sion of cell-mediated immunity.[4] Pitt *et al* have recently studied the risk factors in 155 consecutive patients undergoing biliary surgery for both benign and malignant conditions.[5] Of the 15 clinical or laboratory features studied, eight associated with increased mortality were selected as the basis of a simple scoring system: age greater than 60, malignant obstruction, hypoalbuminaemia (≤ 30 g/l), packed cell volume less than 30%, leucocytosis, and raised serum concentrations of total bilirubin, creatinine, and activities of alkaline phosphatase above defined limits. No patient with two or fewer risk factors died, but as more than three factors became positive mortality increased progressively. Ten of the 12 patients who died had five or more risk factors.

Until recently attempts to reduce the mortality of biliary surgery for obstructive jaundice have centred on antibiotic cover to reduce the risk of sepsis; maintenance of urine flow by an adequate fluid intake, supplemented when necessary by agents such as mannitol; and correction of anaemia and coagulation deficits. Nevertheless, the high operative mortality has obliged surgeons to consider staged operations with preliminary decompression of the biliary tract at laparotomy for those requiring major surgical procedures such as pancreaticoduodenectomy.

An important recent advance has been the development of non-operative techniques for biliary decompression as a prelude to definitive surgery. These include percutaneous transhepatic biliary drainage,[6 7] endoscopic insertion of a catheter into the biliary system from the duodenum,[8] and endoscopic papillotomy to allow internal biliary drainage in patients with carcinoma in or around the papilla of Vater.[9] Of the options available, percutaneous transhepatic biliary drainage has probably been used most extensively and should be within the compass of a radiologist experienced in the technique for percutaneous transhepatic cholangiography.

As described by Nakayama and colleagues,[7] percutaneous transhepatic biliary drainage is preceded by cholangiography carried out through the right flank by the Chiba technique. A dilated intrahepatic duct is then punctured with a steel needle, a guide wire inserted into the biliary system, and the needle removed. A polyethylene catheter is advanced over the guide wire, which is then withdrawn. Two catheters may be used to ensure drainage of both right and left hepatic ductal systems in patients with lesions causing obstruction at the confluence of the two systems. Complications have been reported in 5% of patients,[7 10] and include leakage of bile into the peritoneal or pleural cavities, intraperitoneal bleeding, pneumothorax, haematobilia, and dislocation of the catheter. Percutaneous transhepatic biliary drainage reduced serum concentrations of bilirubin in patients with malignant obstruction at the same rate as cholecystostomy in a group of comparable patients reviewed retrospectively.[7] Nakayama and colleagues successfully achieved percutaneous transhepatic biliary drainage in 104 of 105 patients with obstruction due to benign (21) or malignant disease (84), proceeding to laparotomy in 69 cases after intervals ranging from eight to 70 days. The operative mortality (defined as death within one month of surgery or caused by a condition directly attributable to operation) was 6% for the 69 patients as a whole, and 8% for the subgroup of 49 patients with malignant disease. Retrospective assessment of 148 patients operated on for malignant obstruction without prior decompression showed an operative mortality of 28%, most of the deaths being attributed to hepatic failure or rapid debilitation. The limitations of such retrospective comparison are obvious: no fewer than 35 of the 104 patients undergoing percutaneous transhepatic biliary drainage did not go on to laparotomy. Nevertheless, on the available evidence, a prospective controlled trial of preliminary percutaneous transhepatic biliary drainage versus no preliminary drainage in patients undergoing biliary surgery for obstructive jaundice would be difficult to justify on ethical grounds.

A number of authors[11–14] have described the use of the percutaneous transhepatic route to pass a guide wire through the site of malignant obstruction, allowing subsequent dilatation of the strictured area and insertion of an endoprosthesis to allow bile to drain into the common bile duct or duodenum. Further study will be needed to define the place of long-term percutaneous transhepatic biliary drainage as an alternative to surgical intervention in patients with malignant obstruction. Non-operative drainage may prove to be a valuable palliative procedure in poor-risk patients with malignant obstruction, but the attractions of the technique must not be allowed to deny curative resection to selected patients in this group or to permit unnecessary procrastination.

1 O'Connor MJ, Schwartz ML, McQuarrie DG, Sumner HW. Cholangitis due to malignant obstruction of biliary outflow. *Ann Surg* 1981;**193**:341-5.

2 Allison MEM, Prentice CRM, Kennedy AC, Blumgart LH. Renal function and other factors in obstructive jaundice. *Br J Surg* 1979;**66**:391-7.

