The use of endoscopy in liver diseases

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The use of fibre-optic endoscopy has greatly facilitated the management of some patients with chronic liver disease. Upper endoscopy plays a pivotal role in the diagnosis and management of oesophageal and gastric varices. With the use of reflectance septrophotometry, gastroduodenal mucosal haemoglobin concentration and oxygen saturation can be more precisely measured. Recently, it has been shown that acute gastroduodenitis is associated with a lower pre-treatment mucosal oxygen concentration in the antrum and the first part of the duodenum. Endoscopic ultrasound is increasingly being used to detect varices and in the staging of gastrointestinal tumours. Endoscopic retrograde cholangiopancreatography plays an important role in the diagnosis of recurrent pyogenic cholangitis and endoscopic sphincterotomy is a useful form of treatment. Laparoscopy, with the aid of ultrasound and biopsy is helpful in staging chronic liver disease, identifying focal lesions, and diagnosing peritoneal disease.

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Introduction

Endoscopy, principally flexible endoscopy of the gastrointestinal tract and diagnostic laparoscopy, is increasingly being used in the investigation and management of liver diseases. Progress in instrumentation and active clinical research continue to expand the potential clinical utility of endoscopic ultrasound (EUS). It is the purpose of this article to review the role of upper gastrointestinal endoscopy, endoscopic ultrasound, endoscopic retrograde cholangiopancreatography (ERCP), and laparoscopy in hepatology.

Upper gastrointestinal endoscopy

Oesophageal varices

Oesophageal varices are dilated collaterals in the lower oesophagus that interconnect portal and systemic circulation in patients with portal hypertension. Endoscopically, oesophageal varices are usually unmistakable and appear as irregular, serpiginous, bluish structures running longitudinally in the submucosa of the oesophageal wall (Fig 1). Occasionally, it can be difficult to differentiate small varices from oesophageal folds and EUS can be helpful. The presence of varices in patients with cirrhosis is also an independent risk factor for survival.[1]

The appearance of oesophageal varices is not diagnostic of the cause of portal hypertension and does not allow differentiation between portal hypertension secondary to cirrhosis, pre-sinusoidal hypertension, or portal or splenic vein thrombosis. The majority of patients with cirrhosis develop varices and approximately one third bleed at some point.[2] The mortality from this first bleed is approximately 30% to 50% and two thirds of these patients die within one year.[3] With this high mortality associated with bleeding, prophylactic measures have been vigorously pursued. To this end, prophylactic shunt surgery and sclerotherapy have not been shown to be of benefit and banding ligation is as yet untested.

As only one third of patients will bleed, the accurate targeting of those at risk is important. The most popular model used is that devised by the North Italian Endoscopic Club for the Study and Treatment of Oesophageal Varices.[4] The severity of the underlying liver disease, the presence or absence of red markings on the varices, and the size of the varices are the most important risk factors for bleeding.

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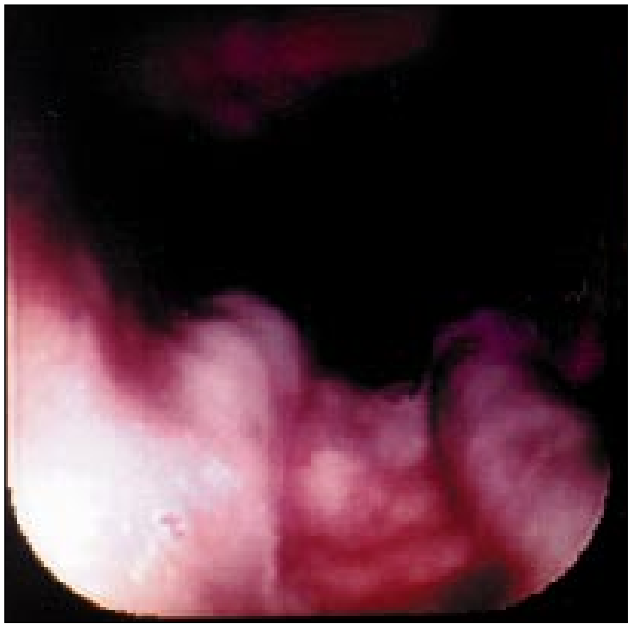

