Type B injuries because the risk of late diagnosis with infected bile collections is more common. Altogether, injuries to the right posterior sectoral duct during LC, either with occlusion or division, make up about 11% of all BDI associated with LC [85]. If the injury is recognised intraoperatively, and there is specialist HPB assistance available, consideration should be given to immediate reconstruction with a roux-en-y loop. The size of the right posterior sectoral duct is an important determinant in this decision and generally if the duct is less than about 2 mm, the risk of subsequent stricturing is high and it may be a disadvantage to try and repair a small duct due to the possible introduction of bacteria. There is limited data available on the long term outcome of these repaired cases but it would appear that at least one third of cases do develop strictures [83, 86]. In the circumstance where no specialist HPB assistance is available, it is preferable to place a drain and transfer the patient to a specialist centre. In the situation where there is specialist assessment available and the RPSD is too small for reconstruction, then ligation to convert the injury to a Type B injury is appropriate.

Most of these injuries are not recognised intra-operatively and present with bile in the drain (Fig. 22.12) or biliary peritonitis. The initial management of biliary peritonitis when diagnosed early is discussed in Chap. 22. At laparoscopy if a sector duct injury is suspected only a lavage and drain should be performed, unless the surgeon is an experienced HPB surgeon. Where the diagnosis of biliary peritonitis is delayed and the patient resents with sepsis and established collections the most pressing problem is treatment of sepsis with appropriate antibiotics and adequate drainage of bilomas which can be accomplished with the placement of percutaneous drains after CT assessment. Once sepsis and the bile leak are controlled the next step is appropriate imaging with MRCP/ERCP to delineate the injury and the placement of an endoscopic stent to decrease the biliary drainage from the distal portion of the injury. Often with this treatment the biliary fistula can be managed non-operatively in the first instance and some of these fistulas will seal with corresponding atrophy of the liver segments. If the patient is well, it may be reasonable to wait up to 8 weeks with this conservative approach [84] for the fistula to close (Fig. 23.2). This is more likely if the initial diagnosis is made early, the bile peritonitis treated before sepsis is established. If at the end of this period the biliary fistula has not closed, then an operative approach with either delayed reconstruction, ligation or, rarely, liver resection can be performed.

Type D Injury

The critical issues in deciding the appropriate treatment for this type of injury are whether there has been any associated thermal injury to the biliary tree, the involved circumference of the bile duct and the diameter of the bile duct. Clearly if there have been significant thermal injury conservative measures are likely to fail early or result in subsequent stricturing. If the injury is recognised intra-operatively and if the involved diameter of the injury is no more than 25%, there is no loss of ductal tissue, no thermal injury and the diameter of the bile duct is at least 4 mm, then primary repair at the time is indicated. The repair may take the form of either primary suture with 5/0 or 6/0 PDS sutures if the defect is very small, or more commonly a repair over a fine bore T-tube or the placement of an antigrade biliary stent across the ampulla. The procedure can be done laparoscopically but is more frequently done with an open procedure. In the circumstance where there has been significant damage to the bile duct with probable thermal injury, then an hepatico-jejunostomy (HCJ) is indicated. If the injury is diagnosed at some time after the index LC, then an endoscopic approach may be feasible depending on the circumstances.

It is generally accepted that there is little place for an end-to-end repair over a T-tube because of the 50–60% incidence of late stricture formation [41, 87]. However, there is now extensive experience in end-to-end biliary anastomoses in liver transplantation [88, 89]. One of the advantages of this technique is that it facilitates endoscopic access and dilatation if it is required. Additionally, there may be a disadvantage of HCJ in that it is less physiologic and may be associated with introduction of bacteria into the biliary tract, malabsorption and impaired ability to gain weight [90, 91]. However, end-to-end repair is contraindicated when the bile duct is less than 4 mm in diameter, where the loss of circumference is more than 30% of the bile duct diameter, where the injury is located less than 2 cm from the ductal confluence, where there is a thermal component to the injury and where there is an associated vascular injury [92–94]. It requires specialist HPB experience to determine the appropriate surgical repair and in practical terms is rarely used.

Type E Injury

Strasberg type E injuries are the most difficult injuries to deal with and are generally associated with the poor long term results. In these injuries there is a complete transection of the bile duct and/or the hepatic ducts. Less than a third of these serious injuries are recognised intra-operatively [15, 17, 21, 95]. This is important as there is strong evidence that intra-operative diagnosis and immediate management is associated with better early and long term outcomes and the avoidance of post-operative complications such as bilomas, cholangitis and strictures. Bilomas and cholangitis are associated with the development of sepsis which is the major cause of mortality in this group [41, 96, 97]. As previously stated there is evidence that routine IOC may be associated with a higher rate of intra-operative diagnosis and less severe injuries [65, 67–70]. If the injury is recognised and there is specialist HPB assistance available, then the injury should be repaired immediately. The principles are outlined in the algorithm in Fig. 23.3. In the circumstance where there is no assistance available, then it is appropriate that no further dissection occur, a drain be placed and the patient transferred to a specialist unit. Some units are able to offer an outreach service for immediate repair of intra-operatively recognised BDI [98].

The majority of these injuries present post-operatively with either biliary peritonitis, bile in the drain or obstructive jaundice. The management of biliary peritonitis is as discussed in Chap. 22.

Unfortunately, type E injuries are often associated with vascular injuries which can influence the outcomes of repair. The most common associated vascular injury is when the right hepatic artery (RHA) is damaged at the time of the bile duct injury. This accounts for 90% of vasculobiliary injuries and is probably because of its proximity to the common hepatic duct (Figs. 1.14a and 1.15) [93, 99, 100]. The RHA may be clipped or diathermied during dissection if it is mistaken for the cystic artery [37]. Additionally, it may be inadvertently damaged if there is bleeding. The RHA is probably damaged more frequently than appreciated and may be asymptomatic. In an autopsy study following open cholecystectomy, it was shown that the RHA was injured in 7% of cases [101]. Likewise, in the era of open cholecystectomy it was shown that the incidence of hepatic arterial injury associated with a BDI was 13.8% [102]. In the era of LC it would appear that the incidence is similar with about 12–17% of patients with a BDI having an injury to the RHA [4, 103]. However, these figures may understate the true incidence and in the circumstance where routine angiography is performed in patients with BDI following LC the incidence has been reported as 47% [104].

Isolated RHA occlusion is rarely associated with significant hepatic ischaemia because of the portal flow and collateral arterial supply. When it is associated with a BDI, the morbidity may be increased. This may be due to the loss of collateral arterial supply via the marginal arteries along the bile duct and the hilar marginal artery (Chap. 1, Fig. 1.15) [93, 104–108]. As estimated by Strasberg et al. the incidence of liver infarction in patients with a RHA/BDI is probably about 10% and this tends to occur

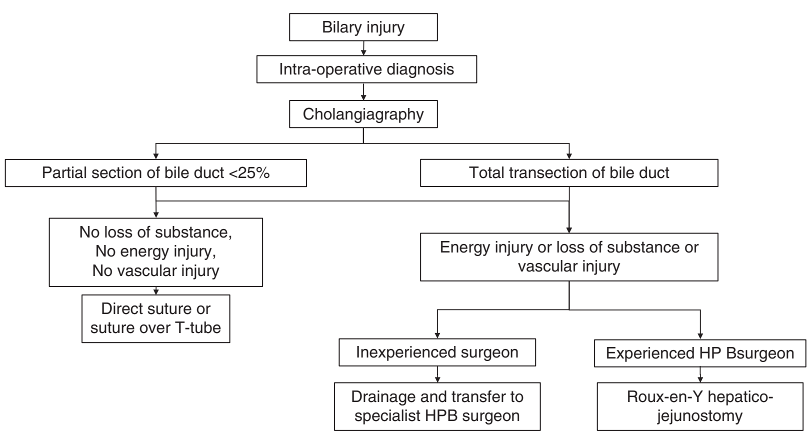

Fig. 23.3 Algorithm for the management of biliary injury diagnosed intra-operatively

slowly over some weeks [93]. Most of these patients have been treated with liver resection [37, 103, 109, 110].

The effect of a RHA injury on the subsequent outcome of a biliary reconstruction is controversial. It has been suggested that the incidence of stricturing and subsequent failure of the bilioenteric anastomosis is higher with an associated RHA injury due to ischaemia and may be up to 50–60% [111–113]. However, there are two larger contemporary series examining this issue and neither found a worse outcome with biliary reconstruction with an associated RHA injury. Stewart et al. [37] studied a series of 84 patients with combined BDI/RHA injuries and found no difference in the long term success of biliary reconstruction although the incidence of hepatic ischaemia was 11% with 5% of the patients requiring a liver resection. Similarly, Alves et al. in a series of 55 patients found no difference in the short or long term outcome of biliary reconstruction in patients with a combined RHA/BDI [104]. Using a Hepp-Couinaud reconstruction (Fig. 23.4) with the left hepatic duct may be the reason that there were less ischaemic strictures in these two series and a 93% overall success rate was achieved.

Other vascular injuries during LC involving the portal vein with or without the common hepatic artery are rare. Sixteen cases were identified by Strasberg et al. in their review [93]. Half of these patients developed rapid hepatic necrosis and liver failure requiring resection or transplantation and 50% of these patients died. It is likely that although these cases are rare they are underreported. Another study by Strasburg et al. suggested that these “extreme vasculobiliary injuries” may be more common in the situation where a severely inflamed gallbladder is removed at open operation by a fundus-down approach (Chaps. 1, 16 and 19) [114].

