Along with bile cholesterol supersaturation and enhanced crystal nucleation, impaired gallbladder motility is one of the main factors involved in gallstone formation. In response to either a meal or cholecystokinin administration, gall bladders of patients with gall stones contract less well than normal gall bladders. This impaired motility arises not as a consequence of the presence of stones in the gall bladder – animal models of gall stones demonstrate that impaired gallbladder motility actually precedes gallstone formation. In humans, in clinical situations where there is impairment of gallbladder motility, there is an enhanced rate of gallstone formation. For instance, in patients receiving total parental nutrition and patients receiving octreotide there is an enhanced rate of gallstone formation. After successful lithotripsy gallbladder stone recurrence is greater in patients with impaired gallbladder motility. In animal models it has also been shown that improving gallbladder motility prevents gall stones. Improving gallbladder motility in patients receiving total parental nutrition prevents sludge formation.

Thus, factors that adversely affect gallbladder motility can promote gallstone formation and the corollary is also true. With this in mind, Sugiyama and Atomi have sought to determine how endoscopic sphincterotomy might affect gallbladder motility and their results are published in this edition of Gut. They found that gallbladder fasting volume was less and gallbladder contractility, in response to caerulein – a cholecystokinin mimetic – was improved both at seven days and five years after endoscopic sphincterotomy. Thus sphincterotomy, actually, has a positive effect on gallbladder motility and may even prevent further gallstone formation. This study therefore removed the niggling doubt in many peoples' mind that sphincterotomy might impair gallbladder motility and therefore increase the possibility of gall stones.

Although not the main objective of the study, clinical follow up indicated that 14 of 15 patients with gallbladder stones remained asymptomatic in the five years after sphincterotomy. This and the motility findings provide further reassurance that the vast majority of patients, especially elderly patients, with problems attributable to duct stones are not in need of a cholecystectomy once the duct stones have been dealt with successfully.

This reassurance regarding endoscopic sphincterotomy receives further support from recent papers describing the short-term and long term clinical outcome in patients who have had this procedure. Follow up of 2347 patients who underwent endoscopic sphincterotomy in 17 institutions in North America demonstrated that the overall death rate in the month after the procedure is 2-3%, with 0.4% due directly or indirectly to the procedure. The commonest complication is clinical pancreatitis occurring in 5-4% of patients. Suspected sphincter of Oddi dysfunction and pre-cut sphincterotomy were the main risk factors for developing severe pancreatitis. Forty one per cent of patients undergoing sphincterotomy with suspected sphincter of Oddi dysfunction developed clinical pancreatitis. Clinically significant haemorrhage occurred in 2% of patients; 0.5% having severe haemorrhage. The main risk factors for the development of haemorrhage were coagulopathy prior to procedure, anticoagulation after procedure, cholangitis, and inexperience of the endoscopist. Clinical recognition of haemorrhage was delayed greater than 24 hours in 52% of cases. Cholangitis (1%), bowel perforation (0.3%), and cholecystitis (0.5%) were the other main complications.

Long term outcome after endoscopic sphincterotomy has also recently been reported on by Prat and colleagues who retrospectively followed up 156 patients for a mean period of 9.5 years – follow up rate 92%. Only 4% of patients had biliary complications definitely related to previous endoscopic sphincterotomy (three patients with papillary stenosis, two of which had common bile duct stones; two cholangitis, one benign common bile duct stricture). Another 3% of patients had biliary tract disease during the 9.5 year follow up that could not be attributed directly to endoscopic sphincterotomy.

At this point in time, it is clear that although endoscopic sphincterotomy is not without its complications and surgery is a viable alternative in many instances, it is often sufficient treatment for elderly patients with clinical problems solely related to common bile duct stones, it does not have a deleterious effect on gallbladder motility and long term complications are uncommon.

This reassurance is particularly welcome for many endoscopists at this juncture when laparoscopic surgery is fuelling an increasing demand for pre-operative and postoperative endoscopic sphincterotomy. However, more studies comparing the efficacy and safety of the endoscopic approach and bile duct surgery are needed with patient stratification according to presenting problem and co-existing morbidity.

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Sphincterotomy and the gall bladder--a slice of luck.

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