3 Bailey ME. Endotoxin, bile salts and renal function in obstructive jaundice. *Br J Surg* 1976;**63**:774-8.

4 Gianni L, Di Padova F, Zuin M, Podda M. Bile acid-induced inhibition of the lymphoproliferative responses to phytohemagglutinin and pokeweed mitogen: an in vitro study. *Gastroenterology* 1980;**78**:231-5.

5 Pitt HA, Cameron JL, Postier RG, Gadacz TR. Factors affecting mortality in biliary tract surgery. *Am J Surg* 1981;**141**:66-72.

6 Burcharth F. A new endoprosthesis for nonoperative intubation of the biliary tract in malignant obstructive jaundice. *Surg Gynecol Obstet* 1978;**146**:76-8.

7 Nakayama T, Ikeda A, Okuda K. Percutaneous transhepatic drainage of the biliary tract. Technique and results in 104 cases. *Gastroenterology* 1978;**74**:554-9.

8 Osnes M, Odd G, Grönoeth K, Havig Ø. Nonoperative internal drainage of obstructive common bile ducts. *Arch Surg* 1979;**114**:862-5.

9 Alderson D, Lavelle MI, Venables CW. Endoscopic sphincterotomy before pancreaticoduodenectomy for ampullary carcinoma. *Br Med J* 1981;**282**:1109-11.

10 Hansson JA, Hoevels J, Simert G, Tylen U, Vang J. Clinical aspects of nonsurgical percutaneous transhepatic bile drainage in obstructive lesions of the extrahepatic bile ducts. *Ann Surg* 1979;**189**:58-61.

11 Smale BF, Ring EJ, Frieman DB, *et al*. Successful long-term percutaneous decompression of the biliary tract. *Am J Surg* 1981;**141**:73-6.

12 Burcharth F, Jensen LI, Olesen K. Endoprosthesis for internal drainage of the biliary tract. Technique and results in 48 cases. *Gastroenterology* 1979;**77**:133-7.

13 Sarrafizadeh MS, Philip PK, Goldman ML. Percutaneous transhepatic T-tube placement for antegrade biliary drainage. A new technique. *Radiology* 1980;**137**:545-6.

14 Pereiras RV, Rheingold OJ, Hutson D, *et al*. Relief of malignant obstructive jaundice by percutaneous insertion of a permanent prosthesis in the biliary tree. *Ann Intern Med* 1978;**89**:589-93.

Metastases in the liver

Liver metastases are an all too common finding at laparotomy for intra-abdominal cancer. Oxley and Ellis,[1] in a report typical of many, found that 112 (18%) patients out of a total of 640 undergoing surgery for carcinoma of the large bowel had deposits in the liver detected by palpation at operation.

The presence of liver metastases has a dramatic effect on prognosis and will considerably affect the surgeon's management of the case. So can we make a confident preoperative assessment of the state of the liver, and how accurate is the surgeon's palpating hand at the time of operation?

Biochemical tests of liver function give some guidance. For example, alkaline phosphatase activity is raised in about a third of patients with liver deposits and false-positive results are unusual in Britain.[2] The likelihood of a false-positive result is, however, higher in populations with a high prevalence of alcoholism and cirrhosis. Combining the liver enzyme estimations with the serum acid phosphatase value greatly refines the accuracy of the biochemical assessment, though again false-positive and false-negative results may be found in around 20% of cases.[3]

Techniques for imaging the liver are becoming more and more refined. They include ultrasound, computed tomography, scanning with radioactive materials, and arteriography. Nevertheless, both false-positive and false-negative results are still found. The common cause for a false-negative diagnosis is that small lesions are difficult to visualise, so that a tumour of less than 2 cm diameter may easily be overlooked. A false-positive finding may be due to benign focal lesions in the liver, such as angiomas, cysts, or adenomas. False-positive scans may be seen in patients with cirrhosis and may be given by an enlarged gall-bladder fossa, the thinned-out or small left lobe of the liver, and peculiar shapes of the liver.[4 5]