The consequence of all of the above, however, is that an associated vascular injury must be suspected in every case of a Type E injury. Duplex

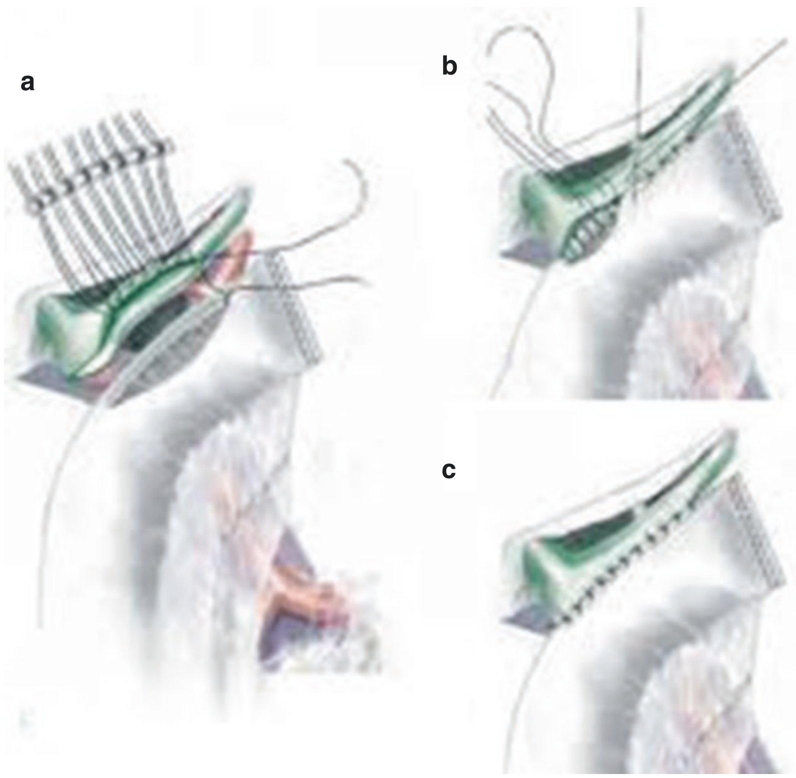

Fig. 23.4 Hepp-Couinard reconstruction. (**a**) The bile ducts are opened from the confluence along the left hepatic duct after this hepatic plate is lowered. The sutures held upward are pre-paced in the anterior wall prior to suturing the posterior wall. (**b**) closing the anterior wall. (**c**) completed anastomosis

ultrasonography will give some information regarding the blood flow to the liver but is unreliable in diagnosing specific injuries [112]. Although coeliac angiography may be the gold standard, CT or MR angiography will normally provide an appropriate assessment.

It is controversial as to whether an associated RHA should be repaired and whether this influences the eventual outcome of the biliary repair. The artery may be injured in different ways: transection, occlusion by clips or thrombosis. There are reports of successful biliary repair without reconstruction of the associated RHA injury [37, 104, 115] but certainly, proximal damage to the biliary tree associated with a concomitant arterial injury may be associated with biliary ischaemia and necrosis [109, 111, 115]. Li et al., adapted a policy of routine arterial reconstruction when possible in their series of 10 patients with RHA injury in a total series of 60 patients with BDI. They were only able to repair the artery in five patients and significantly, three of the ten patients died [103].

Timing of Repair

The timing of operative repair for these injuries has been a source of controversy. It is generally agreed that the best results are achieved with early recognition and early repair by an experienced HPB surgeon [97, 116, 117] particularly when it involves a complex more proximal injury [118]. Only 17% of repairs performed by the primary surgeon were successful [87]. Additionally, in a large series of BDI reported by Thomson et al., 74% of the patients who had undergone repair by the primary surgeon required further surgery [119]. The fact that many older series included repairs by both specialist HPB and non-specialist surgeons may have skewed the results, and may be the reason that early repairs have been reported to have poorer outcomes [120, 121]. Generally, in more recent series, which may involve earlier referral to tertiary centres, there is no advantage in delaying repair in most patients and good results can be achieved [97, 118, 119, 122, 123].

More importantly than the timing of repair is the preparation of the patient for surgery. In the patient where there has be a significant delay in diagnosis and where there are undrained bile collections with surrounding inflammation and infection, early biliary repair may be associated with more complications and poorer long term outcomes [124]. Undrained bile collections can become infected after only a few days [87]. It is important that all intra-abdominal collections are drained by CT or US guided percutaneous drains to control infection and inflammation. Generally, in these patients proximal drainage with a percutaneous trans-hepatic tube is indicated and helps to control the intra-abdominal infection and inflammation by decreasing the bile leak [125]. The definitive repair in patients presenting with a delayed diagnosis and established sepsis should be delayed until there has been a period of normalisation of the inflammatory process.

Patients presenting with a controlled bile leak usually will not have sepsis and can be repaired early. Similarly, patients presenting with jaundice and no bile leak can have the definitive repair early as sepsis is usually not present. If there is cholangitis this can be managed with a PTC. Unfortunately, most patients present with a leak and sepsis.

As pointed out by Connor et al. [15] delayed diagnosis of a major BDI may be associated with significant nutritional problems. It may precipitate systemic inflammatory response and a low serum albumin which is associated with poorer operative outcomes [126–128]. Also, prolonged periods of biliary-enteric disconnection are associated with impaired response and predisposition to infection [129]. In these types of patients, clinical judgement as to when repair should be undertaken is paramount.

Technique of Surgical Repair

Where there has been a complete transection of the CHD/CBD, it is important to realise that this involves an interruption to the anastomotic blood supply to the biliary tract. This is particularly the case with proximal injuries. As previously noted RHA injuries are commonly seen in major BDI but even if the RHA is intact there is impaired blood supply. This relates to the division of the marginal arteries which ascend from the postero-superior pancreatico-duodenal arteries (Fig. 1.15) These arise from the gastroduodenal artery and form a rich plexus of blood supply around the biliary tract [107]. Previous reports of end to end bile duct repair have shown a re-stricture rate of 100% and this is mainly due to ischaemia [87]. Although good results were reported by Sir Rodney Smith with his mucosal graft procedure in the 1970s [130], there is really little place for this technique in contemporary practice.

A successful repair requires that the anastomosis is as wide as possible, tension free, well vascularised and performed with fine sutures which are absorbable and create minimal reaction [131]. It is important that all ischaemic and necrotic tissue be debrided back to a healthy duct. In practice, for injuries where the biliary confluence is intact (Strasberg E1–3 injuries), this usually requires an anastomosis as proximal as possible The best results will usually involve an HCJ using the Hepp-Couinaud technique (Fig. 23.4) [132]. In most cases of major BDI, the biliary tract is not dilated nor fibrosed from obstruction and this may involve a difficult anastomosis to a thin walled and narrow bile duct. To improve this situation Hepp described the technique of dropping the hilar plate to expose the left hepatic duct in its extra hepatic course at the base of segment 4 aided by the anatomical studies of Couinaud [133] (Fig. 23.4). In this way, the left hepatic duct can be opened longitudinally usually for a distance of 2–3 cm from the confluence to allow a side-to-side anastomosis using a Roux-en-Y loop. It is important that the left hepatic dochotomy not be taken too far to the left as the segment 4 artery usually passes anterior to the left hepatic duct from the left hepatic artery and may be injured. As stated by Strasberg et al., this usually means that the extent of the dochotomy to the left is just to the right of the umbilical fissure [131]. Similarly, the segment 4 duct may enter close to the confluence and must be avoided [134]. Generally, the incision in the left hepatic duct is placed anteriorly and posterior dissection is avoided to prevent bleeding. The anastomosis is then done using a single layer of interrupted or continuous fine sutures (such as 5/0 PDS) under magnification (Fig. 23.4). It is often helpful to secure the Roux-en-Y loop to the liver to prevent twisting and to decrease the tension on the anastomosis.

Occasionally, access will still be problematic and this can be improved by a partial resection of segments 5 and 4b [135]. Where the confluence is intact it is required in a minority of circumstances [131, 136]. However, in circumstances where the right and left biliary systems are separated (Bismuth IV, Strasberg E 4 and 5), it is more commonly used to facilitate identification and mobilisation of the RHD particularly. The results of reconstruction where the confluence is preserved are superior to those when the confluence is disconnected [137]. There are essentially three options of how to reconstruct these injuries with loss of the biliary confluence:

– construction of a neoconfluence with a Roux-en-Y hepatico-jejunostomy,
– a double anastomosis to a Roux-en-Y hepatico-jejunostomy, or
– a porto-enterostomy (modified Kasai procedure) [138].

The use of stents is controversial but in the author's opinion are rarely required. Liver resection must be considered in circumstances where there is a major vascular injury and bad quality major ducts which may be related to diathermy injury. This is probably indicated in no more than 5–15% of injuries and may be indicated soon after the BDI, most typically if the RHD is non reconstructable and there is a major vascular injury. It may also be indicated later if there is recurrent cholangitis or intrahepatic bile duct strictures. In highly selected cases excellent long term results may be achieved although there is significant post-operative morbidity [139–143].

Management of Late Presentation of a Biliary Stricture

An unknown proportion of patients will present months to years following LC with abnormal liver functions tests, cholangitis and/or jaundice and will be found to have a biliary stenosis. This may be on the basis of a thermal injury with late stricture formation, a poorly applied clip narrowing the bile duct, or ischaemia related to excessive or unnecessary dissection of the bile duct. A proportion of these patients will also have a history of bile leakage which may have exacerbated the inflammatory response and subsequent stricturing process.