Fig 1. Oesophageal varices identified endoscopically

Emergency endoscopy is the key investigation in the management of oesophageal variceal bleeding. It serves to confirm that the bleeding is from the oesophageal varices and to exclude other causes of upper gastrointestinal bleeding. Emergency endoscopic sclerotherapy[5-8] stops active bleeding effectively and prevents early re-bleeding. Long term treatment with repeat sessions of sclerotherapy also significantly decreases re-bleeding and there are some reports that it leads to improved survival.[9-12] The sclerosants most commonly employed include sodium morrhuate (5% solution), sodium tetradecylsulfate (STS, 1% or 3% solution), ethanolamine oleate and polidocanol. All of these may be injected into or around the oesophageal varices with the exception of polidocanol, which should not be injected intravenously and is usually injected into the paravariceal tissue. However, re-bleeding is still common in patients receiving sclerotherapy and complications can arise after it. These include retrosternal pain, fever, oesophageal ulceration, sympathetic pleural effusion, oesophageal perforation, mediastinitis, and venous embolization of sclerosant, or infection. Surgery[11,12] or transjugular intrahepatic portosystemic shunts (TIPS)[13] should be considered if more than two injection sessions are required to stop the bleeding.

Endoscopic ligation was developed in an attempt to provide an endoscopic therapy that would be at least as effective as sclerotherapy but would have fewer complications.[14-16] It involves placing pre-stretched rubber bands on variceal columns, using a delivery device mounted at the end of an endoscope. The oesophageal mucosa and submucosa, containing varices, are ensnared, causing subsequent strangulation, sloughing, and eventual fibrosis, and resulting in the obliteration of the varices.[14-16] Randomised trials comparing endoscopic variceal ligation with sclerotherapy suggest that ligation is at least as effective as sclerotherapy.[1 7-19] In a recent meta-analysis, endoscopic ligation, when compared with endoscopic sclerotherapy, was shown to have a lower re-bleeding rate, mortality rate, rate of death due to bleeding, and oesophageal stricture rate.[20] The use of intravenous octreotide with endoscopic ligation might further decrease the re-bleeding rate.[21] The major disadvantage of endoscopic ligation is the need for an overtube, which decreases the endoscopic field of view and may allow pooling of blood. These can be partially circumvented by using newly modified banding devices such as the multiple band ligator (Microvasive, Boston Scientific Corporation, Watertown, Ma, US) [Fig 2].

Gastric varices

Gastric varices are submucosal structures that can have a mottled appearance or even resemble clusters of grapes (Fig 3).[22] The bluish colour that is characteristic of oesophageal varices is usually absent in the stomach. Gastric varices may be confused with enlarged folds, except when they run perpendicularly to the axes of the folds. Isolated gastric varices may develop as a consequence of pancreatic disorders that obstruct the splenic vein. Much confusion about the grading and classification of gastric varices exists because of the multiplicity of schemes that have been suggested.[23] The most commonly used classification recognises three