Clearly, therefore, when the surgeon comes to perform a laparotomy on a patient with suspected intra-abdominal cancer he remains in considerable doubt about the state of the patient's liver. He will have learnt from bitter experience that despite a battery of negative findings from the most advanced tests he may yet find a liver studded with deposits, and he will also be familiar with cases where the gloomiest of preoperative prognoses are confounded at the time of surgery. The surgeon is all too aware, however, that he himself is far from omniscient. Forty years ago Goligher[6] analysed a consecutive series of 893 cases of rectal cancer coming to laparotomy at St Mark's Hospital, London, and found that the surgeon detected hepatic deposits in 103 of these (12%). Thirty-one of the patients whose livers had been found to be smooth and free from palpable metastases at operation, but who died in the immediate postoperative period, were submitted to necropsy and in five a secondary deposit was present in the depths of the liver. This represented an incidence of concealed liver metastases of roughly one in six of the cases coming to necropsy. Later studies were more in the surgeon's favour. Hogg and Pack[7] studied 100 patients dying within 30 days of laparotomy for a variety of intra-abdominal tumours at the Memorial Hospital for Cancer, New York. In each case the liver had been palpated and pronounced clear. Only five of these patients were subsequently found to have liver metastases at necropsy. Of these, three were not seen by the pathologist at naked-eye examination of the cut slices of liver but were detected only histologically. Recently, Gray[8] has analysed 116 patients with gastrointestinal cancer submitted to laparotomy at St Vincent's Hospital, Melbourne, who had died within one month of surgery and who had had a necropsy performed. In 78 patients the liver was assessed by the surgeon as being free of tumour and in 38 patients it was considered to contain metastases. Those assessments proved to be wrong in nine of the 116 cases. In six patients the liver had been assessed as being free of tumour but necropsy showed the presence of deposits; in one of these, however, there was no macroscopic evidence of tumour and malignant cells were identified only on microscopical study. In three patients the surgeon's diagnosis of metastases at laparotomy was disproved at necropsy.

From time to time all surgeons will continue carrying out radical resections of abdominal cancers in the hope of cure only to be disappointed by the rapid appearance of liver metastases, signifying that the preoperative and operative assessment of a normal liver was mistaken. This is unfortunate but inevitable until even more sensitive methods of liver imaging are developed. The mere presence of suspicious nodules in the liver must not deter the surgeon from carrying out a resection of the primary lesion, since experience has shown that this is usually the best way of achieving maximum palliation.[1] To biopsy one or more of these "deposits" is good practice. This will provide histological documentation—and every now and then both the surgeon and the patient will have the pleasant surprise of finding the apparent death sentence replaced by the news that the so-called metastases are some entirely benign lesion.

1 Oxley EM, Ellis H. Prognosis of carcinoma of the large bowel in the presence of liver metastases. *Br J Surg* 1969;**56**:149-52.
2 Ellis H, Cheng B. Palliative resection of the large bowel in the presence of liver metastases. *Proc R Soc Med* 1970;**63**:751.
3 Cederqvist C, Nielsen J. Value of liver function tests in the diagnosis of hepatic metastases in patients with gastric cancer. *Acta Chir Scand* 1972;**138**:604-8.
4 DuPriest RW, Haines JE, Rösch J, Krippaehne WW. A comparison of scintiscans and arteriograms for identifying metastatic intrahepatic tumours. *Surg Gynecol Obstet* 1973;**136**:705-10.
5 DeWinter J. Computed axial tomography in abdominal visceral disease. In: Maingot R, ed. *Abdominal operations*. Vol 2. 7th ed. New York: Appleton Century Crofts, 1980:2478-508.
6 Goligher JC. The operability of carcinoma of the rectum. *Br Med J* 1941; ii:393-7.
7 Hogg L, Pack GT. Diagnostic accuracy of hepatic metastases at laparotomy. *Arch Surg* 1956;**72**:251-2.
8 Gray BN. Surgeon accuracy in the diagnosis of liver metastases at laparotomy. *Aust NZ J Surg* 1980;**50**:524-6.

Hodgkin's disease and viruses

Since the first description of Hodgkin's disease both its nature and its cause have been contentious. At first the debate questioned whether the disease was a cancer or an unusual infection, possibly an atypical form of tuberculosis.[1] More recently there has been a general consensus that it is a malignant disease. The Reed-Sternberg cells—the putative cancer cells[2]—have been grown in culture and characterised by Kaplan and Gartner.[3]

Reports of sporadic clusters of Hodgkin's disease have appeared intermittently for 20 years, though some workers have doubted the validity of such an experimental approach.[4] Recent epidemiological studies of the disease have been more rigorous. Gutensohn and Cole[4] have suggested that a delayed and infrequent infection by a common virus may play a part in the development of the disease.[5] Their analogy between the epidemiological features of poliomyelitis and of Hodgkin's disease has been drawn before, but they have amplified the evidence that indirectly supports a viral origin of Hodgkin's disease.

In both poliomyelitis and Hodgkin's disease the peak age of incidence becomes delayed as living conditions improve, and increased risk is associated with higher social class and small family size. If Hodgkin's disease is a rare consequence of a common viral infection the environmental factors in childhood that influence exposure to infectious agents should be associated with Hodgkin's disease in young adults.

Gutensohn and Cole studied 225 patients (cases) and 447 controls (15-39 years of age) and found several factors in