Over the last 20 years, endoscopic treatment of these strictures (Fig. 23.5) has virtually replaced initial surgical treatment (Fig. 23.6) which was previously regarded as the gold standard [144–146]. The endoscopic approach during the early 1990s was to insert one or two stents and change them every 3 months for 1 year [147–149]. However, this changed to treat these injuries more aggressively with multiple stents to hold the strictured segment fully open and 3 monthly changes [150, 151]. There is evidence to support the idea that initial insertion of multiple stents has a lower rate of obstruction and may decrease the need for ERCP procedures [152]. More recently, stricture dilatation and use of self-expanding, fully covered, removable metal stents have been used to treat biliary strictures from a variety of causes with good results [153, 154] (Fig. 23.6).

It is important that these injuries be managed in large specialised centres with access to multidisciplinary teams. In a large series from the Netherlands that included 500 patients referred with BDI between 1990 and 2005 there were 110 patients with a bile duct stricture. The patients were referred at a median of 75 days' post LC (range 4–2899). These patients were treated with progressive balloon dilatation and stenting. After a median duration of stenting/dilatation of 15 months (range 8–21), 20% of patients were judged to have failed treatment and were referred for definitive surgery [144].

It would appear that endoscopic treatment will be successful in 80–90% of cases with rates of cholangitis less than 10% [155, 156]. De Reuver et al. identified a number of factors that could be predictive of a successful outcome: no previous stenting, the injury below the biliary confluence and an increasing number of stents inserted at the first procedure [144]. It does appear that if treatment is successful, there are excellent long term results with a low recurrence rate [144, 149].

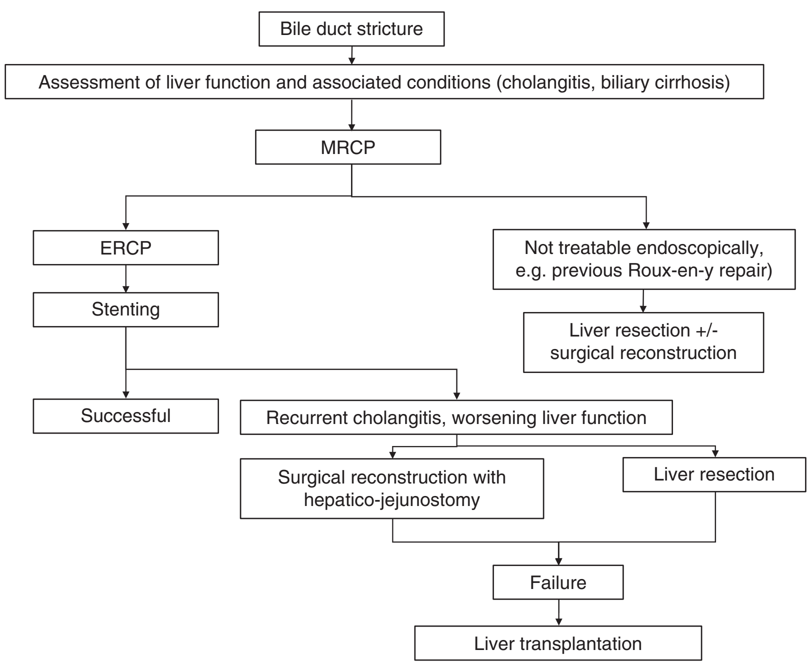

Fig. 23.5 Algorithm for the management of biliary structure with delayed diagnosis

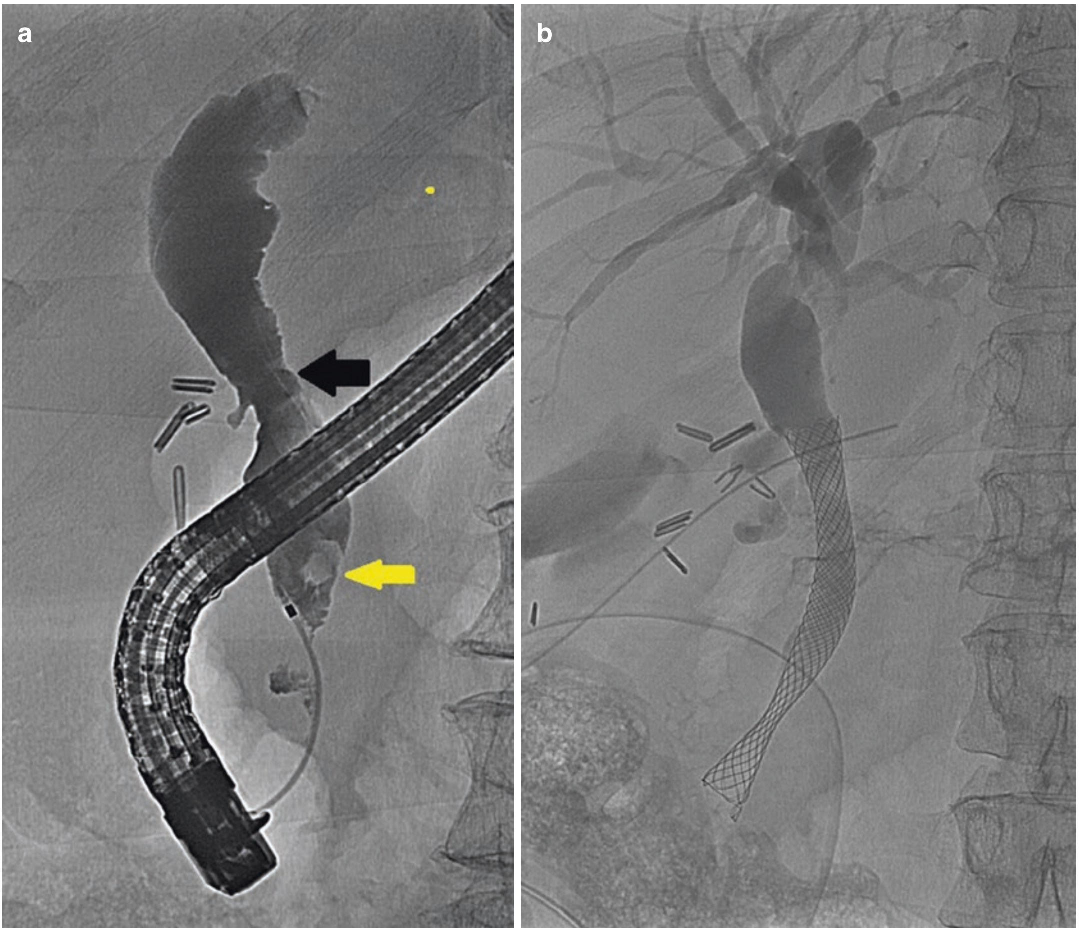

Fig. 23.6 A 58 year old man had a "difficult" subtotal cholecystectomy for severe acute cholecystitis. He had a bile leak managed expectantly that dried up after 6 days. He presented 4 months later with cholangitis. (**a**) The ERCP demonstrated a distal stone (yellow *arrow*) and a slight stricture of the mid CBD. The stone was removed after a sphincterotomy. He represented 18 months later with a further episode of cholangitis. The repeat ERCP demonstrated no stones but a tight stricture. (**b**) This was dilated and an expandable metal stent inserted. This was removed 8 months later and he has been well for 4 years

Long Term Results of Surgical Repair

A number of prognostic factors help determine the outcomes of surgical repair of major BDI. The factors which may negatively influence the outcome of surgical repair include:

- the proximal extent of the injury particularly with loss of the biliary confluence (Strasberg E4) [87, 121, 124, 137],
- delayed referral to a specialist centre, and
- the performance of therapeutic interventions prior to transfer [97, 121].

There is good evidence that repair of BDIs should be done in specialised centres by specialist HPB surgeons. Dr. Way's group showed that in their experience a successful outcome was achieved in 94% of cases, whereas a repair performed by the primary surgeon was only successful in 17% of the patients [87]. A more recent report from the same group showed a success rate of only 21% if the repair was done by the primary surgeon [118]. Perera et al. showed that repairs done by non-specialist HPB surgeons were associated with a statistically significantly higher rate of recurrent cholangitis, re-stricturing, morbidity and the need for an additional surgical procedure [97]. Additionally, repair by a non-specialist HPB surgeon is associated with an increased death rate [8, 161].

The presence of sepsis at the time of repair is associated with increased morbidity and, as previously stated, it is important that sepsis is controlled and treated prior to repair [118]. Sulpice et al. examined 38 major BDIs referred to their centre between 1992 and 2010 and found that amongst 17 variables examined, sepsis and biliary cirrhosis were correlated with major morbidity after operative repair [158].

An anastomotic stricture that develops following surgical repair may occur up to 15 years following surgery [136, 159], but about a third will occur within the first 3 years [160]. In more modern series, the incidence of post-operative stricture formation ranges from 6% to 17% [124]. It would appear that in most specialist centres the incidence of stricture formation post-operatively is 10–15% [122, 158]. Most of these strictures can be managed with a combination of radiologic and endoscopic management. Anastomotic strictures can lead to chronic biliary obstruction and cholangitis. If this is recurrent it can lead to secondary sclerosing cholangitis [161], with the formation of intrahepatic stones, abscesses and further stricture formation which may require liver resection [140, 162].