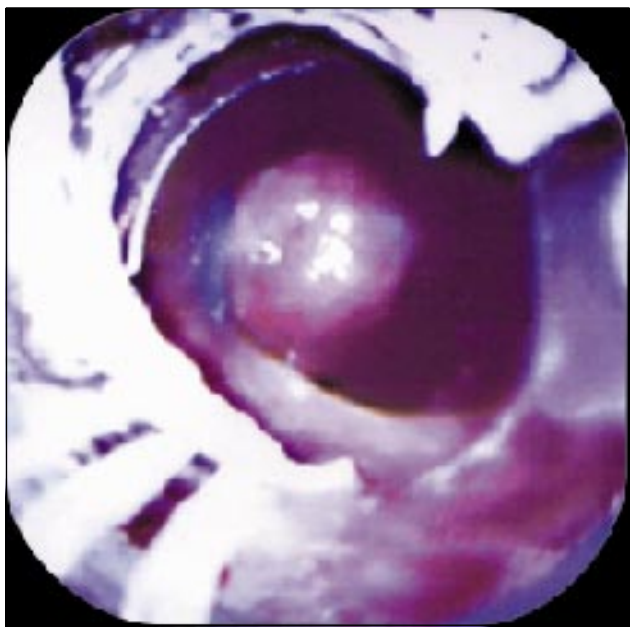

Fig 2. Application of multiple band ligator to oesophageal varices

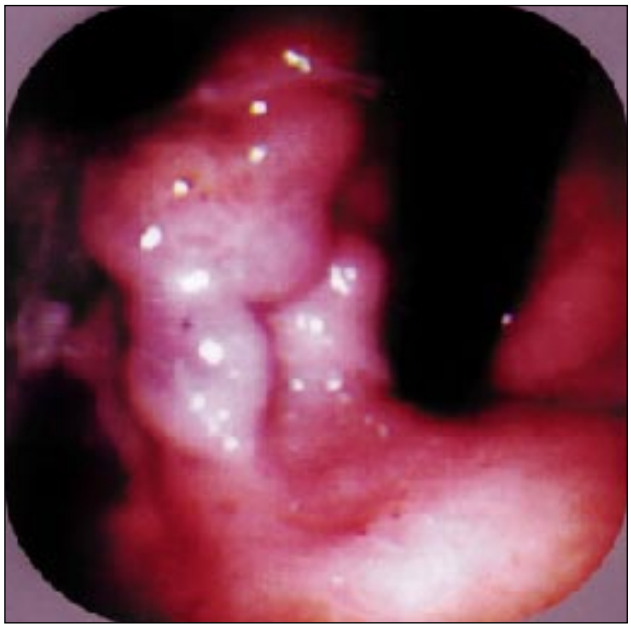

Fig 3. Grape-like bunches of gastric varices at the cardia

types of gastric varices. These include possible varices where there is doubt regarding their differentiation from folds, cardial oesophageal varices or cardial varices in continuity with oesophageal varices, and true gastric varices that are localised in the fundus and/or the cardial area and may extend to the body.

The importance of red colour signs on gastric varices is controversial. The disagreement is over whether these colour signs are related to the varix or to the overlying portal hypertensive gastropathy. Some reports have previously suggested that red spots on gastric varices are risk factors for haemorrhage.[24]

The rationale for devising a classification was to offer guidance on treatment. Type 1 varices should respond successfully to standard sclerotherapy or banding. Type 2 varices require either specialist endoscopic techniques such as the application of tissue adhesive (e.g. n-butyl-2-cyanoacrylate) [Histocryl], thrombin injection, decompression by surgery, or performance of TIPS. Gastric varices are believed to be much less common than oesophageal varices; the reported incidence of gastric varices varies from 6% to 100%.[25] Approximately 75% of gastric varices occur along the lesser curve of the stomach and only 9% are localised to the fundus.[26]

Ectopic varices

The prevalence of ectopic varices in cirrhotic patients ranges from 1% to 5%. Ectopic varices are predominantly located in the digestive tract.[27] Much less frequently,

Table. Distribution of bleeding varices as reported in the literature[27]

Sites of ectopic varices	Percentage of total
Duodenum	17
Jejunum and ileum	18
Colon	15
Rectum	9
Ileostomy or colostomy site	27
Peritoneum	10
Common bile duct and gallbladder	2
Vagina	<1
Bladder	<1

they occur in the peritoneum, biliary tract, vagina, and the bladder (Table). These often have a grape-like appearance. Sometimes, they appear as tumours and if biopsied, could lead to fatal torrential bleeding. In patients who have had an ileostomy or colostomy and have portal hypertension, ectopic varices might develop near the stoma site due to the presence of a porto-systemic connection.[28] Such varices can easily be inspected by the gentle passage of a narrow endoscope a few centimetres into the stoma.

Previous abdominal surgery is also a risk factor for the development of ectopic varices as the adhesions act as variceal conduits. With enteroscopy, jejunal and ileal varices can now be diagnosed directly rather than by angiographic studies. The incidence of colonic varices is unaffected by the type of portal hypertension

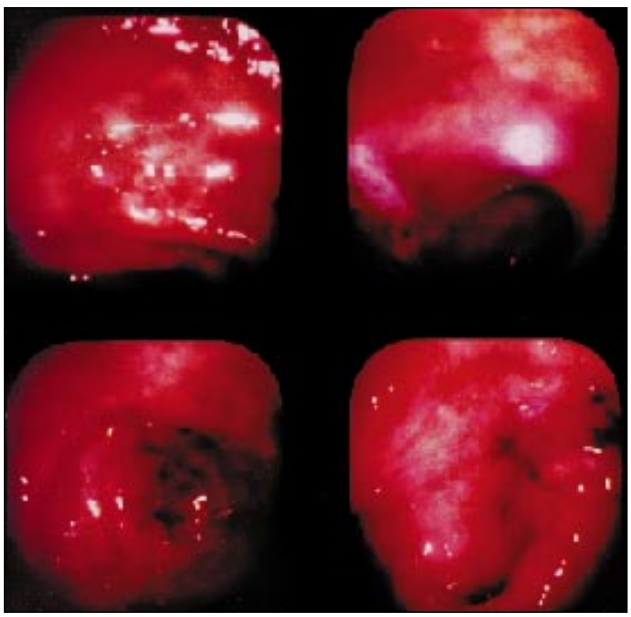

Fig 4. Acute gastroduodenitis after transcatheter oily chemoembolization

but they are more common in patients who have had previous surgery. Differentiation between varices and tumours is more of a problem in the colon because tumours are not uncommon here. In patients with suspected portal hypertension, any suspicious lesion without definitive evidence of malignancy should only be biopsied following angiographic studies to exclude varices.