One of the most feared long term complications of a major BDI is the development of secondary biliary cirrhosis (SBC) with associated portal hypertension. This more commonly develops in patients who have a history of repeated bouts of cholangitis, with multiple interventions and who are referred late to specialist centres [163]. Generally, the prolonged biliary obstruction is associated with the development of severe fibrosis and subsequent cirrhosis which then proceeds to portal hypertension but may occur prior to the presence of cirrhosis [164]. The duration of chronic biliary obstruction resulting in SBC ranges from 15 to 62 months with an average of about 4 years [162, 165, 166]. The incidence of chronic liver disease resulting from a major BDI is uncertain and may be overestimated as they tend to be referred to specialist centres. The incidence in the literature ranges from a low of 6% up to about 25% [122, 124, 162, 163]. Portal hypertension may be associated with a benign bile duct stricture in 15–25% of patients and is associated with significant morbidity and mortality [159, 166, 167].

Liver transplantation is rarely required as a consequence of a major BDI. Sulpice et al. [158] reported that 2 of their 38 (5.3%) referred patients required liver transplantation, whereas Schmidt et al. [124] reported that 2 of 54 (3.7%) patients required liver transplantation at 12 and 36 months following initial surgical repair.

Long Term Health Consequences of a BDI

There remains controversy regarding the long term health effects of a BDI even if it is repaired successfully. This reflects the subjectivity of how quality of life (QOL) is measured and how it may be affected by population assessment, age, gender and ethnicity [169, 170]. Controversy currently exists over the type of QOL assessment and indeed the appropriate response rate to ensure accuracy. As previously stated there is often a significant delay in definitive treatment and the morbidity rate is high. These factors would tend to support the concern that there is a significant diminution in QOL after a major BDI. Sicklick et al. [96] found that the mean time from BDI to definitive treatment was 42 weeks. Multiple studies have documented morbidity rates of up to 50% following definitive management [71, 96, 121, 124, 171] and it would not be surprising if there were a significant diminution in QOL.

Boerma et al. initially looked at 106 patients referred with a BDI sustained during LC and found a significantly decreased QOL both mentally and physically at a median follow up time of 70 months [172]. However, there were some significant problems with the design of this study and it included a high proportion of minor bile leaks and a control group which was not strictly comparable [170, 173]. Only 25 patients underwent a surgical HCJ and the rest were treated endoscopically which suggests much less severe injuries. Only duration of treatment was associated with a poorer mental QOL on multivariate analysis.

It does appear that physical QOL metrics improve the longer the time from definitive treatment, and may return to that of a control population some years following treatment [174]. Mental QOL metrics however do appear to be affected in the long term compared to physical QOL metrics. Melton et al. [175] reported on 54 patients with BDI and found similar physical and social QOL but impaired mental QOL compared to a control group of patients undergoing an uncomplicated LC. De Reuver et al. [176, 177] assessed 403 patients and reported a decreased QOL compared to a control group undergoing uncomplicated LC and the difference did not improve with longer follow up.

There has been one meta-analysis on the long term effect of BDI on health related QOL. This looked at 6 studies and concluded that after controlling for follow up time, patients who had sustained a BDI were more likely to have reduced mental QOL (OR=38.42, 95% CI: 19.14–77.10; $p<0.001$) but not physical, compared to uncomplicated LC patients [178]. A number of authors have also noted that patients who are involved in litigation related to their BDI may have impaired physical and mental QOL metrics [175, 177].

Finally, in a large group of 167 patients followed up by the Hopkins group for a median of 169 months (interquartile range 125–222 months), it was shown that symptoms of depression and low energy improved significantly after definitive surgical repair ($p < 0.05$). In the longer term however, mental health related QOL problems were more common. They concluded optimal multi-disciplinary management of BDI patients could restore patients to pre-injury levels of QOL [179].

Litigation

Litigation following a BDI is potentially an extremely stressful event for both the patient and the surgeon. It does appear that the rate of litigation following LC has increased to that which was present in the OC era. This may reflect a number of factors including: a higher rate of BDI in the laparoscopic era, increased patient expectations, easier access to legal advice and possibly increased advertisement of plaintiff legal services as well as cases being taken on a no-win no-fee basis. In the United States civil law courts, over a 21 year period, from 1970 to 1991, Kern identified 68 cases of BDI of which 49 were from OC and found that the settlement payout was approximately US$500,000 in successfully litigated cases versus US$250,000 in settled cases [180]. It does appear that there has been an increase in litigation related to BDI shortly after the introduction of LC and that the settlement amount increased [181].

It remains uncertain what percentage of BDI result in litigation. The percentage also varies between countries. For example, in the United States, the reported incidence may be increasing and up to 70% of patients with a BDI will resort to litigation [179]. A comparable study from the Netherlands reported an incidence of litigation of 31% [177].

In the United Kingdom, figures are easier to obtain as the majority of cases are perpetrated in the National Health Service (NHS) and are defended by the National Health Service Litigation Authority. Since 2002, all NHS hospitals have been required to report claims of negligence. Between 1995 and 2009, 418 claims related to LC were made and of these 43% were related to a BDI. Overall, the success rate of the claims was 65% and the average payout was US$168,337 which is about 25% of the average payout in the United States. In the United Kingdom, the most common complaints related to intra-operative error and delay in diagnosis [182]. Similarly, Perera et al. [183] reported that one third of patients with major BDI would resort to litigation and that the predictors of litigation included age <52 years ($p=0.03$), associated vascular injury ($p=0.014$), immediate non specialist repair ($p=0.009$) and incomplete recovery ($p=0.017$).

In Australia, the incidence of litigation following a BDI is unknown but it appears that the majority of cases are settled without going to court. The author is not aware of a case of BDI successfully defended in court in Australia. In a recent Australian case of BDI (*Belokozovski v Magary* [2014] NSWDC 5), the court found a surgeon negligent for not having performed an operative cholangiogram.

In the author's opinion there may be examples of BDI that could be successfully defended. This is reflected in the author's experience where a minority of cases transferred for specialist repair have resulted in litigation. One of the problems in a successful defense would be the selection process of expert witnesses. Although there is a long tradition in using expert witnesses in western common law, their reliability is not well established and there are certainly instances where the experts would not be judged as such by their surgical peers [184, 185]. There are also many reasons for a lack of agreement amongst experts which include hindsight bias and attitudes of other practitioners towards their colleagues [186–188].

Conclusion

BDI remains a serious complication of LC. All efforts should be made to avoid this complication due to the significant associated morbidity and mortality. In the circumstance where they occur early, transfer to a specialist multidisciplinary HPB service is required for the best results to be achieved. I have included three algorithms setting out the approach to the clinical problem below.

Key Points

- Bile duct injury continues to occur at an unacceptable rate following laparoscopic cholecystectomy.
- Early recognition and management in a multidisciplinary HPB service will produce the best results.
- In those cases of bile duct transection requiring operative intervention, Hepp-Couinaud hepatico-jejunostomy will produce the best results.
- Mortality of a major bile duct transection requiring operative intervention is significantly greater compared to a routine laparoscopic cholecystectomy.

The long term health consequences of a bile duct injury may be significant.

References

1. Perissat J, Collet DR, Belliard R. Gallstones: laparoscopic treatment, intracorporeal lithotripsy followed by cholecystostomy or cholecystectomy—a personal technique. Endoscopy. 1989;21(Suppl 1):373–4.
2. Dubois F, Icard P, Berthelot G, Levard H. Coelioscopic cholecystectomy. Preliminary report of 36 cases. Ann Surg. 1990;211(1):60–2.
3. Reddick EJ, Olsen DO. Laparoscopic laser cholecystectomy. A comparison with mini-lap cholecystectomy. Surg Endosc. 1989;3(3):131–3.
4. Deziel DJ, Millikan KW, Economou SG, Doolas A, Ko ST, Airan MC. Complications of laparoscopic cholecystectomy: a national survey of 4,292 hospitals and an analysis of 77,604 cases. Am J Surg. 1993;165(1):9–14.
5. Lee VS, Chari RS, Cucchiaro G, Meyers WC. Complications of laparoscopic cholecystectomy. Am J Surg. 1993;165(4):527–32.
6. Richardson AJ, Brancatisano R, Avramovic J, Roney W, Little JM. Injuries to the bile duct resulting from laparoscopic cholecystectomy. Aust N Z J Surg. 1993;63(9):684–9.
7. Adamsen S, Hansen OH, Funch-Jensen P, Schulze S, Stage JG, Wara P. Bile duct injury during laparoscopic cholecystectomy: a prospective nationwide series. J Am Coll Surg. 1997;184(6):571–8.
8. Flum DR, Cheadle A, Prela C, Dellinger EP, Chan L. Bile duct injury during cholecystectomy and survival in medicare beneficiaries. JAMA. 2003;290(16):2168–73.
9. Cagir B, Rangraj M, Maffuci L, Herz BL. The learning curve for laparoscopic cholecystectomy. J Laparoendosc Surg. 1994;4(6):419–27.
10. Moore MJ, Bennett CL. The learning curve for laparoscopic cholecystectomy. The Southern Surgeons Club. Am J Surg. 1995;170(1):55–9.
11. Voitk AJ, Tsao SG, Ignatius S. The tail of the learning curve for laparoscopic cholecystectomy. Am J Surg. 2001;182(3):250–3.
12. Hobbs MS, Mai Q, Knuiman MW, Fletcher DR, Ridout SC. Surgeon experience and trends in intraoperative complications in laparoscopic cholecystectomy. Br J Surg. 2006;93(7):844–53.
13. Archer SB, Brown DW, Smith CD, Branum GD, Hunter JG. Bile duct injury during laparoscopic cholecystectomy: results of a national survey. Ann Surg. 2001;234(4):549–58; discussion 58–9.
14. Francoeur JR, Wiseman K, Buczkowski AK, Chung SW, Scudamore CH. Surgeons' anonymous response after bile duct injury during cholecystectomy. Am J Surg. 2003;185(5):468–75.

15. Connor S, Garden OJ. Bile duct injury in the era of laparoscopic cholecystectomy. Br J Surg. 2006;93(2):158–68.
16. Morgenstern L, McGrath MF, Carroll BJ, Paz-Partlow M, Berci G. Continuing hazards of the learning curve in laparoscopic cholecystectomy. Am Surg. 1995;61(10):914–8.
17. Richardson MC, Bell G, Fullarton GM. Incidence and nature of bile duct injuries following laparoscopic cholecystectomy: an audit of 5913 cases. West of Scotland Laparoscopic Cholecystectomy Audit Group. Br J Surg. 1996;83(10):1356–60.
18. MacFadyen BV Jr, Vecchio R, Ricardo AE, Mathis CR. Bile duct injury after laparoscopic cholecystectomy. The United States experience. Surg Endosc. 1998;12(4):315–21.
19. Waage A, Nilsson M. Iatrogenic bile duct injury: a population-based study of 152 776 cholecystectomies in the Swedish Inpatient Registry. Arch Surg. 2006;141(12):1207–13.
20. Rystedt J, Lindell G, Montgomery A. Bile Duct Injuries Associated With 55,134 Cholecystectomies: Treatment and Outcome from a National Perspective. World J Surg. 2016;40:73–80.
21. Karvonen J, Salminen P, Gronroos JM. Bile duct injuries during open and laparoscopic cholecystectomy in the laparoscopic era: alarming trends. Surg Endosc. 2011;25(9):2906–10.
22. Harboe KM, Bardram L. The quality of cholecystectomy in Denmark: outcome and risk factors for 20,307 patients from the national database. Surg Endosc. 2011;25(5):1630–41.
23. Chuang KI, Corley D, Postlethwaite DA, Merchant M, Harris HW. Does increased experience with laparoscopic cholecystectomy yield more complex bile duct injuries? Am J Surg. 2012;203(4):480–7.
24. Worth PJ, Kaur T, Diggs BS, Sheppard BC, Hunter JG, Dolan JP. Major bile duct injury requiring operative reconstruction after laparoscopic cholecystectomy: a follow-on study. Surg Endosc. 2015;30(5):1839–46.
25. Joseph M, Phillips MR, Farrell TM, Rupp CC. Single incision laparoscopic cholecystectomy is associated with a higher bile duct injury rate: a review and a word of caution. Ann Surg. 2012;256(1):1–6.
26. Strasberg SM. Single incision laparoscopic cholecystectomy and the introduction of innovative surgical procedures. Ann Surg. 2012;256(1):7–9.
27. Cheah SW, Yuan S, Mackay S, Grigg M. Single incision laparoscopic cholecystectomy is associated with a higher bile duct injury rate: a review and word of caution. Ann Surg. 2015;261(2):e54.
28. Khan MH, Howard TJ, Fogel EL, Sherman S, McHenry L, Watkins JL, et al. Frequency of biliary complications after laparoscopic cholecystectomy detected by ERCP: experience at a large tertiary referral center. Gastrointest Endosc. 2007;65(2):247–52.
29. Walsh RM, Henderson JM, Vogt DP, Mayes JT, Grundfest-Broniatowski S, Gagner M, et al. Trends in bile duct injuries from laparoscopic cholecystectomy. J Gastrointest Surg. 1998;2(5):458–62.
30. Windsor JA, Pong J. Laparoscopic biliary injury: more than a learning curve problem. Aust N Z J Surg. 1998;68(3):186–9.
31. Sedlack JD. The utilization of six sigma and statistical process control techniques in surgical quality improvement. J Healthc Qual. 2010;32(6):18–26.
32. Pyzdek T. The Six Sigma handbook. 2nd ed. New York, NY: McGraw Hill and Co.; 2003.
33. Bismuth H. In: Blumgart LH, editor. Postoperative strictures of the bile ducts. New York, NY: Churchill-Livingstone; 1982.
34. Bismuth H, Majno PE. Biliary strictures: classification based on the principles of surgical treatment. World J Surg. 2001;25(10):1241–4.
35. McMahon AJ, Fullarton G, Baxter JN, O'Dwyer PJ. Bile duct injury and bile leakage in laparoscopic cholecystectomy. Br J Surg. 1995;82(3):307–13.
36. Csendes A, Navarrete C, Burdiles P, Yarmuch J. Treatment of common bile duct injuries during laparoscopic cholecystectomy: endoscopic and surgical management. World J Surg. 2001;25(10):1346–51.
37. Stewart L, Robinson TN, Lee CM, Liu K, Whang K, Way LW. Right hepatic artery injury associated with laparoscopic bile duct injury: incidence, mechanism, and consequences. J Gastrointest Surg. 2004;8(5):523–30; discussion 30–1.
38. Bergman JJ, van den Brink GR, Rauws EA, de Wit L, Obertop H, Huibregtse K, et al. Treatment of bile duct lesions after laparoscopic cholecystectomy. Gut. 1996;38(1):141–7.
39. Neuhaus P, Schmidt SC, Hintze RE, Adler A, Veltzke W, Raakow R, et al. Classification and treatment of bile duct injuries after laparoscopic cholecystectomy. Chirurg. 2000;71(2):166–73.
40. Lau WY, Lai EC. Classification of iatrogenic bile duct injury. Hepatobiliary Pancreat Dis Int. 2007;6(5):459–63.
41. Strasberg SM, Hertl M, Soper NJ. An analysis of the problem of biliary injury during laparoscopic cholecystectomy. J Am Coll Surg. 1995;180(1):101–25.
42. Mercado MA, Dominguez I. Classification and management of bile duct injuries. World J Gastrointest Surg. 2011;3(4):43–8.
43. Bektas H, Schrem H, Winny M, Klempnauer J. Surgical treatment and outcome of iatrogenic bile duct lesions after cholecystectomy and the impact of different clinical classification systems. Br J Surg. 2007;94(9):1119–27.
44. Fingerhut A, Dziri C, Garden OJ, Gouma D, Millat B, Neugebauer E, et al. ATOM, the all-inclusive, nominal EAES classification of bile duct injuries during cholecystectomy. Surg Endosc. 2013;27(12):4608–19.
45. Krahenbuhl L, Sclabas G, Wente MN, Schafer M, Schlumpf R, Buchler MW. Incidence, risk factors, and prevention of biliary tract injuries during laparoscopic cholecystectomy in Switzerland. World J Surg. 2001;25(10):1325–30.
46. Olsen D. Bile duct injuries during laparoscopic cholecystectomy. Surg Endosc. 1997;11(2):133–8.