Rectal varices are true variceal vessels that occur in the walls of the rectum above the anus. Bleeding rectal varices are uncommon but can on occasion be exsanguinating.[29] Either rigid or flexible sigmoidoscopy allows the diagnosis to be made with ease and it should be remembered that balloon tamponade in the rectum is as effective as it is in the oesophagus.

Portal hypertensive enteropathy

Portal hypertension affects the gastrointestinal tract and it may lead to portal hypertensive gastropathy (PHG) and portal hypertensive colopathy. The former condition was first described by Osler[30] as chronic gastritis associated with cirrhosis and later redefined by McCormack[31] as a vascular disorder rather than inflammatory condition. It is the cause of chronic anaemia in some cirrhotic patients and can be classified into two main types.[23] Type I typically has a mosaic-like pattern of small polygonal areas that slightly protrude in the centre surrounded by a whitish or yellow depressed border. The mosaic-like pattern can be mild with a diffusely pink areola, moderate with a flat red spot in the centre of the pink areola or severe, with a diffusely red areola. Type II produces mucosal red marks. These can occur in isolation or in association with the mosaic-like pattern.They are red lesions of variable diameter and are either flat or slightly protruding into the lumen of the stomach. The lesions can be diffuse, discrete, or confluent. Black or brown spots are not diagnostic of PHG, but are indicative of recent bleeding. Type I is the more common of the two types and is unlikely to be associated with gastrointestinal haemorrhage and can occur in the absence of portal hypertension, particularly the mild form. Type II is much less common and can cause chronic intestinal blood loss. It appears to be much more specific as a marker of PHG.

The pathogenesis of PHG is unclear, although the presence of portal hypertension is important.[32] It is not the only factor, however, as there is no relationship between the degree of portal pressure and the presence or absence of PHG.[33] Portocaval shunt surgery cures PHG but propanolol has been shown to be highly effective in controlling bleeding from this condition and should now be considered the treatment of choice.[34] Other factors, such as the presence of *Helicobacter pylori* infection,[35] do not appear to be important.

Portal hypertensive colopathy is present in approximately 70% of patients with portal hypertension. Colonoscopy reveals vascular ectasias, irregularity, dilatation, solitary red spots, diffuse red spots, and haemorrhoids. Patients with portal hypertensive colopathy more frequently have oesophageal varices and PHG.[34]

Post-transcatheter oily chemoembolization gastroduodenitis

Transcatheter oily chemoembolization (TOCE) has been used to treat hepatocellular carcinoma (HCC).[36] In a recent prospective study, the effect of TOCE on the gastroduodenal microcirculation and the relationship between pre-TOCE gastroduodenal microcirculation and post-TOCE acute gastroduodenitis was examined. Acute gastroduodenitis was defined endoscopically as mucosal erosions with or without haemorrhages (Fig 4). Thirty-six per cent of patients receiving TOCE were found to develop acute gastroduodenitis. This was associated with a lower pre-TOCE mucosal oxygen saturation level in the antrum and the first part of the duodenum.[37]

Endoscopic ultrasound

Endoscopic ultrasound is useful for detecting varices. Varices are imaged within the submucosa and in the perioesophageal or perigastric soft tissue.[38] In the oesophagus, EUS does not appear to be superior to endoscopy in the detection of oesophageal varices. On the other hand, EUS is invaluable in differentiating gastric varices from submucosal tumours and prominent gastric folds. For submucosal malignancy such as linitis plastica or infiltrating lymphoma, there is wide infiltration and distortion of the third and fourth mucosal layers, whereas gastric mucosal folds are limited to the mucosal layers. In addition, the role of EUS in determining whether or not varices are obliterated is under evaluation.

Endoscopic ultrasound has been shown to be at least as sensitive and specific for the diagnosis of choledocholithiasis as endoscopic retrograde cholangiography (ERCP)[39] and EUS is a very safe procedure with a complication rate of approximately 0.05%. The major complication is perforation, which is related to the relatively large diameter and stiffness of the endoscope used. The greatest risk of perforation exists when an ultrasound endoscope is passed through a malignant oesophageal stricture

in an attempt to achieve accurate staging. In addition to perforation, heavy bleeding after EUS has been reported.