47. Way LW, Stewart L, Gantert W, Liu K, Lee CM, Whang K, et al. Causes and prevention of laparoscopic bile duct injuries: analysis of 252 cases from a human factors and cognitive psychology perspective. Ann Surg. 2003;237(4):460–9.
48. Dekker SW, Hugh TB. Laparoscopic bile duct injury: understanding the psychology and heuristics of the error. ANZ J Surg. 2008;78(12):1109–14.
49. Hugh TB. New strategies to prevent laparoscopic bile duct injury—surgeons can learn from pilots. Surgery. 2002;132(5):826–35.
50. Strasberg SM. Biliary injury in laparoscopic surgery: part 1. Processes used in determination of standard of care in misidentification injuries. J Am Coll Surg. 2005;201(4):598–603.
51. National Institutes of Health Consensus Development conference statement on gallstones and laparoscopic cholecystectomy. Am J Surg. 1993;165(4):390–8.
52. Troidl H. Disasters of endoscopic surgery and how to avoid them: error analysis. World J Surg. 1999;23(8):846–55.
53. Strasberg SM, Brunt LM. Rationale and use of the critical view of safety in laparoscopic cholecystectomy. J Am Coll Surg. 2010;211(1):132–8.
54. Buddingh KT, Nieuwenhuijs VB. The critical view of safety and routine intraoperative cholangiography complement each other as safety measures during cholecystectomy. J Gastrointest Surg. 2011;15(6):1069–70. author reply 71
55. Buddingh KT, Nieuwenhuijs VB, van Buuren L, Hulscher JB, de Jong JS, van Dam GM. Intraoperative assessment of biliary anatomy for prevention of bile duct injury: a review of current and future patient safety interventions. Surg Endosc. 2011;25(8):2449–61.
56. Buddingh KT, Morks AN, ten Cate Hoedemaker HO, Blaauw CB, van Dam GM, Ploeg RJ, et al. Documenting correct assessment of biliary anatomy during laparoscopic cholecystectomy. Surg Endosc. 2012;26(1):79–85.
57. Eikermann M, Siegel R, Broeders I, Dziri C, Fingerhut A, Gutt C, et al. Prevention and treatment of bile duct injuries during laparoscopic cholecystectomy: the clinical practice guidelines of the European Association for Endoscopic Surgery (EAES). Surg Endosc. 2012;26(11):3003–39.
58. Haynes AB, Weiser TG, Berry WR, Lipsitz SR, Breizat AH, Dellinger EP, et al. A surgical safety checklist to reduce morbidity and mortality in a global population. N Engl J Med. 2009;360(5):491–9.
59. Connor SJ, Perry W, Nathanson L, Hugh TB, Hugh TJ. Using a standardized method for laparoscopic cholecystectomy to create a concept operation-specific checklist. HPB (Oxford). 2014;16(5):422–9.
60. Giger U, Ouaissi M, Schmitz SF, Krahenbuhl S, Krahenbuhl L. Bile duct injury and use of cholangiography during laparoscopic cholecystectomy. Br J Surg. 2011;98(3):391–6.
61. Daly SC, Deziel DJ, Li X, Thaqi M, Millikan KW, Myers JA, et al. Current practices in biliary surgery: do we practice what we teach? Surg Endosc. 2016;30(8):3345–50.
62. Ragulin-Coyne E, Witkowski ER, Chau Z, Ng SC, Santry HP, Callery MP, et al. Is routine intraoperative cholangiogram necessary in the twenty-first century? A national view. J Gastrointest Surg. 2013;17(3):434–42.
63. Massarweh NN, Flum DR. Role of intraoperative cholangiography in avoiding bile duct injury. J Am Coll Surg. 2007;204(4):656–64.
64. Ford JA, Soop M, Du J, Loveday BP, Rodgers M. Systematic review of intraoperative cholangiography in cholecystectomy. Br J Surg. 2012;99(2):160–7.
65. Flum DR, Dellinger EP, Cheadle A, Chan L, Koepsell T. Intraoperative cholangiography and risk of common bile duct injury during cholecystectomy. JAMA. 2003;289(13):1639–44.
66. Flum DR, Koepsell T, Heagerty P, Sinanan M, Dellinger EP. Common bile duct injury during laparoscopic cholecystectomy and the use of intraoperative cholangiography: adverse outcome or preventable error? Arch Surg. 2001;136(11):1287–92.
67. Fletcher DR, Hobbs MS, Tan P, Valinsky LJ, Hockey RL, Pikora TJ, et al. Complications of cholecystectomy: risks of the laparoscopic approach and protective effects of operative cholangiography: a population-based study. Ann Surg. 1999;229(4):449–57.
68. Ludwig K, Bernhardt J, Steffen H, Lorenz D. Contribution of intraoperative cholangiography to incidence and outcome of common bile duct injuries during laparoscopic cholecystectomy. Surg Endosc. 2002;16(7):1098–104.
69. Tornqvist B, Stromberg C, Persson G, Nilsson M. Effect of intended intraoperative cholangiography and early detection of bile duct injury on survival after cholecystectomy: population based cohort study. BMJ. 2012;345:e6457.
70. Alvarez FA, de Santibanes M, Palavecino M, Sanchez Claria R, Mazza O, Arbues G, et al. Impact of routine intraoperative cholangiography during laparoscopic cholecystectomy on bile duct injury. Br J Surg. 2014;101(6):677–84.
71. Pekolj J, Alvarez FA, Palavecino M, Sanchez Claria R, Mazza O, de Santibanes E. Intraoperative management and repair of bile duct injuries sustained during 10,123 laparoscopic cholecystectomies in a high-volume referral center. J Am Coll Surg. 2013;216(5):894–901.
72. Nickkholgh A, Soltaniyekta S, Kalbasi H. Routine versus selective intraoperative cholangiography during laparoscopic cholecystectomy: a survey of 2,130 patients undergoing laparoscopic cholecystectomy. Surg Endosc. 2006;20(6):868–74.
73. Sandha GS, Bourke MJ, Haber GB, Kortan PP. Endoscopic therapy for bile leak based on a new classification: results in 207 patients. Gastrointest Endosc. 2004;60(4):567–74.
74. Kaffes AJ, Hourigan L, De Luca N, Byth K, Williams SJ, Bourke MJ. Impact of endoscopic intervention in 100 patients with suspected postcholecystectomy bile leak. Gastrointest Endosc. 2005;61(2):269–75.
75. Carr-Locke AD. Biliary stenting alone versus biliary stenting plus sphincterotomy for the treatment of

post-laparoscopic cholecystectomy bile leaks. Eur J Gastroenterol Hepatol. 2006;18(10):1053–5.
76. Kapoor VK. Management of bile duct injuries: a practical approach. Am Surg. 2009;75(12):1157–60.
77. Taourel P, Bret PM, Reinhold C, Barkun AN, Atri M. Anatomic variants of the biliary tree: diagnosis with MR cholangiopancreatography. Radiology. 1996;199(2):521–7.
78. Puente SG, Bannura GC. Radiological anatomy of the biliary tract: variations and congenital abnormalities. World J Surg. 1983;7(2):271–6.
79. Meyers WC, Peterseim DS, Pappas TN, Schauer PR, Eubanks S, Murray E, et al. Low insertion of hepatic segmental duct VII-VIII is an important cause of major biliary injury or misdiagnosis. Am J Surg. 1996;171(1):187–91.
80. Lillemoe KD, Petrofski JA, Choti MA, Venbrux AC, Cameron JL. Isolated right segmental hepatic duct injury: a diagnostic and therapeutic challenge. J Gastrointest Surg. 2000;4(2):168–77.
81. Christensen RA, van Sonnenberg E, Nemcek AA Jr, D'Agostino HB. Inadvertent ligation of the aberrant right hepatic duct at cholecystectomy: radiologic diagnosis and therapy. Radiology. 1992;183(2):549–53.
82. Hwang S, Lee SG, Lee YJ, Ha TY, Ko GY, Song GW. Delayed-onset isolated injury of the right posterior segment duct after laparoscopic cholecystectomy: a report of hepatic segmental atrophy induction. Surg Laparosc Endosc Percutan Tech. 2007;17(3):203–5.
83. Colovic RB. Isolated segmental, sectoral and right hepatic bile duct injuries. World J Gastroenterol. 2009;15(12):1415–9.
84. Perera MT, Monaco A, Silva MA, Bramhall SR, Mayer AD, Buckels JA, et al. Laparoscopic posterior sectoral bile duct injury: the emerging role of nonoperative management with improved long-term results after delayed diagnosis. Surg Endosc. 2011;25(8):2684–91.
85. Babel N, Sakpal SV, Paragi P, Wellen J, Feldman S, Chamberlain RS. Iatrogenic bile duct injury associated with anomalies of the right hepatic sectoral ducts: a misunderstood and underappreciated problem. HPB Surg. 2009;2009:153269.
86. Perini RF, Uflacker R, Cunningham JT, Selby JB, Adams D. Isolated right segmental hepatic duct injury following laparoscopic cholecystectomy. Cardiovasc Intervent Radiol. 2005;28(2):185–95.
87. Stewart L, Way LW. Bile duct injuries during laparoscopic cholecystectomy. Factors that influence the results of treatment. Arch Surg. 1995;130(10):1123–8; discussion 9.
88. Ishiko T, Egawa H, Kasahara M, Nakamura T, Oike F, Kaihara S, et al. Duct-to-duct biliary reconstruction in living donor liver transplantation utilizing right lobe graft. Ann Surg. 2002;236(2):235–40.
89. Yamamoto S, Sato Y, Oya H, Nakatsuka H, Kobayashi T, Hara Y, et al. Risk factors and prevention of biliary anastomotic complications in adult living donor liver transplantation. World J Gastroenterol. 2007;13(31):4236–41.
90. Jablonska B, Lampe P, Olakowski M, Gorka Z, Lekstan A, Gruszka T. Hepaticojejunostomy vs. end-to-end biliary reconstructions in the treatment of iatrogenic bile duct injuries. J Gastrointest Surg. 2009;13(6):1084–93.
91. Jablonska B, Olakowski M, Lampe P, Gorka Z, Buldak L. Quality-of-life assessment in the treatment of iatrogenic bile duct injuries: hepaticojejunostomy versus end-to-end biliary reconstructions. ANZ J Surg. 2012;82(12):923–7.
92. Gazzaniga GM, Filauro M, Mori L. Surgical treatment of iatrogenic lesions of the proximal common bile duct. World J Surg. 2001;25(10):1254–9.
93. Strasberg SM, Helton WS. An analytical review of vasculobiliary injury in laparoscopic and open cholecystectomy. HPB (Oxford). 2011;13(1):1–14.
94. Jablonska B. End-to-end ductal anastomosis in biliary reconstruction: indications and limitations. Can J Surg. 2014;57(4):271–7.
95. Mirza DF, Narsimhan KL, Ferraz Neto BH, Mayer AD, McMaster P, Buckels JA. Bile duct injury following laparoscopic cholecystectomy: referral pattern and management. Br J Surg. 1997;84(6):786–90.
96. Sicklick JK, Camp MS, Lillemoe KD, Melton GB, Yeo CJ, Campbell KA, et al. Surgical management of bile duct injuries sustained during laparoscopic cholecystectomy: perioperative results in 200 patients. Ann Surg. 2005;241(5):786–92; discussion 93–5.
97. Perera MT, Silva MA, Hegab B, Muralidharan V, Bramhall SR, Mayer AD, et al. Specialist early and immediate repair of post-laparoscopic cholecystectomy bile duct injuries is associated with an improved long-term outcome. Ann Surg. 2011;253(3):553–60.
98. Silva MA, Coldham C, Mayer AD, Bramhall SR, Buckels JA, Mirza DF. Specialist outreach service for on-table repair of iatrogenic bile duct injuries—a new kind of 'travelling surgeon'. Ann R Coll Surg Engl. 2008;90(3):243–6.
99. Brittain RS, Marchioro TL, Hermann G, Waddell WR, Starzl TE. Accidental hepatic artery ligation in humans. Am J Surg. 1964;107:822–32.
100. Davidoff AM, Pappas TN, Murray EA, Hilleren DJ, Johnson RD, Baker ME, et al. Mechanisms of major biliary injury during laparoscopic cholecystectomy. Ann Surg. 1992;215(3):196–202.
101. Halasz NA. Cholecystectomy and hepatic artery injuries. Arch Surg. 1991;126(2):137–8.
102. Chapman WC, Halevy A, Blumgart LH, Benjamin IS. Postcholecystectomy bile duct strictures. Management and outcome in 130 patients. Arch Surg. 1995;130(6):597–602; discussion 602–4.
103. Li J, Frilling A, Nadalin S, Paul A, Malago M, Broelsch CE. Management of concomitant hepatic artery injury in patients with iatrogenic major bile duct injury after laparoscopic cholecystectomy. Br J Surg. 2008;95(4):460–5.
104. Alves A, Farges O, Nicolet J, Watrin T, Sauvanet A, Belghiti J. Incidence and consequence of an hepatic artery injury in patients with postcholecystectomy

bile duct strictures. Ann Surg. 2003;238(1): 93–6.
105. Redman HC, Reuter SR. Arterial collaterals in the liver hilus. Radiology. 1970;94(3):575–9.
106. Stapleton GN, Hickman R, Terblanche J. Blood supply of the right and left hepatic ducts. Br J Surg. 1998;85(2):202–7.
107. Vellar ID. The blood supply of the biliary ductal system and its relevance to vasculobiliary injuries following cholecystectomy. Aust N Z J Surg. 1999;69(11):816–20.
108. de Santibanes E, Palavecino M, Ardiles V, Pekolj J. Bile duct injuries: management of late complications. Surg Endosc. 2006;20(11):1648–53.
109. Buell JF, Cronin DC, Funaki B, Koffron A, Yoshida A, Lo A, et al. Devastating and fatal complications associated with combined vascular and bile duct injuries during cholecystectomy. Arch Surg. 2002;137(6):703–8; discussion 8–10.
110. Slater K, Strong RW, Wall DR, Lynch SV. Iatrogenic bile duct injury: the scourge of laparoscopic cholecystectomy. ANZ J Surg. 2002;72(2):83–8.
111. Gupta N, Solomon H, Fairchild R, Kaminski DL. Management and outcome of patients with combined bile duct and hepatic artery injuries. Arch Surg. 1998;133(2):176–81.
112. Koffron A, Ferrario M, Parsons W, Nemcek A, Saker M, Abecassis M. Failed primary management of iatrogenic biliary injury: incidence and significance of concomitant hepatic arterial disruption. Surgery. 2001;130(4):722–8; discussion 8–31.
113. Schmidt SC, Settmacher U, Langrehr JM, Neuhaus P. Management and outcome of patients with combined bile duct and hepatic arterial injuries after laparoscopic cholecystectomy. Surgery. 2004;135(6): 613–8.
114. Strasberg SM, Gouma DJ. 'Extreme' vasculobiliary injuries: association with fundus-down cholecystectomy in severely inflamed gallbladders. HPB (Oxford). 2012;14(1):1–8.
115. Bilge O, Bozkiran S, Ozden I, Tekant Y, Acarli K, Alper A, et al. The effect of concomitant vascular disruption in patients with iatrogenic biliary injuries. Langenbeck's Arch Surg. 2003;388(4): 265–9.
116. Gouma DJ, Obertop H. Management of bile duct injuries: treatment and long-term results. Dig Surg. 2002;19(2):117–22.
117. Goykhman Y, Kory I, Small R, Kessler A, Klausner JM, Nakache R, et al. Long-term outcome and risk factors of failure after bile duct injury repair. J Gastrointest Surg. 2008;12(8):1412–7.
118. Stewart L, Way LW. Laparoscopic bile duct injuries: timing of surgical repair does not influence success rate. A multivariate analysis of factors influencing surgical outcomes. HPB (Oxford). 2009;11(6): 516–22.
119. Thomson BN, Parks RW, Madhavan KK, Wigmore SJ, Garden OJ. Early specialist repair of biliary injury. Br J Surg. 2006;93(2):216–20.
120. Chaudhary A, Chandra A, Negi SS, Sachdev A. Reoperative surgery for postcholecystectomy bile duct injuries. Dig Surg. 2002;19(1):22–7.
121. de Reuver PR, Grossmann I, Busch OR, Obertop H, van Gulik TM, Gouma DJ. Referral pattern and timing of repair are risk factors for complications after reconstructive surgery for bile duct injury. Ann Surg. 2007;245(5):763–70.
122. Walsh RM, Henderson JM, Vogt DP, Brown N. Long-term outcome of biliary reconstruction for bile duct injuries from laparoscopic cholecystectomies. Surgery. 2007;142(4):450–6; discussion 6–7.
123. Felekouras E, Petrou A, Neofytou K, Moris D, Dimitrokallis N, Bramis K, et al. Early or delayed intervention for bile duct injuries following laparoscopic cholecystectomy? A dilemma looking for an answer. Gastroenterol Res Pract. 2015;2015: 104235.
124. Schmidt SC, Langrehr JM, Hintze RE, Neuhaus P. Long-term results and risk factors influencing outcome of major bile duct injuries following cholecystectomy. Br J Surg. 2005;92(1):76–82.
125. Lillemoe KD, Martin SA, Cameron JL, Yeo CJ, Talamini MA, Kaushal S, et al. Major bile duct injuries during laparoscopic cholecystectomy. Follow-up after combined surgical and radiologic management. Ann Surg. 1997;225(5):459–68; discussion 68–71.
126. Blumgart LH, Kelley CJ, Benjamin IS. Benign bile duct stricture following cholecystectomy: critical factors in management. Br J Surg. 1984;71(11): 836–43.
127. Parekh NR, Steiger E. Percentage of weight loss as a predictor of surgical risk: from the time of Hiram Studley to today. Nutr Clin Pract. 2004;19(5):471–6.
128. Aahlin EK, Trano G, Johns N, Horn A, Soreide JA, Fearon KC, et al. Risk factors, complications and survival after upper abdominal surgery: a prospective cohort study. BMC Surg. 2015;15:83.
129. Kamiya S, Nagino M, Kanazawa H, Komatsu S, Mayumi T, Takagi K, et al. The value of bile replacement during external biliary drainage: an analysis of intestinal permeability, integrity, and microflora. Ann Surg. 2004;239(4):510–7.
130. Wexler MJ, Smith R. Jejunal mucosal graft: a sutureless technic for repair of high bile duct strictures. Am J Surg. 1975;129(2):204–11.
131. Winslow ER, Fialkowski EA, Linehan DC, Hawkins WG, Picus DD, Strasberg SM. "Sideways": results of repair of biliary injuries using a policy of side-to-side hepatico-jejunostomy. Ann Surg. 2009;249(3):426–34.
132. Hepp J, Couinaud C. Approach to and use of the left hepatic duct in reparation of the common bile duct. Presse Med. 1956;64(41):947–8.
133. Hepp J. Hepaticojejunostomy using the left biliary trunk for iatrogenic biliary lesions: the French connection. World J Surg. 1985;9(3):507–11.
134. Couinaud C. Exposure of the left hepatic duct through the hilum or in the umbilical of the liver: anatomic limitations. Surgery. 1989;105(1):21–7.