Endoscopic retrograde cholangiopancreatography

Endoscopic retrograde cholangiopancreatography plays a major role in the diagnosis of recurrent pyogenic cholangitis (RPC).[40] This is a condition in which there is a primary bacterial cholangitis, characterised clinically by recurrent attacks of fever, abdominal pain, and jaundice.[41] The extrahepatic and intrahepatic ducts frequently show dilatation and stricture, and may contain calculi, debris (mainly bile pigments, shed epithelial cells, and mixed exudates), and sometimes pus.[42] Endoscopic sphincterotomy is a safe and effective form of treatment in patients with RPC whose calculi are confined to the common bile duct.[43] Endoscopic retrograde cholangiopancreato-graphy is invaluable in diagnosing biliary tract obstruction that may aggravate underlying liver disease. Therapeutic procedures to relieve biliary obstruction, such as endoscopic sphincterotomy, insertion of nasobiliary catheters, stenting, stone extraction, and balloon dilatation of biliary stricture can all be performed endoscopically.[44] Endoscopic retrograde cholangiopancreatography is also important in diagnosing primary sclerosing cholangitis. This condition gives rise to diffuse multifocal short strictures with patchy distribution throughout the liver and the large bile ducts. There is usually some degree of focal dilatation, which gives a characteristic beaded appearance. In addition, there might be slight irregularities in the walls of the intrahepatic ducts. Primary biliary cirrhosis may cause cholangiographic appearances similar to those of primary sclerosing cholangitis. However, the stricture is less pronounced and is more peripheral in location and the extrahepatic ducts are usually not affected. In patients with cirrhosis, the radiographic appearance is often abnormal with a wide spectrum of ductal abnormalities that are not diagnostic of the underlying cause of cirrhosis and are relatively non-specific.

Laparoscopy

Laparoscopy involves the creation of a pneumo-peritoneum via a small incision through the abdominal wall, followed by the insertion of a fibre-optic laparoscope into the peritoneal cavity. This procedure can be performed safely under local anaesthesia and sedation.[45] It is a well tolerated procedure that can be readily performed in the endoscopy suite and allows direct visualisation of the peritoneal cavity and its contents. Biopsies can be performed using a Silverman or Trucut needle.

The use of laparoscopy in the evaluation of liver disease has recently gained popularity in European countries and in Japan. In the investigation of patients with chronic liver disease, it is more accurate than histology alone in staging the severity of disease and adds valuable prognostic information. In patients with focal liver disease, it has a high degree of sensitivity and this is likely to increase with technological advances, such as laparoscopy ultrasound.[46-48] Recently, its application has been expanded by the introduction of a fibre-optic laparoscope that has a diameter of only 2 mm.

Complications and contraindications

The complication rate of laparoscopy is low. Most complications are related to the biopsy rather than to the laparoscopic procedure. Overall, the mortality rate of diagnostic laparoscopy is between 0.02% and 0.05%, which is similar to that for a percutaneous liver biopsy. Laparoscopic complications, other than those related to the liver biopsy, include bleeding from the abdominal wall stab site, pneumo-omentum, surgical emphysema, and infection.

Contraindications can be divided into absolute and relative ones. Absolute contraindications include an uncooperative patient, abdominal wall sepsis, and gross coagulopathy. Relative ones comprise patients with marked obesity, a large ventral hernia, previous right upper quadrant surgery, poor cardiorespiratory function, or ascites.

Using laparoscopy in hepatology

Laparoscopy can be used in hepatology to stage chronic liver disease, diagnose non-cirrhotic liver disease, identify focal lesions, and to diagnose peritoneal disease. Diagnostic laparoscopy is useful in identifying and providing prognostic information of cirrhosis. Histological examination fails or is inconclusive in diagnosing cirrhosis in approximately 10% of cirrhotic patients, principally because the small biopsy size may miss diseased areas in patients with macronodular cirrhosis. Laparoscopy, on the other hand, approaches 100% sensitivity and 90% specificity in the diagnosis of cirrhosis.[49]

Macronodular changes are easier to identify than micronodular changes in which disruption of light reflex may be a valuable clue. Close inspection of the anterior edge is important as early cirrhosis may be revealed by irregularity of the normally smooth convex

edge. In addition, left lobe hypertrophy compared with the right, the completeness of the regenerating nodule, the presence of splenomegaly, and the development of lymphatic cysts on the liver surface are all predictors of reduced survival.[50] It is hoped that further progress will be made in this area, perhaps by means of image analysis, which might allow volume measurements to be calculated.

The accuracy of laparoscopy in the diagnosis of non-cirrhotic liver disease has been studied less, but is probably as accurate as histology. In a recent study, laparoscopy was compared with histology and showed a sensitivity and specificity for detecting fatty change of 96.4% and 100%, for fibrosis of 100% and 95%, and for in-flammatory activity of 94% and 95%, respectively.[51]

One of the great advantages of modern laparoscopy is its high level of resolution. Lesions of only 1 mm or 2 mm can be readily recognised and this represents a distinct advantage over both ultrasonography and computed tomography (CT) scanning. Malignancy has been identified by laparoscopy in up to 30% to 50% of patients thought to have hepatic or peritoneal malignancy but who have normal ultrasound or CT scans. In addition, laparoscopy can help to confirm the presence of focal lesions identified by conventional ultrasound or CT scans. Another advantage of laparoscopy in identifying focal lesions is the recognition of features not apparent using other techniques.[52] For example, laparoscopy can locate yellow nodules undergoing fatty change in cirrhotic livers, an early indicator of HCC.

Laparoscopy can identify novel features in the investigation of non-malignant disease. It has been reported that dark, red, patchy markings on the liver surface of patients with chronic active hepatitis B signifies reduced hepatitis B replication.[53] Laparoscopy ultrasound has also improved the detection rate of occult liver lesions, cysts, tumours, various benign focal hypoplasias, and granulomata.[54] As advances in technology allow the diameter of instruments to be reduced, it is likely that laparoscopy ultrasound will be performed without general anaesthesia. In addition to the detection of small lesions, laparoscopy is invaluable in staging lymphoma.[55,56] Two other situations in which laparoscopy has been reported as useful is in the investigation of ascites and in patients with pyrexia of unknown origin. In these situations, advances in other non-invasive diagnostic tools have reduced the requirements for laparoscopy. However, when the cause of ascites or pyrexia of unknown origin is unclear, despite extensive investigation, laparoscopy is still a valuable diagnostic procedure.

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References

1. Lebrec D, De Fleury P, Rueff B, Nahum H, Benhamou JP. Portal hypertension, size of esophageal varices and risk of gastrointestinal bleeding in alcoholic cirrhosis. Gastroenterology 1980;79:1139-44.
2. Christensen E, Fauerholdt L, Schlichting P, Juhl E, Poulsen H, Tygstrup N. Aspects of the natural history of gastrointestinal bleeding in cirrhosis and the effect of prednisone. Gastroenterology 1981;81(5):944-52.
3. Rigo GP, Merighi A, Chahin NJ, et al. A prospective study of the ability of three endoscopic classifications to predict hemorrhage from esophageal varices. Gastrointest Endosc 1992;38:425-9.
4. The North Italian Endoscopic Club for the Study and Treatment of Esophageal Varices. Prediction of first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices. N Engl J Med 1988;15:983-9.
5. Larson AW, Cohen H, Zweiban B, et al. Acute esophageal variceal sclerotherapy. Results of a prospective randomized controlled trial. JAMA 1986;255:497-500.
6. Moreto M, Zaballa M, Bernal A, Ibanez S, Ojembarrena E, Rodriguez A. A randomized trial of tamponade or sclerotherapy as immediate treatment of bleeding esophageal varices. Surg Gynecol Obstet 1988;167:331-4.
7. Paquet KJ, Feussner H. Endoscopic sclerosis and esophageal balloon tamponade in acute hemorrhage from esophagogastric varices: a prospective controlled randomized trial. Hepatology 1985;5:580-3.
8. Barsoum MS, Bolous Fl, El-Rooby AA, Rizk-Allah MA, Ibrahim AS. Tamponade and injection sclerotherapy in the management of bleeding esophageal varices. Br J Surg 1982; 69:76-8.
9. Westaby D, Macdougall BR, Williams R. Improved survival following injection sclerotherapy for esophageal varices: final analysis of a controlled trial. Hepatology 1985;5:827-30.
10. Pagliaro L, Burroughs AK, Sorensen TL, et al. Therapeutic controversies and randomised controlled trials (RCTs): prevention of bleeding and rebleeding in cirrhosis. Gastroenterol Int 1989;2:71-84.
11. McCormick PA, Kaye GL, Greenslade L, et al. Esophageal staple transection as a salvage procedure after failure of acute injection sclerotherapy. Hepatology 1992;15:403-6.
12. Henderson JM, Kutner MH, Millikan WJ Jr, et al. Endoscopic variceal sclerosis compared with distal splenorenal shunt to prevent recurrent variceal bleeding in cirrhosis. A prospective, randomized trial. Ann Intern Med 1990;112:262-9.
13. Zemel G, Katzen BT, Becker GJ, Benenati JF, Sallee DS. Percutaneous transjugular portosystemic shunt. JAMA 1991; 266:390-3.
14. Laine L. Rolling review: upper gastrointestinal bleeding. Aliment Pharmacol Ther 1993;7:207-32.
15. Van Stiegmann G, Cambre T, Sun JH. A new endoscopic elastic band ligating device. Gastrointest Endosc 1986;32:230-3.
16. Stiegmann GV, Goff JS, Sun JH, Davis D, Bozdech J. Endoscopic variceal ligation: an alternative to sclerotherapy. Gastrointest Endosc 1989;35:431-4.