135. Mercado MA, Orozco H, de la Garza L, Lopez-Martinez LM, Contreras A, Guillen-Navarro E. Biliary duct injury: partial segment IV resection for intrahepatic reconstruction of biliary lesions. Arch Surg. 1999;134(9):1008–10.
136. Mercado MA, Chan C, Orozco H, Villalta JM, Barajas-Olivas A, Erana J, et al. Long-term evaluation of biliary reconstruction after partial resection of segments IV and V in iatrogenic injuries. J Gastrointest Surg. 2006;10(1):77–82.
137. Mercado MA, Chan C, Orozco H, Hinojosa CA, Podgaetz E, Ramos-Gallardo G, et al. Prognostic implications of preserved bile duct confluence after iatrogenic injury. Hepato-Gastroenterology. 2005;52(61):40–4.
138. Mercado MA, Vilatoba M, Contreras A, Leal-Leyte P, Cervantes-Alvarez E, Arriola JC, et al. Iatrogenic bile duct injury with loss of confluence. World J Gastrointest Surg. 2015;7(10):254–60.
139. Thomson BN, Parks RW, Madhavan KK, Garden OJ. Liver resection and transplantation in the management of iatrogenic biliary injury. World J Surg. 2007;31(12):2363–9.
140. Laurent A, Sauvanet A, Farges O, Watrin T, Rivkine E, Belghiti J. Major hepatectomy for the treatment of complex bile duct injury. Ann Surg. 2008;248(1):77–83.
141. Truant S, Boleslawski E, Lebuffe G, Sergent G, Pruvot FR. Hepatic resection for post-cholecystectomy bile duct injuries: a literature review. HPB (Oxford). 2010;12(5):334–41.
142. Li J, Frilling A, Nadalin S, Broelsch CE, Malago M. Timing and risk factors of hepatectomy in the management of complications following laparoscopic cholecystectomy. J Gastrointest Surg. 2012;16(4):815–20.
143. Pekolj J, Yanzon A, Dietrich A, Del Valle G, Ardiles V, de Santibanes E. Major liver resection as definitive treatment in post-cholecystectomy common bile duct injuries. World J Surg. 2015;39(5):1216–23.
144. de Reuver PR, Rauws EA, Vermeulen M, Dijkgraaf MG, Gouma DJ, Bruno MJ. Endoscopic treatment of post-surgical bile duct injuries: long term outcome and predictors of success. Gut. 2007;56(11):1599–605.
145. Vitale GC, Tran TC, Davis BR, Vitale M, Vitale D, Larson G. Endoscopic management of postcholecystectomy bile duct strictures. J Am Coll Surg. 2008;206(5):918–23; discussion 24–5.
146. Weber A, Prinz C, Gerngross C, Ludwig L, Huber W, Neu B, et al. Long-term outcome of endoscopic and/or percutaneous transhepatic therapy in patients with biliary stricture after orthotopic liver transplantation. J Gastroenterol. 2009;44(12):1195–202.
147. Weber A, Feussner H, Winkelmann F, Siewert JR, Schmid RM, Prinz C. Long-term outcome of endoscopic therapy in patients with bile duct injury after cholecystectomy. J Gastroenterol Hepatol. 2009;24(5):762–9.
148. Abdel-Raouf A, Hamdy E, El-Hanafy E, El-Ebidy G. Endoscopic management of postoperative bile duct injuries: a single center experience. Saudi J Gastroenterol. 2010;16(1):19–24.
149. Brunet M, Blouin Y, Mosimann F. Biliary strictures after cholecystectomy: long term results of a novative endoscopic treatment. Hepato-Gastroenterology. 2014;61(136):2203–8.
150. Costamagna G, Pandolfi M, Mutignani M, Spada C, Perri V. Long-term results of endoscopic management of postoperative bile duct strictures with increasing numbers of stents. Gastrointest Endosc. 2001;54(2):162–8.
151. Kuzela L, Oltman M, Sutka J, Hrcka R, Novotna T, Vavrecka A. Prospective follow-up of patients with bile duct strictures secondary to laparoscopic cholecystectomy, treated endoscopically with multiple stents. Hepato-Gastroenterology. 2005;52(65):1357–61.
152. Lawrence C, Romagnuolo J, Payne KM, Hawes RH, Cotton PB. Low symptomatic premature stent occlusion of multiple plastic stents for benign biliary strictures: comparing standard and prolonged stent change intervals. Gastrointest Endosc. 2010;72(3):558–63.
153. Poley JW, Cahen DL, Metselaar HJ, van Buuren HR, Kazemier G, van Eijck CH, et al. A prospective group sequential study evaluating a new type of fully covered self-expandable metal stent for the treatment of benign biliary strictures (with video). Gastrointest Endosc. 2012;75(4):783–9.
154. Tarantino I, Mangiavillano B, Di Mitri R, Barresi L, Mocciaro F, Granata A, et al. Fully covered self-expandable metallic stents in benign biliary strictures: a multicenter study on efficacy and safety. Endoscopy. 2012;44(10):923–7.
155. Bergman JJ, Burgemeister L, Bruno MJ, Rauws EA, Gouma DJ, Tytgat GN, et al. Long-term follow-up after biliary stent placement for postoperative bile duct stenosis. Gastrointest Endosc. 2001;54(2):154–61.
156. Costamagna G, Shah SK, Tringali A. Current management of postoperative complications and benign biliary strictures. Gastrointest Endosc Clin N Am. 2003;13(4):635–48. ix
157. Fischer CP, Fahy BN, Aloia TA, Bass BL, Gaber AO, Ghobrial RM. Timing of referral impacts surgical outcomes in patients undergoing repair of bile duct injuries. HPB (Oxford). 2009;11(1):32–7.
158. Sulpice L, Garnier S, Rayar M, Meunier B, Boudjema K. Biliary cirrhosis and sepsis are two risk factors of failure after surgical repair of major bile duct injury post-laparoscopic cholecystectomy. Langenbeck's Arch Surg. 2014;399(5):601–8.
159. Pottakkat B, Vijayahari R, Prakash A, Singh RK, Behari A, Kumar A, et al. Factors predicting failure following high bilio-enteric anastomosis for post-cholecystectomy benign biliary strictures. J Gastrointest Surg. 2010;14(9):1389–94.

160. Bottger T, Junginger T. Long-term results after surgical treatment of iatrogenic injury of the bile ducts. Eur J Surg. 1991;157(8):477–80.
161. Ruemmele P, Hofstaedter F, Gelbmann CM. Secondary sclerosing cholangitis. Nat Rev Gastroenterol Hepatol. 2009;6(5):287–95.
162. Johnson SR, Koehler A, Pennington LK, Hanto DW. Long-term results of surgical repair of bile duct injuries following laparoscopic cholecystectomy. Surgery. 2000;128(4):668–77.
163. Nordin A, Halme L, Makisalo H, Isoniemi H, Hockerstedt K. Management and outcome of major bile duct injuries after laparoscopic cholecystectomy: from therapeutic endoscopy to liver transplantation. Liver Transpl. 2002;8(11):1036–43.
164. Ibrarullah M, Sikora SS, Agarwal DK, Kapoor VK, Kaushik SP. 'Latent' portal hypertension in benign biliary obstruction. HPB Surg. 1996;9(3):149–52.
165. Negi SS, Sakhuja P, Malhotra V, Chaudhary A. Factors predicting advanced hepatic fibrosis in patients with postcholecystectomy bile duct strictures. Arch Surg. 2004;139(3):299–303.
166. Barbier L, Souche R, Slim K, Ah-Soune P. Long-term consequences of bile duct injury after cholecystectomy. J Visc Surg. 2014;151(4):269–79.
167. Christophi C, Dudley F, McInnes IE. Surgical treatment of biliary stricture with portal hypertension using veno-venous bypass. Aust N Z J Surg. 1991;61(4):316–8.
168. de Santibanes E, Ardiles V, Gadano A, Palavecino M, Pekolj J, Ciardullo M. Liver transplantation: the last measure in the treatment of bile duct injuries. World J Surg. 2008;32(8):1714–21.
169. Ragins AI, Shan J, Thom DH, Subak LL, Brown JS, Van Den Eeden SK. Effects of urinary incontinence, comorbidity and race on quality of life outcomes in women. J Urol. 2008;179(2):651–5; discussion 5.
170. Hogan AM, Hoti E, Winter DC, Ridgway PF, Maguire D, Geoghegan JG, et al. Quality of life after iatrogenic bile duct injury: a case control study. Ann Surg. 2009;249(2):292–5.
171. Robinson TN, Stiegmann GV, Durham JD, Johnson SI, Wachs ME, Serra AD, et al. Management of major bile duct injury associated with laparoscopic cholecystectomy. Surg Endosc. 2001;15(12):1381–5.
172. Boerma D, Rauws EA, Keulemans YC, Bergman JJ, Obertop H, Huibregtse K, et al. Impaired quality of life 5 years after bile duct injury during laparoscopic cholecystectomy: a prospective analysis. Ann Surg. 2001;234(6):750–7.
173. Moore DE, Feurer ID, Holzman MD, Wudel LJ, Strickland C, Gorden DL, et al. Long-term detrimental effect of bile duct injury on health-related quality of life. Arch Surg. 2004;139(5):476–81; discussion 81–2.
174. Dominguez-Rosado I, Mercado MA, Kauffman C, Ramirez-del Val F, Elnecave-Olaiz A, Zamora-Valdes D. Quality of life in bile duct injury: 1-, 5-, and 10-year outcomes after surgical repair. J Gastrointest Surg. 2014;18(12):2089–94.
175. Melton GB, Lillemoe KD, Cameron JL, Sauter PA, Coleman J, Yeo CJ. Major bile duct injuries associated with laparoscopic cholecystectomy: effect of surgical repair on quality of life. Ann Surg. 2002;235(6):888–95.
176. de Reuver PR, Sprangers MA, Gouma DJ. Quality of life in bile duct injury patients. Ann Surg. 2007;246(1):161–3.
177. de Reuver PR, Sprangers MA, Rauws EA, Lameris JS, Busch OR, van Gulik TM, et al. Impact of bile duct injury after laparoscopic cholecystectomy on quality of life: a longitudinal study after multidisciplinary treatment. Endoscopy. 2008;40(8):637–43.
178. Landman MP, Feurer ID, Moore DE, Zaydfudim V, Pinson CW. The long-term effect of bile duct injuries on health-related quality of life: a meta-analysis. HPB (Oxford). 2013;15(4):252–9.
179. Ejaz A, Spolverato G, Kim Y, Dodson R, Sicklick JK, Pitt HA, et al. Long-term health-related quality of life after iatrogenic bile duct injury repair. J Am Coll Surg. 2014;219(5):923–32. e10
180. Kern KA. Medicolegal analysis of bile duct injury during open cholecystectomy and abdominal surgery. Am J Surg. 1994;168(3):217–22.
181. Chandler JG, Voyles CR, Floore TL, Bartholomew LA. Litigious consequences of open and laparoscopic biliary surgical mishaps. J Gastrointest Surg. 1997;1(2):138–45; discussion 45.
182. Alkhaffaf B, Decadt B. 15 years of litigation following laparoscopic cholecystectomy in England. Ann Surg. 2010;251(4):682–5.
183. Perera MT, Silva MA, Shah AJ, Hardstaff R, Bramhall SR, Issac J, et al. Risk factors for litigation following major transectional bile duct injury sustained at laparoscopic cholecystectomy. World J Surg. 2010;34(11):2635–41.
184. Goldman RL. The reliability of peer assessments of quality of care. JAMA. 1992;267(7):958–60.
185. Andrew LB. Expert witness testimony: the ethics of being a medical expert witness. Emerg Med Clin North Am. 2006;24(3):715–31.
186. Localio AR, Lawthers AG, Brennan TA, Laird NM, Hebert LE, Peterson LM, et al. Relation between malpractice claims and adverse events due to negligence. Results of the Harvard Medical Practice Study III. N Engl J Med. 1991;325(4):245–51.
187. Hugh TB, Tracy GD. Hindsight bias in medicolegal expert reports. Med J Aust. 2002;176(6):277–8.
188. de Reuver PR, Dijkgraaf MG, Gevers SK, Gouma DJ, Group BS. Poor agreement among expert witnesses in bile duct injury malpractice litigation: an expert panel survey. Ann Surg. 2008;248(5):815–20.