17. Stiegmann GV, Goff JS, Michaletz-Onody PA, et al. Endoscopic sclerotherapy as compared with endoscopic ligation for bleeding esophageal varices. N Engl J Med 1992;326:1527-32.
18. Laine L, el-Newihi HM, Migikovsky B, Sloane R, Garcia F. Endoscopic ligation compared with sclerotherapy for the treatment of bleeding esophageal varices. Ann Intern Med 1993;119:1-7.
19. Gimson AE, Ramage JK, Panos MZ, et al. Randomised trial of variceal banding ligation versus injection sclerotherapy for bleeding oesophageal varices. Lancet 1993;342:391-4.
20. Laine L, Cook D. Endoscopic ligation compared with sclerotherapy for treatment of esophageal variceal bleeding: a meta-analysis. Ann Intern Med 1995;123:280-7.
21. Sung JJ, Chung SC, Yung MY, et al. Prospective randomised study of effect of octreotide on rebleeding from oesophageal varices after endoscopic ligation. Lancet 1995;346:1666-9.
22. Mathur SK, Dalvi AN, Someshwar V, Supe AN, Ramakantan R. Endoscopic and radiological appraisal of gastric varices. Br J Surg 1990;77:432-5.
23. Spina GP, Arcidiacono R, Bosch J, et al. Gastric endoscopic features in portal hypertension: final report of a consensus conference, Milan, Italy, September 19, 1992. J Hepatol 1994;21(3):461-7.
24. Hashizume M, Kitano S, Yamaga H, Koyanagi N, Sugimachi K. Endoscopic classification of gastric varices. Gastrointest Endosc 1990;36:276-80.
25. Clark AW, Macdougall BR, Westaby D, et al. Prospective controlled trial of injection sclerotherapy in patient with cirrhosis and recent variceal haemorrhage. Lancet 1980;2: 552-4.
26. Mathur SK, Dalvi AN, Someshwar V, Supe AN, Ramakantan R. Endoscopic and radiological appraisal of gastric varices. Br J Surg 1990;77:432-5.
27. Lebrec D, Benhamou JP. Ectopic varices in portal hypertension. Clin Gastroenterol 1985;14:105-21.
28. Ricci RL, Lee KR, Greenberger NJ. Chronic gastrointestinal bleeding from ileal varices after total protocolectomy for ulcerative colitis: correction by mesocaval shunt. Gastroenterology 1980;78:1053-8.
29. Herman BE, Baum S, Denobile J, Volpe RJ. Massive bleeding from rectal varices. Am J Gastroenterol 1993;88(6):939-42.
30. Pentland YJ, editor. Principles and practice of medicine. 3rd ed. Edinburgh: Lipton, 1898.
31. McCormack TT, Sims J, Eyre-Brook I, et al. Gastric lesions in portal hypertension: inflammatory gastritis or congestive gastropathy? Gut 1985;26:1226-32.
32. Panes J, Bordas JM, Pique JM, et al. Increased gastric mucosal perfusion in cirrhotic patients with portal hypertensive gastropathy. Gastroenterology 1992;103:1875-82.
33. Brinch K, Moller S, Henriksen JH, Becker PU. Portal hypertensive gastropathy. Ugeskr Laeger 1995;157(46):6421-5.
34. Yamakado S, Kanazawa H, Kobayashi M. Portal hypertensive colopathy: endoscopic findings and the relation to portal pressure. Intern Med 1995;34:153-7.
35. Parikh SS, Desai SB, Prabhu SR, et al. Congestive gastropathy: factors influencing development, endoscopic features, *Helicobacter pylori* infection, and microvessel changes. Am J Gastroenterol 1994;89:1036-42.
36. Ngan H, Lai CL, Fan ST, Lai EC, Yuen WK, Tso WK. Treatment of inoperable hepatocellular carcinoma by transcatheter arterial chemoembolization using an emulsion of cisplatin in iodized oil and gelfoam. Clin Radiol 1993;47:315-20.
37. Lau GK, Hui WM, S Hu, Ngan H, Lam SK. Occurrence of acute gastroduodenitis after transcatheter oily chemo- embolization (TOCE): role of gastroduodenal microcirculation [abstract]. Gastroenterology 1996;110(4):1246A.
38. Yasuda K, Cho E, Nakajima M, Kawai K. Diagnosis of submucosal lesions of upper gastrointestinal tract by endoscopic ultrasonography. Gastrointest Endosc 1990;36(2 Suppl):17S-20S.
39. Prat F, Amouyal G, Amouyal P, et al. Prospective controlled study of endoscopic ultrasonography and endoscopic retrograde cholangiography in patients with suspected common-bile duct lithiasis. Lancet 1996;347:75-9.
40. Lam SK, Wong KP, Chan PK, Ngan H, Ong GB. Recurrent pyogenic cholangitis: a study by endoscopic retrograde cholangiography. Gastroenterology 1978;74:1196-203.
41. Cook J, Hou PC, Ho HC, et al. Recurrent pyogenic cholangitis. Br J Surg 1954;42:188-203.
42. Ong GB. A study of recurrent pyogenic cholangitis. Arch Surg 1962;84:199-225.
43. Lam SK. A study of endoscopic sphincterotomy in recurrent pyogenic cholangitis. Br J Surg 1984;71:262-6.
44. Siegel JH. Endoscopic retrograde cholangio-pancreatography: technique, diagnosis, and therapy. New York: Raven Press, 1992.
45. Plevris JN, Hayes PC. Laparoscopy in the investigation of liver disease. Hospital Update 1994;20:128-38.
46. Machi J, Schwartz JH, Zaren HA, Noritomi T, Sigel B. Technique of laparoscopic ultrasound examination of the liver and pancreas. Surg Endosc 1996;10(6):684-9.
47. Van Stiegmann G, McIntyre RC Jr. Laparoscopic ultrasonography. Has it extended the horizon? Gastrointest Endosc Clin North Am 1995;5(4):869-78.
48. Lirici MM, Caratozzolo M, Urbano V, Angelini L. Laparoscopic ultrasonography: limits and potential of present technologies. Endosc Surg Allied Technol 1994;2(2):127-33.
49. Herrera JL, Brewer TG, Peura DA. Diagnostic laparoscopy: a prospective review of 100 cases. Am J Gastroenterol 1989; 84:1051-4.
50. Tameda Y, Yoshizawa N, Takase K, Nakano T, Kosaka Y. Prognostic value of peritoneoscopic findings in cirrhosis of the liver. Gastrointest Endosc 1990;36:34-8.
51. Jalan R, Harrison DJ, Dillon JF, Elton RA, Finlayson ND, Hayes PC. Laparoscopy and histology in the diagnosis of chronic liver disease [abstract]. J Pathol 1994;173:184A.
52. Kameda Y, Shinji Y. Early detection of hepatocellular carcinoma by laparoscopy: yellow nodules as diagnostic indicators. Gastrointest Endosc 1992;38:554-9.
53. Michitaka K, Onji M, Horiike N, Nadano S, Ohta Y. Hepatitis B virus replication in dark reddish patchy markings appearing on the hepatic surface. Gastrointest Endosc 1990;36:15-8.
54. Fukuda M, Hirata K, Mima S. Preliminary evaluation of sonolaparoscopy in the diagnosis of liver disease. In: Boyce HW, Henning H, editors. Endoscopy. Stuttgart: Georg Thieme Verlag, 1992:701-8.
55. Devita VT Jr, Bagley CM Jr, Goodell B, O'Kieffe DA, Trujillo NP. Peritoneoscopy in the staging of Hodgkin's disease. Cancer Res 1971;31:1746-50.
56. Coleman M, Lightdale CJ, Vinciguerra VP, et al. Peritoneoscopy in Hodgkin disease. Confirmation of results by laparotomy. JAMA 1976;236:2634-6.